Multidisciplinary Spine Care

Carl E. Noe
Editor

Springer

Multidisciplinary Spine Care

Carl E. Noe
Editor

Multidisciplinary Spine Care

Springer

Editor
Carl E. Noe
Medical Center, Anesthesiology
The University of Texas Southwestern Med
Dallas, TX, USA

ISBN 978-3-031-04989-7 ISBN 978-3-031-04990-3 (eBook)
https://doi.org/10.1007/978-3-031-04990-3

© The Editor(s) (if applicable) and The Author(s), under exclusive license to Springer Nature Switzerland AG 2022
This work is subject to copyright. All rights are solely and exclusively licensed by the Publisher, whether the whole or part of the material is concerned, specifically the rights of translation, reprinting, reuse of illustrations, recitation, broadcasting, reproduction on microfilms or in any other physical way, and transmission or information storage and retrieval, electronic adaptation, computer software, or by similar or dissimilar methodology now known or hereafter developed.
The use of general descriptive names, registered names, trademarks, service marks, etc. in this publication does not imply, even in the absence of a specific statement, that such names are exempt from the relevant protective laws and regulations and therefore free for general use.
The publisher, the authors and the editors are safe to assume that the advice and information in this book are believed to be true and accurate at the date of publication. Neither the publisher nor the authors or the editors give a warranty, expressed or implied, with respect to the material contained herein or for any errors or omissions that may have been made. The publisher remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

This Springer imprint is published by the registered company Springer Nature Switzerland AG
The registered company address is: Gewerbestrasse 11, 6330 Cham, Switzerland

Introduction

Multidisciplinary spine care is organized into six sections: (1) non-operative care, (2) spine injections and procedures, (3) perioperative care, (4) operative care, (5) pediatric care, and (6) special topics. Our operative team includes neurosurgeons, orthopedic surgeons, and physician assistants. The non-operative team includes pain management specialists, physical medicine and rehabilitation physicians, nurses, and nurse advanced practice practitioners. The perioperative team includes psychologists, internists, anesthesiologists, and intensivists. Our interdisciplinary pain program team includes pain management physicians, psychologists, physical therapists, and case managers. This book presents multiple aspects of spine care from the perspective of different disciplines. Each chapter has been written by a clinician whose active practice involves the topic of their chapter. The authors hope the reader will find the book to be clinically relevant and easy to read. The advantages of having a multidisciplinary spine program under one roof include closer collaboration between operative and non-operative care physicians, improved communication, research, education, and training. The importance of these strengths have only been highlighted during the pandemic.

Chronic back pain continues to be a challenging problem, and the biopsychosocial approach to patients with chronic back pain using an interdisciplinary pain rehabilitation program is an important treatment option in a comprehensive spine program. Interdisciplinary rehabilitation programs have long been recognized as a safe and cost-effective treatment for chronic back pain [1–3]. These programs provide cognitive behavioral therapy and physical therapy in addition to medical treatment. Gatchel has shown that acute back pain is also effectively treated with an interdisciplinary approach in patients who are at high risk of developing chronic pain [4–7]. High-risk patients who are treated with an early intervention have similar outcomes to low-risk patients and much less pain, disability, opioid use, and healthcare utilization compared to high-risk patients treated with usual care. Interdisciplinary treatment is also an alternative to lumbar fusion surgery [8–11]. Multiple randomized trials have shown patients have similar long-term functional status after either interdisciplinary treatment or lumbar fusion. Neck pain is similarly responsive to interdisciplinary treatment [12–18]. Several trials have shown that interdisciplinary treatment is more effective than usual care for neck pain. Interdisciplinary programs are also an effective alternative to long-term opioid therapy [19, 20]. Many patients are able to discontinue or reduce opioids without

adverse outcomes. Reducing long-term opioid use is likely safer for patients and reduces the risk of diversion, substance use disorder, and overdose. For these clinical needs, interdisciplinary programs, with group education, medical, psychological, and physiotherapy, are an important capability of multidisciplinary spine care.

References

1. Salathé CR, Melloh M, Crawford R, Scherrer S, Boos N, Elfering A. Treatment efficacy, clinical utility, and cost-effectiveness of multidisciplinary biopsychosocial rehabilitation treatments for persistent low back pain: a systematic review. Glob Spine J. 2018;8(8):872–86. https://doi.org/10.1177/2192568218765483. Epub 2018 Apr 19. PMID: 30560041; PMCID: PMC6293434.
2. AlMazrou SH, Elliott RA, Knaggs RD, AlAujan SS. Cost-effectiveness of pain management services for chronic low back pain: a systematic review of published studies. BMC Health Serv Res. 2020;20(1):194. Published 2020 Mar 12. https://doi.org/10.1186/s12913-020-5013-1.
3. Kamper SJ, Apeldoorn AT, Chiarotto A, Smeets RJEM, Ostelo RWJG, Guzman J et al. Multidisciplinary biopsychosocial rehabilitation for chronic low back pain: cochrane systematic review and meta-analysis. BMJ. 2015;350:h444. https://doi.org/10.1136/bmj.h444.
4. Gatchel RJ, Polatin PB, Noe C, Gardea M, Pulliam C, Thompson J. Treatment- and cost-effectiveness of early intervention for acute low-back pain patients: a one-year prospective study. J Occup Rehabil. 2003;13(1):1–9. https://doi.org/10.1023/a:1021823505774. PMID: 12611026.
5. Whitfill T, Haggard R, Bierner SM, et al. Early intervention options for acute low back pain patients: a randomized clinical trial with one-year follow-up outcomes. J Occup Rehabil. 2010;20:256–63. https://doi.org/10.1007/s10926-010-9238-4.
6. Marin TJ, Van Eerd D, Irvin E, Couban R, Koes BW, Malmivaara A, van Tulder MW, Kamper SJ. Multidisciplinary biopsychosocial rehabilitation for subacute low back pain. Cochrane Database Syst Rev. 2017;6(6):CD002193. https://doi.org/10.1002/14651858.CD002193.pub2. PMID: 28656659; PMCID: PMC6481490.
7. Rogerson MD, Gatchel RJ, Bierner SM. A cost utility analysis of interdisciplinary early intervention versus treatment as usual for high-risk acute low back pain patients. Pain Pract. 2010;10(5):382–95. https://doi.org/10.1111/j.1533-2500.2009.00344.x. PMID: 20384967.
8. Brox JI, Nygaard ØP, Holm I, Keller A, Ingebrigtsen T, Reikerås O. Four-year follow-up of surgical versus non-surgical therapy for chronic low back pain. Ann Rheum Dis. 2010;69(9):1643–8. https://doi.org/10.1136/ard.2009.108902. Epub 2009 Jul 26. PMID: 19635718; PMCID: PMC2938881.
9. Fairbank J, Frost H, Wilson-MacDonald J, Yu LM, Barker K, Collins R; Spine Stabilisation Trial Group. Randomised controlled trial to compare surgical stabilisation of the lumbar spine with an intensive rehabilitation programme for patients with chronic low back pain: the MRC spine stabilisation trial. BMJ. 2005;330(7502):1233. https://doi.org/10.1136/bmj.38441.620417.8F. Epub 2005 May 23. Erratum in: BMJ. 2005;330(7506):1485. PMID: 15911537; PMCID: PMC558090.
10. Mannion AF, Brox JI, Fairbank JC. Comparison of spinal fusion and nonoperative treatment in patients with chronic low back pain: long-term follow-up of three randomized controlled trials. Spine J. 2013;13(11):1438–48. https://doi.org/10.1016/j.spinee.2013.06.101. Epub 2013 Nov 5. PMID: 24200413.
11. Saltychev M, Eskola M, Laimi K. Lumbar fusion compared with conservative treatment in patients with chronic low back pain: a meta-analysis. Int J Rehabil Res. 2014;37(1):2–8. https://doi.org/10.1097/MRR.0b013e328363ba4b. PMID: 23820296.
12. Persson LC, Lilja A. Pain, coping, emotional state and physical function in patients with chronic radicular neck pain. A comparison between patients treated with surgery, physiotherapy or

neck collar--a blinded, prospective randomized study. Disabil Rehabil. 2001;23(8):325–35. https://doi.org/10.1080/09638280010005567. PMID: 11374522.
13. Jensen IB, Bergström G, Ljungquist T, Bodin L. A 3-year follow-up of a multidisciplinary rehabilitation programme for back and neck pain. Pain. 2005;115(3):273–83. https://doi.org/10.1016/j.pain.2005.03.005. Epub 2005 Apr 19. PMID: 15911154.
14. Jensen IB, Busch H, Bodin L, Hagberg J, Nygren Å, Bergström G. Cost effectiveness of two rehabilitation programmes for neck and back pain patients: a seven year follow-up. Pain. 2009;142(3):202–8. https://doi.org/10.1016/j.pain.2008.12.015. Epub 2009 Feb 13. PMID: 19217717.
15. Bergström C, Jensen I, Hagberg J, Busch H, Bergström G. Effectiveness of different interventions using a psychosocial subgroup assignment in chronic neck and back pain patients: a 10-year follow-up. Disabil Rehabil. 2012;34(2):110–8. https://doi.org/10.3109/09638288.2011.607218. Epub 2011 Oct 11. PMID: 21988525.
16. Myhre K, Marchand GH, Leivseth G, Keller A, Bautz-Holter E, Sandvik L, Lau B, Røe C. The effect of work-focused rehabilitation among patients with neck and back pain: a randomized controlled trial. Spine (Phila Pa 1976). 2014;39(24):1999–2006. https://doi.org/10.1097/BRS.0000000000000610. PMID: 25271499.
17. Monticone M, Ambrosini E, Rocca B, Cazzaniga D, Liquori V, Pedrocchi A, Vernon H. Group-based multimodal exercises integrated with cognitive-behavioural therapy improve disability, pain and quality of life of subjects with chronic neck pain: a randomized controlled trial with one-year follow-up. Clin Rehabil. 2017;31(6):742–52. https://doi.org/10.1177/0269215516651979. Epub 2016 May 31. PMID: 27246516.
18. Moll LT, Jensen OK, Schiøttz-Christensen B, Stapelfeldt CM, Christiansen DH, Nielsen CV, Labriola M. Return to work in employees on sick leave due to neck or shoulder pain: a randomized clinical trial comparing multidisciplinary and brief intervention with one-year register-based follow-up. J Occup Rehabil. 2018;28(2):346–56. https://doi.org/10.1007/s10926-017-9727-9. PMID: 28836120; PMCID: PMC5978826.
19. Crisostomo RA, Schmidt JE, Hooten WM, Kerkvliet JL, Townsend CO, Bruce BK. Withdrawal of analgesic medication for chronic low-back pain patients: improvement in outcomes of multidisciplinary rehabilitation regardless of surgical history. Am J Phys Med Rehabil. 2008;87(7):527–36. https://doi.org/10.1097/PHM.0b013e31817c124f. PMID: 18574345.
20. Cunningham JL, Rome JD, Kerkvliet JL, Townsend CO. Reduction in medication costs for patients with chronic nonmalignant pain completing a pain rehabilitation program: a prospective analysis of admission, discharge, and 6-month follow-up medication costs. Pain Med. 2009;10(5):787–96. https://doi.org/10.1111/j.1526-4637.2009.00582.x. Epub 2009 Mar 19. PMID: 19302437.

Contents

1 Lifestyle Management of Spine Patient 1
Kavita Trivedi and Esther Yoon

2 Evaluation of a New Spine Patient 35
Kegan J. Cunniff and G. Sunny Sharma

3 Electromyography in the Spine Patient 57
Denizen Kocak and Thiru M. Annaswamy

4 Conventional and Advanced Imaging Evaluation of Spine 73
Girish Boraiah and Avneesh Chhabra

5 Spondyloarthritis 109
Nilofar Syed, Fatemah Ezzati, and Lesley Davila

6 Soft Tissue Pain 121
Robert S. Ackerman, Patrick B. Ahern, Biral T. Patel, and Carl E. Noe

7 Non Pharmacological Treatments 137
Renee Enriquez and Isabel Huang

8 Pharmacology 171
Daltry Dott

9 Interdisciplinary Pain Management 199
Richard C. Robinson

10 Spine Injections in the Management of Painful Spinal Conditions . . . 211
Stephanie Jones, Ivan N. Chew, and Judy Yang

11 Vertebral Compression Fractures 227
Ankit Patel and Brent Page

12 Epidural Lysis of Adhesions 251
Gabor Bela Racz and Gabor J. Racz

13 Pre-surgical Psychological Evaluation of Spine Surgery Candidates . 317
Jeffrey Dodds

14 Pre and Postoperative Spine Rehabilitation 329
Nasser Ayyad and Denizen Kocak

15 Preoperative Optimization and Intraoperative Enhanced Recovery Principles for Patients Undergoing Spine Surgery 355
Megan G. Maxwell, Kehinde O. Odedosu, and Bryan T. Romito

16 ICU Care for the Spine Patient 381
Jia W. Romito and Bryan T. Romito

17 Postoperative Care of the Spine Surgery Patient 399
Lori A. Tappen

18 The Neurosurgical Management of Pain 413
Eric Montgomery, Cody Wolfe, Jeffrey Traylor, Salah G. Aoun, and Mazin Al Tamimi

19 Endoscopic Spine Surgery 439
Omar Akbik, Peter Shin, and Mazin Al Tamimi

20 Minimally Invasive Spine Surgery 455
Carl Youssef, Salah G. Aoun, and Mazin Al Tamimi

21 Cervical Spine Problems 469
Michael Van Hal

22 Thoracic Degenerative Conditions 485
Ammar Adenwalla, Olatunde Badejo, Kylan Larsen, Nisha Reddy, Neha Mulpuri, and Shaleen Vira

23 Lumbar Spinal Stenosis 499
Carl Youssef, Sai Chilakapati, and Owoicho Adogwa

24 Spine Vascular Lesions of the Bone and the Epidural Space 507
James P. Caruso, Salah G. Aoun, and Kevin C. Morrill

25 Trauma 517
Douglas Dickson

26 Post Surgical Spinal Deformities 525
Sai Chilakapati, James P. Caruso, Zachary Johnson, and Owoicho Adogwa

27 Patient Reported Outcomes in Spine Surgery 541
Jose Marin Sanchez, Salah G. Aoun, Kristen Hall, and Carlos A. Bagley

28 Scoliosis in Children 549
Karl Rathjen

29 Anesthesia for Pediatric Spinal Deformity 667
Christopher Bryan McLeod

30 Regenerative Therapies 711
Nenna Nwazota and Amr Hegazi

31 Epidural Catheter Infusion for Post-operative Analgesia for Major Spine Surgery 723
Anthony Machi and Enas Kandil

32 Telemedicine Physical Examination for Spine 731
Daltry Dott, Jason Zafereo, Stephanie Jones, and Carl E. Noe

33 The Future of Spine Care 749
Kathleen R. Bell, Hunt Batjer, and Charles Whitten

Index 765

Contributors

Robert S. Ackerman Department of Anesthesiology and Pain Management, University of Texas Southwestern Medical Center, Dallas, TX, USA

Ammar Adenwalla Department of Orthopaedic Surgery, UT Southwestern Medical Center, Dallas, TX, USA

Owoicho Adogwa Department of Neurological Surgery, UT Southwestern Medical Center, Dallas, TX, USA

Patrick B. Ahern Department of Anesthesiology and Pain Management, University of Texas Southwestern Medical Center, Dallas, TX, USA

Omar Akbik Department of Neurological Surgery, UT Southwestern Medical Center, Dallas, TX, USA

Mazin Al Tamimi Department of Neurological Surgery and Spine Center, UT Southwestern Medical Center, Dallas, TX, USA

Thiru M. Annaswamy Department of Physical Medicine & Rehabilitation, VA North Texas Health Care System and UT Southwestern Medical Center, Dallas, TX, USA

Physical Medicine & Rehabilitation Service, VA North Texas Health Care System, Dallas, TX, USA

Salah G. Aoun Department of Neurological Surgery and Spine Center, UT Southwestern Medical Center, Dallas, TX, USA

Nasser Ayyad Department of Physical Medicine and Rehabilitation, UT Southwestern Medical Center Dallas Texas, Dallas, TX, USA

Olatunde Badejo Department of Orthopaedic Surgery, UT Southwestern Medical Center, Dallas, TX, USA

Carlos A. Bagley University of Texas Southwestern Medical Center, Department of Neurological Surgery, Dallas, TX, USA

University of Texas Southwestern Medical Center, Spine Center, Dallas, TX, USA

University of Texas Southwestern Medical Center, Department of Orthopedic Surgery, Dallas, TX, USA

Hunt Batjer University of Texas Southwestern Medical Center, Dallas, TX, USA

Kathleen R. Bell Department of Physical Medicine and Rehabilitation, University of Texas Southwestern Medical Center, Dallas, TX, USA

Girish Boraiah Dr. Chandramma Dayananda Sagar Institute of Medical Education & Research, Harohalli, Ramanagara, Karnataka, India

James P. Caruso The University of Texas Southwestern, Department of Neurological Surgery, Dallas, TX, USA

Department of Neurological Surgery, UT Southwestern Medical Center, Dallas, TX, USA

Avneesh Chhabra University of Texas at Southwestern Medical Center, Dallas, TX, USA

Johns Hopkins University, Walton Center of Neurosciences, Liverpool, UK

Sai Chilakapati Department of Neurological Surgery, UT Southwestern Medical Center, Dallas, TX, USA

Kegan J. Cunniff University of Texas Southwestern Medical Center, Dallas, TX, USA

Lesley Davila Division of Rheumatology, Department of Internal Medicine, UT Southwestern Medical Center, Dallas, TX, USA

Douglas Dickson Department of Orthopaedic Surgery, UT Southwestern Medical Center, Dallas, TX, USA

Jeffrey Dodds Division of Psychology, Department of Psychiatry, UT Southwestern Medical Center, Dallas, TX, USA

Daltry Dott Department of Anesthesiology and Pain Management, University of Texas Southwestern Medical Center, Dallas, TX, USA

Renee Enriquez University of Texas Southwestern, Dallas, TX, USA

Fatemah Ezzati Division of Rheumatology, Department of Internal Medicine, UT Southwestern Medical Center, Dallas, TX, USA

Kristen Hall University of Texas Southwestern Medical Center, Spine Center, Dallas, TX, USA

Amr Hegazi Department of Physical Medicine and Rehabilitation, UT Southwestern Medical Center, Dallas, TX, USA

Isabel Huang University of Texas Southwestern, Dallas, TX, USA

Zachary Johnson Department of Neurosurgery, UT Southwestern Medical School, Dallas, TX, USA

Stephanie Jones Department of Anesthesiology and Pain Management, University of Texas Southwestern Medical Center, Dallas, TX, USA

Ivan N. Chew Department of Anesthesiology and Pain management, University of Texas Southwestern Medical Center, Dallas, TX, USA

Judy Yang Department of Anesthesiology and Pain management, University of Texas Southwestern Medical Center, Dallas, TX, USA

Enas Kandil Department of Anesthesiology and Pain Management, University of Texas Southwestern Medical Center, Dallas, TX, USA

Denizen Kocak Department of Physical Medicine & Rehabilitation, VA North Texas Health Care System and UT Southwestern Medical Center, Dallas, TX, USA

Department of Physical Medicine and Rehabilitation, UT Southwestern Medical Center Dallas Texas, Dallas, TX, USA

Kylan Larsen Department of Orthopaedic Surgery, UT Southwestern Medical Center, Dallas, TX, USA

Anthony Machi Department of Anesthesiology and Pain Management, University of Texas Southwestern Medical Center, Dallas, TX, USA

Megan G. Maxwell University of Texas Southwestern Medical Center, Dallas, TX, USA

Christopher Bryan McLeod Scottish Rite for Children, Dallas, TX, USA

Eric Montgomery Department of Neurological Surgery and Spine Clinic, UT Southwestern Medical Center, Dallas, TX, USA

Kevin C. Morrill The University of Texas Southwestern, Department of Neurological Surgery, Dallas, TX, USA

Neha Mulpuri Department of Orthopaedic Surgery, UT Southwestern Medical Center, Dallas, TX, USA

Carl E. Noe Department of Anesthesiology and Pain Management, University of Texas Southwestern Medical Center, Dallas, TX, USA

Nenna Nwazota Department of Anesthesiology and Pain Management, UT Southwestern Medical Center, Dallas, TX, USA

Kehinde O. Odedosu University of Texas Southwestern Medical Center, Dallas, TX, USA

Parkland Health and Hospital Systems, Dallas, TX, USA

Brent Page UT Southwestern Medical Center, Dallas, TX, USA

Ankit Patel UT Southwestern Medical Center, Dallas, TX, USA

Biral T. Patel Department of Anesthesiology and Pain Management, University of Texas Southwestern Medical Center, Dallas, TX, USA

Gabor Bela Racz Department of Anesthesiology, Texas Tech University Health Science Center, Lubbock, TX, USA

Gabor J. Racz Epimed International, Dallas, TX, USA

Karl Rathjen Department of Orthopaedics, UT Southwestern Medical School and Scottish Rite for Children, Dallas, TX, USA

Nisha Reddy Department of Orthopaedic Surgery, UT Southwestern Medical Center, Dallas, TX, USA

Richard C. Robinson Division of Psychology, Department of Psychiatry, UT Southwestern Medical Center, Dallas, TX, USA

Bryan T. Romito Department of Anesthesiology & Pain Management, University of Texas Southwestern Medical Center, Dallas, TX, USA

Jia W. Romito Departments of Anesthesiology & Pain Management, Neurological Surgery, and Neurology, University of Texas Southwestern Medical Center, Dallas, TX, USA

Jose Marin Sanchez University of Texas Southwestern Medical Center, Department of Neurological Surgery, Dallas, TX, USA

G. Sunny Sharma University of Texas Southwestern Medical Center, Dallas, TX, USA

Peter Shin Department of Neurological Surgery, University of New Mexico Hospital, Albuquerque, NM, USA

Nilofar Syed Division of Rheumatology, Department of Internal Medicine, UT Southwestern Medical Center, Dallas, TX, USA

Lori A. Tappen Department of Orthopedic Surgery, UT Southwestern Medical Center, Dallas, TX, USA

Jeffrey Traylor Department of Neurological Surgery and Spine Clinic, UT Southwestern Medical Center, Dallas, TX, USA

Kavita Trivedi University of Texas Southwestern Medical Center, Dallas, TX, USA

Michael Van Hal Department of Orthopaedic Surgery, UT Southwestern Medical Center, Dallas, TX, USA

Shaleen Vira Department of Orthopaedic Surgery, UT Southwestern Medical Center, Dallas, TX, USA

Charles Whitten Department of Anesthesiology and Pain Management, University of Texas Southwestern Medical Center, Dallas, TX, USA

Cody Wolfe Department of Neurological Surgery and Spine Clinic, UT Southwestern Medical Center, Dallas, TX, USA

Esther Yoon University of Texas Southwestern Medical Center, Dallas, TX, USA

Carl Youssef Department of Neurological Surgery, UT Southwestern Medical Center, Dallas, TX, USA

Jason Zafereo Department of Physical Therapy, University of Texas Southwestern Medical Center, Dallas, TX, USA

Lifestyle Management of Spine Patient

1

Kavita Trivedi and Esther Yoon

Introduction

The average human spine has 33 vertebra, 100 joints, 120 muscles and 220 ligaments. It is literally our backbone and is central to help keep us upright and moving. Therefore, it is important that we take care of our spine in all that we do. This includes our activities at home, at work, at school, during the times that we play and socialize, both during the day and at night. It is important to understand the anatomy of our spine in order to recognize how all the different aspects of our lives affect our spine health.

The spinal column is composed of 7 cervical, 12 thoracic, 5 lumbar and 5 fused sacral vertebrae, along with 5 coccygeal bones. The cervical, thoracic and lumbar vertebrae are similar in structure except for the first (atlas) and second (axis) cervical vertebrae. Each "standard" vertebra is composed of a body, two pedicles, two lamina, four articular facets, and a spinous process. The atlas is composed of a ring of bone without a body, whereas the axis has an odontoid process around which the atlas rotates. Between each pair of vertebrae are two openings, the foramina, through which pass a spinal nerve, radicular blood vessels, and the sinuvertebral nerves (recurrent meningeal nerves). Each foramen is bordered superiorly and inferiorly by pedicles, anteriorly by the intervertebral disc and adjacent vertebral body surfaces, and posteriorly by the facet joint [34] (Figs. 1.1, 1.2 and 1.3).

The spinal canal itself is formed posterolaterally by the laminae and ligamentum flavum, anterolaterally by the pedicles, and anteriorly by the posterior surfaces of the vertebral bodies and intervertebral discs [34]. One retrospective study determined that patients with a sagittal cervical spinal canal diameter of less than 10 mm had myelopathy and those with a diameter of 10–13 mm had pre-myelopathic changes. Additionally, they found that patients with a diameter of 13–17 mm were

K. Trivedi (✉) · E. Yoon
University of Texas Southwestern Medical Center, Dallas, TX, USA
e-mail: kavita.trivedi@utsouthwestern.edu; Eyoon@neurosurgeryone.org

© The Author(s), under exclusive license to Springer Nature Switzerland AG 2022

C. E. Noe (ed.), *Multidisciplinary Spine Care*,
https://doi.org/10.1007/978-3-031-04990-3_1



Fig. 1.1 Posterior view of spine



Fig. 1.2 Lumbar vertebrae

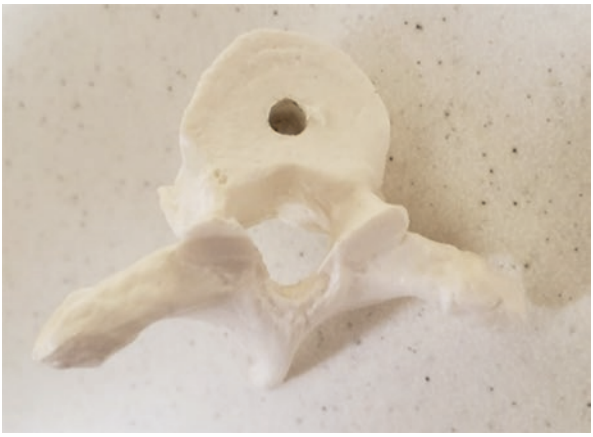


Fig. 1.3 Thoracic vertebrae

less prone to myelopathy but were prone to symptomatic cervical spondylosis and those with a diameter of greater than 17 mm were not prone to development of cervical spondylosis [39]. With regard to the lumbar canal, the midsagittal diameter is approximately 18 mm. Narrowing as a result of spondylosis coupled with extension can compromise the cauda equina and the accompanying vasculature, producing symptoms of neurogenic claudication [34].

In addition to anatomic variations that can cause a spinal problem in patients, there are other mechanical conditions in the spine which can be problematic. One contributor to mechanical issues in the spine is the intervertebral disc. The intervertebral disc is a fibrocartilaginous structure whose main function is to transmit compressible load between the vertebral bodies, while also providing flexibility [38]. Mechanical causes of discogenic pain include torsional injury and internal disc disruption. Torsion injury is believed to result from forcible rotation of the intervertebral joint [14]. A study by Farfan indicated that about 90% of the torque strength of an intervertebral joint is provided by its disc and the two joints between the articular processes with the disc and the joints between the articular processes contributing almost equal portions [41]. Lumbar disc herniation is a common low back disorder and the most common cause of sciatica in adults [43]. In addition to lumbar spine disc herniation, cervical spine disc herniation is a common source of cervical radiculopathy [122]. Various factors have been ascribed to the etiology of lumbar disc herniation, such as compressive stress, aging, overweight and toxic factors [54]. Cigarette smoking generates toxic substances, which may play a role in degeneration of the disc [102]. A 2016 systematic review demonstrated that cigarette smoking had a statistically significant association with increased risk of lumbar disc herniation [59]. This review conducted further analysis which showed that both male and female smokers had a similar risk of lumbar disc herniation and that current smokers showed a higher risk of developing lumbar disc herniation than former smokers. Furthermore, smokers from Asia were most likely to develop lumbar disc herniation, followed by those from Europe and North America. Another disc issue that can cause spine pain is degenerative disc disease. Degenerative disc disease is

a multifaceted progressive irreversible condition and an inevitable part of aging, which has been found to be a contributing factor for low back pain and might cause radiculopathy, myelopathy, spinal stenosis, degenerative spondylolisthesis and herniations. Although the genetic influence is more dominant, the occupational and mechanical influences still persist as a major risk factor [57]. Internal disc disruption results from lumbar disc degradation, its nuclear components, and development of radial fissures that extend from the nucleus into the annulus [127]. Internal disc disruption is thought to be the most common type of discogenic pain and is not to be confused with degenerative changes which are a normal part of aging [14] (Fig. 1.4).

Another mechanical issue of the spine which can cause spine pain is facet-mediated pain. This refers to pathology of the diarthrodial zygapophyseal joint or the facet joint. This joint is located at either side of the posterior vertebral body. The facet joint's opposing bony surfaces are covered by a layer of hyaline articular cartilage, and the joint is encapsulated by the synovium and fibrous capsule [107]. Facet joints work in pairs, along with the intervertebral disc, to constrain the motion of the vertebra while aiding in the transmission of spinal loads [110]. This is often



Fig. 1.4 Side view of lumbar discs; arrow indicates lumbar disc herniation at L4–L5 level

referred to as the three-joint complex. The facet joint ensures that the spinal column resists joint distraction, shear forces, and lateral or anteroposterior translation and imparts sufficient torsional stiffness [66]. The main causes of facet-mediated pain are trauma, where a facet joint can dislocate or fracture, and degeneration which can be trauma or age-induced. Anatomical studies have found that as aging occurs, the joints become weaker and more biplanar, transitioning from a largely coronal orientation to a more prominent sagittal positioning. This is known as tropism and has been found to occur in 20–40% of the general population [99]. The three most caudal facet joints, L3–L4, L4–L5, and L5–S1, are exposed to the greatest strain during lateral bending and forward flexion and are thus more prone to repetitive strain, inflammation, joint hypertrophy, and osteophyte formation [25] (Fig. 1.5).

Additionally, lumbar strain is another mechanical issue that can cause spine pain. A similar process can occur in the cervical and thoracic spine. Lumbar strain or sprain is defined as diffuse pain in the lumbar muscles which can involve radiation to the buttocks. Lumbar strain or sprain accounts for more than 70% of mechanical low back pain [68]. There are numerous causes of spine strain such as participation in certain sports including (but not limited to) weight lifting, football, cycling, running, baseball, ballet, rowing, gymnastics and wrestling. Low back pain is more common in some athletes than in others [15]. One prospective study found that

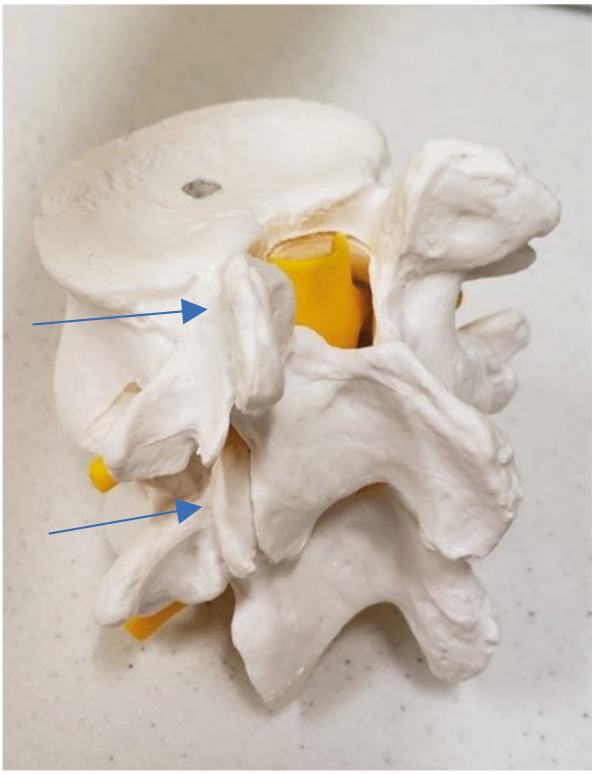


Fig. 1.5 Oblique view of portion of the lumbar spine; arrows indicate lumbar facet joints

wrestlers had the highest rate of severe low back pain, while rates were lower for tennis and soccer players [83]. Another study found that in comparison to other athletes, gymnasts appear to be the most likely to report severe back pain [128]. In addition, certain occupations that require frequent periods of static lumbar flexion are known to be risk factors for the development of a low back disorder [121]. Such professions include surgeons, dentists and certain industrial jobs. Although the task requirement, surgical equipment and operating theatre set up individualized to procedures differs across specialties, low back pain is a consistent finding across all surgical specialties [141]. In terms of industrial jobs, one study found that an increase in the magnitude of lifting frequency, load movement, trunk lateral velocity, trunk twisting velocity, and trunk sagittal angle significantly increases the risk of a low back disorder [88]. Other conditions including pregnancy, metastatic disease to the spine and previous spine surgery can cause cervical, thoracic or lumbar strain. The recent increase in working from home has forced individuals to find spaces in their homes which can be used as an office. This often involves a suboptimal ergonomic design which leads to prolonged periods of poor posture.

How Posture Affects the Spine

Many of us have been told to "sit up straight" or "stop slouching" by a parent or teacher at some point in our lives. That little piece of advice has become so relevant in today's society where a majority of the population spends a significant amount of time sitting, whether that be in front of a desk, computer, television, tablet or phone. Individuals who sit throughout the majority of the day often develop low back pain (LBP) and other spinal issues. Sustained body postures and postures outside of neutral have been identified as risk factors for the development of LBP as it leads to prolonged muscle contractions [56] and changes in intervertebral disc pressure [146]. According to the 2016 analysis of health care spending in the United States (US), the *Journal of the American Medical Association* (JAMA) reported that health care amounted to more than 17% of the US economy and low back pain and neck pain accounted for the third highest amount of health care spending at $87.6 billion from 1996 through 2013 [36]. The total cost of back pain in the US was estimated to be $100–200 billion in 2006 with two-thirds of the estimated amount due to indirect costs from productivity loss and absence of work [67]. In addition, low back pain was the leading cause of industrial disability payments and the second leading medical cause of industrial work loss [20]. As spinal issues continue to grow in prevalence and have an immense burden on the health care system and economy, it is essential to counsel patients on easily accessible and inexpensive conservative measures such as posture training for prevention as well as management of pain and function.

In terms of anatomy, the spine consists of the cervical, thoracic, lumbar, sacral, and coccygeal vertebral regions as well as the intervertebral discs, ligaments, rib cage, and spinal musculature [144]. Components of the whole spine work in

conjunction and serve to protect the spinal cord, support and bear load, and provide motion. In a normal, neutral spine, the cervical and lumbar regions have lordotic curves while the thoracic and sacral/coccygeal regions have kyphotic curves. Newer studies also account for the anatomical relationship between the spine, pelvis, and hip joints as an important factor when describing the positioning of the whole spine. The relationship between these parts is referred to as sagittal balance, which is the anterior-posterior position of C7 with respect to the sacrum [74]. Deviations from the sagittal balance has been shown to correlate with clinical symptoms. In addition, the Spine Stabilizing System hypothesis posed by Panjabi in 1992 stated that the role of the complex neuromuscular system is to keep the spine mechanically stable to avoid injury that eventually leads to pain [111]. This suggests that proper positioning and posture to maintain a neutral spine in sagittal balance keeps the spine mechanically stable and protects it from injury and pain.

In the 1960s, Nachemson measured lumbar intradiscal pressures in different positions of the body [100]. This study revealed that the intradiscal pressure in supine position is about 25% of the pressure in standing position, whereas sitting position increases the intradiscal pressure by 40% compared to the pressure in standing position. It also showed that leaning forward substantially increased intradiscal pressure, whether it be in the sitting or standing position. Similarly, lifting a 10 kg weight in the forward flexed position further increased intradiscal pressure, both in sitting and standing. Intradiscal pressure increases in sitting because of the increased trunk moment when the pelvis rotates backwards and because of disk deformation from flattening of the lumbar spine, both of which also disrupts the sagittal balance [113]. With prolonged standing, muscle activity is required to maintain an upright posture and any shift in the center of gravity, such as an outstretched arm or external weight, requires active counterbalance to maintain equilibrium [113]. When fully flexing the spine, the lumbar extensor muscles become inactive and unable to support anterior shear forces which are highly correlated to the risk of back injury [91]. When these studies are applied outside of a clinical setting, they suggest that jobs or activities that require prolonged sitting, such as an office worker or truck driver, or jobs that require constant forward flexed postures, such as construction, warehouse workers and gardeners, have a higher risk of back injury.

As previously mentioned, prolonged sitting results in posterior rotation of the pelvis and flattening of the lumbar lordosis. This results in posterior migration of the nucleus pulposus [4] and increased passive strain on the posterior spinal elements [2], which together increases the risk of disc herniation [114]. A study by Wilke in the 1990s sought to measure intradiscal pressure during various activities and positions with a more advanced transducer than used by Nachemson in the 1960s [147]. As far as sitting posture, this study found, as predicted, that sitting relaxed without a backrest produced 0.46 MPa of intradiscal pressure, whereas sitting with maximum flexion produced 0.83 MPa of intradiscal pressure. However, the study also found that sitting slouched into the backrest of the chair actually decreased intradiscal pressure, producing only 0.27 MPa of pressure. Wilke concluded that more load was transferred through the backrest with increased

slouching, accounting for the decreased pressure recordings of the study. The results of this study suggest that increasing the contact of the backrest on the low back allows for more load transfer to help decrease lumbar intradiscal pressure during sitting position and may also allow for reduction in the passive strain on the posterior elements. Similarly, the use of a lumbar support cushion can increase the contact on the low back as well as maintain the natural lordotic curvature to decrease posterior element strain [85]. One study measured the effects of a lumbar support pillow on comfort and posture during a prolonged seated task in subjects with and without low back pain [51]. This study indicated that a lumbar support pillow was better at increasing the natural lumbar lordosis as well as neutral thoracolumbar curvature during sitting in both populations. In addition, this study had a postural amplitude difference of about 2–3° in the lumbar region. A separate study has demonstrated that small changes of 2–3° may significantly impact the compressive load at the L4/5 level [23]. However, the study by Grondin did not report any significant improvements in reported comfort. In another study, significant improvements were demonstrated in the visual analog scales for low back pain, stiffness, and fatigue in volunteers who used a fixed lumbar support or a continuous passive motion device with prolonged sitting compared to volunteers who had no lumbar support with prolonged sitting [6]. A systematic review of the effects of chair backrests on low back pain, low back discomfort, and trunk muscle activation found moderate evidence that chair backrests decrease paraspinal muscle activation and limited evidence that they reduce low back discomfort [28]. Therefore, although there are several studies that demonstrate improvements in the sagittal balance with the use of lumbar support as well as reduced pain and discomfort, the overall evidence is limited for the use of backrests for management of low back pain. As low back pain is often multifactorial, management will likely require more than an isolated modification of sitting posture. However, as there are minimal risks with using a seated lumbar support, it is reasonable to discuss its use as a part of a patient's lifestyle management and complete care of their low back pain (Fig. 1.6).

In addition to the sagittal imbalance and increase in intervertebral disc pressure, prolonged seated posture leads to inactivity that, in itself, may be injurious as lack of movement leads to accumulation of metabolites that may accelerate degeneration of discs and increase risk of disc herniation [113]. It also results in tightening of lower extremity muscles, decreased circulation, and deconditioning, which results in increased pain and stiffness [31]. A study aiming to determine whether sitting on a stability ball rather than a chair affects LBP from prolonged sitting resulted in improvement of core endurance in the sagittal plane but no direct significant effects on low back pain or disability [40]. The Stand Back trial in 2018 sought to evaluate the feasibility and effect of decreasing sedentary behavior in desk workers with chronic LBP through use of a sit-stand desk, movement breaks, and pain self-management [9]. This 6-month trial resulted in a 50% decrease in disability in the treatment group compared to a 14% decrease in the controls. However, this study did not show a statistically significant reduction in pain scores in the treatment



Fig. 1.6 Sagittal view of spine

group compared to the controls. Another study examined the effect of office ergonomic intervention in reducing musculoskeletal symptoms [5]. Subjects who received a highly adjustable chair along with office ergonomics training had reduced symptom growth over the workday at 1-year follow-up. In addition, the average pain levels were reduced in both the treatment group who received the adjustable chair and ergonomics training as well as the treatment group who received just the ergonomics training without the adjustable chair compared to the control group who received neither. These studies imply that frequent movement from sitting, whether that be from sit to stand, a stability ball, or an adjustable chair, is an important factor for core muscle endurance, low back pain, and disability. Prolonged immobility, on the other hand, can be detrimental in chronic LBP patients as it can lead to further deconditioning of core muscles and injury to the intervertebral discs. Therefore, proper ergonomics and posture, along with frequent changes in position, is an important component in the management of chronic LBP, especially in patients who are sedentary for the majority of the day (Fig. 1.7).



Fig. 1.7 Ergonomic work station with laptop elevated so it is at eye level to prevent neck strain and an adjustable chair

The Impact of Exercise on the Spine

As physicians, we often refer our spine patients to physical therapy or recommend them to exercise and stay active. In the past, it was common practice to advise patients with acute LBP and sciatica to rest in bed as initial management. However, currently, almost all clinical guidelines for acute LBP management favors staying active above bedrest [70]. One study published in the New England Journal of Medicine trialed patients with acute, nonspecific LBP to one of three treatments – bedrest for 2 days, back-mobilization exercises, or continuing normal daily activities as tolerated – and assessed them at 3 and 12 weeks [86]. This study found that patients who continued their normal activities had statistically significant improvements in duration of pain, pain intensity, lumbar flexion, ability to work, Oswestry back-disability index, and number of days absent from work. A Cochrane review analyzing staying active compared to bedrest in acute LBP found moderate-quality evidence that advice to stay active resulted in small benefits in pain relief and functional improvement in patients with acute LBP compared to advice for bedrest but found little to no difference in patients with sciatica [29]. Overall, the findings of this review did not oppose the recommendations in the current clinical guidelines but did show that greater scientific evidence is needed for more conclusive results. When comparing advice to stay active to formal exercise or physiotherapy, the Cochrane review revealed there was low-quality evidence that suggested little to no difference in symptoms and function. Another systematic review and meta-analysis from 2015 analyzing patients with sciatica revealed low-quality evidence that exercise resulted in small but superior results to leg pain in the short term when compared with just giving advice to stay active [42]. However, in the long term, there was moderate-quality evidence showing no difference in leg pain and disability between exercise and advice to stay active. Therefore, in the setting of acute LBP, it is essential that physicians counsel patients on the importance of at least staying

active in terms of pain reduction and functional improvement. In terms of nonspecific chronic LBP, a review of current literature and meta-analysis revealed no evidence of effectiveness using bedrest as a therapeutic option [16]. In this review, only movement therapy postulated a level I evidence for long-term effect in treating nonspecific chronic LBP. There have been multiple studies performed over the years assessing different methods of movement therapy and its effect on LBP, including core strengthening programs, McKenzie method of diagnosis and therapy (MDT), yoga therapy, tai chi, aerobic exercises, as well as many others. Overall, the evidence of these different exercises and physiotherapy methods are limited or inconclusive and need further investigation, but some of the existing results are promising and are important options when discussing treatment plans with patients.

As part of the exercise prescription for patients with LBP, especially in patients with chronic LBP, core strengthening exercises for trunk stability is often included. The theory behind core strengthening is to train the deep trunk muscles that help to ensure mobility and stability of the lumbopelvic region that are often weak or insufficient in patients with chronic LBP [73]. In a study of 79 participants with chronic LBP, participants were divided into two groups: an experimental group performing specific trunk balance exercises along with standard trunk flexibility exercises and a control group performing general strengthening exercises and the standard trunk flexibility exercises [46]. This study followed the participants for 6 weeks and concluded that the combination of trunk balance and flexibility exercises were significantly more effective than the combination of general strengthening and flexibility exercises in decreasing disability and improving the physical component in the quality of life of chronic LBP patients. However, this study did not show a difference in improvements in visual analog scale (VAS) pain scores between the two groups. Similarly, another study looked at 120 participants and assigned them to a core stabilization exercise group or a routine physical therapy group [3]. However, this study did find significant reduction in pain on VAS in the core stabilization group compared to the routine physical therapy group. A meta-analysis and systematic review in 2017 looked at randomized controlled trials that compared the effectiveness of core stability exercise to general exercise on chronic low back pain [26]. The analysis found a significant reduction in pain intensity and increase in back-specific functional status at 3 months but not at 6 months. This analysis implies that core stability exercises may be more effective than general exercise in the short term. However, in the long term, overall staying active – whether this be through core strengthening or general exercise and conditioning – may be an essential component in reducing pain and improving function in chronic LBP patients. Another systematic review explored the effectiveness of various core strengthening strategies in patients with chronic LBP, identifying four types of core strength training – trunk balance exercises, stabilization, segmental stabilization, and motor control exercises [24]. Compared to typical resistance training, core strength training was easier to learn for chronic LBP patients, patients were able to independently practice core strength training at home more accessibly as it did not require any special equipment, and there were less injuries overall. Of the various types of core strengthening exercises, training of the deep trunk muscles seemed to have the greatest

effect on pain alleviation. This correlates with the findings of weakening in the deep trunk muscles such as the lumbar multifidus and transverse abdominis with insufficient motor control in chronic LBP patients [118].

The use of McKenzie method of mechanical diagnosis and therapy (MDT) is often added to the therapeutic prescription for patients who complain of back pain with radiation or directionality. MDT is an assessment and treatment model used to classify patients with low back pain in order to direct treatment. MDT classifies low back pain patients into three mechanical subgroups – derangement, dysfunction, or postural syndrome. Derangement is the most common subgroup and is associated with directional preference where repeated movements or sustained positions produce improvement in symptoms such as centralization [92]. A meta-analysis from 2006 of randomized controlled trials evaluating the effectiveness of the McKenzie method for low back pain resulted in limited evidence to support the use of MDT as it concluded that the MDT method did not produce clinically significant differences in pain and disability [84]. However, this review did not separate acute low back pain from chronic low back pain, exclude studies with untrained therapists, or analyze if patients were classified into subgroups prior to receiving specific treatments. A more recent meta-analysis in 2018 did include these factors and came to a different conclusion [75]. For this review, MDT did seem to be more effective at decreasing pain level than manual therapy plus exercise in patients with acute LBP. However, it is important to note that the sample size was small and it did not result in any significant difference in improvement of disability compared to other interventions. In chronic LBP patients, MDT was more effective at decreasing pain level and disability compared to other rehabilitation interventions. However, compared to exercise, MDT was only superior in decreasing disability but not pain. When manual therapy and exercise were combined, MDT was not more effective in reducing disability or pain. Again, when managing patients with chronic LBP, the evidence reveals that exercise is a key component to reducing disability and pain that can be just as effective as other formal physical therapy methods.

Yoga is a discipline of mind-body practices that originated in ancient India [120]. According to the US National Health Interview Survey (NHIS), yoga has become increasingly popular in the US with over 13 million adults reporting use of yoga in 2007 to over 21 million adults in 2012 [8, 27]. Of the people who reported use of yoga in 2012, 19.7% stated the use was specifically for management of back pain [27]. As yoga has become a widely used practice and form of exercise in the US, there have been multiple studies of its effects on chronic back pain. One study followed 90 subjects who were randomized to a yoga or control group [149]. The yoga group participated in 24 weeks of biweekly yoga class while the control group continued with standard medical care. Results revealed significantly greater reductions in functional disability, pain intensity, and depression in the yoga group at the end of their course as well as at 6 months post-intervention. Similarly, another study offered a 12-week yoga program to adults with chronic or recurrent LBP and compared them to another group who only received usual care and an educational booklet [132]. In this study, there was little difference in back pain, but the yoga group had improvement in back function compared to the standard group at 3, 6, and

12 months. There was also a Cochrane Review of yoga for the treatment of nonspecific chronic low back pain [145]. When yoga was compared to non-exercise controls, there was moderate evidence of improved function in the intermediate term and low evidence at short and long term. There was also moderate evidence of pain reduction in the short-to-intermediate term. Interestingly, there was moderate-certainty evidence that yoga produced an adverse effect of exacerbation of back pain more commonly when compared to the non-exercise control groups. When compared to exercise controls, there was very-low certainty of improved back-related function and pain reduction in the yoga group due to risk of bias, inconsistency, and imprecision of the studies evaluated. Although there are many studies that demonstrate the benefits of yoga in patients with chronic LBP, many of these studies compare yoga to a non-exercise control. Just as with core strengthening exercises and McKenzie based therapy, yoga fairs similarly with the exercise controls in terms of pain reduction and function, which again demonstrates the overlying importance of exercise in chronic LBP patients (Fig. 1.8).

Tai Chi is another mind-body discipline that has gained more popularity around the world. It is a low-moderate intensity exercise that originated from China with research demonstrating similar health benefits in terms of aerobic fitness, resting energy expenditure, body composition, and self-perceived physical health as aerobic exercise with lower energy metabolism [60]. A systematic review and meta-analysis evaluated the effects of Tai Chi alone or as additional therapy for low back pain [115]. The review suggested significantly decreased pain intensity and improvement of functional disability with practice of Tai Chi alone or as a supplement to routine physical therapy. However, due to location bias, lack of blinding, lack of

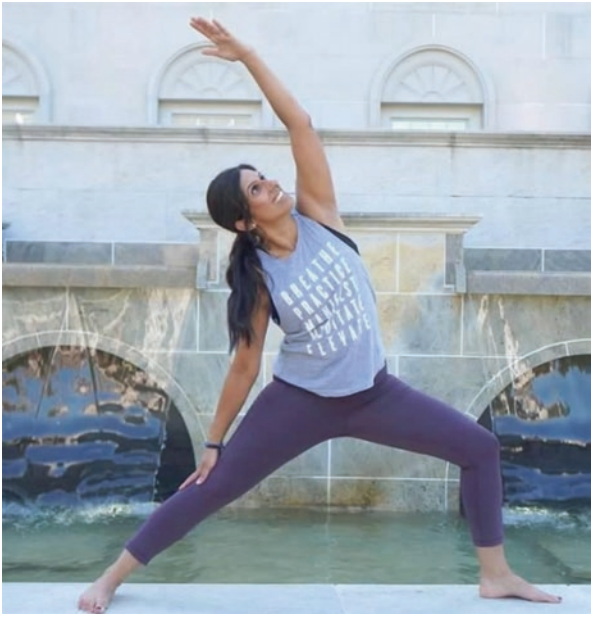


Fig. 1.8 Reverse warrior yoga pose

long-term follow-up, and the wide variation in Tai Chi styles included in the review, the conclusions were drawn cautiously and further trials with more regulations are needed. However, as Tai Chi is a relatively safe exercise that is convenient and inexpensive, it should still be considered when discussing lifestyle modifications and back pain management with patients.

According to the United Kingdom (UK) National Institute for Health and Care Excellence, general strength, conditioning, and resistance training of the spinal musculature, including aerobic exercises, have one of the best results in reducing pain and disability in patients with chronic LBP in both the short and long term. A systematic review in 2015 revealed that aerobic exercise leads to significant reduction in both pain and disability measurements as well as decreases in depression and anxiety, indicating that aerobic exercise may be beneficial to both physical and psychological function in patients with chronic LBP [94]. This study hypothesized that aerobic exercise may decrease resting beta-endorphin levels, contributing to improved mood. In addition, aerobic exercise increases endurance and strength while preventing fatigue, so it helps maintain proper body mechanics, resulting in decreased LBP and injury [139]. Another systematic review from 2016 concluded that aerobic exercise is beneficial in decreasing pain level in patients with chronic low back pain when combined with other forms of therapy and exercises such as stabilization, strengthening, and flexibility [48]. Other forms of aerobic exercise, such as aquatic therapy, have also shown beneficial results in patients with LBP. A systematic review analyzed clinical trials studying all types of therapeutic aquatic exercise in adults suffering from low back pain [140]. Although more high-quality trials are needed, the review concluded there was sufficient evidence to suggest potential benefit of therapeutic aquatic exercise in patients with chronic LBP as well as pregnancy-related LBP.

The term “aerobic exercise” is often defined as a relatively low intensity exercise with longer duration ranging between 15 and 60 continuous minutes and intensity of 60–90% the maximum heart rate [123]. A more recent systematic review suggested that higher training intensities may be a more effective exercise training for patients with chronic non-specific LBP [143]. A randomized controlled trial looked to compare the effects of high-intensity training (HIT) to moderate-intensity training (MIT) on disability, pain, function, exercise capacity, and trunk muscle strength in subjects with chronic non-specific low back pain [135]. HIT demonstrated benefit in all of these categories in persons with chronic non-specific LBP and specifically resulted in greater improvements in disability and exercise capacity when compared to MIT. Another study evaluated the effectiveness of various modes of HIT exercises and compared differences between the modes in terms of pain intensity, disability, and physical performance in subjects with chronic non-specific LBP [136]. Subjects were randomized into four HIT groups – cardiorespiratory interval training plus general resistance training, core strength training, combined general resistance and core strength training, or mobility exercises. After a 12-week program, improvements in each category measured were seen within all groups, but no differences were found between groups. The study concluded that HIT with cardiorespiratory interval training improves chronic LBP rehabilitation outcomes when combined



Fig. 1.9 Treadmill which can be used for jogging and running forms of exercise

with other HIT modes or mobility exercises and various HIT modes can be considered when discussing exercise treatment options with chronic LBP patients.

In terms of intervertebral disc (IVD) health, both animal and human studies have shown a positive relationship of IVD composition and running. One study compared IVD tissue quality of long-distance runners and joggers to that of non-athletic individuals. This study found that the athletic individuals had better disc hydration and glycosaminoglycan levels [11]. The study also reinforced the concept of a "likely anabolic window" for the IVD, with fast walking or slow running providing the strongest anabolic stimulus for IVD adaptation in humans, while slow walking provided minimal IVD benefits compared to no activity and fast running or high-impact jumping activities resulted in high-impact loading considered detrimental to the IVD and vertebral end-plates. In addition, it revealed better IVD hydration, glycosaminoglycan content, and hypertrophy in the exercise group within this "anabolic window", despite repetitive loading, contradicting the previous theories that repetitive loading contributes to IVD degeneration (Fig. 1.9).

The Effects from Nutrition on the Spine

The Oxford dictionary defines nutrition as the process of providing or obtaining food and nourishment necessary for health and growth [109]. Good nutrition helps to keep an individual healthy while poor nutrition often correlates with the degradation of health accompanied by a burden of diseases. What individuals put into their bodies and expose themselves to can have an impact on multiple body systems. For example, eating certain diets can increase cholesterol levels and cause build-up of atherosclerotic plaques. This, in turn, can result in blockage of arteries, marking the deterioration of a patient's cardiovascular health. In the context of a spine patient, nutrition refers to the positive and negative extrinsic factors that affect spine mechanics and correlate with "spine health" or structural changes that affect spine stability, pain, and functionality. These extrinsic factors may correlate with different

spinal issues such as disc deterioration and herniation as well as play a role in spinal pain perception.

As previously mentioned, cigarette smoking is associated with an increased risk of lumbar disc herniation and it creates toxic substances that are correlated to disc degeneration. There have been several studies demonstrating the association between cigarette smoking and disc degeneration and herniation. In a study comparing pairs of identical twins discordant for tobacco exposure, the smoking group had an 18% greater average disc degeneration throughout the lumbar spine [10]. Another study demonstrated that subjects who smoked at least 20 cigarettes per day had greater estimated prevalence rates for low back pain at some time in the past year, low back pain for at least 30 days in the past year, disc degeneration, and degeneration with signs of marrow changes consistent with vertebral inflammatory process [77]. Thirdly, in a prospective study with 11-year follow-up that attempted to identify risk factors for lumbar discectomy secondary to disc herniation in a young and relatively healthy population, daily smoking was the strongest risk factor for lumbar discectomy in the male subjects [90]. There are two main theories of how tobacco use contributes to intervertebral disc degeneration. One theory hypothesizes that tobacco use leads to vascular disease, which causes vascular disruption and anoxia of the nutritional supply to the discs, resulting in malnutrition [65]. The other theory proposes that the chemical compounds found in cigarette smoke can result in direct dose-dependent morphologic changes to the intervertebral disc cells [138] as well as histologic changes such as increased production of interleukin 1B, leading to the upregulation of matrix-degrading metalloproteinases [104]. Although separate in approach, both theories result in alteration of intervertebral discs. Animal studies with gross examination of disc tissues exposed to cigarette smoke have shown necrosis and fibrosis involving the nucleus pulposus as well as changes to the composition of the annulus fibrosis [61, 134]. With the available evidence, it is reasonable to postulate that cigarette smoking leads to pathology within the intervertebral discs that may make them more prone to tearing. According to the degenerative cascade proposed by Kirkaldy-Willis in the 1970s, tearing in the discs leads to disc dysfunction, increasing the risk of herniation [69]. Therefore, it is not surprising that the evidence shows a correlation of disc degeneration and herniation to cigarette smoking.

Spine patients are often instructed by their providers to lose weight in order to decrease the load added onto their spine. Weight loss is often a difficult endeavor that must include the patient's motivation, but may also include a multifaceted approach that includes nutritionists to assess the patient's diet, physical therapists and trainers to provide the patient with activity instructions, counsellors and psychiatrists to evaluate the patient's mood, and many more. Although creating an effective weight loss program or regimen with patients is challenging, it is important because the biophysical effects of obesity on the spine's curvature can have long-term downstream effects of degeneration and increased risk of herniation. In the case of increased disc pressure, one study found that the estimated L4 compressive loading was highest in subjects with obesity for standing, holding, and lifting [7]. In a different cross-sectional study from 2014, thoracolumbar curvatures were

compared between participants with obesity and normal weight individuals according to their waist circumference. This study found that there were significant differences in thoracic kyphosis between the two groups, which was speculated to be due to the increase in upper limb mass as well as the requirement for biomechanical rebalancing of forces upon the body [47]. It is likely that an increased kyphotic positioning of the thoracic spine will produce increased pressure on the intervertebral discs, leading to risk of degenerative disc disease and disc herniations. In one study, subjects with a BMI greater than 25 kg/m^2, categorized as overweight, had imaging with strong association of disc degeneration at 4-year follow-up, which was indicated by decreased signal intensity of the nucleus pulposus on magnetic resonance imaging (MRI) [81]. In addition, the study showed that overweight subjects at the age of 25 had statistically significant increased risk of progression of degenerative changes. In a population-based cross-sectional study, there was a significant increase in the number of levels with disc degeneration, global severity of disc degeneration, and evidence of disc space narrowing in overweight and obese subjects [119]. This study demonstrated a positive linear trend between BMI and evidence of disc degeneration with an odds ratio (OR) = 1.30 in overweight subjects and OR = 1.79 in obese subjects, with significantly more pronounced disc space narrowing in obese subjects with an adjusted OR = 1.72. Lastly, in a retrospective study of 165 patients with a mean age of 21.2 years at the time of surgical treatment for lumbar disc herniation, surgical patients had a statistically significant greater body mass index (BMI) compared to a patient population of similar age [112]. These studies revealed degenerative changes of the intervertebral discs as well as increased incidence of lumbar disc herniations in the obese population as early as their teenage years and into their twenties. This implies that early intervention and maintenance of weight loss may be a key component in retarding further disc degeneration and occurrences of disc herniations.

On the other hand, significantly low BMI can also have detrimental effects to a patient's spine health. In patients with anorexia nervosa (AN), the spine is the skeletal site of greatest bone loss [95]. In one study of subjects with AN, both trabecular and cortical bone sites were affected with low bone mineral density (BMD), with overall data suggesting that female sites of trabecular bone – such as the lumbar spine – were more severely affected [97]. In another study looking at 18 women with AN between 19 and 36 years old, the 18 anorectic women had significantly lower cortical BMD compared to the 28 normal controls, 2 anorectic women had multiple non-traumatic vertebral compression fractures and loss of height, and 1 of these women underwent an iliac-crest bone biopsy that revealed thinned trabeculae and minimal osteoblast and osteoclast activity consistent with osteoporosis [116]. Similarly, when compared to lean and obese women, women with AN had lower integral volumetric BMD and estimated vertebral strength [7]. These studies demonstrate that extremely low BMI is correlated with low BMD and decreased vertebral strength, which in turn can lead to bone disease such as osteoporosis as well as bone injury such as vertebral compression fractures. Interestingly, the latter study also found higher L4 compressive loading during lateral bending in women with AN compared to the lean and obese women with activation of additional muscle

groups in those with AN that was not seen in the lean or obese groups. Therefore, in addition to the low vertebral strength, women with AN also have high applied disc load during lateral bending, increasing the overall load-to-strength ratio and further increasing the vertebral fracture risk in this population.

Patients often inquire about herbal therapies and dietary supplements that may help to modulate their pain and degenerative disease. Especially in the setting where traditional medications such as NSAIDs may be contraindicated or not tolerated, these natural substances may be a potential alternative. Turmeric is often mentioned due to its anti-inflammatory properties. When used as an alternative medicine, turmeric is typically used as an extract that primarily contains its derivative, curcumin, which modifies NF-kB signaling and proinflammatory cytokines such as interleukin production [30]. IL-1 is an inflammatory factor that has shown to reduce the regenerative and repairing capability of the injured cartilage in osteoarthropathy and arthritis [117]. One study suggested that intervertebral disc degeneration could be prevented or reversed by decreasing IL-1 content or inhibiting NF-kB, which were shown to be attenuated by curcumin *in vitro* [152]. A systematic review and meta-analysis revealed that 8–12 weeks of standardized turmeric extracts of 1000 mg/day of curcumin can reduce inflammation-related arthritis symptoms with similar results as ibuprofen and diclofenac sodium [30]. However, the studies that were represented were moderate in quality with small sample sizes that were insufficient to be conclusive. Omega-3 fatty acids have also been shown to have anti-inflammatory properties that play an important role in regulation and reduction in symptoms of osteoarthritis [52]. A recent study investigated the effects of a daily omega-3 fatty acid diet in rats with lumbar disc degeneration [101]. Results revealed a reduction in blood arachidonic acid/eicosapentaenoic acid (AA/EPA) ratios after 1 month of supplementation, micro-MRI analysis showed attenuation of injury-induced reduction of IVD hydration, and histological evaluation demonstrated decreased severity in the destruction of the nucleus pulposus tissue in response to needle puncture injury in the omega-3 fatty acid diet group. The study concluded that results suggested omega-3 fatty acid dietary supplementation may have potential protective effects against intervertebral disc degeneration. However, a systematic review found that there was low or very low-quality data suggesting that increasing omega-3 may increase lumbar spine BMD but did reveal that is has little or no effect on total bone mass or measures of functional status [1]. Glucosamine is another supplement that has anti-inflammatory properties and has been hypothesized to have cartilage restorative effects [76]. In one randomized controlled study of the effects of glucosamine on low back pain, improvements in quality of life as well as reduction of pain at rest and lumbar stiffness was greater in the group that received glucosamine complex compared with the control group [130]. However, in a larger randomized controlled trial studying the effects of glucosamine in patients with chronic low back pain (LBP) and degenerative lumbar osteoarthritis (OA), no significant difference was found between the glucosamine and placebo group, making it difficult to recommend glucosamine supplementation to patients with chronic LBP and lumbar OA [148]. Many other herbal therapies including *Harpagophytum procumbens* (devil's claw) and *Salix alba* (white willow bark) has been studied for their potential effects



Fig. 1.10 Different supplements

on chronic LBP, but evidence for these substances were moderate quality at best [45]. Therefore, although some dietary supplements and herbal therapies show promising findings of anti-inflammatory, analgesic, and protective effects against the progression of disc degeneration, most of the evidence is low in quality and further investigation is needed before healthcare professionals are able to provide clear recommendations. However, as most of these supplements have mild side effects, they may be worth considering in patients who are unable to comply with traditional medications and therapies (Fig. 1.10).

The Effects of Sleep on the Spine

What is sleep? The operational definition of sleep is that of a natural state characterized by a reduction in voluntary motor activity, a decreased response to stimulation (i.e., increased arousal threshold), and stereotypic posture. Sleep is readily distinguishable from other states of altered "consciousness," such as coma and anesthesia, in that it is easily reversible and self-regulating [44]. Sleep research has shown that sleep consists of different cycles. During wakefulness, the cortical electroencephalogram (EEG) typically contains desynchronized high-frequency,

low-amplitude waves in the 14–30 Hz range (i.e., beta waves), presumably reflecting differences in the timing of processing of cognitive, motor and perceptual functions. During "quiet rest," when the eyes are closed, EEG oscillations predominate in the 8–12 Hz range and are referred to as alpha waves. At the onset of non-rapid eye movement (NREM) sleep, the waves become larger in amplitude (reflecting increased cortical firing synchrony), and the EEG frequency slows [44]. NREM sleep is conventionally subdivided into four stages. Stage 1 sleep usually persists for only a few (1–7) minutes and is associated with a low arousal threshold. Therefore, sleep can be easily discontinued during this stage by softly calling a person's name or quietly closing a door. Stage 1 sleep also occurs as a transitional stage throughout the night. Stage 2 NREM sleep is signaled by sleep spindles or K-complexes on the EEG. This stage continues for about 10–25 min and requires a more intense stimulus to produce arousal. In Stage 3 NREM sleep, high-voltage slow-wave activity accounts for 20–50% of the EEG activity. This stage lasts a few minutes and is transitional to Stage 4. In Stage 4 NREM sleep, high-voltage slow-wave activity accounts for more than 50% of the EEG activity. Stages 3 and 4 are often referred to as slow-wave sleep, delta sleep or deep sleep [18]. During rapid eye movement (REM) sleep, which cycles with NREM sleep, the cortical EEG transitions to a high-frequency, low-amplitude activity that resembles the desynchronized pattern of Stage 1 sleep and wake in humans. In contrast to wake, however, the electrooculogram (EOG) reflects rapid eye movements, and the electromyogram (EMG) evidences profound atonia (of the skeletal muscle tissue, only the extraocular, inner ear and respiratory muscles are unaffected).

Sleep is a vital part of our lives and is essential to good mental and physical health. Experts recommend that adults need 7 or more hours of sleep per night for the best health and wellbeing [142]. The Centers for Disease Control and Prevention (CDC) report that adults who were short sleepers (less than 7 h of sleep per 24-h period) were more likely to report being obese, physically inactive, and current smokers compared to people who got enough sleep (7 or more hours per 24-h period) [22]. A 2016 study found cumulative deficits in sustained attention, working memory/executive function and speed of processing as well as impaired alertness and positive mood in adolescents when their sleep opportunity decreased to 5 h for 7 nights [82]. Sleep has become a public health concern and several industries cater to the notion of a "good night's sleep." For example, hotels have upgraded mattresses, provide a variety of pillow options, and installed blackout shades/curtains, individual temperature controls and sound proofed walls. The airline industry, likewise, provides earplugs, eye masks, socks, and blankets on long trips to maximize the comfort of its passengers and to promote sleep [17].

The quality of sleep, the amount of sleep and comfort during sleep can affect one's musculoskeletal health including the spine. Better sleep at night leads to a greater sense of wellbeing, which may affect the perception of pain during the day [12]. Several factors can affect this including sleep surface and sleep position. Many companies advertise optimal sleep surfaces including soft, firm and medium-firm mattresses. A key issue in state of the art sleep systems (i.e. mattress and supporting structure) is how the optimization of bed design affects the manifestation of sleep in healthy human beings [137]. Although much of the data regarding the optimum

mattress type for sleep is inconsistent, several studies have shown that a medium-firm sleep surface is ideal. A double-blind, randomized, controlled multicenter trial found that after 90 days, patients sleeping on a medium-firm mattress had better pain-related outcomes than those sleeping on the most firm mattress [72]. However, a definitive conclusion that orthopedic (firm) mattresses cause more pain cannot be made because knowledge about the age of the mattress or any softening material applied to the top of the mattress would be needed to firmly reach a cause-and-effect relationship [87]. One study suggests that the age of their participant's beds (9.5 years) may have contributed to a slow progression of poor sleep and musculoskeletal discomfort due to a deterioration of support provided by the beds over the years [62]. Longevity and durability of mattresses are difficult to determine, but the Better Sleep Council suggests that the "life" of a mattress depends on original quality, usage and recommends that the sleep surface be evaluated after 5–7 years [64]. This study supports that medium-firm mattresses are suitable in providing sleep quality and comfort. The participants experienced considerable improvement in quality sleep (73%), shoulder pain (75%), back pain (70%) and joint/muscle stiffness (57%) following 12 weeks of sleeping on the new mattress [64]. Another study by Jacobson found that using a medium-firm surface found immediate and significant improvements in all areas of physical pain, sleep comfort and sleep quality among participants [63] (Fig. 1.11).



Fig. 1.11 Mattress

Sleep position is another factor that can affect spine health. We, as human beings, eventually adopt a habitual and consistent sleep position. Habitual sleep postures may influence the amount of load applied to spinal tissues when sleeping [19]. Sustained non-symmetrical sleep postures can induce structural spinal changes in humans [58]. An inadequate posture while sleeping and the presence of nervous tension can lead to pain [33]. Sleep position is a modifiable factor for spine health. Gracovetsky considers the best sleeping position as one that reduces stress, relaxes muscles decreasing their activity, and promotes a better body balance [50]. Several studies have found that sleep posture can increase or decrease spinal pain, and that addressing sleep posture could reduce the development of spinal pain [19]. The primary positions addressed in studies that investigate the best sleep positions include prone, supine and lateral (or side lying). The prone position tends to be the least favorable sleep position because this position is believed to increase load on spinal tissues, reducing recovery and provoking waking spinal symptoms [50]. Cary's review study found that side lying was the sleep posture that least likely provokes cervical or lumbar symptoms [19]. The lateral position is the most adopted sleeping posture, and it is able to support the human spine correctly when both the sleep system and pillow are well conceived: The spinal column is a straight line when projected in a frontal plane, while natural curves (cervical lordosis, thoracic kyphosis, and lumbar lordosis) are maintained [55]. An epidemiological study established that the side sleep position is significantly protective of waking cervicothoracic symptoms and is associated with significantly higher sleep quality ratings when compared with all other sleep positions [49]. The side lying and supine positions were the sleep postures recommended for those with lumbar spinal pain [33].

The Effects of Stress on the Spine

The term "stress" is a curious term that has numerous meanings and implications. There is the physical definition of stress that is used in physics. The American Heritage Dictionary defines this type of stress as the internal distribution of force per unit of area within a body subject to an applied force or system of forces [105]. However, when discussing the effects of stress on health, we are referring to the physiologic definition of stress. The term "stress," as it is used in the physiologic sense, was coined by Dr. Hans Selye in 1936, who defined it as "the non-specific response of the body to any demand for change" [131]. The relationship between stress and disease is well established. Dr. Selye distinguished acute stress from the total response to chronically applied stressors, terming the latter condition as "general adaptation syndrome" [129]. General adaptation syndrome (GAS) involves the theory that all living organisms can respond to stress and the basic reaction pattern is always the same irrespective of the agent used to produce stress [125]. GAS develops in three stages: the Alarm Reaction, the Stage of Resistance, and the Stage of Exhaustion. Most of the characteristic manifestations of the Alarm Reaction (tissue catabolism, hypoglycemia, gastro-intestinal erosions, discharge of secretory granules from the adrenal cortex, haemoconcentration, etc) disappear or are actually reversed during the stage of resistance, but reappear in the stage of exhaustion. This

suggests that the ability of living organisms to adapt themselves to changes in their surroundings, their adaptability or "adaptation energy," is a finite quantity; its magnitude appears to depend largely on genetic factors. In the biological sense, stress is the interaction between damage and defense, just as in physics tension or pressure represents the interplay between a force and the resistance offered to it [124].

There is a clear connection between various types of stress and pain. The neuromatrix theory provides a reasonable mechanism whereby psychological stresses may provide the basis for chronic pain. Stressors have destructive effects on muscle, skeletal and hippocampal neural tissue, which may become the immediate basis of pain. It is possible that psychological stress alone can become a cause of chronic pain because it produces substances that have destructive effects on body tissues. Prolonged stressful events can leave a memory etched into bone, muscle, and nerve tissue, just as an injury sculpts a neuronal pattern into the neuromatrix [93].

There have been investigations into the relationship between stress and its effects on our spine and musculoskeletal system. Marras et al. identified that a potential pathway between psychosocial stress and spine loading may explain how psychosocial stress increases the risk of low back disorders. The introduction of stress increased muscle activation, hip kinematics and spine loads. This study also found that certain individual factors, such as gender and personality traits, dictate how psychosocial stress manifests itself – with increases in muscle coactivity and spine loading [89]. This finding applies in a variety of situations including the workplace. The National Academy of Sciences has proposed a model in which workplace issues such as physical workplace design (external loading), organizational factors, and social context can interact to influence the body's biomechanical response, thereby, potentially initiating an injury pathway leading to low back pain. Similarly, individual factors inherent to the worker such as personality, size, strength, perception, and the like can interact with the biomechanical system to mediate or exacerbate the influence of the workplace factors on the biomechanical response [103]. Another study found that simultaneous mental processing had a large impact on spine load. In this study, mental processing stress acted as a catalyst for the biomechanical responses, leading to intensified spine loading. They found that mental stress appeared to occur as a function of time pressures on task performance and resulted in less controlled movements and increases in trunk muscle coactivation. These adjustments significantly increased spine loading [32]. Similar findings have been reported for the cervical spine. Mentally demanding tasks performed prior and subsequent to heavy physical exertion significantly increased the activity of the upper trapezius, sternocleidomastoid, and cervical trapezius muscles during the physical exertion [106].

The Effects of Overuse of Technology on the Spine

The technology industry is constantly changing with new and improved products introduced to the marketplace for consumer use. Smartphone use has increased rapidly worldwide over the last several years. In 2019, there were an estimated 3.5 billion smartphone users worldwide. This is expected to rise to 3.8 billion in 2021



Fig. 1.12 Different technology devices

[79]. Text messaging is the most used data service in the world [98]. The number of text messages sent continues to increase. The Cellular Telecommunications and Internet Association (CTIA) reported that 63,000 text messages are sent every second and 5.5 billion text messages are sent per day in the United States [21]. Another source reports that 22 billion text messages are sent every day worldwide [37]. In 2019, the CTIA reported wireless data traffic increased over 82% in 2018 alone and is more data than was used in the first 6 and a half years of the decade combined (from 2010 to 2016) (Fig. 1.12).

In addition to the technology boom in the twenty-first century, the 2020 pandemic due to the SARS-CoV-2 virus (Severe Acute Respiratory Syndrome Coronavirus 2), has forced individuals to use technology on a more regular basis for their jobs, education, healthcare and socialization. Many people all over the globe shifted their job to WFH (Working From Home). A 2020 study showed that the rates of low back pain were higher in people who stayed at home during the lockdown (due to SARS-CoV-2 virus) compared to those who continued to work from the workplace [133]. Schools including universities and colleges had to modify their curriculum to offer online courses. Medical practices incorporated telehealth for patients. Telehealth refers to the entire spectrum of activities used to deliver care at a distance without physical contact with the patient. Telehealth can take place synchronously (telephone and video), asynchronously (patient portal messages), through virtual agents (chatbots) and wearable devices [150]. In May 2020, Forbes magazine reported that virtual events are up by 1000% [71].

This steep rise in personal device use has led to people spending hours a day on some type of mobile device. This can lead to different spine and musculoskeletal overuse issues. Musculoskeletal disorders are caused by sudden exertion or prolonged exposure to physical factors such as prolonged, forceful, low amplitude, repetitive use of such devices [126]. The areas of pain can include not only the neck,

upper back and lower back but also the base of the thumbs, wrists, elbows and shoulders. A 2006 case report from Finland discussed a 48-year old male who developed isolated pain at the base of his thumbs (left more than right) secondary to excessive mobile phone use and active texting. His symptoms resolved after excision arthroplasty of the left carpometacarpal (CMC) joint [96].

A factor that contributes to spine pain while using a personal/mobile device is positioning and posture while using the device. Most mobile device tasks require users to look sharply down or to hold arms out in front of them to read the screen, both of which could lead to fatigue and pain in the neck and shoulders [13]. When using photogrammetry to determine head and cervical postures when viewing a mobile phone screen, subjects display a more head forward posture when viewing a mobile phone screen compared to a neutral standing position [53]. Participants automatically stabilize trunks, tighten their necks and shoulders, breathe shallowly as well as thrust their heads forward in order to read a small screen. In an earlier study, 83% of participants reported neck pain while texting [80]. Text neck is a term that is commonly used for a repetitive stress injury where excessive texting or mobile device use are believed to be the primary cause. Common symptoms associated with text neck include pain in the neck/upper back/shoulder, forward head posture and rounded shoulders, reduced mobility and tightness of the neck/upper back/shoulders, headache and increased pain with neck flexion [35]. The flexion posture of the neck and the weight of the head while using a mobile phone are disturbing the balance of the spine. According to the degree of flexion, the stress in the cervical region increases, loads on discs increase and loss of lordosis and degenerative processes accelerate [108]. A 2014 study quantitatively assessed head flexion during the three common smartphone tasks of text messaging, web browsing and video watching. This study found that head flexion angle was significantly larger for text messaging than the other two tasks. This study also determined that the head flexion angle was significantly larger while sitting than while standing [78]. If these types of devices are used incorrectly for long periods of time, one can develop chronic neck and shoulder pain. The association between smartphone use and chronic neck-shoulder pain was explored by studying the spine while doing three tasks: (1) texting with one hand, (2) texting with two hands and (3) typing on a desktop computer. The results of this study suggest that altered kinematics may be associated with pain during all text-entry tasks due to significantly increased angles of cervical right side flexion and postural changes. Two-handed texting was associated with increased cervical flexion and one-handed texting was associated with an asymmetric neck posture. This study concluded that both text-entry methods are not favorable in terms of spinal posture [151] (Fig. 1.13).

Different applications have been developed which can be downloaded on a smart device. These applications sense the user's head and neck positioning and alerts the user if their positioning is suboptimal. This reminds the user to change and optimize their position to avoid spine and musculoskeletal pain.



Fig. 1.13 Picture of individual incorrectly using mobile device which can lead to "Text Neck"; picture on the right illustrates how to correctly use a mobile device

Conclusions

Many aspects of our lives affect our spine health. Maintaining a healthy lifestyle is important in keeping our spine and bodies in optimal shape. This includes staying active with a regular exercise program, eating healthy, getting enough sleep, being aware of increased stress levels, trying to reduce unnecessary tension and working in the best environments possible in relation to our work stations and use of mobile devices.

References

1. Abdelhamid A, Hooper L, Sivakaran R, Hayhoe RPG, Welch A, PUFAH Group. The relationship between omega-3, omega-6 and total polyunsaturated fat and musculoskeletal health and functional status in adults: a systematic review and meta-analysis of RCTs. Calcif Tissue Int. 2019;105(4):353–72. https://doi.org/10.1007/s00223-019-00584-3.
2. Adams MA, Dolan P. Time-dependent changes in the lumbar spine's resistance to bending. Clin Biomech (Bristol, Avon). 1996;11(4):194–200. https://doi.org/10.1016/0268-0033(96)00002-2. PMID: 11415620.
3. Akhtar MW, Karimi H, Gilani SA. Effectiveness of core stabilization exercises and routine exercise therapy in management of pain in chronic non-specific low back pain: a randomized controlled clinical trial. Pak J Med Sci. 2017;33(4):1002–6. https://doi.org/10.12669/pjms.334.12664.
4. Alexander LA, Hancock E, Agouris I, Smith FW, MacSween A. The response of the nucleus pulposus of the lumbar intervertebral discs to functionally loaded positions. Spine (Phila Pa 1976). 2007;32(14):1508–12. https://doi.org/10.1097/BRS.0b013e318067dccb. PMID: 17572620.

5. Amick BC 3rd, Robertson MM, DeRango K, Bazzani L, Moore A, Rooney T, Harrist R. Effect of office ergonomics intervention on reducing musculoskeletal symptoms. Spine (Phila Pa 1976). 2003;28(24):2706–11. https://doi.org/10.1097/01.BRS.0000099740.87791.F7. PMID: 14673374.
6. Aota Y, Iizuka H, Ishige Y, Mochida T, Yoshihisa T, Uesugi M, Saito T. Effectiveness of a lumbar support continuous passive motion device in the prevention of low back pain during prolonged sitting. Spine (Phila Pa 1976). 2007;32(23):E674–7. https://doi.org/10.1097/BRS.0b013e318158cf3e. PMID: 17978643.
7. Bachmann KN, Bruno AG, Bredella MA, et al. Vertebral strength and estimated fracture risk across the BMI spectrum in women. J Bone Miner Res. 2016;31(2):281–8. https://doi.org/10.1002/jbmr.2697.
8. Barnes PM, Bloom B, Nahin RL. Complementary and alternative medicine use among adults and children: United States, 2007. Natl Health Stat Report. 2008;(12):1–23.
9. Barone Gibbs B, Hergenroeder AL, Perdomo SJ, Kowalsky RJ, Delitto A, Jakicic JM. Reducing sedentary behaviour to decrease chronic low back pain: the stand back randomised trial. Occup Environ Med. 2018;75(5):321–7. https://doi.org/10.1136/oemed-2017-104732. Epub 2018 Jan 12. Erratum in: Occup Environ Med. 2018;75(6):474. PMID: 29330230.
10. Battié MC, Videman T, Gill K, et al. 1991 Volvo Award in clinical sciences. Smoking and lumbar intervertebral disc degeneration: an MRI study of identical twins. Spine (Phila Pa 1976). 1991;16(9):1015–21.
11. Belavý DL, Quittner MJ, Ridgers N, Ling Y, Connell D, Rantalainen T. Running exercise strengthens the intervertebral disc. Sci Rep. 2017;7:45975. https://doi.org/10.1038/srep45975. PMID: 28422125; PMCID: PMC5396190.
12. Bergholdt K, Fabricius RN, Bendix T. Better back by better beds? Spine. 2008;33(7):703–8.
13. Berolo S, Wells R, Amick B. Musculoskeletal symptoms among mobile hand-held device users and their relationship to device use: a preliminary study in a Canadian university population. Appl Ergon. 2011;42:371–8.
14. Bogduk N. Clinical and radiologic anatomy of the lumbar spine. 5th ed. New York: Churchill Livingstone; 2012.
15. Bono C. Low back pain in athletes. J Bone Joint Surg Am. 2004;86(2):382–96.
16. Bredow J, Bloess K, Oppermann J, Boese CK, Löhrer L, Eysel P. Konservative Therapie beim unspezifischen, chronischen Kreuzschmerz: Evidenz der Wirksamkeit – eine systematische Literaturanalyse [Conservative treatment of nonspecific, chronic low back pain: evidence of the efficacy – a systematic literature review]. Orthopade. 2016;45(7):573–8. https://doi.org/10.1007/s00132-016-3248-7. Erratum in: Orthopade. 2016;45(7):627. PMID: 27075679.
17. Capezuti EA. The power and importance of sleep. Geriatr Nurs. 2016;37(6):487–8.
18. Carskadon MA, Dement WC. Monitoring and staging human sleep: Chapter 2 – Normal human sleep an overview. In: Principles and practice of sleep medicine. 5th ed. St. Louis: Elsevier Saunders; 2011. p. 16–26.
19. Cary D, Briffa K, McKenna L. Identifying relationships between sleep posture and non-specific spinal symptoms in adults: a scoping review. BMJ Open. 2019;9:e027633.
20. Cats-Baril WL, Frymoyer JW. The economics of spinal disorders. In: Frymoyer JW, et al., editors. The adult spine. New York: Raven; 1991. p. 85–105.
21. Cellular Telecommunications and Internet Association (CTIA). 2019 Annual survey highlights. 20 Jun 2019. https://ctia.org/news/2019-annual-survey-highlights.
22. Centers for Disease Control and Prevention. Short sleep duration among US adults. Content Source National Center for Chronic Disease Prevention and Health Promotion, Division of Population Health. Last reviewed May 2017. http://cdc.gov/sleep/data_statistics/html.
23. Chaffin DB, Andersson GBJ, Martin BJ. Occupational biomechanics. 4th ed. New York: Wiley; 2006.
24. Chang WD, Lin HY, Lai PT. Core strength training for patients with chronic low back pain. J Phys Ther Sci. 2015;27(3):619–22. https://doi.org/10.1589/jpts.27.619.
25. Cohen SP, Srinivasa RN. Pathogenesis, diagnosis, and treatment of lumbar zygapophyseal (facet) joint pain. Anesthesiology. 2007;106(3):591–614.

26. Coulombe BJ, Games KE, Neil ER, Eberman LE. Core stability exercise versus general exercise for chronic low back pain. J Athl Train. 2017;52(1):71–2. https://doi.org/10.4085/1062-6050-51.11.16.
27. Cramer H, Ward L, Steel A, Lauche R, Dobos G, Zhang Y. Prevalence, patterns, and predictors of yoga use: results of a U.S. nationally representative survey. Am J Prev Med. 2016;50(2):230–5. https://doi.org/10.1016/j.amepre.2015.07.037.
28. Curran M, O'Sullivan L, O'Sullivan P, Dankaerts W, O'Sullivan K. Does using a chair backrest or reducing seated hip flexion influence trunk muscle activity and discomfort? A systematic review. Hum Factors. 2015;57(7):1115–48. https://doi.org/10.1177/0018720815591905. Epub 2015 Jul 14. PMID: 26175544.
29. Dahm KT, Brurberg KG, Jamtvedt G, Hagen KB. Advice to rest in bed versus advice to stay active for acute low-back pain and sciatica. Cochrane Database Syst Rev. 2010;(6):CD007612. https://doi.org/10.1002/14651858.CD007612.pub2. PMID: 20556780.
30. Daily JW, Yang M, Park S. Efficacy of turmeric extracts and curcumin for alleviating the symptoms of joint arthritis: a systematic review and meta-analysis of randomized clinical trials. J Med Food. 2016;19(8):717–29. https://doi.org/10.1089/jmf.2016.3705.
31. Dankaerts W, O'Sullivan P, Burnett A, Straker L. Differences in sitting postures are associated with nonspecific chronic low back pain disorders when patients are subclassified. Spine (Phila Pa 1976). 2006;31(6):698–704. https://doi.org/10.1097/01.brs.0000202532.76925.d2. PMID: 16540876.
32. Davis KG, Marras WS, Heaney CA, et al. The impact of mental processing and pacing on spine loading. Spine. 2002;27(23):2645–53.
33. Desouzart G, Matos R, Melo F, et al. Effects of sleeping position on back pain in physically active seniors: a controlled pilot study. Work. 2015;53:235–40.
34. Devereaux MW. Anatomy and examination of the spine. Neurol Clin. 2007;25(2):331–51.
35. DeWitt D. Text neck symptoms and diagnosis. 26 Oct 2018. https://spine-health.com/conditions/neck-pain/text-neck-symptoms-and-diagnosis.
36. Dieleman JL, Baral R, Birger M, et al. US spending on personal health care and public health, 1996-2013. JAMA. 2016;316(24):2627–46. https://doi.org/10.1001/jama.2016.16885.
37. Domo. Data Never Sleeps 5.0. 2017. https://domo.com/learn/data-never-sleeps-5.
38. Dowdell J, et al. Intervertebral disc degeneration and repair. Neurosurgery. 2017;80(3S):S46–54.
39. Edward WC, LaRocca H. The developmental segmental sagittal diameter of the cervical spinal canal in patient with cervical spondylosis. Spine. 1983;8(1):20–7.
40. Elliott TL, Marshall KS, Lake DA, Wofford NH, Davies GJ. The effect of sitting on stability balls on nonspecific lower back pain, disability, and core endurance: a randomized controlled crossover study. Spine (Phila Pa 1976). 2016;41(18):E1074–80. https://doi.org/10.1097/BRS.0000000000001576. PMID: 27010995.
41. Farfan HF, Cossette JW, Robertson GH, et al. The effects of torsion on the lumbar intervertebral joints: the role of torsion in the production of disc degeneration. J Bone Joint Surg Am. 1970;52:468–97.
42. Fernandez M, Hartvigsen J, Ferreira ML, Refshauge KM, Machado AF, Lemes ÍR, Maher CG, Ferreira PH. Advice to stay active or structured exercise in the management of sciatica: a systematic review and meta-analysis. Spine (Phila Pa 1976). 2015;40(18):1457–66. https://doi.org/10.1097/BRS.0000000000001036. PMID: 2616521.
43. Frymoyer JW. Back pain and sciatica. N Engl J Med. 1988;318:291–300.
44. Fuller PM, Gooley JJ, Saper CB. Neurobiology of the sleep-wake cycle: sleep architecture, circadian regulation, and regulatory feedback. J Biol Rhythm. 2006;21(6):482–93.
45. Gagnier JJ, Oltean H, van Tulder MW, Berman BM, Bombardier C, Robbins CB. Herbal medicine for low back pain: a Cochrane review. Spine (Phila Pa 1976). 2016;41(2):116–33. https://doi.org/10.1097/BRS.0000000000001310.
46. Gatti R, Faccendini S, Tettamanti A, Barbero M, Balestri A, Calori G. Efficacy of trunk balance exercises for individuals with chronic low back pain: a randomized clinical trial. J Orthop Sports Phys Ther. 2011;41(8):542–52. https://doi.org/10.2519/jospt.2011.3413.

47. González-Sánchez M, Luo J, Lee R, Cuesta-Vargas AI. Spine curvature analysis between participants with obesity and normal weight participants: a biplanar electromagnetic device measurement. Biomed Res Int. 2014;2014:935151. https://doi.org/10.1155/2014/935151.
48. Gordon R, Bloxham S. A systematic review of the effects of exercise and physical activity on non-specific chronic low back pain. Healthcare (Basel). 2016;4(2):22. https://doi.org/10.3390/healthcare4020022. PMID: 27417610; PMCID: PMC4934575.
49. Gordon S, Grimmer K, Trott P. Sleep position, age, gender, sleep quality and waking cervicothoracic symptoms. Internet J Allied Health Sci Pract. 2007;5(1):Article 6. Retrieved July 11, 2020. Available from http://ijahsp.nova.edu/articles/vol5num/gordon2.pdf.
50. Gracovetsky S. The resting spine: a conceptual approach to the avoidance of spinal reinjury during rest. Phys Ther. 1987;67(4):549–53.
51. Grondin DE, Triano JJ, Tran S, et al. The effect of a lumbar support pillow on lumbar posture and comfort during a prolonged seated task. Chiro Man Therap. 2013;21(1):21. https://doi.org/10.1186/2045-709X-21-21.
52. Gruenwald J, Petzold E, Busch R, Petzold HP, Graubaum HJ. Effect of glucosamine sulfate with or without omega-3 fatty acids in patients with osteoarthritis. Adv Ther. 2009;26(9):858–71. https://doi.org/10.1007/s12325-009-0060-3.
53. Guan X, Fan G, Wu X, et al. Photographic measurement of head and cervical posture when viewing mobile phone: a pilot study. Eur Spine J. 2015;24:2892–8.
54. Hadjipavlou AG, et al. The pathophysiology of disc degeneration. J Bone Joint Surg Br. 2008;90B:1261–70.
55. Haex B. Back and bed: ergonomic aspects of sleeping. Boca Raton: CRC Press; 2005.
56. Hedman TP, Fernie GR. Mechanical response of the lumbar spine to seated postural loads. Spine (Phila Pa 1976). 1997;22(7):734–43. https://doi.org/10.1097/00007632-199704010-00004. PMID: 9106313.
57. Hemanta D, et al. Etiology for degenerative disc disease. Chin Med Sci J. 2016;31(3):185–91.
58. Hill S, Goldsmith J. Biomechanics and prevention of body shape distortion. Tizard Learn Disabil Rev. 2010;15:15–32.
59. Huang W, et al. Is smoking a risk factor for lumbar disc herniation? Eur Spine J. 2016;25:168–76.
60. Hui SS, Xie YJ, Woo J, Kwok TC. Practicing Tai Chi had lower energy metabolism than walking but similar health benefits in terms of aerobic fitness, resting energy expenditure, body composition and self-perceived physical health. Complement Ther Med. 2016;27:43–50. https://doi.org/10.1016/j.ctim.2016.05.006. Epub 2016 May 20. PMID: 27515875.
61. Iwahashi M, Matsuzaki H, Tokuhashi Y, Wakabayashi K, Uematsu Y. Mechanism of intervertebral disc degeneration caused by nicotine in rabbits to explicate intervertebral disc disorders caused by smoking. Spine (Phila Pa 1976). 2002;27(13):1396–401. https://doi.org/10.1097/00007632-200207010-00005.
62. Jacobson BH, Gemmell HA, Hayes BM, et al. Effectiveness of a selected bedding system on quality of sleep, low back pain, shoulder pain, and spine stiffness. J Manipulative Physiol Ther. 2002;25:88–92.
63. Jacobson BH, Wallace TJ, Smith DB, et al. Grouped comparisons of sleep quality for new and personal bedding systems. Appl Ergon. 2008;39(2):247–54.
64. Jacobson BH, Boolani A, Dunklee G, et al. Effect of prescribed sleep surfaces on back pain and sleep quality in patients diagnosed with low back and shoulder pain. Appl Ergon. 2010;42(1):91–7.
65. Jakoi AM, Pannu G, D'Oro A, et al. The clinical correlations between diabetes, cigarette smoking and obesity on intervertebral degenerative disc disease of the lumbar spine. Asian Spine J. 2017;11(3):337–47. https://doi.org/10.4184/asj.2017.11.3.337.
66. Jaumard NV, Welch WC, Winkelstein BA. Spinal facet joint biomechanics and mechanotransduction in normal, injury and degenerative conditions. J Biomech Eng. 2011;133(7):071010.
67. Katz JN. Lumbar disc disorders and low-back pain: socioeconomic factors and consequences. J Bone Joint Surg Am. 2006;88(Suppl 2):21–4. https://doi.org/10.2106/JBJS.E.01273. PMID: 16595438.

68. Kinkade S. Evaluation and treatment of acute low back pain. Am Fam Physician. 2007;75(8):1181–8.
69. Kirkaldy-Willis WH, Wedge JH, Yong-Hing K, Reilly J. Pathology and pathogenesis of lumbar spondylosis and stenosis. Spine (Phila Pa 1976). 1978;3(4):319–28. https://doi.org/10.1097/00007632-197812000-00004.
70. Koes BW, van Tulder M, Lin CW, Macedo LG, McAuley J, Maher C. An updated overview of clinical guidelines for the management of non-specific low back pain in primary care. Eur Spine J. 2010;19(12):2075–94. https://doi.org/10.1007/s00586-010-1502-y. Epub 2010 Jul 3. PMID: 20602122; PMCID: PMC2997201.
71. Koetsier J. Virtual events up 1000% since covid-19, with 52,000 on just one platform. Forbes Magazine. 27 May 2020.
72. Kovacs FM, Abraira V, Pena A, et al. Effect of firmness of mattress on chronic non-specific low back pain: randomized, double-blind, controlled, multicenter trial. Lancet. 2003;362:1599–604.
73. Kumar SP. Efficacy of segmental stabilization exercise for lumbar segmental instability in patients with mechanical low back pain: a randomized placebo controlled crossover study. N Am J Med Sci. 2011;3(10):456–61. https://doi.org/10.4297/najms.2011.3456.
74. Lafage V, Schwab F, Patel A, Hawkinson N, Farcy JP. Pelvic tilt and truncal inclination: two key radiographic parameters in the setting of adults with spinal deformity. Spine (Phila Pa 1976). 2009;34(17):E599–606.
75. Lam OT, Strenger DM, Chan-Fee M, Pham PT, Preuss RA, Robbins SM. Effectiveness of the McKenzie method of mechanical diagnosis and therapy for treating low back pain: literature review with meta-analysis. J Orthop Sports Phys Ther. 2018;48(6):476–90. https://doi.org/10.2519/jospt.2018.7562.
76. Largo R, Alvarez-Soria MA, Díez-Ortego I, et al. Glucosamine inhibits IL-1beta-induced NFkappaB activation in human osteoarthritic chondrocytes. Osteoarthr Cartil. 2003;11(4):290–8. https://doi.org/10.1016/s1063-4584(03)00028-1.
77. Leboeuf-Yde C, Kjaer P, Bendix T, Manniche C. Self-reported hard physical work combined with heavy smoking or overweight may result in so-called Modic changes. BMC Musculoskelet Disord. 2008;9:5. https://doi.org/10.1186/1471-2474-9-5.
78. Lee S, Kang H, Shin G. Head flexion angle while using a smartphone. Ergonomics. 2015;58(2):220–6.
79. Lin Y. 10 Mobile usage statistics every marketer should know in 2020. Oberlo. 22 Apr 2020. https://oberlo.com/blog/mobile-usage-statistics.
80. Lin I, Peper E. Psychophysiological patterns during cell phone text messaging: a preliminary study. Appl Psychophysiol Biofeedback. 2009;34:53–7.
81. Liuke M, Solovieva S, Lamminen A, et al. Disc degeneration of the lumbar spine in relation to overweight. Int J Obes. 2005;29(8):903–8. https://doi.org/10.1038/sj.ijo.0802974.
82. Lo JC, Ong JL, Leong RL, et al. Cognitive performance, sleepiness, and mood in partially sleep deprived adolescents: the need for sleep study. Sleep. 2016;39(3):687–98.
83. Lundin O, Hellstrom M, Nilsson I, et al. Back pain and radiological changes in the thoraco-lumbar spine of athletes. A long-term follow-up. Scand J Med Sci Sports. 2001;11:103–9.
84. Machado LA, de Souza MV, Ferreira PH, Ferreira ML. The McKenzie method for low back pain: a systematic review of the literature with a meta-analysis approach. Spine (Phila Pa 1976). 2006;31(9):E254–62. https://doi.org/10.1097/01.brs.0000214884.18502.93.
85. Makhsous M, Lin F, Hendrix RW, Hepler M, Zhang LQ. Sitting with adjustable ischial and back supports: biomechanical changes. Spine (Phila Pa 1976). 2003;28(11):1113–21; discussion 1121–2. https://doi.org/10.1097/01.BRS.0000068243.63203.A8. PMID: 12782977.
86. Malmivaara A, Häkkinen U, Aro T, Heinrichs ML, Koskenniemi L, Kuosma E, Lappi S, Paloheimo R, Servo C, Vaaranen V, et al. The treatment of acute low back pain--bed rest, exercises, or ordinary activity? N Engl J Med. 1995;332(6):351–5. https://doi.org/10.1056/NEJM199502093320602. PMID: 7823996.
87. Marin R, Cyhan T, Miklos W. Sleep disturbance in patients with chronic low back pain. Am J Phys Med Rehabil. 2006;85(5):430–5.

88. Marras WS, Lavender SA, Leurgans SE, et al. Biomechanical risk factors for occupationally related low back disorders. Ergonomics. 1995;38(2):377–410.
89. Marras WS, Davis KG, Heaney CA, et al. The influence of psychosocial stress, gender, and personality on mechanical loading of the lumbar spine. Spine. 2000;25(23):3045–54.
90. Mattila VM, Saarni L, Parkkari J, Koivusilta L, Rimpelä A. Early risk factors for lumbar discectomy: an 11-year follow-up of 57,408 adolescents. Eur Spine J. 2008;17(10):1317–23. https://doi.org/10.1007/s00586-008-0738-2.
91. McGill SM, Hughson RL, Parks K. Changes in lumbar lordosis modify the role of the extensor muscles. Clin Biomech (Bristol, Avon). 2000;15(10):777–80. https://doi.org/10.1016/s0268-0033(00)00037-1. PMID: 11050362.
92. McKenzie R, May S. The lumbar spine: mechanical diagnosis and therapy. 2nd ed. Wellington: Spinal Publications; 2003.
93. Melzack R. Chapter 6 – Pain and stress: a new prespective. In: Gatchel RJ, Turk DC, editors. Psychosocial factors in pain: clinical perspectives. New York: The Guilford Press; 1999. p. 89–106.
94. Meng XG, Yue SW. Efficacy of aerobic exercise for treatment of chronic low back pain: a meta-analysis. Am J Phys Med Rehabil. 2015;94(5):358–65. https://doi.org/10.1097/PHM.0000000000000188. PMID: 25299528.
95. Miller KK, Grinspoon SK, Ciampa J, Hier J, Herzog D, Klibanski A. Medical findings in outpatients with anorexia nervosa. Arch Intern Med. 2005;165(5):561–6. https://doi.org/10.1001/archinte.165.5.561.
96. Ming Z, Pietikainen S, Hanninen O. Excessive texting in pathophysiology of first carpometacarpal joint arthritis. Pathophysiology. 2006;13:269–70.
97. Misra M, Klibanski A. Anorexia nervosa and bone. J Endocrinol. 2014;221(3):R163–76. https://doi.org/10.1530/JOE-14-0039.
98. Morreale M. Daily SMS mobile usage statistics. SMS Eagle. 6 Mar 2017. https://smseagle.eu/2017/03/06/daily-sms-mobile-statistics.
99. Murtagh FR, Paulsen RD, Rechtine GR. The role and incidence of facet tropism in lumbar spine degenerative disc disease. J Spinal Disord. 1991;4(1):85–9.
100. Nachemson A. The load on lumbar disks in different positions of the body. Clin Orthop Relat Res. 1966;45:107–22. PMID: 5937361.
101. NaPier Z, Kanim LEA, Arabi Y, et al. Omega-3 fatty acid supplementation reduces intervertebral disc degeneration. Med Sci Monit. 2019;25:9531–7. https://doi.org/10.12659/MSM.918649.
102. Nasto LA, et al. Investigating the role of DNA damage in tobacco smoking-induced spine degeneration. Spine J. 2014;14:416–23.
103. National Academy of Science. Musculoskeletal disorders and the workplace: low back and upper extremities. Washington, DC: National Academy Press; 2001.
104. National Guideline Centre (UK). Low back pain and sciatica in over 16s: assessment and management. London: National Institute for Health and Care Excellence (UK); 2016. PMID: 27929617.
105. Nichols B. The American Heritage dictionary. 5th ed. New York: Houghton Mifflin Harcourt Publishing Company; 2012. p. 812.
106. Nimbarte AD, Al Hassan MJ, Guffey SE, et al. Influence of psychosocial stress and personality type on the biomechanical loading of neck and shoulder muscles. Int J Ind Ergon. 2012;42(5):397–405.
107. O'Leary SA, Paschos NK, Link JM, et al. Facet joints of the spine: structure-function relationships, problems and treatments, and the potential for regeneration. Annu Rev Biomed Eng. 2018;20:145–70.
108. Ogrenci A, Koban O, Yaman O, et al. The effect of technological devices on cervical lordosis. Maced J Med Sci. 2018;6(3):467–71.
109. Oxford University Press. 2020 nutrition. Lexico.com. Available at: https://www.lexico.com/en/definition/nutrition. Accessed 14 July 2020.

110. Pal GP, Routal RV. Transmission of weight through the lower thoracic and lumbar regions of the vertebral column in man. J Anat. 1987;152:93–105.
111. Panjabi MM. The stabilizing system of the spine. Part I. Function, dysfunction, adaptation, and enhancement. J Spinal Disord. 1992;5(4):383–9; discussion 397.
112. Pietilä TA, Stendel R, Kombos T, Ramsbacher J, Schulte T, Brock M. Lumbar disc herniation in patients up to 25 years of age. Neurol Med Chir (Tokyo). 2001;41(7):340–4. https://doi.org/10.2176/nmc.41.340.
113. Pope MH, Goh KL, Magnusson ML. Spine ergonomics. Annu Rev Biomed Eng. 2002;4:49–68. https://doi.org/10.1146/annurev.bioeng.4.092101.122107. Epub 2002 Mar 22. PMID: 12117750.
114. Porter JM, Gyi DE. The prevalence of musculoskeletal troubles among car drivers. Occup Med (Lond). 2002;52(1):4–12. https://doi.org/10.1093/occmed/52.1.4. PMID: 11872788.
115. Qin J, Zhang Y, Wu L, He Z, Huang J, Tao J, Chen L. Effect of Tai Chi alone or as additional therapy on low back pain: systematic review and meta-analysis of randomized controlled trials. Medicine (Baltimore). 2019;98(37):e17099. https://doi.org/10.1097/MD.0000000000017099. PMID: 31517838; PMCID: PMC6750325.
116. Rigotti NA, Nussbaum SR, Herzog DB, Neer RM. Osteoporosis in women with anorexia nervosa. N Engl J Med. 1984;311(25):1601–6. https://doi.org/10.1056/NEJM198412203112503.
117. Robbins JR, Thomas B, Tan L, et al. Immortalized human adult articular chondrocytes maintain cartilage-specific phenotype and responses to interleukin-1beta. Arthritis Rheum. 2000;43(10):2189–201. https://doi.org/10.1002/1529-0131(200010)43:10<2189::AID-ANR6>3.0.CO;2-S.
118. Rozenberg S. Lombalgie chronique, définition et prise en charge [Chronic low back pain: definition and treatment]. Rev Prat. 2008;58(3):265–72. French. PMID: 18536200.
119. Samartzis D, Karppinen J, Chan D, Luk KD, Cheung KM. The association of lumbar intervertebral disc degeneration on magnetic resonance imaging with body mass index in overweight and obese adults: a population-based study. Arthritis Rheum. 2012;64(5):1488–96. https://doi.org/10.1002/art.33462.
120. Saper RB, Sherman KJ, Cullum-Dugan D, Davis RB, Phillips RS, Culpepper L. Yoga for chronic low back pain in a predominantly minority population: a pilot randomized controlled trial. Altern Ther Health Med. 2009;15(6):18–27.
121. Sbriccoli P, Solomonow M, Zhou B, et al. Static load magnitude is a risk factor in the development of cumulative low back disorder. Muscle Nerve. 2004;29(2):300–8.
122. Schoenfeld AJ, et al. Incidence and epidemiology of cervical radiculopathy in the United States military: 2000 to 2009. J Spinal Disord Tech. 2012;25:17–22.
123. Schreuder L, Peters G, Nijhuis-van der Sanden R, Morava E. Aerobic exercise in children with oxidative phosphorylation defects. Neurol Int. 2010;2(1):e4. https://doi.org/10.4081/ni.2010.e4. PMID: 21577340; PMCID: PMC3093217.
124. Selye H. Stress and the general adaptation syndrome. Br Med J. 1950;1(4667):1383–92.
125. Selye H. The general adaptation syndrome. Annu Rev Med. 1951;2:327–42.
126. Sharan D, Ajeesh P. Risk factors and clinical features of text message injuries. Work. 2012;41:1145–8.
127. Sherman KJ, Cherkin DC, Erro J, Miglioretti DL, Deyo RA. Comparing yoga, exercise, and a self-care book for chronic low back pain: a randomized, controlled trial. Ann Intern Med. 2005;143(12):849–56. https://doi.org/10.7326/0003-4819-143-12-200512200-00003.
128. Sherman KJ, Cherkin DC, Wellman RD, et al. A randomized trial comparing yoga, stretching, and a self-care book for chronic low back pain. Arch Intern Med. 2011;171(22):2019–26. https://doi.org/10.1001/archinternmed.2011.524.
129. Tan SY, Yip A. Hans Selye (1907-1982): founder of the stress theory. Singap Med J. 2018;59(4):170–1.
130. Tant L, Gillard B, Appelboom T. Open-label, randomized, controlled pilot study of the effects of a glucosamine complex on low back pain. Curr Ther Res Clin Exp. 2005;66(6):511–21. https://doi.org/10.1016/j.curtheres.2005.12.009.

131. The American Institute of Stress. What is stress. Last reviewed 2020. http://stress.org/what-is-stress.
132. Tilbrook HE, Cox H, Hewitt CE, et al. Yoga for chronic low back pain: a randomized trial. Ann Intern Med. 2011;155(9):569–78. https://doi.org/10.7326/0003-4819-155-9-201111010-00003.
133. Toprak Celenay S, Karaaslan Y, Mete O, et al. Coronaphobia, musculoskeletal pain, and sleep quality in stay-at home and continued-working persons during the 3-month covid-19 pandemic lockdown in Turkey. Chronobiol Int. 2020;37(12):1778–85. https://doi.org/10.1080/07420528.2020.1815759.
134. Uematsu Y, Matuzaki H, Iwahashi M. Effects of nicotine on the intervertebral disc: an experimental study in rabbits. J Orthop Sci. 2001;6(2):177–82. https://doi.org/10.1007/s007760100067.
135. Verbrugghe J, Agten A, Stevens S, Hansen D, Demoulin C, Eijnde BO, Vandenabeele F, Timmermans A. Exercise intensity matters in chronic nonspecific low back pain rehabilitation. Med Sci Sports Exerc. 2019;51(12):2434–42. https://doi.org/10.1249/MSS.0000000000002078. PMID: 31269004.
136. Verbrugghe J, Agten A, Stevens S, Hansen D, Demoulin C, Eijnde BO, Vandenabeele F, Timmermans A. High intensity training to treat chronic nonspecific low back pain: effectiveness of various exercise modes. J Clin Med. 2020;9(8):2401. https://doi.org/10.3390/jcm9082401. PMID: 32727108; PMCID: PMC7465397.
137. Verhaert V, Haex B, De Wilde T, et al. Ergonomics in bed design: the effect of spinal alignment on sleep parameters. Ergonomics. 2011;54(2):169–78.
138. Vo N, Wang D, Sowa G, et al. Differential effects of nicotine and tobacco smoke condensate on human annulus fibrosus cell metabolism. J Orthop Res. 2011;29(10):1585–91. https://doi.org/10.1002/jor.21417.
139. Voet NB, van der Kooi EL, Riphagen II, Lindeman E, van Engelen BG, Geurts ACH. Strength training and aerobic exercise training for muscle disease. Cochrane Database Syst Rev. 2010;(1):CD003907. https://doi.org/10.1002/14651858.CD003907.pub3. Update in: Cochrane Database Syst Rev. 2013;7:CD003907. PMID: 20091552.
140. Waller B, Lambeck J, Daly D. Therapeutic aquatic exercise in the treatment of low back pain: a systematic review. Clin Rehabil. 2009;23(1):3–14. https://doi.org/10.1177/0269215508097856. PMID: 19114433.
141. Wartski M, Williams CM, Broughton NS, et al. Quantifying the lumbar spine movements of surgeons during surgical lists in a teaching hospital. ANZ J Surg. 2019;89(3):153–8.
142. Watson NF, Badr MS, Belenky G, et al. Consensus conference panel-joint consensus statement of the American Academy of Sleep Medicine and Sleep Research Society on the recommended amount of sleep for a healthy adult: methodology and discussion. Sleep. 2015;38:1161–83.
143. Wewege MA, Booth J, Parmenter BJ. Aerobic vs. resistance exercise for chronic non-specific low back pain: a systematic review and meta-analysis. J Back Musculoskelet Rehabil. 2018;31(5):889–99. https://doi.org/10.3233/BMR-170920. PMID: 29889056.
144. White AA, Panjabi MM. Clinical biomechanics of the spine. 2nd ed. Lippincott; 1990.
145. Wieland LS, Skoetz N, Pilkington K, Vempati R, D'Adamo CR, Berman BM. Yoga treatment for chronic non-specific low back pain. Cochrane Database Syst Rev. 2017;1(1):CD010671. https://doi.org/10.1002/14651858.CD010671.pub2.
146. Wilder DG, Pope MH, Frymoyer JW. The biomechanics of lumbar disc herniation and the effect of overload and instability. J Spinal Disord. 1988;1(1):16–32. PMID: 2980059.
147. Wilke HJ, Neef P, Caimi M, Hoogland T, Claes LE. New in vivo measurements of pressures in the intervertebral disc in daily life. Spine (Phila Pa 1976). 1999;24(8):755–62. https://doi.org/10.1097/00007632-199904150-00005. PMID: 10222525.
148. Wilkens P, Scheel IB, Grundnes O, Hellum C, Storheim K. Effect of glucosamine on pain-related disability in patients with chronic low back pain and degenerative

lumbar osteoarthritis: a randomized controlled trial. JAMA. 2010;304(1):45–52. https://doi.org/10.1001/jama.2010.893.
149. Williams K, Abildso C, Steinberg L, et al. Evaluation of the effectiveness and efficacy of Iyengar yoga therapy on chronic low back pain. Spine (Phila Pa 1976). 2009;34(19):2066–76. https://doi.org/10.1097/BRS.0b013e3181b315cc.
150. Wosik J, Fudim M, Cameron B, et al. Telehealth transformation: COVID-19 and the rise of virtual care. J Am Med Inform Assoc. 2020;27(6):957–62.
151. Xie YF, Szeto G, Madeleine P, et al. Spinal kinematics during smartphone texting. Appl Ergon. 2018;68:160–8.
152. Yu ZG, Xu N, Wang WB, Pan SH, Li KS, Liu JK. Interleukin-1 inhibits Sox9 and collagen type II expression via nuclear factor-kappaB in the cultured human intervertebral disc cells. Chin Med J. 2009;122(20):2483–8.

Evaluation of a New Spine Patient

2

Kegan J. Cunniff and G. Sunny Sharma

Introduction

Back and neck pain are among the most prevalent and persistent conditions in the world, and the leading contributor for disability of musculoskeletal origin. In the United States, low back pain alone has an annual prevalence of 10–30% and a lifetime prevalence up to 80% [1]. Similarly, neck pain has an annual prevalence of upwards of 30% and is the fourth leading cause of disability [2]. In this chapter, we will focus on the comprehensive evaluation of patients with spine pain, including obtaining a detailed patient history and performing a thorough physical examination.

An understanding of spinal anatomy is essential in the evaluation and treatment of spine patients. The anatomy of the spine and its related structures is complex given the number and variety of bones, muscles, ligaments, and nerves of which it is comprised. This anatomic complexity along with the heterogeneity of spine conditions can make the diagnosis and treatment of neck and back pain difficult. A complete description of the spinal anatomy is beyond the scope of this chapter; however, we will provide a brief review.

The bony spine is composed of 24 vertebrae, the sacrum, and the coccyx which are anatomically separated into four distinct segments: the cervical, thoracic, lumbar, and sacrococcygeal regions. The cervical spine comprises the spinal components of the neck. Bogduk and Mercer further subdivide the cervical spine into three zones based on their bony structure and anatomical function [3]. The suboccipital zone is comprised of the C1 vertebra, also known as the atlas, and mainly functions as the connection between the skull and bony spine. The structure of the C1 is unique, being ring shaped with two lateral masses that superiorly project articular

K. J. Cunniff · G. S. Sharma (✉)
University of Texas Southwestern Medical Center, Dallas, TX, USA
e-mail: Kegan.Cunniff@UTSouthwestern.edu; Gaurav.Sharma3@utsouthwestern.edu

© The Author(s), under exclusive license to Springer Nature Switzerland AG 2022

C. E. Noe (ed.), *Multidisciplinary Spine Care*,
https://doi.org/10.1007/978-3-031-04990-3_2

processes that interface with the occiput. These processes allow for only a small amount of flexion-extension in terms of motion [3]. The transitional zone, made up of the C2 vertebra (the axis) is a zone where early lateral rotation is achieved. The axis projects an odontoid process, known as the dens, superiorly where it is housed within the atlas forming the medial atlantoaxial joint. The typical zone includes the remaining C3–C7 vertebrae and provides terminal lateral rotation, as well as flexion-extension in both the lateral and anterior-posterior directions [4]. The vertebrae in the typical zone take on the standard anatomical structure that is also replicated throughout the thoracic and lumbar spine. These vertebrae have an anterior vertebral body with an intervertebral disc, two articular pillars united by the laminae in the posterior elements, two transverse processes that extend from the articular pillars, and a dorsally directed spinous process. The articular pillars of consecutive vertebrae communicate superiorly and inferiorly via synovial joints referred to as zygapophyseal or facet joints [4].

The thoracic spine replicates this structure throughout its 12 vertebrae. The defining feature of the thoracic spine is the presence of the ribs which articulate with the vertebral body, intervertebral disc, and transverse processes. The thoracic region functions in providing the support and flexibility of the chest region [4].

The main function of the lumbosacral spine is to support the upper body and transmit loads to the pelvis and lower extremities. It also adds additional range of motion between the thoracic spine and pelvis. The vertebrae of the lumbar segment display the typical structure as described above. However, these vertebrae are significantly larger which allow them to transmit greater axial loads. The natural lordosis of the lumbar spine increases the load-bearing tolerance of the region as well [4]. The sacrococcygeal region makes up the caudal terminus of the spine. The sacrum is a large triangular bone comprised of five fused vertebrae. It articulates laterally with the two ilia, caudally with the coccyx and superiorly with the last lumbar vertebra. The final lumbar vertebra is typically L5, however, transitional anatomy may be present in some patients where the L5 vertebra is fused to the sacrum (“sacralized”) or the S1 vertebral body does not fuse with the sacrum (“lumbarization”).

The nervous system of the spine includes central and peripheral components. The central nervous system of the spine is comprised of the spinal cord which lies within the central canal of the vertebral column. Projecting from the spinal cord are 31 pairs of nerve roots which serve as the beginning of the peripheral nervous system. These nerve roots are named based upon their exiting location from the spinal column. In the cervical spine, nerve roots exit above their correspondingly numbered vertebra. For example, the C5 nerve root exits above the C5 vertebra. There is a transition zone between the C7 and T1 vertebrae, where the C8 nerve root exits. The remaining spinal nerve roots exit below their correspondingly numbered vertebra. For example, the T6 nerve root exits below the T6 vertebra in the neuroforamen formed between T6 and T7. This naming convention holds true for the remainder of the spinal cord as it travels inferiorly until the final nerve root pair, Co1, which exits at the coccyx.

Patient History

A thorough patient history is an important component of any new patient evaluation. This is essential in patients with spine pathology given these conditions can present similarly. Given the complex and multifactorial nature of spinal complaints, it can be helpful in clinical decision making to use the history to divide patients into three categories: (1) patients with nonspecific spine pain, (2) spine pain associated with radiculopathy or spinal stenosis, or (3) spine pain potentially associated with red flag symptoms or other specific causes [5]. Appropriate clinical management for these categories is discussed later in this chapter.

An adequate initial history should gather a variety of information regarding the patient's symptoms. Onset and duration of symptoms can provide insight into the acuity of the condition, which can be helpful in diagnosis and management. For example, a patient with a 5-day history of axial lumbar pain that began suddenly while lifting a heavy object is more likely to have an acute strain or sprain than an elderly patient with a 20-year history of chronic lumbar pain. Note that onset of pain not only captures when symptoms began, but also the nature of how the pain began. The character and radiation of pain can aid in determining which structures are generating pain. Lancinating, burning, or electric-type pain that radiates down a leg is more suggestive of lumbar radiculopathy, whereas localized aching or dull low back pain is more suggestive of facet or SI joint mediated symptoms. Inquiring about alleviating and aggravating factors can also aid in identifying possible pain generators. For example, pain from spinal stenosis classically improves with forward flexion and worsens with extension. On the other hand, pain from a herniated intervertebral disc may worsen with forward flexion and improve with extension. Severity of pain can be subjectively evaluated using pain scales, such as the Visual Analog Scale (VAS) or Numerical Rating Scale (NRS). The patient's occupation and prior level of independence with activities of daily living (ADLs) can also add helpful context to the patient's history.

Red Flag Symptoms

While gathering a patient's history, the clinician should be on the lookout for any alarming "red flag" symptoms or factors that may indicate serious or urgent underlying pathologies. A history of malignancy is the leading risk factor for painful bony metastases [1]. History of immunosuppression, intravenous drug use, recent spinal injection, epidural catheters, and constitutional symptoms such as fevers, chills, and malaise should raise suspicion for infection. Bowel incontinence, urinary retention, gait instability, and saddle anesthesia can indicate spinal cord compression or cauda equina syndrome [1]. History of prolonged morning stiffness, skin rashes, iritis, urethritis, colitis, peripheral joint involvement, and family history of rheumatologic conditions should raise concern for an underlying autoimmune or inflammatory etiology, such as ankylosing spondylitis or psoriatic arthritis. Other red flag signs

Table 2.1 Examples of "red flag" signs from patient history or physical examination

Red flag signs
Fever, night sweats or systemic symptoms
Recent spinal injection
Intravenous drug use
Indwelling epidural catheters
History of malignancy
Immunocompromised condition
Unintentional weight loss
History of systemic steroid use
Spinal deformity
Age of onset less than 18 or greater than 55 years
Inflammatory symptoms or family history concerning for rheumatologic conditions
Bowel or bladder dysfunction
Gait instability
Saddle anesthesia
Focal neurologic signs
Upper motor neuron signs
Muscle atrophy
Nonmechanical pain (pain with little or no movement)
Traumatic injury

include age of onset before age 18 or after age 55, nonmechanical symptoms (pain is not affected by movement), nocturnal pain, past trauma, sudden or progressive limb weakness, spasticity, or atrophy [6]. Common red flag signs and symptoms are listed in Table 2.1.

It is also important during the course of the initial evaluation to gather information on the patient's attitude and behaviors towards their symptoms and recovery as these can negatively influence the patient's prognosis. Examples of these attitudes and behaviors include a lengthy time off from work due to symptoms, catastrophic thinking, impaired sleep, avoidance of usual activities, subjective stress, anxiety, and fear that activity/exercise will worsen back symptoms [7]. Extensive education and expectation setting should be performed for patients who display these attitudes as they are associated with poorer performance on functional testing, more severe pain at follow up visits, and increased time off work or on disability. Additionally, psychologic evaluation and/or treatments should be considered in these patients, particularly prior to invasive or irreversible interventions [8–10].

Physical Examination

After a thorough patient history has been obtained, the physical examination is vital in the assessment of the new spine patient and developing the differential diagnosis. The examination should be comprehensive, but should also be guided by the history obtained from the patient. In addition to general observations, an adequate spine exam should include inspection, palpation, range of motion assessment, and neurological testing. Special tests and provocative maneuvers should then be performed

as indicated by the patient's history or other examination findings. Additionally, the examiner should briefly evaluate the patient's mental status by assessing level of consciousness, orientation, behavior, mood, and affect [11].

Inspection

Inspection of the spine patient should begin with examination of the skin, including the upper and lower extremities. Findings such as abrasions, swelling, bruises, or lacerations may indicate recent trauma. Scars may indicate old trauma or prior surgical intervention. Erythema, rashes, or discoloration may indicate infectious or inflammatory processes. Next, observe for any deformities or postural abnormalities. Large dimples, hairy patches, or dark spots over the spine are common deformities associated with spina bifida occulta, for example. The patient's spinal alignment should be assessed for scoliosis, kyphosis, or exaggerated lordosis.

The muscular structures of the spine and extremities should be assessed for bulk and symmetry. A variety of etiologies can cause asymmetry or irregularities in muscle group bulk, such as tendon tears, nerve injuries, or neuromuscular disorders. For example, unilateral muscular atrophy along a spinal root's myotome may suggest chronic or severe radiculopathy due to nerve root compression. As such, identifying myotomal muscle atrophy can be useful in localizing affected nerve roots. More diffuse and symmetrical atrophy, muscle fasciculations or tremors could indicate underlying neuromuscular pathology.

Palpation

Palpation of the spine regions can be done with the patient seated or standing, but is best performed with the patient lying prone in order to maximally relax the musculature. The examiner should systematically palpate the bony landmarks and soft tissues of the cervical, thoracic, and lumbar spine, as well as any nearby structures. In the examination of cervical spine pain, the shoulder musculature, periscapular region, and upper ribs should be palpated as musculoskeletal syndromes affecting these regions have pain patterns that often overlap with cervical etiologies. Similarly, there is significant overlap in pain arising from the lumbar region, sacroiliac region, hip region, and iliotibial band region that may be differentiated by tenderness to palpation. Pain with palpation or percussion of the spinous processes can be seen in patients with vertebral compression fractures, abscesses, or metastases [1, 6]. Tenderness or trigger points in the paraspinal soft tissues may indicate myofascial or referred pain from facetogenic etiology [12].

Range of Motion

In assessing the range of motion, it is important to evaluate range of motion of the axial spine as well as the upper and lower extremities. Where appropriate, patients

Table 2.2 Normal ranges of motion of the spine, shoulder, and hip

Joint	Range of motion	Degrees
Neck	Flexion	0–45
	Extension	0–25
	Rotation	0–35
	Lateral flexion	0–45
Shoulder	Flexion	0–180
	Extension	0–60
	Abduction	0–180
	Internal rotation	0–70
	External rotation	0–90
Thoracolumbar	Flexion	0–80
	Extension	0–25
	Rotation	0–35
	Lateral flexion	0–45
Hip	Flexion	0–120
	Extension	0–30
	Abduction	0–45
	Adduction	0–30
	Hip rotation	0–45
	Hip rotation	0–45

Source: American Academy of Orthopaedic Surgeons [14]

should be asked to actively range the evaluated joints. If the patient is unable to achieve a normal range of motion, the examiner should then attempt to passively range the joint in order to differentiate between weakness and mechanical impediment.

There are multiple methods to evaluate the range of motion of the spine. Some examples include single or double inclinometry, goniometry, Schober's skin distraction test, measuring distance of fingers from the floor, and radiographic measurement [13]. Please see Table 2.2 for normal ranges of motion of the spinal regions, as well as the nearby hips and shoulders [14].

Neurological Testing

Because of the anatomy of the spine and its neurological contents, the physical examination of the spine must include neurological testing and a mechanical examination of the upper and/or lower extremities. The examiner should assess the strength, sensation, and reflexes of the limbs as well as range of motion and provocative testing of the nearby joints, such as the hips or shoulders.

There are a number of ways to manually test the strength of the limbs during the neurological exam of the spine. The most common method used is that of the Medical Research Council Scale in which the muscles responsible for specific joint movements are measured on a zero to five point scale [15]. The test joints are isolated and then tested to see their strength in opposition to gravity and resistance applied by the examiner. Zero points denotes the absence of any muscle activity. One point indicates flickers of activity without movement of the joint. Two points

Table 2.3 Motor examination key nerve roots, muscle groups and associated muscle action

Nerve root	Key muscles	Muscle action
C5	Biceps, brachialis	Elbow flexion
C6	Extensor carpi radialis longus and brevis	Wrist extension
C7	Triceps	Elbow extension
C8	Flexor digitorum profundus	Finger flexion
T1	Abductor digiti minimi	Small finger abduction
L2	Iliopsoas	Hip flexion
L3	Quadriceps femoris	Knee extension
L4	Tibialis anterior	Ankle dorsiflexion
L5	Extensor hallucis longus	Toe extension
S1	Gastrocnemius, soleus	Ankle plantarflexion

indicates the ability to move the tested joint with gravity eliminated. Three points indicates the ability to move the joint against gravity but not with any additional resistance applied. Four points indicates the ability to move the tested joint against gravity and moderate resistance from the examiner. Five points indicates full strength against examiner resistance [15]. Using the key muscle groups described in the International Standards for Neurological Classification of Spinal Cord Injury (ISNCSCI) examination, the examiner can localize affected nerve roots [15]. For example, testing the strength of the triceps assesses the C7 nerve root. Refer to Table 2.3 for a list of key muscle groups used in manual muscle strength testing.

Sensory testing should be performed regionally based upon the portion of the spine affected. Sensory deficits in a dermatomal pattern can help localize lesions to the corresponding spinal level or nerve root. The appropriate dermatomal testing points are generally consistent with those used in the ISNCSCI examination [16, 17]. For example, decreased sensation in the thumb region might suggest an underlying C6 radiculopathy or peripheral nerve pathology [16]. In the lumbar region, decreased sensation along the lateral calcaneal region may suggest an S1 radiculopathy [16]. Light touch can be assessed with a soft cotton swab or similar material. Sharp and dull discrimination can be performed with a small and sharp object such as a safety pin. Proprioception is best assessed by passively moving the fingers and/or toes up or down while the patient's eyes are closed and asking the patient which direction the digit was moved [18].

Deep tendon and upper motor neuron reflexes can be of great aid to the examiner and help guide diagnostic workup. The grading of deep tendon reflexes is described in Table 2.4. Note that reflexes graded 1+ or 3+ can be normal variants in some patients. These grades would be considered abnormal if found only unilaterally or in patients previously found to have 2+ reflexes. For example, hyperreflexia can assist in the identification and localization of an upper motor neuron lesion. The Babinski reflex, Hoffmann's sign, and clonus are also exam findings suggestive of upper motor neuron involvement [11]. These findings may prompt and support ordering advanced diagnostic imaging such as an MRI. In the setting of known spinal canal stenosis, these findings may warrant surgical referral for further evaluation of underlying myelopathy and possible decompression. Conversely,

Table 2.4 Deep tendon reflex grading scale

Grade	Muscle response
0	No response (areflexia)
1+	Diminished hypoactive response
2+	Expected response (normal)
3+	Brisk hyperactive response
4+	Brisk hyperactive response with clonus

Table 2.5 Commonly tested deep tendon reflexes and their primary nerve root innervation

Deep tendon reflex	Nerve root
Biceps	C5
Brachioradialis	C6
Triceps	C7
Patellar	L4
Medial hamstring	L5
Achilles	S1

hyporeflexia or areflexia may indicate a lower motor neuron lesion [11]. For example, a depressed unilateral patellar reflex may suggest compression of the L4 nerve root. Commonly tested deep tendon reflexes and their associated spinal level may be referenced in Table 2.5.

Functional Testing

Myotomal weakness can be further evaluated with functional screening tests, such as toe and heel walking. Difficulty with toe walking due to calf muscle weakness may indicate radiculopathy affecting the S1 nerve root, whereas difficulty with heel walking may indicate an L4–5 radiculopathy resulting in tibialis anterior muscle weakness [19, 20]. Difficulty with sit to stand transfers may also indicate lower extremity weakness [18]. Trendelenburg sign on single leg standing often signals weakness of the contralateral hip abductors innervated by the L4, L5 and S1 nerve roots [20, 21].

Evaluation of gait is another test that can provide insight into a patient's overall functional status. Take notice of the patient's balance, walking speed, gait pattern, stride length, and posture. Certain gait patterns may indicate underlying spinal pathology. For example, a steppage gait may be due to foot drop from an L4–5 radiculopathy, while an ataxic gait may indicate cerebellar dysfunction or proprioceptive abnormalities [18].

Special Maneuvers and Provocative Tests

We will review common special maneuvers and provocative tests for the spine exam that can assist clinicians in the evaluation and diagnosis of spine conditions. Some common exam maneuvers and their associated sensitivities and specificities may be seen in Table 2.6.

Table 2.6 Spine special examination maneuvers and their associated sensitivities and specificities

Exam maneuver	Positive finding	Pathology	Sensitivity (%)	Specificity (%)
Spurling	Reproduction of cervical radicular pain	Cervical radiculitis	38–97	89–100
Bakody	Decrease of cervical radicular pain	Cervical radiculitis	17–44	92–100
Cervical distraction	Decrease of cervical radicular pain	Cervical radiculitis	43–44	90–100
Straight leg raise	Reproduction of lumbar radicular pain	Lumbar radiculitis	52	89
Seated slump	Reproduction of lumbar radicular pain	Lumbar radiculitis	84	89
Facet load	Reproduction of axial lumbar pain	Lumbar facet arthropathy	34.5–45.5	46.9–47.2
Gaenslen's	Reproduction of pain over SI joint/PSIS	SI joint dysfunction	31	94
Thigh thrust	Reproduction of pain over SI joint/PSIS	SI joint dysfunction	90.70	76.40

Cervical Region

Spurling

The Spurling test is utilized in the evaluation of patients in whom cervical radiculopathy is suspected, and attempts to replicate radicular symptoms by compressing the neuroforamen around the cervical nerve roots. The Spurling test is performed with the patient sitting upright. The patient's neck is placed in slight extension and then ipsilaterally flexed and rotated, with respect to the patient's radicular symptoms. The examiner than compresses the cervical spine by applying a downward directed light pressure, approximately 15 lbs., through the top of the skull [22–24]. A positive Spurling test is denoted by reproduction of the patient's radicular symptoms on compression. While this test has been found to have variable sensitivity for cervical radiculopathy on systematic review, its specificity has been reported to range between 89% and 100% [25, 26].

Distraction

The axial manual traction test, or cervical distraction, also functions in the evaluation of cervical radiculopathy. The test is performed with the patient lying supine. Then, 10–15 kg of superiorly-directed traction is applied to the cervical spine by the examiner pulling from the base of the occiput [24]. A positive tested is denoted by a decrease in the patient's usual radicular symptoms, likely as a result of decreasing nerve root impingement at the neuroforamen. The axial manual traction test has relatively poor sensitivity, but excellent specificity [25, 27].

Bakody

The shoulder abduction test, commonly known as Bakody's sign, is performed with the patient sitting upright. The patient is then directed to abduct their shoulder until their hand or forearm is resting on the top of their head [24]. Resolution or improvement in radicular symptoms is considered a positive test and thought to alleviate pain by decreasing neural tension across irritated nerve roots. The shoulder abduction test has been found to have relatively low sensitivity, but high specificity in prior studies [28].

Upper Limb Tension Test

The upper limb tension test (ULTT) is a sensitive, but poorly specific examination maneuver in the assessment of cervical radiculopathy and brachial plexus involvement. The maneuver is performed in slightly different ways depending on the target nerve being tested. One way involves placing the patient in a supine position. Next, the examiner depresses the scapula while externally rotating and abducting the shoulder to roughly 100°, followed by extension of the wrist and fingers with the elbow in flexion. With the wrist and fingers held in extension, the elbow is passively extended while the patient laterally flexes the neck to the contralateral side [24]. A positive test is denoted by reproduction of the patient's radicular symptoms, a decrease in symptoms with ipsilateral neck flexion, an increase in symptoms with contralateral neck flexion, and/or if there is greater than 10° difference in elbow extension between the patient's arms [24]. Wainner et al. found the ULTT to be a highly sensitive exam maneuver in the detection of cervical radiculopathy, but only achieved 22% specificity [28].

Valsalva

The Valsalva maneuver can also be a highly specific examination maneuver in the assessment of cervical radiculopathy. The procedure is performed with the patient sitting upright. Direct the patient to take a deep breath, hold it, and then bear down [24]. Reproduction of the patient's radicular pains denotes a positive test. This test boasts a high specificity at 94%, but was found to only have a sensitivity of 22% [28].

Lumbosacral Region

Straight Leg Raise

The straight leg raise is a specific test aimed at identifying lumbar nerve root compression or impingement through the application of neural tension. The test is performed with the patient lying supine. The examiner passively flexes the hip by lifting the straightened leg from the ankle. The hip should be flexed to up to 90°, or as maximally tolerated by the patient. A positive straight leg raise is defined as reproduction of lower extremity radicular symptoms radiating from the lower back to the ankle. If stretching pain is produced in the posterior thigh, this is not a positive straight leg raise and is more consistent with hamstring tightness [1, 29]. The sensitivity and specificity were reported by Majlesi et al. to be 52% and 89% respectively

[30]. If while performing the straight leg raise there are radicular symptoms reproduced in the contralateral leg, this is referred to as a positive crossed straight leg raise maneuver. This test has a lower sensitivity of 28% when compared to the straight leg raise, but boasts a similar specificity of 90% [31].

Seated Slump Test

The seated slump test is another neural tension test used in the identification of lumbar nerve root compression responsible for radicular symptoms. To perform the test, the patient is seated upright at the edge of an exam table. The patient is then instructed to slump the thoracolumbar spine by flexing forward while looking straight ahead. Next, fully flex the cervical spine and then instruct the patient to fully extend one knee. Finally, instruct the patient to maximally dorsiflex the ankle of the extended leg. Reproduction of the patient's radicular pain is considered a positive test. Extension of the neck after production of pain may reduce the provoked pain by reducing neural tension. The test should be repeated on the opposite leg, as well. While similar in specificity to the straight leg raise, the slump test boasts a superior sensitivity in one study at 84% [30].

Gaenslen's

Gaenslen's test is a high specificity, low sensitivity examination maneuver used in the identification of pain stemming from the sacroiliac joints [32]. This test is performed with the patient lying supine. The asymptomatic hip is maximally flexed by having the patient pull their knee to their chest, and the symptomatic hip is maximally extended by the examiner gently lowering the leg off the side of the exam table and applying downward pressure on the extended leg [1]. A positive Gaenslen's test is denoted by the reproduction of pain at the sacroiliac joint on the symptomatic side. Both sides should be tested.

Facet Loading

This exam maneuver has many names. Facet loading, Kemp's test, or Extension-Rotation test, is an examination maneuver designed to reproduce a patient's lumbar facetogenic symptoms by increasing load-bearing through the facet joints. To perform the test, the examiner guides the patient into extension, lateral flexion, and lateral rotation towards the symptomatic side of the spine being tested. Overpressure is then applied by the examiner through the ipsilateral shoulder with one hand while stabilizing the ipsilateral ileum with the other hand. The examination is then performed on the opposite side. A positive test is denoted as a reproduction of the patient's axial lumbar pain [33]. In patients with radicular symptoms, this maneuver may also reproduce radiating symptoms if facet loading compromises the nerve root at the neural foramen. This test has been widely used in the initial evaluation of facet arthropathy. Although some studies have boasted sensitivity as high as 100%, Stuber et al. showed a sensitivity and specificity of 34.5–45.5% and 46.9–47.2%, respectively [34]. A similar facet loading maneuver may be used in the cervical region to evaluate for cervical facet arthropathy.

FABER Test

The Flexion Abduction External Rotation (FABER) test, also known as Patrick's sign, can be used in both the evaluation of intraarticular hip pathology and sacroiliac joint arthropathy. This test is performed with the patient lying in a supine position. The examiner places the patient's leg on the symptomatic side in a figure-four position by flexing the hip and knee and then placing the lateral malleolus of the flexed leg on top of the knee of the contralateral extended leg. Next, the hip of the flexed leg is externally rotated and abducted by directing the flexed knee toward the exam table. The examiner then adds additional external rotation with the application of gentle pressure through the medial aspect of the flexed knee. A positive test is indicated by reproduction of pain in the region of the SI joint on the symptomatic side. In the detection of SI joint arthropathy, a positive Faber test was found to have a sensitivity of 31% and specificity of 94% [32].

Posterior Superior Iliac Spine (PSIS) Joint Distraction

The PSIS distraction test is a newer examination maneuver used in the assessment of SI joint arthropathy. The test can be performed with the patient either standing or lying prone. From this position, a medial-to-laterally directed force is applied to the PSIS and is considered positive with reproduction of the patient's SI joint pain. This more recently developed examination maneuver has been found to have sensitivity as high as 100% and specificity of 94% [32].

SI Compression/Distraction

The SI joint compression test is performed with the patient in a side-lying position with the hips flexed to approximately 45° and knees flexed to 90°. The examiner, standing behind the patient, applies pressure through the iliac crest toward the contralateral iliac crest. A positive test is denoted by provocation of the patient's usual pain [35]. A 2009 systematic review found the SI joint compression test to have a sensitivity of 62.8% and specificity of 69.2% [36].

The SI joint distraction test is performed with the patient lying supine. With both hands on the patient's anterior superior iliac spines, the examiner applies pressure in a posterolateral direction which stresses the anterior sacroiliac ligaments [35]. Again, reproduction of the patient's pain denotes a positive test. SI joint distraction has a reported sensitivity 60% and specificity of 81% [37].

Thigh Thrust

To perform the thigh thrust, begin with the patient in a supine position. Next, flex the patient's hip to 90° with the knee remaining relaxed. With one hand placed between the patient's sacrum and the examination table, the examiner applies downward force through the long axis of the femur. This test should be repeated on the contralateral side [38]. Reproduction of the patient's pain is considered a positive sacroiliac joint test and has been determined to have a sensitivity of 90.7% and specificity of 76.4% on systematic review [36].

General Overview of Clinical Decision Making

As mentioned previously, clinical decision making in the initial evaluation of the patient with spine pain can be improved by stratifying patients into one of three categories: patients with nonspecific spine pain, spine pain with symptoms of radiculopathy or spinal stenosis, and spine pain associated with red flag symptoms or other specific conditions.

Nonspecific Spine Pain

In patients who have back and neck pain for less than 4 weeks without signs or symptoms of radiculopathy, spinal stenosis, functional impairment, or red flags on history and examination, a conservative approach is appropriate for initial management. Encourage patients to maintain an active lifestyle and provide education on diet, posture, and a home exercise program. If not recently completed, a referral to physical therapy is recommended. For pain control, counsel the patient on nonpharmacologic pain relief options such as superficial heat, topical analgesic ointments (i.e. Bengay, Salonpas, etc.), massage, transcutaneous electrical nerve stimulation (TENS), posture aids, and activity modification. In terms of medications, acetaminophen is recommended as a first-line analgesic due to its favorable side effect profile. If insufficient in providing relief, nonsteroidal anti-inflammatory drugs (NSAIDs) and muscle relaxants can be effective in the short term and can be considered in patients without contraindications to either medication class [5]. Opioid analgesics may be considered in patients with severe pains refractory to the prior medications, but should be used cautiously. Advanced imaging or invasive procedures are usually not recommended at this initial evaluation [5].

Ideally, the patient should follow-up in 4 weeks to assess for improvement in symptoms. If the patient's symptoms are improving, continue conservative management, provide further education, and follow up as necessary. If there is no improvement in symptoms or the patient subsequently develops signs of radiculopathy, spinal stenosis, or red flag symptoms, imaging studies should be considered. MRI is the preferred imaging method to evaluate for disc or nerve root pathology. However, CT can be considered in scenarios in which MRI is contraindicated or unavailable. Additionally, standing x-rays with flexion and extension views can provide insight into the structural stability of the spine [5].

Radiculopathy and Spinal Stenosis

In patients presenting with radiculopathy or neurogenic claudication concerning for spinal stenosis, initial management in the acute phase (<4 weeks) is similar to that of nonspecific spinal pain described above. Although more concerning than nonspecific pain, evidence suggests that initial management with conservative self-care techniques is safe and appropriate [29]. Emphasis is again placed on education,

self-care, activity modification, conservative pain management, and physical therapy. Medications to reduce neurogenic symptoms, such as gabapentin or pregabalin, are an additional treatment option in the initial management of this population as well.

Imaging is not typically recommended in the acute symptom phase, however, should be considered if there are neurological deficits present or red flag symptoms [29]. Patients should be reevaluated after 4 weeks of conservative management. If the patient's symptoms persist or worsen, imaging should be considered for those who are candidates for interventions such as epidural steroid injections or surgery. MRI is again the imaging modality of choice for these patients, but CT scan is an acceptable alternative when MRI is not appropriate.

Red Flags and/or Specific Conditions

For patients with red flags on history or physical examination or for those in whom a specific diagnosis is suspected, a more aggressive diagnostic work-up should be pursued. Examples of specific diagnoses may be suspected vertebral compression fracture, spinal infection, malignancy, cauda equina syndrome, or myelopathy to name a few. The goal of this work-up is to rapidly identify and treat the underlying etiology.

Advanced imaging is recommended in the initial evaluation of these patients, with MRI being the preferred imaging modality for the initial evaluation. For patients with progressive focal neurologic deficits or concern for conditions such as cauda equina syndrome or compression fractures, non-contrast MRI is appropriate. Those patients with red flags suggestive of infection or malignancy should undergo MRI of the spine with and without contrast. CT or CT myelogram are appropriate alternatives in those unable to undergo MRI.

In addition to imaging, laboratory testing should be considered to help narrow or confirm a suspected diagnosis. Complete blood count, basic metabolic panel, c-reactive protein, and erythrocyte sedimentation rate are appropriate in most cases [5]. Human leukocyte antigen B27 and serum inflammatory markers should be checked in those whom autoimmune or inflammatory processes are suspected. For patients in whom multiple myeloma is suspected, serum protein electrophoresis should be performed. Furthermore, electromyography and nerve conduction studies can provide additional information and aid in the diagnosis of neuropathic or myopathic disorders [5].

Common Clinical Syndromes

After the appropriate work-up is performed and a specific diagnosis is made, treatment targeted at that diagnosis may be considered. We will discuss a brief overview of some common presenting conditions and pain generators of the spine. Some of these topics will also be discussed in greater detail later in this textbook.

Myotendinous Pain

Strains and Sprains

Injury to the muscles and ligaments of the cervical spine can be caused by a variety of mechanisms. In the neck, myotendinous injuries account for the majority of neck pain caused by traumatic injury. These injuries are most commonly caused by motor vehicle accidents or sports injuries, but can be caused by any trauma which places extreme stress on the neck musculature, causing a cervical strain. A sprain of the musculature occurs when these stresses cause an excessive stretching or tearing of ligaments [39, 40].

In the lumbosacral spine, sprains and strains are also typically preceded by a traumatic injury. Classically, these injuries tend to occur when lifting a heavy object or with truncal twisting while holding a heavy object. Pain from these injuries is usually limited to the lumbosacral region and does not radiate into the extremities. Additionally, the lumbosacral paraspinal musculature is typically sore and tender to palpation. Over 90% of patients with a low back strain/sprain recover after 12 weeks with conservative management with NSAIDs, muscle relaxers, and activity modification [41].

Myofascial Pain Syndrome and Trigger Points

Myofascial pain syndrome (MPS) is a prevalent and painful condition involving the musculature and its surrounding fascia. It typically manifests as a tight band of muscle with a discretely palpable and tender nodule within the muscle band referred to as a trigger point [42, 43]. MPS often manifests with both regional and referred pain patterns, and reproduction of these pain patterns on palpation of the trigger point is a hallmark finding [43]. Myofascial trigger points and their pain patterns have been most extensively catalogued by Travell and Simmons in their textbook about myofascial pain [44].

Arthropathy

Facet Arthropathy

The zygapophyseal joints, also known as facet joints, are the diarthrodial joints that articulate between adjacent spinal levels. The facet joints and their corresponding intervertebral discs form a “three-joint complex” that allows for the motion of the spine. Facet arthropathy is a common cause of back and neck pain and is seen in association with degenerative disc disease given the relationship of facet joints and intervertebral discs in regards to spinal motion. Facet arthropathy is most commonly due to osteoarthritis, but can have other etiologies. Rheumatoid arthritis has been seen to affect the cervical spine joints. Spondyloarthritis such as ankylosing

spondylitis or psoriatic arthritis can also be implicated in facet mediated pain conditions. Pain from the facet joint typically manifests as a dull neck or back pain. Referred pain patterns from facet arthropathy are also common and the distribution of referred symptoms depends on the affected facets, with overlap seen between adjacent facet joints. Pain from the upper cervical facet joints can refer to the face and occipital region of the head, sometimes manifesting as cervicogenic headaches. In the mid to lower cervical spine, painful facets can spread pain to the shoulder girdle and periscapular region. Lumbar facet pain patterns are variable, but typically radiate into the buttock and thigh region and rarely extend below the knees [45]. On examination, painful facets are often exacerbated by maneuvers that place the spine in hyperextension or extension and rotation, such as the facet loading test. Treatment of symptomatic facet joints can include conservative measures such as physical therapy, minimally invasive interventions such as medial branch radiofrequency ablation, or surgical interventions.

It is important to note that severe facet arthropathy can also result in radicular symptoms or neurogenic claudication. This is due to facet joint hypertrophy and overgrowth resulting in stenosis of the neural foramen or central canal. In these patients, treatment interventions such as epidural steroid injection, foraminotomy, and laminectomy may be indicated to optimize symptom relief [46].

Sacroiliac Joint Dysfunction

Pain from sacroiliac joint dysfunction affects 15–30% of patients with chronic, non-radicular pain. Prevalence of sacroiliac joint dysfunction is greatest in young athletes and the elderly, and is also commonly seen in pregnant women [47, 48]. While the classic sacroiliac joint pain pattern has been described as extending from the PSIS through the buttocks and posterolateral thigh, true pain patterns from sacroiliac joint dysfunction can vary greatly. For example, some patients report radiation to the knee or groin region [48–50]. The value of the physical examination in sacroiliac joint dysfunction has been questioned given the heterogeneity of patient symptoms, but multiple studies have concluded that a cluster of provocative tests with at least three being positive are useful in both diagnosis and predicting positive response to SI joint injection [37, 48]. One commonly used cluster described by Laslett et al. combines the SI joint compression, SI joint distraction, thigh thrust, sacral thrust and Gaenslen's maneuvers to achieve high sensitivity and specificity in the assessment of SI joint dysfunction [37].

Multiple conservative treatment options exist for sacroiliac joint dysfunction including physical therapy, activity modification, bracing and NSAIDs. Minimally invasive treatment options include intra- and extraarticular corticosteroid injection, lateral branch radiofrequency nerve ablation, and sacroiliac joint fusion [48]. Prolotherapy has also been explored in sacroiliac joint pain as an avenue to strengthen the sacroiliac ligaments and reduce pain symptoms [51]. New treatments have also been explored including regenerative medicine treatments as well as peripheral nerve stimulation [51, 52].

Discogenic Pain

Another common pain generator of back and neck pain are the intervertebral discs. Discogenic pain is often axial in nature. Diagnosis and treatment of discogenic back pain is difficult as symptoms are often vague and the underlying pathology may involve the degeneration of multiple discs and their surrounding structures. The quality of discogenic pain is often described as bandlike and worsened with spinal flexion, but can also present similarly to facet mediated pain and has been found to have similar referral patterns [53, 54]. Diagnosis of discogenic pain can be aided with advanced imaging studies which may reveal loss of disc height, Modic endplate changes, Schmorl nodes and disc herniation, among others. Provocative or CT discography can be used to further evaluate discogenic pain but should be used cautiously given the potential risks of discography such as post-procedural disc herniation or infection [55]. While epidural steroid injections and surgical interventions are potential treatment options for refractory pain, most patients are expected to improve with conservative management [6].

Radiculopathy

Radiculopathy is defined as dysfunction of the nerve root(s) which can manifest as pain, hyporeflexia, paresthesia, numbness and/or weakness which are referred in a predictable pattern [56]. Classically, radicular referral patterns are described as following the distribution of the affected nerve root. While these distributions can be useful in localizing a nerve root generating radicular symptoms, a recent study has revealed potential overlap in radicular referral patterns [57]. Therefore, suspected radiculopathy can be confirmed with imaging and/or electrodiagnostic studies to ensure clinical correlation prior to an invasive procedure such as epidural steroid injection or surgical intervention.

Myelopathy

Myelopathy results from injuries to the spinal cord and can arise from chronic compression or more acute clinical conditions such as trauma, infection, or autoimmune disorders [58]. It can present with long-tract signs such as hyperreflexia, increased muscle tone, clonus, and pathologic reflexes (such as the Babinski reflex or Hoffmann's sign) as well as weakness, numbness, and pain in the associated limbs [56]. Patients can present with impairments in gait, difficulty with fine motor control, muscle atrophy, and loss of proprioception as well. In the cervical spine, cervical myelopathy can also present as central cord syndrome which is denoted by motor impairment greater in the upper extremities than the lower extremities, urinary retention, and/or sensory impairments below the level of the lesion [59]. Treatment varies based on the underlying cause of myelopathy. Compression from degeneration, trauma or tumors may require surgical decompression, whereas autoimmune, infectious, and nutritional myelopathies may be managed medically [60].

Spinal Stenosis

Spinal stenosis is described as narrowing of the central spinal canal, lateral recess, or foraminal region that can result in irritation or compression of the spinal cord or its nerve roots. Spinal stenosis most frequently occurs at the lumbar region [61]. Spinal stenosis can be congenital but is more frequently degenerative in nature. Patients with central spinal stenosis classically present with symptoms of neurogenic claudication which manifests as weakness, heaviness, and radicular pain in the limbs that worsens with activity and improves with rest [62]. Patients often report symptoms are exacerbated with standing or spinal extension, and symptoms are improved with forward flexion of the spine such as stooping forward or sitting. Neurogenic claudication can present similarly to vascular claudication, but it is typically differentiated from vascular claudication by the presence of normal distal pulses on physical examination [63]. Often, patients with neurogenic claudication may not have active symptoms on examination, but may develop these after exercise or ambulation. Management of spinal stenosis typically begins with physical therapy and conservative treatments. If symptoms persist, then treatment options such as epidural steroid injection and surgical decompression can be considered [63].

Vertebral Compression Fractures

Vertebral fractures are among the most common fractures related to conditions such as osteoporosis that result in low bone mass. As such, the incidence of these fractures increases with age for both men and women. Women are significantly more likely to suffer vertebral fractures after the age of 60 years [64]. The most common site for vertebral compression fracture is at the thoracolumbar junction [64]. Vertebral fractures are most commonly caused by osteoporosis or trauma. In all cases, the cause of the vertebral fracture should be considered as a fraction are the result of malignancy or infiltrative diseases that weaken the vertebrae [64]. Clinically, vertebral fractures can be difficult to recognize. Pain with percussion or palpation of the bony midline may aid in identifying these fractures, but their diagnosis is often confirmed on radiographic studies. Treatment varies based on the type, severity, and acuity of the identified fracture. Potential treatment options include rest, physical therapy, medications, and vertebral augmentation.

References

1. Urits I, Burshtein A, Sharma M, Testa L, Gold PA, Orhurhu V, et al. Low back pain, a comprehensive review: pathophysiology, diagnosis, and treatment. Curr Pain Headache Rep. 2019;23(3):23. https://doi.org/10.1007/s11916-019-0757-1.
2. Cohen SP. Epidemiology, diagnosis, and treatment of neck pain. Mayo Clin Proc. 2015;90(2):284–99. https://doi.org/10.1016/j.mayocp.2014.09.008.
3. Bogduk N, Mercer SR. Biomechanics of the cervical spine. I: Normal kinematics. Clin Biomech. 2000;15:633–48.

4. Bogduk N. Functional anatomy of the spine. Handb Clin Neurol. 2016;136:675–88. https://doi.org/10.1016/B978-0-444-53486-6.00032-6.
5. Forseen SE, Corey AS. Clinical decision support and acute low back pain: evidence-based order sets. J Am Coll Radiol. 2012;9(10):704–12. https://doi.org/10.1016/j.jacr.2012.02.014.
6. Barr KP, Concannon LG, Harrast MA. Low back pain. In: Cifu DX, editor. Braddom's physical medicine & rehabilitation. 5th ed. Philadelphia: Elsevier; 2016.
7. Sweeney T, Prentice C, Saal JA, et al. Cervicothoracic muscular stabilizing technique. Phys Med Rehabil State Art Rev. 1990;4:335–59.
8. Al-Obaidi SM, Al-Zoabi B, Al-Shuwaie N, et al. The influence of pain and pain-related fear and disability beliefs on walking velocity in chronic low back pain. Int J Rehabil Res. 2003;26(2):101–8.
9. Al-Obaidi SM, Nelson RM, Al-Awadhi S, et al. The role of anticipation and fear of pain in the persistence of avoidance behavior in patients with chronic low back pain. Spine. 2000;25(9):1126–31. https://doi.org/10.1097/00007632-200005010-00014.
10. Waddell G. The back pain revolution. 2nd ed. Edinburgh: Churchill Livingstone; 2004.
11. Scholten P, Chekka K, Benzon HT. Physical examination of the patient with pain. In: Fishman S, Cohen SP, Hurley RW, Benzon HT, Raja S, Liu SS, editors. Essentials of pain medicine. 4th ed. Philadelphia: Elsevier; 2018.
12. Partanen JV, Ojala TA, Arokoski JPA. Myofascial syndrome and pain: a neurophysiological approach. Pathophysiology. 2010;17:19–28.
13. McGregor AH, McCarthy ID, Hughes SP. Motion characteristics of the lumbar spine in the normal population. Spine. 1995;20(22):2421–8.
14. American Academy of Orthopaedic Surgeons. Joint motion: methods of measuring and recording. 6th ed. Edinburgh: Churchill Livingstone; 1972.
15. Paternostro-Sluga T, Grim-Stieger M, Posch M, Schuhfried O, Vacariu G, Mittermaier C, et al. Reliability and validity of the Medical Research Council (MRC) scale and a modified scale for testing muscle strength in patients with radial palsy. J Rehabil Med. 2008;40(8):665–71. https://doi.org/10.2340/16501977-0235.
16. Kirshblum SC, Burns SP, Biering-Sorensen F, Donovan W, Graves DE, Jha A, et al. International standards for neurological classification of spinal cord injury (revised 2011). J Spinal Cord Med. 2011;34(6):535–46. https://doi.org/10.1179/204577211X13207446293695.
17. Hales M, Biros E, Reznik JE. Reliability and validity of the sensory component of the international standards for neurological classification of spinal cord injury (ISNCSCI): a systematic review. Top Spinal Cord Inj Rehabil. 2015;21(3):241–9. https://doi.org/10.1310/sci2103-241.
18. O'Dell MW, Lin CD, Singh JR, Christolias GC. The physiatric history and physical examination. In: Cifu DX, editor. Braddom's physical medicine & rehabilitation. 5th ed. Philadelphia: Elsevier; 2016.
19. Bickley LS. The nervous system. In: Bickley LS, editor. Bates' guide to physical examination and history taking. 10th ed. Philadelphia: Lippincott, Williams and Wilkins; 2009. p. 655–735.
20. Wong CK, Johnson EK. A narrative review of evidence-based recommendations for the physical examination of the lumbar spine, sacroiliac and hip joint complex. Musculoskeletal Care. 2012;10(3):149–61. https://doi.org/10.1002/msc.1012.
21. Gogu S, Gandbhir VN. Trendelenburg sign. In: StatPearls [Internet]. StatPearls Publishing; 2020. https://www.ncbi.nlm.nih.gov/books/NBK555987/?report=reader. Accessed 30 Nov 2020.
22. Anekstein Y, Blecher R, Smorgick Y, Mirovsky Y. What is the best way to apply the Spurling test for cervical radiculopathy? Clin Orthop Relat Res. 2012;470(9):2566–72. https://doi.org/10.1007/s11999-012-2492-3.
23. Tong HC, Haig AJ, Yamakawa K. The Spurling test and cervical radiculopathy. Spine. 2002;27(2):156–9. https://doi.org/10.1097/00007632-200201150-00007.
24. Hippensteel KJ, Brophy R, Smith MV, Wright RW. A comprehensive review of physical examination tests of the cervical spine, scapula, and rotator cuff. J Am Acad Orthop Surg. 2019;27(11):385–94. https://doi.org/10.5435/JAAOS-D-17-00090.

25. Viikari-Juntura E, Porras M, Laasonen EM. Validity of clinical tests in the diagnosis of root compression in cervical disc disease. Spine. 1989;14(3):253–7. https://doi.org/10.1097/00007632-198903000-00003.
26. Thoomes EJ, van Geest S, van der Windt DA, Falla D, Verhagen AP, Koes BW, et al. Value of physical tests in diagnosing cervical radiculopathy: a systematic review. Spine J. 2018;18(1):179–89. https://doi.org/10.1016/j.spinee.2017.08.241.
27. Viikari-Juntura E. Interexaminer reliability of observations in physical examinations of the neck. Phys Ther. 1987;67(10):1526–32. https://doi.org/10.1093/ptj/67.10.1526. PMID: 3659137.
28. Wainner RS, Fritz JM, Irrgang JJ, Boninger ML, Delitto A, Allison S. Reliability and diagnostic accuracy of the clinical examination and patient self-report measures for cervical radiculopathy. Spine. 2003;28(1):52–62. https://doi.org/10.1097/00007632-200301010-00014.
29. Chou R, Qaseem A, Snow V, et al. Diagnosis and treatment of low back pain: a joint clinical practice guideline from the American College of Physicians and the American Pain Society. Ann Intern Med. 2007;147:478–91.
30. Majlesi J, Togay H, Unalan H, Toprak S. The sensitivity and specificity of the Slump and the Straight Leg Raising tests in patients with lumbar disc herniation. J Clin Rheumatol. 2008;14(2):87–91. https://doi.org/10.1097/RHU.0b013e31816b2f99.
31. van der Windt DA, Simons E, Riphagen II, Ammendolia C, Verhagen AP, Laslett M, et al. Physical examination for lumbar radiculopathy due to disc herniation in patients with low-back pain. Cochrane Database Syst Rev. 2010;(2):CD007431. https://doi.org/10.1002/14651858.CD007431.pub2.
32. Werner CM, Hoch A, Gautier L, König MA, Simmen HP, Osterhoff G. Distraction test of the posterior superior iliac spine (PSIS) in the diagnosis of sacroiliac joint arthropathy. BMC Surg. 2013;13:52. https://doi.org/10.1186/1471-2482-13-52.
33. Lyle MA, Manes S, McGuinness M, Ziaei S, Iversen MD. Relationship of physical examination findings and self-reported symptom severity and physical function in patients with degenerative lumbar conditions. Phys Ther. 2005;85(2):120–33.
34. Stuber K, Lerede C, Kristmanson K, Sajko S, Bruno P. The diagnostic accuracy of the Kemp's test: a systematic review. J Can Chiropr Assoc. 2014;58(3):258–67.
35. Telli H, Telli S, Topal M. The validity and reliability of provocation tests in the diagnosis of sacroiliac joint dysfunction. Pain Physician. 2018;21(4):E367–76. PMID: 30045603.
36. Szadek KM, van der Wurff P, van Tulder MW, Zuurmond WW, Perez RS. Diagnostic validity of criteria for sacroiliac joint pain: a systematic review. J Pain. 2009;10(4):354–68. https://doi.org/10.1016/j.jpain.2008.09.014.
37. Laslett M, Aprill CN, McDonald B, Young SB. Diagnosis of sacroiliac joint pain: validity of individual provocation tests and composites of tests. Man Ther. 2005;10(3):207–18. https://doi.org/10.1016/j.math.2005.01.003. PMID: 16038856.
38. Arnbak B, Jurik AG, Jensen RK, Schiøttz-Christensen B, van der Wurff P, Jensen TS. The diagnostic value of three sacroiliac joint pain provocation tests for sacroiliitis identified by magnetic resonance imaging. Scand J Rheumatol. 2017;46(2):130–7. https://doi.org/10.1080/03009742.2016.1184308.
39. DePalma MJ, Gasper JJ, Slipman CW. Common neck problems. In: Cifu DX, editor. Braddom's physical medicine & rehabilitation. 5th ed. Philadelphia: Elsevier; 2016.
40. Jackson R. Cervical trauma: not just another pain in the neck. Geriatrics (Basel, Switzerland). 1982;37(4):123–6.
41. Borenstein DG. Chronic low back pain. Rheum Dis Clin N Am. 1996;22(3):439–56. https://doi.org/10.1016/s0889-857x(05)70281-7.
42. Shah JP, Thaker N, Heimur J, Aredo JV, Sikdar S, Gerber L. Myofascial trigger points then and now: a historical and scientific perspective. PM R. 2015;7(7):746–61. https://doi.org/10.1016/j.pmrj.2015.01.024.
43. Gerwin RD. Classification, epidemiology, and natural history of myofascial pain syndrome. Curr Pain Headache Rep. 2001;5(5):412–20. https://doi.org/10.1007/s11916-001-0052-8.

44. Travell JG, Simons DG. Myofascial pain and dysfunction: the trigger point manual. 1st ed. Philadelphia: Williams & Wilkins; 1983.
45. Gellhorn AC, Katz JN, Suri P. Osteoarthritis of the spine: the facet joints. Nat Rev Rheumatol. 2013;9(4):216–24. https://doi.org/10.1038/nrrheum.2012.199.
46. Cohen SP, Huang JH, Brummett C. Facet joint pain--advances in patient selection and treatment. Nat Rev Rheumatol. 2013;9(2):101–16. https://doi.org/10.1038/nrrheum.2012.198.
47. Palsson TS, Beales D, Slater H, O'Sullivan P, Graven-Nielsen T. Pregnancy is characterized by widespread deep-tissue hypersensitivity independent of lumbopelvic pain intensity, a facilitated response to manual orthopedic tests, and poorer self-reported health. J Pain. 2015;16:270–82.
48. Cohen SP, Chen Y, Neufeld NJ. Sacroiliac joint pain: a comprehensive review of epidemiology, diagnosis and treatment. Expert Rev Neurother. 2013;13(1):99–116. https://doi.org/10.1586/ern.12.148.
49. Slipman CW, Jackson HB, Lipetz JS, Chan KT, Lenrow D, Vresilovic EJ. Sacroiliac joint pain referral zones. Arch Phys Med Rehabil. 2000;81(3):334–8.
50. Fortin JD, Dwyer AP, West S, Pier J. Sacroiliac joint: pain referral maps upon applying a new injection/arthrography technique. Part I: asymptomatic volunteers. Spine. 1994;19(13):1475–82.
51. Soto Quijano DA, Otero Loperena E. Sacroiliac joint interventions. Phys Med Rehabil Clin N Am. 2018;29(1):171–83. https://doi.org/10.1016/j.pmr.2017.09.004.
52. Guentchev M, Preuss C, Rink R, Peter L, Sailer MHM, Tuettenberg J. Long-term reduction of sacroiliac joint pain with peripheral nerve stimulation. Oper Neurosurg. 2017;13(5):634–9. https://doi.org/10.1093/ons/opx017.
53. Grubb SA, Kelly CK. Cervical discography: clinical implications from 12 years of experience. Spine (Phila Pa 1976). 2000;25(11):1382–9. https://doi.org/10.1097/00007632-200006010-00010. PMID: 10828920.
54. Slipman CW, Plastaras CT, Palmitier RA, Huston CW, Sterenfeld EB. Symptom provocation of fluoroscopically guided cervical nerve root stimulation. Are dynatomal maps identical to dermatomal maps? Spine. 1998;23(20):2235–42. https://doi.org/10.1097/00007632-199810150-00019.
55. Fujii K, Yamazaki M, Kang JD, Risbud MV, Cho SK, Qureshi SA, et al. Discogenic back pain: literature review of definition, diagnosis, and treatment. JBMR Plus. 2019;3(5):e10180. https://doi.org/10.1002/jbm4.10180.
56. Harrop JS, Hanna A, Silva MT, Sharan A. Neurological manifestations of cervical spondylosis: an overview of signs, symptoms, and pathophysiology. Neurosurgery. 2007;60(1 Supp1 1):S14–20.
57. Furman MB, Johnson SC. Induced lumbosacral radicular symptom referral patterns: a descriptive study. Spine J. 2019;19(1):163–70. https://doi.org/10.1016/j.spinee.2018.05.029.
58. Oyinkan Marquis B, Capone PM. Myelopathy. Handb Clin Neurol. 2016;136:1015–26. https://doi.org/10.1016/B978-0-444-53486-6.00052-1.
59. Schneider RC, Cherry G, Pantek H. The syndrome of acute central cervical spinal cord injury; with special reference to the mechanisms involved in hyperextension injuries of cervical spine. J Neurosurg. 1954;11(6):546–77. https://doi.org/10.3171/jns.1954.11.6.0546.
60. Bhattacharyya S. Spinal cord disorders: myelopathy. Am J Med. 2018;131(11):1293–7. https://doi.org/10.1016/j.amjmed.2018.03.009.
61. Babb A, Carlson WO. Spinal stenosis. S D Med. 2006;59(3):103–5.
62. Messiah S, Tharian AR, Candido KD, Knezevic NN. Neurogenic claudication: a review of current understanding and treatment options. Curr Pain Headache Rep. 2019;23(5):32. https://doi.org/10.1007/s11916-019-0769-x.
63. Bagley C, MacAllister M, Dosselman L, Moreno J, Aoun SG, El Ahmadieh TY. Current concepts and recent advances in understanding and managing lumbar spine stenosis. F1000Res. 2019;8:F1000 Faculty Rev-137. https://doi.org/10.12688/f1000research.16082.1.
64. Schousboe JT. Epidemiology of vertebral fractures. J Clin Densitom. 2016;19(1):8–22. https://doi.org/10.1016/j.jocd.2015.08.004.

Electromyography in the Spine Patient

3

Denizen Kocak and Thiru M. Annaswamy

Electromyography (EMG) is a useful tool in the localization, classification, and identification of radiculopathy and other neuromuscular conditions in the spine population. This chapter will seek to provide an overview of EMG, discuss its utility in the evaluation of the spine patient, and highlight various clinical pearls.

Electromyography, often referring to both Nerve Conduction Studies (NCS) and needle EMG in combination, is an in-office procedure that evaluates peripheral nerves and muscles. EMG is an objective and valid test of the peripheral nervous system, but elements of the study can be subjective, making interpretation of the study challenging at times. Additionally, needle EMG is a dynamic study and the choice of which muscles to sample is a real time decision based on clinical presentation, physical exam, and findings in muscles which have already been sampled. Planning an EMG study is a skill in and of itself, and some studies may be less diagnostically accurate, especially when performed by an inexperienced electromyographer.

Much like the history, the physical exam, and imaging, EMG is another piece of the clinical puzzle that must be taken in context. EMG has the best diagnostic value when used as an extension of the history and physical exam. EMG has poor diagnostic utility as a standalone test, bereft of a history and physical exam. For example, a remote axonal nerve injury consistent with radiculopathy can be detected on EMG in a patient who does not clinically present with radiculopathy symptoms or

D. Kocak
Department of Physical Medicine and Rehabilitation, VA North Texas Health Care System and UT Southwestern Medical Center, Dallas, TX, USA

T. M. Annaswamy (✉)
Department of Physical Medicine & Rehabilitation, VA North Texas Health Care System and UT Southwestern Medical Center, Dallas, TX, USA

Physical Medicine & Rehabilitation Service, VA North Texas Health Care System, Dallas, TX, USA

© The Author(s), under exclusive license to Springer Nature Switzerland AG 2022

C. E. Noe (ed.), *Multidisciplinary Spine Care*,
https://doi.org/10.1007/978-3-031-04990-3_3

signs. Without the context of a past history of radicular symptoms that have since resolved, this diagnostic information obtained on EMG is of much less value. When placed in context of a relevant history and physical examination, however, EMG has tremendous value and provides diagnostic information to evaluate the presence, type, chronicity, and severity of nerve damage and can be used to help guide treatment planning.

This chapter will seek to provide an overview of the study for the provider, identify strengths and limitations of EMG, and information on outcome prognostication with EMG.

Overview of the Study

The examiner begins the test by performing NCS in the extremity or extremities of interest. This consists of stimulating peripheral nerves to evoke a "supramaximal" response. For motor studies, the response is termed the compound muscle action potential (CMAP) and for sensory nerve the response is termed the sensory nerve action potential (SNAP). The response of the nerve is assessed both for latency (time to onset for motor nerves and time to peak for sensory nerves) and amplitude and both are compared against a reference table of normal values. This can help to assess for demyelination as well as axonal loss.

One important point to note is that in radiculopathy, sensory nerve conduction studies are generally normal (because the dorsal root ganglion containing the cell bodies of the sensory axons are usually located distal to the site of radicular nerve impingement) [1]. Abnormality in a sensory nerve conduction study should alert the examiner to the presence of underlying conditions such as peripheral nerve entrapment or peripheral neuropathy.

One nerve conduction study, not routinely performed by examiners, which can be abnormal in radiculopathy is the F wave. F-wave is a late response, which evaluates the nerve over the entire length of the nerve and can suggest a more proximal lesion if distal studies are normal. In lumbosacral radiculopathy which was surgically confirmed in 95/100 patients studied, the sensitivity of the F-wave study appeared to be about 70% [2]. One caveat to this study is that it does not localize the proximal lesion, which may be in the proximal nerve, plexus, or roots. This study is also typically only performed on the median (Fig. 3.1) and ulnar nerves in the upper extremities (which may be abnormal in C8 or T1 radiculopathy, but would be normal in radiculopathy of the more common, higher cervical levels) and the tibial and peroneal nerves in the lower extremities (which may be abnormal in L5–S1 radiculopathies, but normal in other levels).

Another less commonly performed nerve conduction study is the H Reflex, which is most commonly only obtained by stimulating the tibial nerve in the popliteal fossa and measuring the response in the soleus muscle. It studies a reflex arc with afferent Ia sensory fibers, and efferent motor fibers. Although not commonly used for the diagnosis of radiculopathy, the soleus H-reflex is useful to evaluate for S1 radiculopathy as it is an electric analog of the Achilles stretch reflex, which one

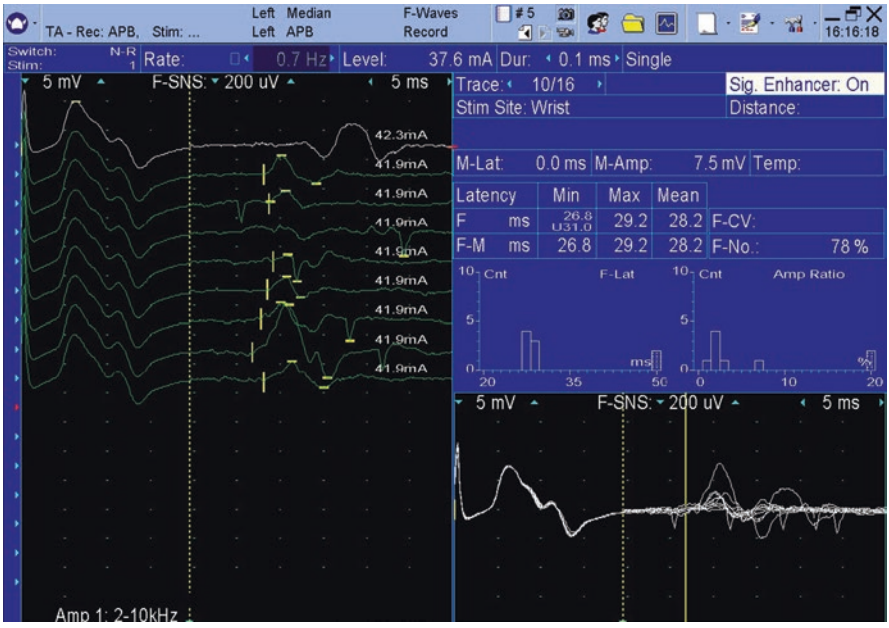


Fig. 3.1 Median F-wave recording. Left half: A raster of 9 Median CMAP and F-wave recordings are displayed here. F-waves are marked on the right side of the split screen. Top right: F-wave latency measures are listed. Bottom right: Superimposed traces with F-wave latency bar (solid line)

could expect to be abnormal in S1 radiculopathy. Of note, if the Achilles reflex is absent on physical exam, the corresponding soleus H-reflex is likely absent as well. The real value of the H-reflex study comes from side-to-side comparison where a difference in latency of 1.8 ms or greater or absence on one side can suggest S1 radiculopathy with roughly 50% sensitivity and 91% specificity.

Needle EMG is the second portion of the test, and for our purposes, the much more interesting study. This test is performed by inserting a small needle (either a concentric needle or a monopolar needle) in order to evaluate motor unit action potentials (MUAPs). The choice of needle is often dictated by examiner familiarity and preference and there is little difference between the measured potentials with the two needles.

For each muscle, spontaneous and insertional activity are examined with the patient at rest, and then the MUAPs are examined with the patient contracting the muscle in question both in submaximal and maximal fashion. For in-depth discussion of the various types of waveforms and abnormalities, refer to the list of references at the end of the chapter. This chapter will attempt to summarize the findings to providers who do not perform the studies to better help them interpret results of EMGs that they may refer patients for. We will break down the findings into examination with the patient at rest (examination of spontaneous and insertional activity) and with the patient activating the desired muscle (evaluation of MUAPs).

With the patient at rest, spontaneous and insertional activity are examined. Insertional activity can be rated as decreased, increased, or normal and there are standardized rating scales which electromyographers use. Briefly, the needle is moved very slightly within the muscle in order to irritate the muscle membrane and record the response. Increased insertional activity is indicative only of increased membrane instability and can be seen in both neuropathic and myopathic conditions. Decreased insertional activity is more commonly seen with fatty or fibrous replacement of normal muscle tissue. Increased insertional activity alone is not indicative of abnormality.

Abnormal spontaneous activity is the hallmark of active denervation. For the purposes of examination of radiculopathy, we will primarily concern ourselves with fibrillation potentials and positive sharp waves, both of which represent spontaneous discharges due to depolarization of the muscle fiber, seen in active denervation. In interpreting EMGs, one will often find these referred to simply as "fibs" and "sharps" in reports. These represent muscle fibers that are no longer innervated, and are therefore discharging spontaneously. The standard rating for fibs and sharps ranges from 0 (none present) to 4+ (baseline entirely obscured by spontaneous potentials) (Fig. 3.2). They represent active axonal pathology and denervation of the muscle and can help to define chronicity, which will be discussed in more detail later in the chapter.

Less commonly, one may encounter complex repetitive discharges (CRDs) which represent a cycle of depolarization in adjacent muscle fibers based on ephaptic spread. This is commonly seen in the setting of repeated denervation, reinnervation, and denervation of adjacent muscle fibers which can occur in radiculopathy, though less commonly than simple sharps and fibs. Additionally, fasciculation potentials may sometimes hint at prior reinnervation if they have an abnormal morphology though the presence of fasciculation potentials alone is not usually indicative of radiculopathy.

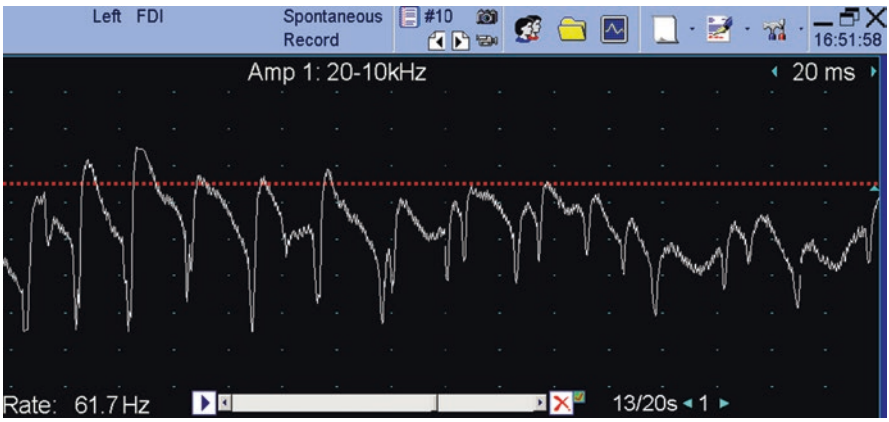


Fig. 3.2 Positive sharp waves, 4+ rating

With the patient activating the muscle, MUAPs are examined. The measured MUAP represents the depolarization of an entire motor unit, including the multiple muscle fibers that make up that same motor unit. They are assessed for morphology, stability, and firing pattern.

The components of MUAP morphology are typically duration, phasicity, and amplitude. For the purposes of radiculopathy, all three are important to examine. Duration is reflective of the time it takes for all of the muscle fibers within a motor unit to fire, and in the setting of denervation followed by reinnervation (e.g., chronic radiculopathy), the number of muscle fibers innervated by a single motor unit increases, leading to increased MUAP duration. Additionally, duration of the MUAP can help to suggest the degree of chronicity as early reinnervation tends to have longer duration due to more asynchronous firing and more chronic reinnervation tends to have a more normal duration as the new, enlarged, motor unit is able to better fire in synchrony. Phasicity is a description of how in-sync the firing muscle fibers of a motor unit are. With a large degree of asynchrony, the waveform will have many turns that cross the baseline, and be called polyphasic (Fig. 3.3). With time and more chronic reinnervation, the reinnervated motor unit tends to fire more synchronously and polyphasic potentials may turn into serrated potentials (many turns that do not cross the baseline) and eventually even into MUAPs with normal phasicity as the motor unit fires with less asynchrony. Interpretation of amplitude can also be taken into consideration in this context as more chronic reinnervation will tend to have a narrower waveform with taller amplitude as more muscle fibers are firing simultaneously, and the measured potentials are additive. It may help to thing of the wide, polyphasic, MUAP being compressed from the sides and the amplitude increasing as a result. Do be aware that polyphasia, increased MUAP duration, and changes in amplitude are not exclusive to neuropathic conditions and can also be seen in myopathic pathology, though amplitude will tend to be decreased in myopathic disorders and increased in neuropathic disorders.

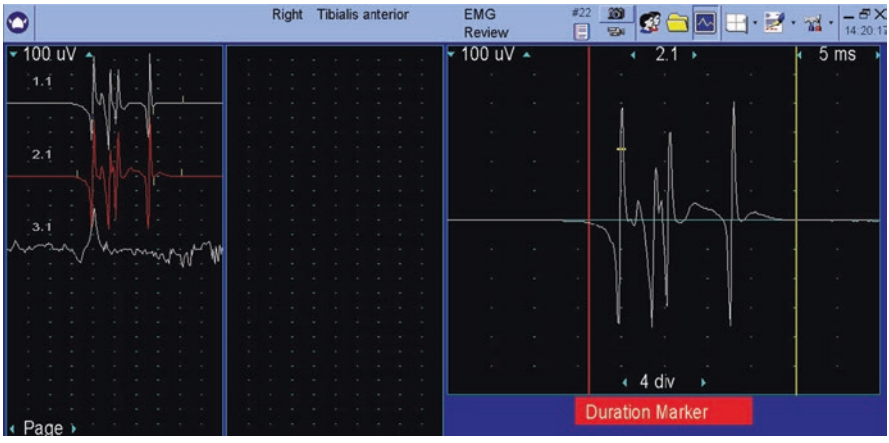


Fig. 3.3 Polyphasic motor unit potential. Left half: 3 motor units are recorded. Units 1.1 and 2.1 are polyphasic. Right half: Unit 2.1 is displayed in more detail demonstrating high polyphasicity

The next item which is observed is the recruitment pattern. Decreased recruitment is typical of a neuropathic cause and decreased activation is characteristic of a more central cause (pain, spinal cord lesion, stroke, etc.). In radiculopathy, one would expect a normal or decreased recruitment pattern. Without any structural change to the muscle fibers there is no change in the force-generating ability of a single muscle fiber. Instead, in radiculopathy, there is an interference in the affected nerve root's ability to recruit additional motor units and muscle fibers to generate more force when needed. As a result, the available motor unit will increase its firing rate (normal recruitment rate is 5–10 Hz) in order to generate the required force. This is referred to as decreased recruitment. In contrast, in myopathic conditions, force-generating ability of individual muscle fibers is affected but the ability to communicate with additional muscle fibers to recruit them is not. Thus, a pattern of normal or increased recruitment develops where additional units are recruited early in order to generate more force because the force-generating ability of each motor unit is lesser than normal.

Utility of EMG in Identifying Radiculopathy

Due to lack of a universally accepted and reliable reference standard, the true sensitivity and specificity of EMG in determining the presence of lumbosacral radiculopathy is difficult to establish. A wide range of sensitivities from 30% to >80% have been reported for lumbosacral radiculopathy [3] and a range of about 50–70% sensitivity has been reported for the identification of cervical radiculopathy [4].

The more interesting measure for EMG is the specificity of the test. In order to diagnose radiculopathy on needle electromyography, at least two muscles innervated by the same spinal nerve root but by different peripheral nerves must show abnormalities. This can be a combination of muscles in the limbs and paraspinal muscles, and the specificity for various combinations of muscles has been studied. The definition of an "abnormality" in these muscles is quite broad and deserves to be further characterized. Mainly, one will be looking at the presence of positive sharp waves, fibrillation potentials, and polyphasic potentials, though there are other indicators of radiculopathy. For any combination of muscles listed below, the specificity for diagnosing lumbosacral radiculopathy was 100% when only considering the presence of positive sharp waves and/or fibrillation potentials. The specificity for using the threshold of at least 30% polyphasia in the muscle to quantify it as abnormal led to a slightly reduced but still quite excellent specificity as detailed in Table 3.1 [5].

Table 3.1 Specificity of EMG in lumbosacral radiculopathy

Muscles showing abnormalities	Sharps/Fibs	>30% Polyphasia
Paraspinal + Two limb muscles	100	97
Two limb muscles	100	90
Paraspinal + One limb muscle	100	87

The inherent variability in the choice of muscles to sample and what to call abnormal, based on a preferences and decisions by the electromyographer, may account for the wide range of reported sensitivities. Given this fact, it is important to understand what a high-quality study looks like and identify ones that are more likely to either accurately identify or more likely to miss radiculopathy. In short, the more muscles that are sampled, the higher the likelihood that one will accurately identify the presence of a radiculopathy but this also comes with additional discomfort to the patient and prolongs the duration of the study, which is often not practical for the electromyographer.

Attempts have been made to determine an adequate number of muscles to study in order to identify radiculopathy in the patient who has previously electrodiagnostically proven radiculopathy. The American Academy of Neuromuscular and Electrodiagnostic Medicine (AANEM) recently published a monograph detailing recommendations for the evaluation of suspected radiculopathy [6, 7]. The exact sensitivities for specific algorithms and number of muscles studied is beyond the scope of this chapter and can be found in the AANEM monograph but we will briefly discuss them here. Their strongest recommendation was that the paraspinal muscles should be included in the radiculopathy evaluation, and in order to reach adequate sensitivity without the paraspinals, eight limb muscles must be studied. Their recommendation, made easier to remember with a handy rhyme, was "To minimize harm, six in the leg and six in the arm" with the caveat that one of the studied muscles was a paraspinal muscle and that the other sampled muscles represented the nerve roots in the affected limb (C5–T1 in the arm or L2–S2 in the leg). This of course, is simply a recommendation for initial screening studies for radiculopathy, and further evaluation of additional muscles is directed by real-time interpretation of abnormalities on EMG.

In a study of 50 patients with surgically defined single root cervical radiculopathy who were evaluated with EMG and imaging (either CT myelogram or MRI), there was good concordance in the affected level which was identified. Within this cohort, 35 patients had mixed sensorimotor symptoms, nine had sensory complaints only, and six had motor complaints only. Imaging results matched surgical results in all but five of the patients, and generally correlates with imaging findings in 65–85% of patients [4]. Discussion of the electrodiagnostic findings is slightly more nuanced. In that study, the C5, C7, and C8 radiculopathies had a more standard pattern of involved muscles listed in the table below but the C6 radiculopathy had a significant overlap with the both the C5 and the C7 radiculopathy patterns of muscles that showed abnormal results [8].

For lumbosacral radiculopathy, the patterns of abnormal muscles were examined by the same group and they found that L2–L4 radiculopathies were the most difficult to confirm, likely in the setting of fewer distal muscles and overlap among innervation of L2–L4 root innervation in muscles. Although many root innervation tables will include other root levels, Tsao et al. suggested that the gastrocnemius was predominantly S1 innervated, the biceps femoris was exclusively S1 innervated, and the tibialis anterior was predominantly L5 innervated [9]. This information can be used to help guide electrodiagnostic interpretation of findings.

Additionally, needle EMG is more likely to be abnormal in patients who present with motor deficits (this will make sense as we discuss limitations of sensory predominant radiculopathy below) and patients with motor complaints or objective weakness on exam are more likely to have EMG correlates [4].

Limitations of EMG in Radiculopathy

Despite the ability of EMG to add to the clinical diagnosis of a patient with radiculopathy, there are several inherent limitations of the study that the spine provider should be aware of when ordering or performing an EMG.

Chief among these is the limitation that EMG will not always be able to define the specific level of the radiculopathy. In almost all cases, the EMG will be able to localize the level to within +/− one level but due to the multi-nerve root level innervations of muscles a single muscle is not enough to localize the affected nerve root. In general, the goal should be to sample enough muscles innervated by a specific nerve root but by different peripheral nerves to feel confident that the pathological findings are attributable to a single nerve root. For an EMG to suggest radiculopathy at a certain root level, at least two muscles supplied by the same root should have abnormal findings suggestive of denervation or reinnervation potentials. In order to provide a stronger case for a specific level, more muscles must be studied. Consider for example a sample of the tibialis anterior (L4/L5 nerve roots, deep peroneal nerve) and the peroneus longus (L5/S1 nerve roots, superficial peroneal nerve) which both show evidence of reinnervation potentials suggestive of a radiculopathy. One could surmise that this would be enough evidence for an L5 radiculopathy as the L5 nerve root is traditionally common to both muscles but in order to rule out a common peroneal neuropathy additional muscles should be examined. Take a moment to review Table 3.2 for an illustrative example.

Table 3.2 Example of EMG/NCS helping with lesion localization

	Subacute axonal L5 radiculopathy	Subacute axonal common peroneal neuropathy
Nerve conduction study		
Tibial motor to AH	Normal	Normal
Peroneal motor to EDB	May be abnormal	Likely abnormal
Peroneal motor to TA	May be abnormal	Likely abnormal
Superficial peroneal sensory	Normal	Likely normal
Sural sensory	Normal	Normal
EMG		
Tibialis anterior	May be abnormal	Likely abnormal
Peroneus longus	May be abnormal	Likely abnormal
Medial gastrocnemius	Normal	Normal
Tibialis posterior	May be abnormal	Normal
Gluteus medius	May be abnormal	Normal
L5 Paraspinal	Likely abnormal	Normal

An example of what would be strong enough to suggest radiculopathy would be to have evidence of denervation and or reinnervation potentials in the biceps brachii (C5/C6 nerve roots, musculocutaneous nerve) and the pronator teres (C6/C7 nerve roots, median nerve), and involvement of the paraspinals would localize the lesion as a radiculopathy.

It should be noted that needle EMG is not ideal for lesion localization. This means that abnormal findings in a muscle of interest suggests that the lesion is proximal to the muscle, but absence of abnormalities does not necessarily localize the lesion distal to the muscle. Especially in peripheral nerve injuries in the limb, nerve conduction studies are much more specifically able to localize the lesion (for example, by performing ulnar inching where the skilled electromyographer can localize a cubital tunnel lesion to within an inch of the lesion).

Knowing this, let us look at the example above where the biceps brachii and the pronator teres showed abnormalities. These findings allow us to say that there is a lesion proximal to the branching point from the musculocutaneous nerve to the biceps and proximal to the branching point from the median nerve to the pronator teres. This could be consistent with a plexopathy, radiculopathy, or less commonly isolated mononeuropathies. Now take the same scenario, but let us assume that the corresponding paraspinals (C6 level paraspinals) demonstrated evidence of active denervation (fibrillation potentials and/or positive sharp waves). This means that there is an axonal lesion proximal to the branching point of the nerve that innervates the paraspinals (medial branch or dorsal rami dependent on level studied). This allows us to confidently state that the lesion is very unlikely to be due to a brachial plexopathy.

Although the paraspinal muscles are valuable in the evaluation of radiculopathy, they present with some specific challenges that are unique to electromyographic evaluation. Firstly, in the paraspinal muscles only spontaneous and insertional activity are evaluated, meaning that a fully reinnervated paraspinal muscles will not be defined as abnormal. Second, patients must be able to relax fully, and patients are less able to consciously relax the paraspinals. An experienced electromyographer can help patients relax using simple repositioning, but it is not uncommon to have a background of motor activity that interferes with the electromyographer's ability to adequately evaluate the presence of denervation potentials. Third, paraspinal EMG is relatively contraindicated in patients on anticoagulation due to the increased risk of hematoma. Lastly, in patients who have had spinal surgery which utilizes a posterior approach, the paraspinal muscles may show persistent abnormal needle findings for a number of years after surgery due to iatrogenic nerve and muscle injury, which can remain false-positive for 2–3 years post-operatively.

Additionally, other conditions can lead to findings of sharp waves and fibrillation potentials, as they signify spontaneous depolarization of the muscle fibers. This can occur in other neuropathic conditions like peripheral neuropathies, inflammatory myopathies, muscular dystrophies, and neuromuscular junction disorders such as botulism. One needs to be aware that sharps and fibs do not signify denervation from radiculopathy, but instead signify membrane instability, which often occurs in a denervated muscle.

It is important for the spine provider to examine EMG reports with a critical eye and take them in the clinical context of the patient to look at the quality of the diagnosis of radiculopathy based on EMG.

One must also be aware of the possibility of a prefixed and postfixed brachial plexus. Although a completely pre or post-fixed brachial plexus are rare, studies have suggested that about 50% of patients will have some variation relative to the "typical brachial plexus" in a study of about 200 sampled plexuses. This same study suggested that about 25% of plexuses were prefixed and about 2.5% were postfixed. A review of multiple similar studies has suggested a prevalence of prefixed plexuses ranging from 10% to 60% and prevalence of postfixed plexuses from less than 1% all the way up to over 50%. With such a wide range, and variations in the definition of a prefixed and postfixed plexus, it is difficult to establish a true prevalence but it is something that must be considered as it will affect the nerve roots that innervate specific muscles. For example, in a prefixed plexus, one might expect the deltoid (traditionally considered C5 and C6 innervated) to be innervated by the C4 and C5 nerve roots. This is just one example but a similar principle can be applied to other muscles of the arm. Similarly, in a postfixed plexus, one would expect there to be contribution from the C7 nerve root to the deltoid [10, 11].

Additionally, it is not uncommon for radiculopathy to have relative preservation of some fascicles which may lead to an unexpectedly normal muscle study in the setting of high clinical and electromyographic suspicion (based on muscles already studied) for radiculopathy. It may also lead to relative sparing of one muscle despite clear evidence of radiculopathy based on the other muscles studied. Another interesting case to consider is if the radiculopathy affects only sensory fascicles while sparing the motor fascicles in the nerve root. In this case, needle EMG will be normal. Additionally, a purely demyelinating lesion may not be discovered on needle EMG without adequate assessment of recruitment and firing rate, because denervation and reinnervation potentials are dependent on the presence of axonal injury.

Understanding these scenarios in which EMG may not reveal a radiculopathy, it is important to be aware that a normal study does not negate the clinical diagnosis of radiculopathy.

Increasing the accuracy of determination of the level of the affected nerve root comes at the cost of studying more muscles. This means more discomfort for the patient, a longer exam, and in the case of specific muscles, increased risk of injury to the patient. For example, differentiation of a C5 vs C6 radiculopathy is more difficult as both the deltoid and biceps traditionally have both C5 and C6 innervation. One could study the rhomboids which are traditionally C4/C5 innervated to further differentiate C5 vs C6 radiculopathy though this comes with the increased risk of pneumothorax for the patient. One must weigh the relative clinical benefit vs the risk of studying further muscles.

Though not a direct limitation of EMG, we have included mimickers of radiculopathy in Table 3.3 as it is important to highlight that the electrodiagnostic evaluation of a patient is an extension of the physical exam and other clinical data and that one must be aware of common pain referral patterns and other etiologies of sensory complaints. This is not an exhaustive list, but seeks to identify some common confounders.

Table 3.3 Mimickers of radiculopathy

Cervical facet arthropathy	Referral to shoulder/scapula
Lumbosacral facet arthropathy	Referral to posterior thigh and groin [12]
Hip osteoarthritis	Referral to groin/thigh
SI Joint pain	Referral to groin/leg/foot [13]
Peripheral neuropathy	Often distal > proximal and symmetric but varies with etiology
Peripheral mononeuropathy	Neuropathic pain/weakness in nerve distribution
Myopathy	Weakness and distal pain

Timing of Findings in Radiculopathy

It is also important to be aware of *when* certain findings will become apparent on EMG in the course of radiculopathy. With an acute inciting event that leads to radiculopathy, there will be a somewhat predictable time course of the presentation of various abnormalities on EMG. A routine electrodiagnostic evaluation for radiculopathy is less likely to yield false negative results after 6 weeks from initial onset of symptoms to allow for axonal injury changes to become more apparent. In the section below, we will detail the expected progression of EMG findings in radiculopathy.

A detailed history and physical will help to better interpret the electrodiagnostic findings. Getting details on onset and progression of current symptoms is as important as getting the history of prior radicular symptoms even if they have since resolved. Such information will enable one to better attribute findings of a combination of both acute denervation and chronic reinnervation. The onus of obtaining this nuanced history also falls onto the electromyographer performing the study, but any spine provider needs to keep this in mind while interpreting the studies they ordered.

In the acute phase from injury to about 10–14 days, there will not be evidence of active denervation on needle EMG as there has not been sufficient time for Wallerian degeneration, and thus distal denervation to occur. One will typically see a pattern of reduced recruitment as some axons will not fire if there is axonal injury. Fibrillation potentials will then begin to develop, and while needle EMG is not highly specific for delineating the timing of the injury, some general trends can be used as surrogate markers for chronicity. The amplitude of fibrillation potentials can suggest the duration of denervation with very early fibrillation potentials being as large as 1 mV in size, often decreasing to 600 μV within the first 2 months, and subsequently decreasing to 100 μV or smaller at 1 year. In general, it may suffice to recognize that larger amplitudes suggest an injury that occurred just a few months ago whereas smaller amplitudes suggest an injury greater than a year ago [14].

The first signs of reinnervation which occur will be nascent potentials on EMG as terminal collateral sprouting is just barely reaching the denervated muscle fibers. The sprouting occurs from distal motor units and therefore, nerves do not have to grow far to reach their new target and nascent potentials can be seen as early as 6–8 weeks. For reinnervation from axonal regrowth as opposed to collateral sprouting, the time will roughly mimic the time it takes for nerve regrowth to occur (roughly 1 inch per month), and nascent potentials will be seen much sooner in more proximal muscles relative to distal ones.

Following nascent potentials, more polyphasic and above average amplitude potentials will begin to develop (remember this is asynchronous firing from early reinnervation). These may persist for quite some time, and it is difficult to characterize a specific time course based on polyphasic potentials alone. With time, these new motor units will begin to fire more synchronously and potentials will summate to produce larger amplitude motor unit action potentials. After several years have passed, they may summate to the degree that they become "giant motor units" which can reach amplitudes of greater than 20 mV and are the most chronic finding on needle electromyography.

Role of EMG in Lumbar Spinal Stenosis

Although electrodiagnosis is less commonly used in the diagnosis of lumbar spinal stenosis relative to lumbosacral or cervical radiculopathy, it can also be an effective tool in examining patients with suspected symptomatic lumbar spinal stenosis. Haig and colleagues have spent extensive time validating and detailing paraspinal mapping as a technique to evaluate lumbar spinal stenosis on EMG. Originally, they utilized an extremely intensive paraspinal mapping technique which required mapping more areas but they concluded that the MiniPM technique provided similar sensitivity and specificity with the benefit of less patient discomfort and less chance of iatrogenic injury as fewer samples were needed. The L3, L4, and L5 levels were sampled bilaterally. To localize the first three points of needle insertion, the inferior border of the lumbar spinous process was palpated and the needle was inserted 2.5 cm laterally and 1 cm cranially to this point for the L3, L4, and L5 levels. For the last insertion point, the needle was placed 2.5 cm laterally to the midpoint between the posterior superior iliac spines. At all locations, the needle was advanced in three different cephalocaudal angles (45° cranial, 0°, and 45° caudal) while being directed 45–60° towards the midline. Observed muscle membrane instability was rated on a scale from 0 to 4+. Based on their sample of 150 patients which included asymptomatic patients, patients with back pain but no suspected stenosis, and patients with clinical spinal stenosis, a paraspinal mapping score of >4 had 100% specificity and a 30% sensitivity for lumbar spinal stenosis when compared to the axial pain and asymptomatic groups [15].

In addition to paraspinal mapping, needle EMG of the limb when taken in composite with the paraspinals appears to increase the sensitivity to 47.8% but reduced specificity slightly to 87.5% and the H-Wave showed a sensitivity of about 36.4% with a specificity of 91% [15].

When EMG Is Not Helpful

In addition to knowing when to order an EMG, it is important to know the conditions in which EMG is not likely to provide additional information. The AANEM published a set of Choosing wisely recommendations in 2015 which highlights

some of these [16]. Chief for the spine provider is that electromyography is not likely to be helpful for axial neck or back pain without symptoms (weakness/numbness/pain) in the associated limbs. It is a test of the nerves, neuromuscular junction, and associated muscle fibers so unless involvement of these is suspected, EMG would be expected to be normal, or if abnormal, may result in additional unnecessary workup and testing for the patient with additional associated risks and costs.

Significance of EMG in Predicting Clinical Outcomes

While we still advocate for the use of conservative treatment (therapy, medications, etc.) of uncomplicated lumbosacral or cervical radiculopathy, it is interesting to note that some studies have shown that patients with electrodiagnostically confirmed radiculopathy have better outcomes with both injections and surgical intervention compared to patients with negative EMG findings.

For epidural steroid injections targeting pain from radiculopathy, it appeared that patients with positive EMG findings were more likely to respond to them. In a study of 170 patients with lumbosacral or cervical radiculopathy who received transforaminal epidural steroid injections, pain at 30 days and opioid use were examined. It appeared that a statistically significantly larger percentage of patients with lumbosacral radiculopathy reported >50% pain relief lasting longer than 30 days (37% versus 17%). For patients with cervical radiculopathy, this trend did not hold but of note, sample size in the cervical radiculopathy group was only 22 patients. Additionally, patients with positive EMG findings trended toward using less opioids but this did not reach statistical significance [17]. In another study of patients with lumbosacral radiculopathy who underwent transforaminal epidural steroid injections, disability score improvement was statistically significantly better in patients with electrodiagnostically positive radiculopathy. This was measured by the Oswestry Disability Index (ODI) in a cohort of 39 patients [18].

A similar trend was shown for interlaminar epidural steroid injections as well, which included 89 patients which showed statistically significantly improved pain scores at follow up visit and significantly improved function as defined by the Pain Disability Questionnaire (PDQ) score [19].

In patients with lumbar spinal stenosis, a pilot study of 11 patients undergoing interlaminar epidural steroid injection for lumbar spinal stenosis demonstrated improved Pain Disability Questionnaire results at 1 months for the subset of patients who had a positive pre-injection EMG [20].

For surgical outcomes, the evidence is weaker in the ability to predict outcomes after surgery but at least one study has shown that it may be useful in predicting post-surgical outcomes in cervical radiculopathy. In a set of 20 patients who underwent anterior approach cervical discectomy and fusion, patients who had positive EMG findings had better functional outcomes as defined by the Prolo Score and had better satisfaction scores [21].

Conclusions

In summary, EMG (nerve conduction study and needle electromyography) is a valuable tool that adds diagnostic value to the clinical history and physical exam, and complements imaging studies. It can help to diagnose radiculopathy, can localize the level of radiculopathy, and can suggest the severity and chronicity of the lesion. Patients with EMG findings of radiculopathy have improved pain and reduced disability following lumbosacral epidural steroid injections and tend to do better after surgery. It is however, important that the ordering spine provider be aware of the limitations of EMG studies and be able to interpret the results in order to make informed clinical decisions.

References

1. Preston DC. Electromyography and neuromuscular disorders: clinical-electrophysiologic correlations. 3rd ed. London: Elsevier Saunders; 2012.
2. Toyokura M, Murakami K. F-wave study in patients with lumbosacral radiculopathies. Electromyogr Clin Neurophysiol. 1997;37(1):19–26.
3. Reza Soltani Z, Sajadi S, Tavana B. A comparison of magnetic resonance imaging with electrodiagnostic findings in the evaluation of clinical radiculopathy: a cross-sectional study. Eur Spine J. 2014;23(4):916–21.
4. So YT. Guidelines in electrodiagnostic medicine. Practice parameter for needle electromyographic evaluation of patients with suspected cervical radiculopathy. Muscle Nerve Suppl. 1999;8:S209–21.
5. Tong HC, Haig AJ, Yamakawa KS, Miner JA. Specificity of needle electromyography for lumbar radiculopathy and plexopathy in 55- to 79-year-old asymptomatic subjects. Am J Phys Med Rehabil. 2006;85(11):908–12; quiz 913–905, 934.
6. Dillingham TR, Annaswamy TM, Plastaras CT. Evaluation of persons with suspected lumbosacral and cervical radiculopathy: electrodiagnostic assessment and implications for treatment and outcomes (Part I). Muscle Nerve. 2020;62(4):462–73.
7. Dillingham TR, Annaswamy TM, Plastaras CT. Evaluation of persons with suspected lumbosacral and cervical radiculopathy: electrodiagnostic assessment and implications for treatment and outcomes (Part II). Muscle Nerve. 2020;62(4):474–84.
8. Levin KH, Maggiano HJ, Wilbourn AJ. Cervical radiculopathies: comparison of surgical and EMG localization of single-root lesions. Neurology. 1996;46(4):1022–5.
9. Tsao BE, Levin KH, Bodner RA. Comparison of surgical and electrodiagnostic findings in single root lumbosacral radiculopathies. Muscle Nerve. 2003;27(1):60–4.
10. Pellerin M, Kimball Z, Tubbs RS, et al. The prefixed and postfixed brachial plexus: a review with surgical implications. Surg Radiol Anat. 2010;32(3):251–60.
11. Uysal II, Seker M, Karabulut AK, Büyükmumcu M, Ziylan T. Brachial plexus variations in human fetuses. Neurosurgery. 2003;53(3):676–84; discussion 684.
12. Gellhorn AC, Katz JN, Suri P. Osteoarthritis of the spine: the facet joints. Nat Rev Rheumatol. 2013;9(4):216–24.
13. Schneider BJ, Rosati R, Zheng P, McCormick ZL. Challenges in diagnosing sacroiliac joint pain: a narrative review. PM R. 2019;11(Suppl 1):S40–s45.
14. Feinberg J. EMG: myths and facts. HSS J. 2006;2(1):19–21.
15. Haig AJ, Tong HC, Yamakawa KSJ, et al. The sensitivity and specificity of electrodiagnostic testing for the clinical syndrome of lumbar spinal stenosis. Spine. 2005;30(23):2667–76.
16. Foundation A. American Association of Neuromuscular & Electrodiagnostic Medicine. Ten things physicians and patients should question. ABIM Foundation. Choosing wisely:

an initiative of the ABIM Foundation website. https://www.choosingwisely.org/societies/american-association-of-neuromuscular-electrodiagnostic-medicine/. Published 2021. Accessed 1 Jan 2021. 2021.
17. McCormick Z, Cushman D, Caldwell M, et al. Does electrodiagnostic confirmation of radiculopathy predict pain reduction after transforaminal epidural steroid injection? A multicenter study. J Nat Sci. 2015;1(8):e140.
18. Fish DE, Shirazi EP, Pham Q. The use of electromyography to predict functional outcome following transforaminal epidural spinal injections for lumbar radiculopathy. J Pain. 2008;9(1):64–70.
19. Annaswamy TM, Bierner SM, Chouteau W, Elliott AC. Needle electromyography predicts outcome after lumbar epidural steroid injection. Muscle Nerve. 2012;45(3):346–55.
20. Lin CK, Borresen A, Kroll M, Annaswamy TM. Predicting response to epidural steroid injections for lumbar spinal stenosis with biomarkers and electromyography. PM R. 2020;12(7):663–70.
21. Alrawi MF, Khalil NM, Mitchell P, Hughes SP. The value of neurophysiological and imaging studies in predicting outcome in the surgical treatment of cervical radiculopathy. Eur Spine J. 2007;16(4):495–500.

Conventional and Advanced Imaging Evaluation of Spine

4

Girish Boraiah and Avneesh Chhabra

History of Spine Imaging

X ray discovery by Roentgen happened in the year 1895. But, the use of Spine x-rays started around 1920s and immediately, it was followed by X-ray tomography. Earliest clinical use of fluoroscopy, then called Cineradiography, was for the evaluation of joint movement in 1905. Subsequently fluoroscopy was incorporated into surgery and also for the evaluation of artificial limb fitted stump. Spine Fluoroscopy was gradually incorporated into intraoperative imaging over the next few years [1]. Use of Spine x-rays to assess trauma, spine instability and screening of many disease entities like infections, developmental aberrations and tumors has retained relevance to this day. Tomography (before being replaced mostly by CT) has the advantage over plain X-rays for the detection of subtle osseous abnormalities, such as complex fractures, bone fragments within the spinal canal, and cortical erosions. Later pneumoencephalography, pneumomyelography and myelogram using Lipiodol type contrast slowly emerged as additional tools to diagnose intracranial tumors, spinal cord tumors and hydrocephalus. For myelogram, thorium dioxide (Thorotrast), iophendylate (Pantopaque) and Meglumine were used for decades with less frequent adverse reactions or severe complications. Subsequently in the early 1970s, less toxic nonionic water-soluble contrast

G. Boraiah
Dr. Chandramma Dayananda Sagar Institute of Medical Education & Research, Harohalli, Ramanagara, Karnataka, India

A. Chhabra (✉)
University of Texas at Southwestern Medical Center, Dallas, TX, USA

Johns Hopkins University, Walton Center of Neurosciences, Liverpool, UK
e-mail: avneesh.chhabra@utsouthwestern.edu

© The Author(s), under exclusive license to Springer Nature Switzerland AG 2022

C. E. Noe (ed.), *Multidisciplinary Spine Care*,
https://doi.org/10.1007/978-3-031-04990-3_4

Metrizamide and in 1980s, iohexol and iopamidol came into use. Spinal angiography started around 1970s. Selective catheterization using the Seldinger technique and subtraction technique was developed subsequently for assessing small abnormal vessels and related spinal vascular malformations. Till today, digital spinal angiography remains the study of choice due to an inherent advantage of superior spatial resolution and multiple angiographic runs on table, even though CT (computed tomography) angiography and MR (magnetic resonance) angiography are widely available. First EMI CT scanner introduced in 1973 could image only the head. In 1975, CT imaging of the spine became available followed by introduction of post-myelographic CT. In early 1980s, Lumbar spine CT with contrast was extensively used in post-operative/degenerative spine disease, tumors and infection assessment. Even with low field, 0.15 T–0.6 T MRI scanners in the early 1980s, the contrast resolution was superior to the best available CT scanners at that time. With the introduction of intravenous Gadolinium based contrast agents (GBCA) in 1988, the spinal cord and its pathology were better visualized on contrast enhanced MRI than CT, despite the fact that only low field magnets were available. Ultrasound was also gaining popularity and it was prudently used for spinal assessment in infants for meningocele, tethered cord, etc. Ultrasound has though limited role in adult spine imaging [2].

With advances like 3D (dimensional) imaging, dual energy CT scanning, metal artifact reduction techniques, fast scanning machines / techniques, low dose scanning (using automated iterative approaches and dose modulation), dynamic contrast scanning and so on, the capabilities of CT imaging have been at the new frontiers. Intra-operative CT and CT fluoroscopy have been made real-time imaging possible with excellent surgical and interventional radiology planning. Even with such advancements, the main drawbacks of CT are radiation exposure and poor soft tissue contrast, when compared to MRI. Hence, MRI with latest technology like 3-T (tesla) imaging, multi-channel spine coils, metal artifact reduction, fast scanning techniques with turbo spin-echo, 3D imaging, motion studies using gradient-echo sequences, echo-planar imaging, and parallel imaging, etc. has become the first choice for advanced spinal imaging in almost all conditions. Newer sequences like DWI (diffusion weighted imaging), DTI (diffusion tensor imaging), CSF flow studies, MR Neurography (MRN), MR spectroscopy (MRS), and perfusion MR imaging have provided abundance of imaging details, which were not otherwise possible. Navigational MRI systems are also being exploited similar to CT imaging for assistance with robotic and interventional procedures [3].

This chapter will focus on the commonly used imaging modalities for the evaluation of spinal pathologies so that the reader can learn their appropriate indications and role in different spinal conditions. Imaging appearances of various pathologies are discussed with relevant case examples. Finally, advanced and emerging imaging modalities in the domain of spine imaging are also highlighted.

Morbidity Related to Spine

By Medical Expenditure Panel Survey, approximately 6% of US adults reported an ambulatory visit for a primary diagnosis of a back or neck condition (13.6 million people in the year 2008). Between the years 1999 and 2008, the mean inflation adjusted annual expenditures on medical care, chiropractic care, and physical therapy (three of the most common ambulatory health services utilized by individuals with spine conditions) for these patients increased by 95% (from $487 to $950 per patient per year). Approximately $90 billion is spent on the diagnosis and management of low back pain, and an additional $10 to $20 billion is attributed to economic losses in productivity each year.

The frequency of ambulatory visits for Intervertebral disk disorders, sprains and strains, and for disease related to spinal curvature is at 18.7%, 7.0%, and 2.8%, respectively [4]. The 2010 global burden of disease study estimated that low back pain is among the top ten diseases and injuries that account for the highest number of disability-adjusted life years (DALY) worldwide.

Imaging Evaluation

Following a through clinical assessment, radiography is the first line screening modality. This may be supplemented with fluoroscopy as needed. Advanced imaging with CT and MRI is indicated in specific circumstances, as outlined in the subsequent sections.

Radiographic Evaluation of Spine

The radiographic (X-ray) imaging of cervical, thoracic and lumbosacral spine is commonly performed using frontal (anteroposterior, AP), lateral, and bilateral oblique views. X-rays serve as the first and cost-effective screening modality for spinal evaluation in almost all conditions, except in emergent post-traumatic assessment where CT might be chosen as the initial screening modality. AP view allows optimal assessment of the scoliosis, vertebral count, lumbosacral transitional vertebra (LSTV), transverse process fracture, pedicular involvement/injury, paravertebral soft tissues, uncovertebral joint, and sacroiliac (SI) joint disease. Lateral view is optimal for the evaluation of spinal curvature, sagittal balance assessment, evaluation of short pedicles, vertebral compression and spinal process fractures, vertebral retropulsion, Baastrup's disease, spinal listhesis, atlanto-axial dislocation and prevertebral soft tissues. Oblique views are optimal for facet joints, spondylolysis, SI joint, and neural foraminal assessment. Libson et al. concluded that 20% of pars interarticularis fractures (spondylolysis) were detected only on the oblique views

[5]. Special X-ray views are obtained for different regions and various indications, e.g. bending views for scoliosis, and flexion and extension views for listhesis and potential spinal instability. More than 2 mm anteroposterior motion and asymmetrical disc narrowing are indicators of anteroposterior and rotational instability, respectively. Anterolisthesis is graded from I to IV based on 4 quarters of end-plate widths of the inferior vertebra, e.g. grade IV anterolisthesis is >75% slippage of the vertebra. Spondyloptosis is referred to as Grade V by many. More than grade II anterolisthesis usually has spondylolysis in association. Retrolisthesis is graded from I to III based on degree of neural foraminal stenosis in thirds, i.e. mild, moderate, or severe stenosis correspond to the grade I–III. LSTV is classified based on Castellvi classification into class I–IV. The diagnostic standard for Lumbar segmental instability (LSI) is excessive translational or rotational movements between lumbar vertebrae, accomplished by using functional (flexion–extension) radiographs, with development of 2 mm or more listhesis or more than 5–10° of rotation component, the latter is though more difficult to identify.

In the cervical spine, main features to evaluate are vertebral body height, transverse processes, overlapping articular processes of facet joint, uncovertebral joint, equal intervertebral spaces, centrally placed spinous processes and medial ends of upper ribs. Also visible are soft tissues mainly muscles, lung apices and the central air-filled trachea. Less than or more than 50% anterolisthesis is associated with unilateral or bilateral facet dislocations, respectively. The 1st (atlas) and 2nd (axis) cervical vertebrae are best assessed on the open mouth view. Open mouth view is an AP projection which shows central odontoid peg of the axis, bilateral lateral masses of atlas along with bilateral atlantoaxial joints equidistantly placed from the midline (peg) (Fig. 4.1). Open mouth view can identify C1 burst (Jefferson's) fracture, C2 Odontoid (Dens) fracture), alar or transverse ligament injury and basilar invagination. Fuch's view of the Odontoid process can be used as an alternative in cases with no history of acute spinal injury. If there is widening of one side, say right sided lateral atlantoaxial space, it should correspond to rotation of the face towards right

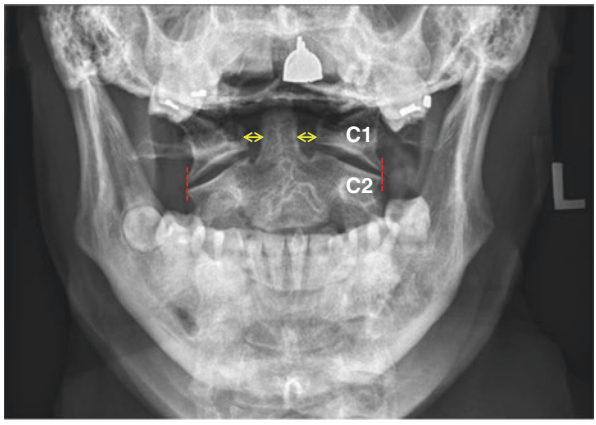


Fig. 4.1 Open mouth view. Equal distance between dens to ring of C1 on either side (↔). Normal alignment of lateral margins of C1 and C2 (red lines)

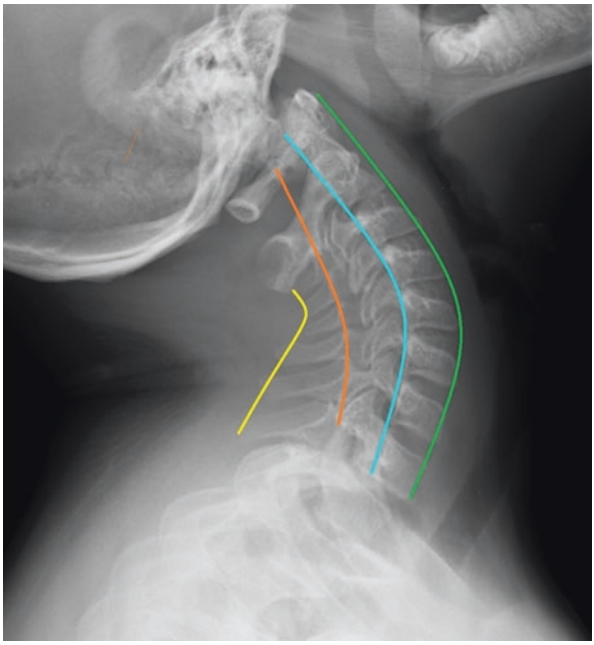


Fig. 4.2 Cervical spinal contour lines. 1. Anterior vertebral line (Green). 2. Posterior vertebral line (Blue). 3. Spinolaminar line (Orange). 4. Posterior Spinous line (Yellow)

side. If the face is rotated to the other side, rotary subluxation diagnosis can be confirmed.

Lateral view shows the prevertebral soft tissue thickness/space (it is abnormal if it measures >7 mm at C2 level and >22 mm in adults or >14 mm in children (<15 years) at C6, or is roughly larger than the corresponding vertebral body width or if there is focal dense soft tissue bulge at any level), atlantoaxial interval (normal is up to 2.5 mm in adults and 5 mm in children), intervertebral spaces, continuous C2 ring (Harris ring), spinous processes, dens, anterior arch of the atlas, facet joints with superior and inferior articulating surfaces. C2 vertebra has the largest body and C3 represents the reference vertebral height to compare for the evaluation of compression fractures. C7 has the largest spinous process. Look for parallelism of the facet joint articulations. It is important to note four spinal contour lines to evaluate instability or malalignments, especially in the trauma setting (Fig. 4.2). Step-off in the contours of these lines is pathologic except in pseudolisthesis, which can be seen at C2–3 and C7–T1 levels.

1. Anterior vertebral line connecting the anterior margins of the vertebrae.
2. Posterior vertebral line connecting the posterior margin of the vertebrae.
3. Spinolaminar line connecting the confluence of bases of the spinous processes and the posterior margin of laminae, depicts the posterior margin of the spinal canal. This line represents the most important line during alignment evaluation as it is not disrupted in pseudolisthesis.

4. Posterior Spinous line connecting the posterior margin of the spinous processes. If space between spinous processes is widened, it suggests interspinous ligamentous injury in setting of trauma.

Os odontoideum is a congenital variant of the axis, which is variable in size and shape, well corticated, smooth and separate from rest of short odontoid process. It can be identified and differentiated by X-rays with the open mouth, anteroposterior, and lateral views. Dynamic lateral flexion and extension views may provide information about atlanto-axial instability. CT/MRI may be necessary in few cases to glean additional information [7]. Atlantooccipital assimilation is a partial or complete congenital fusion between the atlas and the base of the occiput, which often requires CT for complete evaluation [8]. If apex of dens breaches the plane of the foramen magnum, then basilar impression is suspected. When the dens protrudes above the foramen magnum, a basilar invagination is diagnosed, which can result in chronic headaches, limited neck motion, and acute neurologic deterioration [9]. These entities are optimally assessed by CT/MRI. Craniometry [10] through MRI plays a crucial role in evaluation and management of these craniovertebral junction anomalies.

Denis [11] divided spine into three columns to assess spine instability secondary to trauma, which was an improvisation of the prior two column classification from Holdsworth (1970). Anterior column comprises anterior half of vertebral body, anterior half of annulus fibrosus and anterior longitudinal ligament. Middle column comprises posterior half of vertebral body, posterior half of annulus fibrosus and posterior longitudinal ligament. Posterior column comprises posterior bony arch of vertebra, supraspinous ligament, interspinous ligament, ligamentum flavum and facet joint capsule. Involvement of two or more columns is associated with instability and reduced load carrying capacity (Fig. 4.3) [12]. A lateral spine x-ray can identify 75% of fractures with a sensitivity of 85%. The sensitivity increases to over 90% when a full series of AP, lateral, oblique and open mouth X-rays are obtained (Fig. 4.4) [6]. Cervicothoracic (swimmer's view) lateral projection of cervical spine with arm by side of the head allows better visualization of C-7, T-1, and T-2 vertebrae due to uncovering of the spine from the bony and soft tissues of the shoulder girdle (Fig. 4.5).

Radiography in Scoliosis

Angulation of the lateral spinal curvature with Cobb angle of 10° or more is referred to as Scoliosis. If Cobb angle is less than 10°, it is called spinal asymmetry. Frontal X-rays are used to grade the vertebral rotation by Nash-Moe method, to measure coronal balance and to evaluate Cobb angle (Figs. 4.6 and 4.7). Cobb's angle changes with standing frontal, supine rightward- and leftward-bending radiographic views. These views along with standing lateral view (used to measure sagittal balance) are employed to classify scoliosis by Lenke system, which is widely used in guiding surgery. Another study by Alberto Ofenhejm Gotfryd et al. suggested using

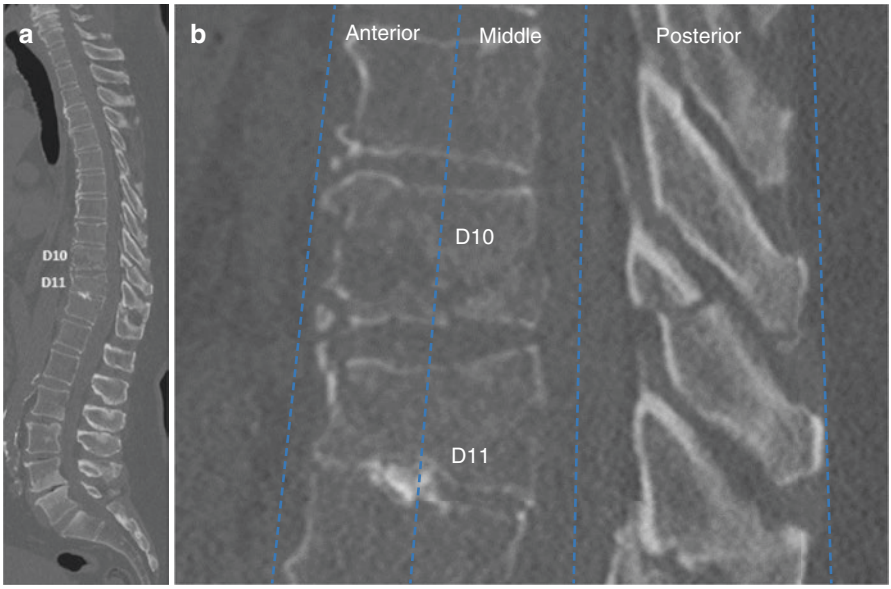


Fig. 4.3 Three column fractures of the spine involving D11 and D10 respectively, on sagittal CT. (**a**) shows sagittal reconstruction from 3D CT and (**b**) shows zoomed view of the fractures at D10/11 level

a lateral oblique view radiograph in supine position to predict the percentage operative correction achievable using pedicle screws for the main thoracic curve, in patients with Adolescent idiopathic scoliosis of Lenke types 1A and 1B. For idiopathic scoliosis in adolescent and adults with Cobb angles of less than 20° and 30° respectively, follow-up imaging at 4–12-month intervals suffices. Bracing and surgery are generally opted when the Cobb angle is between 20° to 45° and greater than 45°, respectively. Such therapeutic decisions are also based on the age and scoliosis progression [13].

In a study by Hasegawa et al. [14] showed that in x-ray and CT imaging, the measurements (Pelvic tilt, pelvic incidence, Cobb and rotation angles of the major curve) especially of the thoracolumbar area, were significantly greater in the standing position than in the supine position. Whereas the sacral slope was significantly smaller in the standing position than in the supine position.

Digital Video Fluoroscopy

Digital video fluoroscopy (DVF) to assess normal and abnormal lumbar spinal motions in vivo has been suggested by many as being superior to the static flexion–extension views [15]. Lumbar flexion-extension motion has been assessed with simultaneous use of electrogoniometer and videofluoroscopy. Cost and practical feasibility renders flexion-extension radiography being preferred over

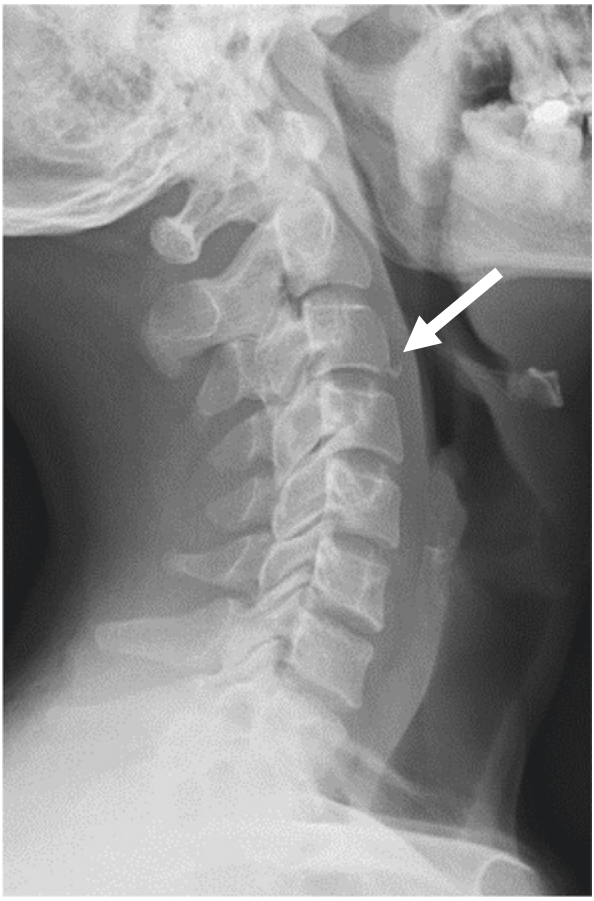


Fig. 4.4 Extension teardrop injury at C3 – Most often, it is a stable injury (arrow)

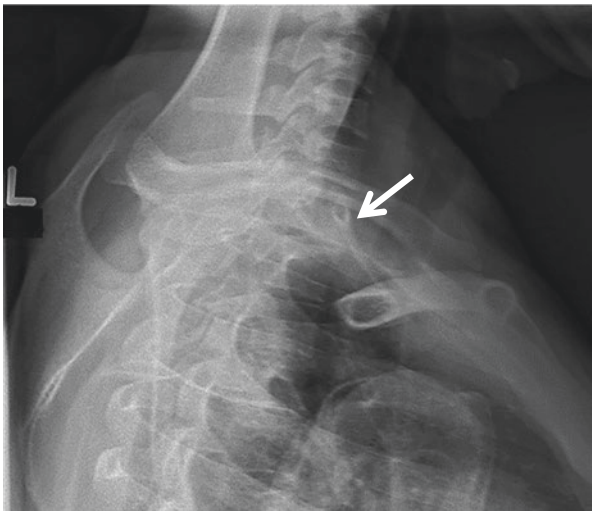


Fig. 4.5 Swimmer's view enables good visualization of the cervicothoracic junction



Fig. 4.6 Whole spine X-ray for scoliosis assessment. Major rotatory dextroscoliosis centered in the mid-lower thoracic spine

video-fluoroscopy. Flexion-extension radiographs have limited utility in the acute setting with high false-negative and false-positive rates [16]. A study comparing flexion-extension radiographs with CT also concluded that they are not efficacious when a negative CT has been performed in blunt trauma without neurological findings [17]. Dynamic fluoroscopy also does not identify additional fractures or instability that has not been identified on CT imaging [18].

Various forms of fluoroscopy have been in use for a long time in orthopedic surgery, especially during spinal surgery. Earlier form of fluoroscopy was with x-ray tube with fluorescent screen [19] and later X-ray Image Intensification with Television became available in the late 1950s [20]. Mobile C-arm image amplifier with television fluoroscopy became available around 1975 [21]. Recently automated C-arm positioning by deep learning process has been shown to improve accuracy on synthetic images derived during the procedure [22].

Fluoroscopy has now become part and parcel of many spine surgical procedures especially spine fixation [23], facet or epidural injection, cage placement and vertebroplasty. Fluoroscopy aids in radiological visualization of the bony structures and instrumentation allowing minimal invasiveness, and thus doing away with direct operative visualization / large operative exposure of the tissues. Fluoroscopy-based procedures have led to safer procedures, shorter procedural time, reduced blood



Fig. 4.7 Whole spine X-ray for scoliosis assessment. S-shaped scoliosis curvature of the thoracolumbar spine and major levoscoliosis of the mid-lower lumbar spine

loss, and early recovery of patients (Fig. 4.8). Main drawbacks of fluoroscopy are steep learning curve for the beginners and radiation exposure, especially to personnel who are involved in long duration procedures or multiple fluoroscopic procedures routinely [24, 25]. With newer technology in fluoroscopy like isocentric 3D C-arm or O-arm with computer-based navigation system in one pass, it is possible to provide 3D reconstruction of the spine, and the image acquisition could be done without the staff being in the operative room, thereby limiting the radiation exposure [26]. These systems have been shown to decrease the overall procedural time while reducing the radiation exposure to the staff [26–29].

Transforaminal extradural and interlaminar epidural steroid and /or anesthetic injections are used to treat cervical radiculopathy (Fig. 4.9). Similarly, radiofrequency ablation (RFA) of medial branch of the spinal dorsal ramus aka Facet joint denervation / rhizotomy, and intra-articular facet steroid injection addresses facetogenic pain. According to one study, fluoroscopically guided lumbar spine epidural injections led to inadvertent intravascular injection in 12%, and it was more common with transforaminal injections [30]. Similarly cervical fluoroscopy guided transforaminal injections was associated with some complications including epidural hematoma [31]. Many studies have provided sufficient evidence to state that

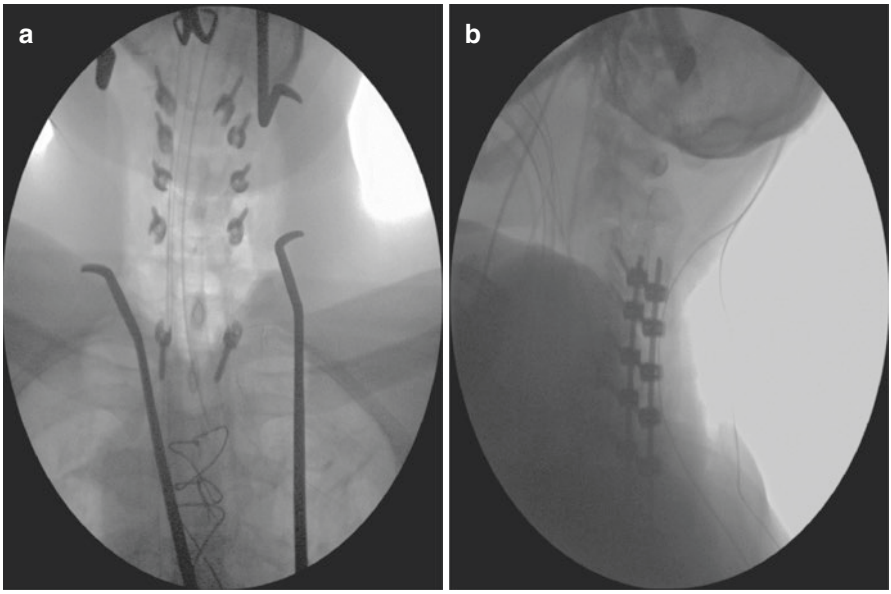


Fig. 4.8 C-arm fluoroscopy of intraoperative cervical spine fixation (**a**) shows pedicular screws with tissue retractors in the middle of fixation procedure and (**b**) shows confirmation of position of rods with screws at the end of fixation

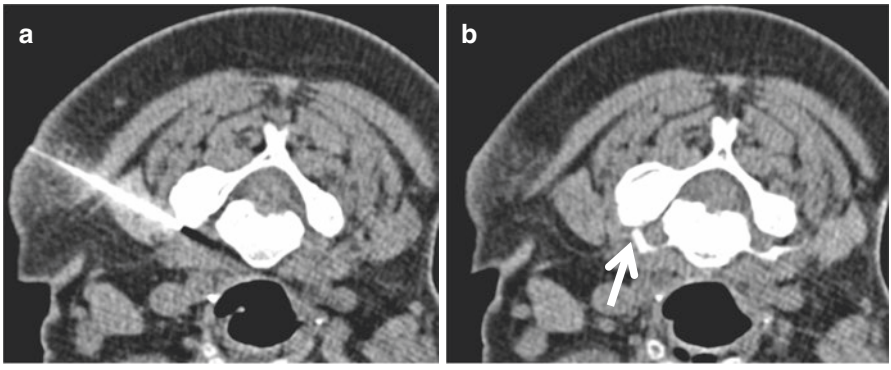


Fig. 4.9 CT guided transforaminal epidural cervical spinal injection (**a**) shows needle tip in epidural space and (**b**) shows contrast spread in epidural space (arrow)

CT guided approach to these injections is safer and effective, though more expensive (Fig. 4.10) [32–35].

In cervical transforaminal injection, the needle tip needs to be placed in junctional location between the foraminal zone and extraforaminal zones. For facet joint injection, needle trajectory should match the joint line curvature (Figs. 4.9 and 4.10). For medial branch block or RFA, the needle needs to be placed between the superior articular process and transverse process.

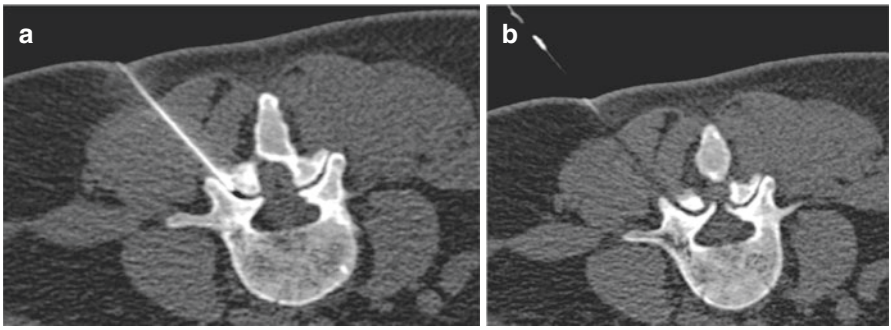


Fig. 4.10 CT guided cervical facet joint injection. (**a**) shows needle tip in facet joint and (**b**) shows minimal contrast in joint space

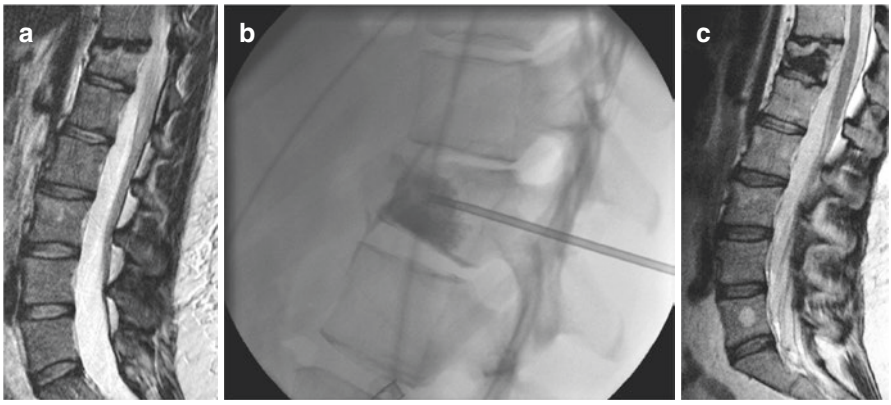


Fig. 4.11 Cementoplasty of wedge compression (**a**) wedge compression of T12vertebra with a hemangioma that deteriorated over months, (**b**) shows fluoroscopic cementoplasty using a 13-gauge Osteo-Site needle via transpedicular approach and (**c**) shows maintenance of height even after 7 years after cementoplasty. Newly formed hemangioma of L5 can also be visualized

Percutaneous cementoplasty or vertebroplasty replaces part of the diseased vertebral body with acrylic cement (polymethylmethacrylate [PMMA]). This prevents vertebral body collapse and its untoward consequences like exiting nerve root compression or sometimes impingement of the spinal cord, thereby alleviating pain. Percutaneous cementoplasty was first performed by Deramond et al. in 1984. Relevant indications for this procedure are severe painful osteoporosis, painful vertebral body / sacral tumors and acetabular tumors, and symptomatic vertebral angioma (Fig. 4.11). During cementoplasty, PMMA polymerizes releasing energy as heat and, also by its cytotoxic nature coagulates the adjacent tumoral cells. Bleeding and infection are the main contraindications. Other complications include cement leak into spinal canal or adjacent veins, infection, post-procedural pain, adjacent segment fractures, and allergic reactions [36].

Discography is a method in which contrast is injected into intervertebral disk using a fine needle, which can reproduce the patient's back pain. The assumption is that if the disc disease is the cause of back pain, then injection of contrast would recreate the pain by stimulating the nerve innervating the annulus fibrosus. CT or MRI based discography can also be performed using corresponding contrast for injection. Whereas fluoroscopy-based discography shows leak and location of the tear, CT and MRI also demonstrate anatomical details of the tears, which might be useful for treatment and surgical planning. Discography was first described by Swedish radiologist Lindblom in 1940s to assess the primary discogenic source of back pain. Main drawbacks for discography are duration involved in the procedure, radiation (if Fluoroscopy or CT used), low diagnostic sensitivity as per many studies, and a complication rate of about 2% for lumbar and 13% for cervical discography. Discography has also been advocated prior to surgery (nucleotomy, spondylodesis) where clinical and imaging could not accurately identify the level of a disc pathology as the causative factor of the back pain and is also used to differentiate scar tissue from recurrent disc prolapse [37–40]. It is however well known that back pain is a result of chronic degenerative changes of the intervertebral discs that occurs at multiple levels, and discography might not be able to identify the exact level for maximal pain. Though MRI identifies many aspects of the disc disease, such pathologies might not translate into defining the cause of pain.

Computed Tomography (CT)

CT is an important modality for the diagnosis of multiple diseases of the spine with a predominant current role in trauma. CT detects subtle fractures, canal / foraminal stenosis, retropulsed bony fragments within canal / foramina, osteophytes, subluxation, spondylolysis, spondylolisthesis and multiple other bony pathologies in an acute setting of trauma. Main advantages of CT lie in capability of 3D reconstruction in multiple planes with better visualization of bony structures than MRI (Figs. 4.3, 4.12, and 4.13). Another important advantage of CT especially in scenario of trauma is faster acquisition and ability to do high resolution angiogram concomitantly. Main drawbacks of CT are radiation exposure, and suboptimal evaluation of the spinal cord, ligamentous and soft tissue structures. Thus, CT plays a key role in spine trauma to achieve early diagnosis and aids in instituting early management. CT also complements MRI in subacute setting and provides additional information e.g. about calcific changes and bony structures, which may be crucial for treatment and pre surgical planning. CT with metal artifact reduction techniques might be better than MRI in assessing spine for bony structures, implant and canal size after spine instrumentation (Fig. 4.12).

CT imaging of the thoracolumbar spine can be concomitantly obtained when thorax or abdomen-pelvis imaging is done in trauma patients for possible visceral, soft-tissue or vascular injuries. Separate dedicated thoracolumbar imaging is not required and bone window reconstruction from abdominothoracic trauma protocol

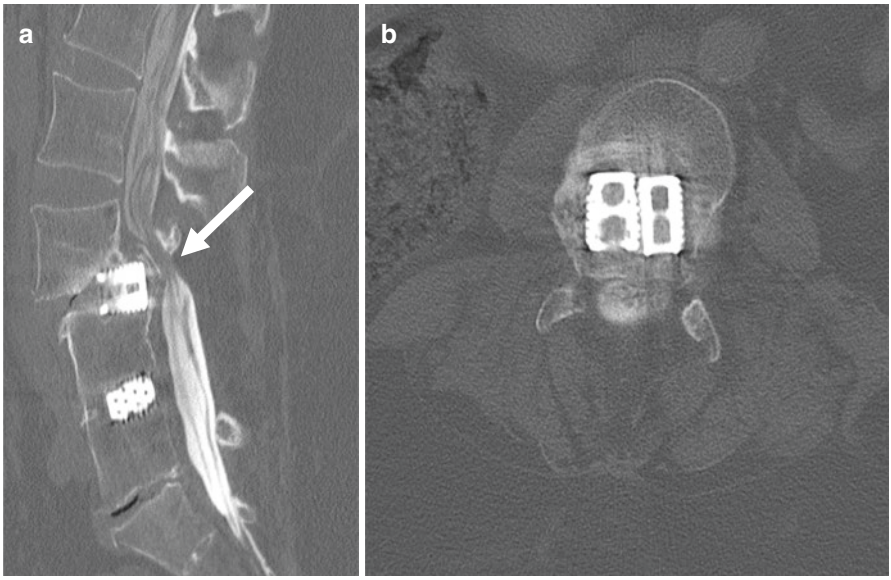


Fig. 4.12 CT myelogram with metal reduction technique. (**a**) Sagittal reformat and (**b**) axial image shows thecal sac narrowing (arrow) at L3–L4 with anterolisthesis and interbody spacer despite spinal decompression. There is no evidence of CSF leak

delivers a sensitivity of 98% and specificity of 97% for the detection of spinal fractures [41]. CT findings in posterior column distraction and potentially unstable posterior ligamentous complex injury includes compression fracture with loss of more than 40% vertebral body height, more than 25° kyphotic angle, interspinous distance widening, posterior column fractures with horizontal orientation, facet joint diastasis, facet joint subluxation or facet joint dislocation. Abdominal hollow visceral injury, mesenteric injury and seat belt injury are often associated with chance-type and transverse process fractures, which may be missed unless evaluated using multiplanar reconstructions [42].

To reduce implant related artifacts and achieve better images, several modifications and techniques are used during CT imaging, including higher peak voltage (>120–140 KVp), higher tube current, lower pitch, smooth reconstruction kernels, metal artifact reduction reconstruction algorithms and dual-energy data acquisition with virtual monoenergetic extrapolation postprocessing. Immediately after spinal instrumentation surgery, CT is performed to assess the proper reduction of fracture, implant placement and also to rule out significant hematoma (which can compress on spinal cord or other important neural structures). Short term or long term follow up with CT is done to evaluate the implant itself, its position, osteolysis around and changes in adjacent soft tissues including collection / particle disease (adverse local tissue reaction) (Fig. 4.12). Though soft tissue can be best evaluated by MRI, initial evaluation for infection and other changes in soft tissue can be best done with CT with lesser artifacts. Variable position of the implant can lead to varied adverse

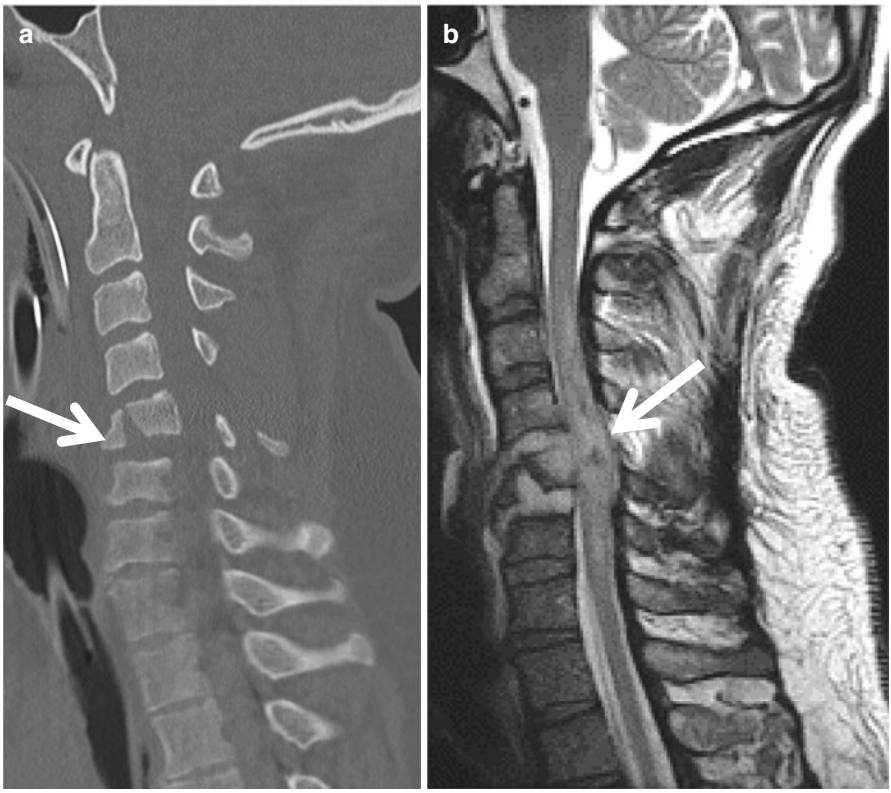


Fig. 4.13 Flexion teardrop injury at C5 – Most often, it is unstable (associated with hemorrhagic spinal cord in this patient, arrows)

outcomes and symptoms based on the position of the implant in relation to the adjacent important structures like thecal sac, neural elements and other soft tissues. Implant failure can occur due to altered dynamics or repetitive stress leading to fractures and / or disengagement of fixation construct [43].

CT myelography even though invasive, has advantages over conventional MR in its ability to obtain dynamic images, postoperative imaging for metallic implant related complications, evaluate slow cerebrospinal (CSF) leaks, superficial siderosis and in cases where MRI is contraindicated. CT myelography best demonstrates the pathologies that contact or narrow the spinal thecal sac or cord (Fig. 4.12), or displace the spinal cord, nerve roots and thecal sac. CT myelography is useful in assessing compressive cystic lesions like spinal arachnoid cysts, spontaneous cord herniation, arachnoid webs, and other intradural cystic lesions. It helps to differentiate such entities and diseases and hence is useful for surgical planning. Filling the arachnoid cyst intrathecally with contrast material also helps exclude neuroenteric cysts. Spinal cord herniation is another important differential to exclude and an absence of CSF ventral or ventrolateral to the cord, lack of CSF loculation dorsal to

the cord, and on postmyelography CT an absence of delayed myelographic CSF opacification dorsal to the cord are important findings that suggest cord herniation rather than an arachnoid cyst [44]. Spinal canal narrowing from Calcium Pyrophosphate Deposition Disease or Ossification of the Posterior Longitudinal Ligament and its effect on thecal sac is also better assessed with CT myelography [45].

CT myelography has disadvantages of being an invasive procedure with risks associated with intrathecal contrast injection, exposure to radiation, and requirement of patient mobilization for contrast to diffuse to the point of interest [46]. Significant spinal extra-arachnoid fluid collections on preprocedural spinal MR imaging can be evaluated with either dynamic CT or digital subtraction myelography [47]. Dynamic CT Myelography is better for assessment of Fast Spinal CSF Leaks [48].

Magnetic Resonance Imaging (MRI)

Role of MRI in spinal imaging is to identify and differentiate pathologies due to vascular, ischemic, infective, inflammatory, neoplastic, demyelination, degenerative, congenital, traumatic and metabolic causes. MRI provides superior soft tissue contrast than other modalities, has multiplanar capability with no radiation exposure, and can specifically demonstrate individual tissue characteristics, such as water, blood, fat, infarction, proton diffusion and perfusion using a multitude of sequences. MRI clearly provides pathologic information about spinal cord, intervertebral disc, ligaments, tendons and paraspinal muscles. It can characterize disc disease into disc bulge, protrusion, extrusion, sequestration, annular fissure and so on (Figs. 4.14, 4.15, and 4.16). Disc dessication is common with aging. In most subjects, lumbar disc levels tend to dessicate beyond the age of 40 years and cervical discs beyond 20–25 years. Disc bulge on MRI demonstrates as extension of the disc material beyond the margins of the vertebra. Posterior disc bulges are important as they can impinge on the thecal sac or the nerve roots. Bulge, by definition, involves more than 90 degrees of the posterior one-half of the disc circumference and is caused by inner annular fissures. Outer annular fissures result in disc herniations, with 45–90° extent, being referred to as a broad-based disc herniation. In general, extrusions and sequestrations of discs are more frequently symptomatic than protrusions. Disc protrusion is broad based and displays obtuse angles with the parent disc on the axial image, doesn't extend beyond the margins of the disc on the sagittal image, maintains dessicated dark T2 disc signal and can demonstrate coexistent annular fissure, which themselves may persist for a long time. Extrusions exhibit a narrower neck with the parent disc, money bag appearance, extend above and below the disc level on sagittal image, are larger, and demonstrate increased T2 signal alteration due to the frequent associated peri-discal inflammatory tissue. The latter can also show rim enhancement on post-contrast images and is also a significant contributor of reduced disc herniation appearance on follow-up MRI with conservative management and physical therapy. Sequestration involves disruption of disc

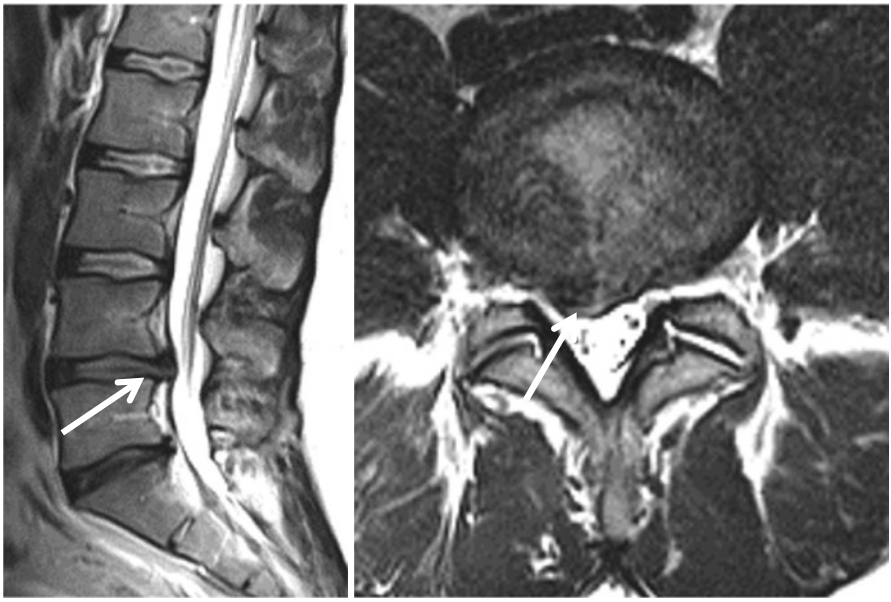


Fig. 4.14 Sagittal and axial T2W MR images show right paracentral disc protrusion

material from the parent disc and superior or inferior migration. Presence of rim enhancement distinguishes it from an extra- or intra-dural tumor (Fig. 4.16). Another important role of MRI is to evaluate the edematous changes in the bone from stress changes. End-plate changes are classically divided into three Modic types (I) edema, (II) fatty metamorphosis, (III) sclerosis, but often, a mixture exists, with edema like T2 signal more associated with pain symptoms. Since, spine spondylosis is part and parcel of normal aging, MRI is also useful to identify coexisting inflammation, infection, or enthesopathy.

MRI has a substantial role in the evaluation of spinal infections. Most common causes include- Staph Aureus, Tuberculosis (TB), Brucellosis and so on, each one with some recognizable patterns of spine involvement. X-rays usually do not provide much information about infection till advanced stage of infection when there is enough destruction of vertebral bone, disc space loss, and significant soft tissue collection/gas formation. CT identification of early infection is suboptimal and often indirect through periosteal reaction, bone erosions, gas formation in soft tissue or sufficient soft tissue collection to be able to identify rim enhancement on contrast study. MRI can identify infection at an early stage by identifying the edema pattern or minimal collection in bone/soft tissue structures. Thin rim contrast enhancement and associated central diffusion restriction suggests infection over neoplasm. Involvement of two adjacent vertebral bodies along with intervening disc and soft tissue phlegmon or abscess (Fig. 4.17) often depicts pyogenic etiology. Multiple vertebral involvements can often be seen in tuberculosis, brucellosis and fungal etiology. Relative sparing of the disc with gibbus formation favors TB. Mixed

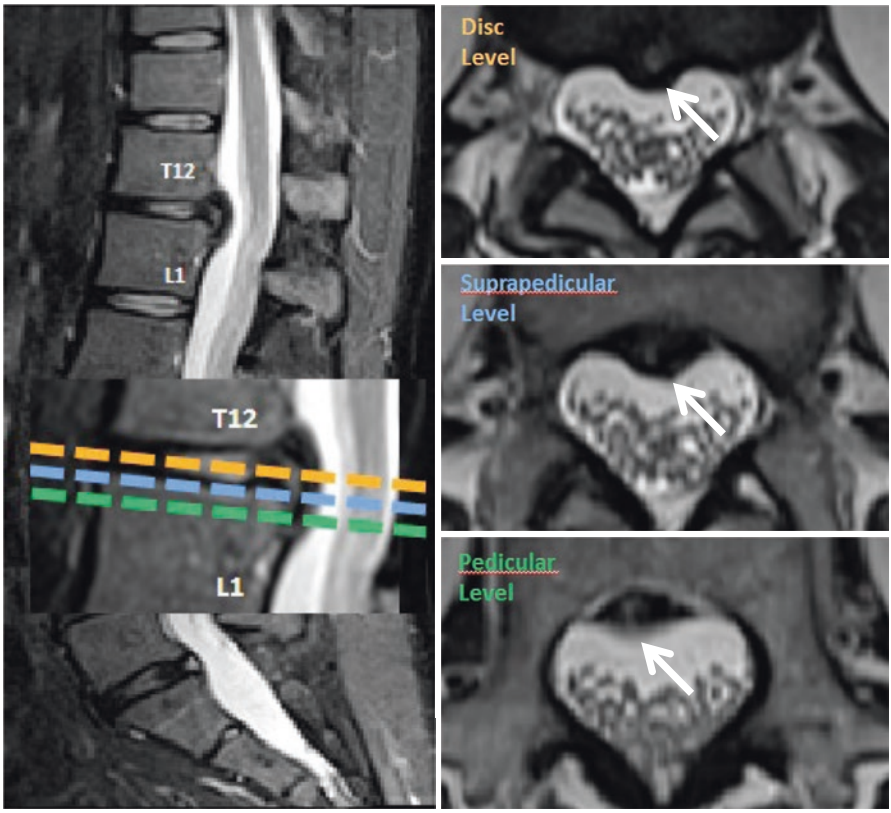


Fig. 4.15 Sagittal and sequential axial T2W MR images show a disc extrusion (arrows) with caudal migration (from disc (Orange) to Pedicular (Green) level)

intensity, less fluidy collections and associated lung lesions or calcification on X-rays favors TB as well. Brucellosis shows involvement of entire vertebral body with sclerosis on plain films and periostitis while gibbus is rare. Hypointense fungal elements in soft tissues along with lytic and sclerotic changes in vertebral body are characteristic of fungal etiology. MRI is useful in finding collections for drainage and can guide the site of biopsy. It is also helpful in follow-up of patients for resolution of infection.

Inflammatory/autoimmune disease of the spine can be distinguished into one involving the spinal cord specifically and the other involving the bony spine. Diseases involving the spinal cord include multiple sclerosis (MS), acute disseminated encephalomyelitis (ADEM), transverse myelitis (TM), neuromyelitis optica spectrum disorders (NMOS) and so on. Inflammatory disease of the bony spine or axial spondyloarthropathy occur specifically secondary to ankylosing spondylitis, psoriasis, lupus arthritis, and rheumatoid arthritis. X-ray and CT overall has no significant role in the diagnosis of inflammatory or autoimmune disease of the spinal cord. In axial spondyloarthropathy, X-ray and CT provide diagnostic and prognostic

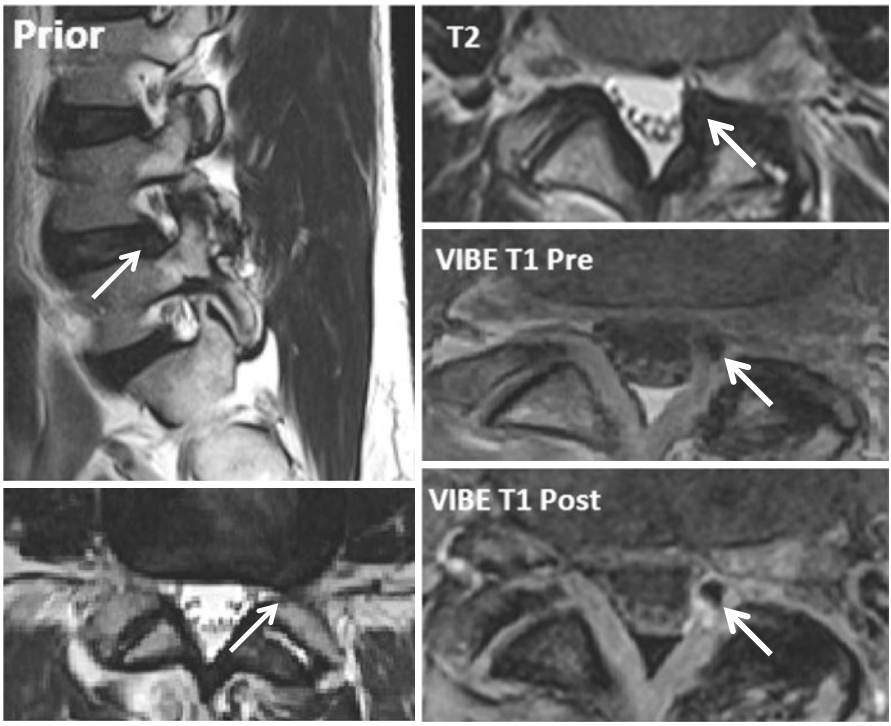


Fig. 4.16 Sequestrated migrated disc material. Extradural peripherally enhancing fragment of extruded migrated disc lying at same level as previous foraminal disc extrusion. VIBE- volume interpolated breath hold examination

information only in the later stages of the disease. MRI remains the cornerstone (Fig. 4.18) of early diagnosis and management of inflammatory / autoimmune disease of both the spinal cord and also the bony spine [49, 50].

Multiple short segment peripheral cord involvement is seen in MS. Whereas long segment holocord involvement is more typically seen in the setting of ADEM, NMOS and transverse myelitis. Using imaging alone, it is difficult to differentiate between multiple sclerosis and NMO. Nonspecific imaging features which support NMO are optic neuritis, less frequent involvement of brain (confluent large hyperintensities in NMOS, when compared to oval lesions in MS), and contiguous long segment central cord involvement. According to one meta-analysis [51] for NMOS, Aquaporin 4 antibody has sensitivity and specificity based on type of assay of about 70 and 95, respectively. TM unlike ADEM does not involve brain, whereas ADEM can involve spinal cord in one-third of the patients. The inflammatory diseases of the spine can be identified with specific enthesopathy patterns (for example, Romanus and Andersson lesions in ankylosing spondylitis reflecting anterosuperior/anteroinferior corner erosions and end plate erosions at discovertebral junctions, respectively), location involved (cervical spine involvement in rheumatoid arthritis and sacroiliac joint in seronegative arthropathies). Use of multiparametric

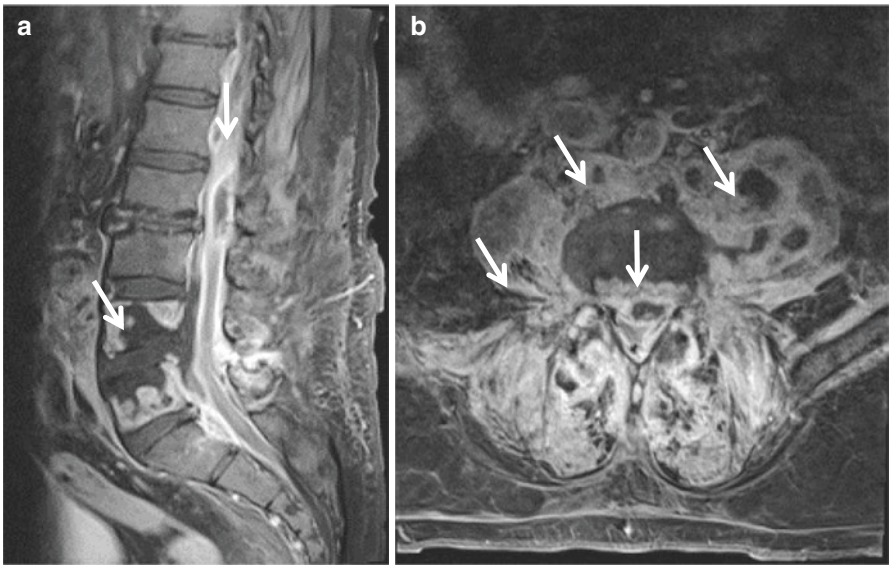


Fig. 4.17 Discitis-osteomyelitis of L4–L5 along with paravertebral abscesses (arrows) and epidural abscesses (arrow-heads) on T1 FS post-contrast (**a**) Sagittal image show the full craniocaudal extent of vertebral involvement and (**b**) Axial image shows epidural phlegmonous and abscess component (arrowhead) compressing on spinal cord

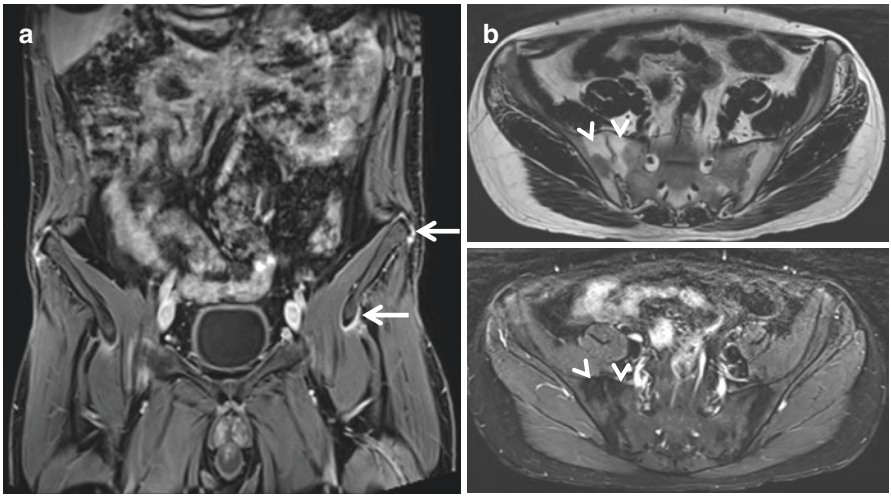


Fig. 4.18 Spondyloarthropathy spectrum (**a**) Coronal T1 FS post-contrast image shows bilateral anterosuperior and anteroinferior iliac spine enhancement of tendinous attachment (arrows) depicting acute enthesitis and (**b**) Axial without and with fat saturation images shows fatty metamorphosis (arrow-heads) and partial ankylosis of bilateral SI joints confirming long-standing spondyloarthritis

and 3D rheumatology lumbosacral MR imaging (MRLI) protocol helps in accurate diagnosis of various stages (acute, subacute and chronic) of inflammatory activity in bones, entheses, ligaments, tendons and joints [49, 50]. Acute lesions typically demonstrate bone marrow edema and chronic lesions show fatty changes/sclerosis. Thus, MRI provides information about the stage of disease and disease activity, which is not only useful in treatment but is also extremely relevant in prognosis and follow-up strategy [52–54].

Tumors of spine may involve the spinal cord (intramedullary, extramedullary and extradural) or the bony spine. Primary neoplasms of the bony spine are rare when compared to the metastatic lesions, except benign hemangiomas. They occur with a incidence of about 5 per 1,00,000 person-years [55]. Most of the primary bony spine neoplasms are benign, most common being enostosis and hemangioma. The incidence of spinal cord tumors is less (Less than 1 per 1,00,000 person-years [56]) compared to the bony spinal tumors. Most common spinal cord related tumors being glioma and ependymoma, and the intra-dural mass lesions include- meningioma and schwannoma/neurofibroma. On radiography, while bony lesions can demonstrate typical missing pedicle of metastasis, punched out lesions of myeloma, corduroy appearance of hemangioma, and sclerotic metastases of prostate cancer, etc.; for spinal cord tumors, x-ray provides only indirect evidence in some tumors due to scalloping and erosion of the bony spine. CT for bony spinal tumor adds more information compared to x ray by providing additional details about matrix characteristics throughout the tumor including calcification of chondrosarcoma, and ossification of osteosarcoma, as well as details of gross dimension of the tumor and bony spinal canal narrowing. CT is most helpful for pre-surgical planning before spinal decompression and/or fusion as well as for CT guided biopsy. For spinal cord tumors, CT provides suboptimal evidence of only thickened spinal cord or calcification if present. The modality is inadequate to differentiate tumor from other etiologies, like infection or inflammation. For bony spinal tumors, MRI is better than CT to evaluate the extent of bone marrow involvement (Fig. 4.19) and also provides high resolution details about extraosseous soft tissue and neurovascular bundle involvement. Though MRI provides extensive details about the matrix, it might not provide details about subtle calcification and ossification. Apart from common metastatic, lymphoma and myeloma lesions, typical locations of bone tumors include- vertebral body (T2 bright lesions- hemangioma (Fig. 4.11), chordoma and chondrosarcoma, T2 dark lesion- Giant cell tumor), pedicles (osteoid osteoma <1.5 cm size and osteoblastoma >1.5 cm), posterior elements (chondrosarcoma, osteochondroma, and aneurysmal bone cyst with fluid-fluid levels). MRI helps in localization of tumor into intramedullary (glioma- ill-defined margins, cervicothoracic area location; ependymoma- sharp margins, cystic changes and hemorrhage, cervical and lumbar locations, and hemangioblastoma- cyst with a vascular nodule), intradural-extramedullary (meningioma, peripheral nerve sheath tumor, and angiolipoma) and extramedullary (metastasis, myeloma, lymphoma, etc.). MRI provides comprehensive details about composition of the tumor, accurate dimensions and extent of the lesion.

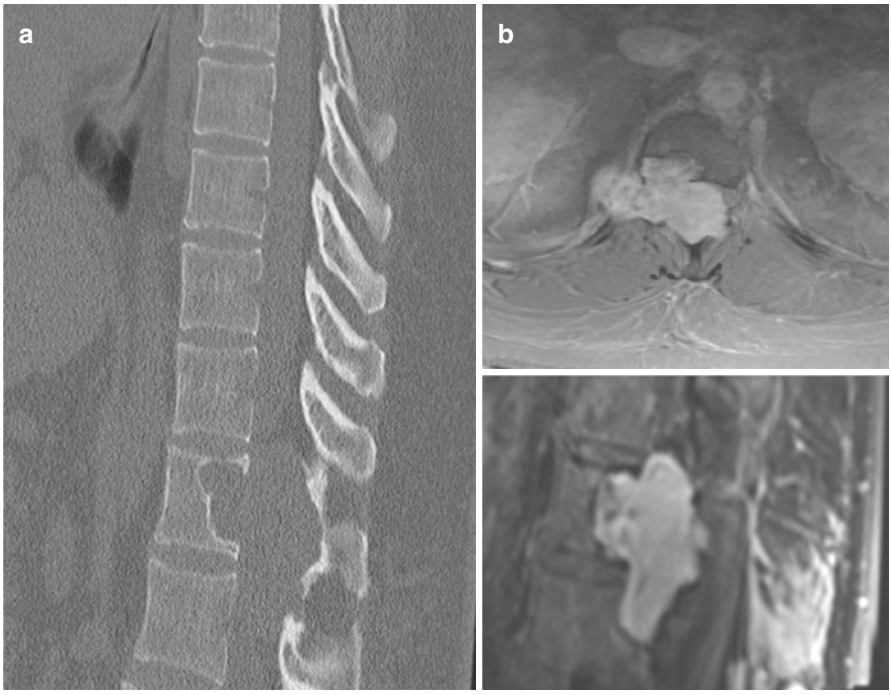


Fig. 4.19 Schwannoma (**a**) Sagittal CT reformat shows vertebral lytic lesion with large intraspinal component and (**b**) T1 FS post-contrast axial and sagittal images however shows homogenous enhancing nerve sheath tumor centered around right neural foramen with dumbbell like extraforaminal and intraspinal component along with anterior vertebral body scalloping

Vascular disorders include vascular malformations, hemorrhage and infarction. Plain x rays provide no significant information in the diagnosis of vascular disorders of the spine (which are rare and constitute about 1–2% of vascular neurologic pathologies) [57]. However digital subtraction angiography (DSA) plays a key role in the diagnosis and management of spinal vascular lesions including arteriovenous fistula and arteriovenous malformation, which are most common such lesions. CT angiogram is useful in the evaluation of hemorrhage and spinal vascular pathologies. CT has no role in spinal cord ischemia (contributing to less than 1% of all strokes) [57]. MRI is the imaging modality of choice for spinal vascular malformation and the angiogram can be obtained using time of flight (2D/3D) and contrast-enhanced sequences. MRI also provides details about congestive spinal cord edema secondary to the vascular malformation and details the soft tissue component of the vascular malformation. Apart from various sequences MRI, susceptibility weighted imaging (SWI) is sensitive in identifying spinal cord bleed and diffusion weighted imaging (DWI) is sensitive in identifying infarctions [57].

Congenital and developmental spine abnormalities range from craniovertebral junction abnormalities to various neural tube anomalies. Congenital bony spine abnormalities can manifest as alterations in normal size and shape of vertebra,

which occur mainly due to variation in fusion of ossification centers, and rarely due to absence of the ossification center itself. Congenital bony spine abnormalities include hemivertebra, butterfly vertebra, block vertebra, spur of diastematomyelia, hypoplasia and aplasia.

Spinal dysraphism is a large set of congenital anomalies secondary to defective neural arch with herniation of meninges or neural elements and associated clinical manifestations. Herniation of neural elements to skin surface can be open (spina bifida aperta) or can be covered by skin, closed (spina bifida occulta) dysraphism. Closed Spinal Dysraphism may go undetected throughout life, as most are asymptomatic. Complex bony spine abnormalities are often associated with spinal cord anomalies [58] and are more common (10 per 10,000 live births) than spinal dysraphisms (3.2–4.6 per 10,000 births) [59]. Ultrasound when performed prenatally, may detect many open neural tube defects as early as 11 weeks and segmentation anomalies of vertebra around 16 weeks. Postnatally, ultrasound is useful in infants, but less so in later ages. Plain x rays can identify congenital bony spine anomalies. CT can identify more subtle bony spine anomalies along with providing some information about soft tissue (especially lipomatous) component of the neural elements. Congenital lumbar canal stenosis (CLSS) and transitional vertebrae (Fig. 4.20) can be identified on CT and MRI [60]. MRI is the imaging modality of choice for spinal dysraphism as it can provide most of the details including the composition of the open neural tube defect and it also helps in identification of subtle entities like neuroenteric cysts, meningeal cysts, syringohydromyelia and so on. MRI is essential in

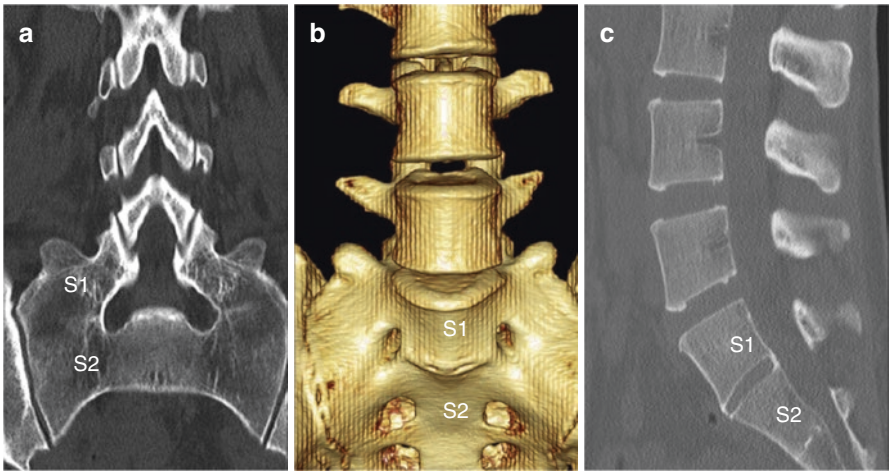


Fig. 4.20 Transverse process of lumbarised S1 (transitional vertebra) fused bilaterally with the sacral transverse process below - Castellvi type IIIb seen on (**a**) CT Coronal reformat, (**b**) Volume rendered image and (**c**) CT Sagittal reformat. (Castellvi types I, II and III depends on transverse processes being large-dysplastic, showing pseudo-articulation with sacrum and complete osseous fusion with sacrum, respectively. Subtypes are further classified unilateral (I/II/III a) or bilateral (I/II/III b). Whereas type IV (with no subtype) is combination of unilateral type II and contralateral type III)



Fig. 4.21 MR myelogram using Coronal heavily T2W steady state sequence shows preganglionic avulsion of the left C7–T1 nerve roots with associated pseudomeningoceles (arrow)

preoperative planning of open neutral tube defects. Fetal MRI is able to identify many of the spinal dysraphisms in utero, differentiate between open and closed spinal dysraphism [61] and also identify fusion anomalies of the spine.

MRI is also the modality of choice for failed back surgery syndrome and other miscellaneous causes of cord abnormalities, such as subacute combined degeneration. Other advances of MR imaging include- MR myelography (Fig. 4.21) and upright MRI. MR myelogram can be performed using heavily T2 weighting or following injection of intrathecal Gadolinium. MR myelogram has uses like CT myelogram and carries other advantages, such as better depiction of neural structures and no radiation. MRI myelography has been shown of value in conditions like tethered cord, adhesive arachnoiditis, disc herniation, spinal arteriovenous malformation, post-traumatic pseudomeningoceles and so on. With improvements in conventional MRI resolution, a common use of myelogram has been to non-invasively detect CSF leak. Off label use of intrathecal gadolinium is also shown to have high rate of detection of CSF leaks compared to CT [47]. The concerns of encephalopathy or seizures after MR myelogram are rare with newer contrast agents [62].

Apart from supine MRI, various other position-based MRI techniques are being evaluated, e.g. (1) Positional MRI (pMRI)-Imaging in varying weight-bearing

positions (e.g. standing, seated or in the positions that worsen symptoms), (2) Kinetic MRI-Static imaging of kinetic maneuvers (e.g., flexion, extension, rotation, lateral bending), and (3) Dynamic MRI with images acquired while the spine is in real-time motion. Serial images played as cine loops nicely show the dynamic movements of the spinal column and pathologic alterations [63, 64]. MRI with neck flexion and extension can reveal dynamic stenosis or Hirayama disease (flexion myelopathy). Upright MRI is believed to replicate the expected effects of body weight and posture has on the spinal curvature and important spinal structures like neural foramina and spinal canal. Recent studies claim that upright MRI can portray occult stenosis, disc protrusion, or instability, which otherwise would have not been clearly assessed in supine MRI. Study by Ferreiro Perez et al. [65] showed posterior disc herniation was underestimated on supine MRI when compared to upright MRI. Similar results of the disc pathology at lumbar spine (L5–S1 followed by L4–L5 and L3–L4) were seen well on upright MRI, as was shown by Gilbert et al. [66]. Meakin et al. [67] showed an increase in curvature under load during upright MRI, when compared against supine MRI. Tarantino et al. [68] showed that dynamic MRI with an open-configuration using low-field tilting MRI system, permits visualization of occult spine and disc pathologies in patients with acute or chronic low back pain who had MRI in the recumbent position or in patients with pain only in the upright position. However, other studies show that no significant difference in various spinal parameters in upright MRI when compared to supine MRI, and thus, the modality has not gained widespread acceptance.

Special MRI Sequences and Applications in Spine Imaging

Diffusion-weighted imaging (DWI) and Diffusion-tensor imaging (DTI) help assess the isotropic and anisotropic diffusion of water molecules respectively, thereby interrogating proton diffusion at a cellular level. DWI renders early visualization and diagnosis of spinal cord and brain infarcts, and identification of small spinal tumors with utmost confidence. Advances in DWI have led to its use in benign and malignant bony spinal lesions, spinal cord lesions, pre and post treatment in infections or malignant lesions. Recently in 2018 Park et al. [69] showed that differentiation of multiple myeloma and metastases is possible with axial diffusion-weighted MR imaging (Fig. 4.22). Daghighi et al. [70] showed that with DWI, it is possible

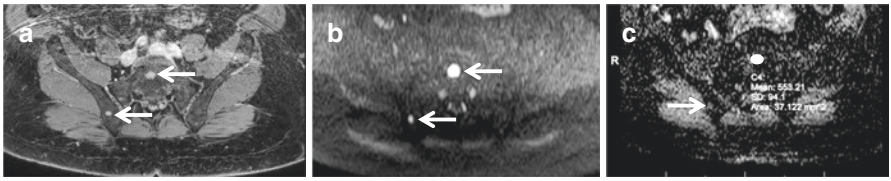


Fig. 4.22 Multiple myeloma (**a**) shows T1 FS post-contrast axial image are seen as discrete enhancing lesions of the sacrum and right ilium, (**b**) shows bright lesions on DWI and (**c**) shows lesions with restricted diffusion (ADC of 0.6×10^{-3} mm^2/s)

to differentiate acute infectious spondylitis from degenerative Modic type 1 change. Significant diffusion restriction is seen with highly cellular, higher grade, and round cell tumors. Pus also restricts as compared to simple fluid collection. Myxoid and chondroid lesions however do not significantly restrict. With evolution of DTI, spinal cord tracts can be evaluated that could provide a road map for conservative surgery to preserve critical (usually motor) neural tracts. DWI of the spine correlates well with the presence or absence of spinal infection and may complement conventional magnetic resonance imaging (MRI) with median ADC value being 740×10^{-6} mm^2/s for patients with positive microbiological sampling and 1980×10^{-6} mm^2/s for patients with negative microbiological sampling ($p < 0.001$) [71]. DWI and DTI usefulness has also been shown in immune-mediated encephalitis, neuritis and neurodegenerative disorders [72]. Finally, DWI has been shown to be useful in detection and quantification (useful for follow-up) of subtle inflammatory changes in Spondyloarthropathy not seen on other MR conventional images (Fig. 4.23) [52–54, 73].

MR Neurography (MRN) is an imaging dedicated to diagnosing peripheral neuropathy and is being rapidly used for characterizing neuromuscular diseases. With advances in fat suppression, fast MRI techniques, 3 T MR scanners and 3D imaging; rapid acquisition of images without temporal degradation in image quality is possible with good isotropic resolution in the range of 0.9–1.5 mm. 3D anatomic nerve-selective MR Neurography results in effective vascular signal suppression and differentiates the nerves from vascular structures within a neurovascular bundle. Fat suppressed 3D DW PSIF (reversed fast imaging in steady state free precession) is one of excellent nerve-selective MRN techniques [74]. Role of MRN has been established in peripheral neuropathy, nerve injury, nerve sheath tumor, nerve entrapment or impingement. MRN also has been used to exclude neuropathy in pathologies mimicking neuropathy and to provide imaging guidance for perineural medication injections [75]. MRN has a significant role in anatomically complex brachial [76] and lumbosacral plexus pathologies [77]. MRN has been applied in diagnosis, treatment and follow-up of nerve related pathologies due to various etiologies (Fig. 4.24). Recently, the technology has been used in diagnosis of greater occipital nerve neuropathy in patients with unilateral occipital migraines with a

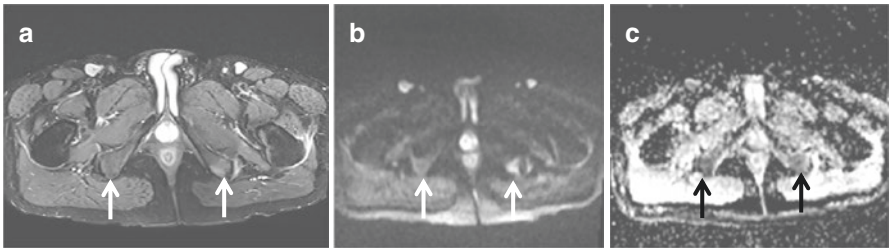


Fig. 4.23 Bilateral (Left > right) ischial tuberosity enthesitis (**a**) shows STIR axial image with subtle edema visible only on left, (**b**) shows bright signal bilaterally on DWI and (**c**) shows altered diffusion bilaterally

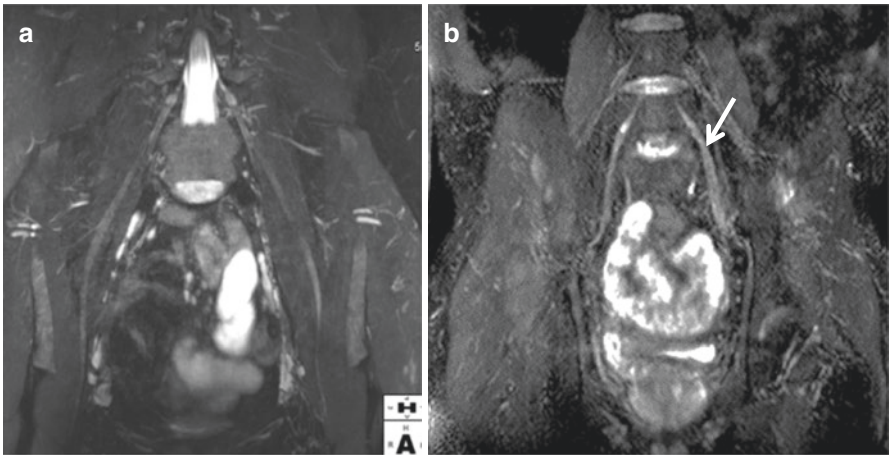


Fig. 4.24 Coronal MIP images of 3D STIR in different patients (**a**) shows bilaterally symmetrical normal femoral nerves, (**b**) shows abnormal thickening of left L5 nerve with obscuration of the left dorsal nerve root ganglion in this case of left L5 radiculopathy (arrow)

good correlation of imaging findings to the clinical presentation [78]. One study on MRN of Lumbosacral Plexus in Failed Back Surgery Syndrome (FBSS) has found neuroforaminal stenosis, iatrogenic nerve injuries, and neuropathy in substantial number of patients who had non-contributory conventional spine MRI so that specific treatment approaches could address the issue of FBSS [79]. DTI employed as part of MRN also reveals neuropathy with reduced fractional anisotropy and increased apparent diffusion coefficient of the affected nerves.

CSF flow imaging of the spine using phase-contrast MRI sequence obtains signal contrast between flowing and stationary nuclei by using opposite gradient sensitization at two different time points. The sequence yields signal from the moving nuclei and nulls signal from the stationary nuclei. Using magnitude and phase images, quantitative and directionality assessments can be done. To distinguish motion, this sequence applies anticipated velocity encoding (VENC) which is the expected maximum CSF flow, generally 5–8 cm per second. Lower VENC like 2–4 cm per second is useful to differentiate communicating versus non-communicating arachnoid cysts and is also useful to evaluate VP shunts for possible obstruction. Higher VENC of 20–25 cm per second depicts high velocity CSF flow, as seen within cerebral aqueduct in normal pressure hydrocephalus [66]. As CSF flow is pulsatile and synchronous with the cardiac cycle, either prospective or retrospective cardiac gating yields the best imaging and assessment [67]. Craniovertebral junction pathologies both congenital and acquired alter CSF flow, which is the main cause for the development of hydrocephalus and symptoms. Improved CSF velocity in such cases after surgery are associated with favorable response. If CSF flow is seen within syringomyelic cysts, it provides a clue to the possibility of further enlargement and helps to distinguish it from myelomalacia, which is a close differential on conventional imaging [68, 69].

Chemical shift imaging (CSI) makes use of the differences in precession frequencies of lipid and water protons within the same imaging voxel acquired using different echo times. CSI leads to output where lipid and water signals are additive (in-phase) or subtracted (opposed-phase). This helps to assess vertebral bone marrow fat content in benign processes (osteoporosis, hemangiomas, degenerative endplate changes, etc.) versus malignant infiltrative processes (e.g. leukemia, lymphoma and metastasis), thereby potentially avoiding biopsy in a significant percentage of patients. Signal drop-out of 20% as a cut-off can be used to differentiate benign lesions from malignant lesions (Fig. 4.25) [80–83]. CSI can also be used to differentiate vertebral compression fractures of benign from malignant etiologies [84].

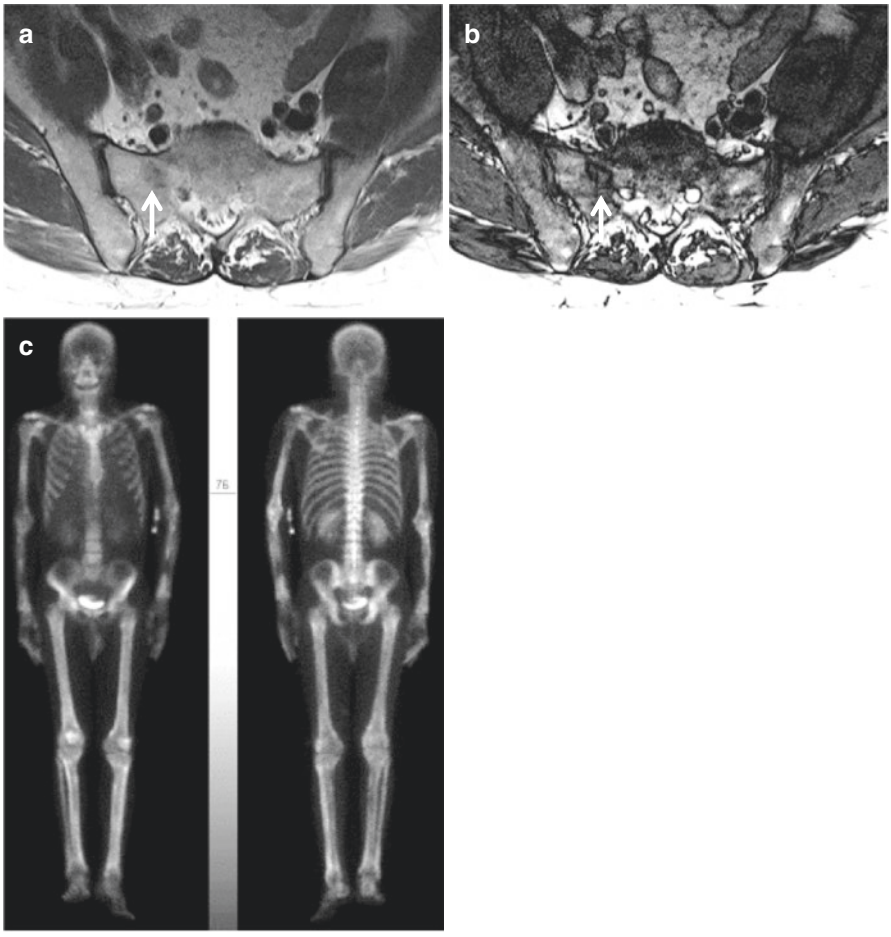


Fig. 4.25 Focal lesion in Midline of sacrum (**a**) Shows In-phase image with altered signal, (**b**) Shows Out-of-phase image with loss of signal of more than 20% in corresponding area. and (**c**) Shows bone scan without corresponding uptake. This lesion was unchanged over years and was classified as focal red marrow conversion

MR perfusion-weighted imaging (PWI) assesses the amount of blood flow into tissues and thus, also assesses biologic behavior of neoplasms, identifies ischemic/infarcted regions and aids in characterization of other lesions/diseases. Perfusion MRI techniques can be done with or without using an exogenous contrast agent. Dynamic susceptibility contrast (DSC) assesses signal loss in T2 or T2* by the passage of the bolus of contrast agent through the tissue. Dynamic contrast enhanced (DCE) assesses increase in signal on T1 before and after passage of the bolus of contrast agent in the tissue. Without using contrast, arterial spin-labeling (ASL) assesses magnetically labeled blood on T1 to estimate perfusion. ASL can be used as pulsed or continuous. ASL is used to assess cerebral blood flow (CBF) and takes about 5–8 min to acquire. ASL has also been used experimentally in patients with discogenic pain and to evaluate vascularity of spinal tumors. PWI has thus been used to assess spinal neoplasm - primary malignant, metastatic lesions, and benign lesions like hemangioblastoma to evaluate tumor biology and vascularity. PWI also been used to predict outcomes of spinal lesions with encouraging results [85]. Using perfusion studies, ischemia and hypoxia has been studied in the pathogenesis of myelopathy and to suggest early intervention to prevent full blown myelopathy and future disability [86].

Future Directions

Extensive research is happening at a faster pace in various parts of the world, bringing newer technologies and uses in spinal imaging for a variety of pathologies. To mention a few, Paraspinal Muscle and extremity muscle segmentation on CT or MRI using automated computer software with Atlas-based tools. Apart from muscle bulk measurement, it also provides information about the amount of fatty infiltration. Manual annotation of the muscles is time-consuming and laborious. Automated pseudo-coloring technique or histogram analysis would likely lead to easy and accurate assessment of the different muscles and its pathologies [87]. The surrogate quantitative imaging markers can serve as treatment response and prognostic indicators.

Artificial intelligence has been tried in spine fracture detection on plain radiographs. With everyday improving robust and powerful computational power, the deep neural networks will become more advanced and there will be an extraordinary change, the way imaging is being interpreted and used. Mundane and repeated tasks can be accomplished by machine and the imaging interpretations of specific tasks, e.g. spine fracture or detection of compression fracture, will likely be done in an equivalent manner to expert radiologists. This will especially help medical care in remote locations, aiding in timely management of trauma and other patients [88, 89]. Artificial intelligence has also been shown to predict fractures in predisposed patients [90].

Role of Magnetization Transfer MRI, Diffusion Tensor Imaging, Diffusion non-Tensor Imaging (q-space), Myelin Water Imaging, fMRI and Perfusion in detailed evaluation of spinal cord in trauma are also being investigated [91]. Functional MRI

has been tried in spinal cord similar to what has been already established in brain imaging [92]. Diffusion tensor imaging (DTI) has been employed to assess microstructure of muscle tissue in its physiological and pathological stages. Thus, track subtle changes of muscle tissue composition especially in important muscles like back muscles. These strategies are being aimed at early interventions that can prevent occurrence or help better treat related pathologies affecting these muscles [93]. Dixon based fatty changes in muscles are shown equivalent to MR spectroscopy, a metabolic imaging quantitative technique [94].

Multiple studies have shown the application of Hybrid SPECT with CT fusion to identify potential sites for treatment in patients with axial neck and back pain. Presurgical assessment for hypermetabolic foci on spinal SPECT imaging correlating with back pain sites and similar post-operative assessment have been show to produce better outcomes in early investigations [95].

ASL can be used to assess marrow perfusion and hence biological changes within the bones. It might have role in finding bone loss, fatty conversion, directing interventions, and evaluate therapy response and prognosis based on perfusion changes [96].

To conclude, radiologic imaging of spine has come a long way with many advanced techniques being currently in use and many on horizon. Gaining understanding of optimal indications of different imaging modalities is essential for a reader to prudently apply these technologies in their practice for the benefit of patients.

Disclosures AC serves as a consultant for ICON Medical and Treace Medical Concepts Inc. AC receives royalties from Jaypee and Wolters.

Conflicts of Interest None.

References

1. Gillis L. Cineradiography in orthopaedic surgery. Br Med J. 1947;2(4516):140.
2. Hoeffner EG, Mukherji SK, Srinivasan A, Quint DJ. Neuroradiology back to the future: spine imaging. AJNR Am J Neuroradiol. 2012;33(6):999–1006.
3. Nouh MR. Imaging of the spine: where do we stand? World J Radiol. 2019;11(4):55–61.
4. Davis MA. Where the united states spends its spine dollars: Expenditures on different ambulatory services for the management of back and neck conditions. Spine. 2012;37(19):1693–701.
5. Libson E, Bloom RA, Dinari G, Robin GC. Oblique lumbar spine radiographs: importance in young patients. Radiology. 1984;151(1):89–90.
6. West OC, Anbari MM, Pilgram TK, Wilson AJ. Acute cervical spine trauma: diagnostic performance of single-view versus three-view radiographic screening. Radiology. 1997;204(3):819–23.
7. Jumah F, Alkhdour S, Mansour S, He P, Hroub A, Adeeb N, et al. Os odontoideum: a comprehensive clinical and surgical review. Cureus. 2017;9(8):e1551.
8. Electricwala AJ, Harsule A, Chavan V, Electricwala JT. Complete atlantooccipital assimilation with basilar invagination and atlantoaxial subluxation treated non-surgically: a case report. Cureus. 2017;9(6):e1327.

9. Donnally III CJ, Munakomi S, Varacallo M. Basilar invagination. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2020 [cited 2020 Aug 9]. Available from: http://www.ncbi.nlm.nih.gov/books/NBK448153/.
10. Smoker WR. Craniovertebral junction: normal anatomy, craniometry, and congenital anomalies. Radiographics. 1994;14(2):255–77.
11. Denis F. The three column spine and its significance in the classification of acute thoracolumbar spinal injuries. Spine. 1983;8(8):817–31.
12. Haher TR, Tozzi JM, Lospinuso MF, Devlin V, O'Brien M, Tenant R, et al. The contribution of the three columns of the spine to spinal stability: a biomechanical model. Paraplegia. 1989;27(6):432–9.
13. Kim H, Kim HS, Moon ES, Yoon C-S, Chung T-S, Song H-T, et al. Scoliosis imaging: what radiologists should know. Radiographics. 2010;30(7):1823–42.
14. Hasegawa K, Okamoto M, Hatsushikano S, Caseiro G, Watanabe K. Difference in whole spinal alignment between supine and standing positions in patients with adult spinal deformity using a new comparison method with slot-scanning three-dimensional X-ray imager and computed tomography through digital reconstructed radiography. BMC Musculoskelet Disord [Internet]. 2018 Dec 6 [cited 2020 May 26];19. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6284293/.
15. Ahmadi A, Maroufi N, Behtash H, Zekavat H, Parnianpour M. Kinematic analysis of dynamic lumbar motion in patients with lumbar segmental instability using digital videofluoroscopy. Eur Spine J. 2009;18(11):1677–85.
16. Harris TJ, Blackmore CC, Mirza SK, Jurkovich GJ. Clearing the cervical spine in obtunded patients. Spine. 2008;33(14):1547–53.
17. Como JJ, Diaz JJ, Dunham CM, Chiu WC, Duane TM, Capella JM, et al. Practice management guidelines for identification of cervical spine injuries following trauma: update from the eastern association for the surgery of trauma practice management guidelines committee. J Trauma. 2009;67(3):651–9.
18. Padayachee L, Cooper DJ, Irons S, Ackland HM, Thomson K, Rosenfeld J, et al. Cervical spine clearance in unconscious traumatic brain injury patients: dynamic flexion-extension fluoroscopy versus computed tomography with three-dimensional reconstruction. J Trauma. 2006;60(2):341–5.
19. Schueler BA. The AAPM/RSNA physics tutorial for residents: general overview of fluoroscopic imaging. Radiographics. 2000;20(4):1115–26.
20. Koslowski L, Weller S. The application of x-ray image intensification to orthopaedic surgery. Ger Med Mon. 1966;11(2):61–2.
21. Berci G, Zheutlin N. Improving radiology in surgery. Med Instrum. 1976;10(2):110–4.
22. Kausch L, Thomas S, Kunze H, Privalov M, Vetter S, Franke J, et al. Toward automatic C-arm positioning for standard projections in orthopedic surgery. Int J Comput Assist Radiol Surg. 2020;15(7):1095–105.
23. Magerl FP. Stabilization of the lower thoracic and lumbar spine with external skeletal fixation. Clin Orthop. 1984;189:125–41.
24. Herscovici D, Sanders RW. The effects, risks, and guidelines for radiation use in orthopaedic surgery. Clin Orthop. 2000;375:126–32.
25. Salvia JCL, de Moraes PR, Ammar TY, Schwartsmann CR. Fluoroscopy duration in orthopedic surgery. Rev Bras Ortop. 2011;46(2):136–8.
26. Narain AS, Hijji FY, Yom KH, Kudaravalli KT, Haws BE, Singh K. Radiation exposure and reduction in the operating room: perspectives and future directions in spine surgery. World J Orthop. 2017;8(7):524–30.
27. Hott JS, Papadopoulos SM, Theodore N, Dickman CA, Sonntag VKH. Intraoperative Iso-C C-arm navigation in cervical spinal surgery: review of the first 52 cases. Spine. 2004;29(24):2856–60.
28. Kim CW, Lee Y-P, Taylor W, Oygar A, Kim WK. Use of navigation-assisted fluoroscopy to decrease radiation exposure during minimally invasive spine surgery. Spine J. 2008;8(4):584–90.

29. Smith HE, Welsch MD, Sasso RC, Vaccaro AR. Comparison of radiation exposure in lumbar pedicle screw placement with fluoroscopy vs computer-assisted image guidance with intraoperative three-dimensional imaging. J Spinal Cord Med. 2008;31(5):532–7.
30. Husseini JS, Simeone FJ, Staffa SJ, Palmer WE, Chang CY. Fluoroscopically guided lumbar spine interlaminar and transforaminal epidural injections: inadvertent intravascular injection. Acta Radiol. 2020;61(11):1534–40.
31. Engel A, King W, MacVicar J, Standards Division of the International Spine Intervention Society. The effectiveness and risks of fluoroscopically guided cervical transforaminal injections of steroids: a systematic review with comprehensive analysis of the published data. Pain Med. 2014;15(3):386–402.
32. Cyteval C, Thomas E, Decoux E, Sarrabere M-P, Cottin A, Blotman F, et al. Cervical radiculopathy: open study on percutaneous periradicular foraminal steroid infiltration performed under CT control in 30 patients. AJNR Am J Neuroradiol. 2004;25(3):441–5.
33. Depriester C, Setbon S, Larde A, Malaquin E, Vanden Abeele B, Bocquet J. CT-guided transforaminal cervical and lumbar epidural injections. Diagn Interv Imaging. 2012;93(9):704–10.
34. Wald JT, Maus TP, Geske JR, Carter RE, Diehn FE, Kaufmann TJ, et al. Safety and efficacy of CT-guided transforaminal cervical epidural steroid injections using a posterior approach. AJNR Am J Neuroradiol. 2012;33(3):415–9.
35. Gossner J. Safety of CT-guided lumbar nerve root infiltrations. Analysis of a two-year period. Interv Neuroradiol. 2014;20(5):533–7.
36. Gangi A, Guth S, Imbert JP, Marin H, Dietemann J-L. Percutaneous vertebroplasty: indications, technique, and results. Radiographics. 2003;23(2):e10.
37. Bernard TN. Lumbar discography followed by computed tomography. Refining the diagnosis of low-back pain. Spine. 1990;15(7):690–707.
38. McCutcheon ME, Thompson WC. CT scanning of lumbar discography. A useful diagnostic adjunct. Spine. 1986;11(3):257–9.
39. Kluner C, Kivelitz D, Rogalla P, Putzier M, Hamm B, Enzweiler C. Percutaneous discography: comparison of low-dose CT, fluoroscopy and MRI in the diagnosis of lumbar disc disruption. Eur Spine J. 2006;15(5):620–6.
40. Walker J, El Abd O, Isaac Z, Muzin S. Discography in practice: a clinical and historical review. Curr Rev Musculoskelet Med. 2008;1(2):69–83.
41. Roos JE, Hilfiker P, Platz A, Desbiolles L, Boehm T, Marincek B, et al. MDCT in emergency radiology: is a standardized chest or abdominal protocol sufficient for evaluation of thoracic and lumbar spine trauma? AJR Am J Roentgenol. 2004;183(4):959–68.
42. Raniga SB, Skalski MR, Kirwadi A, Menon VK, Al-Azri FH, Butt S. Thoracolumbar spine injury at CT: trauma/emergency radiology. Radiographics. 2016;36(7):2234–5.
43. Ghodasara N, Yi PH, Clark K, Fishman EK, Farshad M, Fritz J. Postoperative spinal CT: what the radiologist needs to know. Radiographics. 2019;39(6):1840–61.
44. Ospina Moreno C, Vela Marín AC, Castán Senar A, Montejo Gañán I, Cózar Bartos M, Marín Cárdenas MA. Radiological diagnosis of spinal arachnoid cysts: a pictorial essay. J Med Imaging Radiat Oncol. 2016;60(5):632–8.
45. Patel DM, Weinberg BD, Hoch MJ. CT myelography: clinical indications and imaging findings. Radiographics. 2020;40(2):470–84.
46. Song K-J, Choi B-W, Kim G-H, Kim J-R. Clinical usefulness of CT-myelogram comparing with the MRI in degenerative cervical spinal disorders: is CTM still useful for primary diagnostic tool? J Spinal Disord Tech. 2009;22(5):353–7.
47. Chazen JL, Talbott JF, Lantos JE, Dillon WP. MR myelography for identification of spinal CSF leak in spontaneous intracranial hypotension. AJNR Am J Neuroradiol. 2014;35(10):2007–12.
48. Luetmer PH, Schwartz KM, Eckel LJ, Hunt CH, Carter RE, Diehn FE. When should I do dynamic CT myelography? Predicting fast spinal CSF leaks in patients with spontaneous intracranial hypotension. AJNR Am J Neuroradiol. 2012;33(4):690–4.
49. Taurog JD, Chhabra A, Colbert RA. Ankylosing spondylitis and axial spondyloarthritis. N Engl J Med. 2016;374(26):2563–74.

50. Alian A, Omar H, Chhabra A. Cross-sectional imaging for inflammatory arthropathy of the pelvis. Semin Ultrasound CT MR. 2017;38(3):279–90.
51. Ruiz-Gaviria R, Baracaldo I, Castañeda C, Ruiz-Patiño A, Acosta-Hernandez A, Rosselli D. Specificity and sensitivity of aquaporin 4 antibody detection tests in patients with neuromyelitis optica: a meta-analysis. Mult Scler Relat Disord. 2015;4(4):345–9.
52. Chhabra A, Ezzati F, Taurog JD, Xi Y, Pezeshk P. Three tesla and 3D multiparametric combined imaging evaluation of axial spondyloarthritis and pelvic enthesopathy. Eur J Radiol. 2020;126:108916.
53. Sanal HT, Yilmaz S, Kalyoncu U, Cinar M, Simsek I, Erdem H, et al. Value of DWI in visual assessment of activity of sacroiliitis in longstanding ankylosing spondylitis patients. Skelet Radiol. 2013;42(2):289–93.
54. Zhao Y, Li S, Liu Z, Chen X, Zhao X, Hu S, et al. Detection of active sacroiliitis with ankylosing spondylitis through intravoxel incoherent motion diffusion-weighted MR imaging. Eur Radiol. 2015;25(9):2754–63.
55. Dreghorn CR, Newman RJ, Hardy GJ, Dickson RA. Primary tumors of the axial skeleton. Experience of the Leeds Regional Bone Tumor Registry. Spine. 1990;15(2):137–40.
56. Schellinger KA, Propp JM, Villano JL, McCarthy BJ. Descriptive epidemiology of primary spinal cord tumors. J Neuro-Oncol. 2008;87(2):173–9.
57. Krings T, Lasjaunias PL, Hans FJ, Mull M, Nijenhuis RJ, Alvarez H, et al. Imaging in spinal vascular disease. Neuroimaging Clin N Am. 2007;17(1):57–72.
58. Trenga AP, Singla A, Feger MA, Abel MF. Patterns of congenital bony spinal deformity and associated neural anomalies on X-ray and magnetic resonance imaging. J Child Orthop. 2016;10(4):343–52.
59. Alexander PG, Tuan RS. Role of environmental factors in axial skeletal dysmorphogenesis. Birth Defects Res C Embryo Today. 2010;90(2):118–32.
60. Soldatos T, Chalian M, Thawait S, Belzberg AJ, Eng J, Carrino JA, et al. Spectrum of magnetic resonance imaging findings in congenital lumbar spinal stenosis. World J Clin Cases. 2014;2(12):883–7.
61. Nagaraj UD, Bierbrauer KS, Peiro JL, Kline-Fath BM. Differentiating closed versus open spinal dysraphisms on fetal MRI. AJR Am J Roentgenol. 2016;207(6):1316–23.
62. Nacar Dogan S, Kizilkilic O, Kocak B, Isler C, Islak C, Kocer N. Intrathecal gadolinium-enhanced MR cisternography in patients with otorhinorrhea: 10-year experience of a tertiary referral center. Neuroradiology. 2018;60(5):471–7.
63. Hansen BB. Introducing standing weight-bearing MRI in the diagnostics of low back pain and degenerative spinal disorders. Dan Med J. 2017;64(10):B5416.
64. Hansen BB, Nordberg CL, Hansen P, Bliddal H, Griffith JF, Fournier G, et al. Weight-bearing MRI of the lumbar spine: spinal stenosis and spondylolisthesis. Semin Musculoskelet Radiol. 2019;23(6):621–33.
65. Ferreiro Perez A, Garcia Isidro M, Ayerbe E, Castedo J, Jinkins JR. Evaluation of intervertebral disc herniation and hypermobile intersegmental instability in symptomatic adult patients undergoing recumbent and upright MRI of the cervical or lumbosacral spines. Eur J Radiol. 2007;62(3):444–8.
66. Gilbert JW, Martin JC, Wheeler GR, Storey BB, Mick GE, Richardson GB, et al. Lumbar disk protrusion rates of symptomatic patients using magnetic resonance imaging. J Manip Physiol Ther. 2010;33(8):626–9.
67. Meakin JR, Smith FW, Gilbert FJ, Aspden RM. The effect of axial load on the sagittal plane curvature of the upright human spine in vivo. J Biomech. 2008;41(13):2850–4.
68. Tarantino U, Fanucci E, Iundusi R, Celi M, Altobelli S, Gasbarra E, et al. Lumbar spine MRI in upright position for diagnosing acute and chronic low back pain: statistical analysis of morphological changes. J Orthop Traumatol. 2013;14(1):15–22.
69. Park GE, Jee W-H, Lee S-Y, Sung J-K, Jung J-Y, Grimm R, et al. Differentiation of multiple myeloma and metastases: use of axial diffusion-weighted MR imaging in addition to standard MR imaging at 3T. PLoS One. 2018;13(12):e0208860.

70. Daghighi MH, Poureisa M, Safarpour M, Behzadmehr R, Fouladi DF, Meshkini A, et al. Diffusion-weighted magnetic resonance imaging in differentiating acute infectious spondylitis from degenerative Modic type 1 change; the role of b-value, apparent diffusion coefficient, claw sign and amorphous increased signal. Br J Radiol. 2016;89(1066):20150152.
71. Dumont RA, Keen NN, Bloomer CW, Schwartz BS, Talbott J, Clark AJ, et al. Clinical utility of diffusion-weighted imaging in spinal infections. Clin Neuroradiol. 2019;29(3):515–22.
72. Drake-Pérez M, Boto J, Fitsiori A, Lovblad K, Vargas MI. Clinical applications of diffusion weighted imaging in neuroradiology. Insights Imaging. 2018;9(4):535–47.
73. Bozgeyik Z, Ozgocmen S, Kocakoc E. Role of diffusion-weighted MRI in the detection of early active sacroiliitis. AJR Am J Roentgenol. 2008;191(4):980–6.
74. Chhabra A, Zhao L, Carrino JA, Trueblood E, Koceski S, Shteriev F, et al. MR neurography: advances. Radiol Res Pract. 2013;2013:809568.
75. Chhabra A, Andreisek G, Soldatos T, Wang KC, Flammang AJ, Belzberg AJ, et al. MR neurography: past, present, and future. AJR Am J Roentgenol. 2011;197(3):583–91.
76. Chhabra A, Thawait GK, Soldatos T, Thakkar RS, Del Grande F, Chalian M, et al. High-resolution 3T MR neurography of the brachial plexus and its branches, with emphasis on 3D imaging. AJNR Am J Neuroradiol. 2013;34(3):486–97.
77. Soldatos T, Andreisek G, Thawait GK, Guggenberger R, Williams EH, Carrino JA, et al. High-resolution 3-T MR neurography of the lumbosacral plexus. Radiographics. 2013;33(4):967–87.
78. Hwang L, Dessouky R, Xi Y, Amirlak B, Chhabra A. MR neurography of greater occipital nerve neuropathy: initial experience in patients with migraine. AJNR Am J Neuroradiol. 2017;38(11):2203–9.
79. Dessouky R, Khaleel M, Khalifa DN, Tantawy HI, Chhabra A. Magnetic resonance neurography of the lumbosacral plexus in failed back surgery syndrome. Spine. 2018;43(12):839–47.
80. Zampa V, Cosottini M, Michelassi C, Ortori S, Bruschini L, Bartolozzi C. Value of opposed-phase gradient-echo technique in distinguishing between benign and malignant vertebral lesions. Eur Radiol. 2002;12(7):1811–8.
81. Douis H, Davies AM, Jeys L, Sian P. Chemical shift MRI can aid in the diagnosis of indeterminate skeletal lesions of the spine. Eur Radiol. 2016;26(4):932–40.
82. Suh CH, Yun SJ, Jin W, Park SY, Ryu C-W, Lee SH. Diagnostic performance of in-phase and opposed-phase chemical-shift imaging for differentiating benign and malignant vertebral marrow lesions: a meta-analysis. AJR Am J Roentgenol. 2018;211(4):W188–97.
83. Pezeshk P, Alian A, Chhabra A. Role of chemical shift and Dixon based techniques in musculoskeletal MR imaging. Eur J Radiol. 2017;94:93–100.
84. Eito K, Waka S, Naoko N, Makoto A, Atsuko H. Vertebral neoplastic compression fractures: assessment by dual-phase chemical shift imaging. J Magn Reson Imaging. 2004;20(6):1020–4.
85. Jahng G-H, Li K-L, Ostergaard L, Calamante F. Perfusion magnetic resonance imaging: a comprehensive update on principles and techniques. Korean J Radiol. 2014;15(5):554–77.
86. Ellingson BM, Woodworth DC, Leu K, Salamon N, Holly LT. Spinal cord perfusion MR imaging implicates both ischemia and hypoxia in the pathogenesis of cervical spondylosis. World Neurosurg. 2019;128:e773–81.
87. Li H, Luo H, Liu Y. Paraspinal muscle segmentation based on deep neural network. Sensors. 2019;19(12):2650.
88. Kim DH, MacKinnon T. Artificial intelligence in fracture detection: transfer learning from deep convolutional neural networks. Clin Radiol. 2018;73(5):439–45.
89. Cheng C-T, Ho T-Y, Lee T-Y, Chang C-C, Chou C-C, Chen C-C, et al. Application of a deep learning algorithm for detection and visualization of hip fractures on plain pelvic radiographs. Eur Radiol. 2019;29(10):5469–77.
90. Badgeley MA, Zech JR, Oakden-Rayner L, Glicksberg BS, Liu M, Gale W, et al. Deep learning predicts hip fracture using confounding patient and healthcare variables. NPJ Digit Med. 2019;2:31.
91. Smith SA, Pekar JJ, van Zijl PCM. Advanced MRI strategies for assessing spinal cord injury. Handb Clin Neurol. 2012;109:85–101.

92. Choe AS. Advances in spinal functional magnetic resonance imaging in the healthy and injured spinal cords. Curr Phys Med Rehabil Rep. 2017;5(3):143–50.
93. Klupp E, Cervantes B, Schlaeger S, Inhuber S, Kreuzpointer F, Schwirtz A, et al. Paraspinal muscle DTI metrics predict muscle strength. J Magn Reson Imaging. 2019;50(3):816–23.
94. Fischer MA, Nanz D, Shimakawa A, Schirmer T, Guggenberger R, Chhabra A, et al. Quantification of muscle fat in patients with low back pain: comparison of multi-echo MR imaging with single-voxel MR spectroscopy. Radiology. 2013;266(2):555–63.
95. Brusko GD, Perez-Roman RJ, Tapamo H, Burks SS, Serafini AN, Wang MY. Preoperative SPECT imaging as a tool for surgical planning in patients with axial neck and back pain. Neurosurg Focus. 2019;47(6):E19.
96. Xing D, Zha Y, Yan L, Wang K, Gong W, Lin H. Feasibility of ASL spinal bone marrow perfusion imaging with optimized inversion time. J Magn Reson Imaging. 2015;42(5):1314–20.

Spondyloarthritis

5

Nilofar Syed, Fatemah Ezzati, and Lesley Davila

Introduction

The spondyloarthritides are a collection of disorders characterized by axial or peripheral inflammatory arthritis and associated clinical manifestations. This includes axial spondyloarthritis, psoriatic arthritis, reactive arthritis, inflammatory bowel disease related arthritis, and juvenile spondyloarthritis. These disorders all have overlapping symptoms and similar clinical features.

Classification

The Assessment of Spondyloarthritis International Society (ASAS) developed classification criteria in 2009 categorizing spondyloarthritis into two subcategories: axial spondyloarthritis and peripheral spondyloarthritis [1]. Axial spondyloarthritis (axSpA) is the chronic inflammatory arthritis primarily affecting the axial skeleton. It is important to recognize axSpA as one of many causes of chronic low back pain and the focus of this chapter will be on axial spondyloarthritis.

Patients with axSpA are classified as having either of two subtypes: (1) radiographic axial spondyloarthritis (r-axSpA) or ankylosing spondylitis (AS) and (2) non-radiographic axial spondyloarthritis (nr-axSpA). The basis for the classification as AS or nr-axSpA is the presence or absence of structural changes on radiographs.

N. Syed (✉) · F. Ezzati · L. Davila
Division of Rheumatology, Department of Internal Medicine, UT Southwestern Medical Center, Dallas, TX, USA
e-mail: Nilofar.Syed@UTSouthwestern.edu; Fatemah.Ezzati@UTSouthwestern.edu; Lesley.Davila@UTSouthwestern.edu

© The Author(s), under exclusive license to Springer Nature Switzerland AG 2022

C. E. Noe (ed.), *Multidisciplinary Spine Care*,
https://doi.org/10.1007/978-3-031-04990-3_5

Epidemiology

The prevalence of AS is approximately around 1% of the population in the United States [2]. AS is more common in males compared to females with a ratio of 2–3:1 but among patients with nr-axSpA there is a higher proportion of females and the ratio of males to females is closer to 1:1.

The prevalence of HLA B27 antigen is about 6% in the United States but the prevalence of AS in the HLA-B27-positive population is only about 5 percent [3, 4]. Conversely however, 90% of patients with AS have associated HLA B27 positivity and it is felt to play a role in the pathogenesis. The frequency of HLA-B27 in nr-axSpA may be slightly lower than in AS with an estimated prevalence of about 75–85 percent in nr-axSpA [5].

Clinical Manifestations

Symptoms can sometimes be present for years before a diagnosis is made and thus it is important to recognize the clinical features that distinguish axSpA from non-inflammatory causes of back pain.

In most cases, inflammatory back pain is the initial manifestation for axSpA, and a good history is a critical component of the evaluation. This can be performed with a few simple questions regarding timing of pain, aggravating/alleviating factors and associated stiffness. Inflammatory back pain is usually chronic with a duration longer than 3 months with an onset typically before the age of 45. Patients with inflammatory back pain will often report an insidious onset of symptoms in the lower back or buttock region. Pain can often be present at night waking them up from sleep and patients may also report alternating buttock pain. There is typically improvement with exercise and worsening with periods of inactivity. Associated prolonged morning stiffness is also commonly reported and stiffness usually lasts over 30 minutes.

With progression of the disease, patients may report pain, stiffness and decreased mobility in the cervical or thoracic spine and they may also report chest pain due to involvement of the costovertebral joints (Table 5.1).

Patients may also have associated symptoms of peripheral arthritis which can accompany the symptoms of spinal involvement. Peripheral arthritis is typically oligoarticular and asymmetric affecting larger joints such as hips, knees, or ankles.

Other manifestations include enthesitis which is inflammation where tendons or ligaments insert into bone (entheses) and dactylitis where "sausage" like swelling of

Table 5.1 Inflammatory Back Pain Features

1. Chronic course, lasting >3 months
2. Earlier age of onset, <45 years-old
3. Nocturnal awakening due to pain
4. Alternating buttock pain
5. Improvement with exercise
6. Pain exacerbated with inactivity
7. Morning stiffness lasting >30 minutes

digits can occur, the latter mainly seen in psoriatic arthritis (PsA). Sites of enthesitis can include Achilles tendon or plantar fascia insertion, greater and lesser trochanters, sacroiliac and costovertebral joints.

Extra-articular manifestations include uveitis, psoriasis or inflammatory bowel disease. About one third of patients with axSpA can develop acute anterior uveitis which can be unilateral and associated with pain, photophobia, redness or blurry vision. Inflammatory bowel disease can be seen in approximately 5–10% of patients with axSpA but a higher number of patients may have asymptomatic inflammation in the large intestine [6]. Axial involvement can also be seen in patients with psoriatic arthritis and it can occur alone in about 5% of patients or in conjunction with other areas of arthritis in up to 40% of PsA patients. It is typically asymmetric, affecting one sacroiliac joint. The onset of joint pain can also precede the onset of psoriasis in about 15–20% of patients [7].

Physical Exam

On examination patients may exhibit sacroiliac joint tenderness, decrease in mobility of the spine and signs of inflammation in the peripheral joints or entheses. Certain physical exam techniques are helpful in evaluating for AS. Occiput to wall test evaluates cervical range of motion. With the patient standing with their back against a wall, measurement is made of the distance between the occiput and wall.

Chest expansion may be decreased due to costovertebral inflammation and is measured at the fourth intercostal level with less than 2.5 cm chest expansion considered abnormal.

A Modified Schober test assesses flexion of the lumbar spine. This is performed by making a a horizontal line at the level of the superior iliac spines and a second mark 10 cm above that while the patient is standing. The patient is then asked to bend forward and the difference in length between the two marks is measured. A measurement of 5 cm or more is considered normal [8]. The FABER (or Patrick's) test evaluates for sacroiliac pain. The patient lies supine and places their heel on the opposite knee resulting in the hip being in a flexed, abducted and externally rotated position. The examiner then applies pressure down onto the flexed leg which elicits increased pain in the sacroiliac joint.

Diagnosis

Lab Testing

There are no specific laboratory tests that are diagnostic of axSpA, although two types of tests can contribute to making the diagnosis which include HLAB 27 and acute phase reactants.

A thorough medical history, physical examination should be obtained in patients suspected of axSpA based upon the presence of one or more features of disease. HLA-B27 testing should be considered in patients suspected of having SpA, and

can be useful to increase the confidence of a diagnosis of axSpA in patients in whom plain radiographs or magnetic resonance imaging (MRI) also exhibit abnormalities consistent with axSpA. It is important to keep in mind that a positive test for HLA-B27 alone is not diagnostic for axSpA, and a negative test for HLA-B27 does not exclude the diagnosis.

Acute phase reactants – Acute phase reactants, including the erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP), should be measured in patients suspected of axSpA. Acute phase reactants are one of the 11 SpA features used for determining the diagnosis of axSpA

Elevated CRP levels are observed in almost 40 percent of patients with axSpA, with a higher percentage of patients with elevated levels in the subgroup with AS compared with the nr-axSpA-subpopulation [9]. However, a normal ESR or CRP does not exclude a diagnosis of axSpA or that the disease is in an active state. Other lab tests such as rheumatoid factor and ANA are often negative and not indicated as part of the evaluation.

Imaging Studies

The appropriate use and accurate interpretation of sacroiliac and other joint images are very important in the diagnosis of axSpA. The following approach should be used in patients suspected of axSpA based upon the medical history and examination:

- **Plain radiography** – All patients suspected of axSpA should have an anterior-posterior (AP) plain radiograph of the pelvis to visualize the sacroiliac (SI) joints. Plain radiography of the pelvis continues to be the most widely used imaging technique for diagnostic evaluation. This is frequently sufficient to identify sacroiliitis in chronic cases.

  On imaging in sacroiliitis, the lower anterior part of the sacroiliac joints is mostly affected. The presence of erosions, changes in joint width, or sclerosis strongly supports the diagnosis of axSpA. With advanced disease the sacroiliac joints may be fused (ankylosis). Changes in the spine can lead to loss of lordosis, squaring of the vertebrae and syndesmophytes which is characterized as bony bridging between the vertebrae leading to a "bamboo spine" appearance on radiographs. Patients classified as having nr-axSpA do not have evidence of x-ray findings. However approximately 5–10 percent of patients with nr-axSpA can develop evidence of radiographic involvement within about 2 years and about 20 percent after 5 years [10]. Figure 5.1 shows radiographic changes with sacroiliitis.
- **MRI** – It may take years for abnormalities to be evident on pelvic radiographics and so MRI is useful in detecting abnormalities earlier. MRI of the SI joints is usually indicated only in patients without evidence of sacroiliitis on plain radiographs in whom axSpA is still suspected based on other symptoms and findings to help establish the diagnosis of nr-axSpA. It is important to recognize that positive MRI findings by themselves are not sufficient to make the diagnosis of axSpA in the absence of other features of SpA; thus, an MRI should only be ordered if there is a reasonable degree of suspicion of axSpA.



Fig. 5.1 Frontal pelvic radiographs show widening and erosive changes in the bilateral sacroiliac joints, more prominent on the left, consistent with sacroiliitis. (Image provided by Dr. Parham Pezeshk)

The ESSR Arthritis Subcommittee recommendations for appropriate scanning protocols for MRI of the pelvis in the diagnosis of axSpA include simultaneous evaluation of T1-weighted (T1W) and fat-suppressed MRI sequences (such as short tau inversion recovery (STIR) and T2-weighted fat-suppressed turbo spin echo (T2-FS) sequences) [11].

Findings of sacroiliitis on MRI are demonstrated as bone marrow edema of the sacroiliac joints. Erosions of the sacroiliac joint on T1 weighted sequencing increases sensitivity. Contrast enhancement is not necessary, unless the findings without contrast are uncertain and a high suspicion of axSpA remains. Figure 5.2 shows MRI changes with sacroiliitis.

Imaging of enthesitis When SpA is suspected, ultrasound or MRI may be used to detect peripheral enthesitis, which may support the diagnosis of SpA.

Algorithm

The subsequent diagnostic steps are adapted from the strategy referred to as the 2013 Assessment of Spondyloarthritis International Society (ASAS) modified Berlin algorithm [12]:

A patient with chronic low back pain (at least three months) and onset before the age of 45 should have an anterior-posterior (AP) plain radiograph of the pelvis to examine the sacroiliac joints. A diagnosis of axSpA can be made if the imaging meets criteria for sacroiliitis (at least grade 2 bilaterally or grade 3 unilaterally) and the patient has at least one other SpA-typical parameter.

In patients who are not positive for sacroiliitis by plain radiography of the pelvis, the presence or history of each of 11 features of SpA should be ascertained; a patient with at least 4 of the 11 SpA features can usually be diagnosed with nr-axSpA, but preferably such patients should have positive imaging and/or positive test for HLA-B27; the absence of both of these two findings makes SpA less likely in such patients.

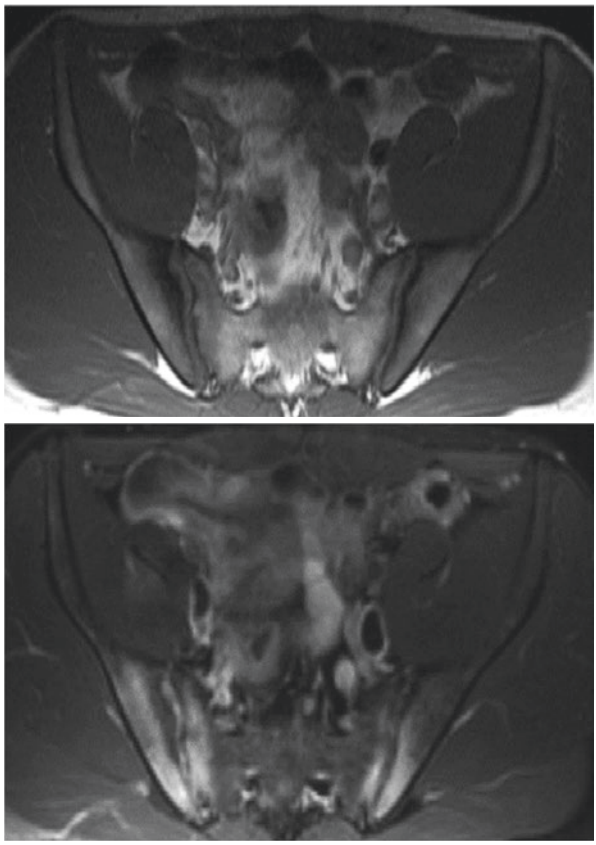


Fig. 5.2 Axial T1 and STIR images in a 24 year-old male with back pain. Mild widening of the right SI joint with adjacent moderate marrow edema representing sacroiliitis. Left SI joint is less severely involved. (Images provided by Dr. Parham Pezeshk)

These 11 features characteristic of SpA are:

- Inflammatory back pain
- Heel pain (enthesitis)
- Dactylitis
- Uveitis
- Positive family history for SpA
- Inflammatory bowel disease
- Alternating buttock pain
- Psoriasis
- Asymmetric arthritis
- Positive response to nonsteroidal anti-inflammatory drugs (NSAIDs)
- Elevated acute phase reactants (ESR or CRP)

For patients with fewer than four SpA features and without radiographic sacroiliitis it is recommended to undergo HLA-B27 testing.

- Patients without radiographic sacroiliitis but with two to three SpA features and positive HLA-B27 testing may generally be diagnosed with nr-axSpA.

- A diagnosis of axSpA is relative unlikely in those with two or three SpA features who are negative for HLA-B27 and lack radiographic sacroiliitis. However, in patients in whom the clinical suspicion of axSpA remains high, an MRI should be obtained to evaluate for evidence of sacroiliitis, which will support the diagnosis of nr-axSpA
- Patients without radiographic sacroiliitis and with no or only one SpA feature, but a positive test for HLA-B27, should be evaluated for sacroiliitis by MRI. An MRI that is positive for sacroiliitis supports the diagnosis of nr-axSpA in such patients but is itself not diagnostic and should be interpreted in the context of the patient's symptoms and other findings.

Differential Diagnosis

Conditions that cause chronic low back and spinal pain may present in a similar fashion to axSpA and should be considered in the differential diagnosis:

1. **Acute or chronic mechanical back pain**:
   Obtaining a good history may help to differentiate this from inflammatory back pain. This can be readily distinguished from axSpA by the absence of inflammatory characteristics in the majority of patients and also lacking associated features of SpA; however, a minority of patients with these conditions may have a pattern of discomfort that is typical of inflammatory back pain.
2. **Diffuse idiopathic skeletal hyperostosis**:
   Patients with diffuse idiopathic skeletal hyperostosis (DISH) may have musculoskeletal pain and stiffness in affected areas, including the neck, back, and sometimes the extremities. Reduced spinal motion, especially in the thoracic spine, is present in all patients in advanced cases. The hallmarks of DISH are radiographic abnormalities. DISH is characterized on imaging by the ossification of paravertebral ligaments and peripheral entheses. Unlike patients with axSpA, the SI joints are usually spared, the apophyseal joints are not ankylosed, exuberant osteophyte formation and flowing calcification occur, at least four contiguous vertebral bodies are typically ossified, and disc height is maintained.
3. **Vertebral compression fracture** – This typically occurs in patients with low bone mass of the spine, especially with osteoporosis or trauma. Patients are typically older than those with SpA, and the pain is usually more acute and more severe than in SpA and is localized to the spine.
4. **Osteitis condensans ilii** –
   Osteitis condensans ilii (OCI) is a self-limiting condition marked by sclerosis of the iliac bone, found either incidentally on imaging in asymptomatic patients or those presenting with lower back pain. Imaging and clinical findings are localized to the sacroiliac joints. It spares the joint space, is not progressive, and most commonly presents in the absence of lab value abnormalities.

Treatment Options

- Treatment of SpA is quite extensive. Goals of treatment include reducing pain and stiffness, improving physical function, mobility and activities of daily living as well as reducing potential joint and spine damage [13]. Treatment can be divided up into both pharmacologic and nonpharmacologic categories and can differ based on the subtype of SpA. It is important that patients are seen by a rheumatologist as many of the pharmacologic options can prevent radiographic progression of disease and slow down pain and disability. Untreated, the disease can lead to irreversible structural damage and disability, causing a great impact on physical and social quality of life of the patients and even on their families [14]. Treatment decisions should be made based on shared decision making between the patient and rheumatologist. Additionally, it is important to screen patients for extra articular manifestations of disease (psoriasis, GI manifestations, ocular disease, etc.) which may influence treatment choices.

Nonpharmacologic Treatment

All patients with SpA should be offered physical therapy, exercise and patient education about their disease in addition to pharmacologic treatment.

Patient education: Patient education should include topics such as maintaining healthy posture and weight, regular exercise, the need for regular office visits to evaluate disease activity and monitor medications. Any patient started on pharmacologic therapy should receive information about specific medications they will be taking and risks associated with that therapy.

- *Physical therapy:* All patients newly diagnosed with axSpA should be encouraged to participate in physical therapy to learn proper posture, strengthening exercises and lifelong habits of stretching to help prevent loss of spinal mobility. Additionally, patients who already have lost mobility of the spine may be able to regain function through individual or group-based classes.
- *Exercise:* Studies suggest that exercise plays an important role in SpA, reducing pain, improving mobility and function as well has having a positive role on mental health. Specific exercise recommendations can be given in physical therapy, but it is important the patient develops a lifelong habit of maintaining spine mobility for best outcomes [15].
- *Role for surgical intervention:* In many patients with longstanding spondyloarthropathy, there is substantial damage to the hip joints causing pain and restriction of movement. These patients may be good candidates for total hip arthroplasty which can reduce pain and restore movement and functionality. Additionally, patients with kyphosis and loss of neck function may be referred to spinal surgeon for options such as laminectomy (in the case of nerve

root compression) or osteotomy and fixation which rarely can be used to fuse the spine in a straight position.

Pharmacologic Treatment

- **Nonsteroidal anti-inflammatory drugs (NSAIDs):** NSAIDs have been a cornerstone of therapy for SpA for decades. Ideally patients should have a trial of one or two full strength daily NSAIDs for treatment of axSpA before moving on to other medication options [16]. Scheduled NSAIDs are preferred over as needed dosing in terms of relieving pain and stiffness and potentially reducing spinal disease. In many patients NSAIDs are the only medication required and the majority have improvement in back pain and stiffness [17].
- **TNF inhibition**: In patients who have inadequate response to NSAIDs, the next choice of therapy is a tumor necrosis factor (TNF) alpha inhibitor for axial disease. These medications include etanercept, adalimumab, infliximab, certolizumab pegol and golimumab (as well as their biosimilars) [18]. TNF inhibition has been shown in clinical trials to reduce the inflammatory component of axSpA including reduction in pain and stiffness, improved spinal mobility, peripheral arthritis, enthesitis, as well as reduction in radiographic progression of disease. These medications are generally well-tolerated although can increase risk of infections and patients need to be screened for tuberculosis prior to initiation [19]. There is no preference of one TNF inhibitor over another in terms of efficacy but in patients with other manifestations including uveitis or IBD, a TNF receptor inhibitor such as etanercept is not recommended due to lack of efficacy. In patients who responded well to a TNF inhibitor but had gradual loss of response over time, switching to an alternative TNF inhibitor is recommended.
- **Anti-IL17 antibody:** Secukinumab and Ixekizumab are anti-IL17A monoclonal antibody medications that are approved for treatment of axial spondyloarthritis. The anti-IL17A monoclonal antibody are shown to reduce symptoms as well as radiographic progression of disease. In patients with no response to a trial of TNF inhibitor for at least 3 months, switching to secukinumab or ixekizumab is recommended [16]. As compared to the TNF inhibitors, the anti-IL17A monoclonal antibody medications are not effective for Crohn's disease and are typically avoided in these patients.
- **JAK inhibitor:** Tofacinitib which is a JAK inhibitor, is also a treatment option approved for axSpA after failure of TNF inhibitors but it is associated with increased risk of cardiovascular disease, cancer, and thrombosis [20].
- **Disease modifying anti-rheumatic drugs (DMARDs):** DMARDs have some benefit in the treatment of peripheral arthritis but there is not adequate benefit for axial involvement. In patients who continue to have peripheral joint involvement the initiation of sulfasalazine or methotrexate can be considered.

- **Systemic glucocorticoids:** There is no role for the use of systemic glucocorticoids for treatment of axSpA, although they can be of use for other manifestations of disease such as peripheral arthritis, eye disease or GI disease.

References

1. Rudwaleit M, van der Heijde D, Landewé R, Listing J, Akkoc N, Brandt J, Braun J, Chou CT, Collantes-Estevez E, Dougados M, Huang F, Gu J, Khan MA, Kirazli Y, Maksymowych WP, Mielants H, Sørensen IJ, Ozgocmen S, Roussou E, Valle-Oñate R, Weber U, Wei J, Sieper J. The development of assessment of SpondyloArthritis international Society classification criteria for axial spondyloarthritis (part II): validation and final selection. Ann Rheum Dis. 2009;68(6):777–83.
2. Reveille, et al. Prevalence of axial spondyloarthritis in the United States: estimates from a cross-sectional survey. Arthritis Care Res. 2012;64:905–10.
3. Reveille JD, Hirsch R, Dillon CF, Carroll MD, Weisman MH. The prevalence of HLA-B27 in the US: data from the US National Health and Nutrition Examination Survey, 2009. Arthritis Rheum. 2012;64(5):1407–11.
4. Braun B M, Remlinger G, Eggens U, Rudwaleit M, Distler A, Sieper J. Prevalence of spondylarthropathies in HLA-B27 positive and negative blood donors. Arthritis Rheum. 1998;41(1):58.
5. Kiltz U, Baraliakos X, Karakostas P, Igelmann M, Kalthoff L, Klink C, Krause D, Schmitz-Bortz E, Flörecke M, Bollow M, Braun J. Do patients with non-radiographic axial spondylarthritis differ from patients with ankylosing spondylitis? Arthritis Care Res (Hoboken). 2012;64(9):1415.
6. Van Praet L, Van den Bosch FE, Jacques P, Carron P, Jans L, Colman R, Glorieus E, Peeters H, Mielants H, De Vos M, Cuvelier C, Elewaut D. Microscopic gut inflammation in axial spondyloarthritis: a multiparametric predictive model. Ann Rheum Dis. 2013;72(3):414–7.
7. Klippel JH, et al. Primer on the rheumatic diseases. 13th ed. New York: Springer; 2008.
8. Landewe R, van Tubergen A. Clinical tools to assess and monitor Spondyloarthritis. Curr Rheumatol Rep. 2015;17:47.
9. Rudwaleit M, Haibel H, Baraliakos X, Listing J, Märker-Hermann E, Zeidler H, Braun J, Sieper. The early disease stage in axial spondylarthritis: results from the German Spondyloarthritis Inception Cohort. Arthritis Rheum. 2009;60(3):717.
10. Constantino F, Zeboulon N, Said-Nahal R, Breban M. Radiographic sacroiliitis develops predictably over time in a cohort of familial spondyloarthritis followed longitudinally. Rheumatology (Oxford). 2017;56:811.
11. Sudoł-Szopińska I, et al. Recommendations of the ESSR Arthritis Subcommittee for the use of magnetic resonance imaging in musculoskeletal rheumatic disease. Semin Musculoskelet Radiol. 2015;19:396–411.
12. van den Berg R, de Hooge M, Rudwaleit M, et al. ASAS modification of the Berlin algorithm for diagnosing axial spondyloarthritis: results from the SPondyloArthritis Caught Early (SPACE)-cohort and from the Assessment of SpondyloArthritis international Society (ASAS)-cohort. Ann Rheum Dis. 2013;72:1646–53.
13. Van der Heijde D, Ramiro S, Landewé R, Baraliakos X, Van den Bosch F, Sepriano A, Regel A, Ciurea A, Dagfinrud H, Dougados M, van Gaalen F, Géher P, van der Horst-Bruinsma I, Inman RD, Jongkees M, Kiltz U, Kvien TK, Machado PM, Marzo-Ortega H, Molto A, Navarro-Compàn V, Ozgocmen S, Pimentel-Santos FM, Reveille J, Rudwaleit M, Sieper J, Sampaio-Barros P, Wiek D, Braun J. 2016 update of the ASAS-EULAR management recommendations for axial spondyloarthritis. Ann Rheum Dis. 2017;76(6):978–91.

14. Singh JA, Strand V. Spondyloarthritis is associated with poor function and physical health-related quality of life. J Rheumatol. 2009;36(5):1012–20.
15. Reimold AM, Chandran V. Nonpharmacologic therapies in spondyloarthritis. Best Pract Res Clin Rheumatol. 2014;28(5):779–92.
16. Ward MM, Deodhar A, Gensler LS, Dubreuil M, Yu D, Khan MA, Haroon N, Borenstein D, Wang R, Biehl A, Fang MA, Louie G, Majithia V, Ng B, Bigham R, Pianin M, Shah AA, Sullivan N, Turgunbaev M, Oristaglio J, Turner A, Maksymowych WP, Caplan L. 2019 update of the American College of Rheumatology/Spondylitis Association of America/Spondyloarthritis research and treatment network recommendations for the treatment of ankylosing spondylitis and nonradiographic axial Spondyloarthritis. Arthritis Care Res (Hoboken). 2019;71(10):1285–99.
17. Song IH, Poddubnyy DA, Rudwaleit M, Sieper J. Benefits and risks of ankylosing spondylitis treatment with nonsteroidal antiinflammatory drugs. Arthritis Rheum. 2008;58(4):929–38.
18. Callhoff J, Sieper J, Weiß A, Zink A, Listing J. Efficacy of TNFα blockers in patients with ankylosing spondylitis and non-radiographic axial spondyloarthritis: a meta-analysis. Ann Rheum Dis. 2015;74(6):1241–8.
19. Koo S, Marty FM, Baden LR. Infectious complications associated with immunomodulating biologic agents. Infect Dis Clin N Am. 2010;24(2):285–306.
20. Deodhar A, Sliwinska-Stanczyk P, Xu H, Baraliakos X, Gensler L, Fleishaker D, Wang L, Wu J, Menon S, Wang C, Dina O, Fallon L, Kanik K, van der Heijde D. Tofacitinib for the treatment of adult patients with Ankylosing spondylitis: primary analysis of a phase 3, randomized, double-blind, placebo-controlled study. Arthritis Rheumatol. 2020;72(suppl 10):1004–14.

Soft Tissue Pain

6

Robert S. Ackerman, Patrick B. Ahern, Biral T. Patel, and Carl E. Noe

Introduction

Soft tissue is an important source of pain located near the spine. Many patients diagnosed with non-specific low back pain for failed back syndrome often have soft tissue pain syndromes that may be successfully treated. Soft tissue pain may be a primary soft tissue disorder or may be secondary to a spinal condition and often manifest due to mechanical disorders or systemic inflammation [1]. Kuslich studied pain responses to stimulation of different back tissues during lumbar spine surgery performed with local anesthesia, examining the heterogeneity of pain impulses and stimuli and how they may present symptomatically with some congruency [2]. Compressed nerve roots were sensitive to stimulation in 99% of patients followed by 74% with stimulation of the central annulus, 71% with the central lateral annulus and 61% with the vertebral end plate. In addition, 41% of patients were sensitive to paravertebral muscle stimulation, 30% to facet capsule stimulation, 25% to supraspinous ligament stimulation, and 17% with lumbar fascia stimulation [2]. Spine structures are more sensitive to stimulation, but soft tissues are a significant potential source of back pain.

The sacroiliac joint was examined as a potential source of low back pain, noting its role in regulating reflex muscle activation and locomotion, controlling body posture and trunk mobility. Gluteus maximus and quadratus lumborum muscle activation was noted after stimulation of the ventral sacroiliac joint in pigs; stimulation of the joint capsule produced activation of the multifidus muscles [3]. Thus, muscle spasms can result from a primary pain source in the spine. Patients with discogenic, ligamentous, or facet pain often have associated muscle spasm pain, for which treatment of can significantly reduce overall pain [4].

R. S. Ackerman · P. B. Ahern · B. T. Patel (✉) · C. E. Noe
Department of Anesthesiology and Pain Management, University of Texas Southwestern Medical Center, Dallas, TX, USA
e-mail: rackerman@usf.edu; Patrick.Ahern@PHHS.Org; biral.patel@utsouthwestern.edu

© The Author(s), under exclusive license to Springer Nature Switzerland AG 2022

C. E. Noe (ed.), *Multidisciplinary Spine Care*,
https://doi.org/10.1007/978-3-031-04990-3_6

Common soft tissue pain problems consist of myofascial pain, bursitis, tendinitis, enthesitis, among others [1]. Furthermore, soft tissue pain may present as referred pain from distant sources. A thorough and wide differential diagnosis of acute soft tissue pain is needed to prioritize and create a diagnostic impression and hypothesize an appropriate treatment strategy.

Differential Diagnosis of Acute Pain

Soft tissue pain disorders can occur in all regions of the body. In the head and neck, causes include occipital neuralgia, torticollis, and thoracic outlet syndrome. In the chest wall, costochondritis is notable [1]. In the upper extremities, diagnoses include rotator cuff tendinopathy and tear, subacromial bursitis, lateral and medial epicondylitis, De Quervain disease, and Dupuytren contracture; in the lower extremities, consider trochanteric and ischial bursitis, patellofemoral syndrome, Achilles tendinopathy, and plantar fasciitis [1]. Weiner's textbook Differential Diagnosis for Acute Pain Syndromes comprehensives describes common, uncommon, and rare causes for acute pain problems [5].

Posterior Neck Pain

Common causes of acute posterior neck pain include meningitis, myositis, occipital migraine, cervical disc pathologies, and rheumatoid arthritis. Uncommon conditions include myofascial pain, torticollis, osteomyelitis, and ankylosing spondylitis. Rare diagnoses include epidural abscess and hematoma, pathologic fracture, retropharyngeal and chest wall abscesses, vertebral artery dissection or pseudoaneurysm, and occipital neuralgia [5].

Interscapular and Scapular Pain

Common causes of acute interscapular and scapular pain include myofascial pain, trauma, and vertebral compression fracture. Uncommon conditions include myocardial infarction, common bile duct stone, herpes zoster, and osteomyelitis. Rare diagnoses include transverse myelitis, pleuritis, and pyelonephritis [5].

Midline Thoracolumbar Pain

Common causes of acute midline thoracolumbar pain include vertebral compression fractures, myofascial pain, pancreatitis, and soft tissue injuries. Uncommon conditions include duodenal ulcers, herpes zoster, and pathologic fracture due to cancer. Rare diagnoses include epidural abscess and pathologic fracture due to osteomyelitis [5].

Lumbosacral Pain

Common causes of acute lumbosacral pain include spondylosis and spondylolisthesis, facet syndrome, osteoporotic fractures, myofascial pain, diverticulitis, pelvic inflammatory disease, lumbosacral strain, and disc degeneration. Uncommon conditions include sacroiliitis, Paget's disease, hyperextension injury, and intraspinal synovial cyst. Rare diagnoses include cauda equina syndrome, conus medullaris infarction, epidural abscess, and hematoma [5].

Unilateral Buttock Pain

Common causes of acute unilateral buttock pain include facet syndrome, disc degeneration, myofascial pain, trochanteric bursitis, and furuncle. Uncommon conditions include sacroiliitis due to Reiter's disease, ischial bursitis, hamstring strain, piriformis syndrome, and anorectal abscess. Rare diagnoses include infectious sacroiliitis and epidural abscess [5].

Bilateral Buttocks Pain

Common causes of acute bilateral buttocks pain include facet syndrome, trauma, disc conditions, furuncles, and sacroillitis due to Reiter's disease. Uncommon conditions include horseshoe abscess, spondylolisthesis, and ischial bursitis. Rare diagnoses include conus medullaris infarction and epidural abscess and hematoma [5].

Myofascial Pain

The Simons and Travell criteria of myofascial pain are as follows: a localized tender spot within a taut band of muscle; an expected pattern of referred pain; tenderness to palpation; reduced range of motion; and a local twitch in response to needle insertion into or snapping of the myofascial trigger point [6]. This widely accepted description includes both acute and chronic durations and single or multiple trigger points. As many as one-third of patients with musculoskeletal disorders have a component of myofascial pain.

Etiologies

Simons' hypothesis includes many myofascial, biomechanical, and central nervous factors that could account for the clinical characteristics. Abnormal pre- and postsynaptic depolarizations, dysfunction in acetylcholinesterase activity, acetylcholine release, or receptor activity, as well as abnormalities in muscle spindle function and the motor end plate are all theorized to contribute [7]. The treatment of myofascial

pain includes both pharmacologic and non-pharmacologic strategies. Determining an underlying perpetuating cause is important. Abnormal posture, muscle imbalances, hormonal and nutritional problems (such as Vitamin D deficiency) are frequently involved.

Treatments

Medication classes (and common medications) used to treat myofascial pain include muscle relaxants (cyclobenzaprine, tizanidine), sedatives and hypnotics (clonazepam, alprazolam, diazepam), antidepressants (amitriptyline, nortriptyline, fluoxetine, duloxetine, venlafaxine), anticonvulsants (gabapentin, pregabalin), topical analgesics (lidocaine, menthol, and diclofenac patches), and anti-inflammatory medications (ibuprofen, diclofenac, aspirin, acetaminophen) [7]. Other treatment strategies include transcutaneous electrical nerve stimulation (TENS), ultrasound therapy, laser therapy, magnetic stimulation, and needle-based interventions such as acupuncture, dry needling, and trigger point injections [7, 8].

Exercise triggers the release of beta-endorphins from the pituitary gland and hypothalamus, enabling the peripheral and central analgesic effects from the mu-opioid receptor [9]. Acupuncture, like dry needling, works to inhibit pain perception via an increase in central nervous opioid peptides [8]. Ultrasound therapy works to heat the effected tissue, causing a prolongation of sarcomere contraction [8].

Sharan developed a continuum of care for the treatment of myofascial low back pain [9]:

- Phase I: Severe Discomfort. Goal: pain relief. Treatments: ultrasound, muscle therapy, sensory desensitization.
- Phase II: Moderate Discomfort. Goal: flexibility restoration. Treatments: yoga, self-stretching exercises, spine mobilization.
- Phase III: Mild Discomfort. Goal: strengthening and conditioning. Treatments: aquatic therapy, body mechanics, EMG biofeedback, progressive strengthening exercises.
- Phase IV: Maintenance. Goal: functional restoration. Treatments: pilates, Tai Chi, functional and vocational rehabilitation, strength training.

Trigger point injections should be performed with short needles, especially in the thoracic region due to the risk of pneumothorax. This is one of the most common reasons for lawsuits related to injections. Large patients should be injected using imaging, if necessary. The target for a trigger point injection is not the taut band, but the trigger point itself. The trigger point must be localized accurately and may be marked with a pen or localized with the index finger to elicit a “jump sign.” The trigger point is injected with local anesthetic to anesthetize and relax the muscle [8, 9]. Independent exercise is the goal for patients with myofascial pain and injections should not be repeated endlessly if patients are not progressing toward a home

exercise program. A study of the occurrence and inter-rater reliability of myofascial trigger points in the quadratus lumborum and gluteus medius muscles suggested the clinical usefulness is greatest when localized tenderness, the "jump sign," or patient's recognition of pain are used as criteria for the presence of trigger points [10].

Fibromyalgia

Fibromyalgia syndrome (FMS), with an incidence of 2% in the United States, is characterized by persistent and widespread musculosketal pain of non-inflammatory origin. Originally FMS was tough to define due to many concomitant symptoms and related comorbidities, such as fatigue, depression, memory issues, and insomnia. The American College of Rheumatology definitions of the syndrome have been continuously refined, with Galvez-Sanchez suggesting new diagnostic criteria for fibromyalgia syndrome [11]:

- Core Diagnostic Criteria. The presence of pain in six or more body sites from nine possible locations, in addition to fatigue and sleep disturbance.
- Common Features. Includes musculoskeletal stiffness, environmental sensitivity, dyscognition (forgetfulness, slow thinking), and tenderness.
- Common Medical and Psychiatric Comorbidities. Chronic headaches, depression and anxiety disorders, irritable bowel syndrome, chronic fatigue syndrome, chronic pelvic pain, restless leg syndrome, etc.
- Neurobiological, Psychosocial, and Functional Consequences. Includes medical cost of fibromyalgia syndrome, morbidity, and mortality.
- Putative Neurobiological and Psychosocial Mechanisms, Risk Factors, and Protective Factors. Focuses on comorbidities and pathophysiological components.

Fibromyalgia is considered a nociplastic pain syndrome. Multiple theories have emerged to explain this disorder. The immune system may be involved triggering the onset of fibromyalgia, as well as the human microbiota. Skin and thalamic cells' release of neuro-inflammatory mediators may influence microglia. An association between the severity of symptoms and oxidative stress may help explain the role of genetics [12].

Several medications have been studied, with memantine, tapentadol, and duloxetine described as being most useful. Further study into mirogabalin, cannabis, and the glutamate co-agonist NYX2915 are being studied [12, 13]. In the United States, pregabalin, duloxetine, and milnacipran are approved by the Food and Drug Administration for the treatment of FMS. Complementary therapies, such as TENS, ozone, stress reduction, attachment-based compassion therapy, guided meditation, hyperbaric oxygen, and biofeedback, are also being explored [12]. Interdisciplinary treatment – including psychological, physiotherapeutical, educational, and medical interventions, have been shown to improve the health-related quality of life in patients with fibromyalgia [14].

Cervical Whiplash

For patients who experienced soft tissue neck injuries from motor vehicle accidents, positive correlations existed between poor outcomes and risk factors such as numbness or pain in an upper extremity, restricted motion at one level on cervical flexion/extension plain films, reversal of cervical lordosis on lateral plain films, greater than 3 month use of cervical collars, and recurrent need for physical therapy after the initial course [15]. While the majority (57%) of patients recovered in this study, several developed degenerative changes after the injury. Other studies have found paresthesia, thoracolumbar back pain, and the presence of multiple symptoms to be predictive of continuing symptoms and symptom severity after soft tissue injury of the cervical spine [16]. Examining cervical spine radiographs, degenerative changes were found in 68% of patients, and 87% of those with symptoms. 20% of patients with normal X-rays had symptoms [16].

The Quebec Task Form team classified whiplash injuries by severity of clinical symptoms from Whiplash-Associated Disorder (WAD) 1 to WAD 5 [17]:

- WAD 1: No neck discomfort. No abnormal signs.
- WAD 2: Neck pain, stiffness, or only tenderness. No abnormal signs.
- WAD 3: Neck symptoms. Musculoskeletal signs.
- WAD 4: Neck symptoms. Neurological signs.
- WAD 5: Neck symptoms. Fractures or dislocations.

Experimental cervical trauma studies have shown injury to the deepest posterior cervical muscles, in particular the musculus obliquus [18]. Hyperextension injuries can damage the anterior longitudinal ligament and facet capsule ligaments. Hyperflexion injuries damage the ligamentum flavum and interspinous ligaments. Whiplash injuries can tear anterior longitudinal ligaments and cause anterior disk detachments that are seen on MRI [18]. Compression injury of facet joints can also occur. Rear end injuries can damage cervical facet capsular ligaments. The transverse ligament and posterior atlanto-occipital membrane are more commonly injured in frontal motor vehicle accidents compared to rear end accidents. Lateral displacement injuries can tear the transverse atlantal ligament and produce atlanto-axial instability. If the distance between the dens and the anterior atlas exceeds 3 mm, the transverse ligament and the alar ligament may be ruptured [18].

Injuries of the tectorial membrane, alar ligaments and transverse ligaments can be imaged following high energy accidents. While the normal tectorial membrane and transverse ligament are routinely seen, the normal alar ligaments may be difficult to visualize. These ligamentous structures are important stabilizers in the cranio-cervical junction and occipito-cervical fusion is necessary in cases of instability and potential myelopathy [19].

However, for stable soft tissue whiplash injuries, muscle energy technique has been used to treat upper trapezius and levator scapulae muscles in patients with soft tissue neck pain. The muscle energy technique involves static stretching of the

muscles by the therapist placing one hand on the patients shoulder for stabilization and using the other hand to move the head to stretch the muscles [20].

Cervical Dystonia and Torticollis

The use of botulinum neurotoxin type A (BoNT/A) in conjunction with physical therapy is efficacious in obviating the need for more invasive treatment modalities in treating congenital torticollis [21]. BoNT/A has also demonstrated sustained significant clinical benefit to patients suffering from cervical dystonia [22]. Use of botulinum neurotoxin reduces neck pain by decreasing excessive muscle spindle activity, inhibiting retrograde neuronal flow to the central nervous system, inhibiting release of neuropeptides by nociceptors and blocking release of acetylcholine at nerve endings without interfering with nerve conduction [23]. Despite its role in improving outcomes in cervical dystonia and congenital torticollis, current evidence has demonstrated no similar benefit in treating patients with chronic neck pain [24].

Notalgia Paresthetica

Notalgia paresthetica is a syndrome of refractory neuropathic pain and pruritus commonly located unilaterally in the infrascapular region. Interestingly, there is growing evidence of its association with degenerative or traumatic cervicothoracic disc disease. Initial work-up of notalgia paresthetica associated with cervical disease may require cervical spinal imaging. Treatment options range from conservative modalities (e.g. physical therapy, cervical muscle strengthening, spinal manipulation, etc.) to pharmacotherapies (NSAIDs, muscle relaxants) to surgical interventions [25].

Paraspinal Muscles

The interspinalis and lumbar multifidus muscles, core paraspinal muscles, are thought to be important sources of soft tissue-related low back pain. Theories regarding the nature of injury to these muscles include muscle avulsion, muscle atrophy or prolonged neurological inhibition of paraspinal muscles [26]. Lumbar multifidus muscles are particularly key in providing muscular stabilization of the neutral zone range of motion in the lower back. Dysfunction of the lumbar multifidus muscles – a consequence of inhibition of spinal pain – is strongly associated with low back pain and, in most cases, persists even after resolution of pain [27]. Imaging may show atrophy of paraspinal muscles. However because of variability of study quality and approaches, the relationship between atrophy and low back pain remains unclear [28].

The vast majority of acute episodes of low back pain resolve within 2–4 weeks, however recurrence rates following initial insult range between 60% and 86%. The large recurrence rate is related to instability at the spinal segmental levels, further illustrating the importance of well-functioning paraspinal muscles in improving outcomes. Evidence shows that patients receiving specific lumbar stabilization therapy in addition to medical management and resumption of normal daily activity experience fewer recurrences of low back pain compared to those who receive only medicant management and resumption of normal activity [29].

Quadratus Lumborum

The quadratus lumborum muscle, an important lateral stabilizer of the spine, originates near the iliac crest and attaches to the transverse processes of the lumbar spine through the thoracolumbar fascia. Injury to the muscle is associated with stressful twisting motions, such as with golfing, that overload the muscle. Patients present with flank pain and tenderness of the buttock, lower back and lateral hip. They may cite coughing or sneezing as occurrences where their pain is exacerbated. Initial treatment includes myofascial therapy that aims to restore quadratus lumborum muscle function in addition to joint manipulation of related dysfunctional areas [4].

Thoracolumbar Fascia

The thoracolumbar fascia itself represents a source of idiopathic pain generation in those with lumbar disorders. The mechanisms theorized to elicit the low back pain include: nociceptive input from other tissues that innervate the same spinal segment, microinjuries that cause irritation to nociceptive nerve endings, and restructured tissue that alters proprioceptive signaling following injury, immobility or chronic overload, thereby lowering the threshold for pain. Histologic evidence of morphological changes in the lumbodorsal fascia exists in those with chronic low back pain [30].

Greater Trochanteric Pain Syndrome

Greater trochanteric pain syndrome – a regional pain syndrome – consists of the following diagnoses: trochanteric bursitis (the most common cause), gluteal muscle tendinopathy and external coxa saltans. Traditionally, trochanteric bursitis was considered the central source of soft tissue-related lateral hip pain. However, advances in imaging studies have demonstrated that many patients diagnosed with trochanteric bursitis have concomitant hip abductor tendinopathy [31].

The clinical features of greater trochanteric pain syndrome include chronic intermittent pain over the buttock region and lateral thigh. Patients with trochanteric

bursitis tend to note particular difficulty sleeping on the affected side, while those with gluteal muscle tendinopathy will present with hip abductor weakness [32]. Greater trochanteric pain syndrome's "pseudoradiculopathy" does not extend distal to the proximal tibia (at the insertion of the iliotibial tract at Gerdy's tubercle). The lateral hip pain is exacerbated with activity and can be reproduced through palpation of the greater trochanter, termed the "jump sign". A positive Trendelenburg sign is the most sensitive physical finding for posterior gluteus medius tendinopathy [4].

Repetitive friction between the greater trochanter and iliotibial (IT) band causes microtrauma of the gluteal tendons that insert into the greater trochanter. Blunt trauma and idiopathic sources can cause similar damage. The etiology of GTPS is due to this damage that results in inflammation of adjacent bursae, increased IT band tension and gluteal tendon degeneration [32]. Moreover, habitual preference of the tensor fascia lata in performing hip abduction movements results in posterior gluteus medius muscle disuse weakness and atrophy, further increasing the risk for injury in the future [4].

While imaging may be helpful in ruling out alternate causes of hip pain, diagnosis is based on history and physical. The management of greater trochanteric pain syndrome should be step-wise in accordance with clinical response. Initial treatment options for trochanteric bursitis include NSAIDs, physiotherapy and corticosteroid injections. Further treatment options include shockwave therapy and platelet-rich plasma injections [32]. Rarely used, surgical intervention is reserved for treatment-refractory cases [33]. The initial management of gluteal tendinopathy includes physical therapy focused on strengthening the posterior gluteus medius, with options such as cold pack, ultrasound and iontophoresis, and posture changes aiming to decrease stretch on the muscle. Bursal or trigger point injection may be pursued as an adjunct to better facilitate physical therapy. If tendon rupture is discovered on imaging, surgical repair or hip sica bracing could be indicated [4].

Gluteal Bursitis

Gluteal bursitis presents with pain while walking or going up stairs and is localized to the buttock region with or without radiation to the trochanteric region, posterolateral thigh and calf. It is produced with passive or resisted maneuvers of external rotation and/or abduction. These movements elicit pain secondary to compression of hip bursae [4]. In contrast to the traditional idea of a single "trochanteric bursa" lying over the lateral aspect of the greater trochanter, cadaveric studies suggest the presence of on average 6 bursae per hip and buttock region [34]. Injection of local anesthetic and/or corticosteroid to the site of maximum tenderness serves as a source of both diagnostic and therapeutic utility [4, 34]. Given the highly variable location and quantity of buttock and hip bursae, an improved ability to differentiate between bursa would be advantageous for limiting diagnosis and refractory nature of gluteal bursitis [34].

Ischial Bursitis

Ischial bursitis presents as a source of gluteal or upper posterior thigh pain following periods of prolonged sitting or exercise. The ischial bursa is located between the ischial tuberosity and the gluteus maximus and serves to limit the frictional forces between the two. However prolonged activities that put pressure on the ischium cause an inflammatory reaction resulting in swelling and tenderness. The condition will often mimic the symptoms of sciatica or hamstring tendonitis. Ischial bursitis that is refractory to rest, lifestyle modifications and NSAIDs may require a step-up in therapy with injections of mixed steroid and local anesthetic [35].

Neuropathic Back Pain

Neuropathic back pain, one of the most common chronic pain conditions worldwide, describes pain from injury or disease that affects nerve roots innervating the spine and lower limbs in addition to the invasive innervation of damaged lumbar discs. While noted for its wide variability in presentation, features of neuropathic back pain commonly include pain arising without stimulus, allodynia and hyperalgesia. Current guidelines advise on how to best manage the nociceptive and behavioral components of chronic low back pain, however modalities specifically targeting the neuropathic component is limited. Conflicting evidence exists on the efficacy of antidepressants and anticonvulsants in the treatment of neuropathic back pain. Topical capsaicin and lidocaine are attractive options given that their use has achieved significant reductions in pain scores with limited adverse effects [36]. Enteral tapentadol, a dual μ-opioid receptor agonist and noradrenaline reuptake inhibitor, demonstrates significant reductions in quantity and duration of neuropathic low back pain "attacks", however has an adverse effect incidence that is less well-tolerated [37].

Shingles

Herpes zoster and postherpetic neuralgia is most commonly localized to thoracic- or cranial-associated spinal ganglions. Reactivation of the varicella zoster virus in the L5 dorsal root ganglion is rare, however should be considered part of one's differential diagnosis for lumbosciatic pain with nonspecific discovertebral changes on MRI. Skin changes and blistering following the prodromal pain and paresthesias typically allow for a quick diagnosis [38]. However in the setting of absent skin manifestations with zoster sine herpete, an atypical herpes zoster presentation, the possibility of herpes zoster must still be considered [39]. Prolonging diagnosis increases the risk of postherpetic neuralgia and postherpetic neuropathies - which recover in only 50% of cases - in addition to more damaging and fatal complications [38]. Treatment consists of initiation of an antiviral agent (acyclovir or valacyclovir) as soon as the diagnosis is made, in combination with analgesic agents. Management

of postherpetic neuralgia involves NSAIDs, gabapentinoids, opioids, corticosteroids and tricyclic antidepressants [39]. Shingles prevention via vaccination is currently recommended in adults greater than 50 years old by the United States CDC [40].

Nerve Entrapment

The differential diagnosis for chronic localized back pain should include entrapment of cutaneous branches of the posterior rami of thoracic spinal nerves. Analogous to localized neuropathic anterior abdominal pain and its associated diagnosis of anterior cutaneous nerve entrapment syndrome (ACNES), posterior cutaneous nerve entrapment syndrome (PCNES) is a relatively novel concept associated with neuropathic pain accompanied by sensations of hyperalgesia and allodynia along the distribution of the nerve. These symptoms are related to irritation of the cutaneous branches by the entrapment. Results from a relatively small sample size suggest a step-up diagnostic and treatment regimen of local anesthetic and corticosteroid injections and neurectomy of lateral branch of the posterior primary ramus leads to long-term pain relief [41, 42].

Pain that is reproducible or aggravated by palpation or movement around the lumbar region and buttocks is also suggestive of superior or middle cluneal nerve entrapment. These cutaneous nerves, which are purely sensory, can be entrapped near the iliac crest and produce numbness and radiating pain when their trigger point is compressed. Due to small size of the nerves, imaging modalities such as CT and MRI are not very informative. Thus when suspected, diagnosis is likely with positive Tinel sign upon trigger point compression followed by symptomatic relief with cluneal nerve block. Patients with pain refractory to conservative treatment and nerve blockage may be candidates for surgical release or percutaneous neurolysis [43].

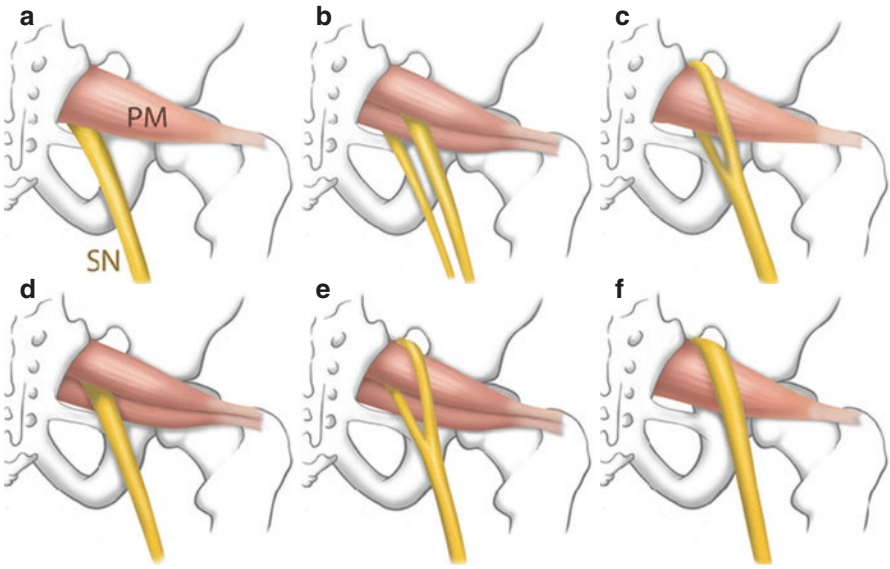
Sciatic nerve entrapment can present with symptoms radiating anywhere in the lower extremity along the sciatic nerve distribution. However pain located specifically in the buttock is commonly a predictor of sciatic nerve entrapment. Various physical exam maneuvers are highly sensitive and specific for sciatic nerve entrapment. However other diagnostic modalities such as MRI, ultrasound, EMG, nerve conduction velocity tests and anesthetic injections may improve diagnostic confidence. Beyond conservative management, more advanced treatment options include local injections, neurolysis and neurectomy [44].

Piriformis Syndrome

Piriformis syndrome is a clinical condition caused by sciatic nerve entrapment that presents with reports of pain in the buttock region. It is commonly associated with shooting, burning or aching discomfort along the back of the leg that is exacerbated by hip movement or prolonged sitting. Additional complaints include numbness in the buttock region and tingling along the distribution of the sciatic nerve [45].

The piriformis muscle originates anterior to the S2 – S4 vertebrae and superior margin of the greater sciatic foramen and inserts onto the greater trochanter of the femur. Entrapment of the sciatic nerve occurs either anterior to the piriformis muscle or posterior to the gemelli-obturator internus complex around the level of the ischial tuberosity [45]. Due to their adjacent proximity, sciatic nerve irritation occurs following periods of piriformis muscle overuse, stress or inflammation most commonly related to chronically poor body mechanics or an acute injury with forceful internal hip rotation. A full list of causes includes [45, 46]:

- Prolonged periods of sitting
- Piriformis muscle hypertrophy
- Trauma to the hip or buttock region
- Anatomic anomalies
  - Bipartite piriformis muscle
  - Sciatic nerve course variations with the most common variation being the common fibular nerve branching superior through the piriformis muscle. Notably, as much as 75% of the population have variation of connection between the two branching nerves – the common fibular nerve and the tibial nerve [47]. (See the variations in image)
  - Tumor invasion
  - Inferior gluteal artery aneurysm
  - Abscess formation
- Myofascial trigger points
- Secondary to laminectomy
- Klippel-Trenaunay syndrome
- Myositis ossificans of the piriformis muscle
- Femoral nailing [46]



The diagnosis of piriformis syndrome, one of exclusion, is based on history and clinical presentation [47]. Differential diagnosis includes facet arthropathy, lumbar canal stenosis, disc inflammation, herniated disc, lumbar muscle strain, iliac vein thrombosis, renal stones and pelvic causes. Diagnostic imaging modalities and EMG may be helpful in excluding other conditions, while therapeutic modalities that will be discussed below can also serve a diagnostic purpose [45, 46].

Like most causes of soft tissue pain, piriformis syndrome typically responds to conservative treatment. Cases refractory to conservative treatment often require interventional modalities. Piriformis muscle injections utilizing landmark-based technique, with the concomitant use of ultrasound, CT, nerve stimulation or electromyography, are common next-step modalities. The injection solution is composed of either botulinum toxin or local anesthetic with or without long-acting corticosteroid [46]. There is evidence showing no additional benefit with the inclusion of corticosteroid to the injectate despite its frequent use [48]. Surgical intervention is historically reserved for severe and intractable cases that is refractory to conservative and less invasive interventional modality, however the introduction of botulinum toxin injections has limited to need to pursue surgery [45, 46].

Referred Pain

Referred pain – back pain referred from a non-spinal source – accounts for approximately 2% of chronic low back pain and is commonly mistaken for soft tissue pain. The causes of referred back pain include gastrointestinal disease (pancreatitis, cholecystitis, penetrating ulcer, colonic diverticulitis), renal disease (nephrolithiasis, pyelonephritis, perinephric abscess), diseases of pelvic organs (prostatitis, endometriosis, chronic pelvic inflammatory disease), pregnancy and aortic aneurysm [49]. Given its innervation from the sciatic, femoral and obturator nerves, the hip joint has been identified as a source of potential pain referral. Studies suggest symptomatic hip joints refer pain to the buttock in more than 70% of cases [50]. Lower thoracic vertebral fractures can also serve as a source of referred lumbosacral pain [51].

Treatment Guidelines

Given the lack of specific treatment recommendations for the wide range of causes of low back pain, current guidelines have pivoted toward grouping treatment modalities based on chronic vs acute and subacute pain.

The vast majority of episodes of acute and subacute low back pain improve over time regardless of treatment. Thus, current guidelines recommend initially utilizing nonpharmacologic treatment options. Such modalities include massage, acupuncture or spinal manipulation. Should the provider deem pharmacologic therapy indicated, nonsteroidal anti-inflammatory drugs or skeletal muscle relaxants are recommended.

For those suffering from chronic low back pain, current guidelines recommend initially selecting nonpharmacologic treatment options that include: exercise, multidisciplinary rehabilitation, acupuncture, tai chi, yoga, mindfulness-based stress reduction, motor control exercise, progressive relaxation, electromyography biofeedback, low-level laser therapy, operant therapy, cognitive behavioral therapy or spinal manipulation. Should the provider deem pharmacologic therapy indicated, nonsteroidal anti-inflammatory drugs are first-line therapy. Second-line pharmacologic therapy includes tramadol or duloxetine. Pursuing opioid-based therapy should only be considered for those who have failed prior therapeutic modalities, when benefits outweigh the risks and after the patient has been advised on its risks and realistic benefits [52].

References

1. Hubbard MJ, Hildebrand BA, Battafarano MM, Battafarano DF. Common soft tissue musculoskeletal pain disorders. Prim Care. 2018;45(2):289–303.
2. Kuslich SD, Ulstrom CL, Michael CJ. The tissue origin of low back pain and sciatica: a report of pain response to tissue stimulation during operations on the lumbar spine using local anesthesia. Orthop Clin North Am. 1991;22(2):181–7.
3. Indahl A, Kaigle A, Reikerås O, Holm S. Sacroiliac joint involvement in activation of the porcine spinal and gluteal musculature. J Spinal Disord. 1999;12(4):325–30.
4. Borg-Stein J, Wilkins A. Soft tissue determinants of low back pain. Curr Pain Headache Rep. 2006;10(5):339–44.
5. Weiner SL. Differential diagnosis of acute pain by body region. New York: McGraw Hill; 1993. p. 845.
6. Simons D, Travell J, Simons L. Travell & Simons' myofascial pain and dysfunction: the trigger point manual. Pennsylvania: Williams & Wilkins; 1999.
7. Annaswamy TM, De Luigi AJ, O'Neill BJ, Keole N, Berbrayer D. Emerging concepts in the treatment of myofascial pain: a review of medications, modalities, and needle-based interventions. PM R. 2011;3(10):940–61.
8. Ramsook RR, Malanga GA. Myofascial low back pain. Curr Pain Headache Rep. 2012;16(5):423–32.
9. Sharan D, Rajkumar JS, Mohandoss M, Ranganathan R. Myofascial low back pain treatment. Curr Pain Headache Rep. 2014;18(9):449.
10. Hua NK, Van der Does E. The occurrence and inter-rater reliability of myofascial trigger points in the quadratus lumborum and gluteus medius: a prospective study in non-specific low back pain patients and controls in general practice. Pain. 1994;58(3):317–23.
11. Galvez-Sánchez CM, Reyes Del Paso GA. Diagnostic criteria for fibromyalgia: critical review and future perspectives. J Clin Med. 2020;9(4):1219.
12. Bazzichi L, Giacomelli C, Consensi A, Giorgi V, Batticciotto A, Di Franco M, et al. One year in review 2020: fibromyalgia. Clin Exp Rheumatol. 2020;38 Suppl 123(1):3–8.
13. Clauw DJ. Fibromyalgia: a clinical review. JAMA. 2014;311(15):1547–55.
14. Martín J, Torre F, Padierna A, Aguirre U, González N, Matellanes B, et al. Interdisciplinary treatment of patients with fibromyalgia: improvement of their health-related quality of life. Pain Pract. 2014;14(8):721–31.
15. Hohl M. Soft-tissue injuries of the neck in automobile accidents. Factors influencing prognosis. J Bone Joint Surg Am. 1974;56(8):1675–82.
16. Watkinson A, Gargan MF, Bannister GC. Prognostic factors in soft tissue injuries of the cervical spine. Injury. 1991;22(4):307–9.

17. Spitzer WO, Skovron ML, Salmi LR, Cassidy JD, Duranceau J, Suissa S, et al. Scientific monograph of the Quebec Task Force on Whiplash-Associated Disorders: redefining "whiplash" and its management. Spine (Phila Pa 1976). 1995;20(8 Suppl):1s–73s.
18. Li F, Liu N-S, Li H-G, Zhang B, Tian S-W, Tan M-G, et al. A review of neck injury and protection in vehicle accidents. Transp Saf Environ. 2019;1(2):89–105.
19. Benedetti PF, Fahr LM, Kuhns LR, Hayman LA. MR imaging findings in spinal ligamentous injury. AJR Am J Roentgenol. 2000;175(3):661–5.
20. Phadke A, Bedekar N, Shyam A, Sancheti P. Effect of muscle energy technique and static stretching on pain and functional disability in patients with mechanical neck pain: a randomized controlled trial. Hong Kong Physiother J. 2016;35:5–11.
21. Limpaphayom N, Kohan E, Huser A, Michalska-Flynn M, Stewart S, Dobbs MB. Use of combined botulinum toxin and physical therapy for treatment resistant congenital muscular torticollis. J Pediatr Orthop. 2019;39(5):e343–8.
22. Kessler KR, Skutta M, Benecke R. Long-term treatment of cervical dystonia with botulinum toxin A: efficacy, safety, and antibody frequency. J Neurol. 1999;246:265–74.
23. Langevin P, Lowcock J, Weber J, Nolan M, Gross AR, Peloso PM, Roberts J, Graham N, Goldsmith CH, Burnie SJ, Haines T, Cervical Overview Group. Botulinum toxin intramuscular injections for neck pain: a systematic review and metaanalysis. J Rheumatol. 2011;38(2):203–14.
24. Langevin P, Peloso PMJ, Lowcock J, Nolan M, Weber J, Gross A, Roberts J, Goldsmith CH, Graham N, Burnie SJ, Haines T. Botulinum toxin for subacute/chronic neck pain. Cochrane Database Syst Rev. 2011;(7):CD008626.
25. Alai NN, Skinner HB, Nabili ST, Jeffes E, Shahrokni S, Saemi AM. Notalgia paresthetica associated with cervical spinal stenosis and cervicothoracic disk disease at C4 through C7. Cutis. 2010;85:77–81.
26. Faur C, Patrascu JM, Haragus H, Anglitoiu B. Correlation between multifidus fatty atrophy and lumbar disc degeneration in low back pain. BMC Musculoskelet Disord. 2019;20(1):414.
27. Freeman MD, Woodham MA, Woodham AW. The role of the lumbar multifidus in chronic low back pain: a review. PM R. 2010;2(2):142–6.
28. Jeffrey R, Cooley BFW, Ardakani EM, Kjaer P, Jensen TS, Hebert JJ. Relationships between paraspinal muscle morphology and neurocompressive conditions of the lumbar spine: a systematic review with meta-analysis. BMC Musculoskelet Disord. 2018;19(351):21.
29. Hides JA, Jull GA, Richardson CA. Long-term effects of specific stabilizing exercises for first-episode low back pain. Spine. 2001;26(11):E243–8.
30. Wilke J, Schleip R, Klingler W, Stecco C. The lumbodorsal fascia as a potential source of low back pain: a narrative review. Biomed Res Int. 2017;2017:5349620.
31. Redmond JM, Chen AW, Domb BG. Greater trochanteric pain syndrome. J Am Acad Orthop Surg. 2016;24(4):231–40.
32. Reid D. The management of greater trochanteric pain syndrome: a systematic literature review. J Orthop. 2016;13(1):15–28.
33. Seidman AJ, Varacallo M. Trochanteric bursitis. StatPearls; 2020.
34. Woodley SJ, Mercer SR, Nicholson HD. Morphology of the bursae associated with the greater trochanter of the femur. J Bone Joint Surg Am. 2008;90(2):284–94.
35. Johnson DB, Varacallo M. Ischial bursitis. StatPearls; 2022.
36. Baron R, Binder A, Attal N, Casale R, Dickenson AH, Treede RD. Neuropathic low back pain in clinical practice. Eur J Pain. 2016;20:861–73.
37. Steigerwald I, Müller M, Davies A, Samper D, Sabatowski R, Baron R, Rozenberg S, Szczepanska-Szerej A, Gatti A, Kress HG. Effectiveness and safety of tapentadol prolonged release for severe, chronic low back pain with or without a neuropathic pain component: results of an open-label, phase 3b study. Curr Med Res Opin. 2012;28:911–36.
38. Hackenberg RK, von den Driesch A, König DP. Lower back pain with sciatic disorder following L5 dermatome caused by herpes zoster infection. Orthop Rev. 2015;7:6046:81–83.
39. Junli Zhou JL, Ma L, Cao S. Zoster sine herpete: a review. Korean J Pain. 2020;33(3):208–15.

40. Dooling KL, Guo A, Patel M, Lee GM, Moore K, Belongia EA, Harpaz R. Recommendations of the advisory committee on immunization practices for use of herpes zoster vaccines. MMWR Morb Mortal Wkly Rep. 2018;67:103–8.
41. Oliver B, Boelens RCM, Scheltinga MR, van Laarhoven K, Roumen RM. Chronic localized back pain due to posterior cutaneous nerve entrapment syndrome (POCNES): a new diagnosis. Pain Physician. 2017;20:455–8.
42. Maatman RC, Boelens OB, Scheltinga MRM, Roumen RMH. Chronic localized back pain due to entrapment of cutaneous branches of posterior rami of the thoracic nerves (POCNES): a case series on diagnosis and management. J Pain Res. 2019;12:715–23.
43. Toyohiko Isu KK, Morimoto D, Iwamoto N. Superior and middle cluneal nerve entrapment as a cause of low back pain. Neurospine. 2018;15:25–32.
44. Martin RR, Martin HD, Kivlan BR. Nerve entrapment in the hip region: current concepts review. Int J Sports Phys Ther. 2017;12(7):1163–73.
45. Hicks BL, Lam JC, Varacallo M. Piriformis syndrome. StatPearls; 2020.
46. Jankovic D, Peng P, van Zundert A. Brief review: piriformis syndrome: etiology, diagnosis, and management. Can J Anesth. 2013;60:1003–12.
47. Samara Lewis JJ, Lee C, Lewis R, Gest T. Anatomical variations of the sciatic nerve, in relation to the piriformis muscle. Transl Res Anat. 2016;5:15–9.
48. Misirlioglu TO, Akgun K, Palamar D, Erden MG, Erbilir T. Piriformis syndrome: comparison of the effectiveness of local anesthetic and corticosteroid injections: a double-blinded, randomized controlled study. Pain Physician. 2015;18:163–71.
49. Last AR, Hulbert K. Chronic low back pain: evaluation and management. Am Fam Physician. 2009;79(12):1067–74.
50. John M, Lesher PD, Hager N, Kaplan M, Furman M. Hip joint pain referral patterns: a descriptive study. Pain Med. 2008;9(1):22–5.
51. Tae-Hoon Doo D-AS, Kim H-I, Shin D-G, Kim H-J, Chung J-H, Lee J-O. Clinical relevance of pain patterns in osteoporotic vertebral compression fractures. J Korean Med Sci. 2008;23(6):1005–10.
52. Amir Qaseem M, Wilt TJ, McLean RM, Forciea MA, for the Clinical Guidelines Committee of the American College of Physicians*. Noninvasive treatments for acute, subacute, and chronic low back pain: a clinical practice guideline from the American College of Physicians. Ann Intern Med. 2017;166:514–30.

Non Pharmacological Treatments

7

Renee Enriquez and Isabel Huang

Introduction

Spine pain can be extremely complex and commonly requires a multimodal treatment approach for successful outcomes. Treatment of spine pain can be successful when non-invasive therapeutic treatments are utilized. In this chapter, we review common non-interventional modalities in the current practice of neck and low back pain. We will briefly define the conservative therapeutics and review the efficacy of these treatments to assist you in making informed decisions when formulating a treatment plan. First, we will explore the active therapeutics, which can be self-guided, commonly supervised exercise program, which also happens to be an integral part of the physical therapy program. Then we will discuss passive therapeutics, with much of these treatments requiring a trained professional to assist in its administration. And finally, we will explore the psychological treatments used for spine pain, which in fact requires equal participation from both the provider and the participant. As for all treatments, patient compliance and economic burden must be considered when developing a treatment program. Incorporating just one treatment may not sufficiently manage pain but utilizing multiple effective modalities could potentially reduce pain to a tolerable level so individuals can enjoy an improved quality of life.

Active Component: Therapeutic Exercise

A formalized physical therapy program generally consists of two components: an active component and a passive component. That active component of a physical therapy treatment plan is therapeutic exercise. Therapeutic exercise is the

R. Enriquez (✉) · I. Huang
University of Texas Southwestern, Dallas, TX, USA
e-mail: Renee.enriquez@utsouthwestern.edu; isabel.huang@utsouthwestern.edu

© The Author(s), under exclusive license to Springer Nature Switzerland AG 2022

C. E. Noe (ed.), *Multidisciplinary Spine Care*,
https://doi.org/10.1007/978-3-031-04990-3_7

self-guided or assisted systematic and planned body movements to strengthen, improve endurance and gain flexibility with the goal to ultimately restore normal physical functioning and decrease symptoms. Therapeutic exercise is the most important part of a physical therapy plan for the treatment of some spinal pain, depending on pathology and chronicity. It is also a prescribed self-guided treatment plan with hopes that it will not only improve spine pain but help prevent it.

Acute spine pain is generally accepted as pain that is present for 4 weeks or less; subacute spine pain is pain lasting from 4 to 12 weeks; and chronic spine pain is pain experienced for more than 12 weeks. Therapeutic exercise can be beneficial depending on the chronicity of spine pain and location of pain.

Exercise and Acute Low Back Pain

For acute and subacute low back pain, many patients improve over time without any formalized therapeutics and thus a referral to a structured physical therapy program may not be warranted for acute pain management. In 2017, the American College of Physicians guideline updated current evidence for non-pharmacologic treatment options for low back pain based on systematic review [1]. The most recent review on exercise treatment for acute or subacute low back pain showed there were no difference in pain relief between exercise therapy and no exercise therapy [1], regardless of radicular signs. The benefits, if any, were determined to be modest and short term [1]. However, some physical activity should be recommended in the acute and subacute phase. Historically, bed rest was prescribed as a treatment for low back pain as recent as 1980 [2]. This recommendation is obsolete as current established clinical practice supported by scientific evidence is to have patient return to normal activity [3]. Additional recommendations can be made for self-management of back pain including education given to the patient about self-guided exercises [4]. It is also worth noting that although there may not be strong evidence to recommend a supervised exercise program for acute low back pain, for those who received therapy, there were no reports of serious adverse events [1].

Exercise and Acute Neck Pain

There is a lack of strong evidence to support recommending a formalized therapeutic exercise program in the setting of acute neck pain, regardless of cause. However, clinical practice guidelines recommend self-guided exercise for the management of neck pain, including whiplash associated neck pain [5]. A Cochrane Database Systematic Review on exercises for mechanical neck disorders found no evidence to support exercise only for acute neck pain [6]. However, a systematic review of exercise for neck pain provided by the Ontario Protocol for Traffic Injury Management Collaboration (OPTIMa) did note some benefit of home exercise in

acute neck pain without radicular symptoms, although it did not prove superior to multi-modal manual therapy or medications [7, 8]. In regards to acute neck pain with neurologic signs, OPTIMa did find that there is evidence supporting a supervised graded strengthening exercise program compared to a home exercise program but not superior to other interventions, including a treatment with a cervical collar and rest [7, 9]. Clinical Practice Guidelines for Neck Pain as per the American Physical Therapy Association did recommend a formalized physical therapy program with exercise for acute neck pain with moderate evidence to support these findings [10]. The types of exercise vary, from home exercise program instruction to assisted exercises, but it is supported with moderate evidence for acute neck pain with mobility deficits, associated with whiplash, headaches, and/or radiating pain [10]. A systematic review on preventative exercise therapy for neck pain in office workers revealed there was strong evidence for muscle strengthening and endurance exercise for treating neck pain and reducing disability attributed to neck pain [11]. Again, it should be noted that with many of the above recommendations, the exercises are coupled with a multimodal approach, and the recommendations are inconsistent. Thus, an exercise program in the acute neck pain with and without neurologic signs can be prescribed as a formal therapy program or self-administered program prescribed with education. But other treatment options are available and are shown to be just as effective for neck pain treatments.

Exercise and Chronic Spine Pain

There is a general consensus that exercise should be prescribed for chronic low back pain and neck pain. It is a first line treatment, especially in chronic low back pain, and is recommended in all current clinical practice guidelines [12]. Exercise therapy improves not only pain but overall function with those suffering from chronic low back pain. Although not completely understood, the effects of exercise are likely multi-faceted, by improving neurologic, psychologic and musculoskeletal function, which are supported by numerous animal and human studies. It is in the type of exercise, formal or informal, that guidelines vary extensively.

Current clinical guidelines by various medical organizations endorse the use of exercise in chronic low back pain. The recent clinical practice guideline from the American College of Physicians based on a large systematic review study showed moderate evidence that exercise resulted in a small improvement in pain relief and function when compared to the no exercise group [1]. There were no clear differences between different exercise regimens in more the 20 randomized control studies in patients with low back pain [1]. These exercise routines that were reviewed included Pilates, Tai chi, yoga, motor control exercises, and will be discussed further in the chapter. Choice for the type of non-therapeutic exercise should be patient dependent and preference [12]. The recommendations overall for those with chronic low back pain should include non-pharmacologic therapeutics, including exercise; however, if inadequate, pharmacotherapy should be

initiated [1]. Prior reviews have also concluded that there is no single exercise technique or program that has proven beneficial to another for patients with subacute and chronic low back pain [13].

There is plenty of evidence that walking and aerobic exercise, amongst others are effective in reducing low back pain. In a 2015 meta-analysis of 26 studies, there were clinically significant improvements in low back pain and function at 12 months with walking [14]. However, when compared to other types of exercise, outcomes were similar to other exercises and combining exercise with walking did not improve outcomes [15]. Similar findings were noted with aerobic exercise as being an effective activity to decrease symptoms of low back pain [16].

For chronic low back pain with radiculopathy, recommendations vary based on clinical guidelines. Passive therapies, including spinal manipulation, acupuncture and/or massage are generally not endorsed [7]. However, others have endorsed the use of exercise and spinal manipulation [17], which will be reviewed later in the chapter.

Chronic neck pain also responds well to exercise, either alone or when coupled with other therapeutics. As per the Clinical Practice Guidelines of the American Physical Therapy Association, physical therapy incorporating exercise as well as therapeutics have shown moderate benefit in improving patients' neck pain with mobility deficits and chronic neck pain with headache [10]. For patients with chronic neck pain associated with movement coordination impairments, including whiplash associated disorders, recommendations were to continue patient education, mobilization exercises, and TENS unit. However, neck pain due to whiplash associated disorders responds better during the acute phase, which is supported with moderate level evidence [10]. These exercise recommendations are for a formal therapy program and usually coupled with other modalities.

Supervised exercise alone is also beneficial for chronic neck pain with associated disorders. At least three randomized control trials suggested the supervised Qigong exercise was more effective than no treatment in reducing neck pain and disability for chronic neck pain without neurologic signs [7]. When comparing between types of supervised exercise, the Qi gong group versus general exercise sessions consisting of improving range of motion, strengthening, and flexibility, there was no clinical significance in the difference in outcomes [7, 18]. Supervised Yoga was also proven to be more beneficial than education and home exercise for short-term improvement of pain [7]. When combining a standardized program of strengthening, range of motion and flexibility exercises under direct guidance from a professional, again there is moderate evidence showing improvement in reduction of pain and disability [7] when compared to no intervention.

Self-directed exercise or unsupervised exercise has not been shown to be as beneficial for those with chronic neck pain, regardless of radicular signs or headaches [6, 7]. An updated systematic review of exercises in neck pain did not find high quality evidence to support exercises for home exercise program and no substantial evidence to support a stretching only exercise program for neck pain [6].

Exercise and Prevention of Spine Pain

Clinical practice guidelines in multiple specialties do strongly support therapeutic exercise alone as a treatment plan for spine pain, however, there is strong evidence supporting its incorporation of within a multimodal treatment plan. Not only can therapeutic exercises improve spine pain, there is also evidence that it can be effective in regards to prevention of spine pain.

For low back pain, exercise can be effective in preventing low back pain [19]. A meta-analysis in 2016 showed moderate level evidence for the use of exercise with or without education in the prevention of low back pain [20]. Shiri R, et al. in a 2017 meta-analysis with 13 randomized controlled trials and three non-randomized control trials found that exercise reduced risk of developing low back pain by 33 percent [21]. In addition, a meta-analysis review of 9 observational studies demonstrating post-treatment exercises consisting of strengthening and flexibility was more effective in reducing low back pain recurrence at one year than with no intervention [22].

Exercise has also been shown to have a benefit for preventing neck pain, mostly studied in office workers. There is moderate evidence to support the use of exercise to reduce the risk of new episodes of neck pain as per meta-analysis in 2018 by de Campos [23]. Two randomized, controlled trials of 500 participants found moderate quality evidence that exercise reduces the risk of recurrent neck pain [23]. Even a daily walking program has been shown to be effective in reducing neck pain [24]. A 6-month prospective cluster-randomized control trial enrolled high-risk office workers and randomized them to a walking program or a control group [24]. Once adjusted for confounding factors, a statistically significant preventative effect on neck pain was observed in the walking intervention group [24].

Exercise Based Exercises for Spine Pain

Overall, clinical recommendations guidelines support the use of physical activity and most exercise programs for treatment of spine pain. This might be related to the similar positive effects that all movement-guided treatments have on a body's neurologic or inflammatory system. There are also psychological benefits with exercise. Exercise upregulates neurotrophins, lowers cortisol levels and increases serotonin and norepinephrine levels, and improves oxygenation saturation and angiogenesis in the brain [25]. Finally, there is evidence suggesting that physical activity and exercise intervention have few adverse events that may reduce pain severity, improve physical function, and as a consequent, increase quality of life [26].

Without high level evidence that one particular exercise program is more beneficial than another, prescribing an exercise program should be individualized to the patient, taking into consideration their physical capabilities, preferences and available social and financial resources.

Walking

Frequently prescribed exercises with the least number of barriers to participation is aerobic activity, such as walking. Walking is known to decrease various forms of musculoskeletal pain. A meta-analysis in 2015 showed walking improved pain and function in low back pain and function at 12 months [14]. A walking program has also been shown to be effective in preventing neck pain [24]. A prescribed walking program will vary depending on a person's physical conditioning, but most experts agree that depending on your current fitness level, an increase in pace and time should occur slowly. There is no one specific walking program that is proven to be superior to others, although there are many resources and guidelines provided by government health agencies and nationally recognized medical agencies that are easily accessible online, including sample walking programs available through the National Institutes of Health and the American College of Sports Medicine [27, 28].

Aerobic Exercise

Aerobic exercise also includes bicycling, swimming, and elliptical training. Without high level of evidence to endorse one activity over another, a 2015 meta-analysis of eight cohort studies examining aerobic exercise and back pain showed a significant reduction in Roland-Morris Disability Questionnaire, Oswestry Disability Questionnaire, Hospital Anxiety and Depression Scale, and McGill Pain Questionnaire after aerobic exercise, indicating that aerobic exercise reduces back pain and disability of those with chronic low back pain [16].

Stretching Exercise

There are a wide variety of stretching exercises that prove to be modestly helpful in improving back pain when incorporated in a stretching program, although there are no recent systematic reviews providing high quality evidence to suggest stretching alone is beneficial in improving back pain. There is some evidence that Slump stretching may have a positive effect on people with low back pain demonstrated in a recent 2018 meta-analysis [29]. Slump stretching is an adaptive treatment maneuver based on the slump test, which is a diagnostic test to help differentiate radicular vs non-radicular mediated pain. The maneuvers are thought to reduce edema and hypoxia around the injured neuronal structures [29]. The review of slump stretching on low back pain, including 12 eligible studies with 515 patients with short-term follow up, did reveal a large effect size and significant effect with the use of slump stretching to reduce low back pain and disability improvement, although the evidence was considered of low to moderate quality [29].

A stretching program has been beneficial in reducing chronic non radicular neck pain and disability associated with chronic neck pain; however, the evidence

is supported when stretching was combined with supervised strengthening, range-of-motion, and flexibility exercises [7]. This is consistent with the American Physical Therapy Association Guidelines recommending stretching exercise program coupled with a multimodal treatment plan that is supported by moderate and low level evidence for acute and chronic neck pain with and without neurologic findings [10].

Pilates

Pilates was created by Joseph Pilates, German citizen during the first World War. He attached bed springs to the hospital bed to support and strengthen the limbs, which was the early version of the "reformer", a principal piece of equipment in Pilates for performing instructor trained exercises [30]. When he emigrated to the US in the early 1920s, Pilates continued to teach this method which focuses on the primary principles of breathing, controlled whole body movement with core strengthening and flow of movement outward to the limbs [16]. A recent systematic review in 2018 endorsed the benefits of Pilates [31]. The review included 23 studies, which did show Pilates to be an effective rehabilitation tool for reducing pain and disability [31]. However, it has similar efficacy to other exercise regimens for the treatment of low back pain [32]. Pilates was no more effective than other exercise treatments for chronic low back pain as per another systematic review in 2015, which included 29 studies [32]. There are multitudes of studies that show no evidence to low quality evidence for the reduction in pain with Pilates. Thus, the Clinical Guidelines Recommendations for Noninvasive Treatments of Chronic Low Back pain provided by the American College with Pilates does not directly recommend solely Pilates, but is recommended as a form of exercise due to the mild effects on chronic back pain [1]. Pilates is considered an effective exercise to help reduce chronic low back pain, however, no more superior than other exercise programs.

Yoga

Yoga is a physical, mental and spiritual practice that originated in India some 3000–5000 years B.C. It is a popular form of exercise and meditation throughout the world and has evolved to different styles and forms, thus making adaptable to all levels of expertise and physical functioning. It has been a common exercise that providers recommend for treatment of back pain, but like many self-guided and supervised exercises, there are no high level evidence studies to support that Yoga is superior to other exercises, especially as a form of treatment for chronic low back pain. A recent Cochrane review showed slight functional improvement and pain in non-specific back pain, although there were reports of increased pain with Yoga when compared to non-exercise control groups [33]. And when compared to other non-yoga exercises, Yoga was not more effective in treatment for low back pain.

Neck pain can also be treated with yoga with low level evidence showing a significant improvement in symptoms. A 2019 meta-analysis on the effects of Yoga in patients with chronic, non-specific neck pain consisting of 10 trials and 686 participants showed a positive effect on neck pain intensity, pain-related functional disability, cervical range of motion, mood and quality of life [34]. Yoga was superior to exercise for improving cervical range of motion, although the evidence was limited [34].

Tai Chi

Tai chi originated in ancient China as a self-defense technique that has evolved into a graceful form of exercise. It consists of self-paced, slow but constant movements performed in a focused manner that allows for gentle exercise and stretching. Tai chi is commonly prescribed for elderly patients since it is low impact and considered relatively safe for most if not all fitness levels.

Although the practice is usually performed in a standing position, it can also be performed in a sitting position for those who have functional and balance impairments. Tai chi can also improve balance and thus one can progress from seated to standing practice since tai chi has been shown to reduce elderly falls in the community. A 2017 systematic review found that Tai Chi lead to a decreased risk of falls in the elderly [35]. It is currently endorsed as a preventative exercise for the elderly to reduce falls by the United States Department of Health and Human Services and the English National Health Service. Furthermore, a randomized control trial in 2018 by Li and colleagues compared effects of tai chi/qigong to multimodal exercise intervention in preventing falls in the community dwelling high-risk elderly. The study consisted of 670 participants that were delegated to 3 exercise groups of either tai chi/qigong, multimodal standard exercises consisting weight training, aerobics and stretching, or stretching exercises alone. The tai chi/qigong group showed lower incidence of falls compared to the other groups and showed improved cognitive functioning as well as physical performance function scores when compared to stretching alone [36]. Thus, there is evidence to support the use of tai chi for balance, but tai chi has also shown some benefit in treating spine pain.

Not only does tai chi show evidence for improving balance, there is also moderate evidence that tai chi can be effective in reduction in pain and disability in chronic nonspecific back pain [37]. A randomized control trial with sample size of 160 compared tai chi to non-treatment for patients with chronic non-specific back pain. Although the results were only assessed immediately post-treatment, there was a statistically significant decrease in pain and disability in the tai chi group [38]. A 2017 systematic review for an American College of Physicians Clinical Practice Guidelines included 2 fair quality randomized control trials that showed participants in the tai chi group had great improvement in function when compared to waitlist or no treatment [39].

Non-specific neck pain can potentially improve with tai chi although there is less supportive data to support its use. The systematic review by Yuan Q and colleagues reviewing traditional Chinese medicine for neck and back pain did not find any randomized controlled trials in regards to tai chi based on their criteria [37]. There is a randomized control trial on the effects of tai chi and neck exercises for the treatment of chronic neck pain. Lauche and colleagues [40] assigned 114 participants with non-specific neck pain into a 12-week treatment plan of tai chi, neck exercises or waitlist. After 12 weeks, tai chi participants exhibited a significant decrease in pain in comparison to the waitlist group [40]. Tai chi group also had improvement in functional disability and quality of life, and overall tai chi was thought to be more effective in relieving non-specific neck pain. Despite these promising findings, the authors concluded that tai chi was no more effective than conventional exercises for neck pain in this study [40].

Qigong

Qigong is a focused breathing and meditation practice coupled with coordinated body posture and movement that improves health and spirituality and is also incorporated in martial arts training. It is similar to tai chi in that they are both ancient Chinese practices focusing on mind and body. Its purpose is to increase and restore the flow of qi energy and regain balance, which promotes good health [41]. It has the potential to be helpful in neck pain but has not proven superior to other exercises programs when compared in quality studies. There appears to be less evidence to support its benefit in reducing low back pain, at least when compared to studies focusing on benefit of the therapeutic for neck pain.

The systematic review of traditional Chinese medicine incorporated two randomized controlled studies comparing Qigong and waitlist/no treatment for neck pain and showed pain improvement in the treatment group, with moderate evidence to support. But comparing Qigong to exercise showed no difference in the short-term and intermediate term, suggesting that it is not superior to other treatments for neck pain [37]. In vonTrott P et al. randomized control study demonstrated clinically important reductions in neck pain at 3 months compared to waitlist group and at 6 months when compared to seated qigong and a waitlist group; however, no clinically important differences were noted between the qigong group and supervised exercise group [42].

Physical Therapy

Physical therapy (PT) is a major component to a multidisciplinary, multimodal non-interventional treatment plan of spine pain. The primary portion of therapy is the active component with supervised therapeutic exercise. Many different physical

therapy programs developed with changes in their approach to exercise. There is good evidence supporting most exercise programs as successful treatments of pain, although there is variability in the evidence supporting one program over another.

The Merriam Webster dictionary defines physical therapy as an, "[intervention] for the preservation, enhancement, or restoration of movement and physical function impaired or threatened by disease, injury, or disability that utilizes therapeutic exercise, physical modalities, assistive devices, and patient education and training" [43]. Physical therapy was officially brought into existence in 1813 by Per Henrik Ling, known as the "father of Swedish gymnastics", where he founded the royal Central Institute of Gymnastics for massage, manipulation and exercise [44]. The term physiotherapy was first presented to the English-speaking world to the general population in the second edition of the Oxford English Dictionary in 1905. However, the term was used as early as 1894 by Dr. Edward Playter of Canada during a presentation and later published an article stating the importance of natural remedies such as food, clothing, rest, exercise, and massage, may be considered natural therapeutics, or "physiotherapy" [45]. The field of physical therapy subsequently progressed in the early 1900's during the polio epidemic with passive range of motion treatments being utilized, as well as the use of modalities administered in a structured medical program [44]. The use of "natural therapeutics" flourished in the US with its involvement in World War I and II due the increasing needs to treat chronically injured soldiers.

The field has since flourished to be a part of an accepted treatment approach prescribed by physicians and providers conservatively treating musculoskeletal diseases, pathology, and symptoms, including spine pain. Currently, there are 223,751 licensed physical therapists in US [46], many focusing on the rehabilitation of those with spine pain in light of the high prevalence of back and neck pain in the US. The majority of pain-related PT guidelines are for pain associated with spinal pathology [47]. The following sections will examine the elements of physical therapeutics provided by therapists either actively or passively, as well as review its efficacy in the treatment of neck and low back pain.

Physical Therapist Lead Exercise Programs

Professional or supervised exercise is a major component to physical therapy. As noted earlier in the chapter, exercise can be beneficial for reducing spine pain, but evidence shows that supervised exercise is superior in the treatment of chronic ailments afflicting the neck and back. The benefit of a guided exercise treatment plan is the one-on-one supervision with a professional to motivate, teach, and help prevent injury. There is also a passive component to physical therapy, which allows the therapist to reduce pain and improve function with modalities, many of which can augment the effects of pain reduction provided by the exercise. Those modalities and treatments will be discussed later in the chapter. Although not an exhaustive list, we will review review supervised exercise and stretching programs frequently prescribed by spine providers and administered by certified physical therapists.

Types of PT Programs

McKenzie Therapy

The McKenzie Method of Mechanical Diagnosis and Therapy (MDT) is a classification system and popular treatment approach for spine pain. The McKenzie method was developed by a physiotherapist named Robin McKenzie from New Zealand. In the 1960s, Robin McKenzie developed a diagnosis and management approach for spine pain that involves centralization of pain. Due to the popularity of his program, Robin McKenzie created the McKenzie Institute International in 1981. The institute certifies other physical therapist as an official McKenzie certified therapist if they have completed the required training certification [48].

McKenzie exercises are generally thought of as spinal extension exercises; however, this is not the primary mechanism for treatment. Instead, MDT is a standardized approach to both the assessment, classification, treatment and prevention of low back pain with or without radicular pain. MDT focuses its treatment on self-treatment strategies, and minimizes manual therapy procedures with the McKenzie-trained therapist who instead supports the patient with passive procedures. McKenzie organization state that self-treatment is the best way to achieve a lasting improvement of back pain [48].

The initial approach of the program is to classify the patient into 3, or potentially 4 groups of disorders: derangement, dysfunction, postural, or other. It is through this classification that the therapist determines treatment. One of the most common groups is the derangement group, which refers to pain that is caused by disruption of the normal anatomical position of the spine. The repeated maneuvers in one direction cause a production of symptoms or distal migration of symptoms that is sub optimal or pathologic. It is the opposite of that maneuver, or the one direction of repeated maneuvers, which decreases the referred symptoms or centralizes it. This maneuver is a clinically induced directional preference of the patient and the target of the therapist [49]. The most common directional preference that results in centralization is extension of the back, and why the MDT is considered a spine extension program. The presence of centralization itself has shown to be a good prognosis for patients with low back pain, and when obtained through an appropriate MDT program, there are improvements in patient outcomes [50]. It is generally accepted that the current assessment model for classifying patients with low back pain has demonstrated good inter-examiner reliability on previous studies, but evidence for treatment effectiveness for back and neck pain is limited [51].

A recent literature review and meta-analysis in 2018 examined the McKenzie program for the treatment of acute and chronic low back pain [51]. For acute low back pain, 3 RCT were included and there was good quality evidence showing MDT was not clinically superior to other exercise interventions or just education for the treatment of acute LBP [51]. However, the same review found moderate to high – quality evidence that MDT was superior to other rehabilitation interventions in reducing pain and disability for chronic low back pain [51]. A 2012 systematic review found that lumbar centralization was associated with a better recovery prognosis in terms of pain, short- and long-term disability, and the decreased likelihood

of undergoing surgery in the following year [52]. A more recent systematic review compared MDT to manual therapy for the treatment of low back pain [53]. It did find that McKenzie method fared better in pain outcomes in the short term and disability measures in the long term, but acknowledged that the evidence is low quality due to limitations, which included the 5 RCT with small sample sizes [53].

In regards to MDT and acute or chronic neck pain, a systematic review of efficacy of McKenzie therapy for spine pain determined that there is insufficient data available on the efficacy of MDT for neck pain, regardless of chronicity [54]. Another systematic review examining the reliability of the classification system in MDT found that there is limited data to determine the inter-rated reliability of the MDT classification system for neck and thoracic spine pain [55]. There was also limited evidence for identifying directional preference in patients with neck pain [55], which will ultimately have an impact on the diagnostic component of MDT and subsequently the treatment plan.

Movement Control

- Movement control exercises (MCE).

Non-specific back pain may be as a result of movement impairment and abnormal spinal alignment. This leads to trauma to tissues due to improper tissue loading [56]. The repetition of altered alignments and movements result in localized tissue stress and continuous nociceptive irritability [57]. The proposed mechanism of pain is called movement control impairment [58]. The therapeutic approach to treat this impairment is called movement control exercises [58] also known as *Spinal Stabilization.* Therapists treating those thought to have movement control impairments aim to restore movement, correct movement patterns and avoid postural patterns provoking pain. Most of the motor control exercises focus on muscle recruitment patterns and retrain the movement of muscles to an optimal pattern in a progressive manner [59]. Another term that has been used interchangeably is motor control exercise [60]; however, *motor* control exercise is disputed in the literature as different exercise when compared to *movement* control exercise [58]. Although the difference might appear to be minor, *motor* control exercises focus on individual muscles, whereas, *movement* control exercises and interventions focus on deep trunk musculature as a whole with strengthening in a more coordinated manner [58]. Regardless of the differences, the goal of treatment is to improve back pain by focusing on activation, coordination and control of pelvic, core and deep spinal musculature. There have also been variations of these exercises overtime, although no one approach has been shown to be superior versus another.

This therapeutic approach is very popular and commonly prescribed by physicians and other medical providers for the treatment of non-specific low back pain. A Cochrane Review study in 2016, which included 29 randomized control trials with a total of 2431 participants, evaluated the effectiveness of motor control exercise in patients with non-specific low back pain [61]. When compared to minimal intervention, there was low to moderate quality evidence that pain was improved at short, intermediate and long-term follow up [61]. There is low quality of evidence that

motor control exercises have more of a clinical effect compared to exercise with or without modalities [61]. There is also moderate to high quality evidence that motor control exercises provide similar outcomes to manual therapies [61].

The most recent systematic review in 2018 by Luomajoki et al. reviewed the benefit of motor control exercise on patients with non-specific back pain [58]. The study included 11 randomized control trials with total of 781 participants receiving movement control exercises versus variable control groups. The authors claimed that the effect of motor control exercise treatment on disability was improved at 12 months but there was no difference in pain after 12 months [58]. The quality of the evidence was very low to moderate [19] and part of this could be due to the heterogeneity of the studies included and small samples sizes in the selected studies [58].

Although limited studies exist, there appears to be a benefit of motor control exercises combined with education, including cognitive functional therapy, for treatment of chronic back pain [57]. This will be discussed later in the chapter.

Williams Flexion

Williams Flexion Exercise (WFE) or Williams Lumbar Flexion Exercise is a therapeutic exercise program that focuses on enhancement of lumbar flexion and strengthening of the core and pelvic muscles. It was created by an orthopedic surgeon, Dr. Eugene Regen, in the 1930s. It was as a form of non-surgical spine care, mostly targeted at men younger than 50 and women younger than 40 [62]. He proposed that those with hyperlordosis of the lumbar spine leads to increase pain and pathology and flexion mediated exercises would reduce pressure on the posterior elements of the spine and relieve pain by correcting the mal-alignment [63]. WFE is considered a flexion based exercise program, contradictory to the McKenzie extension approach, although both programs do not solely rely on one positional direction for pain when individualized for the patient and pathology. A small preliminary study compared MDT with WFE in 1984 for the treatment of low back pain. The MDT group had better outcomes, including improvements in low back pain and pain-free range of motion, although the quality of evidence of the study was low [63]. In 1991, another study comparing the effects of spinal flexion exercises to spinal extension exercises on patients with low-back pain showed no difference in low back pain severity [64]. Another prospective RCT comparing the effects of flexion and extension back exercises among soldiers with acute low back pain again found no difference between the groups [65].

Overall, there is limited data or systematic reviews evaluating WFE, although the principals of the treatment are still used by therapists today.

Back School

Back schools are educational and training programs provided for treating and preventing spine pain and injuries. Back school classes are commonly taught by therapists within the occupational health care setting. It was implemented in 1969 with a target population of those at risk for injury to the low back, those with non-specific back pain, intermittent acute or chronic back pain, and post-surgical patients [66].

There are multiple back schools, with multiple educational methods and different exercises, but most tend to follow a typical content and structure. They discuss back anatomy, common musculoskeletal disorders, the adverse effects of mechanical strain and poor positioning, the need for exercising and strengthening the back and core with practical applications.

A Cochrane review in 2004 demonstrated moderate evidence supporting the benefit of back schools, particularly in the occupational setting, with reduction of pain and improved function and return to work status when compared to other therapeutic treatments [67]. However, a systematic review and meta-analysis in 2016 evaluating back schools for the treatment of chronic low back pain refuted those claims and found that the evidence in support for the use of back schools for treatment of low back pain was weak [66]. A more recent Cochrane review published in 2017 examining back school for the treatment of chronic low back pain reinforced the prior review in 2016. The review included 30 trials with 4105 participants comparing back school to no treatment, medical care, passive physiotherapy, or exercise therapy. The meta-analysis showed minimal to no difference in efficacy to favor back school [68]. In fact, at long-term follow-up, low quality evidence suggested that the passive physiotherapy was better than back school [19, 68].

Passive Component: Manipulation and Modalities

Besides a supervised exercise program, physical therapists incorporate other components into their comprehensive treatment plan. There is manual therapy and manipulation, which is also provided by other specialties, including chiropractors and doctors of osteopathic medicine. Therapeutic modalities are another major component to the treatment plan. Therapeutic modalities utilize thermal, mechanical, and light energies to decrease pain, improve range of motion, tissue healing, and muscle activation [69]. These passive therapies, when provided by a trained professional, may improve short- and long-term outcomes for spine pain.

There is a difference between physical therapy manipulation and chiropractic manipulation but that is beyond the scope of this discussion. Similar principals within the two practices are applicable to this discussion and thus a differentiation of such treatments is not necessary in this section.

Mobilization and Manipulation

Manipulation is a passive technique where the provider applies a specific directed manual force to a joint at or near the end of its passive range of motion. A common feature that patients note is a “crack” or “pop” within the joint. That pop is thought to be secondary to cavitation, where gas bubbles that have formed in the joints are released via manipulation [70]. The physiological effects of spinal manipulation are thought to be caused by the changes on the inflow of sensory information to the central nervous system [71]. Specifically for low back pain and associated

diagnoses, it is believed that it impacts primary afferent neurons from paraspinal tissues, motor control and pain processing [71]. When the participant is evaluated and deemed appropriate by the therapist, manipulation can be an effective for both acute and chronic neck and back pain.

In acute and subacute neck pain with and without mobility impairments and without neurologic signs, the Clinical Practical Guidelines for Neck Pain recommend thoracic spinal manipulation with moderate level of evidence to support [10]. For chronic neck pain with and without neurologic deficits, cervical and thoracic manipulation is recommended, which is supported by moderate level evidence [10]. For subacute and chronic neck pain with headache, cervical manipulation is supported. These guidelines are based on multiple studies, including the Cochrane review of cervical and thoracic manipulation and mobilization for neck pain to assess if it improves clinical outcomes in patients with acute, subacute or chronic pain [72]. 51 trials were included, with a total of 2920 participants. Measures of improvement were based on pain, function and disability, patient satisfaction, quality of life and global perceived effect in adults. The review demonstrated with moderate quality evidence that manipulation and mobilization both provided improvement in pain, function and satisfaction for sub-acute and chronic neck pain at short term and intermediate follow up [72]. There is low quality evidence that manipulation versus control provided intermediate and short-term relief of acute or chronic pain [72]. Another systematic review demonstrated a superior benefit when manipulation is used in conjunction with other treatments [73].

Spinal manipulation has shown similar benefits in acute low back pain and superior benefits with chronic low back pain. A Cochrane review in 2012 showed that spinal manipulative therapy was no more effective in acute low back pain than other recommended therapies [74]. The same author later updated the review with high-quality evidence suggesting that there is no clinically relevant difference between spinal manipulative therapy and other interventions with pain reduction and functional status in those with chronic low back pain [75]. A more recent systematic review and meta-analysis (2018) of the benefits, including efficacy, effectiveness, and safety of various manipulation therapies for treatment of low back pain proved supportive of these measures for treatment [76]. There were 9 trials with 1176 participants, and mobilization and manipulation were compared to other active therapies [76]. Seven out of the nine trials demonstrated a decrease in disability at a clinically significant level. Subgroup analysis showed mobilization versus other treatment was superior in decreasing pain, where a subgroup analysis of manipulation showed a statistically significant decrease in pain and disability. There was moderate quality evidence that manipulation and mobilization can reduce pain and improve function in those with low back pain [76]. Both manipulation and mobilization were deemed safe treatment options.

Massage

Therapeutic massage is defined as manipulation of soft tissue to improve upon an individual's well-being. There are various forms of massage techniques. *Effleurage* incorporates gliding strokes along the muscle. *Petrissage* involves pressure or

kneading over the muscle. *Friction* involves quick movements to generate warmth on the muscles. *Tapotement* involves rhythmic tapping on the body to stimulate the muscles. *Vibration* involves rhythmic quick movements over skin. Stretching either active or passive, and muscle energy techniques where the patient is asked to move against the provider can be incorporated. Reflexology is yet another form of massage technique which focuses on putting pressure on areas of feet and hands, which per reflexology theory corresponds to the body. Pressure on the feet and hand is thought to bring healing to the corresponding region of the body [77]. Additionally, fascia, which is a thin connective tissue that surrounds muscles, can be released utilizing fascia release techniques.

Per the Cochrane Review where 25 trials were reviewed in a total of 3096 participants suffering from non-specific low back pain. One low quality study with only 51 participants indicated massage was not better than inactive controls. For sub-acute and chronic low back pain, the evidence was unclear whether or not massage was better than active control. The 2015 Cochrane reviewed 12 new randomized control trials of massage for low back pain that indicated there is very little confidence that massage was able to effectively treat low back pain. For acute low back pain, massage did seem to help with short-term pain but did not seem to improve function. For sub-acute and chronic low back pain the improvements noted in pain and function appeared solely short-term when compared to inactive controls. Generally, studies reviewed in the 2015 Cochrane review were deemed as "low" to "very low" quality of evidence which differed from the prior 2008 Cochrane Review which noted that the studies reviewed at that time was "moderate" in quality and findings indicated more positive results for massage therapy. Difficulties with blinding studies was the biggest barrier to producing studies with strong methodologies. Amongst the studies reviewed, there were also a large number of differing massage techniques [78].

In regards to neck pain, Cochrane Review in 2012 reviewing 15 trials was inconclusive for managing neck pain but as a stand alone treatment massage was noted to provide short-term relief for pain. The studies that were reviewed had methodology that was low grade and just as the Cochrane reviews for low back pain, the trials generally did not adequately describe the massage techniques utilized. One study indicated that nearly one-fourth of the participants noted low blood pressure after the treatments [79].

Although it is unclear from studies whether or not massage has been helpful for low back pain, one can consider utilizing this modality to treat neck or back pain especially since there are very minimal adverse events associated with massage.

Passive Therapeutics

Therapeutic modalities, also called biophysical agents [80], have long been an approach to physical therapy and rehabilitation. The modalities use various forms of energy, including the following: mechanical, like traction and compression; thermal, like heat and cold; and electromagnetic, like electrotherapy, diathermy,

ultraviolet, and infrared light. They provide therapeutic benefits with minimal participation of the patient. There are multiple benefits for healing and recovery, including wound, soft tissue and muscle recovery after injury or surgery, pain modulation, decrease inflammation, improve connective tissue extensibility, improve circulation and tissue perfusion [80]. Although most modalities expose patients to a low risk of adverse events, the following conditions should be considered a contraindication or at minimum a precaution for utilization of many modalities including: acute deep vein thrombosis or thrombophlebitis, hemorrhagic conditions, impaired sensation, impaired cognition, electrical implants, pregnancy, and malignancy [80]. Overall, modalities are an accepted part of a multimodal treatment plan used for spine pain, and thus, the most common modalities used for spine pain will be discussed below. Again, this is not an exhaustive list and includes the modalities that are utilized frequently and its efficacy has been studied extensively.

Thermal Modalities

Cryotherapy is the use of cold for a therapeutic benefit by lowering tissue temperature. It decreases tissue metabolism and blood flow and is commonly used for acute injuries with bleeding and rapid inflammation, which reduces bleeding and swelling. Cold also reduces pain by desensitizing peripheral afferent nerve endings [81], which is beneficial in an acute or chronic injury or pain state. It can be applied through many vehicles, and is a considered first line treatment option for acute injuries.

Thermotherapy is superficial heat that is conveyed through conduction or convection. It has similar benefits as superficial cold but it does so by opposite mechanisms. It increases tissue temperature and blood flow, facilitating tissue healing, and relaxes skeletal muscles thereby decreasing spasm and pain. It is also applied through multiple vectors and has been thought of as a first line modality for chronic pain.

In regards to the benefit of either thermotherapy or cryotherapy for treatment of neck and back pain, there have been conflicting recommendations from national medical organizations. A Cochrane review in 2009 concluded that the evidence is limited to support the use of superficial heat and cold for low back pain [82]. One study showed heat wraps moderately improved pain relief and disability for low back pain in the short term compared to placebo, which was supported with moderate quality evidence [82]. Another study show low quality evidence that a heat wrap provided effective relief compared to ibuprofen and Tylenol after 1 to 2 days [83]. Another low-level study showed that heat and exercise provided greater relief at one week compared to exercise alone [84].

For chronic neck pain, a systematic review in 2013 showed there was no difference between hot packs and mobilization, manipulation at improving pain, function or patient satisfaction [83]. In fact, there was no added benefit when hot packs were combined with mobilization, manipulation, or electrical stimulation [83]. A large systematic review article on the effects of treatments for different types of neck pain, including acute and chronic whiplash, neck pain with radiculopathy, and non-specific neck pain, which included 91 systematic reviews, randomized control trials

or observational studies found that there is no information regarding if heat or cold is better than active treatment [86].

Thermal modalities are frequently prescribed by medical providers and used in physical therapy treatments. They are relatively safe options for pain treatment with low quality evidence to support their use. Although they are not proven superior to other treatments, it may provide some temporary relief for symptomatic patients.

Transcutaneous Electrical Nerve Stimulation

Transcutaneous electrical nerve stimulation (TENS) is a form of electrotherapy, like percutaneous electrical nerve stimulation, that is used in the treatment of pain, especially spine pain. It works, in theory, on stimulating large afferent fibers, which inhibits the small nociceptive fibers to reduce the sensation of pain through gate control mechanisms [85]. The low threshold large afferent fibers can be stimulated selectively with low intensity current stimulation, and does not activate the small pain fibers [86]. TENS is a commonly prescribed modality for patients with neck and back pain with conflicting levels of evidence in the literature in support of its use and demonstrated efficacy for the treatment of spine pain.

The evidence to support the use of TENS unit in the reduction of low back pain is variable. The clinical practice guidelines from the American College of Physicians reported in 2017 that there was insufficient evidence to determine the efficacy of TENS [1]. A more recent systematic review and meta-analysis examining TENS and interferential current (IFC) examined this treatment in adults with chronic low back pain. IFC is a newer form of electrical stimulation modality that summates two alternating current signals at different frequencies resulting in a frequency of about 4000 Hz that can penetrate the tissues deeper with less pain than with TENS modulating a frequency of 125 Hz. The 2017 study looked at nine randomized controlled trials with 655 participants receiving TENS and IFC interventions versus control groups [87]. The electrotherapy group had better pain relief during therapy but not immediately after, or at 1–3 months post treatment and there was no effect on disability. Like many other systematic reviews, the quality of clinic trials is low and findings remain consistent amongst other systematic reviews [85–88].

Similar to TENS, there is electrical muscle stimulation (EMS), which is also a form of electrotherapy that is used to help with muscle pain, reduce spasms and improve strength [86]. EMS transmits a strong current that causes visible muscle fiber contraction but subsequently causes activation of both large afferent fibers and small nociceptive fibers, which increases pain sensations [86]. Like TENS, multiple systematic reviews have failed to show efficacy in pain relief with EMS [1, 19]. However, a randomized controlled double blind study in 1997 showed that, TENS and EMS produced greater pain reduction in low back pain in combination than when utilized independently [89].

One systematic review in 2013 examining physical modalities for neck pain and associated disorders did review TENS therapy with inclusion of 3 randomized controlled trials and a total of 88 participants [90]. There was pain reduction with TENS compared to placebo for whiplash-associated disorders immediately following treatment. TENS was also found to significantly reduce pain better in acute

whiplash-associated disorders when compared to standard physiotherapy [90]. One randomized controlled trial did show a benefit with less pain immediately after treatment with TENS in chronic neck pain when compared to electrical muscle stimulation [86, 90].

Despite low quality evidence showing efficacy of electrotherapy [91], meta-analyses of TENS benefits tend to show positive outcomes when compared to placebo but rarely do they reach statistical significant and unclear the clinical significance [85]. Therefore, TENS and other forms of electrotherapy should be considered for acute and chronic neck and back pain as it may reduce pain during and immediately after treatment, and also has a low risk profile in the correct patient population [85] .

Spinal Traction

Spinal traction modality includes mechanical or manual traction, which applies distractive forces to decrease or reduce compression on structures of the spine. It reduces mechanical pressure on the spinal segments and associated peripheral nerves, joints, and vasculature, and presumed to then decrease pain, increase range of motion, improve range of motion, among other benefits [92]. There is very limited data demonstrating the benefit of traction therapy [85].

For radicular back pain, a Cochrane review in 2013 reviewed 32 studies with 2762 participants with acute, subacute and chronic low back pain with and without sciatica, with a majority of studies having up to a 4- month follow-up [93]. Mechanical or manual therapy with or without physical therapy was no more effective for treating low back pain than all other interventions including sham, and other modalities [93]. Notably, seven of the studies in the review reported side effects including increased pain, aggravation of neurologic side effects and surgery [93].

Review of traction for the treatment of neck pain had similar results as seen with low back pain. Graham et al. 2013 showed there was no difference between continuous traction and placebo for improving pain or function in patient with acute and chronic pain [90]. Another systematic review of neck pain and traction compared with sham traction did not proved more effective pain relief in 4 weeks or 3 months, nor did it improve sleep, social functioning and activities of daily life [94]. In that same systematic review, four randomized controlled trials, two of which were of sufficient quality, compared traction versus sham traction, placebo tablets, exercise, acupuncture, heat, collar or analgesics. There was no consistent difference in pain between traction and the other treatments [94]. The incidences of serious adverse events in the studies were low [94].

Thus spinal traction has not proven superior to other therapeutic modalities and there is data to support that it is no better than placebo. Some studies suggest that traction is not effective for the treatment of back pain [85].

Low Level Laser Therapy

Laser is an acronym for Light Amplification by Stimulated Emission of Radiation. Low level laser therapy is an electric modality that produces wave length of light, that is thought to affect the function of fibroblasts to accelerate connective tissue

repair and reduce inflammation and pain [95]. Variable wavelengths of 660 nm and 905 nm are used in the treatment of spine pain as well as other musculoskeletal disorders [95]. It is FDA approved for temporary relief of muscle and joint pain but with limited evidence to support its use. The American Academy of Orthopedic Surgeons has no recommendations for or against its use [95]. The ICON systematic review found one randomized controlled trail with 55 participants showing no benefit of laser when compared to placebo for chronic pain syndrome [90, 96]. In another RCT, a specialized form of laser therapy was inferior to manipulation for cervicogenic headache [90]. However, there is a randomized controlled trial of low-quality evidence that showed a combination of low level laser therapy and NSAIDS do decrease pain and moderately improve function compared to sham therapy and NSAIDs in acute and subacute pain [1, 97].

Therapeutic Ultrasound

Ultrasound therapy (US) is the use of sound waves that deliver deep heat energy to deep tissues to treat musculoskeletal orders including spine pain. It is a form of mechanical energy but still considered an electrotherapy. It has been used by physiotherapists since the 1940s. It is thought to reduce local swelling, promote soft tissue healing, soften scar tissues and increase soft tissue elasticity.

A 2020 Cochrane review assessed if ultrasound is an effective treatment for chronic non-specific low back pain [98]. 10 studies were included in the review with 1025 participants treated for chronic non-specific low back pain. Studies compared ultrasound to placebo, no treatment, electrical impulses, manipulation, laser therapy and exercise [98]. Based on the review, there was low and very-low evidence that ultrasound improves pain and well-being more than placebo; and when compared to other interventions, the studies were poorly conducted or were too imprecise for the authors to make a conclusion [98]. An additional review by Graham et al. demonstrated that pulsed ultrasound was no better than placebo at changing function or global perceived effect immediately post treatment in patients with acute whiplash associated disorders or chronic myofascial neck pain [90]. US was also inferior to mobilization for subacute and chronic neck pain immediately after treatment [90]. An additional systematic review in 2011 evaluating the efficacy and safety of ultrasound and shockwave therapies for the treatment of back pain also found that the evidence from their review does not support the effectiveness of ultrasound or shock wave therapy for treatment of low back pain although no adverse events were reported [99].

Currently, existing evidence does not support the use of therapeutic ultrasound in the management of neck and back pain.

Bracing

This is often a topic of debate as to whether or not bracing is helpful. When an individual is suffering, the treating provider often tries to incorporate a multimodality approach to treat the pain and external supports are often considered. Bracing is often requested by patients since the idea of bracing an injured portion of the body is seen time and time again in a variety of other situations such as bracing for fractures, bracing for painful knees, tennis elbows, dysfunctional sacroiliac joints etc.

Cervical collars, either soft or hard, are routinely discouraged for acute cervical neck pain as these may delay recovery [100, 101]. In a systematic review of 11 randomized trials of patients reporting acute neck pain secondary to whiplash, subjects who were recommended to rest and immobilize with a soft collar suffered from neck pain for a longer duration than those who engaged in active physiotherapy [102].

Just as cervical collars, lumbar supports can also come in a flexible or rigid form and are often employed especially in the workplace to prevent low back injuries. They can be ordered in custom or off-the-shelf format, and worn all day or part of the day. There are several goals lumbar support aims to achieve, which includes the following: limit movement of the spine, provide external support, correct deformities, improve stabilization, and provide compression or massaging effects [103]. Additionally, bracing potentially can be a psychological support and provide proprioceptive feedback for an individual, cautioning against certain painful movements. There is a concern that bracing could result in core muscle weakness from disuse or conversely can help relieve muscle tension when worn; however, these hypotheses were not supported when the electromyographic of the erector spinae muscles activity did not decrease while wearing a lumbar brace [104].

Generally, for acute to subacute low back pain, there is poor evidence indicating improvement in pain or function with the addition of lumbar support [105]. When reviewing participants in multiple studies, 550 people said it did not help, but 410 individuals felt that lumbar bracing was more helpful than no intervention. There is no clear conclusion whether or not lumbar bracing helps. Such a decision should be decided upon between the patient and provider on a case-by-case basis. In regards to prevention of low back pain, seven randomized control trials indicated that there is moderate evidence that the lumbar support does not help reduce low back pain and has not been shown to be more effective in preventing low back pain as compared to education in proper lifting techniques. Another study focused on industry workers at high risk for mechanical low back pain. These assembly line workers often stand while performing repetitive movements and lifting movements with their arms. For these industry workers, no benefit was shown versus placebo in terms of low back pain level when wearing a soft lumbar brace versus placebo [106]. A Cochrane review indicated that the effectiveness in treatment of low back pain indicated limited evidence that lumbar supports can reduce pain and increase functionality but no more than if no intervention was taken [107].

The cost for back braces varies widely from $40 to $1000 depending on the material, shape, features, adjustability, customized or prefabricated. Insurance plans may or may not cover braces and if covered the typical out-of-pocket cost is a copay of 20% (reference).

Acupuncture

The word “acupuncture” originates from Latin with the root word puncture meaning to needle and prefix acus meaning needle. Modern acupuncture is generally thought to have originated in China though the exact date is unknown. Publications of textbooks discussing the manipulation of qi within one’s body by means of needling were recovered dating back to the 5th and 8th centuries AD [108]. In Eastern

medicine, qi is thought to be a life force or energy that flows within one's body. The actual Chinese word for acupuncture is *Zhenjiu* which actually includes various techniques in addition to needles such as electro-acupuncture, laser acupuncture, microsystem acupuncture, acupressure and moxibustion [109]. By definition, dry needling, which is often incorporated in physical and occupational therapy, can also be considered as acupuncture though not often described as such.

In a meta-analysis of 75 randomized control trials, findings noted there were reductions in pain amongst chronic low back pain sufferers immediately as well as short term relief. Immediate relief in back pain was noted for acute low back pain. For chronic neck pain, when compared to sham acupuncture (where the needle was punctured superficially) there was both reduction in pain and disability immediately after treatment as well as one-month after treatment [37]. In another meta-analysis looking only at high quality trials at an individual patient level indicated that patients who underwent acupuncture experienced less pain as compared to sham controls for back and neck pain [110]. In terms of functionality, a large trial in Germany with 3093 patients suffering from an average of 7 years of low back pain were either assigned to acupuncture or no acupuncture. Those in the acupuncture group showed a significant difference in functionality improvement than no acupuncture [111].

Practitioners who are licensed to practice typically have secured a diploma from the National Certification Commission for Acupuncture and Oriental Medicine. Physicians can also perform acupuncture once they've progressed through a medical acupuncture program.

Needles can be placed along traditional acupuncture points or along trigger spots. Once inserted, the needles can be manipulated by twisting, adjusting the depth, connected to electrical current to perform electroacupuncture or moxibustion by burning the herb artemisia vulgaris at the end of the acupuncture needle.

There are minimal reactions and toxic side effects [109]. Contraindications include clotting, bleeding disorders, anticoagulant usage, local skin infections, and significant psychiatric conditions. Minor adverse events include pain near the needle insertion point, nausea, vomiting, fainting and dizziness. Over more than 760,000 sessions there were 2 cases of pneumothorax where one was treated conservatively and one required a drain [112]. When patients have exhausted traditional treatments for back and neck pain, acupuncture can potentially be a relatively safe and effective method for pain relief. Sessions are typically twice a week for several months. After initial treatments, occasional sessions can be continued every month or every other month. If no improvements are noted in 10 to 12 sessions then acupuncture should be discontinued. The cost is generally $65 to $125 per session [113].

Psychological Treatments and Therapies

Pain is defined by Webster dictionary as an unpleasant or distressing sensation that can be perceived as a prick, tingle burning, aching, or stinging from a part of one's body. Even if the external inciting factor for pain is the same, the actual perception of pain will differ between individuals as each individual has a unique past

experience, comorbidities, body composition, tolerance, coping mechanism etc. Multiple studies have noted the connection between the evolvement of chronic pain and one's prior, current and future psychological composition [114–119]. The psyche of an individual has a profound effect on why for some people the pain is tolerable and for others completely disabling, interfering with their daily activities and inhibiting them from being able to work. Some would even argue that the a bigger component towards the attenuation of chronic back pain is psychological rather than physical [114].

Regarding psychopathology prior to chronic pain, multiple studies have shown higher tendencies for acute to chronic back and neck pain conversions if one does suffer from psychiatric disorders [114–116]. A study indicated that for females that suffered from physical abuse had an increased risk of reporting significant back and neck pain by five-fold and those experiencing sexual abuse noted an increased risk by four-fold [116]. A prior history of social dysfunction (bullying) or social isolation could also result in cross sensitization to regions that perceive pain. Specific brain regions (anterior cingulate cortex) show activation during social distress [117]. This area is similarly activated when experimental pain is inflicted thus potentially resulting in greater pain exacerbation due to cross sensitization [120–122]. Psychopathology that is often associated with chronic low back pain include depression, somatoform disorders, anxiety, substance abuse, and personality disorders. A study comparing chronic low back pain versus acute low back pain sufferers indicated that within the first group, 90% met diagnostic criteria for somatoform pain disorder, 46% met criteria for major depression, and 40% suffered from substance abuse disorder compared to the latter group with only 8% suffering from major depression and 38% suffering from substance abuse disorder [115]. The authors also cite that job loss is often associated with chronic low back pain, which could predispose the individuals to development/worsening of premorbid psychopathology [115].

Regarding psychopathology that develops after chronic low back pain begins, multiple studies indicate that changes to the brain's neurologic pathway occurs in response to such a chronic stressor. The process in which an organism strives to maintain homeostasis when faced with stressors is defined as allostasis. The cost in which the organism has to expend to maintain homeostasis is defined as allostatic load [118]. Generally, pain's function is to guide an organism to move towards positive adaptive behaviors. Essentially, pain directs an organism to avoid the noxious stimuli but when pain becomes chronic, the individual starts to accumulate an allostatic load which results in cumulative and potentially maladaptive changes to the brain and therefore behavior. Such maladaptive changes may result in altered perceptions including hyperalgesia, allodynia, etc. and centralization of pain and therefore avoidance of movement, social withdrawal, emotional lability, decreased productivity and preference towards potentially self-destructive behavior such as substance abuse. Several neural pathways that may be altered include the dorsolateral prefrontal cortex, ventrolateral prefrontal cortex, medial prefrontal cortex which collectively appear to reduce abilities to modulate emotional states [119–121]. Acute pain tends to affect the mesolimbic reward-related pathway and in

contrast to chronic pain, there is less evidence that cognition is significantly altered in individuals suffering from acute pain [122].

Which occurs first? Is the pain causative of the psychopathology and maladaptive behaviors or is the psychopathology causative of the development of chronic low back pain? Some studies have indicated that the depression occurred after the pain supporting what is known as the "consequence hypothesis." Fishbain et al. however also noted that there were conversely a few studies that supported the theory that depression preceded the onset of pain [123]. Polatin et al. reviewed chronic low back pain in relations to depression in 200 patients and noted that 55% of individuals with major depressive disorder developed before the onset of chronic pain and 45% of individuals became depressed after the onset of chronic pain. Essentially, both scenarios are viable possibilities. Screening for depression in those suffering from both acute or chronic low back pain is therefore an important factor to drive an effective plan of care especially since the relative risk of impairments in activities of daily living was 1.67 times higher in those suffering from depression in elderly patients. Mobility impairments for elderly suffering from chronic back pain concomitant with depression was 7.73 times higher [124]. Addressing underlying depression and an individual's social factors could be significantly beneficial to being able to successfully treat chronic low back pain. The concept of identifying "psychosocial flags" which could affect perception of back pain include acknowledging the individuals' ABCDEFW – attitude (positive or catastrophizing), behavior changes, compensation (in terms of pending legal case, monetary gains or financial issues), diagnosis, emotions (psychological history and current response to symptoms), family (support or lack thereof) and work [125].

Knowing this connection between mind and body, one should be cognizant to first focus on preventing the transition from acute to chronic back/neck pain but if chronic neck/back pain does occur, understand how to address the psychological component. Most commonly utilized psychosocial interventions include cognitive behavioral treatment, hypnosis, biofeedback, and mindfulness. Cognitive behavioral therapy focuses on increasing awareness of inaccurate or negative thoughts. After identifying these distorted thoughts, the individual is challenged to reshape and reformulate the thoughts into a more positive thought. Biofeedback works on helping an individual learn how to control one's body when stress is present. Sensors may be used to monitor an individuals' brain waves, respiratory rate, heart rate, tension in muscle, sweat activity, and temperature. An individual is trained to try to modulate their bodies into a more relaxed state thereby controlling their physical response in the face of stressors [126]. Mindfulness encourages individual to intensely focus on being acutely aware of the present and also incorporates guided imagery and envisioning relaxation of the body and mind [127].

How effective are these different psychological treatments? A review looked at 23 studies with a total of 3359 participants with trials utilizing varying types of cognitive behavioral methodology. The studies showed decreased disability and pain for the individuals participating in some form of cognitive behavioral treatment as compared to no treatment at long-term follow up. Another study with 342 participants comparing individuals suffering from chronic low back pain were allocated

either to meditation and cognitive behavioral therapy or usual treatment for chronic low back pain. Results indicated that those receiving cognitive behavioral therapy were able to experience decreased daily pain and decreased physical disability [128]. Another systematic review on 400 articles indicated that psychological interventions were safe and effective treatments for those suffering from pain [129]. Just as chronic pain resulted in alterations to the neurological connections in the brain, so does cognitive behavioral therapy. fMRI scans showing cognitive behavioral therapy associated with increased activity within the ventrolateral prefrontal/lateral orbitofrontal cortex and increased executive cognitive control [121]. Often, time limitations would dissuade individuals from pursuing psychological interventions. Fortunately, another study showed that even a shorter duration (4 week) session has demonstrated benefit in back pain symptoms and frontal lobe regulation when compared to the control group which was allocated to stress reduction reading [130].

Increased ability for an individual to be able to meditate could also address other issues that could be potentially exacerbating or could have occurred as a result of chronic back pain such as sleep, depression, anxiety and stress reactivity [131]. Not only has psychological treatments been shown to reduce chronic pain and decrease disability, these interventions have also been safe without the toxic side effects seen in common treatments for chronic back pain such as the commonly employed usage of opioids. Opioids could result in constipation, itching, risk for addiction, respiratory depression and death, which are side effects not seen with psychological interventions.

The cost of psychological therapy can range significantly from a range of $60 to $200 sessions and even with a short duration of treatment. Coverage for this beneficial treatment from health insurances have unfortunately been on the decline.

References

1. Qaseem A, Wilt TJ, McLean RM, Forciea MA, Clinical Guidelines Committee of the American College of P. Noninvasive treatments for acute, subacute, and chronic low back pain: a clinical practice guideline from the American College of Physicians. Ann Intern Med. 2017;166(7):514–30. https://doi.org/10.7326/M16-2367 [published Online First: Epub Date]|.
2. Wiesel SW, Cuckler JM, Deluca F, Jones F, Zeide MS, Rothman RH. Acute low-back pain. An objective analysis of conservative therapy. Spine (Phila Pa 1976). 1980;5(4):324–30. https://doi.org/10.1097/00007632-198007000-00006 [published Online First: Epub Date]|.
3. Foster NE, Anema JR, Cherkin D, et al. Prevention and treatment of low back pain: evidence, challenges, and promising directions. Lancet. 2018;391(10137):2368–83. https://doi.org/10.1016/S0140-6736(18)30489-6 [published Online First: Epub Date]|.
4. Mork PJ, Bach K, self BC. A decision support system to enhance self-management of low back pain: protocol for the selfBACK Project. JMIR Res Protoc. 2018;7(7):e167 https://doi.org/10.2196/resprot.9379 [published Online First: Epub Date]|.
5. Childs JD, Cleland JA, Elliott JM, et al. Neck pain: clinical practice guidelines linked to the International Classification of Functioning, Disability, and Health from the Orthopedic Section of the American Physical Therapy Association. J Orthop Sports Phys Ther. 2008;38(9):A1-A34. https://doi.org/10.2519/jospt.2008.0303 [published Online First: Epub Date]|.

6. Gross A, Kay TM, Paquin JP, et al. Exercises for mechanical neck disorders. Cochrane Database Syst Rev. 2015;1:CD004250. https://doi.org/10.1002/14651858.CD004250.pub5 [published Online First: Epub Date]|.
7. Southerst D, Nordin MC, Cote P, et al. Is exercise effective for the management of neck pain and associated disorders or whiplash-associated disorders? A systematic review by the Ontario Protocol for Traffic Injury Management (OPTIMa) Collaboration. Spine J. 2016;16(12):1503–23. https://doi.org/10.1016/j.spinee.2014.02.014 [published Online First: Epub Date]|.
8. Bronfort G, Evans R, Anderson AV, Svendsen KH, Bracha Y, Grimm RH. Spinal manipulation, medication, or home exercise with advice for acute and subacute neck pain: a randomized trial. Ann Intern Med. 2012;156(1 Pt 1):1–10. https://doi.org/10.7326/0003-4819-156-1-201201030-00002 [published Online First: Epub Date]|.
9. Kuijper B, Tans JT, Beelen A, Nollet F, de Visser M. Cervical collar or physiotherapy versus wait and see policy for recent onset cervical radiculopathy: randomised trial. BMJ. 2009;339:b3883. https://doi.org/10.1136/bmj.b3883 [published Online First: Epub Date]|.
10. Blanpied PR, Gross AR, Elliott JM, et al. Neck pain: revision 2017. J Orthop Sports Phys Ther. 2017;47(7):A1-A83. https://doi.org/10.2519/jospt.2017.0302 [published Online First: Epub Date]|.
11. Sihawong R, Janwantanakul P, Sitthipornvorakul E, Pensri P. Exercise therapy for office workers with nonspecific neck pain: a systematic review. J Manipulative Physiol Ther. 2011;34(1):62–71. https://doi.org/10.1016/j.jmpt.2010.11.005 [published Online First: Epub Date]|.
12. Kamper SJ, Yamato TP, Williams CM. The prevalence, risk factors, prognosis and treatment for back pain in children and adolescents: an overview of systematic reviews. Best Pract Res Clin Rheumatol. 2016;30(6):1021–36. https://doi.org/10.1016/j.berh.2017.04.003 [published Online First: Epub Date]|.
13. Ferreira ML, Smeets RJ, Kamper SJ, Ferreira PH, Machado LA. Can we explain heterogeneity among randomized clinical trials of exercise for chronic back pain? A meta-regression analysis of randomized controlled trials. Phys Ther. 2010;90(10):1383–403. https://doi.org/10.2522/ptj.20090332 [published Online First: Epub Date]|.
14. O'Connor SR, Tully MA, Ryan B, et al. Walking exercise for chronic musculoskeletal pain: systematic review and meta-analysis. Arch Phys Med Rehabil. 2015;96(4):724–34 e3. https://doi.org/10.1016/j.apmr.2014.12.003 [published Online First: Epub Date]|.
15. Vanti C, Andreatta S, Borghi S, Guccione AA, Pillastrini P, Bertozzi L. The effectiveness of walking versus exercise on pain and function in chronic low back pain: a systematic review and meta-analysis of randomized trials. Disabil Rehabil. 2019;41(6):622–32. https://doi.org/10.1080/09638288.2017.1410730 [published Online First: Epub Date]|.
16. Meng XG, Yue SW. Efficacy of aerobic exercise for treatment of chronic low back pain: a meta-analysis. Am J Phys Med Rehabil. 2015;94(5):358–65. https://doi.org/10.1097/PHM.0000000000000188 [published Online First: Epub Date]|.
17. Chou R, Cote P, Randhawa K, et al. The Global Spine Care Initiative: applying evidence-based guidelines on the non-invasive management of back and neck pain to low- and middle-income communities. Eur Spine J. 2018;27(Suppl 6):851–60. https://doi.org/10.1007/s00586-017-5433-8 [published Online First: Epub Date]|.
18. Rendant D, Pach D, Ludtke R, et al. Qigong versus exercise versus no therapy for patients with chronic neck pain: a randomized controlled trial. Spine (Phila Pa 1976). 2011;36(6):419–27. https://doi.org/10.1097/BRS.0b013e3181d51fca [published Online First: Epub Date]|.
19. Shipton EA. Physical therapy approaches in the treatment of low back pain. Pain Ther. 2018;7(2):127–37. https://doi.org/10.1007/s40122-018-0105-x [published Online First: Epub Date]|.
20. Steffens D, Maher CG, Pereira LS, et al. Prevention of low back pain: a systematic review and meta-analysis. JAMA Intern Med. 2016;176(2):199–208. https://doi.org/10.1001/jamainternmed.2015.7431 [published Online First: Epub Date]|.

21. Shiri R, Coggon D, Falah-Hassani K. Exercise for the prevention of low back pain: systematic review and meta-analysis of controlled trials. Am J Epidemiol. 2018;187(5):1093–101. https://doi.org/10.1093/aje/kwx337 [published Online First: Epub Date]|.
22. Choi BK, Verbeek JH, Tam WW, Jiang JY. Exercises for prevention of recurrences of low-back pain. Occup Environ Med. 2010;67(11):795–6. https://doi.org/10.1136/oem.2010.059873 [published Online First: Epub Date]|.
23. de Campos TF, Maher CG, Steffens D, Fuller JT, Hancock MJ. Exercise programs may be effective in preventing a new episode of neck pain: a systematic review and meta-analysis. J Physiother. 2018;64(3):159–65. https://doi.org/10.1016/j.jphys.2018.05.003 [published Online First: Epub Date]|.
24. Sitthipornvorakul E, Sihawong R, Waongenngarm P, Janwantanakul P. The effects of walking intervention on preventing neck pain in office workers: a randomized controlled trial. J Occup Health. 2020;62(1):e12106. https://doi.org/10.1002/1348-9585.12106 [published Online First: Epub Date]|.
25. Ploughman M. Exercise is brain food: the effects of physical activity on cognitive function. Dev Neurorehabil. 2008;11(3):236–40. https://doi.org/10.1080/17518420801997007 [published Online First: Epub Date]|.
26. Geneen LJ, Moore RA, Clarke C, Martin D, Colvin LA, Smith BH. Physical activity and exercise for chronic pain in adults: an overview of Cochrane Reviews. Cochrane Database Syst Rev. 2017;4:CD011279. https://doi.org/10.1002/14651858.CD011279.pub3 [published Online First: Epub Date]|.
27. Walking: a step in the right direction. Secondary walking: a step in the right direction. (4) https://www.niddk.nih.gov/health-information/weight-management/walking-step-right-direction.
28. Starting a walking program. Secondary starting a walking program. https://www.prescriptiontogetactive.com/static/pdfs/starting-a-walking-program-ACSM.pdf.
29. Pourahmadi M, Hesarikia H, Keshtkar A, et al. Effectiveness of slump stretching on low back pain: a systematic review and meta-analysis. Pain Med. 2019;20(2):378–96. https://doi.org/10.1093/pm/pny208 [published Online First: Epub Date]|.
30. History of Pilates. Secondary History of Pilates 2020. https://www.pilatesfoundation.com/pilates/the-history-of-pilates/.
31. Byrnes K, Wu PJ, Whillier S. Is Pilates an effective rehabilitation tool? A systematic review. J Bodyw Mov Ther. 2018;22(1):192–202. https://doi.org/10.1016/j.jbmt.2017.04.008 [published Online First: Epub Date]|.
32. Patti A, Bianco A, Paoli A, et al. Effects of Pilates exercise programs in people with chronic low back pain: a systematic review. Medicine (Baltimore). 2015;94(4):e383. https://doi.org/10.1097/MD.0000000000000383 [published Online First: Epub Date]|.
33. Wieland LS, Santesso N. A summary of a Cochrane review: yoga treatment for chronic non-specific low back pain. Eur J Integr Med. 2017;11:39–40. https://doi.org/10.1016/j.eujim.2017.03.003 [published Online First: Epub Date]|.
34. Li Y, Li S, Jiang J, Yuan S. Effects of yoga on patients with chronic nonspecific neck pain: a PRISMA systematic review and meta-analysis. Medicine (Baltimore). 2019;98(8):e14649. https://doi.org/10.1097/MD.0000000000014649 [published Online First: Epub Date]|.
35. Lomas-Vega R, Obrero-Gaitan E, Molina-Ortega FJ, Del-Pino-Casado R. Tai Chi for risk of falls. A meta-analysis. J Am Geriatr Soc. 2017;65(9):2037–43. https://doi.org/10.1111/jgs.15008 [published Online First: Epub Date]|.
36. Li F, Harmer P, Fitzgerald K, et al. Effectiveness of a therapeutic Tai Ji Quan intervention vs a multimodal exercise intervention to prevent falls among older adults at high risk of falling: a randomized clinical trial. JAMA Intern Med. 2018;178(10):1301–10. https://doi.org/10.1001/jamainternmed.2018.3915 [published Online First: Epub Date]|.
37. Yuan QL, Guo TM, Liu L, Sun F, Zhang YG. Traditional Chinese medicine for neck pain and low back pain: a systematic review and meta-analysis. PLoS One. 2015;10(2):e0117146. https://doi.org/10.1371/journal.pone.0117146 [published Online First: Epub Date]|.
38. Chou R, Deyo R, Friedly J, et al. Nonpharmacologic therapies for low back pain: a systematic review for an American College of Physicians Clinical Practice Guideline. Ann Intern

Med. 2017;166(7):493–505. https://doi.org/10.7326/M16-2459 [published Online First: Epub Date]|.
39. Hall AM, Maher CG, Lam P, Ferreira M, Latimer J. Tai chi exercise for treatment of pain and disability in people with persistent low back pain: a randomized controlled trial. Arthritis Care Res (Hoboken). 2011;63(11):1576–83. https://doi.org/10.1002/acr.20594 [published Online First: Epub Date]|.
40. Lauche R, Stumpe C, Fehr J, et al. The effects of Tai Chi and neck exercises in the treatment of chronic nonspecific neck pain: a randomized controlled trial. J Pain. 2016;17(9):1013–27. https://doi.org/10.1016/j.jpain.2016.06.004 [published Online First: Epub Date]|.
41. Jahnke R, Larkey L, Rogers C, Etnier J, Lin F. A comprehensive review of health benefits of qigong and tai chi. Am J Health Promot. 2010;24(6):e1-e25. https://doi.org/10.4278/ajhp.081013-LIT-248 [published Online First: Epub Date]|.
42. von Trott P, Wiedemann AM, Ludtke R, Reishauer A, Willich SN, Witt CM. Qigong and exercise therapy for elderly patients with chronic neck pain (QIBANE): a randomized controlled study. J Pain. 2009;10(5):501–8. https://doi.org/10.1016/j.jpain.2008.11.004 [published Online First: Epub Date]|.
43. "Physical Therapy". Merriam Webster.
44. Shaik A, Shemjaz A. The rise of physical therapy: a history in footsteps. Arch Med Health Sci. 2014;2(2):257–60. https://doi.org/10.4103/2321-4848.144367 [published Online First: Epub Date]|.
45. Korobov SA. Towards the origin of the term physiotherapy: Dr Edward Playter's contribution of 1894. Physiother Res Int. 2005;10(3):123–4. https://doi.org/10.1002/pri.1 [published Online First: Epub Date]|.
46. Association APT. APTA Workforce Data: Licensed PTs by State 2019. Dec 31, 2019 ed, 2019.
47. (ASH) ASfH. Report on pain management best practices: updates, gaps, inconsistencies, and recommendations. Secondary report on pain management best practices: updates, gaps, inconsistencies, and recommendations Dec 6, 2019 2019. https://www.hhs.gov/ash/advisory-committees/pain/reports/index.html.
48. The McKenzie Method. Secondary The McKenzie Method. https://mckenzieinstitute.org/about-us/the-legend-of-the-mckenzie-method/.
49. May S, Donelson R. Evidence-informed management of chronic low back pain with the McKenzie method. Spine J. 2008;8(1):134–41. https://doi.org/10.1016/j.spinee.2007.10.017 [published Online First: Epub Date]|.
50. Petersen T, Larsen K, Nordsteen J, Olsen S, Fournier G, Jacobsen S. The McKenzie method compared with manipulation when used adjunctive to information and advice in low back pain patients presenting with centralization or peripheralization: a randomized controlled trial. Spine (Phila Pa 1976). 2011;36(24):1999–2010. https://doi.org/10.1097/BRS.0b013e318201ee8e [published Online First: Epub Date]|.
51. Lam OT, Strenger DM, Chan-Fee M, Pham PT, Preuss RA, Robbins SM. Effectiveness of the McKenzie method of mechanical diagnosis and therapy for treating low back pain: literature review with meta-analysis. J Orthop Sports Phys Ther. 2018;48(6):476–90. https://doi.org/10.2519/jospt.2018.7562 [published Online First: Epub Date]|.
52. May S, Aina A. Centralization and directional preference: a systematic review. Man Ther. 2012;17(6):497–506. https://doi.org/10.1016/j.math.2012.05.003 [published Online First: Epub Date]|.
53. Namnaqani FI, Mashabi AS, Yaseen KM, Alshehri MA. The effectiveness of McKenzie method compared to manual therapy for treating chronic low back pain: a systematic review. J Musculoskelet Neuronal Interact. 2019;19(4):492–9.
54. Clare HA, Adams R, Maher CG. A systematic review of efficacy of McKenzie therapy for spinal pain. Aust J Physiother. 2004;50(4):209–16. https://doi.org/10.1016/s0004-9514(14)60110-0 [published Online First: Epub Date]|.
55. Garcia AN, Costa L, de Souza FS, et al. Reliability of the mechanical diagnosis and therapy system in patients with spinal pain: a systematic review. J Orthop Sports Phys Ther.

2018;48(12):923–33. https://doi.org/10.2519/jospt.2018.7876 [published Online First: Epub Date]|.
56. O'Sullivan PB, Phyty GD, Twomey LT, Allison GT. Evaluation of specific stabilizing exercise in the treatment of chronic low back pain with radiologic diagnosis of spondylolysis or spondylolisthesis. Spine (Phila Pa 1976). 1997;22(24):2959–67. https://doi.org/10.1097/00007632-199712150-00020 [published Online First: Epub Date]|.
57. Smart KM, Blake C, Staines A, Thacker M, Doody C. Mechanisms-based classifications of musculoskeletal pain: part 3 of 3: symptoms and signs of nociceptive pain in patients with low back (+/− leg) pain. Man Ther. 2012;17(4):352–7. https://doi.org/10.1016/j.math.2012.03.002 [published Online First: Epub Date]|.
58. Luomajoki HA, Bonet Beltran MB, Careddu S, Bauer CM. Effectiveness of movement control exercise on patients with non-specific low back pain and movement control impairment: a systematic review and meta-analysis. Musculoskelet Sci Pract. 2018;36:1–11. https://doi.org/10.1016/j.msksp.2018.03.008 [published Online First: Epub Date]|.
59. Aasa B, Berglund L, Michaelson P, Aasa U. Individualized low-load motor control exercises and education versus a high-load lifting exercise and education to improve activity, pain intensity, and physical performance in patients with low back pain: a randomized controlled trial. J Orthop Sports Phys Ther. 2015;45(2):77–85, B1–4. https://doi.org/10.2519/jospt.2015.5021 [published Online First: Epub Date]|.
60. Carol Hartigan M, Kevin K, Bernard MD. Exercise-based therapy for low back pain. In: Steven J, Atlas M, MPH, editors. UpToDate; 2019.
61. Saragiotto BT, Maher CG, Yamato TP, et al. Motor control exercise for chronic non-specific low-back pain. Cochrane Database Syst Rev. 2016(1):CD012004. https://doi.org/10.1002/14651858.CD012004 [published Online First: Epub Date]|.
62. Dydyk AM, Sapra A. Williams Back Exercises. Treasure Island (FL): StatPearls; 2020.
63. Ponte DJ, Jensen GJ, Kent BE. A Preliminary report on the use of the McKenzie protocol versus Williams protocol in the treatment of low back pain. J Orthop Sports Phys Ther. 1984;6(2):130–9. https://doi.org/10.2519/jospt.1984.6.2.130 [published Online First: Epub Date]|.
64. Elnaggar IM, Nordin M, Sheikhzadeh A, Parnianpour M, Kahanovitz N. Effects of spinal flexion and extension exercises on low-back pain and spinal mobility in chronic mechanical low-back pain patients. Spine (Phila Pa 1976). 1991;16(8):967–72. https://doi.org/10.1097/00007632-199108000-00018 [published Online First: Epub Date]|.
65. Dettori JR, Bullock SH, Sutlive TG, Franklin RJ, Patience T. The effects of spinal flexion and extension exercises and their associated postures in patients with acute low back pain. Spine (Phila Pa 1976). 1995;20(21):2303–12. https://doi.org/10.1097/00007632-199511000-00008 [published Online First: Epub Date]|.
66. Straube S, Harden M, Schroder H, et al. Back schools for the treatment of chronic low back pain: possibility of benefit but no convincing evidence after 47 years of research-systematic review and meta-analysis. Pain. 2016;157(10):2160–72 https://doi.org/10.1097/j.pain.0000000000000640 [published Online First: Epub Date]|.
67. Heymans MW, van Tulder MW, Esmail R, Bombardier C, Koes BW. Back schools for non-specific low-back pain. Cochrane Database Syst Rev. 2004(4):CD000261. https://doi.org/10.1002/14651858.CD000261.pub2 [published Online First: Epub Date]|.
68. Parreira P, Heymans MW, van Tulder MW, et al. Back Schools for chronic non-specific low back pain. Cochrane Database Syst Rev. 2017;8:CD011674. https://doi.org/10.1002/14651858.CD011674.pub2 [published Online First: Epub Date]|.
69. Bellew JW. Therapeutic modalities past, present, and future: their role in the patient care management model. In: Bellew JW, Michlovitz SL, Nolan Jr TP, editors. Modalities for therapeutic intervention. 6th ed. New York: McGraw-Hill Education; 2016.
70. Gibbons P, Tehan P. Patient positioning and spinal locking for lumbar spine rotation manipulation. Man Ther. 2001;6(3):130–8 https://doi.org/10.1054/math.2001.0404 [published Online First: Epub Date]|.

71. Pickar JG. Neurophysiological effects of spinal manipulation. Spine J. 2002;2(5):357–71. https://doi.org/10.1016/s1529-9430(02)00400-x [published Online First: Epub Date]|.
72. Gross A, Langevin P, Burnie SJ, et al. Manipulation and mobilisation for neck pain contrasted against an inactive control or another active treatment. Cochrane Database Syst Rev. 2015(9):CD004249. https://doi.org/10.1002/14651858.CD004249.pub4 [published Online First: Epub Date]|.
73. Gross AR, Kay TM, Kennedy C, et al. Clinical practice guideline on the use of manipulation or mobilization in the treatment of adults with mechanical neck disorders. Man Ther. 2002;7(4):193–205. https://doi.org/10.1054/math.2002.0477 [published Online First: Epub Date]|.
74. Rubinstein SM, Terwee CB, Assendelft WJ, de Boer MR, van Tulder MW. Spinal manipulative therapy for acute low back pain: an update of the cochrane review. Spine (Phila Pa 1976). 2013;38(3):E158–77. https://doi.org/10.1097/BRS.0b013e31827dd89d [published Online First: Epub Date]|.
75. Rubinstein SM, van Middelkoop M, Assendelft WJ, de Boer MR, van Tulder MW. Spinal manipulative therapy for chronic low-back pain: an update of a Cochrane review. Spine (Phila Pa 1976). 2011;36(13):E825–46. https://doi.org/10.1097/BRS.0b013e3182197fe1 [published Online First: Epub Date]|.
76. Coulter ID, Crawford C, Hurwitz EL, et al. Manipulation and mobilization for treating chronic low back pain: a systematic review and meta-analysis. Spine J. 2018;18(5):866–79. https://doi.org/10.1016/j.spinee.2018.01.013 [published Online First: Epub Date]|.
77. Brent A, Bauer M. What is reflexology? Can it relieve stress?: Mayo Clinic, 2020.
78. Furlan AD, Giraldo M, Baskwill A, Irvin E, Imamura M. Massage for low-back pain. Cochrane Database Syst Rev. 2015(9):CD001929. https://doi.org/10.1002/14651858.CD001929.pub3 [published Online First: Epub Date]|.
79. Patel KC, Gross A, Graham N, et al. Massage for mechanical neck disorders. Cochrane Database Syst Rev. 2012(9):CD004871. https://doi.org/10.1002/14651858.CD004871.pub4 [published Online First: Epub Date]|.
80. Association APT. Biophysical agents. Secondary biophysical agents December 5, 2020 2020. http://guidetoptpractice.apta.org/content/1/SEC35.extract.
81. Algafly AA, George KP. The effect of cryotherapy on nerve conduction velocity, pain threshold and pain tolerance. Br J Sports Med. 2007;41(6):365–9; discussion 69. https://doi.org/10.1136/bjsm.2006.031237 [published Online First: Epub Date]|.
82. French SD, Cameron M, Walker BF, Reggars JW, Esterman AJ. A Cochrane review of superficial heat or cold for low back pain. Spine (Phila Pa 1976). 2006;31(9):998–1006. https://doi.org/10.1097/01.brs.0000214881.10814.64 [published Online First: Epub Date]|.
83. Nadler SF, Steiner DJ, Erasala GN, et al. Continuous low-level heat wrap therapy provides more efficacy than Ibuprofen and acetaminophen for acute low back pain. Spine (Phila Pa 1976). 2002;27(10):1012–7. https://doi.org/10.1097/00007632-200205150-00003 [published Online First: Epub Date]|.
84. Mayer JM, Ralph L, Look M, et al. Treating acute low back pain with continuous low-level heat wrap therapy and/or exercise: a randomized controlled trial. Spine J. 2005;5(4):395–403. https://doi.org/10.1016/j.spinee.2005.03.009 [published Online First: Epub Date]|.
85. Brandom RL. Physical medicine and rehabilitation. 3rd ed. Saunders Elsevier; 2007.
86. Hsueh TC, Cheng PT, Kuan TS, Hong CZ. The immediate effectiveness of electrical nerve stimulation and electrical muscle stimulation on myofascial trigger points. Am J Phys Med Rehabil. 1997;76(6):471–6. https://doi.org/10.1097/00002060-199711000-00007 [published Online First: Epub Date]|.
87. Resende L, Merriwether E, Rampazo EP, et al. Meta-analysis of transcutaneous electrical nerve stimulation for relief of spinal pain. Eur J Pain. 2018;22(4):663–78. https://doi.org/10.1002/ejp.1168 [published Online First: Epub Date]|.
88. Wu LC, Weng PW, Chen CH, Huang YY, Tsuang YH, Chiang CJ. Literature review and meta-analysis of transcutaneous electrical nerve stimulation in treating chronic back pain.

Reg Anesth Pain Med. 2018;43(4):425–33. https://doi.org/10.1097/AAP.0000000000000740 [published Online First: Epub Date]|.
89. Moore SR, Shurman J. Combined neuromuscular electrical stimulation and transcutaneous electrical nerve stimulation for treatment of chronic back pain: a double-blind, repeated measures comparison. Arch Phys Med Rehabil. 1997;78(1):55–60. https://doi.org/10.1016/s0003-9993(97)90010-1 [published Online First: Epub Date]|.
90. Graham N, Gross AR, Carlesso LC, et al. An ICON overview on physical modalities for neck pain and associated disorders. Open Orthop J. 2013;7:440–60. https://doi.org/10.2174/1874325001307010440 [published Online First: Epub Date]|.
91. Binny J, Joshua Wong NL, Garga S, et al. Transcutaneous electric nerve stimulation (TENS) for acute low back pain: systematic review. Scand J Pain. 2019;19(2):225–33. https://doi.org/10.1515/sjpain-2018-0124 [published Online First: Epub Date]|.
92. Bellew JW, Michlovitz SL, Nolan T. Modalities for therapeutic intervention. F.A. Davis Company; 2016.
93. Wegner I, Widyahening IS, van Tulder MW, et al. Traction for low-back pain with or without sciatica. Cochrane Database Syst Rev. 2013(8):CD003010. https://doi.org/10.1002/14651858.CD003010.pub5 [published Online First: Epub Date]|.
94. Binder AI. Neck pain. BMJ Clin Evid. 2008;2008**.**
95. Cotler HB, Chow RT, Hamblin MR, Carroll J. The use of low level laser therapy (LLLT) For musculoskeletal pain. MOJ Orthop Rheumatol. 2015;2(5). https://doi.org/10.15406/mojor.2015.02.00068 [published Online First: Epub Date]|.
96. Waylonis GW, Wilke S, O'Toole D, Waylonis DA, Waylonis DB. Chronic myofascial pain: management by low-output helium-neon laser therapy. Arch Phys Med Rehabil. 1988;69(12):1017–20.
97. Konstantinovic LM, Cutovic MR, Milovanovic AN, et al. Low-level laser therapy for acute neck pain with radiculopathy: a double-blind placebo-controlled randomized study. Pain Med. 2010;11(8):1169–78. https://doi.org/10.1111/j.1526-4637.2010.00907.x [published Online First: Epub Date]|.
98. Ebadi S, Henschke N, Forogh B, et al. Therapeutic ultrasound for chronic low back pain. Cochrane Database Syst Rev. 2020;7:CD009169. https://doi.org/10.1002/14651858.CD009169.pub3 [published Online First: Epub Date]|.
99. Seco J, Kovacs FM, Urrutia G. The efficacy, safety, effectiveness, and cost-effectiveness of ultrasound and shock wave therapies for low back pain: a systematic review. Spine J. 2011;11(10):966–77. https://doi.org/10.1016/j.spinee.2011.02.002 [published Online First: Epub Date]|.
100. Gennis P, Miller L, Gallagher EJ, Giglio J, Carter W, Nathanson N. The effect of soft cervical collars on persistent neck pain in patients with whiplash injury. Acad Emerg Med. 1996;3(6):568–73. https://doi.org/10.1111/j.1553-2712.1996.tb03466.x [published Online First: Epub Date]|.
101. Rosenfeld M, Gunnarsson R, Borenstein P. Early intervention in whiplash-associated disorders: a comparison of two treatment protocols. Spine (Phila Pa 1976). 2000;25(14):1782–7. https://doi.org/10.1097/00007632-200007150-00008 [published Online First: Epub Date]|.
102. Verhagen AP, Scholten-Peeters GG, van Wijngaarden S, de Bie RA, Bierma-Zeinstra SM. Conservative treatments for whiplash. Cochrane Database Syst Rev. 2007(2):CD003338. https://doi.org/10.1002/14651858.CD003338.pub3 [published Online First: Epub Date]|.
103. Zarghooni K, Beyer F, Siewe J, Eysel P. The orthotic treatment of acute and chronic disease of the cervical and lumbar spine. Dtsch Arztebl Int. 2013;110(44):737–42. https://doi.org/10.3238/arztebl.2013.0737 [published Online First: Epub Date]|.
104. van Poppel MN, de Looze MP, Koes BW, Smid T, Bouter LM. Mechanisms of action of lumbar supports: a systematic review. Spine (Phila Pa 1976). 2000;25(16):2103–13. https://doi.org/10.1097/00007632-200008150-00016 [published Online First: Epub Date]|.
105. Oleske DM, Lavender SA, Andersson GB, Kwasny MM. Are back supports plus education more effective than education alone in promoting recovery from low back pain?: results

from a randomized clinical trial. Spine (Phila Pa 1976). 2007;32(19):2050–7. https://doi.org/10.1097/BRS.0b013e3181453fcc [published Online First: Epub Date]|.
106. Bataller-Cervero AV, Rabal-Pelay J, Roche-Seruendo LE, et al. Effectiveness of lumbar supports in low back functionality and disability in assembly-line workers. Ind Health. 2019;57(5):588–95. https://doi.org/10.2486/indhealth.2018-0179 [published Online First: Epub Date]|.
107. van Duijvenbode IC, Jellema P, van Poppel MN, van Tulder MW. Lumbar supports for prevention and treatment of low back pain. Cochrane Database Syst Rev. 2008(2):CD001823 https://doi.org/10.1002/14651858.CD001823.pub3 [published Online First: Epub Date]|.
108. Ramey D, Buell PD. A true history of acupuncture. Focus Altern Complement Ther. 2004;9(4):269–73. https://doi.org/10.1211/fact.2004.00244 [published Online First: Epub Date]|.
109. Library WHS. Acupuncture: review and analysis of reports on controlled clinical trials. In: Organization WH, editor. . Geneva; 2012.
110. Vickers AJ, Vertosick EA, Lewith G, et al. Acupuncture for chronic pain: update of an individual patient data meta-analysis. J Pain. 2018;19(5):455–74. https://doi.org/10.1016/j.jpain.2017.11.005 [published Online First: Epub Date]|.
111. Witt CM, Jena S, Selim D, et al. Pragmatic randomized trial evaluating the clinical and economic effectiveness of acupuncture for chronic low back pain. Am J Epidemiol. 2006;164(5):487–96. https://doi.org/10.1093/aje/kwj224 [published Online First: Epub Date]|.
112. Melchart D, Weidenhammer W, Streng A, et al. Prospective investigation of adverse effects of acupuncture in 97 733 patients. Arch Intern Med. 2004;164(1):104–5. https://doi.org/10.1001/archinte.164.1.104 [published Online First: Epub Date]|.
113. Berman BM, Langevin HM, Witt CM, Dubner R. Acupuncture for chronic low back pain. N Engl J Med. 2010;363(5):454–61. https://doi.org/10.1056/NEJMct0806114 [published Online First: Epub Date]|.
114. Simmonds MJ, Kumar S, Lechelt E. Psychological factors in disabling low back pain: causes or consequences?. Disabil Rehabil. 1996;18(4):161–8. https://doi.org/10.3109/09638289609166295 [published Online First: Epub Date]|.
115. Kinney RK, Gatchel RJ, Polatin PB, Fogarty WT, Mayer TG. Prevalence of psychopathology in acute and chronic low back pain patients. J Occup Rehabil. 1993;3(2):95–103. https://doi.org/10.1007/BF01078162 [published Online First: Epub Date]|.
116. Linton SJ. A review of psychological risk factors in back and neck pain. Spine (Phila Pa 1976). 2000;25(9):1148–56. https://doi.org/10.1097/00007632-200005010-00017 [published Online First: Epub Date]|.
117. Onoda K, Okamoto Y, Nakashima K, Nittono H, Ura M, Yamawaki S. Decreased ventral anterior cingulate cortex activity is associated with reduced social pain during emotional support. Soc Neurosci. 2009;4(5):443–54. https://doi.org/10.1080/17470910902955884 [published Online First: Epub Date]|.
118. McEwen BS. Stress, adaptation, and disease. Allostasis and allostatic load. Ann N Y Acad Sci. 1998;840:33–44. https://doi.org/10.1111/j.1749-6632.1998.tb09546.x [published Online First: Epub Date]|.
119. Apkarian AV, Sosa Y, Sonty S, et al. Chronic back pain is associated with decreased prefrontal and thalamic gray matter density. J Neurosci. 2004;24(46):10410–5. https://doi.org/10.1523/JNEUROSCI.2541-04.2004 [published Online First: Epub Date]|.
120. Van Essen DC, Ugurbil K, Auerbach E, et al. The Human Connectome Project: a data acquisition perspective. Neuroimage. 2012;62(4):2222–31. https://doi.org/10.1016/j.neuroimage.2012.02.018 [published Online First: Epub Date]|.
121. Jensen KB, Kosek E, Wicksell R, et al. Cognitive Behavioral Therapy increases pain-evoked activation of the prefrontal cortex in patients with fibromyalgia. Pain. 2012;153(7):1495–503. https://doi.org/10.1016/j.pain.2012.04.010 [published Online First: Epub Date]|.

122. Seminowicz DA, Shpaner M, Keaser ML, et al. Cognitive-behavioral therapy increases prefrontal cortex gray matter in patients with chronic pain. J Pain. 2013;14(12):1573–84. https://doi.org/10.1016/j.jpain.2013.07.020 [published Online First: Epub Date]|.
123. Fishbain DA, Cutler R, Rosomoff HL, Rosomoff RS. Chronic pain-associated depression: antecedent or consequence of chronic pain? A review. Clin J Pain. 1997;13(2):116–37. https://doi.org/10.1097/00002508-199706000-00006 [published Online First: Epub Date]|.
124. Rush AJ, Polatin P, Gatchel RJ. Depression and chronic low back pain: establishing priorities in treatment. Spine (Phila Pa 1976). 2000;25(20):2566–71. https://doi.org/10.1097/00007632-200010150-00004 [published Online First: Epub Date]|.
125. Physiopedia. The Flag System. Secondary the Flag System November 19, 2020 November 26, 2020. https://www.physio-pedia.com/index.php?title=The_Flag_System&oldid=260518.
126. Staff MC. Biofeedback. Secondary biofeedback November 11, 2020 2019. https://www.mayoclinic.org/tests-procedures/biofeedback/about/pac-20384664.
127. Staff MC. Mindfulness exercises. Secondary mindfulness exercises. 2020; https://www.mayoclinic.org/healthy-lifestyle/consumer-health/in-depth/mindfulness-exercises/art-20046356#:~:text=Mindfulness
128. Cherkin DC, Sherman KJ, Balderson BH, et al. Effect of mindfulness-based stress reduction vs cognitive behavioral therapy or usual care on back pain and functional limitations in adults with chronic low back pain: a randomized clinical trial. JAMA. 2016;315(12):1240–9. https://doi.org/10.1001/jama.2016.2323 [published Online First: Epub Date]|.
129. Castelnuovo G, Giusti EM, Manzoni GM, et al. Psychological Treatments and Psychotherapies in the Neurorehabilitation of Pain: evidences and Recommendations from the Italian Consensus Conference on Pain in Neurorehabilitation. Front Psychol. 2016;7:115. https://doi.org/10.3389/fpsyg.2016.00115 [published Online First: Epub Date]|.
130. Braden BB, Pipe TB, Smith R, Glaspy TK, Deatherage BR, Baxter LC. Brain and behavior changes associated with an abbreviated 4-week mindfulness-based stress reduction course in back pain patients. Brain Behav. 2016;6(3):e00443. https://doi.org/10.1002/brb3.443 [published Online First: Epub Date]|.
131. Goyal M, Singh S, Sibinga EM, et al. Meditation programs for psychological stress and well-being: a systematic review and meta-analysis. JAMA Intern Med. 2014;174(3):357–68. https://doi.org/10.1001/jamainternmed.2013.13018 [published Online First: Epub Date]|.

Pharmacology

8

Daltry Dott

Overview

Learning Objectives

- Identify medications commonly used to treat pain due to spinal conditions
- Compare the efficacy of different medications and their use in spine-related pain
- Recognize the advantages and disadvantages of medications used to treat spine-related pain
- Understand the dosing strategies for medications used for pain due to spinal conditions

Acetaminophen

Mechanism of Action

The exact mechanism of action of acetaminophen is unknown. It is believed to be a weak inhibitor of prostaglandin synthesis through cyclooxygenase (COX) inhibition. Other mechanisms of action are believed to be due to activation of central descending serotonergic pathways. Acetaminophen targets nociceptive pain. Other mechanisms of action are thought to include enhancement of the cannabinoid signaling pathways [52].

D. Dott (✉)
Department of Anesthesiology and Pain Management, University of Texas Southwestern Medical Center, Dallas, TX, USA
e-mail: Daltry.dott@utsouthwestern.edu

© The Author(s), under exclusive license to Springer Nature Switzerland AG 2022

C. E. Noe (ed.), *Multidisciplinary Spine Care*,
https://doi.org/10.1007/978-3-031-04990-3_8

Table 8.1 Acetaminophen dosing

Medication	Route	Dosing	Maximum daily dose
Acetaminophen	PO IV	325–1000 mg tid 650–1000 mg q4–6h	3000 mg 4000 mg

PO oral, *IV* intravascular

Evidence in Spine

Minimal evidence exists to support the use of acetaminophen alone for the use of pain, but it is commonly recommended in guidelines as first line therapy either alone or in conjunction with other medications. Despite the lack of high-quality evidence, the opioid sparing or synergistic effects of acetaminophen in conjunction with other medications may justify its use [89].

Adverse Effects

Acetaminophen is relatively well tolerated but can lead to gastrointestinal disturbance, such as nausea, vomiting, abdominal pain, diarrhea, constipation. At high doses or when combined with alcohol, acetaminophen can be hepatotoxic, leading to acute hepatotoxicity, liver failure, and death.

Dosing

See Table 8.1 for common dosing of Acetaminophen.

COX-Inhibitors

Mechanism of Action

Two types of COX inhibitor medications are currently available to treat pain: nonsteroidal anti-inflammatory medications (NSAIDs) and selective COX-2 inhibitors. Inhibition of COX-1 causes a decrease in thromboxane, prostacyclin, and prostaglandins, leading to antipyretic, analgesic, antiplatelet, and anti-inflammatory effects. Inhibition of COX-2 causes a decrease in inflammatory prostaglandins, proteases, and reactive oxygen species that lead to inflammation, fever, and pain. The major differences between the available COX-inhibitors are their selectivity to COX-1 or COX-2 and each drug's particular pharmacokinetics and dosing strategies. NSAIDs target nociceptive pain.

Evidence in Spine

NSAIDs are commonly recommended as first line treatment for low back pain with or without radiculopathy as they may be effective for short term relief of chronic

low back pain without radiculopathy [85]. There is conflicting evidence of whether one NSAID produces superior improvement in pain in comparison to others or if changing to another NSAID after a failed trial of one NSAID will prove to be efficacious [45, 85]. Hauk [45] suggests that if a trial with one NSAID is ineffective after a 2–4-week period, a different NSAID can be tried and may be more effective. In patients with chronic inflammatory spine conditions, such as ankyloses spondylitis, with axial low back pain, evidence suggests that NSAIDs may slow progression of disease [106].

Studies for the use of NSAIDs for the treatment of chronic pain or radiculopathy do not support their use. The outcome with the use of NSAIDs versus other commonly used pharmacotherapies, including opioids and muscle relaxants, in the treatment of chronic pain was no different. For the treatment of radicular symptoms, there is a lack of evidence for therapeutic benefit with NSAIDs compared to placebo [85].

Using NSAIDs as part of a multimodal pharmacological treatment strategy may be more efficacious than as monotherapy. Combination therapy with aceclofenac plus tizanidine (vs. aceclofenac alone) or diclofenac plus B vitamins (vs diclofenac alone) was effective in reducing pain intensity in patients with (sub)acute low back pain with or without leg pain but none provided a clinically important difference in pain intensity reduction [64].

Other benefits of NSAID treatment may include improvement in symptoms of other comorbid conditions, including depression. Depression is a common comorbidity in patients with chronic pain. In patients treated with naproxen or aspirin compared to acetaminophen, depression and suicidal ideation were less, but more depression was reported in patients treated with celecoxib compared to acetaminophen. In women but not men, ibuprofen was associated with fewer reports of depression [57].

Adverse Effects

The risks of adverse events may limit the use of NSAIDS. The gastrointestinal (GI) and cardiovascular (CV) systems are two of the most common systems affected by adverse events with the use of NSAIDs. Ho et al. [46] propose an algorithm for choosing an anti-inflammatory medication in patients with a clinical indication for anti-inflammatory therapy but with GI and/or CV risks. In patients with high or low GI risk and high CV risk, they recommend celecoxib 200 mg/day with concomitant use of a proton pump inhibitor (PPI). In patients who are low GI and CV risk, NSAID of choice or celecoxib in combination with a PPI is recommended [46]. Another issue that may arise is that there is some evidence that NSAID use may cause delayed bone healing in patients who have recently undergone a posterolateral lumbar fusion. When administered for >2 days or at high doses, ketorolac may be associated with pseudarthrosis after posterolateral lumbar fusion. Ketorolac's use in smokers is also associated with pseudarthrosis [59].

Table 8.2 Commonly used NSAID and COX-2 inhibitor dosing

Medication	Route	Dosing	Maximum daily dose
Aspirin	PO	350–1000 mg q4–6h	6000 mg
Diclofenac	PO	100–200 mg bid	400 mg
	TD: Gel	2 grams qid	32 g
	TD: Patch	1 patch (180 mg) bid	360 mg
Etodolac	PO	200–400 mg q6–8h	1000 mg
Ibuprofen	PO	200–800 mg tid	2400 mg
Indomethacin	PO	25–50 mg 2–3 times per day	200 mg
Ketorolac	PO	10 mg qid	40 mg
	IV/IM	15–30 mg q6h	120 mg
Nabumetone	PO	500–1000 mg once or twice daily	2000 mg
Naproxen	PO	250 q6–8h or 500 mg twice daily	1000 mg
Piroxicam	PO	20 mg once daily or 10 mg bid	20 mg
Meloxicam	PO	7.5–15 mg daily	15 mg
Celebrex	PO	100–200 mg bid	400 mg

PO oral, *TD* transdermal, *IV* intravenous, *IM* intramuscular

The most common adverse effects of NSAIDs include cardiovascular events, including heart attack; gastrointestinal toxicity, including stomach irritation and ulceration; hematological toxicity, including increased risk of bleeding; nephrotoxicity, more common with NSAIDs than COX-2 inhibitors; and hepatotoxicity.

Topical formulations can greatly reduce renal toxicity and GI effects, but patients who use topical patches or gels may experience skin irritation at the site of application [26].

Dosing

See Table 8.2 for common dosing of commonly used NSAIDs and COX-2 inhibitors.

Antidepressants

Mechanism of Action

Multiple classes of antidepressants, including tricyclic antidepressants (TCAs), serotonin-norepinephrine reuptake inhibitors (SNRIs), and selective serotonin reuptake inhibitors (SSRIs), are used for the treatment of spine-related pain. Tricyclic antidepressants increase the levels of norepinephrine and serotonin in the synaptic cleft and act as an antagonist at histamine and acetylcholine receptors. SNRIs increase the levels of both norepinephrine and serotonin in the synaptic cleft. SSRIs increase the amount of serotonin in the synaptic cleft. Antidepressants have an effect on neuropathic pain. The exact mechanisms for the efficacy of antidepressants for the treatment of neuropathic pain is yet to be elucidated, and it is presumed that the mechanism may be different than the mechanisms for treatment of depression and mood stabilization. The mechanisms underlying antidepressants' ability to

treat neuropathic pain is to increase norepinephrine in the spinal cord as well as act on the locus ceruleus to directly inhibit pain and activate the impaired descending norepinephrine inhibitory system. Dopamine and serotonin are also increased in the central nervous system with antidepressant use and may enhance the inhibitory effects of norepinephrine [73].

Evidence in Spine

Neuropathic pain is most responsive to analgesic effects of antidepressants. TCAs are the most effective and are used as first line agents. SNRIs are considered second line, and the effectiveness of SSRIs is limited for the treatment of persistent pain [29]. At low doses, tricyclic antidepressants may be helpful in treating both leg and back pain related to lumbar spinal stenosis [75]. Venlafaxine has been shown to be an effective treatment for both acute and chronic neuropathic pain [2]. However, milnacipran lacks evidence to support its use in the treatment of neuropathic pain [28].

Although antidepressants may be effective in treating neuropathic pain, multiple systematic reviews have concluded that there is no clear evidence for the efficacy of antidepressants over placebo for the treatment of low back pain [103]. Duloxetine has been shown to be useful in treating low back pain [5]. However, concomitant use of an NSAID or acetaminophen with duloxetine did not significantly change the efficacy of duloxetine alone for the treatment of chronic low back pain [93]. Of the SSRIs, Fluoxetine is the most studied for the treatment of chronic pain. Fluoxetine, fluvoxamine, and escitalopram seem to be the SSRIs that have the most supporting evidence for use in chronic pain treatment [78]. While TCAs may be effective in treating radicular pain, they do not seem to have much effect on chronic low back pain. Low-dose amitriptyline showed a nonsignificant improvement in pain intensity at 6 months [103]. After a single dose of imipramine, there was no analgesic effect in patients with chronic low-back pain. However, the anti-nociceptive effects may depend on CYP2D6 metabolizer status and may need to be take into account [92].

The advantages to using antidepressants in spine related pain may be due to their other effects instead of their direct analgesic effect. The efficacy of TCAs may be more related to mood and sleep modulation instead of a direct analgesic process [92]. Low-dose amitriptyline has been shown to lead to a reduction in disability at 3 months without improvement in pain intensity [103]. Depression is common among patients with spinal cord injury and other types of spine-related pain. Antidepressants may help modulate mood and can help with pain as well as quality of life after spine surgery [13].

In patients with spinal cord injury related pain the results of treatment with amitriptyline are mixed with a significant response in patients with comorbid depression receiving doses up to 150 mg daily [107].

The use of antidepressants may also help to decrease long-term costs and utilization of higher risk medications. The use of duloxetine in patients with chronic low

back pain versus other non-surgical treatment, including other pharmacological therapies, such as narcotics and NSAIDs, and non-invasive therapy, such as chiropractic therapy, physical therapy, and exercises therapy, was associated with reduced rates of non-surgical therapies and similar back surgery rates without increased costs [49]. Patients with chronic low back pain who initiated duloxetine treatment rather than muscle relaxants, gabapentin, pregabalin, venlafaxine, and tricyclic antidepressants had better compliance and lower likelihood of opioid use [5]. In addition, older patients with chronic low back pain and depression who are co-prescribed an opioid with venlafaxine are less likely to have an analgesic response to low-dose venlafaxine than patients who are not on opioid medications. Opioid morphine equivalent dosing was negatively correlated with pain response with venlafaxine [94].

Overall, the guidelines for antidepressant use are variable. TCAs and SNRIs are more effective for neuropathic pain and SNRIs and SSRIs may be more effective for low back pain treatment. In addition, these medications may work through modulation of comorbidities that commonly accompany pain. More studies should be performed to elucidate the best dosing strategy and efficacy for different etiologies of pain.

Adverse Effects

Due to the anticholinergic effects of TCAs, potential adverse effects include cardiotoxicity, weight gain, blurred vision, dry mouth, constipation, sedation, and postural hypotension (dizziness). TCAs also pose a risk of anticholinergic toxicity. TCAs have a small therapeutic dose range and there is a risk of lethal overdose if not used properly. The most common side effects experienced with SNRIs and SSRIs include nausea, dry mouth, headache, dizziness, sedation, constipation, changes in sexual function. When combined with other medications that increase serotonin levels, TCAs, SNRIs, and SSRIs can all lead to serotonin syndrome.

Dosing

See Table 8.3 for common dosing strategies of antidepressants.

Anticonvulsants

Mechanism of Action

The precise mechanism of action of gabapentin and pregabalin is still unknown. Studies have shown that gabapentin and pregabalin bind to voltage-gated calcium channels, but the exact therapeutic mechanism is yet to be elucidated. Topiramate blocks voltage-gated sodium and calcium channels and inhibits the glutamate

Table 8.3 Antidepressant dosing

Medication	Route	Dosing	Maximum daily dose
TCAs			
Nortriptyline	PO	25 mg 3–4 times daily	100 mg, if >100 mg is given, plasma levels should be monitored
Amitriptyline	PO	25–150 mg 1–4 times per day	150 mg
Imipramine	PO	75–150 mg once a day	200 mg
Desipramine	PO	100–200 mg qhs or bid	300 mg
SNRIs			
Duloxetine	PO	30–60 mg daily	60 mg
Venlafaxine	PO	75–225 mg daily	225 mg
SSRIs			
Fluoxetine	PO	20–60 mg daily	80 mg
Sertraline	PO	25–200 mg daily	200 mg
Paroxetine	PO	12.5–60 mg daily	60 mg
Citalopram	PO	20–40 mg daily	40 mg
Escitalopram	PO	10–20 mg daily	20 mg

PO Oral

pathway while enhancing the GABA-pathways. It also inhibits carbonic anhydrase. The exact mechanism of action responsible for treatment of pain remains to be determined. Anticonvulsants target neuropathic pain.

Evidence in Spine

Multiple meta-analyses have found that gabapentin and pregabalin are not effective for the treatment of low back pain either with or without radiculopathy [32, 34]. Specifically, in a randomized double blind cross over study, extended release gabapentin failed to demonstrate any significant decrease in pain score compared to placebo [41], and in a double-blind, placebo-controlled trial, pregabalin showed no increased efficacy than placebo in reducing leg-pain intensity in patients with both acute and chronic moderate to severe sciatica [65]. A randomized controlled trial in the Department of Veterans Affairs Healthcare System failed to demonstrate superiority of gabapentin compared to placebo for the treatment of low back pain [6]. Pregabalin has been shown to be no more effective than placebo in treating lower extremity radiculopathy as well [3]. Of the evidence available, there exists very low-quality evidence for the treatment of radicular leg pain, nerve injury pain, and spinal cord injury related pain with gabapentin [109].

Some evidence shows that gabapentin may be helpful in patients with lumbar radiculopathy pain. In patients with chronic lumbar radiculopathy, gabapentin may provide improvement in pain and quality of life [112]. A multicenter randomized double-blind study comparing the efficacy of gabapentin and epidural steroid injections for lumbosacral radicular pain in joint service military treatment facilities

found a modest improvement in both groups at 1 and 3 months that resulted in modest improvements in pain and function [24].

Topiramate provides a small effect for short term pain treatment [34]. In a double-blind, randomized, 2-period crossover trial of topiramate and diphenhydramine (placebo), topiramate titrated to maximal tolerated dose reduced average back and overall pain, worst leg and back scores, and global pain relief scores [51]. In a case report, topiramate helped a morbidly obese (BMI 61.4 kg/m^2) female with chronic low back pain achieve clinically meaningful and significant weight loss and improvement in her chronic low back pain and functionality [44]. Topiramate is more effective than placebo in the short-term treatment of chronic nonspecific low back pain [70].

In a Cochrane Review, there was little evidence to support the efficacy of oxcarbazepine in neuropathic radicular pain. Some very low-quality evidence suggests that it may provide some efficacy in treatment of pain but adverse effects, serious adverse effects, and adverse effects leading to discontinuation of the medication are more common with oxcarbazepine than placebo and limits its utility [113].

Pregabalin may be more effective when used in combination with other medications and in treating other common comorbidities associated with pain that can help improve patient quality of life. The combination of NSAIDs and pregabalin was no more effective in reducing pain than NSAIDs alone in patients with acute lumbar disc herniations. However, the combination of NSAID plus pregabalin showed to be more effective to treat sleep disturbance than NSAIDs alone. Patient global impressions of change were also significantly improved with a combination of NSAID and pregabalin instead of NSAID alone [72]. In an observational study of patients with chronic refractory cervical pain and radiculopathy, a significant improvement in self-reported sleep interference and pain was observed in patients treated with pregabalin as opposed to treatment with conventional analgesic care (acetaminophen, NSAIDs, opioids, antidepressants, other antiepileptic drugs) [96]. Pregabalin may produce beneficial effects for patients with central and mixed neuropathic pain at doses of 300–600 mg daily [27]. Pregabalin may be an effective treatment for neuropathic pain but adverse effects on balance at initial doses and with dose increases may limit its use [16].

In patients with spinal cord injury-related central pain, there is conflicting evidence for the efficacy of gabapentin (≥1800 mg daily) and pregabalin. Lamotrigine was effective for incomplete spinal cord injury-related pain at and below the level of injury. The data for carbamazepine for central pain is mixed [107].

Adverse Effects

Gabapentinoids are associated with adverse events. There is high level evidence supporting the risk of harms with these medications [34]. Gabapentin and pregabalin have significant adverse effects and lack significant evidence for pain improvement in patients with chronic low back pain [91]. Pregabalin is associated with

higher rates of adverse events than placebo [65]. Gabapentin's use is limited due to the adverse events, including somnolence, dizziness, peripheral edema, and gait disturbance [109].

Gabapentin and pregabalin most commonly can cause unsteadiness, constipation, diarrhea, word-finding difficulties, sedation, nausea, vomiting, and dry mouth. In some rare instances, gabapentin and pregabalin may cause psychiatric disturbances, including suicidal ideation, depression, anxiety, or violent or aggressive behavior.

The most common side effects experienced with topiramate include nausea, anorexia, weight loss, fatigue, and paresthesias.

Common adverse effects of carbamazepine include dizziness, drowsiness, dry mouth, tongue swelling, balance and coordination problems, nausea, and vomiting. Serious adverse effects include severe skin reaction (rash, Stevens-Johnson syndrome, and toxic epidermal necrolysis) as well as neutropenia. If carbamazepine is prescribed, white blood cell count should be monitored regularly during treatment.

Lamotrigine adverse effects include dizziness, headache, ataxia, nausea, blurred vision, somnolence, psychiatric disturbance (depression, anxiety, emotional lability, difficulty concentrating, nervousness), and rash.

Dosing

See Table 8.4 for common dosing strategies for commonly used anticonvulsants.

The anticonvulsant medications are typically administered using titration schedule. These medications should be used with caution in patients who are taking other sedating medications, and patients should not take these medications with other sedating medications. In the elderly, side effects can be amplified, and anticonvulsants should be prescribed with caution.

Table 8.4 Anticonvulsant dosing

Medication	Route	Dosing	Maximum daily dose
Gabapentin	PO (IR)	100–1200 mg tid, titrated	3600 mg
Pregabalin	PO	25–300 mg bid, titrated	600 mg
Topiramate	PO (IR) PO (ER)	25–50 mg once to twice per day 25–100 mg daily	100 mg 100 mg
Lamotrigine	PO	Depends on other anticonvulsants patient is concurrently taking	
Carbamazepine	PO (IR) PO (ER)	100–600 mg bid 400–800 mg daily (initial dose 200 mg daily)	1200 mg 1200 mg

PO oral, *IR* immediate release, *ER* extended release

Muscle Relaxers

Mechanism of Action

The mechanisms of action for the multiple classes of muscle relaxers are unknown. Each muscle relaxer may work through different mechanisms and may be more or less efficacious for patients on an individual basis.

Baclofen is a gamma-aminobutyric acid (GABA) agonist. The precise mechanism of action is unknown. It inhibits both monosynaptic and polysynaptic reflexes at the spinal level and may cause the hyperpolarization of afferent terminals. Other binding sites occur at the supraspinal level and contribute to its clinical effect.

Cyclobenzaprine acts primarily in the brainstem by activating locus ceruleus neurons, increasing the release of norepinephrine in the ventral horn of the spinal cord, leading to increased inhibitory action of norepinephrine on gamma and alpha motor neurons to reduce tonic somatic motor activity.

Methocarbamol act as a general central nervous system depressant and seems to have no direct action on the contractile mechanism of striated muscle, the motor end plate, or the nerves.

Metaxalone does not directly act on skeletal muscle and potentially acts through central nervous system (CNS) depression.

Tizanidine is a central alpha-2-adrenergic receptor agonist and reduces spasms through increased presynaptic inhibition of motor neurons.

Carisoprodol acts via CNS depression.

Benzodiazepines bind to a receptor on GABA-A complexes and increase the frequency of chloride channel opening, leading to an increased inhibitory effect of GABA on neuronal excitability.

Evidence in Spine

Overall, muscle relaxers have mixed effects in treating spine related pain. In a meta-analysis, cyclobenzaprine was more effective than placebo in treating back pain, but the effect is modest, and the risk of adverse effects is significant so it should be used at the lowest effective dose and avoided in elderly [15]. In patients with nonspecific back pain, cyclobenzaprine is effective, and no difference was noted between 5 mg dosing or 10 mg dosing [14]. Once daily cyclobenzaprine ER has also been shown to be effective in treating neck and back pain due to muscle spasm [62].

The use of combination therapy with muscle relaxants and analgesic medications is conflicting. Some studies have shown that combination therapy of cyclobenzaprine with ibuprofen or naproxen has shown no benefit for neck pain or acute nontraumatic low back pain without radiculopathy [14, 20, 37]. In patients with acute, nontraumatic low back pain without radiculopathy, a combination of methocarbamol or orphenadrine with naproxen compared with naproxen plus placebo showed no benefit [36]. In addition to a lack of pain improvement, combination therapy with

baclofen, metaxalone, or tizanidine with ibuprofen failed to lead to an improvement in function as well in patients with acute low back pain [39]. In a randomized, double-blind, comparative efficacy clinical trial, the combination of diazepam and naproxen vs naproxen with placebo, did not improve functional outcomes or pain in patients with acute, nontraumatic, nonradicular low back pain [38]. However, Toth and Urtis [100] found that combination of muscle relaxers, including cyclobenzaprine, metaxalone, and carisoprodol, can be used with analgesic medications to achieve improved pain control. In addition, a double-blind, double-dummy, randomized, multicentric, comparative study showed that patients using combination tizanidine-aceclofenac had significant increases in spinal flexion and improved pain intensity for the treatment of acute low back pain [77]. Also, patients treated with tizanidine and tramadol resulted in improvement in pain at rest and with effort [87].

In patients with spasticity, multiple muscle relaxers have been found to be effective. In a systematic review found fair evidence for treatment with baclofen and tizanidine, and in patients with neck or back pain due to musculoskeletal conditions, there is fair evidence that cyclobenzaprine, carisoprodol, orphenadrine, and tizanidine are effective [22]. Tizanidine is a useful medication in patients suffering from spasticity due to spinal cord injury as well as in patients with chronic myofascial neck and back pain [61].

Another route of administration for the muscle relaxer baclofen is via an intrathecal pump. A retrospective questionnaire of patients who had undergone intrathecal baclofen pump insertion who suffered from chronic mechanical low back pain or failed back pain demonstrated that spinal drug administration systems seem to be of benefit in alleviating pain in these conditions [82].

Adverse Effects

Skeletal muscle relaxants have been shown to be beneficial in nonspecific chronic low back pain, but adverse effects may limit their use [104].

The most common side effects reported with all muscle relaxants are sedation, dizziness, vertigo, headache, memory problems, gastrointestinal disturbance, dry mouth. Sudden cessation of baclofen may lead to life-treating withdrawal. Carisoprodol should be prescribed judiciously as it has addictive properties and may lead to substance abuse.

Dosing

See Table 8.5 for common dosing strategies for commonly prescribed muscle relaxants.

Special care should be taken when prescribing muscle relaxants with other sedating medications. In the elderly, side effects can be amplified, and muscle relaxants should be prescribed with caution.

Table 8.5 Muscle relaxant dosing

Medication	Route	Dosing	Maximum daily dose
Baclofen	PO	5–20 mg tid prn	80 mg
	IT	22–1400 mcg daily infusion	–
Cyclobenzaprine	PO	5–10 mg tid prn	30 mg
Methocarbamol	PO	500–1500 mg 3–4 times per day prn	4500 mg
	IV	1000 mg tid prn	3000 mg
Metaxalone	PO	800 mg 3–4 times per day prn	2400 mg
Tizanidine	PO	2–16 mg tid prn	36 mg
Carisoprodol	PO	250–350 mg qid prn	1400 mg
Diazepam	PO	2–10 mg 3–4 times daily prn	–
	IV/IM	5–10 mg q3–4h prn	–

PO oral, *IT* intrathecal, *IV* intravenous, *IM* intramuscular, *prn* as needed, *tid* three times daily, *qid* four times daily

Corticosteroids

Mechanism of Action

Exogenous corticosteroids mimic the endogenous glucocorticoids produced within the hypothalamic-pituitary-adrenal axis. They bind to receptors in the cytoplasm and migrate into the nucleus where the receptor-steroid complex binds to the DNA and alters genetic synthesis of proteins. Multiple cellular functions are modified, including the production of enzymes that regulate metabolic processes as well as those that regulate the synthesis of inflammatory cytokines.

Evidence in Spine

The evidence for use of corticosteroids in spine related pain is lacking. In a randomized, controlled trial, oral corticosteroids were more effective in pain relief than gabapentin or pregabalin in patients with lumbar radiating pain [53]. However, for patients with acute low back pain, prednisone and oral systemic corticosteroids provided no improvement in pain [35, 42]. Corticosteroids may be effective in treating radicular pain but likely are not effective for low back pain with or without radiculopathy.

Adverse Effects

Short term use is usually well tolerated but can cause hypertension, hyperglycemia, flushing and warmth, and psychological effects, including anxiety, psychosis. Chronic use of corticosteroids can lead to a myriad of complications, including, osteoporosis, decreased immunity, suppression of the HPA axis with the possibility of acute adrenal insufficiency if the exogenous source is abruptly discontinued, postoperative delayed wound healing or infection, weight gain.

Table 8.6 Corticosteroid dosing

Medication	Route	Dosing	Maximum daily dose
Prednisone	PO	6-day taper: 30 mg, 25 mg, 20 mg, 15 mg, 10 mg, 5 mg in divided doses	–
Methylprednisolone (off label)	PO	6-day taper: 24 mg, 20 mg, 16 mg, 12 mg, 8 mg, 4 mg in divided doses	–

PO oral, *IM* intramuscular

Dosing

See Table 8.6 for common dosing strategies for commonly prescribed corticosteroids.

Local Anesthetics

Mechanism of Action

Local anesthetics decrease neuronal excitation by antagonizing sodium channels. Local anesthetics affect neuropathic pain.

Evidence in Spine

Lidocaine infusions have been used in the treatment of chronic pain [68]. A review of patients with intractable neuropathic pain treated with lidocaine infusions demonstrated significant decrease in pain [111]. However, lidocaine infusion has not been shown to be an effective treatment for acute lumbar radicular pain [97] and was no more effective in treating neuropathic pain of failed back surgery syndrome than placebo [76].

Mexiletine is an oral form of a local anesthetic and is not commonly used due to its high rate of adverse effects. In a meta-analysis, mexiletine was found to be an effective treatment for neuropathic pain [101]. Typically, response to a lidocaine infusion is used to identify patients who may have a positive response to mexiletine treatment [17]. More studies need to be performed examining the efficacy of mexiletine and the risks of adverse effects prior to recommending its routine use in treating spine-related pain.

Topical lidocaine formulations may be effective for neuropathic pain. In patients with localized neuropathic pain who previously were unable to tolerate or failed to improve with other medications, including antiepileptics, antidepressants, and opioids, lidocaine medicated plasters were effective in relieving pain [63]. In patients with cervical radiculopathy, lidocaine 5% medicated plaster improved pain symptoms and allowed patients to started rehabilitative treatment with physical therapy sooner [67]. Lidocaine 5% medicated plaster may be a valuable additional approach for the management of neuropathic low back pain [9, 10]. Topical lidocaine formulations are typically well tolerated with a low rate of discontinuation due to adverse effects.

Table 8.7 Local Anesthetic dosing

Medication	Route	Dosing	Maximum daily dose
Mexiletine	PO	150–900 mg daily	900 mg
Lidocaine	IV: Infusion TD: Patch TD: Gel, cream, lotion, ointment	5.5 mg/kg infused over 2 hours 4–5%, apply patch(es) for up to 12 hours within a 12-hour period. Patches may be cut 3–5%, apply a thin film to affected area 2–4 times a day	5 mg/kg maximum of 3 patches at a time 20 g

PO oral, *IV* intravenous, *TD* transdermal

Adverse Effects

Lidocaine infusions are overall well tolerated with side effects resolving quickly with interruption or discontinuation of the infusion [66].

The most common adverse effects with IV formulations include dizziness, nausea, lightheadedness, ringing in the ears, hypotension, cardiac arrhythmias, and local anesthetic systemic toxicity, which can lead to cardiac arrest, and seizure. Side effects with mexiletine include GI upset, headache, blurred vision, dizziness, sedation, paresthesias, weakness, elevated liver function tests. Mexiletine has a high rate of nausea and dizziness, which can limit its clinical use. However, in a recent study, mexiletine seemed to be well tolerated in its use to treat neuropathic pain [86]. Topical lidocaine can produce localized reactions after application.

Dosing

See Table 8.7 for common dosing strategies for commonly used local anesthetics.

Opioids

Mechanism of Action

Opioids exert their effects through binding to mu, delta, and kappa G-protein-coupled receptors, which are widely distributed within the CNS and peripheral nervous system. The range of effect produced by opioids depends on the type and location of the receptor that is stimulated. Agonists of the mu receptors cause analgesia but also leads to sedation, respiratory depression, bradycardia, nausea, vomiting, and a reduction in gastric motility. Activation of the delta receptors causes spinal and supraspinal analgesia and reduced gastric motility. Activation of the kappa receptor produces spinal analgesia, diuresis, and dysphoria. Activation of all of these receptors leads to a net effect of cellular hyperpolarization and reduced neurotransmitter release [79].

Tramadol is an atypical opioid and in addition to its opioidergic effects, also has noradrenergic and serotonergic actions. It also has modulatory effects on several

mediators in pain signaling, such as voltage-gated sodium channels, V1 channels, glutamate receptors, alpha2 adrenoreceptors, adenosine receptors, and mechanisms involving substance P, calcitonin gene-related peptide, prostaglandin E2, and proinflammatory cytokines. Because of its broad spectrum of targets, tramadol can be used to relieve a broad range of pain, including low back, osteoarthritic, and neuropathic pain. In addition, tramadol has anxiolytic and antidepressant effects that improve pain outcomes [8].

Buprenorphine is a unique opioid that is a partial agonist at the mu receptor and a weak kappa receptor antagonist and delta receptor agonist.

The available pharmacologic formulations of opioids are divided into three categories: natural (morphine, codeine, thebaine), semi-synthetic (hydromorphone, hydrocodone, oxycodone), and synthetic (tramadol, methadone, fentanyl, tapentadol).

Evidence in Spine

The evidence for use of opioids in the treatment of spine-related pain is conflicting. In most cases, the evidence suggests that the utility of opioids is limited in the treatment of spine-related pain due to its significant amount of adverse effects and addictive properties and if used, should not be used as a first line treatment option. In a randomized clinical trial in patients with chronic back, hip, or knee osteoarthritis pain, immediate-release opioids (morphine, oxycodone, or hydrocodone/acetaminophen) was not superior to nonopioid (acetaminophen or NSAID) in achieving pain relief at 12 months [55]. A systematic review and meta-analysis of placebo-controlled randomized controlled trials showed moderate quality evidence that opioids reduce pain in the short term. However, clinically relevant pain relief was not observed within a dose range of 40–240 mg morphine equivalents per day. Evidence for long-term efficacy is lacking, and the efficacy of opioid use for acute low back pain is unknown [1]. In a Cochrane Review, there is some evidence for the use of short-term opioids to treat chronic low back pain. However, the trials that compare opioids to NSAIDs or antidepressants did not show any differences did not show any significant differences regarding pain and function. There are no randomized controlled trials using opioids versus placebo to support the efficacy and safety of long-term opioid therapy to treat chronic low back pain [18]. In a Cochrane review, there is insufficient evidence to support or refute that morphine is efficacious for the treatment of neuropathic pain [25].

Combination of opioids with other analgesics has not been shown to be beneficial for spine-related pain treatment. The addition of oxycodone/acetaminophen to naproxen did not improve functional outcomes or pain in patients with acute nontraumatic low back pain without radiculopathy [37]. In a Cochrane Review, there is insufficient evidence to support or refute the use of paracetamol in combination with codeine or dihydrocodeine for neuropathic pain [110].

Tramadol is an opioid with some unique properties and may be more efficacious in treating spine-related pain. When used as part of combination treatment, the combination of tramadol and dexketoprofen resulted in effective treatment for nonspecific acute low back pain, higher compliance, fewer side effects, and less rescue-drug use [81], and the combination of tramadol with NSAID may decrease

the incidence of adverse events and may help prevent the transition of acute low back pain to chronic low back pain [48]. In patients with chronic low back pain, tramadol extended release (ER) was found to be more effective in treating pain than placebo [105]. In a randomized, double-blind, placebo-controlled, parallel-group study, tramadol ER was more effective than placebo in providing pain relief, functional improvements, and improved quality of life in patients with chronic low back pain [56]. Tramadol may also improve other comorbidities that often accompany pain. In a retrospective case-control study of patients with chronic low back pain, tramadol and acetaminophen versus celecoxib alone was more effective in conferring a motivational effect while also being effective in reducing chronic low back pain [99]. The combination of tramadol-acetaminophen was more effective than NSAIDs at in treating pain and also conferred an antidepressant effect that NSAIDs did not [98]. However, in a systematic review, there was no reliable indication for the use of tramadol to treat neuropathic pain [31].

A few studies have demonstrated that long acting opioids may be effective in spine-related pain. Once daily hydrocodone has been shown to be efficacious in treating uncontrolled moderate to severe chronic low back pain and was generally well tolerated [108]. In another study, oxycodone controlled-release was more effective than placebo in providing pain relief and improvements in quality of life and quality of sleep. However, oxycodone had significantly more side effects than placebo [60]. In addition, tapentadol prolonged release was more effective in treating neuropathic pain symptoms and at improving global health status than oxycodone/naloxone prolonged release and had improved gastrointestinal tolerability [9, 10].

A couple of opioids are available in transdermal and buccal formulations that bypass first pass metabolism. Buprenorphine buccal film has been effective in treating moderate to severe pain in patients with chronic low back pain [83], and transdermal fentanyl was noninferior to gabapentin for the treatment of lumbar radicular pain in terms of pain reduction with no difference in patient functional status, depressive symptoms, and the occurrence of adverse events [47].

Opioids may provide a synergistic effect when used in combination with other medications. In patients with chronic low back pain with or without leg pain, tramadol plus paracetamol versus placebo and buprenorphine plus pregabalin versus buprenorphine alone provided significant reduction in pain intensity [64].

Opioids can also be administered via intrathecal pump and may have some neuromodulatory effects. This will be further discussed in the neuromodulation chapter.

Adverse Effects

The medium- and long-term use of opioids for chronic non-cancer pain is associated with a number of adverse events, including serious events [33]. The most common side effects of opioids include sedation, dizziness, nausea, vomiting, constipation, physical dependency, tolerance, addiction, and respiratory depression. Overdose can cause severe and life-threatening respiratory depression.

Table 8.8 Opioid dosing

Medication	Route	Dosing	Maximum daily dose
Codeine	PO	15–60 mg q4–6h prn	360 mg
Tramadol	PO (IR) PO (ER)	50–100 mg q4–6h prn 100 mg daily	400 mg 300 mg
Morphine	PO (IR) PO (ER)	15–30 mg q4–12h prn Once or twice daily dosing depending on formulation	– –
Hydrocodone	PO (IR) PO (ER)	2.5–10 mg q4–12h prn Once daily dosing	– –
Oxycodone	PO (IR) PO (ER)	2.5–30 mg q4–12h prn Twice daily dosing	– –
Hydromorphone	PO (IR) PO (ER)	2–4 mg q4–12h prn Once daily dosing	– –
Methadone	PO	10 mg 2–3 times daily	–
Fentanyl	TD: Patch	12.5–100 mcg/h q72h	–
Tapentadol	PO (IR) PO (ER)	50–100 mg q4–6h prn 50–250 mg bid	– –
Buprenorphine	Buccal TD: Patch	75–900 mg bid 5–20 mcg/h q7d	1000 mg 20 mcg/h

PO oral, *IR* immediate release, *ER* extended release, *TD* transdermal, *bid* twice daily, *prn* as needed

Aside from common side effects, in patients with chronic pain, night-time sleep disturbance is common and may be exacerbated by opioid treatment. In comparison to patients receiving non-opioid medications for chronic back pain, patients taking opioid medications (>100 mg morphine equivalent per day) for chronic back pain demonstrated distinctly abnormal brain activity during sleep [84].

Dosing

See Table 8.8 for commonly prescribed opioid dosing strategies.

Capsaicin

Mechanism of Action

Topical capsaicin is an agonist for TRPV1 receptors, which are activated by high temperatures, pH <6.0, or a combination of the two. TRPV1 receptor activation leads to depolarization of C and A delta fibers, leading to a sensation of warmth, burning, stinging, or itching. Capsaicin generates a persistent effect and increases calcium permeability at multiple levels. Sustained high levels of intracellular calcium activates proteases and an induce the depolymerization of microtubules. High levels of intracellular calcium and chloride lead to osmotic swelling. An additional effect of capsaicin is to disrupt mitochondria respiration. The combination of these actions leads to impaired local nociceptor function and desensitization [4].

Evidence in Spine

Capsaicin comes in a topical formulation that can be helpful in the treatment of neuropathic pain. Topical capsaicin capsaicin 8% patch may be a valuable additional approach for the management of neuropathic low back pain [9, 10]. Application of a high-dose capsaicin 8% patch within innervated territories helped to alleviate painful radiculopathy [11]. However, capsaicin's use is limited due to its high incidence of discontinuation due to intolerable adverse effects.

Adverse Effects

Capsaicin typically does not systemic effects but can cause localized erythema, induration, burning, itching, and pain. Rare side effects include cardiac dysrhythmias and hypertension.

N-Methyl-D-Aspartate (NMDA) Receptor Antagonists

Mechanism of Action

Ketamine is the most commonly known and used NMDA receptor antagonist and alters the actions of glutamate.

Evidence in Spine

Ketamine is most commonly used as an intravenous (IV) infusion in the treatment of pain. For patients with acute pain, ketamine may help to attenuate pain unrelieved by other modalities. In patients with acute pain who became tolerant to opioids and not achieving adequate pain control, ketamine could be used as an adjuvant for severe intractable pain [19]. In patients with acute pain, including low back pain, who presented to the emergency room, low dose-ketamine produced a significant analgesic effect within 5 minutes of administration and provided a moderate reduction in pain for 2 hours. However, this reduction in pain was not superior to morphine when analyzed using the numeric rating scale [69].

Ketamine infusions may be helpful in the treatment of chronic pain and may be aid in decreasing opioid medications. In a review analyzing previous trials of ketamine as a single dose, continuous infusion, patient-controlled analgesia, epidural ketamine with opioids, and studies in children, IV PCA with ketamine versus morphine alone did not improve analgesia. IV infusion or single bolus dose of ketamine resulted in a decrease in opioid requirements in a majority of studies, and a majority of trial with epidural ketamine showed beneficial effects. Low dose

ketamine may be a useful adjuvant in pain treatment [95]. In a retrospective analysis evaluating the efficacy of outpatient IV ketamine infusions for chronic intractable pain, patients obtained a significant decrease in visual analog scale (VAS) score and half of the patients had relief lasting up to 3 weeks with minimal side effects [80]. In patient with chronic whiplash associated pain, 7 out of 20 patients had no improvement with any of the interventions (placebo, placebo/remifentanil, ketamine/placebo, ketamine/remifentanil) but in the patients who did respond, the combination of ketamine and remifentanil showed an analgesic effect on habitual pain and ketamine seemed to enhance the effect of remifentanil on electrical pain thresholds [58].

Topical ketamine has been used to treat neuropathic pain, including lumbar radiculopathy, and patients reported a significant decrease in numerical analogue scale after initial application as well as alterations in temperature sensation, feelings of relaxation, and decreased tension in the area of relaxation. Ketamine gel may be a potential option for patients with chronic neuropathic pain [40].

Adverse Effects

At low doses, ketamine is relatively well tolerated. Adverse effects include depressed mental state, feelings of detachment, slurred speech, hallucinations, dizziness, and nystagmus. Other more serious effects include, chest pain, hypertension, changes in heart rate, amnesia, coma, delirium, hyperthermia, seizures, nausea and vomiting, changes in respiratory rate, salivation, laryngeal spasm, anxiety, and muscle rigidity.

Tumor Necrosis Factor (TNF) Inhibitors (Anti-TNF)

Mechanism of Action

Anti-TNF medications suppress the immune system by blocking the action of TNF, which is a substance that leads to inflammation and auto-immune diseases.

Evidence in Spine

Etanercept is an anti-TNF medication that has been used in patients with axial spondyloarthritis. In this patient population, it seems to slow the progression of sacroiliac joint changes compared to patients not receiving biologics [30] and has been shown to reduce spine and sacroiliac joint inflammation via MRI imaging [43]. In patients with rheumatologic etiologies of spine-pain, anti-TNF medications may be helpful in preventing progression of the disease and evaluation for disease modifying agents may be warranted.

Adverse Effects

The most common side effects of etanercept include GI disturbance (nausea, vomiting, abdominal pain, diarrhea, gastroesophageal reflux), headache, weight changes, weakness, and pain or discomfort at the injection site. Other serious side effects can include seizures, swelling, rash or skin changes, increased risk of infection, GI bleed, joint pain or swelling.

Prostaglandin E1 (PGE1) Derivatives

Mechanism of Action

PGE1 derivatives are agonists of the E1 receptor, a G-protein receptor. PGE1 is a vasodilator and causes angiogenesis. It is thought that the improvement in circulation exerts an analgesic effect [50].

Use in Spine

Limaprost has been used to treat pain due to lumbar spinal stenosis in Japan. Limaprost has also been shown to be effective in treating neuropathic pain [50]. In patients with cervical spondylotic radiculopathy, limaprost was superior to pregabalin in treating arm numbness [74]. Although it is not approved to treat pain in the United States currently, PGE1 derivatives may be an option for patients who fail to improve with other treatments.

Adverse Effects

The most common adverse effects include headache, flushing and GI disturbance (diarrhea, nausea, vomiting, and abdominal pain).

Other

There is a paucity of literature evaluating the efficacy of topical agents for the use of radicular pain. Topical compound formulations may provide localized delivery of medications without the adverse effects that may limit the use of systemic formulations in patients. Safaeian et al. [88] describe the use of topical formulation compound cream composed of diclofenac, ibuprofen, baclofen, cyclobenzaprine, bupivacaine, gabapentin, and pentoxifylline (T7) as an effective treatment for radicular pain.

Conclusion

Many pharmacologic therapies have some evidence for effectiveness in treating chronic and acute low back pain [21]. However, the effects of pain reduction and improvement in function is typically small to moderate and is typically only short lasting [54]. Pharmacological treatment is often based on tradition and personal experience as the literature is lacking in studies that can be used to formulate evidence-based guidelines [71]. Multiple guidelines have been published regarding medication management for the treatment of pain [7, 12, 23, 85, 102]. However, significant differences between guidelines in terms of attitude towards pharmacotherapy, analgesics of first choice, and recommendations for or against the prescription of specific pharmacological treatments exist [90]. One overarching recommendation is that opioids should only be considered a last resort option in cases that all other pharmacological options have failed and after a discussion of risks and benefits has occurred. Of the medications available, type of pain (nociceptive versus neuropathic), diagnosis and origin of pain, safety profile, and patient-specific issues (ex. past medical history and past experiences with similar medications) are important factors that should influence medication choice should pharmacologic intervention be warranted.

References

1. Abdel Shaheed C, Maher CG, Williams KA, Day R, McLachlan AJ. Efficacy, tolerability, and dose-dependent effects of opioid analgesics for low back pain: a systematic review and meta-analysis. JAMA Intern Med. 2016;176(7):958–68. https://doi.org/10.1001/jamainternmed.2016.1251.
2. Aiyer R, Barkin RL, Bhatia A. Treatment of neuropathic pain with venlafaxine: a systematic review. Pain Med. 2017;18(10):1999–2012. https://doi.org/10.1093/pm/pnw261.
3. Alavinia SM, Furlan JC, MacDonald SL, Kumbhare D. Second-order peer reviews of clinically relevant articles for the physiatrist: is pregabalin effective in the treatment of radiating low back pain into the leg? Am J Phys Med Rehabil. 2018;97(9):e78–80. https://doi.org/10.1097/PHM.0000000000000944.
4. Anand P, Bley K. Topical capsaicin for pain management: therapeutic potential and mechanisms of action of the new high-concentration capsaicin 8% patch. Br J Anaesth. 2011;107(4):490–502. https://doi.org/10.1093/bja/aer260.
5. Andrews JS, Wu N, Chen SY, Yu X, Peng X, Novick D. Real-world treatment patterns and opioid use in chronic low back pain patients initiating duloxetine versus standard of care. J Pain Res. 2013;6:825–35. Published 2013 Nov 26. https://doi.org/10.2147/JPR.S50323.
6. Atkinson JH, Slater MA, Capparelli EV, et al. A randomized controlled trial of gabapentin for chronic low back pain with and without a radiating component. Pain. 2016;157(7):1499–507. https://doi.org/10.1097/j.pain.0000000000000554.
7. Attal N. Pharmacological treatments of neuropathic pain: the latest recommendations. Rev Neurol (Paris). 2019;175(1–2):46–50. https://doi.org/10.1016/j.neurol.2018.08.005.
8. Barakat A. Revisiting tramadol: a multi-modal agent for pain management. CNS Drugs. 2019;33(5):481–501. https://doi.org/10.1007/s40263-019-00623-5.

9. Baron R, Binder A, Attal N, Casale R, Dickenson AH, Treede RD. Neuropathic low back pain in clinical practice. Eur J Pain. 2016;20(6):861–73. https://doi.org/10.1002/ejp.838.
10. Baron R, Likar R, Martin-Mola E, et al. Effectiveness of tapentadol prolonged release (PR) compared with oxycodone/naloxone PR for the management of severe chronic low back pain with a neuropathic component: a randomized, controlled, open-label, phase 3b/4 study. Pain Pract. 2016;16(5):580–99. https://doi.org/10.1111/papr.12308.
11. Baron R, Treede RD, Birklein F, et al. Treatment of painful radiculopathies with capsaicin 8% cutaneous patch. Curr Med Res Opin. 2017;33(8):1401–11. https://doi.org/10.1080/03007995.2017.1322569.
12. Bates D, Schultheis BC, Hanes MC, et al. A comprehensive algorithm for management of neuropathic pain. Pain Med. 2019;20(Suppl 1):S2–S12. https://doi.org/10.1093/pm/pnz075.
13. Bayoumi AB, Ikizgul O, Karaali CN, Bozkurt S, Konya D, Toktas ZO. Antidepressants in spine surgery: a systematic review to determine benefits and risks. Asian Spine J. 2019;13(6):1036–46. https://doi.org/10.31616/asj.2018.0237.
14. Braschi E, Garrison S, Allan GM. Cyclobenzaprine for acute back pain. Can Fam Physician. 2015;61(12):1074.
15. Browning R, Jackson JL, O'Malley PG. Cyclobenzaprine and back pain: a meta-analysis. Arch Intern Med. 2001;161(13):1613–20. https://doi.org/10.1001/archinte.161.13.1613.
16. Çağlar Okur S, Vural M, Pekin Doğan Y, Mert M, Sayıner Çağlar N. The effect of pregabalin treatment on balance and gait in patients with chronic low back pain: a retrospective observational study. J Drug Assess. 2019;8(1):32–5. Published 2019 Feb 4. https://doi.org/10.1080/21556660.2019.1579098.
17. Carroll I, Kaplan K, Mackey S. Mexiletine therapy for chronic pain: survival analysis identifies factors predicting clinical success. J Pain Symptom Manag. 2008;35(3):321–6. https://doi.org/10.1016/j.jpainsymman.2007.04.022.
18. Chaparro LE, Furlan AD, Deshpande A, Mailis-Gagnon A, Atlas S, Turk DC. Opioids compared to placebo or other treatments for chronic low-back pain. Cochrane Database Syst Rev. 2013;(8):CD004959. Published 2013 Aug 27. https://doi.org/10.1002/14651858.CD004959.pub4.
19. Chazan S, Ekstein MP, Marouani N, Weinbroum AA. Ketamine for acute and subacute pain in opioid-tolerant patients. J Opioid Manag. 2008;4(3):173–80. https://doi.org/10.5055/jom.2008.0023.
20. Childers MK, Borenstein D, Brown RL, et al. Low-dose cyclobenzaprine versus combination therapy with ibuprofen for acute neck or back pain with muscle spasm: a randomized trial. Curr Med Res Opin. 2005;21(9):1485–93. https://doi.org/10.1185/030079905X61938.
21. Chou R, Deyo R, Friedly J, et al. Systemic pharmacologic therapies for low back pain: a systematic review for an American College of Physicians Clinical Practice guideline. Ann Intern Med. 2017;166(7):480–92. https://doi.org/10.7326/M16-2458.
22. Chou R, Peterson K, Helfand M. Comparative efficacy and safety of skeletal muscle relaxants for spasticity and musculoskeletal conditions: a systematic review. J Pain Symptom Manag. 2004;28(2):140–75. https://doi.org/10.1016/j.jpainsymman.2004.05.002.
23. Clauw D, McCarberg BH. Managing chronic pain with nonopioid analgesics: a multidisciplinary consult. Am J Med. 2012;125(5):S1. https://doi.org/10.1016/j.amjmed.2011.10.003.
24. Cohen SP, Hanling S, Bicket MC, et al. Epidural steroid injections compared with gabapentin for lumbosacral radicular pain: multicenter randomized double blind comparative efficacy study. BMJ. 2015;350:h1748. Published 2015 Apr 16. https://doi.org/10.1136/bmj.h1748.
25. Cooper TE, Chen J, Wiffen PJ, et al. Morphine for chronic neuropathic pain in adults. Cochrane Database Syst Rev. 2017;5(5):CD011669. Published 2017 May 22. https://doi.org/10.1002/14651858.CD011669.pub2.
26. Deng ZH, Zeng C, Yang Y, et al. Topical diclofenac therapy for osteoarthritis: a meta-analysis of randomized controlled trials. Clin Rheumatol. 2016;35(5):1253–61. https://doi.org/10.1007/s10067-015-3021-z.

27. Derry S, Bell RF, Straube S, Wiffen PJ, Aldington D, Moore RA. Pregabalin for neuropathic pain in adults. Cochrane Database Syst Rev. 2019;1(1):CD007076. Published 2019 Jan 23. https://doi.org/10.1002/14651858.CD007076.pub3.
28. Derry S, Wiffen PJ, Kalso EA, et al. Topical analgesics for acute and chronic pain in adults – an overview of Cochrane Reviews. Cochrane Database Syst Rev. 2017;5(5):CD008609. Published 2017 May 12. https://doi.org/10.1002/14651858.CD008609.pub2.
29. Dharmshaktu P, Tayal V, Kalra BS. Efficacy of antidepressants as analgesics: a review. J Clin Pharmacol. 2012;52(1):6–17. https://doi.org/10.1177/0091270010394852.
30. Dougados M, Maksymowych WP, Landewé RBM, et al. Evaluation of the change in structural radiographic sacroiliac joint damage after 2 years of etanercept therapy (EMBARK trial) in comparison to a contemporary control cohort (DESIR cohort) in recent onset axial spondyloarthritis. Ann Rheum Dis. 2018;77(2):221–7. https://doi.org/10.1136/annrheumdis-2017-212008.
31. Duehmke RM, Derry S, Wiffen PJ, Bell RF, Aldington D, Moore RA. Tramadol for neuropathic pain in adults. Cochrane Database Syst Rev. 2017;6(6):CD003726. Published 2017 Jun 15. https://doi.org/10.1002/14651858.CD003726.pub4.
32. Ebell MH. Gabapentin and pregabalin not effective for low back pain with or without radiculopathy. Am Fam Physician. 2019;99(6): 394B–C.
33. Els C, Jackson TD, Kunyk D, et al. Adverse events associated with medium- and long-term use of opioids for chronic non-cancer pain: an overview of Cochrane Reviews. Cochrane Database Syst Rev. 2017;10(10):CD012509. Published 2017 Oct 30. https://doi.org/10.1002/14651858.CD012509.pub2.
34. Enke O, New HA, New CH, et al. Anticonvulsants in the treatment of low back pain and lumbar radicular pain: a systematic review and meta-analysis. CMAJ. 2018;190(26):E786–93. https://doi.org/10.1503/cmaj.171333.
35. Eskin B, Shih RD, Fiesseler FW, et al. Prednisone for emergency department low back pain: a randomized controlled trial. J Emerg Med. 2014;47(1):65–70.
36. Friedman BW, Cisewski D, Irizarry E, et al. A randomized, double-blind, placebo-controlled trial of naproxen with or without orphenadrine or methocarbamol for acute low back pain. Ann Emerg Med. 2018;71(3):348–56.e5. https://doi.org/10.1016/j.annemergmed.2017.09.031.
37. Friedman BW, Dym AA, Davitt M, et al. Naproxen with cyclobenzaprine, oxycodone/acetaminophen, or placebo for treating acute low back pain: a randomized clinical trial. JAMA. 2015;314(15):1572–80. https://doi.org/10.1001/jama.2015.13043.
38. Friedman BW, Irizarry E, Solorzano C, et al. Diazepam is no better than placebo when added to naproxen for acute low back pain. Ann Emerg Med. 2017;70(2):169–76.e1. https://doi.org/10.1016/j.annemergmed.2016.10.002.
39. Friedman BW, Irizarry E, Solorzano C, et al. A randomized, placebo-controlled trial of ibuprofen plus metaxalone, tizanidine, or baclofen for acute low Back pain. Ann Emerg Med. 2019;74(4):512–20. https://doi.org/10.1016/j.annemergmed.2019.02.017.
40. Gammaitoni A, Gallagher RM, Welz-Bosna M. Topical ketamine gel: possible role in treating neuropathic pain. Pain Med. 2000;1(1):97–100. https://doi.org/10.1046/j.1526-4637.2000.00006.x.
41. Gewandter JS, Frazer ME, Cai X, et al. Extended-release gabapentin for failed back surgery syndrome: results from a randomized double-blind cross-over study. Pain. 2019;160(5):1029–36. https://doi.org/10.1097/j.pain.0000000000001478.
42. Goldberg H, Firtch W, Tyburski M, et al. Oral steroids for acute radiculopathy due to a herniated lumbar disk: a randomized clinical trial. JAMA. 2015;313(19):1915–23.
43. Guillot X, Prati C, Sondag M, Wendling D. Etanercept for treating axial spondyloarthritis. Expert Opin Biol Ther. 2017;17(9):1173–81. https://doi.org/10.1080/14712598.2017.1347156.
44. Gupta A, Kulkarni A, Ramanujam V, Zheng L, Treacy E. Improvement in chronic low back pain in an obese patient with topiramate use. J Pain Palliat Care Pharmacother. 2015;29(2):140–3. https://doi.org/10.3109/15360288.2015.1035837.

45. Hauk L. Spondyloarthritis: NICE releases guidelines on diagnosis and treatment. Am Fam Physician. 2017;96(10):677–8.
46. Ho KY, Gwee KA, Cheng YK, Yoon KH, Hee HT, Omar AR. Nonsteroidal anti-inflammatory drugs in chronic pain: implications of new data for clinical practice. J Pain Res. 2018;11:1937–48. Published 2018 Sep 20. https://doi.org/10.2147/JPR.S1681880.
47. Hwang CJ, Lee JH, Kim JH, et al. Gabapentin versus transdermal fentanyl matrix for the alleviation of chronic neuropathic pain of radicular origin: a randomized blind multicentered parallel-group noninferiority trial. Pain Res Manag. 2019;2019:4905013. Published 2019 Feb 4. https://doi.org/10.1155/2019/4905013.
48. Inage K, Orita S, Yamauchi K, et al. Low-dose tramadol and non-steroidal anti-inflammatory drug combination therapy prevents the transition to chronic low back pain. Asian Spine J. 2016;10(4):685–9. https://doi.org/10.4184/asj.2016.10.4.685.
49. Ivanova JI, Birnbaum HG, Kantor E, Schiller M, Swindle RW. Duloxetine use in chronic low back pain: treatment patterns and costs. PharmacoEconomics. 2012;30(7):595–609. https://doi.org/10.2165/11598130-000000000-00000.
50. Kanai A, Osawa S, Suzuki A, Ishimaru R, Hoka S. Effectiveness of prostaglandin E1 for the treatment of patients with neuropathic pain following herpes zoster. Pain Med. 2007;8(1):36–40. https://doi.org/10.1111/j.1526-4637.2007.00249.x.
51. Khoromi S, Patsalides A, Parada S, Salehi V, Meegan JM, Max MB. Topiramate in chronic lumbar radicular pain. J Pain. 2005;6(12):829–36. https://doi.org/10.1016/j.jpain.2005.08.002.
52. Klinger-Gratz PP, Ralvenius WT, Neumann E, et al. Acetaminophen relieves inflammatory pain through CB_1 cannabinoid receptors in the rostral ventromedial medulla. J Neurosci. 2018;38(2):322–34. https://doi.org/10.1523/JNEUROSCI.1945-17.2017.
53. Ko S, Kim S, Kim J, Oh T. The effectiveness of oral corticosteroids for management of lumbar radiating pain: randomized, controlled trial study. Clin Orthop Surg. 2016;8(3):262–7. https://doi.org/10.4055/cios.2016.8.3.262.
54. Koes BW, Backes D, Bindels PJE. Pharmacotherapy for chronic non-specific low back pain: current and future options. Expert Opin Pharmacother. 2018;19(6):537–45. https://doi.org/10.1080/14656566.2018.1454430.
55. Krebs EE, Gravely A, Nugent S, et al. Effect of opioid vs nonopioid medications on pain-related function in patients with chronic back pain or hip or knee osteoarthritis pain: the SPACE randomized clinical trial. JAMA. 2018;319(9):872–82. https://doi.org/10.1001/jama.2018.0899.
56. Lee JH, Lee CS, Ultracet ER Study Group. A randomized, double-blind, placebo-controlled, parallel-group study to evaluate the efficacy and safety of the extended-release tramadol hydrochloride/acetaminophen fixed-dose combination tablet for the treatment of chronic low back pain. Clin Ther. 2013;35(11):1830–40. https://doi.org/10.1016/j.clinthera.2013.09.017.
57. Lehrer S, Rheinstein PH. Nonsteroidal anti-inflammatory drugs (NSAIDs) reduce suicidal ideation and depression. Discov Med. 2019;28(154):205–12.
58. Lemming D, Sörensen J, Graven-Nielsen T, Lauber R, Arendt-Nielsen L, Gerdle B. Managing chronic whiplash associated pain with a combination of low-dose opioid (remifentanil) and NMDA-antagonist (ketamine). Eur J Pain. 2007;11(7):719–32. https://doi.org/10.1016/j.ejpain.2006.11.002.
59. Li J, Ajiboye RM, Orden MH, Sharma A, Drysch A, Pourtaheri S. The effect of ketorolac on thoracolumbar posterolateral fusion: a systematic review and meta-analysis. Clin Spine Surg. 2018;31(2):65–72. https://doi.org/10.1097/BSD.0000000000000613.
60. Ma K, Jiang W, Zhou Q, Du DP. The efficacy of oxycodone for management of acute pain episodes in chronic neck pain patients. Int J Clin Pract. 2008;62(2):241–7. https://doi.org/10.1111/j.1742-1241.2007.01567.x.
61. Malanga G, Reiter RD, Garay E. Update on tizanidine for muscle spasticity and emerging indications. Expert Opin Pharmacother. 2008;9(12):2209–15. https://doi.org/10.1517/14656566.9.12.2209.

62. Malanga GA, Ruoff GE, Weil AJ, Altman CA, Xie F, Borenstein DG. Cyclobenzaprine ER for muscle spasm associated with low back and neck pain: two randomized, double-blind, placebo-controlled studies of identical design. Curr Med Res Opin. 2009;25(5):1179–96. https://doi.org/10.1185/03007990902851753.
63. Martini A, Del Balzo G, Schweiger V, et al. Efficacy of lidocaine 5% medicated plaster (VERSATIS®) in patients with localized neuropathic pain poorly responsive to pharmacological therapy. Minerva Med. 2018;109(5):344–51. https://doi.org/10.23736/S0026-4806.18.05690-2.
64. Mathieson S, Kasch R, Maher CG, et al. Combination drug therapy for the management of low back pain and sciatica: systematic review and meta-analysis. J Pain. 2019;20(1):1–15. https://doi.org/10.1016/j.jpain.2018.06.005.
65. Mathieson S, Maher CG, McLachlan AJ, et al. Trial of pregabalin for acute and chronic sciatica. N Engl J Med. 2017;376(12):1111–20. https://doi.org/10.1056/NEJMoa1614292.
66. Mooney JJ, Pagel PS, Kundu A. Safety, tolerability, and short-term efficacy of intravenous lidocaine infusions for the treatment of chronic pain in adolescents and young adults: a preliminary report. Pain Med. 2014;15(5):820–5. https://doi.org/10.1111/pme.12333.
67. Mattozzi I. Trattamento conservative della radicolopatia cervicale con lidocaina cerotto 5% [Conservative treatment of cervical radiculopathy with 5% lidocaine medicated plaster]. Minerva Med. 2015;106(1):1–7.
68. Mayhew A, Argáez C. Intravenous lidocaine for chronic pain: a review of the clinical effectiveness and guidelines [Internet]. Canadian Agency for Drugs and Technologies in Health: Ottawa; 2018. Available from: https://www.ncbi.nlm.nih.gov/books/NBK531808/.
69. Miller JP, Schauer SG, Ganem VJ, Bebarta VS. Low-dose ketamine vs morphine for acute pain in the ED: a randomized controlled trial. Am J Emerg Med. 2015;33(3):402–8. https://doi.org/10.1016/j.ajem.2014.12.058.
70. Muehlbacher M, Nickel MK, Kettler C, et al. Topiramate in treatment of patients with chronic low back pain: a randomized, double-blind, placebo-controlled study. Clin J Pain. 2006;22(6):526–31.
71. Müller-Schwefe G, Morlion B, Ahlbeck K, et al. Treatment for chronic low back pain: the focus should change to multimodal management that reflects the underlying pain mechanisms. Curr Med Res Opin. 2017;33(7):1199–210. https://doi.org/10.1080/03007995.2017.1298521.
72. Nakashima H, Kanemura T, Ando K, et al. Is pregabalin effective against acute lumbar radicular pain?. Spine Surg Relat Res. 2018;3(1):61–6. Published 2018 Jun 29. https://doi.org/10.22603/ssrr.2018-0003.
73. Obata H. Analgesic mechanisms of antidepressants for neuropathic pain. Int J Mol Sci. 2017;18(11):2483. Published 2017 Nov 21. https://doi.org/10.3390/ijms18112483.
74. Onda A, Kimura M. Comparisons between the efficacy of limaprost alfadex and pregabalin in cervical spondylotic radiculopathy: design of a randomized controlled trial. Fukushima J Med Sci. 2018;64(2):73–81. https://doi.org/10.5387/fms.2018-07.
75. Orbai AM, Meyerhoff JO. The effectiveness of tricyclic antidepressants on lumbar spinal stenosis. Bull NYU Hosp Jt Dis. 2010;68(1):22–4.
76. Park CH, Jung SH, Han CG. Effect of intravenous lidocaine on the neuropathic pain of failed back surgery syndrome. Korean J Pain. 2012;25(2):94–8. https://doi.org/10.3344/kjp.2012.25.2.94.
77. Pareek A, Chandurkar N, Chandanwale AS, Ambade R, Gupta A, Bartakke G. Aceclofenac-tizanidine in the treatment of acute low back pain: a double-blind, double-dummy, randomized, multicentric, comparative study against aceclofenac alone. Eur Spine J. 2009;18(12):1836–42. https://doi.org/10.1007/s00586-009-1019-4.
78. Patetsos E, Horjales-Araujo E. Treating chronic pain with SSRIs: what do we know? Pain Res Manag. 2016;2016:2020915. https://doi.org/10.1155/2016/2020915.
79. Pathan H, Williams J. Basic opioid pharmacology: an update. Br J Pain. 2012;6(1):11–6.

80. Patil S, Anitescu M. Efficacy of outpatient ketamine infusions in refractory chronic pain syndromes: a 5-year retrospective analysis. Pain Med. 2012;13(2):263–9. https://doi.org/10.1111/j.1526-4637.2011.01241.x.
81. Perna A, Ricciardi L, Barone G, Tamburrelli FC, Proietti L, Pola E. Medical management of acute non-specific low back pain: comparison of different medical treatments, one center's retrospective analysis. J Biol Regul Homeost Agents. 2018;32(6 Suppl. 1):121–9.
82. Raphael JH, Southall JL, Gnanadurai TV, Treharne GJ, Kitas GD. Long-term experience with implanted intrathecal drug administration systems for failed back syndrome and chronic mechanical low back pain. BMC Musculoskelet Disord. 2002;3:17. https://doi.org/10.1186/1471-2474-3-17.
83. Rauck RL, Potts J, Xiang Q, Tzanis E, Finn A. Efficacy and tolerability of buccal buprenorphine in opioid-naive patients with moderate to severe chronic low back pain. Postgrad Med. 2016;128(1):1–11. https://doi.org/10.1080/00325481.2016.1128307.
84. Robertson JA, Purple RJ, Cole P, Zaiwalla Z, Wulff K, Pattinson KT. Sleep disturbance in patients taking opioid medication for chronic back pain. Anaesthesia. 2016;71(11):1296–307. https://doi.org/10.1111/anae.13601.
85. Roelofs PD, Deyo RA, Koes BW, Scholten RJ, van Tulder MW. Nonsteroidal anti-inflammatory drugs for low back pain. Cochrane Database Syst Rev. 2008;(1):CD000396.
86. Romman A, Salama-Hanna J, Dwivedi S. Mexiletine usage in a chronic pain clinic: indications, tolerability, and side effects. Pain Physician. 2018;21(5):E573–9.
87. Rossi M, Ianigro G, Liberatoscioli G, et al. Eperisone versus tizanidine for treatment of chronic low back pain. Minerva Med. 2012;103(3):143–9.
88. Safaeian P, Mattie R, Hahn M, Plastaras CT, McCormick ZL. Novel treatment of radicular pain with a multi-mechanistic combination topical agent: a case series and literature review. Anesth Pain Med. 2016;6(2):e33322. Published 2016 Mar 1. https://doi.org/10.5812/aapm.33322.
89. Saragiotto BT, Machado GC, Ferreira ML, Pinheiro MB, Abdel Shaheed C, Maher CG. Paracetamol for low back pain. Cochrane Database Syst Rev. 2016;(6):CD012230.
90. Schreijenberg M, Koes BW, Lin CC. Guideline recommendations on the pharmacological management of non-specific low back pain in primary care – is there a need to change? Expert Rev Clin Pharmacol. 2019;12(2):145–57. https://doi.org/10.1080/17512433.2019.1565992.
91. Shanthanna H, Gilron I, Rajarathinam M, et al. Benefits and safety of gabapentinoids in chronic low back pain: a systematic review and meta-analysis of randomized controlled trials. PLoS Med. 2017;14(8):e1002369.
92. Schliessbach J, Siegenthaler A, Bütikofer L, et al. Effect of single-dose imipramine on chronic low-back and experimental pain. A randomized controlled trial. PLoS One. 2018;13(5):e0195776. Published 2018 May 9. https://doi.org/10.1371/journal.pone.0195776.
93. Skljarevski V, Liu P, Zhang S, Ahl J, Martinez JM. Efficacy and safety of duloxetine in patients with chronic low back pain who used versus did not use concomitant nonsteroidal anti-inflammatory drugs or acetaminophen: a post hoc pooled analysis of 2 randomized, placebo-controlled trials. Pain Res Treat. 2012;2012:296710. https://doi.org/10.1155/2012/296710.
94. Stahl ST, Jung C, Weiner DK, Peciña M, Karp JF. Opioid exposure negatively affects antidepressant response to venlafaxine in older adults with chronic low back pain and depression [published online ahead of print, 2019 Oct 21]. Pain Med. 2019;pnz279. https://doi.org/10.1093/pm/pnz279.
95. Subramaniam K, Subramaniam B, Steinbrook RA. Ketamine as adjuvant analgesic to opioids: a quantitative and qualitative systematic review. Anesth Analg. 2004;99(2). https://doi.org/10.1213/01.ANE.0000118109.12855.07.
96. Taguchi T, Nozawa K, Parsons B, et al. Effectiveness of pregabalin for treatment of chronic cervical radiculopathy with upper limb radiating pain: an 8-week, multicenter prospective observational study in Japanese primary care settings. J Pain Res. 2019;12:1411–24. Published 2019 May 3. https://doi.org/10.2147/JPR.S191906.

97. Tanen DA, Shimada M, Danish DC, Dos Santos F, Makela M, Riffenburgh RH. Intravenous lidocaine for the emergency department treatment of acute radicular low back pain, a randomized controlled trial. J Emerg Med. 2014;47(1):119–24. https://doi.org/10.1016/j.jemermed.2012.12.014.
98. Tetsunaga T, Tetsunaga T, Tanaka M, Ozaki T. Efficacy of tramadol-acetaminophen tablets in low back pain patients with depression. J Orthop Sci. 2015;20(2):281–6. https://doi.org/10.1007/s00776-014-0674-4.
99. Tetsunaga T, Tetsunaga T, Tanaka M, Nishida K, Takei Y, Ozaki T. Effect of tramadol/acetaminophen on motivation in patients with chronic low back pain. Pain Res Manag. 2016;2016:7458534. https://doi.org/10.1155/2016/7458534.
100. Toth PP, Urtis J. Commonly used muscle relaxant therapies for acute low back pain: a review of carisoprodol, cyclobenzaprine hydrochloride, and metaxalone. Clin Ther. 2004;26(9):1355–67. https://doi.org/10.1016/j.clinthera.2004.09.008.
101. Tremont-Lukats IW, Challapalli V, McNicol ED, Lau J, Carr DB. Systemic administration of local anesthetics to relieve neuropathic pain: a systematic review and meta-analysis. Anesth Analg. 2005;101:1738–49.
102. Uhl RL, Roberts TT, Papaliodis DN, Mulligan MT, Dubin AH. Management of chronic musculoskeletal pain. J Am Acad Orthop Surg. 2014;22(2):101–10. https://doi.org/10.5435/JAAOS-22-02-101.
103. Urquhart DM, Wluka AE, van Tulder M, et al. Efficacy of low-dose amitriptyline for chronic low back pain: a randomized clinical trial [published correction appears in JAMA Intern Med. 2019;179(3):457]. JAMA Intern Med. 2018;178(11):1474–81. https://doi.org/10.1001/jamainternmed.2018.4222.
104. van Tulder MW, Touray T, Furlan AD, Solway S, Bouter LM. Muscle relaxants for nonspecific low back pain. Cochrane Database Syst Rev. 2003;(2):CD004252.
105. Vorsanger GJ, Xiang J, Gana TJ, Pascual ML, Fleming RR. Extended-release tramadol (tramadol ER) in the treatment of chronic low back pain. J Opioid Manag. 2008;4(2):87–97. https://doi.org/10.5055/jom.2008.0013.
106. Wang R, Bathon JM, Ward MM. Nonsteroidal Antiinflammatory Drugs as Potential Disease-Modifying Medications in Axial Spondyloarthritis [published online ahead of print, 2019 Nov 9]. Arthritis Rheumatol. 2019. https://doi.org/10.1002/art.41164.
107. Watson JC, Sandroni P. Central neuropathic pain syndromes. Mayo Clin Proc. 2016;91(3):372–85. https://doi.org/10.1016/j.mayocp.2016.01.017.
108. Wen W, Sitar S, Lynch SY, He E, Ripa SR. A multicenter, randomized, double-blind, placebo-controlled trial to assess the efficacy and safety of single-entity, once-daily hydrocodone tablets in patients with uncontrolled moderate to severe chronic low back pain. Expert Opin Pharmacother. 2015;16(11):1593–606. https://doi.org/10.1517/14656566.2015.1060221.
109. Wiffen PJ, Derry S, Bell RF, et al. Gabapentin for chronic neuropathic pain in adults. Cochrane Database Syst Rev. 2017;6(6):CD007938. Published 2017 Jun 9. https://doi.org/10.1002/14651858.CD007938.pub4.
110. Wiffen PJ, Knaggs R, Derry S, Cole P, Phillips T, Moore RA. Paracetamol (acetaminophen) with or without codeine or dihydrocodeine for neuropathic pain in adults. Cochrane Database Syst Rev. 2016;12(12):CD012227. Published 2016 Dec 27. https://doi.org/10.1002/14651858.CD012227.pub2.
111. Wren K, Lancaster R, Walesh M, Margelosky K, Leavitt K, Hudson A, Albala M. Intravenous lidocaine for relief of chronic neuropathic pain. AANA J. 2019;87(5):351–5.
112. Yildirim K, Deniz O, Gureser G, et al. Gabapentin monotherapy in patients with chronic radiculopathy: the efficacy and impact on life quality. J Back Musculoskelet Rehabil. 2009;22(1):17–20. https://doi.org/10.3233/BMR-2009-0210.
113. Zhou M, Chen N, He L, Yang M, Zhu C, Wu F. Oxcarbazepine for neuropathic pain. Cochrane Database Syst Rev. 2017;12(12):CD007963. Published 2017 Dec 2. https://doi.org/10.1002/14651858.CD007963.pub3.

Interdisciplinary Pain Management

9

Richard C. Robinson

Introduction

Pain is a complex sensory and emotional experience that impacts multiple areas of a person's life the longer it persists [4]. As pain becomes chronic, longer than 3–6 months, it impacts multiple psychological and social areas with increasing severity [4]. Specifically, it impacts cognitive domains: attention, concentration, executive functioning and word finding [55]. Chronic pain also impacts mood – increasing the chances of depressed mood and anxiety [5]. Lastly chronic pain has tremendous social effects with regard to work, interpersonal functioning and sense of identity [4]. Chronic pain does not only impact an individual, but also impacts society at large with regard to healthcare costs and lost productivity [3].

Given the complex nature of pain as it becomes chronic, the approach that considers biological, psychological and social impacts of pain – the biopsychosocial approach – remains the most useful manner to address these complex set of physical, emotional and cognitive sequelae [4]. Therefore, to treat a complex condition such as chronic pain requires expertise in each of these facets. The interdisciplinary approach to pain management – where multiple disciplines work in a cohesive fashion in one setting – is arguably the best manner in which to address these complex set of factors [26].

Pain is a debilitating condition that impacts over 100 million people in the United States [3]. Furthermore, the cost of pain is estimated to be somewhere between $500 and $635 billion dollars a year when direct medical costs and lost productivity are taken into account [3]. Furthermore, pain is more costly than any other health condition in the United States aside from cardiovascular disease [3]. Although hundreds of billions of dollars is spent on the treatment of pain a year, the prevalence of pain

R. C. Robinson (✉)
Division of Psychology, Department of Psychiatry, UT Southwestern Medical Center, Dallas, TX, USA
e-mail: Richard.Robinson@UTSouthwestern.edu

© The Author(s), under exclusive license to Springer Nature Switzerland AG 2022

C. E. Noe (ed.), *Multidisciplinary Spine Care*,
https://doi.org/10.1007/978-3-031-04990-3_9

continues to rise, especially with regard to the most common type of pain, chronic low back pain (CLBP) [3].

Pain is meant to serve a biologically adaptive function. Specifically, it alerts one to an injury or illness when it is serving its evolutionary purpose [7]. An individual with a broken leg is alerted by pain to take pressure of the leg, or an individual with an infection is notified by pain to treat the injury. However, the longer pain persists, the less adaptive it becomes especially when there is no clear biological benefit for the pain and it simply interferes with an individual's life [4]. It is an axiom that the longer pain persists, the more important the role of psychosocial factors become in the maintenance and aggravation of pain.

Biopsychosocial

The biopsychosocial approach to chronic illness was first described by George Engel – an internist and psychiatrist at the University of Rochester. This approach is meant to supplement, but not replace, the traditional biomedical model of pain [12]. With regard to the biomedical model of pain, pain is assumed to correspond with the degree of tissue damage. From this model, concepts of "functional" vs. "organic" pain developed which suggested that pain has a biological origin in the case of "organic" pain or it is serving some type of psychological/or other "function" with regard to "functional pain." In other words, the biomedical model assumes pain is either "physical" or "psychological" [7]. This perspective is based on the specificity theory of pain developed by Rene Descartes, which presupposes that the amount of tissue damage should equate to the amount of pain a person experiences [7]. This has led to the unfortunate distinctions mentioned above that could also be summarized as "real" versus "fake" pain by those unfamiliar with the complex nature of pain once it becomes chronic.

Although the biomedical model of pain has led to many advancements in the treatment of pain, one could argue that it has struggled to address the growing epidemic of chronic pain. As mentioned, the biopsychosocial model of pain is not meant to exclude the importance of biological factors such as illness and injury in the experience of pain [4]. Rather, it is meant to elaborate the conception of pain to include psychological and contextual factors. As most practitioners who work with individuals with chronic pain understand, the psychological and contextual factors can serve to impair improvement in the experience of pain [7].

Beecher was one of the first researchers to note how the impact of psychosocial factors on the experience of pain [7]. He worked as a surgeon during World War II in Anzio Italy. He noted that 20% of combat soldiers required powerful analgesics despite not being in shock and with serious injuries [10]. It was concluded that the meaning of the pain, which could be associated with no longer being in danger, was not seen as the alarm that others might have experienced it if they were going to be sent back to life-threatening situations [10]. Other studies have found the importance of mood on pain, with better moods being associated with higher pain tolerance than individuals who were experiencing dysphoric or anxious mood states [5].

The role of cognition has also been long understood to play a role in the pain experience. In fact, when individuals are trained in simple distraction techniques their pain tolerance, as measured by the cold pressor tests increase. In addition, self-talk regarding the nature of pain has long been recognized to play a role in the pain experience [5].

Neuroscience of Pain

Our understanding of the neuroscience of pain has grown exponentially over the last 20 years [15]. Although the insight of the Gate Control Theory of pain remains relative today, we have a much clearer understanding of pain pathways and are beginning to understand the way in which the brain changes in response to effective treatment of pain.

Most patients are surprised to learn that they have no pain receptors, but rather nociceptors. These nociceptors – when stimulated at a sufficient intensity and interpreted by the brain as dangerous – often translate to the experience of pain [13]. An essential component of interdisciplinary care is education and helping patients understand that tissue damage does not always equate to pain and that there can be pain without ongoing tissue damage [4, 13]. For instance, in our interdisciplinary pain program we often use the example of cutting oneself in the garden or garage and not even noticing an injury until you see blood. Also, most people are familiar with the concept of phantom limb pain – where a person experiences pain but without nociception.

Understanding the basic neuroscience of pain is an essential element to managing pain and allows individual to gain a better grasp of their symptoms. As mentioned, "Pain is a complex sensory and emotional experience that can vary widely between people and even with an individual depending on the context and meaning of the pain and the psychological state of the person" [5].

The Gate Control Theory of Pain ushered in the psychosocial aspects of our understanding of pain. The theory developed by Melzack and Wall postulated that both peripheral nerve signals and descending signals from the brain could impact an interneuron in the dorsal horn of the spinal column which served a gate-like mechanism [14]. With regard to peripheral nerve signals, rubbing one's leg after bumping it would serve to partially close this gate like mechanism. Descending nerve fibers from the periaquaductal gray area of the brain could also serve to partly mitigate the transmission of signals within someone who was in a pleasant mood or who was distracted, as just two examples [14].

However, with the development of newer technologies, our understanding of the neuroscience of pain has continued to grow [15]. When nociceptive signals pass through the gate-like mechanism of the dorsal horn of the spinal column they first go to the thalamus of the brain – the major relay station of the brain. Projections then connect the signals from the thalamus to the primary and secondary somatosensory cortex that process where a sensation is occurring in the body as well as the texture of the sensation, e.g., an itch or a crunch [15]. However, if someone were

only to experience nociceptive signals in these areas of the brain, they might be at risk of interpreting the signals as more of a crunching than that of a noxious signal [18].

Pain becomes "pain" in the limbic systems of the brain. The limbic system is the pain and emotional processing center of the brain [15]. Although the idea of a limbic "system" is an area of debate within neuroscience, it remains a useful concept regarding the processing of nociceptive signals from the body. Specifically, the limbic system quickly categorizes external and internal input as positive or negative [17]. For instance, a pleasant meal may quickly be categorized by the positive valence system, while a cut to the leg may be processed by the negative valence system.

The limbic system that includes areas of the brain such as the hippocampus, amygdala, basal ganglia, interior cingulate cortex and anterior cingulate cortex [17]. Christopher DeCharms, Sean Mackey and colleagues conducted a series of studies that allowed participants to see activity in their anterior cingulate cortex (ACC) [18]. Through distraction and other commonly practiced pain management techniques subjects were able to decrease activation in the ACC and reduce the pain they experienced [18]. Individuals with chronic pain were able to decrease their pain by 65% and individuals where pain was induced were able to decrease their pain by 25% [18]. Furthermore, Bushnell and colleagues demonstrated in one study the different parts of the brain that were involved in the management of pain [5]. Specifically, in their study they found that attentional modulation was related to the superior parietal lobe, somatosensory cortex and insula. However, emotional control was related to the periaqueductal grey, and prefrontal cortex [5]. Furthermore, these same areas of the brain demonstrate functional changes when an individual engages in cognitive-behavioral therapy. Finally, newer research is demonstrating changes in grey matter in some of the same areas described above when pain improves, but this work remains preliminary [56].

Psychological Impact of Pain

As mentioned, pain is a complex sensory and emotional experience, and the longer pain persists the greater the impact on an individual's life [4]. Anecdotally, individuals with pain may begin to experience difficulty with sleep, difficulty concentrating and lower distress tolerance. As pain persists, it appears to impact more and more areas of an individual's life. As concentration and sleep become impacted, it is not a surprise that performance at work or school may begin to suffer. As difficulty in occupation functioning increases, one's sense of self may begin to diminish.

The most apt analogy to an emotion regarding pain is to that of anxiety. Anxiety serves as a signal that indicates danger in the external or internal world [17]. Pain serves a similar function as it is meant to signal damage to tissue or an illness that requires attention or a change in behavior [13]. However, the consequences of persistent pain are more commonly associated with depression, with estimates as high of 58% of individuals suffering from major depressive disorder [23].

Cognitive consequences of pain appear clear with regard to attention and concentration, but our understanding of the impact of pain is often confounded by co-occurring factors, such factors include difficulty with sleep, medication and mood state [19]. Working memory also appears impaired by pain, especially regarding working memory involving visual systems [19].

Executive functioning refers to the concept of control and reasoning. However, it should be noted that difficulties in one area of cognitive faculties, such as attention and concentration, may inevitably lead to difficulties with executive functioning to some extent. Executive functioning, "refers to our ability to problem solve, learn from errors and organize input as well as our response" [19]. Evidence of the negative impact of pain on cognitive functioning is observed with the Iowa Gambling Task where individuals attempt to win money by engaging in a simulated gambling activity [20]. This test has been shown to correlate with aspects of executive functioning and emotional problems solving [20].

Consistent with the impact of pain on cognitive functioning, various neuroimaging and testing techniques have also begun to note evidence of anatomical changes within the brain [56]. Findings from the neuroscience literature have noted decreases in grey matter in areas central in processing both pain and emotion. Specifically, changes in grey matter have been noted in the anterior cingulate cortex, insula cortex and prefrontal cortex. Some evidence also suggests change in white matter in similar regions [5]. The exact cause and mechanisms of these changes remain unclear, but some researches have hypothesized it is due to neuroexcitation of these circuits over time [5].

As previously mentioned, the emotional impact chronic pain is self-evident to anyone who has had pain for more than 3–6 months. It should be noted that normal reactions of frustration, irritability and worry are not considered psychological conditions. However, as pain continues to tax coping resources and has more detrimental effects an estimated 59% of individuals develop a disorder that meet full criteria for a behavioral health disorder [22]. Previous studies have reported rates of major depressive disorder that range from 34% to 58% [22–24]. When adjustment disorder with depressed mood is also taken into consideration, these numbers rise. Also, in one study an estimated 35% of individuals suffered from an anxiety disorder [24].

Psychological Treatment of Pain

Before proceeding to a more specific explication of how the biopsychosocial approach to pain management is implemented through an interdisciplinary pain management program, a brief review of treatment of pain from a psychological perspective is warranted. The approach taken for many pain practitioners can be summarized as a "top – down" and "bottom – up" approach to pain management. Specifically, "top – down" interventions refer to interventions such as providing education regarding the nature of pain, distinction between the concepts of "hurt" versus "harm" and providing other useful information, including education on general self-care, sleep, appropriate exercise and nutrition [4, 36]. The "top – down"

approaches also include skills training in techniques that have proven to be effective to manage pain, such as mindfulness meditation, relaxation training, pacing, hypnosis, etc. Approaches categorized as "bottom – up" utilize more traditional therapy techniques to assist in addressing maladaptive thoughts, feelings and beliefs regarding pain [4].

Cognitive-behavioral therapy remains the "gold-standard" for the treatment of chronic pain, but increasing evidence supports the use of therapies such as acceptance commitment therapy and emotional awareness and expressive therapy [4, 50, 51]. Cognitive-behavioral therapy posits that the functioning of the clinically relevant aspects of a person can be divided into thoughts, feelings and behaviors [43]. Also these elements of a person are considered to be bi-directional and operate within a system. Therefore, changing one element of the system – thoughts, feelings or behaviors – can change the functioning of the other elements. For example, addressing what are commonly referred to as "automatic negative thoughts" testing them to reasoning and experimentation can lead to more balance, objective and realistic thoughts [43]. Also, finding ways to reinforce adaptive behavior could impact thinking and feeling.

Turk and Gatchel described goals that have been shown to be helpful for individuals suffering from chronic pain [4]. The first goal is to help an individual with chronic pain begin to identify and change thoughts they may be having about their pain from something that feels out of their control to something that could be impacted positively. The next goal is what was previously described as the "top-down" techniques, where individuals learn how to develop self-regulation over some of their physiological functioning through practices such as relaxation training, biofeedback and mindfulness meditation [4].

The remaining three goals involve improving and strengthening coping and resiliency. Specifically, to help individuals move to more active problem-solving approaches and promote a sense of self-efficacy where they feel that there are options to help manage pain. The last two goals involve the ongoing ability to assess the interaction among their thoughts feelings and behaviors. Lastly, the intended outcome is for an individual to be able to apply the armamentarium of tools in multiple settings in flexible and adaptive manners. For instance, finding ways to pace or meditate at work or during other activities of daily living [4].

Interdisciplinary Care

The biopsychosocial approach to approach to pain includes respect of the biological, psychological and social aspects of pain and posits that to effectively treat pain, all three elements, and sub-elements, must be attended to and addressed as much as feasible [26]. Anecdotally, practitioners unfamiliar with practicing in this model may assume that physical pathology is minimized in favor of psychosocial factors. However, that is an inaccurate assumption and physician's typically lead the interdisciplinary programs. Although we now know that there is a weak correlation between the amount of tissue damage/physical pathology with the experience of

chronic pain, there remains a correlation [7]. Medical staff not only lead the interdisciplinary team in most settings but may order additional diagnostic tests and engage different medical specialties. In addition, medication and interventions may also be recommended and implemented withing the context of interdisciplinary care [26].

Along those lines, physical therapy is an essential element of interdisciplinary care. As most reading this will know, is essential to addressing physical contributors to pain through traditional modalities related to graded exercise, stretching and strengthening [39]. Through the years, other techniques have been refined to assist in the decrease of pain and allow for more progressively challenging activity, e.g., dry-needling, craniosacral therapy, etc. However, physical therapy plays a major role in the psychosocial aspect of treatment as well. Specifically, physical therapy is crucial in helping some individuals struggling with kinesiophobia, fear of movement, and avoidance of activity [38]. Also, physical therapy helps promote a sense of self-efficacy mentioned previously [38].

A distinction is often made between multidisciplinary and interdisciplinary, although this distinction is not readily adopted within the pain management field. However, there are important differences. Specifically, interdisciplinary care involves the delivery of care in one setting, allowing staff and faculty to collaborate in both formal (e.g., case conferences) and informal ways. Multidisciplinary refers to the delivery of services from multiple disciplines, but at disparate locations, making collaboration somewhat more challenging [26].

The evidence for the effectiveness of interdisciplinary pain management has accumulated over decades. Functional restoration is a type of interdisciplinary care that was developed for individuals receiving Worker's Compensation benefits who had failed multiple other treatments. In a landmark study, Mayer, Gatchel et al. compared individuals who had undergone a functional restoration program over the course of 2 years [48]. They found that 87% of individuals who had undergone functional restoration were working at 2-years compared to 41% of the comparison group who received treatment as usual. In addition, the investigators found twice as many surgeries in the comparison group than the functional restoration group. Furthermore, the comparison group engaged in five times more health care visits and were more likely to be reinjured [48]. These findings were replicated both in the US and abroad over three decades [26].

Friederich and colleagues evaluated 93 individuals with chronic low back pain and compared standard exercise to interdisciplinary care [57]. They found that even after 5 years individuals who engaged in interdisciplinary care reported lower pain and disability than individuals who engaged in standard exercise [57].

Fairbank and colleagues engaged in a multicenter randomized control trial for 349 individuals with chronic low back pain and compared spinal fusion to interdisciplinary pain management [58]. Both groups showed improvement with regard to pain ratings and disability on self-report measures. However, the cost of interdisciplinary care was almost half of the cost of spinal fusion [58].

Other efforts examining interdisciplinary care have focused on the intensity of treatment. Skouen and colleagues examined 195 clients with chronic low back pain

and compared a "light" interdisciplinary program, to a more intensive interdisciplinary program to treatment as usual [59]. The investigators found that interdisciplinary care had better outcomes than treatment as usual. Furthermore, they found that there was not differences between light or intensive interdisciplinary pain programs [59].

The long-term effectiveness of interdisciplinary care was also reinforced by the work of Oslund and colleagues through an intensive interdisciplinary pain program that combined physical therapy, cognitive-behavioral therapy, occupational therapy and intensive case management [60]. As one would expect, there were significant improvements in both pain reduction and hours resting after the four-week program. However, these gains were also maintained after 1 year [60].

The cost effectiveness of interdisciplinary care has also been evaluated. For instance, Gatchel and Okifuji found that annual medical costs were reduced by 68% in individuals who engaged in interdisciplinary care [61]. Furthermore, when the costs saving were extrapolated to evaluate the difference between individuals who underwent conventional care compared to interdisciplinary care, the results reflected a significant cost savings [61].

Conclusion

Chronic pain is a costly and debilitating medical condition that impacts multiple areas of an individuals life. The biopsychosocial approach to pain management builds upon the traditional biomedical model and it expands the area of focus to also include psychological and social functioning. The evidence for the effectiveness of interdisciplinary care to address the underlying biological, psychological and social factors have accumulated over decades to demonstrate the effectiveness of this approach.

References

1. Institute of Medicine: Committee on Advancing Pain Research Care, and Education. Relieving pain in America: a blueprint for transforming prevention, care, education, and research. The National Academies Collection; 2011.
2. Prevalence of chronic pain and high-impact chronic pain among adults—United States, 2016 [Internet]. 2016.
3. Gaskin DJ, Richard P. The economic costs of pain in the United States. J Pain. 2012;13(8):715–24.
4. Turk DC, Gatchel RJ. Psychological approaches to pain management: a practioner's handbook. 2nd ed. New York: The Guilford Press; 2002.
5. Bushnell MC, Ceko M, Low LA. Cognitive and emotional control of pain and its disruption in chronic pain. Nat Rev. 2013;14:502–11.
6. Gatchel RJ, Peng YB, Peters ML, Fuchs PN, Turk DC. The biopsychosocial approach to chronic pain: scientific advances and future directions. Psychol Bull. 2007;133(4):581.
7. Baum A, Gatchel RJ, Krantz DS. An introduction to health psychology. Boston: McGraw Hill; 1997.

8. Drossman DA. Functional versus organic: an inappropriate dichotomy for clinical care. Am J Gastroenterol. 2006;101(6):1172–5.
9. Breuer J, Freud S. Studies in hysteria. New York: Nervous and Mental Disease Publishing; 1937.
10. Beecher HK. Pain in men wounded in battle. Ann Surg. 1946;123:96–105.
11. Bingel U. The effect of treatment expectation on drug efficacy: imaging the analgesic benefit of the opioid remifentanil. Sci Transl Med. 2011;3:7–14.
12. Engel GL. The need for a new medical model: a challenge for biomedicine. Science. 1977;196(4286):129–36.
13. Butler D, Moseley L. Explain pain. Adelaide City West: Noigroup Publications; 2003.
14. Melzack R, Wall PD. Pain mechanisms: a new theory. Science. 1965;150(3699):971–9.
15. Gatchel RJ, Robinson RC, Peng YB, Benitez OJ. Pain and the brain: a synthesis of the current understanding of brain response to pain learned from functional magnetic resonance imaging (fMRI). Pract Pain Manag. 2011;8(5).
16. Villemure C, Bushnell MC. Mood influences supraspinal pain processing separately from attention. J Neurosci. 2009;29:705–15.
17. Siegel DJ. The developing mind: how relationships and the brain interact to shape who we are. 2nd ed. New York: Guilford Press; 2012.
18. deCharms RC, Maeda F, Glover GH, Ludlow D, Pauly JM, Soneji D, et al. Control over brain activation and pain learned by using real-time functional MRI. Proc Natl Acad Sci. 2005;102(51):1826–31.
19. Moriarty O, McGuire BE, Finn DP. The effect of pain on cognitive function: a review of clinical and preclinical research. Prog Neurobiol. 2011;93(3):385–404.
20. Tamburin S, Maier A, Schiff S, Lauriola MF, Di Rosa E, Zanette G, et al. Cognition and emotional decision-making in chronic low back pain: an ERPs study during Iowa gambling task. Front Psychol. 2014;5:1350.
21. Whitlock EL, Diaz-Ramirez LG, Glymour MM, Boscardin WJ, Covinsky KE, Smith AK. Association between persistent pain and memory decline and dementia in a longitudinal cohort of elders. JAMA Intern Med. 2017;177(8):1146–53.
22. Polatin PB, Kinney RK, Gatchel RJ, Lillo E, Mayer TG. Psychiatric illness and chronic low back pain: the mind and the spine-which goes first? Spine. 1993;18(1):66–71.
23. Fishbain DA, Cutler RB, Rosomoff HL, et al. Chronic-pain associated depressions: antecedt or consequence of chronic pain? Clin J Pain. 1997;13:116–37.
24. McWilliams LA, Cox BJ, Enns MW. Mood and anxiety disorders associated with chronic pain: an examination in a nationally representative sample. Pain. 2003;106:127–33.
25. Robinson RC. Psychometric testing: the early years and the MMPI. In: Gatchel RJ, Weisberg J, editors. Personality characteristics of patients with pain. Washington, DC: American Psychological Association; 2000.
26. Gatchel RJ, McGeary DD, McGeary CA, Lippe B. Interdisciplinary chronic pain management: past, present, and future. Am Psychol. 2014;69(2):119–30. https://doi.org/10.1037/a0035514.
27. Fields HL. The doctor's dilemma: opiate analgesics and chronic pain. Neuron. 2011;69(4):591–4.
28. Von Korff M, Kolodny A, Deyo RA, Chou R. Long-term opioid therapy reconsidered. Ann Intern Med. 2011;155:325–8.
29. Lee M, Silverman SM, Hansen H, Patel VB, Manchikanti L. A comprehensive review of opioid-induced hyperalgesia. Pain Physician. 2011;14(2):145–61.
30. Chou R, Turner JA, Devine EB, Hansen RN, Sullivan SD, Blazina I, et al. The effectiveness and risks of long-term opioid therapy for chronic pain: a systematic review for a National Institutes of Health Pathways to Prevention Workshop. Ann Intern Med. 2015;162(4):276–86.
31. Robinson RC, Gatchel RJ, Polatin PB, Deschner M, Gajraj N, Noe C. Screening for problematic opioid behavior. Clin J Pain. 2001;17:220–8.
32. Weir K. New solutions for the opioid crisis: how psychologists improve care. Monit Psychol. 2019;50(6):32–9.

33. Gatchel RJ, Theodore BR. Evidence-based outcomes in pain research and clinical practice. Pain Pract. 2008;8(6):452–60.
34. Chou R. 2009 Clinical guidelines from the American Pain Society and the American Academy of Pain Medicine on the use of chronic opioid therapy in chronic noncancer pain: what are the key messages for clinical practice? Polskie Archiwum Medycyny Wewnetrznej. 2009;119(7–8):469–77.
35. Jensen MC, Brant-Zawadzki MN, Obuchowski N, Modic MT, Malkasian D, Ross JS. Magnetic resonance imaging of the lumbar spine in people without back pain. N Engl J Med. 1994;331(2):69–73.
36. Fordyce WE. Behavioral methods for chronic pain and illness. St. Louis: C. V. Mosby Co.; 1976.
37. Gatchel RJ, Robinson RC, Block AR, Benedtto NN. Assessment of pain in primary care settings. In: Maruish ME, editor. Handbook of psychological assessment in primary care settings. New York: Routledge – Taylor & Francis Group; 2017.
38. Vlaeyen JW, Linton SJ. Fear-avoidance model of chronic musculoskeletal pain: 12 years on. Pain. 2012;153(6):1144–7.
39. Mior S. Exercise in the treatment of chronic pain. Clin J Pain. 2001;17(4 Suppl):S77–85.
40. Wandner LD, Prasad R, Ramezani A, Malcore SA, Kerns RD. Core competencies for the emerging specialty of pain psychology. Am Psychol. 2019;74(4):432–44.
41. Taylor SE. Health Psychology. 9th ed. Guilford: New York; 2015.
42. Carver CS, Scheier MF, Fulford D. Self-regulatory processes, stress, and coping. In: John OP, Robins RW, Pervin LA, editors. Handbook of personality theory and research. 3rd ed. New York: Guilford; 2008.
43. Beck AT. Thinking and depression. II. Theory and therapy. Arch Gen Psychiatry. 1964;10:561–71.
44. Turk DC, Rudy TE. Assessment of cognitive factors in chronic pain: a worthwhile enterprise? J Consult Clin Psychol. 1986;54(6):760–8.
45. Lefebvre MF. Cognitive distortion and cognitive errors in depressed psychiatric and low back pain patients. J Consult Clin Psychol. 1981;49(4):517–25.
46. Sullivan MJ, Thorn B, Haythornthwaite JA, Keefe F, Martin M, Bradley LA, et al. Theoretical perspectives on the relation between catastrophizing and pain. Clin J Pain. 2001;17(1):52–64.
47. Wertli MM, Burgstaller JM, Weiser S, Steurer J, Kofmehl R, Held U. Influence of catastrophizing on treatment outcome in patients with nonspecific low back pain: a systematic review. Spine (Phila Pa 1976). 2014;39(3):263–73.
48. Mayer TG, Gatchel RJ, Mayer H, Kishino N, Kelley J, Mooney VA. Prospective two-year study of functional restoration in industrial low back pain. J Am Med Assoc. 1987;258:1181–2.
49. Speed TJ, Parekh V, Coe W, Antoine D. Comorbid chronic pain and opioid use disorder: literature review and potential treatment innovations. Int Rev Psychiatry. 2018;30(5):136–46.
50. McCracken LM, Vowles KE. Acceptance and commitment therapy and mindfulness for chronic pain: model, process, and progress. Am Psychol. 2014;69(2):178–87.
51. Lumley MA, Schubiner H, Lockhart NA, Kidwell KM, Harte SE, Clauw DJ, et al. Emotional awareness and expression therapy, cognitive behavioral therapy, and education for fibromyalgia: a cluster-randomized controlled trial. Pain. 2017;158(12):2354–63.
52. Andrasik F, Flor H, Turk DC. An expanded view of psychological aspects in head pain: the biopsychosocial model. Neurol Sci. 2005;26(Suppl 2):s87–91.
53. Veehof MM, Trompetter HR, Bohlmeijer ET, Schreurs KM. Acceptance- and mindfulness-based interventions for the treatment of chronic pain: a meta-analytic review. Cogn Behav Ther. 2016;45(1):5–31.
54. Sollner W, Schussler G. Psychodynamic therapy in chronic pain patients: a systematic review. Z Psychosom Med Psychother. 2001;47(2):115–39.
55. Block C, Cianfrini L. Neuropsychological and neuroanatomical sequelae of chronic non-malignant pain and opioid analgesia. NeuroRehabilitation. 2013;33:343–66.

56. Rodriguez-Raecke R, Niemeier A, Ihle K, Ruether W, May A. Structural brain changes in chronic pain reflect probably neither damage nor atrophy. PLoS One. 2013;8(2):e54475. https://doi.org/10.1371/journal.pone.0054475.
57. Friedrich M, et al. Long-term effect of a combined exercise and motivational program on the level of disability of patients with chronic low back pain. Spine. 2005;30:995–1000.
58. Fairbank J, et al. Randomised controlled trial to compare surgical stabilisation of the lumbar spine with an intensive rehabilitation programme for patients with chronic low back pain: the MRC spine stabilsation trial. Br Med J. 2005;330:1233.
59. Skouen JS, et al. Relative cost-effectiveness of extensive and light multidisciplinary treatment programs versus treatment as usual for patient with chronic low back pain on long-term sick leave: randomized controlled study. Spine. 2002;27(9):901–9.
60. Oslund S, Robinson RC, et al. Long-term effectiveness of a comprehensive pain management program: strengthening the case for interdisciplinary care. Proc Baylor Univ Med Cent. 2011;22(3):211–4.
61. Gatchel RJ, Okifuji A. Evidence-based scientific data documenting the treatment and cost-effectiveness of comprehensive pain programs for chronic malignant pain. J Pain. 2006;7(11):779–93.

Spine Injections in the Management of Painful Spinal Conditions

10

Stephanie Jones, Ivan N. Chew, and Judy Yang

Introduction

Spinal pain conditions are extremely prevalent. In fact, low back pain is the most common cause of activity limitation and work absence throughout much of the world [1]. Studies show that low back pain causes more global disability than any other condition [2]. Unlike many other conditions, there are multiple pain generators that can contribute to low back pain, sometimes complicating the treatment approach. In an effort to identify the source of pain and develop an appropriate treatment, low back pain can be categorized according to symptoms and/or chronicity. One of the most common ways to separate low back pain is based on radicular or axial complaints. Several conservative treatment modalities can be successfully utilized in the treatment of lumbar spinal pain conditions. However, when such interventions fail to provide adequate relief, specialists can offer more invasive options such as targeted spinal injections in the treatment of both axial and radicular spinal pain syndromes.

Neck pain is also a common cause of disability, ranking fourth in the USA as one of the most common disabling health conditions [3]. Nearly 50% of the general population will succumb to cervicalgia (neck pain) during their lifetime [4]. The treatment of cervical spine pain is similar to low back pain, and interventional spine specialists often utilize similar spinal injections for neck pain as for low back pain.

The goal of this chapter is to review the most common spinal injections utilized in the treatment of various spinal pain conditions, including a review of the evidence supporting the continued use of these injections, and also a review of potential complications from these minimally invasive spinal injections.

S. Jones (✉) · I. N. Chew · J. Yang
Department of Anesthesiology and Pain Management, University of Texas Southwestern Medical Center, Dallas, TX, USA
e-mail: JONES@UTSOUTHWESTERN.EDU; Ivan.chew@utsouthwestern.edu; Judy.yang@utsouthwestern.edu

© The Author(s), under exclusive license to Springer Nature Switzerland AG 2022

C. E. Noe (ed.), *Multidisciplinary Spine Care*,
https://doi.org/10.1007/978-3-031-04990-3_10

Epidural Steroid Injections

Background

Therapeutic substances such as combinations of saline and various local anesthetics have been injected into the lumbar epidural space for over a hundred years in an attempt to treat lumbar spine pain. However, it was not until 1953 that corticosteroids were first administered to the lumbar epidural space to manage lumbar radicular pain by Lievre et al. [5]. Later, the first controlled trial evaluating the efficacy of epidural steroid injections was performed by Swerdlow and Sayle-Creer in 1970 [6].

Mechanism of Action

The mechanism of action of steroids in the treatment of low back pain has been debated for years. Corticosteroids inhibit phospholipase A2, an inflammatory mediator found in high concentrations in herniated discs [7]. However, the primary role of phospholipase A2 is its involvement in the conversion of arachidonic acid to inflammatory prostaglandins, prostacyclins, thromboxanes, and leukotrienes. These mediators increase pain and inflammation, and excite peripheral nociceptors. Through inhibition of phospholipase A2, such inflammatory mediators are reduced by administration of steroids [8]. Corticosteroids also have direct analgesic effects, and have been found to reduce conduction in unmyelinated C fibers [9]. Despite multiple postulated mechanisms to support the use of epidural steroids in the treatment of lumbar spine pain conditions, epidural steroid administration continues to be controversial. Studies have shown that the administration of saline, local anesthetic, or any nonsteroid solution can exert an analgesic effect simply via the "washout" of inflammatory cytokines [10]. A 2015 review by Manchikanti et al. suggested steroid use in epidural injections was not superior to local anesthetic injections [11]. However, subsequent studies have continued to support the use of epidural steroid injections, which counters such a conclusion. An extensive 2018 review showed superiority of epidural steroid injections to local anesthetic alone, especially for short-term pain relief [12].

Lumbar Interlaminar Epidural Steroid Injections

There are primarily three different approaches to deposit steroid into the lumbar epidural space, the interlaminar approach, the transforaminal approach and the caudal approach. The interlaminar approach involves the passage of a needle through the interlaminar space and ligamentum flavum using a "loss of resistance" technique to deliver medication to the epidural space. Classically, this approach was utilized as a blind technique prior to the addition of fluoroscopic-guidance, and continues to be the approach for blind placement of perioperative epidural catheters

for laboring patients or surgical patients receiving epidural analgesia. It is now best practice to perform this technique using fluoroscopic guidance when utilizing epidural steroid injections for therapeutic purposes. Fluoroscopy allows the practitioner to ensure appropriate epidurography with dye spread prior to injecting a therapeutic substance. Studies have shown that even in experienced hands, blind interlaminar epidural injections are inappropriately placed approximately 20% of the time [13].

There are advantages and disadvantages to the interlaminar technique. Advantages of the interlaminar approach include deposition of steroid at more than one spinal level. A single interlaminar injection can also be utilized for bilateral lower extremity pain complaints. Disadvantages to this approach include increased risk of dural puncture, primary spread of injectate in the dorsal epidural space, which is more distant from the anterior epidural space and the proposed primary source of pathology. However, some studies do suggest anterior spread with an interlaminar approach, specifically using a paramedian approach, as illustrated by Candido et al. [14]. Figure 10.1 shows an anterior-posterior fluoroscopic image of an L34 interlaminar epidural injection of radiopaque contrast.



Fig. 10.1 Lumbar interlaminar ESI

Evidence

Interlaminar epidural steroid injections have been studied extensively for their efficacy in treating radicular pain due to a disk herniation, pain due to spinal stenosis, axial back pain in the absence of disk herniation, and failed back surgery syndrome. A 2017 review specifically evaluated the efficacy of lumbar interlaminar epidural steroid injections with fluoroscopic guidance in the treatment of radicular pain related to lumbar disc herniation, radicular pain due to lumbar spinal stenosis, radicular pain of unclear etiology, and back and/or leg pain due to unclear or multiple etiologies [15]. The review of the literature yielded 71 articles on fluoroscopically guided L-ILESI (lumbar interlaminar epidural steroid injections) for treatment of lower extremity and/or low back pain due to multiple etiologies. Only 41 met the stringent inclusion criteria. For all conditions leading to lower extremity and/or low back pain, the quality of evidence is low in accordance with the GRADE (Grades of Recommendation, Assessment, Development and Evaluation) system of evaluating evidence to determine the quality of the evidence. There is lack of randomized controlled trials for fluoroscopically guided lumbar interlaminar epidural steroid injections. The exhaustive search yielded only 12 pragmatic studies addressing the use of fluoroscopically-guided interlaminar injections for radicular pain due to herniated disc, and 5 for spinal stenosis.

The overall quality of the evidence is considered low, but the body of evidence supports fluoroscopically-guided L-ILESI in the treatment of radicular pain from lumbar disc herniation as well as stenosis. The evidence is especially supportive of interlaminar epidural steroid injections in providing short-term pain relief for these conditions. Due to these findings, the intervention seems to be an appropriate treatment modality for acute or subacute radicular pain (due to disc herniation or spinal stenosis), but may be insufficient for chronic radicular pain or neurogenic claudication due to spinal stenosis [15].

Caudal Epidural Steroid Injections

The caudal approach involves passing a needle at the sacral hiatus through the sacrococcygeal ligament into the epidural space. The benefits to this technique are the relative ease of placement, low risk for dural puncture or nerve root injury, and ability to access the epidural space when patients may have significant post-surgical scarring or hardware related to previous spinal surgeries. The disadvantages to this technique include the large volumes often necessary to reach the site of pathology and the injection tends to be less specific to the intended site. Figure 10.2 shows a lateral fluoroscopic image of a caudal epidural placement of a Tuohy needle.

Evidence

Despite extensive study of the efficacy of lumbar epidural steroid injections, there is no consensus opinion on the subject, so it is difficult to say which approach to the



Fig. 10.2 Caudal ESI

lumbar epidural space is most "efficacious" [16]. A 2018 review and meta-analysis by Lee et al. evaluated the efficacy of caudal epidural steroid injections versus transforaminal approach in the treatment of lumbosacral disc herniations. An exhaustive literature search resulted in only 6 quality studies. Four articles supported the superiority of TFESI (transforaminal epidural steroid injection) over caudal epidural steroid injection. However, one of these articles showed no statistically significant difference between the two approaches. Meta-analysis revealed short-term and long-term trends toward improved efficacy with TFESI, but without statistical significance. Overall, the evidence level was low [17].

A 2012 review by Abdi et al. evaluated the efficacy of caudal epidural steroid injections in the treatment of chronic low back pain. Again, the quality of the evidence was deemed to be poor due to an overall paucity of literature. 73 studies were identified, 16 of which met inclusion criteria (including 11 randomized trials and 5 non-randomized trials). For lumbar disc herniation, evidence was good for short and long-term relief of radicular pain with local anesthetics and steroids, and fair relief with local anesthetic only. For other lumbar spine pain conditions, including discogenic pain, spinal stenosis, and post-surgery pain syndrome, evidence was only considered to be fair [18].

Lumbar Transforaminal Epidural Steroid Injections and Selective Nerve Root Blocks

The transforaminal approach allows for a more targeted approach to the epidural space and involved nerve root, with less volume of injectate necessary. To gain access to the foramen, the patient is typically placed prone, and oblique angulation of the c-arm is utilized to view the spinal elements as a "scotty dog" image. The superior articular process appears as the dog's "ear", the transverse process projects over the vertebral body as the "nose", the inferior articular process is the dog's front "leg", the pedicle overlaps the region of the dog's "eye", and the spinous process is the dog's "feet".

Traditionally, a subpedicular approach was recommended, with the needle tip aimed towards the "safe triangle." The borders of the "safe triangle" include the inferior pedicle as the superior border, the lateral vertebral body as the lateral border, and the traversing nerve root as the hypotenuse. By placing the needle in this area, one presumably avoids nerve root trauma and is bordered by the bony landmark of the vertebral body, which reduces incidence of intradiscal placement. However, critics of this approach voice that this target is actually "unsafe" due to a higher incidence of vascular structures in the vicinity, especially spinal radicular arteries, which have been implicated in devastating neurological deficits attributed to vascular penetration and spinal cord ischemia [19]. However, by avoiding particulate steroid in transforaminal steroid injections, the overall risk of such ischemic events have lessened.

An alternative approach for transforaminal epidural steroid injections targets "Kambin's triangle." Kambin's triangle describes the area overlying the posterolateral disc, bounded by the inferior vertebral body (superior border), exiting spinal nerve root (hypotenuse), and traversing nerve root (vertical edge). Utilizing Kambin's triangle as a target may offer the advantage of lower inadvertent vascular injection, but does not fully eliminate risk, including inadvertent violation of the intravertebral disc [20, 21].

The approach for a lumbar selective nerve root block is essentially the same as a transforaminal epidural steroid injection. However, low injectate volumes, ideally 0.5 ml or less, are utilized to hopefully target spread along the nerve root without epidural spread. The goal of a selective nerve root block is to help isolate which nerve root is the etiology of a patient's complaint, especially when multiple areas of spinal pathology are apparent on imaging. Unfortunately, even with such low volumes, studies have shown variable spread to other spinal nerve roots due to the contiguous nature of the spinal nerve root sheaths with the dura mater [22]. Figure 10.3 shows an anterior-posterior fluoroscopic image of a right L5 selective nerve root block with contrast spread into the neural foramen and under the pedicle.

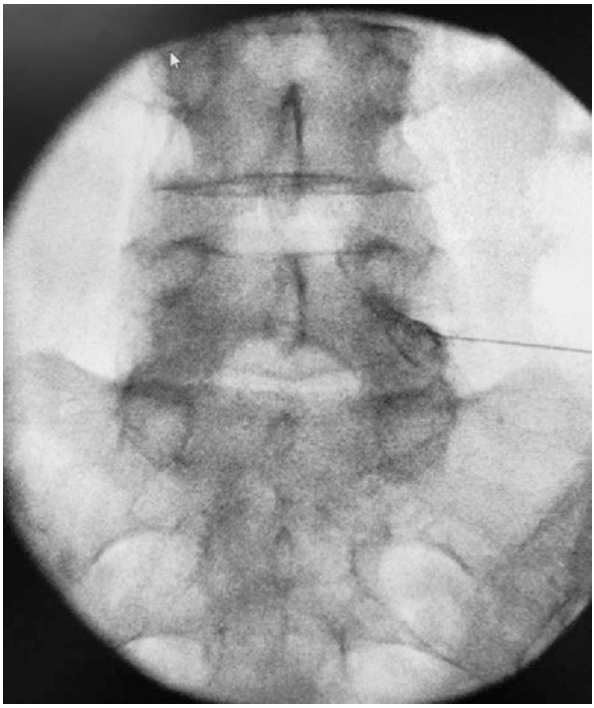


Fig. 10.3 Lumbar transforaminal ESI

Cervical Epidural Steroid Injections

The cervical epidural space is considerably narrower than the lumbar spine, ranging from 1 to 4 mm [23]. There are primarily 2 approaches to deposit steroid into the cervical epidural space, the interlaminar approach and the transforaminal approach.

Cervical Interlaminar Epidural Steroid Injections

Similar to the lumbar spine, the cervical interlaminar approach involves the passage of a needle through the midline interlaminar space. The needle traverses the supraspinous ligament, interspinous ligament, and ligamentum flavum using a "loss of resistance" technique in conjunction with fluoroscopic guidance to deliver medication to the epidural space. The needle is often advanced in the contralateral oblique view, allowing the ability to visualize needle depth as it approaches the spinolaminar line. Typically, the C6–7 or C7–T1 level is accessed due its larger posterior to anterior epidural space diameter at the lower cervical levels [24]. Figure 10.4 shows an anterior-posterior fluoroscopic image of a cervical interlaminar epidural injection of radiopaque contrast.



Fig. 10.4 Cervical interlaminar ESI

Cervical Transforaminal Epidural Steroid Injections and Selective Nerve Root Blocks

The cervical transforaminal approach typically involves the patient in the supine position with the fluoroscope positioned in an oblique view to optimize the intended cervical neuroforamen. The needle is then advanced to target the superior articulating process of the caudad vertebrae of the neuroforamen in question. The needle is then walked ventromedially into the posterior portion of the foramen. Entering in this way maximizes avoiding vascular structures located in the superior aspect of the foramen and the vertebral artery located in the anterior aspect of the foramen [27].

It should be noted, the neurovascular risks can be significant with the cervical transforaminal approach. Known complications include potential injury of a radiculomedullary or vertebral artery, embolic events, and brain and spinal cord infarctions [25, 26]. Recommendations to minimize complications include use of contrast enhanced live fluoroscopy with or without digital subtraction, test dose bolus of short acting local anesthetic with post bolus monitoring for neurological sequelae, minimal to no sedation, and use of nonparticulate steroid [58].

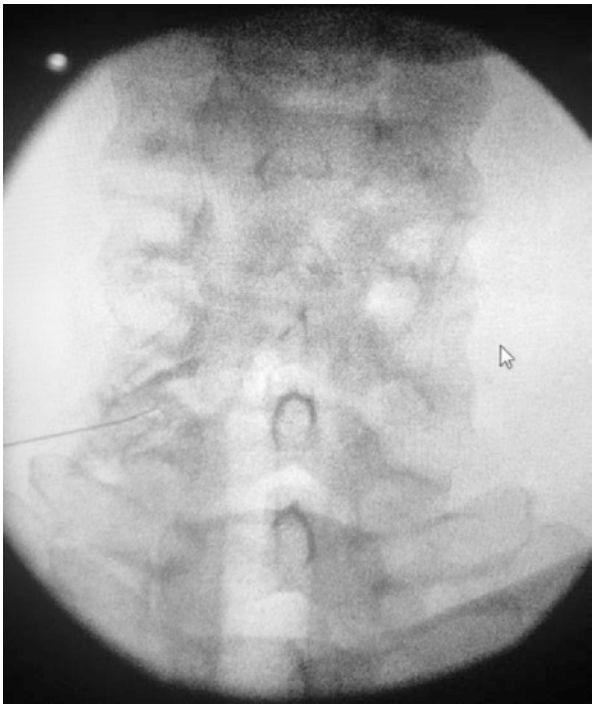


Fig. 10.5 Cervical transforaminal ESI

The term cervical selective nerve root block has been used synonymously with cervical transforaminal epidural steroid injections. Sometimes it is used to describe a diagnostic procedure which does not employ steroids. Other times it can be in reference to cervical extraforaminal injections and cervical selective nerve root injections. Figure 10.5 shows an anterior-posterior fluoroscopic image of a left cervical transforaminal curved blunt needle placement and contrast injection.

Evidence

In a systemic review article published in 2009, Benyamin et al. evaluated the effects of cervical interlaminar epidural injections in managing chronic neck and upper extremity pain [28]. 3 randomized controlled trials were found to show positive results for short-term relief and 2 were positive for long-term relief [29–31]. The evidence for managing cervical radiculopathy with cervical interlaminar epidural steroid injections was deemed moderate for short-term improvement and long-term improvement by Abdi et al. [33]. Similarly, the evidence for cervical transforaminal epidural steroid injections in managing cervical nerve root pain is moderate [32, 33]. Kolstad et al. and Lin et al. were able to show that cervical epidural transforaminal injections can lead to pain relief significant enough to prevent patients from

having to undergo surgery [34, 35]. Overall, it should be noted that randomized controlled studies of cervical epidural steroid injections compared to placebo groups have been sparse and are still needed in the literature.

Facet Joint injections and Medial Branch Nerve Blocks

Background

The facet joints are another common pain generator of the spine. A facet joint, also known as a zygapophysial joint or z joint, is formed from the articulation between the superior articular process of one vertebra and the inferior articular process of the vertebra directly above it. A facet joint forms the connections between the bones of the spine. These joints stabilize and guide spinal motion, and can cause pain in the head, neck, back, hip, buttocks or leg. Each facet is innervated by medial branch nerves which are terminal branches off the dorsal rami [53, 54].

In 1933, Ralph K. Ghormley coined the term "facet syndrome," referring to the pains of the articular facet joints that is responsible for 15–52% of those who complain of chronic low back pain [36, 37]. Cervical facets were first identified as a source of pain by Pawl in 1977 [38]. Today, facet joint pain can be diagnosed and treated with facet joint injections and medial branch nerve blocks. These come only second to epidural steroid injections as the most commonly performed pain procedures in the United States in 2006 [39]. Facet joint injections and medial branch nerve blocks are indicated for the diagnosis and treatment of axial spine pain.

Facet Joint Injections

Lumbar facet injections are performed under fluoroscopic guidance with the patient in the prone position. The fluoroscope is typically positioned in ipsilateral oblique view. Intra-articular steroid injections are targeted at the middle to upper half of the joint. Periarticular injection may also be acceptable in the case that intra-articular injection is proven to be difficult [55]. Cervical facet injections are performed under fluoroscopic guidance with either a posterior or lateral approach. For the posterior approach, the fluoroscope is positioned in an anteroposterior view and tilted caudally to identify the cervical facet joint. The needle is advanced coaxially to the lateral half of the joint to avoid the spinal cord towards the midpoint of the articular pillar. Vital structures such as the vertebral artery, spinal nerve, and dorsal root ganglion are located ventral to the joint so additional multiplanar views should be used to assess depth. The lateral approach involves the patient in the lateral decubitus position with the symptomatic side up. It may be necessary to tilt and oblique the fluoroscope slightly to optimize the view of the facet joint. The lateral approach is arguably technically easier and less soft tissue is traversed however lower cervical levels may be obscured by the shoulders and careful attention must be paid to the apex of the lung [27].

Medial Branch Nerve Blocks

Medial branch nerve blocks are mainly diagnostic injections. After a positive response to diagnostic injections, the most commonly performed treatment for facet mediated pain is radiofrequency denervation.

Each lumbar facet is innervated by the medial branches arising from the dorsal rami from the level above the joint and at the same level. Lumbar medial branch blocks are performed with the patient in the prone position. For the L1–4 medial branches, the target needle destination is the junction of the superior articular process and the transverse process where the medial branch crosses. In the oblique fluoroscopic view, this is just superior to the "eye of the scotty dog". The L5–S1 facet joint derives its innervation through the L4 medial branch and the L5 dorsal ramus. The L5 dorsal ramus lies at the middle of the base at the sacral ala [56, 57].

Cervical facet joints are innervated by the medial branches of the cervical dorsal rami above and below the joint as these branches course around the waist of the articular pillars. The C2–3 facet joint is unique in that it is innervated by 2 different branches of the C3 dorsal ramus [52]. For the lateral approach to cervical medial branch blocks, the patient is in the lateral decubitus position with the symptomatic side up. The fluoroscope is adjusted to align the articular pillar borders and the target destination is the tip of the needle over the center point of the articular pillar. For the posterior approach, views are optimized in either anteroposterior or oblique views such that the tip of the needle's destination is the lateral groove or waist of the articular pillar for the C3–C6 levels. The C7 medial branch lies at the junction of the superior articular process and transverse process much like the lumbar medial branches. Lastly, the C8 medical branch lies over the superolateral transverse process of T1 [27].

It should be noted that medial branch nerve blocks have been associated with a high false positive rate. To enhance the diagnostic validity of each injection, avoid the use of sedation and analgesics, use injectate volumes $\leq$0.5 mL, and limit the volume of superficial local anesthesia. Educating patients regarding correct use of a pain diary can also be helpful in properly chronicling the pain response [59].

Evidence

In a systematic review of 21 randomized controlled trials and 5 observational studies, quality assessment showed that the level of evidence for the effectiveness of lumbar facet joint nerve blocks is fair and level of evidence for lumbar intraarticular injections is limited [40]. A separate systematic review of 17 studies suggested that the level of evidence for the diagnostic accuracy of lumbar facet joint nerve blocks is good [41]. Two high quality RCTs and one moderate to high quality RTC has shown a positive effect of lumbar medial branch nerve blocks. A study of 229 patients conducted by Cohen et al. suggest that facet blocks are not therapeutic, but may provide prognostic value prior to radiofrequency ablation [42].

The data supporting cervical facet injections and cervical medial branch blocks is much more limited. In a systematic review performed by Falco et al. in 2012, 13 out of 32 studies considered for inclusion met criteria. The literature review found one randomized controlled trial evaluating cervical facet joint nerve blocks and 2 randomized controlled trials evaluating intraarticular injections. Furthermore, there was only one observational study evaluating cervical facet joint nerve blocks and no observational studies meeting inclusion criteria evaluating intraarticular injections. The evidence for cervical intraarticular injections was deemed limited and the evidence for cervical medial branch blocks was deemed fair [43].

Sacroiliac Joint Injections

Another common pain generator in the low back is the sacroiliac joint or SI joint. Reported prevalence studies of low back pain originating from the SI joint pain is as high as 45% [44]. The SIJ is the largest axial joint in the body and is formed by the articulation of the sacrum and ilium [45]. The joint is comprised a synovial cartilaginous joint and a fibrous articulation [46]. The joint is not readily mobile and is primarily for stability and weight bearing. The exact innervation of the SI joint is debatable however many suggest that it comes predominantly from the dorsal rami of the S1 through S4 nerve roots. There may be additional contributions from nerve fibers within the joint capsule and adjoining ligaments [47, 48].

Pain from the joint can come from either degenerative or inflammatory processes. The SI joint may also be a referred site of pain from degenerative disc disease at L5/S1, spinal stenosis, or osteoarthritis of the hip [49].

SI joint injections can be useful for both diagnostic and therapeutic purposes. A positive response to an injection is the most accurate means for diagnosing a painful joint complex. SI joint injections are typically done under fluoroscopic guidance with contrast, however CT guidance and ultrasound guidance techniques have also been employed. An SI joint injection is performed with the patient in the prone position with the fluoroscope angled toward the contralateral oblique side. Entry into the joint is typically 1–2 cm above the inferior margin of the joint. Contrast agent is used to verify intra articular spread outlining the margins of the joint. After confirmation, a combination of local anesthetic and steroid is injected. Figure 10.6 shows an oblique fluoroscopic image of a needle placement in the inferior aspect of the sacroiliac joint.

Evidence

A 2015 systematic review of 6 randomized control trials and 8 observational studies by Simpopoulos et al. suggested that the evidence supporting intraarticular and periarticular injections is still limited [50]. Interestingly, in a study comparing intraarticular and periarticular injections for patients with SI joint pain, periarticular injections were found to be more effective and easier to perform than intraarticular injections [51].



Fig. 10.6 Sacroiliac joint injection

Conclusion

Among those afflicted with chronic pain, spinal pain emanating from the cervical, thoracic and lumbosacral regions comprise a large majority of the etiologies. Minimally invasive, targeted interventional techniques are crucial to managing pain and improving the quality of life of the pain sufferers. Spinal injections can be used for both diagnostic and therapeutic purposes and are important in multimodal approaches to pain management.

References

1. Lidgren L. The bone and joint decade 2000–2010. Bull World Health Organ. 2003;81:629.
2. Hoy D, et al. The global burden of low back pain: estimates from the Global Burden of Disease 2010 study. Ann Rheum Dis. 2014;73:968–74.
3. Cohen S. Epidemiology, diagnosis, and treatment of neck pain. Mayo Clin Proc. 2015;90(2):284–99.
4. Fejer R, Kyvik K, Hartvigsen J. The prevalence of neck pain in the world population: a systematic critical review of the literature. Eur Spine J. 2006;15(6):834–48.

5. Lievre J, Bloch-Michel H, Pean G, et al. L'hydrocortisone en injection locale. Rev Rhumat Mal Osteoartic. 1953;20:310Y311.
6. Swerdlow M, Sayle-Creer W. A study of extradural medication in the relief of the lumbosciatic syndrome. Anaesthesia. 1970;25:341Y345.
7. Saal J. The role of inflammation in lumbar pain. Spine. 1995;20:1821Y1827.
8. Petho G, Reeh P. Sensory and signaling mechanisms of bradykinin, eicosanoids, platelet-activating factor, and nitric oxide in peripheral nociceptors. Physiol Rev. 2012;92:1699Y1775.
9. Johansson A, Hao J, Sjolund B. Local corticosteroid application blocks transmission in normal nociceptive C-fibers. Acta Anaesthesiol Scand. 1990;34:335Y338.
10. Jacobs L, Vo N, Kang J. Identifying inflammatory targets for the biologic therapies for spinal pain. PM R. 2011;3(6 suppl 1):S12YS17.
11. Manchikanti L, et al. Epidural injections for lumbar radiculopathy and spinal stenosis: a comparative systematic review and meta-analysis. Pain Physician. 2016;19:E365–410.
12. Lee JH, et al. Comparison of clinical efficacy of epidural injection with or without steroid in lumbosacral disc herniation: a systematic review and meta-analysis. Pain Physician. 2018;21:449–67.
13. White AH. Injection techniques for the diagnosis and treatment of low back pain. Orthop Clin N Am. 1983;14(3):553–67.
14. Candido K, Raghavendra M, Chinthagada M, et al. A prospective evaluation of iodinated contrast flow patterns with fluoroscopically guided lumbar epidural steroid injections: the lateral parasagittal interlaminar epidural approach versus the transforaminal epidural approach. Anesth Analg. 2008;106:638Y644.
15. Sharma AK, et al. The effectiveness and risks of fluoroscopically guided lumbar interlaminar epidural steroid injections: a systematic review with comprehensive analysis of the published data. Pain Med. 2017;18(2):239.
16. Manchikanti L, Singh V, Pampati V, et al. Comparison of the efficacy of caudal, interlaminar, and transforaminal epidural injections in managing lumbar disc herniation: is one method superior to the other? Korean J Pain. 2015;28:11–21.
17. Lee JH, et al. Comparison of clinical efficacy of transforaminal and caudal epidural steroid injection in lumbar and lumbosacral disc herniation: a systematic review and meta-analysis. Spine J. 2018;18(12):2343–53.
18. Parr AT, Manchikanti L, Hameed H, Conn A, Manchikanti KN, Benyamin RM, Diwan S, Singh V, Abdi S. Caudal epidural injections in the management of chronic low back pain: a systematic appraisal of the literature. Pain Physician. 2012;15(3):E159–98.
19. Derby R, Kine G, Saal JA, et al. Response to steroid and duration of radicular pain as predictors of surgical outcome. Spine. 1992;17:S176–83.
20. Park JW, Nam HS, Cho SK, et al. Kambin's triangle approach of lumbar transforaminal epidural injection with spinal stenosis. Ann Rehabil Med. 2011;35:833–43.
21. Trinh KH, Gharibo CG, Aydin SM. Inadvertent intradiscal injection with TFESI utilizing Kambin's retrodiscal approach in the treatment of acute lumbar radiculopathy. Pain Pract. 2016;16(4):E70–3.
22. Castro WH, Grönemeyer D, Jerosch J, et al. How reliable is lumbar nerve root sheath infiltration? Eur Spine J. 1994;3:255–7.
23. Cousins MJ, Bridenbaugh PO. Neural blockade in clinical anesthesia and management of pain. Philadelphia: Lippincott-Raven; 1998. p. 255.
24. Fujinaka MK, Lawson EF, Schulteis G, Wallace MS. Cervical epidural depth: correlation between needle angle, cervical anatomy, and body surface area. Pain Med. 2012;13(5):665–9.
25. Malhotra G, Abbasi A, Rhee M. Complications of transforaminal cervical epidural steroid injections. Spine. 2009;34(7):731–9.
26. Botwin KP, Gruber RD, Bouchlas CG, et al. Complications of fluoroscopically guided transforaminal lumbar epidural injections. Arch Phys Med Rehabil. 2000;81:1045.
27. Berkwits L, Furman MB. Atlas of image-guided spinal procedures. 2nd ed. Philadelphia: Elsevier, Inc.; 2018.

28. Benyamin RM, Singh V, Parr AT, et al. Systematic review of the effectiveness of cervical epidurals in the management of chronic neck pain. Pain Physician. 2009;12:137.
29. Stav A, Ovadia L, Sternberg A, Kaadan M, Weksler N. Cervical epidural steroid injection for cervicobrachialgia. Acta Anaesthesiol Scand. 1993;37:562–6.
30. Castagnera L, Maurette P, Pointillart V, Vital JM, Erny P, Senegas J. Long term results of cervical epidural steroid injection with and without morphine in chronic cervical radicular pain. Pain. 1994;58:239–43.
31. Pasqualucci A, Varrassi G, Braschi A, Peduto VA, Brunelli A, Marinangeli F, Gori F, Colò F, Paladini A, Mojoli F. Epidural local anesthetic plus corticosteroid for the treatment of cervical brachial radicular pain: single injection versus continuous infusion. Clin J Pain. 2007;23:551–7.
32. Conger A, Cushman DM, Speckman RA, Burnham T, Teramoto M, McCormick ZL. The effectiveness of fluoroscopically guided cervical transforaminal epidural steroid injection for the treatment of radicular pain; a systematic review and meta-analysis. Pain Med. 2020 Jan 1;21(1):41–54.
33. Abdi S, Datta S, Trescot AM, Schultz DM, Adlaka R, Atluri SL, Smith HS, Manchikanti L. Epidural steroids in the management of chronic spinal pain: a systematic review. Pain Physician. 2007;10(1):185–212.
34. Kolstad F, Leivseth G, Nygaard O. Transforaminal steroid injections in the treatment of cervical radiculopathy. A prospective outcome study. Acta Neurochir. 2005;147:1065–70.
35. Lin EL, Lieu V, Halevi L, Shamie AN, Wang JC. Cervical epidural steroid injections for symptomatic disc herniations. J Spinal Disord Tech. 2006;19:183–6.
36. Ghormley RK. Low back pain with special reference to the articular facets, with presentation of an operative procedure. JAMA. 1933;101:1773–7.
37. Binder DS, Nampiaparampil DE. The provocative lumbar facet joint. Curr Rev Musculoskelet Med. 2009;2:15–24.
38. Pawl RP. Headache, cervical spondylosis, and anterior cervical fusion. Surg Annu. 1977;9:391.
39. Manchikanti L, Singh V, Pampati V, et al. Analysis of growth of intervention- al techniques in managing chronic pain in the Medicare population: a 10-year evaluation from 1997 to 2006. Pain Physician. 2009;12(1):9–34.
40. Manchikanti L, Kay AD, Boswell MV, et al. A systematic review and best evidence synthesis of the effectiveness of therapeutic facet joint interventions in managing chronic spinal pain. Pain Physician. 2015;18(4):E535–82.
41. Boswell MV, Manchikanti L, Kaye AD, et al. A best-evidence systematic appraisal of the diagnostic accuracy and utility of facet (zygapophysial) joint injections in chronic spinal pain. Pain Physician. 2015;18(4):E497–533.
42. Cohen SP, Doshi TL, Constantinescu OC, et al. Effectiveness of lumbar facet joint blocks and predictive value before radiofrequency denervation: the facet treatment study (FACTS), a randomized, controlled clinical trial. J Anesthesiol. 2018;129(3):517.
43. Falco FJ, Manchikanti L, Datta S, Wargo BW, Geffert S, Bryce DA, Atluri S, Singh V, Benyamin RM, Sehgal N, Ward SP, Helm S 2nd, Gupta S, Boswell MV. Systematic review of the therapeutic effectiveness of cervical facet joint interventions: an update. Pain Physician. 2012;15(6):E839–68.
44. Manchikanti L, Singh V, Pampati V, et al. Evaluation of the relative contributions of various structures in chronic low back pain. Pain Physician. 2001;4:308–16.
45. Forst SL, Wheeler MT, Fortin JD, et al. The sacroiliac joint: anatomy, physiology and clinical significance. Pain Physician. 2006;9:1533–9.
46. Cohen SP. Sacroiliac joint pain: a comprehensive review of anatomy, diagnosis and treatment. Anesth Analg. 2005;101:1440–53.
47. Fortin JD, Kissling RO, O'Connor BL, Vilenesky JA. Sacroiliac joint innervations and pain. Am J Orthop. 1999;28:687–90.
48. Cox RC, Fortin JD. The anatomy of the lateral branches of the sacral dorsal rami: implications for radiofrequency ablation. Pain Physician. 2014;17:459–64.

49. Barros G, McGrath L, Mikhail G. Sacroiliac joint dysfunction in patients with low back pain. Fed Pract. 2019;36(8):370–5.
50. Simopoulos TT, Manchikanti L, Gupta S, et al. Systematic review of the diagnostic accuracy and therapeutic effectiveness of sacroiliac joint interventions. Pain Physician. 2015;18(E):713–56.
51. Murakami E, Tanaka Y, Aizawa T, Ishizuka M, Kokubun S. Effect of periarticular and intraarticular lidocaine injections for sacroiliac joint pain: prospective comparative study. J Orthop Sci. 2007;12(3):274–80.
52. Bogduk N. The clinical anatomy of the cervical dorsal rami. Spine. 1982;7:319–30.
53. Pedersen HE, Blunck CF, Gardner E. The anatomy of the lumbosacral posterior rami and meningeal branches of spinal nerves (sinuvertebral nerves)—with an experimental study of their function. J Bone Joint Surg Am. 1956;38:377–91.
54. Bogduk N. Clinical anatomy of the lumbar spine and sacrum. 3rd ed. Edinburgh: Churchill Livingstone; 1997. p. 127–44.
55. Kershen LM, Nacey NC, Patrie JT, Fox MG. Fluoroscopically guided facet injections: comparison of intra-articular and periarticular steroid and anesthetic injection on immediate and short-term pain relief. AJNR Am J Neuroradiol. 2018;39(11):2161–5.
56. Bogduk N, Wilson AS, Tynan W. The human lumbar dorsal rami. J Anat. 1982;134:383–97.
57. Dreyfuss P, Schwarzer AC, Lau P, Bogduk N. Specificity of lumbar medial branch and L5 dorsal ramus blocks: a computed tomography study. Spine. 1997;22:895–902.
58. Rathmell JP, Benzon HT, Dreyfuss P, Huntoon M, Wallace M, Baker R, Riew KD, Rosenquist RW, Aprill C, Rost NS, Buvanendran A, Kreiner DS, Bogduk N, Fourney DR, Fraifeld E, Horn S, Stone J, Vorenkamp K, Lawler G, Summers J, Kloth D, O'Brien D Jr, Tutton S. Safeguards to prevent neurologic complications after epidural steroid injections: consensus opinions from a multidisciplinary working group and national organizations. Anesthesiology. 2015;122(5):974–84.
59. Hunter JP. Practical management of pain by Honorio Benzon, James P. Rathmell, Christopher L. Wu, Dennis C. Turk, Charles E. Argoff, Robert W. Hurley. Physiother Can. 2016;68(2):208.

Vertebral Compression Fractures

11

Ankit Patel and Brent Page

Introduction

While many patients with acute osteoporotic VCFs achieve significant pain relief with conservative measures, there are some who continue to experience persistent pain. VCFs may contribute to symptoms of back pain, radiculopathy, and/or myelopathy. Patients with VCFs may also experience other medical co-morbidities, functional impairments, and overall reduction in quality of life. For some patients, VCFs can be an incidental finding on spine imaging. Therefore, it is important to identify if a patient has a symptomatic compression fracture as this significantly impacts the treatment plan.

This chapter aims to review the epidemiology and common etiologies of VCFs, as well as the global impact of these fractures on patients. The key points in the clinical and diagnostic imaging evaluation of patients with VCFs will also be highlighted. An evidence-based review will be provided on the various treatment options for acute and persistent pain associated with VCFs. The chapter will also emphasize the importance of a multidisciplinary approach to treatment and prevention of VCFs.

Epidemiology and Risk Factors

A vertebral compression fracture (VCF) is characterized by collapse of trabecular bone within the vertebral body. VCFs have a variety of causes, including osteoporosis, trauma, malignancy, and infection. Osteoporotic fractures are by far the most common, accounting for an estimated 700,000 new VCFs in the United States every year [1]. Osteoporosis is characterized by decreased bone mineral density (BMD).

A. Patel (✉) · B. Page
UT Southwestern Medical Center, Dallas, TX, USA
e-mail: Ankit.Patel@UTSouthwestern.edu

© The Author(s), under exclusive license to Springer Nature Switzerland AG 2022

C. E. Noe (ed.), *Multidisciplinary Spine Care*,
https://doi.org/10.1007/978-3-031-04990-3_11

The World Health Organization defines osteoporosis as a BMD that lies 2.5 standard deviations (SD) or more below the average BMD of a healthy, premenopausal white female (T-score < −2.5 SD). Osteoporosis results in diminished structural support of the spinal column, increasing the risk of fracture. In 2010, approximately 10.2 million older adults in the United States had osteoporosis, placing them at substantially increased risk for VCF compared to their non-osteoporotic peers [2–4]. The rate of osteoporosis is expected to increase more than 30% by the year 2030 as the population ages [4].

Other VCF risk factors are similar to those for osteoporosis and include advanced age, female sex, Asian or Caucasian ethnicity, excessive alcohol consumption, tobacco use, estrogen deficiency, history of falls, lack of physical activity, use of systemic glucocorticoids, and deficiency of calcium and vitamin D [5, 6]. The prevalence of VCF increases with advancing age, affecting approximately 25% of all postmenopausal women [7]. By 80 years of age, the prevalence in women reaches 40% [8]. Elderly men are also at increased risk of VCF, though lifetime fracture risk in men is less than in women [7].

History of prior VCF is also an important risk factor for sustaining a new VCF. Having sustained 1 VCF increases the risk of a subsequent VCF by approximately five-fold in the first year following the initial fracture [9]. In patients with a history of 2 or more VCFs, the subsequent fracture risk increases up to 12-fold [10].

While osteoporosis is the most common cause of VCF in the elderly, any new compression fracture in a young and otherwise healthy patient should prompt further investigation. Again, it is important to assess bone health and to also evaluate for secondary causes of fracture, such as malignancy. Metastatic disease affecting the vertebral body can compromise bone stability and lead to pathologic VCF. Bone metastasis is common in a variety of advanced stage solid tumor malignancies including prostate, lung, renal, breast, and colorectal cancer. For example, bone metastasis is diagnosed in up to 45% of metastatic prostate cancer patients within 12 months of initial cancer diagnosis [11]. Pathologic fracture is also common in Multiple myeloma (MM), a plasma cell malignancy associated with osteolytic bone disease. VCF is the most common type of fracture in MM and is seen in up to 60% of patients at the onset of disease [12].

Global Effects of VCF

More than two-thirds of patients with VCFs are asymptomatic and only identified incidentally, often on standard radiographs of the chest and abdomen [13]. Symptomatic fractures, however, can cause significant acute and chronic back pain, impaired physical functioning, vertebral height loss, and progressive spinal kyphotic deformity [14–17]. Symptomatic VCF negatively impacts quality of life and mental health. Patients often report feelings of anxiety and depression following an acute VCF [18]. Additionally, the loss of ability to participate in recreational activities, secondary to fracture associated pain and debility, can lead to social isolation [18].

Patient perceived deterioration in overall health status is common and adds to dissatisfaction following acute fracture [19]. Not surprisingly, patients with a history of prior VCF have greater disability and worse quality of life after sustaining a subsequent VCF compared to those with a first-time fracture [20]. While VCFs are rarely fatal in the short term, they have been associated with a higher mortality rate which becomes more pronounced in the years following fracture [21].

The social and economic costs of VCFs are also substantial. Direct healthcare costs associated with VCFs were estimated to exceed $1 billion per year in 2005, with costs expected to increase more than 50% by the year 2025 as rates of osteoporosis continue to rise [22]. When patient and caregiver productivity loss is factored in, the costs associated with VCF are substantially higher. Direct economic costs to the patient are also high. In the first 12 months following an initial osteoporotic fracture, average all-cause healthcare costs more than double [23]. Also, in the first 12 months following a fracture, patients are 14 times more likely to require primary care physician services, as compared to the general population [24]. Approximately 10% of patients with acute VCF require hospitalization, with a 6-day length of stay on average [25]. Of those patients requiring hospitalization, approximately half require ongoing skilled care in a nursing facility following hospital discharge [25].

Given the substantial individual, societal, and growing economic burden associated with VCF, the identification and treatment of underlying fracture etiology is paramount. Unfortunately, studies suggest physicians often fail to evaluate bone health nor initiate osteoporosis directed treatment following acute osteoporotic VCF [26]. These missed opportunities, in addition to the known high rates of refracture, highlight the importance of addressing bone health in a timely manner following VCF.

Evaluation

For patients who have back pain and for whom there is a high index of suspicion of a possible compression fracture, imaging can be useful in confirming or ruling out the presence of a vertebral compression deformity. It is imperative that clinical correlation is applied because some patients can have asymptomatic VCFs that are noted incidentally on spine imaging. Features in the patient's history that are suggestive of an acute compression fracture include an acute onset of severe pain in the region of the compression fracture. Acute compression fractures often occur spontaneously or as a result of trivial strain [27]. An accurate diagnosis of an acute VCF can be missed initially and can lead to a delay in appropriate care [27]. For benign acute compression fractures, the pain is typically worse with activity and relieved with rest. The pain can also be aggravated with coughing, sneezing, and activities that jar the body. Some patients may experience symptoms of early satiety and decreased exercise tolerance due to a compression of the abdominal and thoracic cavity from the spinal deformity associated with multiple VCFs [28]. The wide

spectrum of impact that compression fractures can have on patients highlights the importance of obtaining a thorough history of the patient's symptoms, as well as the impact of the fracture on the patient's function and quality of life.

The most common sites of VCFs include the thoracolumbar junction, the mid-thoracic spine, and the lumbar spine [29]. In patients with acute compression fractures, physical examination may reveal point tenderness over the symptomatic spinous process. Patients may have a positive "closed-fist percussion sign" or "supine sign" in the setting of an acute compression fracture. The closed-first percussion sign requires the examiner to percuss over the site of a suspected fracture with the hypothenar aspect of the fist. Reproduction of the back pain is considered to be a positive sign. In order to evaluate for the supine sign, the examiner observes the patient transition to a supine position on the examination table. This sign is considered positive if the patient is unable to lie supine due to severe back pain. The closed-fist percussion sign has been shown to have a sensitivity of 87.5% and a specificity of 90%, while the supine sign has a reported sensitivity of 81.25% and a specificity of 93.33% [30].

Depending on the location, severity, and height loss associated with VCF(s), patients may be noted have kyphosis or loss of lordosis on physical inspection. Some patients with VCFs in the upper back region can develop a rounded-appearing kyphotic deformity known as a dowager's hump [31]. A reduction in overall body height may also be present in patients with severe or multilevel compression fractures.

Neurological compromise due to a VCF is a rare but potentially catastrophic scenario [32]. A comprehensive clinical evaluation and neuromuscular examination is important to rule out radiculopathy, myelopathy, cauda equina, or spinal cord compression. Spinal canal compromise should be suspected in patients who develop lower extremity pain, neurologic signs or symptoms, or bowel or bladder incontinence after the initial diagnosis of acute back pain due to a VCF [32]. These "red-flag" signs and symptoms warrant urgent imaging and surgical consultation. According the American College of Radiology's appropriateness criteria for management of VCFs, surgical consultation should be considered in patients with spinal instability, neurologic deficits, or spinal deformity. Surgical consultation is recommended in the setting of patients with pathologic VCFs who have severe pain, neurologic deficits, spinal deformity, spinal instability, or pulmonary dysfunction [33].

Plain radiographs can be useful for evaluating the presence of a superior and/or inferior endplate compression deformity and to quantify the degree of vertebral body height loss (Figs. 11.1, 11.2, 11.3, and 11.4). Repeating plain radiographs upon patient follow up can be considered to monitor for fracture progression [34]. It should be noted that not all vertebral body deformities are a result of a VCF. Vertebral bodies may appear deformed from other conditions such as Schmorl's nodes, short vertebral height, Scheuermann's disease, and physiologic wedging [35].

Magnetic resonance imaging (MRI) can be useful to assess fracture acuity by evaluating for endplate edema (Figs. 11.5, and 11.6). In a patient with an acute compression fracture, the MRI would be expected to demonstrate marrow edema on



Fig. 11.1 Lateral X-ray demonstrating a T10 VCF

fat-suppressed short tau inversion recovery (STIR) sequences. In the setting of chronic compression fractures, the MRI would not demonstrate high T2, low T1, or STIR signal abnormality in the compression fracture (Fig. 11.7). MRI can be useful in procedural planning and in distinguishing acute versus chronic fractures in patients with multiple wedge deformities and conflicting physical examination findings [33]. MRI can also be considered in the evaluation of symptomatic patients who do not have significant height loss on plain radiographs.

Contrast-enhanced MRI studies can aid in differentiating between osteoporotic and malignant vertebral fracture. MRI features that could suggest the presence of a pathologic VCF include abnormal posterior element signal, epidural/paravertebral soft-tissue mass, expansion of posterior vertebral contour, abnormal enhancement, and replacement of normal marrow signal [36, 37] (Fig. 11.8).

The benefits of an MRI over a computerized tomography (CT) scan or plain radiographs are more optimal soft tissue & bone marrow resolution, as well as avoidance of ionizing radiation. If there is a contraindication to MRI, a CT scan can be useful to evaluate for any bony retropulsion (Fig. 11.9). It should be noted that CT scans will expose the patient to ionized radiation. For assessment of specific

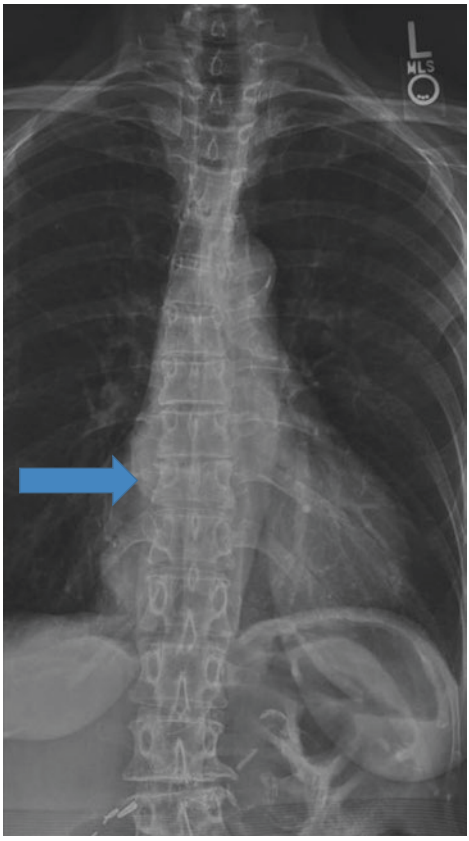


Fig. 11.2 AP X-ray demonstrating a T10 VCF

bony details such as the location and extent of fracture lines, thin-section CT with sagittal reconstructions can be a useful modality [38].

Bone scintigraphy, or bone scan, can be useful in patients who are unable to undergo MRI and in whom a CT scan or clinical history does not confirm the acuity of the compression fracture (Fig. 11.10). A bone scan may show elevated tracer uptake for up to 12 months following a fracture, therefore the results should be correlated clinically [38].

Management

Successful management of VCF often involves a graduated, multimodal approach. Most patients with acute VCF can be treated conservatively and pain typically resolves over a period of 4 to 6 weeks [39]. Comprehensive treatment strategies should address pain control and maintenance of physical functioning. It is also essential to address bone health, when the fracture is osteoporotic in nature, given the high likelihood of subsequent fracture.



Fig. 11.3 Lateral X-ray of a L1 VCF

Medications for Pain Control

Adequate pain control is important to prevent immobility and associated comorbidities including decubitus ulcers, venous thromboembolism, pulmonary disease, and progressive functional decline. First-line analgesics used to manage acute pain from VCF include acetaminophen and nonsteroidal anti-inflammatory drugs (NSAIDs). Appropriate consideration should be taken when prescribing NSAIDs to patients with a history of gastric ulcers, gastrointestinal bleeding, cardiac and renal disease. Selective cyclooxygenase-2 (COX-2) inhibitors, which have a lower risk of gastrointestinal side effects as compared to traditional nonselective NSAIDs, may also be considered [40–42]. There is a theoretical risk of impaired bone healing with the use of NSAIDs, though this has not been confirmed and NSAIDs are commonly used for acute pain control in clinical practice [43, 44]. Other frequently used pharmacotherapies include muscle relaxants, transdermal lidocaine, and various neuropathic pain medications (e.g., gabapentin, pregabalin, and tricyclic antidepressants). Although generally well tolerated, appropriate caution should be taken when prescribing skeletal muscle relaxants and neuropathic pain medications, especially in the elderly. Dizziness, somnolence, and gait disturbance are all documented side

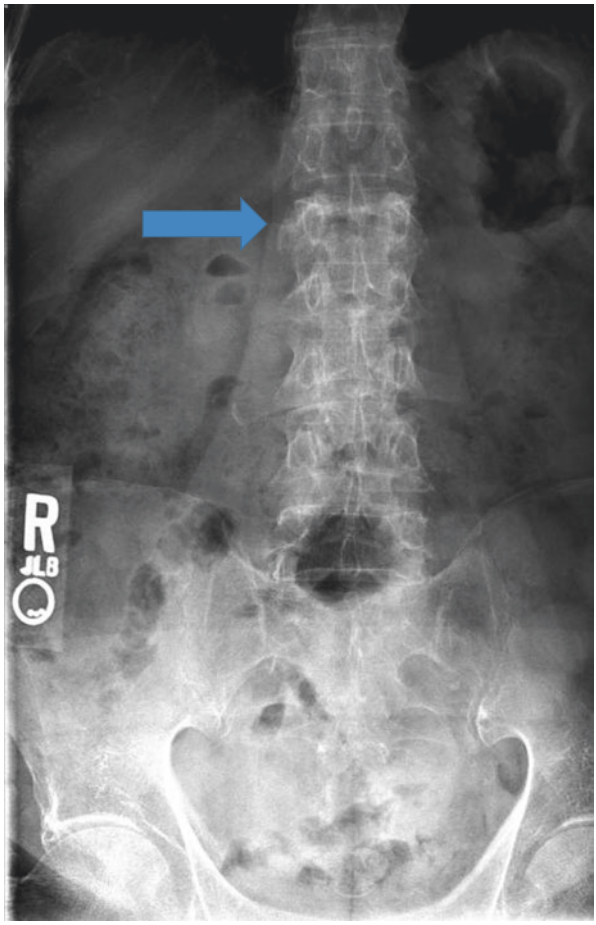


Fig. 11.4 AP X-ray of a L1 VCF

effects of gabapentin and the use of muscle relaxers has been shown to increase hospitalization rates in the elderly [45–47]. Tricyclic antidepressants, such as amitriptyline, reduce pain by inhibiting the reuptake of norepinephrine and serotonin. Tricyclics have demonstrated effectiveness in treating neuropathic pain but their common side effects including urinary retention, sedation, and postural hypotension may limit their use [48, 49].

Opioid pain medications may be required when patients fail to obtain adequate pain control with first-line analgesics and activity modification. Special consideration when prescribing opioids in the elderly include risk of reduced gastrointestinal motility, urinary retention, cognitive slowing, loss of balance, and increased risk of falls [50, 51]. However, a short course of opioid treatment can be an effective means of providing analgesia and preventing immobility secondary to uncontrolled, acute pain. When opioid medications are required a laxative can also be given to prevent constipation as straining with deification can acutely exacerbate VCF pain. As pain subsides, opioids should be tapered gradually while closely monitoring the patient's

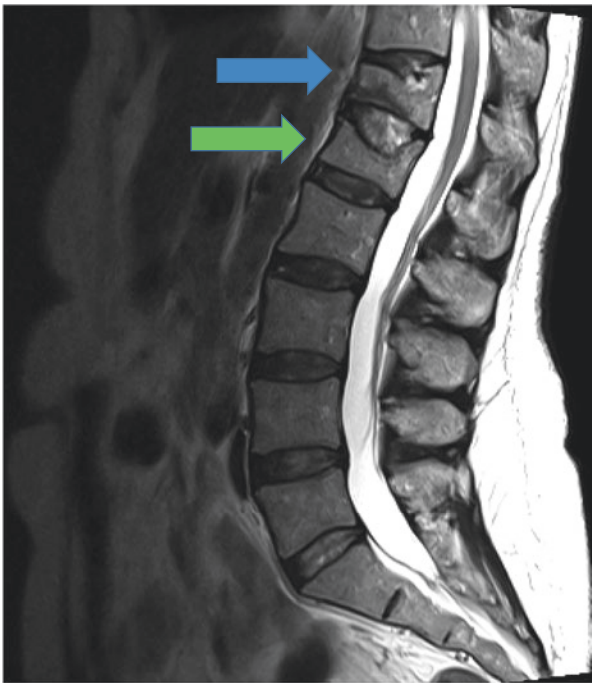


Fig. 11.5 T2-weighted MRI demonstrating an acute T12 VCF (blue arrow) and a chronic L1 VCF (green arrow)

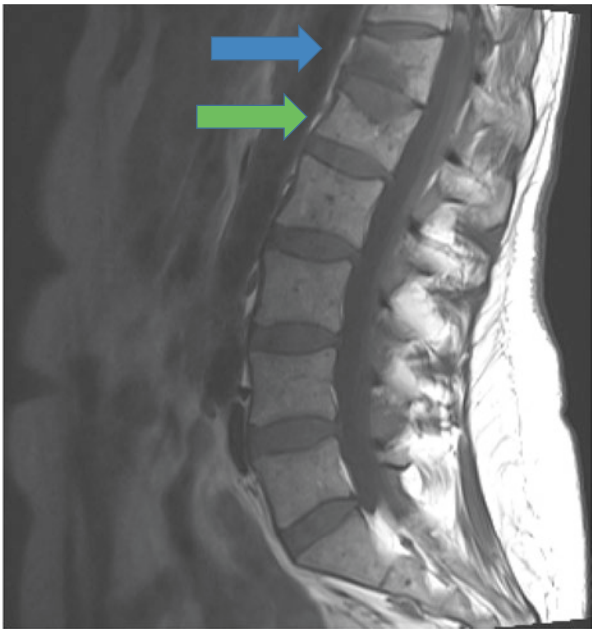


Fig. 11.6 T1-weighted MRI demonstrating an acute T12 VCF (blue arrow) and a chronic L1 VCF (green arrow)

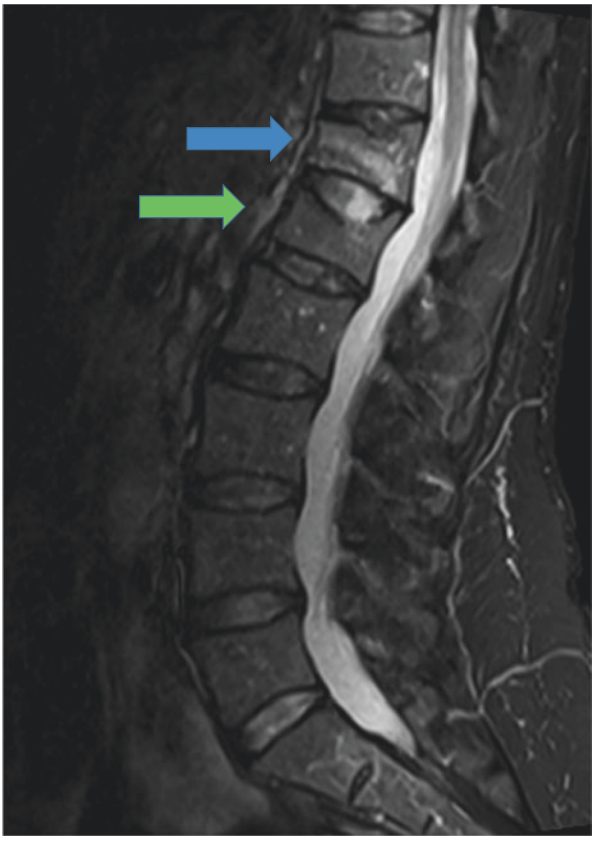


Fig. 11.7 STIR MRI sequence demonstrating increased STIR signal in an acute T12 VCF and normal signal in a chronic L1 VCF

response to dose reduction, including residual pain and functional status. Re-evaluation and optimization of non-opioid analgesics may also be appropriate as opioid analgesics are weaned. The risks and benefits of opioid medications should be carefully considered on a case-by-case basis.

Calcitonin may be used as an adjunct to traditional oral analgesics for pain control in acute VCF. It is also an option for patients with uncontrolled pain who cannot tolerate NSAIDs or opioids. Calcitonin is typically administered intranasally for a two to four-week course. Ideally, treatment should be initiated within 5 days following acute fracture [52, 53]. Although the exact mechanism of analgesia is unknown, calcitonin appears to exert a pain-relieving effect independent of its antiresorptive properties, possibly via a direct central nervous system mechanism involving calcitonin-binding receptors, modulation of peripheral prostaglandin levels, or by increasing plasma β-endorphin release [54, 55]. A meta-analysis by Knopp-Sihota et al., examining the combined results of 13 trials, demonstrated significant pain reduction with calcitonin administration following acute osteoporotic VCF. However, results from the analysis did not show any convincing evidence when calcitonin was used for chronic pain associated with older fractures [56]. Recently, there has been some concern that the long-term use of calcitonin may increase various cancer rates.

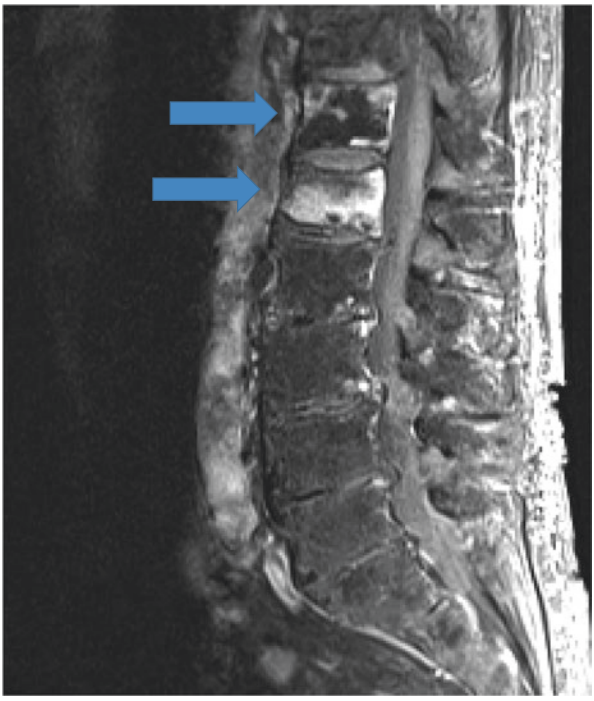


Fig. 11.8 MRI with contrast demonstrating abnormal marrow signal & post-contrast enhancement in pathologic T12 and L1 VCFs

Although a direct causal relationship has not been established there does appear to be a weak association with long-term use [57].

In summary, successful pharmacotherapy for the management of pain in VCF requires an individualized approach based on the intensity, quality, and duration of pain. A thorough understanding of the indications and potential side effects for each medication is also important. Medication indications and dosing regimens should be frequently reviewed as the natural course of pain associated with acute VCF typically improves over subsequent weeks.

Spinal Bracing

Spinal orthoses can also be used to reduce pain in patients following acute VCF. In general, braces are used to limit spinal flexion, thereby decreasing load on the fractured and painful anterior vertebral column [58]. Although high-quality evidence is lacking, bracing may also aid in limiting motion about the injured vertebrae to reduce pain, facilitate bone healing, prevent further vertebral body collapse, and decrease adjacent paraspinal muscle spasm by providing axial support [44, 53, 59–62]. Several bracing options are available for stable fractures including the Jewitt and CASH (Cruciform Anterior Spinal Hyperextension) orthoses. These braces provide a ridged 3-point contact system to promote neutral spine posture and limit flexion of the thoracic spine and thoracolumbar junction [61, 63]. Semi-ridged or flexible orthoses may also be appropriate for some patients and have been shown to



Fig. 11.9 Sagittal CT scan demonstrating a severe T9 VCF with mild posterior retropulsion

provide equivalent outcomes when compared to rigid bracing [58]. As pain subsides, braces should be weaned to avoid weakening of the axial musculature. Though some patients do find benefit from bracing, the most recent American Association of Orthopedic Surgeons guideline was unable to recommend for or against spinal bracing in patients with osteoporotic VCF, citing an overall lack of high-quality evidence [53].

Physical Therapy and Exercise

Physical therapy and directed exercise may also be employed as part of the multimodal treatment plan. Goals should include developing an individualized program focused on axial strengthening, balance, proper mechanics, and pain provoking activity modification. In addition to the positive impact of progressive resistance training on bone mineral density, exercise can also improve quality of life and reduce the risk of falls and fracture recurrence in patients with VCF [64–66]. A

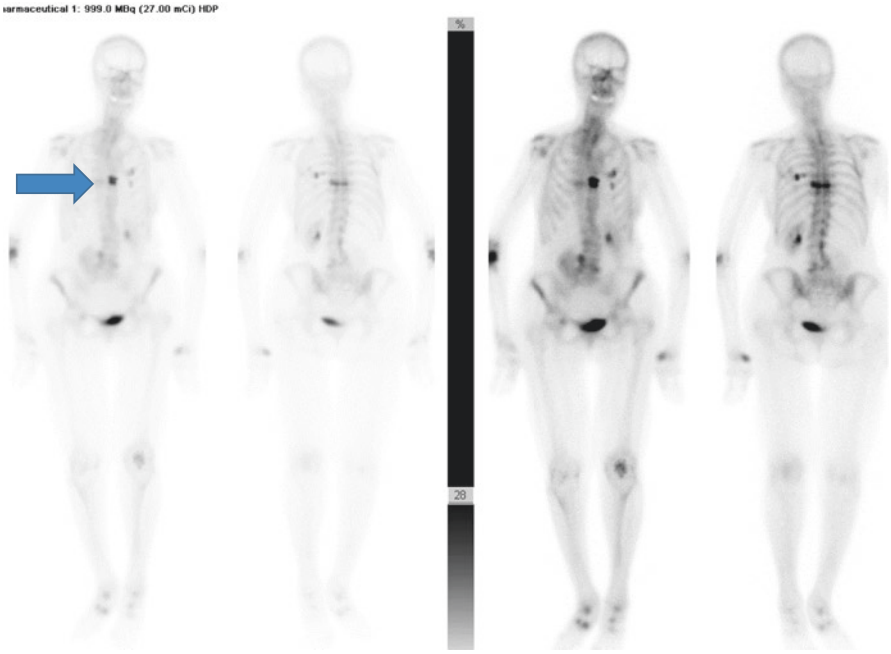


Fig. 11.10 Bone scan demonstrating increased radiotracer activity in a patient with several fractures, including a T9 VCF

retrospective review by Huntoon et al. concluded that a program of isometric back extensor strengthening in combination with proprioceptive postural retraining following osteoporotic VCF significantly decreased fracture recurrence following percutaneous vertebroplasty when compared to percutaneous vertebroplasty alone (4.5 vs. 20.4 months to re-fracture) [67]. Sinaki et al. examined the long-term effects of a 2-year resisted back extension program in healthy postmenopausal women without VCF. At 8-year follow-up, they found participants had a significant reduction in VCF risk and improved bone density compared to controls [66].

While several studies highlight the benefits of therapeutic exercise, a recent Cochran review examining exercise for improving outcomes following VCF, both alone or as part of a structured physical therapy intervention, drew no clinically relevant definitive conclusions [68]. The review included nine trials (749 participants). While some studies were positive and demonstrated improved pain, physical functioning, and quality of life, the overall quality of evidence was deemed weak. Additionally, there is no high-quality data regarding the safety of exercise following VCF or the effect on subsequent fracture risk. However, in general, safe therapeutic exercise programs can be developed based on the patient's current musculoskeletal status and individualized goals. Specific recommendations compiled by an expert consensus panel include limiting physical activity to moderate intensity, incorporating daily balance training, and development of spinal extensor muscle endurance [69]. Additional consensus recommendations included educating patients on proper posture and body mechanics during activities of daily living and stretching muscles

that prevent proper spinal alignment (e.g., tight pectoralis muscles causing exaggerated thoracic kyphosis) [69]. Finally, formal consultation with a physical therapist may be beneficial in patients with significant pain or debility to develop an individualized and graduated exercise plan [69].

Preventative Medicine & Bone Health

Interventions aimed at improving bone quality should also be addressed following an acute osteoporotic VCF. Treatment measures for osteoporosis include nutrition and lifestyle modification and pharmacologic therapy [70]. Lifestyle measures include exercise, smoking cessation, avoidance of excessive alcohol consumption, and fall prevention. Ensuring adequate calcium and vitamin D intake is also essential to bone health. The National Osteoporosis Foundation recommends a total calcium intake of 1200 milligrams per day for women over the age of 50 and men over the age of 70 and 800–1000 IU of vitamin D per day for men and women age 50 and older. Total calcium intake per day should include both dietary and supplemental forms taken in divided doses with meals. Consideration for initiation of pharmacotherapy is also appropriate following osteoporotic VCF [71]. A variety of medications are currently approved for the treatment of osteoporosis, including the bisphosphonates (alendronate, ibandronate, risedronate, and zoledronic acid), recombinant parathyroid hormone (teriparatide), receptor activator of nuclear factor kappa-B (RANK) ligand inhibitor (denosumab), and others. All agents act through either antiresorptive or osteogenic mechanisms. Choice of agent should be individualized and based on efficacy, safety, cost, and patient convenience [70, 72]. Referral to an endocrinologist, osteoporosis specialist, or to a dedicated osteoporosis coordinated care team should be considered to ensure patients who suffer a fracture receive appropriate diagnosis, treatment, education, and follow-up [73–75].

Spinal Injections

A hypothesis of facet-mediated pain following VCFs has been proposed. The posterior elements are thought to be strained biomechanically following a vertebral deformity [76]. A retrospective study evaluating the difference between vertebroplasty and facet medial branch blocks for pain associated with one-level VCFs found similar pain relief between the two groups at 2 years, and more cost-effectiveness in the medial branch block group [77].

Wang, et al., evaluated the difference in clinical outcomes of 206 patients that were randomized to undergo vertebroplasty versus facet blocks for back pain due to VCFs. The results demonstrated significantly better pain relief and functional outcomes a 1 week in the vertebroplasty group compared to the facet block group, however there were no significant differences between the two groups from 1 month to 12 months after the interventions [78]. These studies underscore the need for larger prospective randomized controlled trials evaluating facet blocks versus sham blocks and facet blocks versus vertebral augmentation in this patient population.

For some patients with VCFs, the kyphosis can lead to narrowing of the neural foramina at the level of the fractures. This can cause acute radicular pain symptoms in the distribution of the affected exiting nerve root. Consideration can be given to an epidural steroid injection for persistent or disabling radicular pain, however the potential adverse impact of repeat epidural steroid injections on bone mineral density should be taken into account [79].

Vertebral Augmentation

When conservative management fails to provide adequate pain relief, surgical intervention may be considered. Vertebroplasty and kyphoplasty are minimally invasive, percutaneous vertebral augmentation procedures frequently used to treat refractory pain secondary to osteoporotic and malignant VCF [80]. After a trial of conservative management, patients with persistent, severe back pain and physical exam and advanced imaging findings consistent with acute VCF (tenderness on palpation; vertebral end plate and/or marrow edema on MRI or increased radiotracer uptake on bone scintigraphy) are typically considered for treatment.

Vertebroplasty is a fluoroscopically guided procedure involving the percutaneous infusion of polymethylmethacrylate (PMMA) bone cement into the fractured vertebral body via a transpedicular approach. The objective is to reduce pain, stabilize the fractured elements, and provide structural support to the compromised trabecular bone. Kyphoplasty adds the additional step of inflating a balloon in the vertebral body in order to create a cavity for PMMA injection and to attempt restoration of vertebral height (Figs. 11.11, 11.12, 11.13, and 11.14). Both procedures

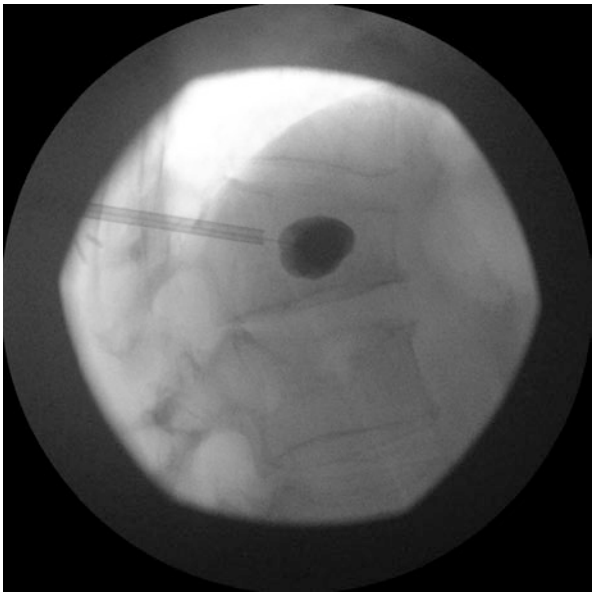


Fig. 11.11 Lateral fluoroscopy image demonstrating transpedicular kyphoplasty balloon inflation

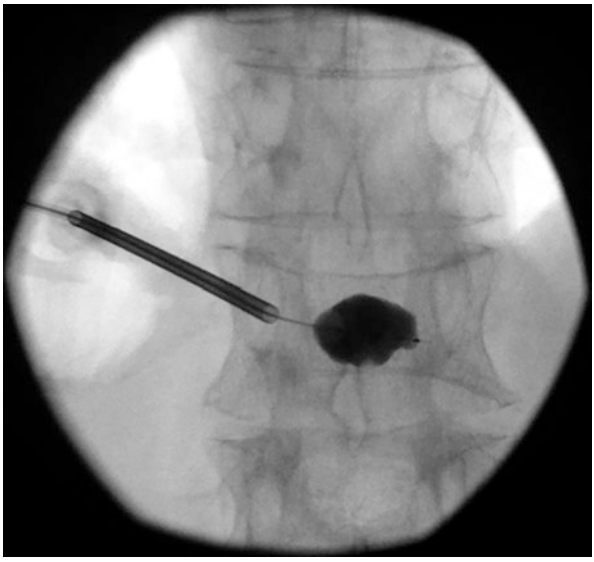


Fig. 11.12 AP fluoroscopy image demonstrating transpedicular kyphoplasty balloon inflation

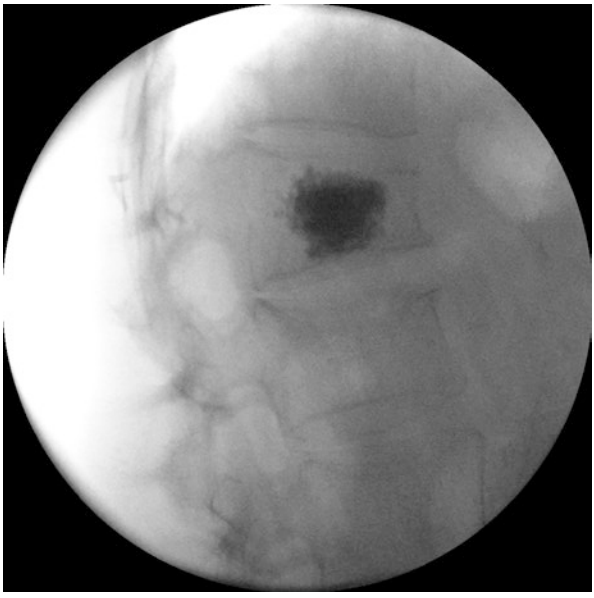


Fig. 11.13 Lateral fluoroscopy image demonstrating successful PMMA injection into a VCF

are typically preformed on an outpatient basis, under light sedation or general anesthesia. Procedural complications are rare, with major complications occurring in <1% of patients [81, 82]. Major complications include hemorrhage, osteomyelitis, cement pulmonary embolism, new procedure-related fractures, and permanent neurologic deficits [81]. Absolute contraindications to vertebral augmentation include

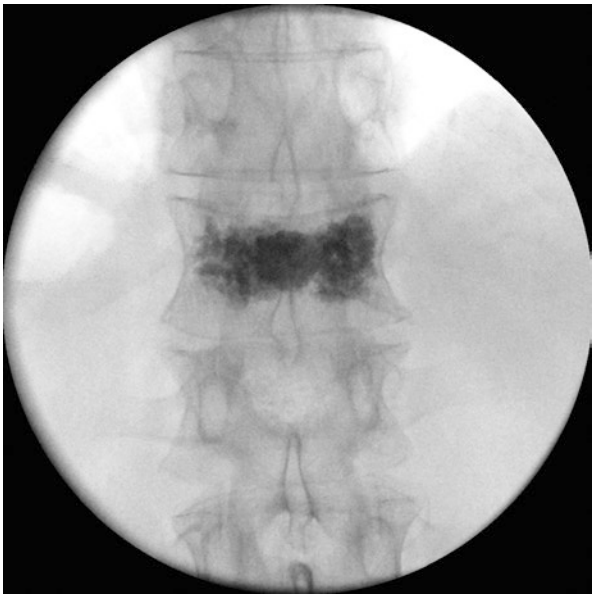


Fig. 11.14 AP fluoroscopy image demonstrating successful PMMA injection into a VCF

asymptomatic VCF, uncontrollable coagulopathy, unstable spinal fracture, active infection, or allergy to bone cement or opacification agents [83].

Although numerous studies have been published on the subject, the efficacy of vertebral augmentation remains controversial. Several early prospective randomized controlled trials (RCT) demonstrated positive results. The Vertebroplasty for Painful Chronic Osteoporotic Vertebral Fractures (VERTOS) trial, published in 2007, was the first prospective RCT comparing vertebroplasty to sham procedure [84]. Subacute and chronic (6–24 weeks) VCFs were included in the analysis. This study found significant improvement in pain scores at 24 hours post-vertebroplasty, but the effect was lost by 2 weeks. VERTOS II followed in 2010 and compared early vertebroplasty with medical management [85]. Inclusion criteria were moderate to severe back pain, fracture age <6 weeks, focal tenderness, and bone edema on MR imaging. At 1 month, there was significant improvement in visual analog scale (VAS) scores in the vertebroplasty group with durability at 1-year follow-up.

The Fracture Reduction Evaluation (FREE) trial, published in 2009, was the first RCT to compare kyphoplasty with medical management for acute and subacute (<3 months) VCFs causing moderate to severe back pain (numeric rating scale [NRS] ≥4/10) [86]. The primary end point, Short-Form-36 physical component summary scores, significantly improved following kyphoplasty at 1 and 6 months but the effect was lost at 24-month follow-up. This study also demonstrated a durable improvement in vertebral height restoration (27%) and kyphosis correction (3.3 degrees) at 24-month follow-up. Studies comparing vertebroplasty to kyphoplasty have generally shown comparable efficacy in reducing pain and disability in VCF [87–89].

While these early studies were overall encouraging, several trials produced negative results. For example, the 2009 Investigational Vertebroplasty Safety and Efficacy Trial (INVEST), designed to compare vertebroplasty with a sham procedure, demonstrated no difference in back pain between the two groups at 1 month [90]. Each of these early trials had limitations including lack of blinding (VERTOS II, FREE), inclusion of chronic fractures (VERTOS, INVEST), and enrollment of patients with moderate pain (VERTOS, VERTOS II, FREE, INVEST). Thus, debate continued regarding the efficacy of vertebral augmentation for painful VCF.

In 2016, the double blinded Vertebroplasty for Acute Painful Osteoporotic Fractures (VAPOUR) trial was designed to compare early vertebroplasty with sham procedure [91]. Patient selection was much more stringent and attempted to control for the limitations identified in prior studies. Inclusion criteria were 60 years of age or older, severe back pain, fracture age <6 weeks, and MR imaging with edema or SPECT CT uptake. One hundred twenty patients were enrolled and randomly assigned to treatment or sham. The primary end point was conversion of pain from severe (NRS ≥7) to mild (NRS <4) at 2-week follow-up. Significantly more patients had an NRS <4 at 2-week follow-up in the vertebroplasty compared to sham group (44% vs. 21%; $p = 0.01$), which was durable to 6 months. Mean NRS scores were also significantly decreased in the vertebroplasty compared to sham group at all time points up to 6 months. Additionally, vertebroplasty resulted in significantly improved disease-specific quality of life and significantly less analgesic use at 3 and 6 months.

Finally, VERTOS IV, published in 2018, is the most recent double-blinded RCT comparing vertebroplasty to sham procedure in VCF [92]. Inclusion criteria included fracture age <6 weeks, VAS score ≥5, focal back pain, and edema on MRI. Due to slow recruitment, inclusion of fractures up to 9 weeks was ultimately allowed. One hundred and eighty patients were randomized to vertebroplasty or sham. Results revealed VAS scores, the primary end point, did not differ between the two groups at any time point from 1-day to 1-year follow-up. Notably, pain in both groups significantly improved at all time points. By 12-month follow-up, mean VAS scores had declined by 5.00 in the vertebroplasty group and 4.75 in the sham group.

Interpretation of the available evidence is challenging given the heterogeneity of study inclusion criteria, open vs. blinded design, and variable use of sham procedure. Questions remain regarding the optimal timing of intervention and which patient characteristics indicate favorable outcome. Overall, evidence has shown that those with acute fractures (<6 weeks) and severe pain may benefit from vertebral augmentation. This statement is consistent with the recommendations of a multisociety interventional spine panel which found vertebral augmentation to be a safe and valid treatment option for painful VCF refractory to medical management [82]. Further high-quality studies may also aid in defining the long-term impact of vertebral augmentation on other important outcome measures such as fall risk, adjacent fracture risk, future vertebral height loss and kyphosis.

In summary, a multimodal approach to the management of painful VCF is often necessary. While most patients achieve adequate pain controlled with conservative measures alone, vertebral augmentation may be considered for those with severe,

refractory pain following acute VCF. In addition to controlling pain and promoting function, timely evaluation and treatment of bone health is of high importance. Successful management may also necessitate coordination across a multidisciplinary team, including the primary care physician, endocrinologist or osteoporosis specialist, oncologist and radiation oncologist when malignancy is known or suspected, and interventional spine specialist [33]. Further high-quality studies are needed to better inform individualized management strategies.

Conclusion

VCFs are a common cause of back pain and disability, especially in the elderly. While osteoporosis in the most likely etiology, other causes, such as malignancy, must not be overlooked. Although most VCFs are asymptomatic, some patients may experience significant fracture-related pain and functional deficits resulting in poor quality of life and high socioeconomic costs. Diagnostic studies may include plain film radiographs or more advanced imagining, such as MRI. The patient history and physical exam are important and often aid in establishing the diagnosis and fracture acuity. Timely evaluation and optimization of bone health following an osteoporotic VCF is important in reducing the risk of new fractures. When indicated, treatment of osteoporosis should be initiated given the high risk of subsequent fracture. Most patients who suffer an acute VCF respond to conservative management, with pain gradually resolving over several weeks. Successful conservative treatment is often multimodal and may include medications for pain control, physical therapy, and spinal bracing. For those patients with an acute fracture who continue to experience significant pain, despite a trial of conservative therapy, vertebral augmentation can be considered.

References

1. Riggs BL, Melton LJ 3rd. The worldwide problem of osteoporosis: insights afforded by epidemiology. Bone. 1995;17(5 Suppl):505S–11S.
2. Cauley JA, Hochberg MC, Lui LY, Palermo L, Ensrud KE, Hillier TA, et al. Long-term risk of incident vertebral fractures. JAMA. 2007;298(23):2761–7.
3. Nevitt MC, Cummings SR, Stone KL, Palermo L, Black DM, Bauer DC, et al. Risk factors for a first-incident radiographic vertebral fracture in women > or = 65 years of age: the study of osteoporotic fractures. J Bone Miner Res. 2005;20(1):131–40.
4. Wright NC, Looker AC, Saag KG, Curtis JR, Delzell ES, Randall S, et al. The recent prevalence of osteoporosis and low bone mass in the United States based on bone mineral density at the femoral neck or lumbar spine. J Bone Miner Res. 2014;29(11):2520–6.
5. Alexandru D, So W. Evaluation and management of vertebral compression fractures. Perm J. 2012;16(4):46–51.
6. Kanis JA. Diagnosis of osteoporosis and assessment of fracture risk. Lancet. 2002;359(9321):1929–36.
7. Melton LJ 3rd. Epidemiology of spinal osteoporosis. Spine (Phila Pa 1976). 1997;22(24 Suppl):2s–11s.

8. Melton LJ 3rd, Lane AW, Cooper C, Eastell R, O'Fallon WM, Riggs BL. Prevalence and incidence of vertebral deformities. Osteoporos Int. 1993;3(3):113–9.
9. Lindsay R, Silverman SL, Cooper C, Hanley DA, Barton I, Broy SB, et al. Risk of new vertebral fracture in the year following a fracture. JAMA. 2001;285(3):320–3.
10. Ross PD, Davis JW, Epstein RS, Wasnich RD. Pre-existing fractures and bone mass predict vertebral fracture incidence in women. Ann Intern Med. 1991;114(11):919–23.
11. Hernandez RK, Wade SW, Reich A, Pirolli M, Liede A, Lyman GH. Incidence of bone metastases in patients with solid tumors: analysis of oncology electronic medical records in the United States. BMC Cancer. 2018;18(1):44.
12. Anselmetti GC, Manca A, Montemurro F, Hirsch J, Chiara G, Grignani G, et al. Percutaneous vertebroplasty in multiple myeloma: prospective long-term follow-up in 106 consecutive patients. Cardiovasc Intervent Radiol. 2012;35(1):139–45.
13. Fink HA, Milavetz DL, Palermo L, Nevitt MC, Cauley JA, Genant HK, et al. What proportion of incident radiographic vertebral deformities is clinically diagnosed and vice versa? J Bone Miner Res. 2005;20(7):1216–22.
14. Cook DJ, Guyatt GH, Adachi JD, Clifton J, Griffith LE, Epstein RS, et al. Quality of life issues in women with vertebral fractures due to osteoporosis. Arthritis Rheum. 1993;36(6):750–6.
15. Jung HJ, Park YS, Seo HY, Lee JC, An KC, Kim JH, et al. Quality of life in patients with osteoporotic vertebral compression fractures. J Bone Metab. 2017;24(3):187–96.
16. Ross PD. Clinical consequences of vertebral fractures. Am J Med. 1997;103(2A):30S–42S; discussion S-3S.
17. Suzuki N, Ogikubo O, Hansson T. The course of the acute vertebral body fragility fracture: its effect on pain, disability and quality of life during 12 months. Eur Spine J. 2008;17(10):1380–90.
18. Gold DT. The clinical impact of vertebral fractures: quality of life in women with osteoporosis. Bone. 1996;18(3 Suppl):185S–9S.
19. Kim KW, Cho KJ, Kim SW, Lee SH, An MH, Im JH. A nation-wide, outpatient-based survey on the pain, disability, and satisfaction of patients with osteoporotic vertebral compression fractures. Asian Spine J. 2013;7(4):301–7.
20. Suzuki N, Ogikubo O, Hansson T. Previous vertebral compression fractures add to the deterioration of the disability and quality of life after an acute compression fracture. Eur Spine J. 2010;19(4):567–74.
21. Cooper C, Atkinson EJ, Jacobsen SJ, O'Fallon WM, Melton LJ 3rd. Population-based study of survival after osteoporotic fractures. Am J Epidemiol. 1993;137(9):1001–5.
22. Burge R, Dawson-Hughes B, Solomon DH, Wong JB, King A, Tosteson A. Incidence and economic burden of osteoporosis-related fractures in the United States, 2005–2025. J Bone Miner Res. 2007;22(3):465–75.
23. Williams SA, Chastek B, Sundquist K, Barrera-Sierra S, Leader D Jr, Weiss RJ, et al. Economic burden of osteoporotic fractures in US managed care enrollees. Am J Manag Care. 2020;26(5):e142–e9.
24. Dolan P, Torgerson DJ. The cost of treating osteoporotic fractures in the United Kingdom female population. Osteoporos Int. 1998;8(6):611–7.
25. Gehlbach SH, Burge RT, Puleo E, Klar J. Hospital care of osteoporosis-related vertebral fractures. Osteoporos Int. 2003;14(1):53–60.
26. Freedman BA, Potter BK, Nesti LJ, Giuliani JR, Hampton C, Kuklo TR. Osteoporosis and vertebral compression fractures-continued missed opportunities. Spine J. 2008;8(5):756–62.
27. Patel U, Skingle S, Campbell GA, Crisp AJ, Boyle IT. Clinical profile of acute vertebral compression fractures in osteoporosis. Br J Rheumatol. 1991;30(6):418–21.
28. Ughwanogho E, Hebela NM. Vertebral compression fractures. In: Pignolo RJ, Keenan MA, Hebela NM, editors. Fractures in the elderly: a guide to practical management. Totowa, NJ: Humana Press; 2011. p. 225–37.
29. Rosen H, Walega D. Osteoporotic thoracolumbar vertebral compression fractures: Clinical manifestations and treatment. Waltham, MA: Up-to-Date [database on the Internet]; 2020. Available from: http://uptodate.com.

30. Langdon J, Way A, Heaton S, Bernard J, Molloy S. Vertebral compression fractures--new clinical signs to aid diagnosis. Ann R Coll Surg Engl. 2010;92(2):163–6.
31. Tay BKB, Freedman BA, Rhee JM, Boden SD, Skinner HB. Chapter 4. Disorders, diseases, and injuries of the spine. In: Skinner HB, PJ MM, editors. Current diagnosis & treatment in orthopedics. 5th ed. New York, NY: The McGraw-Hill Companies; 2014.
32. Heggeness MH. Spine fracture with neurological deficit in osteoporosis. Osteoporos Int. 1993;3(4):215–21.
33. Expert Panels on Neurological Imaging IR, Musculoskeletal I, Shah LM, Jennings JW, CFE K, Hohenwalter EJ, et al. ACR appropriateness criteria((R)) management of vertebral compression fractures. J Am Coll Radiol. 2018;15(11S):S347–S64.
34. Donnally III C, DiPompeo C, Varacallo M. Vertebral compression fractures. Treasure Island, FL: StatPearls Publishing; 2020 [08 Nov 2020]. Available from: https://www.ncbi.nlm.nih.gov/books/NBK448171/.
35. Jarraya M, Hayashi D, Griffith J, Guermazi A, Genant H. Identification of vertebral fractures. In: Guglielmi G, editor. Osteoporosis and bone densitometry measurements. Berlin, Heidelberg: Springer Berlin Heidelberg; 2013. p. 41–55.
36. Mauch JT, Carr CM, Cloft H, Diehn FE. Review of the Imaging features of benign osteoporotic and malignant vertebral compression fractures. AJNR Am J Neuroradiol. 2018;39(9):1584–92.
37. Thawait S, Marcus M, Morrison W, Klufas R, Eng J, Carrino J. Research synthesis: what is the diagnostic performance of magnetic resonance imaging to discriminate benign from malignant vertebral compression fractures? Systematic review and meta-analysis. Spine (Phila Pa 1976). 2012;37(12):E736–44.
38. Stallmeyer MJB, Zoarski GH. Patient evaluation and selection. In: Mathis JM, Deramond H, Belkoff SM, editors. Percutaneous nertebroplasty. New York, NY: Springer New York; 2002. p. 41–60.
39. Silverman SL. The clinical consequences of vertebral compression fracture. Bone. 1992;13(Suppl 2):S27–31.
40. Chan FK, Lanas A, Scheiman J, Berger MF, Nguyen H, Goldstein JL. Celecoxib versus omeprazole and diclofenac in patients with osteoarthritis and rheumatoid arthritis (CONDOR): a randomised trial. Lancet. 2010;376(9736):173–9.
41. Emery P, Zeidler H, Kvien TK, Guslandi M, Naudin R, Stead H, et al. Celecoxib versus diclofenac in long-term management of rheumatoid arthritis: randomised double-blind comparison. Lancet. 1999;354(9196):2106–11.
42. Simon LS, Weaver AL, Graham DY, Kivitz AJ, Lipsky PE, Hubbard RC, et al. Anti-inflammatory and upper gastrointestinal effects of celecoxib in rheumatoid arthritis: a randomized controlled trial. JAMA. 1999;282(20):1921–8.
43. Dodwell ER, Latorre JG, Parisini E, Zwettler E, Chandra D, Mulpuri K, et al. NSAID exposure and risk of nonunion: a meta-analysis of case-control and cohort studies. Calcif Tissue Int. 2010;87(3):193–202.
44. Longo UG, Loppini M, Denaro L, Maffulli N, Denaro V. Conservative management of patients with an osteoporotic vertebral fracture: a review of the literature. J Bone Joint Surg Br. 2012;94(2):152–7.
45. Alvarez CA, Mortensen EM, Makris UE, Berlowitz DR, Copeland LA, Good CB, et al. Association of skeletal muscle relaxers and antihistamines on mortality, hospitalizations, and emergency department visits in elderly patients: a nationwide retrospective cohort study. BMC Geriatr. 2015;15:2.
46. Derry S, Bell RF, Straube S, Wiffen PJ, Aldington D, Moore RA. Pregabalin for neuropathic pain in adults. Cochrane Database Syst Rev. 2019;1(1):CD007076.
47. Wiffen PJ, Derry S, Bell RF, Rice AS, Tolle TR, Phillips T, et al. Gabapentin for chronic neuropathic pain in adults. Cochrane Database Syst Rev. 2017;6(6):CD007938.
48. Abdulla A, Adams N, Bone M, Elliott AM, Gaffin J, Jones D, et al. Guidance on the management of pain in older people. Age Ageing. 2013;42 Suppl 1:i1–57.

49. Finnerup NB, Attal N, Haroutounian S, McNicol E, Baron R, Dworkin RH, et al. Pharmacotherapy for neuropathic pain in adults: a systematic review and meta-analysis. Lancet Neurol. 2015;14(2):162–73.
50. Rolita L, Spegman A, Tang X, Cronstein BN. Greater number of narcotic analgesic prescriptions for osteoarthritis is associated with falls and fractures in elderly adults. J Am Geriatr Soc. 2013;61(3):335–40.
51. Solomon DH, Rassen JA, Glynn RJ, Garneau K, Levin R, Lee J, et al. The comparative safety of opioids for nonmalignant pain in older adults. Arch Intern Med. 2010;170(22):1979–86.
52. Lyritis GP, Paspati I, Karachalios T, Ioakimidis D, Skarantavos G, Lyritis PG. Pain relief from nasal salmon calcitonin in osteoporotic vertebral crush fractures. A double blind, placebo-controlled clinical study. Acta Orthop Scand Suppl. 1997;275:112–4.
53. Esses SI, McGuire R, Jenkins J, Finkelstein J, Woodard E, Watters WC 3rd, et al. American Academy of Orthopaedic Surgeons clinical practice guideline on: the treatment of osteoporotic spinal compression fractures. J Bone Joint Surg Am. 2011;93(20):1934–6.
54. Azria M. Possible mechanisms of the analgesic action of calcitonin. Bone. 2002;30(5 Suppl):80S–3S.
55. Ofluoglu D, Akyuz G, Unay O, Kayhan O. The effect of calcitonin on beta-endorphin levels in postmenopausal osteoporotic patients with back pain. Clin Rheumatol. 2007;26(1):44–9.
56. Knopp-Sihota JA, Newburn-Cook CV, Homik J, Cummings GG, Voaklander D. Calcitonin for treating acute and chronic pain of recent and remote osteoporotic vertebral compression fractures: a systematic review and meta-analysis. Osteoporos Int. 2012;23(1):17–38.
57. Wells G, Chernoff J, Gilligan JP, Krause DS. Does salmon calcitonin cause cancer? A review and meta-analysis. Osteoporos Int. 2016;27(1):13–9.
58. Kato T, Inose H, Ichimura S, Tokuhashi Y, Nakamura H, Hoshino M, et al. Comparison of rigid and soft-brace treatments for acute osteoporotic vertebral compression fracture: a prospective, randomized, multicenter study. J Clin Med. 2019;8(2)
59. Meccariello L, Muzii VF, Falzarano G, Medici A, Carta S, Fortina M, et al. Dynamic corset versus three-point brace in the treatment of osteoporotic compression fractures of the thoracic and lumbar spine: a prospective, comparative study. Aging Clin Exp Res. 2017;29(3):443–9.
60. Pfeifer M, Begerow B, Minne HW. Effects of a new spinal orthosis on posture, trunk strength, and quality of life in women with postmenopausal osteoporosis: a randomized trial. Am J Phys Med Rehabil. 2004;83(3):177–86.
61. Prather H, Watson JO, Gilula LA. Nonoperative management of osteoporotic vertebral compression fractures. Injury. 2007;38(Suppl 3):S40–8.
62. Stadhouder A, Buskens E, Vergroesen DA, Fidler MW, de Nies F, Oner FC. Nonoperative treatment of thoracic and lumbar spine fractures: a prospective randomized study of different treatment options. J Orthop Trauma. 2009;23(8):588–94.
63. Newman M, Minns Lowe C, Barker K. Spinal orthoses for vertebral osteoporosis and osteoporotic vertebral fracture: a systematic review. Arch Phys Med Rehabil. 2016;97(6):1013–25.
64. Sinaki M. Exercise for patients with osteoporosis: management of vertebral compression fractures and trunk strengthening for fall prevention. PM R. 2012;4(11):882–8.
65. Papaioannou A, Adachi JD, Winegard K, Ferko N, Parkinson W, Cook RJ, et al. Efficacy of home-based exercise for improving quality of life among elderly women with symptomatic osteoporosis-related vertebral fractures. Osteoporos Int. 2003;14(8):677–82.
66. Sinaki M, Itoi E, Wahner HW, Wollan P, Gelzcer R, Mullan BP, et al. Stronger back muscles reduce the incidence of vertebral fractures: a prospective 10 year follow-up of postmenopausal women. Bone. 2002;30(6):836–41.
67. Huntoon EA, Schmidt CK, Sinaki M. Significantly fewer refractures after vertebroplasty in patients who engage in back-extensor-strengthening exercises. Mayo Clin Proc. 2008;83(1):54–7.
68. Gibbs JC, MacIntyre NJ, Ponzano M, Templeton JA, Thabane L, Papaioannou A, et al. Exercise for improving outcomes after osteoporotic vertebral fracture. Cochrane Database Syst Rev. 2019;7(7):CD008618.

69. Giangregorio LM, McGill S, Wark JD, Laprade J, Heinonen A, Ashe MC, et al. Too Fit To Fracture: outcomes of a Delphi consensus process on physical activity and exercise recommendations for adults with osteoporosis with or without vertebral fractures. Osteoporos Int. 2015;26(3):891–910.
70. Eastell R, Rosen CJ, Black DM, Cheung AM, Murad MH, Shoback D. Pharmacological management of osteoporosis in postmenopausal women: an endocrine society* clinical practice guideline. J Clin Endocrinol Metab. 2019;104(5):1595–622.
71. Cosman F, de Beur SJ, LeBoff MS, Lewiecki EM, Tanner B, Randall S, et al. Clinician's guide to prevention and treatment of osteoporosis. Osteoporos Int. 2014;25(10):2359–81.
72. Barrionuevo P, Gionfriddo MR, Castaneda-Guarderas A, Zeballos-Palacios C, Bora P, Mohammed K, et al. Women's values and preferences regarding osteoporosis treatments: a systematic review. J Clin Endocrinol Metab. 2019;104(5):1631–6.
73. Camacho PM, Petak SM, Binkley N, Diab DL, Eldeiry LS, Farooki A, et al. American Association of Clinical Endocrinologists/American College of Endocrinology Clinical Practice Guidelines for the Diagnosis and Treatment of Postmenopausal Osteoporosis-2020 Update. Endocr Pract. 2020;26(Suppl 1):1–46.
74. Eisman JA, Bogoch ER, Dell R, Harrington JT, McKinney RE Jr, McLellan A, et al. Making the first fracture the last fracture: ASBMR task force report on secondary fracture prevention. J Bone Miner Res. 2012;27(10):2039–46.
75. Dumitrescu B, van Helden S, ten Broeke R, Nieuwenhuijzen-Kruseman A, Wyers C, Udrea G, et al. Evaluation of patients with a recent clinical fracture and osteoporosis, a multidisciplinary approach. BMC Musculoskelet Disord. 2008;9:109.
76. Bogduk N, MacVicar J, Borowczyk J. The pain of vertebral compression fractures can arise in the posterior elements. Pain Med. 2010;11(11):1666–73.
77. Bae IS, Chun HJ, Bak KH, Yi HJ, Choi KS, Kim KD. Medial branch block versus vertebroplasty for 1-level osteoporotic vertebral compression fracture: 2-year retrospective study. World Neurosurg. 2019;122:e1599–e605.
78. Wang B, Guo H, Yuan L, Huang D, Zhang H, Hao D. A prospective randomized controlled study comparing the pain relief in patients with osteoporotic vertebral compression fractures with the use of vertebroplasty or facet blocking. Eur Spine J. 2016;25(11):3486–94.
79. Nah SY, Lee JH, Lee JH. Effects of epidural steroid injections on bone mineral density and bone turnover markers in patients taking anti-osteoporotic medications. Pain Physician. 2018;21(4):E435–e47.
80. Gray DT, Hollingworth W, Onwudiwe N, Jarvik JG. Costs and state-specific rates of thoracic and lumbar vertebroplasty, 2001–2005. Spine (Phila Pa 1976). 2008;33(17):1905–12.
81. Baerlocher MO, Saad WE, Dariushnia S, Barr JD, McGraw JK, Nikolic B, et al. Quality improvement guidelines for percutaneous vertebroplasty. J Vasc Interv Radiol. 2014;25(2):165–70.
82. Barr JD, Jensen ME, Hirsch JA, JK MG, Barr RM, Brook AL, et al. Position statement on percutaneous vertebral augmentation: a consensus statement developed by the Society of Interventional Radiology (SIR), American Association of Neurological Surgeons (AANS) and the Congress of Neurological Surgeons (CNS), American College of Radiology (ACR), American Society of Neuroradiology (ASNR), American Society of Spine Radiology (ASSR), Canadian Interventional Radiology Association (CIRA), and the Society of NeuroInterventional Surgery (SNIS). J Vasc Interv Radiol. 2014;25(2):171–81.
83. Tsoumakidou G, Too CW, Koch G, Caudrelier J, Cazzato RL, Garnon J, et al. CIRSE guidelines on percutaneous vertebral augmentation. Cardiovasc Intervent Radiol. 2017;40(3):331–42.
84. Voormolen MH, Mali WP, Lohle PN, Fransen H, Lampmann LE, van der Graaf Y, et al. Percutaneous vertebroplasty compared with optimal pain medication treatment: short-term clinical outcome of patients with subacute or chronic painful osteoporotic vertebral compression fractures. The VERTOS study. AJNR Am J Neuroradiol. 2007;28(3):555–60.
85. Klazen CA, Lohle PN, de Vries J, Jansen FH, Tielbeek AV, Blonk MC, et al. Vertebroplasty versus conservative treatment in acute osteoporotic vertebral compression fractures (Vertos II): an open-label randomised trial. Lancet. 2010;376(9746):1085–92.

86. Wardlaw D, Cummings SR, Van Meirhaeghe J, Bastian L, Tillman JB, Ranstam J, et al. Efficacy and safety of balloon kyphoplasty compared with non-surgical care for vertebral compression fracture (FREE): a randomised controlled trial. Lancet. 2009;373(9668):1016–24.
87. Dohm M, Black CM, Dacre A, Tillman JB, Fueredi G, investigators K. A randomized trial comparing balloon kyphoplasty and vertebroplasty for vertebral compression fractures due to osteoporosis. AJNR Am J Neuroradiol. 2014;35(12):2227–36.
88. Evans AJ, Kip KE, Brinjikji W, Layton KF, Jensen ML, Gaughen JR, et al. Randomized controlled trial of vertebroplasty versus kyphoplasty in the treatment of vertebral compression fractures. J Neurointerv Surg. 2016;8(7):756–63.
89. Liu JT, Li CS, Chang CS, Liao WJ. Long-term follow-up study of osteoporotic vertebral compression fracture treated using balloon kyphoplasty and vertebroplasty. J Neurosurg Spine. 2015;23(1):94–8.
90. Kallmes DF, Comstock BA, Heagerty PJ, Turner JA, Wilson DJ, Diamond TH, et al. A randomized trial of vertebroplasty for osteoporotic spinal fractures. N Engl J Med. 2009;361(6):569–79.
91. Clark W, Bird P, Gonski P, Diamond TH, Smerdely P, McNeil HP, et al. Safety and efficacy of vertebroplasty for acute painful osteoporotic fractures (VAPOUR): a multicentre, randomised, double-blind, placebo-controlled trial. Lancet. 2016;388(10052):1408–16.
92. Firanescu CE, de Vries J, Lodder P, Venmans A, Schoemaker MC, Smeets AJ, et al. Vertebroplasty versus sham procedure for painful acute osteoporotic vertebral compression fractures (VERTOS IV): randomised sham controlled clinical trial. BMJ. 2018;361:k1551.

Epidural Lysis of Adhesions

12

Gabor Bela Racz and Gabor J. Racz

Introduction

The goal of the lysis of adhesions chapter is to separate the tissue plane between the nerve root and epidural structures. The sliding motion between the posterior longitudinal ligament and the dura mater can become reduced from disk material leaking into the space and causing inflammation and scarring. Pain from this scarring can be identified by performing a dural tug maneuver. To perform the dural tug maneuver, the patient's spine is maximally flexed and the patient is asked to point to the location of back pain. It is thought that the pain is localized to the painful level where the dura is adhered to the posterior longitudinal ligament. The posterior longitudinal ligament is highly innervated and is a source of back pain [1]. The procedures for caudal, sacral, lumbar transforaminal and cervical lysis of adhesions have been described in detail elsewhere and much of the text and images are reproduced herein [2].

Recent Modifications

Recent modifications to the technique are summarized here. They include obtaining a lateral view during contrast injection to ensure that the ventrolateral epidural space is opened [3]. This is critical and a lack of ventral spread explains many of the failures that have been reported with the procedure. Midline catheter placement has been used for epidural lysis of adhesions however to ensure ventrolateral

G. B. Racz
Department of Anesthesiology, Texas Tech University Health Science Center, Lubbock, TX, USA

G. J. Racz (✉)
Epimed International, Dallas, TX, USA
e-mail: gabor@epimed.com

© The Author(s), under exclusive license to Springer Nature Switzerland AG 2022

C. E. Noe (ed.), *Multidisciplinary Spine Care*,
https://doi.org/10.1007/978-3-031-04990-3_12

medication placement, the midline catheter position is not recommended [4]. Also, in a study of transforaminal injections, 8 ml of injected volume was more effective than 3 ml, with the same steroid dose [5]. This supports the concept that spreading medications around the epidural space is effective. With lysis of epidural adhesions, hypertonic saline is diluted with lidocaine to a final concentration of 10% saline in order to prevent pain with injection of hypertonic saline. Five percent hypertonic has been used and was associated with less pain during infusion but without other advantage [6]. Pain can be effectively prevented by using lidocaine before injecting hypertonic saline and 10% has a longer duration of effect than 5%. The use of additional lidocaine before hypertonic saline injection is helpful to prevent pain in case hypertonic saline breaks out of an anesthetized compartment and flows into an area of the epidural space that is not anesthetized. Additionally, it important to flush hypertonic saline from the catheter to avoid pain that results from injecting the residual hypertonic saline on a subsequent day without local anesthetic in the epidural space.

If success is not apparent within several days, the procedure may be repeated at 6 weeks rather than waiting for 3 months, during which time, scarring can reform. In the past, repeating the procedure was delayed to avoid overtreatment but it has been learned that results are better if a second procedure is done sooner to avoid a return to baseline scarring levels. Finally, the dural flossing exercises should be performed 10 times per day for several weeks and neural flossing exercises shoulder be performed indefinitely. These exercised are described later in the chapter.

Lysis of Epidural Adhesions

Epidural lysis of adhesions employs site-specific catheter placement to the desired ventral lateral epidural space and verifying that the space is opened up at the symptomatic level to achieve freeing up of the nerve root. If this is not achieved, placement of a transforaminal catheter to open up the symptomatic level is performed. Lysis of adhesions with the local steroid and hypertonic saline sequence is performed either as a one-day procedure or as three separate injections. Lysis of epidural adhesions was developed as a three-day technique with injections each day; however, the technique has been adapted to a shorter treatment using injections at least 6 hours apart over a day and a half. Once the lysis procedure has been completed, the patient begins neural-flossing exercises. Since the pulling forces with exercises are relatively small, exercises alone are not sufficient to resolve symptoms associated with epidural adhesions. The physical lysis of adhesion by fluid dispersion in the tissue plane is crucial. Because the most significant innervation to the spinal canal component of the disc is the sinu- vertebral system, ventrolateral spinal canal catheter placement is optimal and this type of pain responds very well to the use of hypertonic saline.

Hypertonic saline has long-term analgesic effects when used in the epidural space for lysis of epidural adhesions. The recovery of action potentials in myelinated neurons has been demonstrated after application of hypertonic saline [7].

Hitchcock reported the use of cold saline and hypertonic saline for the treatment of pain decades ago [8, 9]. Birkenmaier studied hypertonic saline in a human fibroblast cell culture but did not study recovery of action potentials [10]. Hyperosmolar solutions are similar to local anesthetic in that nerve conduction recovers but fibroblasts are inhibited. Heavner, et al. performed a prospective, randomized blinded trial of lesion specific epidural adhesiolysis on 59 patients with chronic intractable low back pain [11]. The combination of hypertonic saline and hyaluronidase has provided the best results with epidural lysis of adhesions [12]. Hyaluronidase enhances the spread of injected medications in the epidural space and has an inhibitory effect on neutrophil infiltration. This occurs to a similar degree in animal extract as well as recombinant hyaluronidase preparations [13]. Using hyaluronidase has been criticized for increasing risks and costs but data to support these conclusions was not presented [14]. Neutrophil infiltration is the first step in the inflammation cascade that leads to edema and early pain. Hyaluronidase is useful in preventing local edema and neuropathic post- procedure sensitivity and pain. In the epidural space, these medications are safe and effective but epidural placement must be confirmed with radiographic imaging and local anesthetic test doses to rule out subdural or other placement.

Applied Anatomy

The spinal epidural space lies within the spinal canal from the foramen magnum to the sacral hiatus. The posterior epidural space is bounded by the ligament flavum posteriorly and the dura mater anteriorly. The epidural space contains veins and fat as well as segmental arteries. The epidural space extends laterally into the neural foramina. The epidural space may be compartmentalized by attachment of the dura mater to the ligamentum flavum lamina or other structures. A negative pressure is produced in the epidural space by the pressure gradient associated with inspiration. The caudal epidural space is entered through the sacral hiatus. The sacral hiatus is easily palpated at the superior aspect of the gluteal fold. The thecal sac extends inferiorly to S3 so needle tip placement should be below this level to avoid dural puncture. The first sacral foramina is oriented in a plane that required fluoroscopic positioning in an ipsilateral oblique and cranial view for optimal visualization. The lumbar neural foramina can be approached inferior to the transverse process. Most of the arteries lie in the more anterior part of the foramen.

The ligamentum flavum in the cervical epidural space is inconsistent so the loss of resistance technique is less reliable compared to the lumbar levels especially superior to the sixth cervical level. A large epidural venous plexus is present at the cervico-thoracic junction, but cervical epidural procedures are performed at this level to avoid the risk of dural puncture at a mid-cervical spinal cord level. Cervical neural foramina increase in size with cervical flexion and decrease with extension. This feature allows flexion –rotation exercises to open the foramina in case of fluid loculation in the epidural space after an injection. This can decompress the epidural space and resolve symptoms of pain after a cervical injection.

Indications

The Epidural lysis of adhesions procedure is indicated for radicular pain syndromes that have not responded to conservative care including medications and physical therapy. A comprehensive list of established indications from previous studies is below:

- Chronic low back pain of 3–6 month duration and failed conservative treatment options
- Back pain with or without radiculopathy
- Radiating Lower Extremity Pain with provocative straight leg raising test
- Failed back surgery syndrome
- Radiographic evidence of pathology such as spondylosis
- Spinal Stenosis
- Osteophyte and radiculopathy
- Lateral recess stenosis and radiculopathy
- Disc herniation and radiculopathy
- Spondylosis and radiculopathy, (MRI, CT)
- Radiculopathy due to Epidural Fibrosis (on enhanced MRI)
- Disco genic back pain and back spasm
- Faded stimulation from Neuromodulator (SCS, spinal narcotics)
- 18 years of age or older (no specific contraindication by age)

Contraindications include:

- Spinal instability
- Spinal Cord Syrinx
- Local infection, unresolved spinal infection
- Chronic infection
- History of gastrointestinal bleeding or ulcers
- Substance use disorder and/or uncontrolled major depression or psychiatric disorders
- Arachnoiditis
- Arterio-venous malformation
- History of adverse reaction to local anesthetic, steroids, contrast or other injected medications.
- Uncontrolled or acute medical illnesses including coagulopathy, renal insufficiency, chronic liver dysfunction, progressive neurological deficit, urinary and sphincter dysfunction, infection, increased intracranial pressure, spinal fluid leak, pseudo tumor cerebri, intracranial tumors, unstable angina and severe chronic obstructive pulmonary disease.
- The use of anti-platelet medications or anticoagulants e.g.: aspirin, Plavix, NSAID's, gingko, ginseng, Vitamin E, garlic, Coumadin, Fish Oil, Mobic, etc. (Laboratory measurements for bleeding and clotting to be in the normal range following discontinuation for appropriate duration.)
- Pregnant or lactating women

In addition to these indications, several new indications are being treated successfully with lysis. Annular tears are associated with back pain but common treatments

are not particularly effective [15]. Annular tears and high intensity zone lesions are another indication for the lysis procedure. Endplate edema and back pain is also responsive to the lysis procedure. Neuromodulation "fade" due to scarring can be reversed by lysis of adhesions and prolong the life of the stimulator by several years. Post-surgical foot drop and bladder urgency secondary to sacral nerve root scarring may respond to S1 or lumbar transforaminal lysis depending on the site localized by the dural tug maneuver.

Techniques

The goal of the procedure is to inject therapeutic medications at the site of pathology. The site of pathology is determined by history, physical examination and imaging studies, including epidurography. Epidurography is performed by injecting radiopaque contrast into the epidural space to demonstrate areas of epidural adhesions that do not fill with contrast normally. A specialized catheter is used to penetrate the adhesion area of the epidural space so that injections will open the space and allow delivery of therapeutic medications. Conventional injections such as single shot interlaminar and transforaminal epidural injections may fail to relief pain because they injected medication never reaches the target of pain. The procedure involves multiple steps and injections. Myelogram grade contrast is used to localize epidural adhesions. Following catheter placement into the scar area, additional contrast is used to open the epidural space and neural foramina. Following this, local anesthetic is injected to provide analgesia and to test for subdural blockade. Hyaluronidase is usually injected to facilitate spreading of injected medications. Hypertonic saline is injected to reduce swelling and provide long term analgesia. The rationale for the procedure is to provide longer term analgesia compared to conventional single shot steroid injections by injecting steroid and other therapeutic medications at specific pathology as opposed to injecting into the posterior epidural space or neural foramen and relying on favorable spread to result in the medication reaching a therapeutic location.

Lysis of Epidural Adhesions via Caudal Approach

The sacral hiatus allows direct access to the sacral epidural space (Fig. 12.1). The Rx™Coude® epidural needle has a specialized tip to allow for catheter repositioning without shearing the catheter. The RX™ Coude® allows for multiple passes of the catheter to achieve the optimal tip placement. The RX™ Coude® tip is designed to reduce the chance of catheter shearing. The opening of the needle tip is completely round allowing free passage of the catheter, unlike the oval tipped Touhy needles or conventional spinal cord stimulator needles. The needle tip and catheter bend must be positioned in the same direction. The catheter used has a wire coil construction with a special coating to prevent kinking and vascular penetration. The Racz® catheters are radiopaque and can be steered in the epidural space to the level

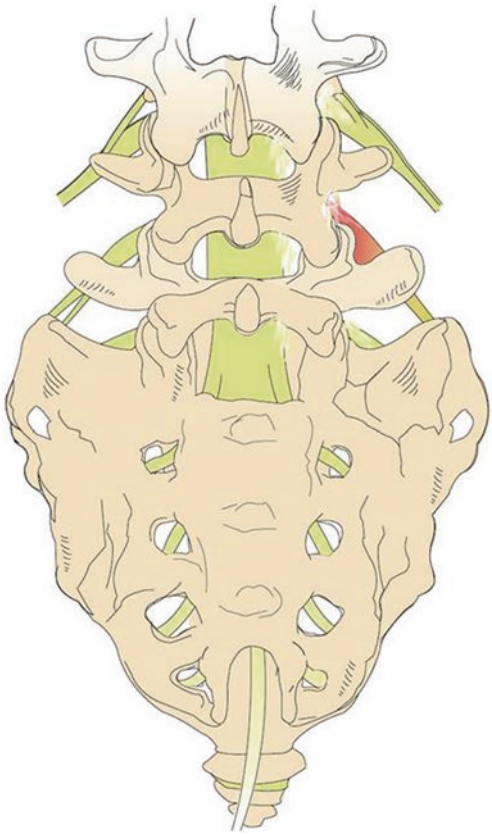


Fig. 12.1 Posterior lumbosacral anatomy with sacral hiatus

and side of pathology in the ventrolateral epidural space. Site-specific injections are far superior to blind non-specific delivery of medications. The ventral lateral epidural space is unique in that fluid injected under pressure follows the path of least resistance and will spread into the scarred perineural space and "free up" the nerve roots. When the target cannot be reached, it is clearly visible on the lateral fluoroscopic views. Subsequent treatment using the transforaminal approach for catheter placement and lysis can further reduce back pain and/or radiculopathy from involvement of the structures that are the most richly innervated by the sinu-vertebral nerve system. For one-day lysis procedures, the skin entry point for needle access may be close to the sacral hiatus in the midline for easier placement. For the three-day technique and repeat injections or continuous infusion therapies, the skin entry is 2 inches inferior and 1-inch lateral of the gluteal cleft on the contralateral side to the pain. This second approach places the skin entry away from the sacral hiatus in order to reduce the chance of infection and allows easier catheter placement on the affected side. Palpation with the index finger is used to locate the sacral horns (cornua) of the sacral hiatus to locate the entry point for the 15ga. or 16 ga. RX-2™ Coude® needle (Fig. 12.2). For a 1 day procedure, the skin entry point may be just

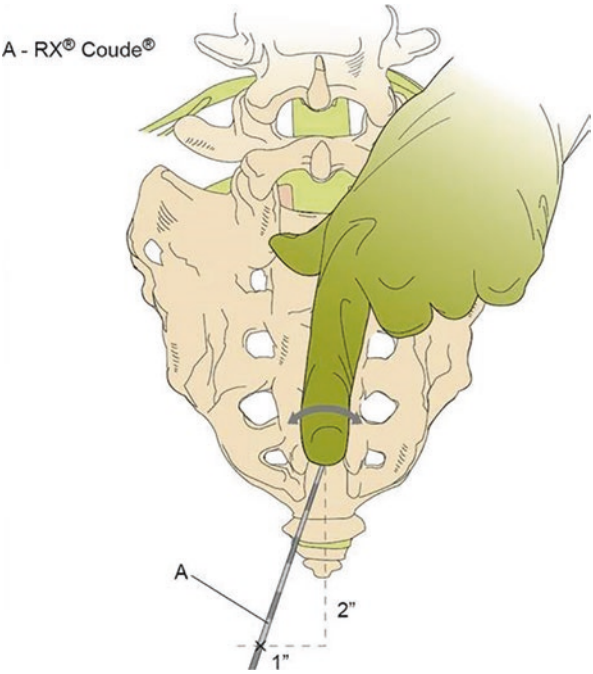


Fig. 12.2 The sacral hiatus is identified by palpation and a skin puncture site is contemplated approximately 2 inches inferior and 1 inch lateral to the hiatus. Skin puncture site is labeled as an X, inferior to the sacral hiatus to allow entry of the RX-Coude needle. The puncture is made off the midline on the side contralateral to the pain in order to direct the catheter to the affected side

inferior to the sacral hiatus. However, for a 3 day procedure, the entry point is 2″ below the sacral hiatus and 1″ from midline (gluteal cleft). This reduces infection risk by this tunneling technique. The anterior-posterior C-arm position may be used to confirm the location of the sacral hiatus (Fig. 12.3). The finger is rolled medially and laterally to confirm the location and the finger is maintained at the sacral hiatus as a guide. A lateral fluoroscopic view should be obtained after skin penetration to avoid needle advancement too anteriorly into the bowel. (Fig. 12.4) After confirming epidural placement, rotate the needle 90° degrees towards the target area (Fig. 12.5a, b). Anterior-posterior and lateral view and injection of contrast confirms good needle placement. The lateral fluoroscopic image should be used to check for circumferential contrast spread. Needle tip placement should be below the S3 neural foramen to avoid the thecal sac. Midline catheter placement in the sacrum can result in subdural placement by penetrating the inferior dural sac at the S3 level. This is avoided by catheter placement off the midline when using the caudal approach. It is important to make a bend at the Racz® bend marker on the catheter 1 inch (second marker on the catheter) proximal to the catheter tip at a 15°–20° angle for optimum steering (Fig. 12.6). With the XL tip catheter the stylet needs to be close to the tip for enhanced steering. If the bend is too short, the catheter tends to buckle. If the bend is too long, it is much harder to steer. The C-Arm is rotated to the anterior-posterior position (Fig. 12.7). In order to direct the catheter to the ventral lateral epidural space, the catheter advancement should be slow, keeping the catheter near the midline and the point on the bend medial to the tip. This allows the catheter to

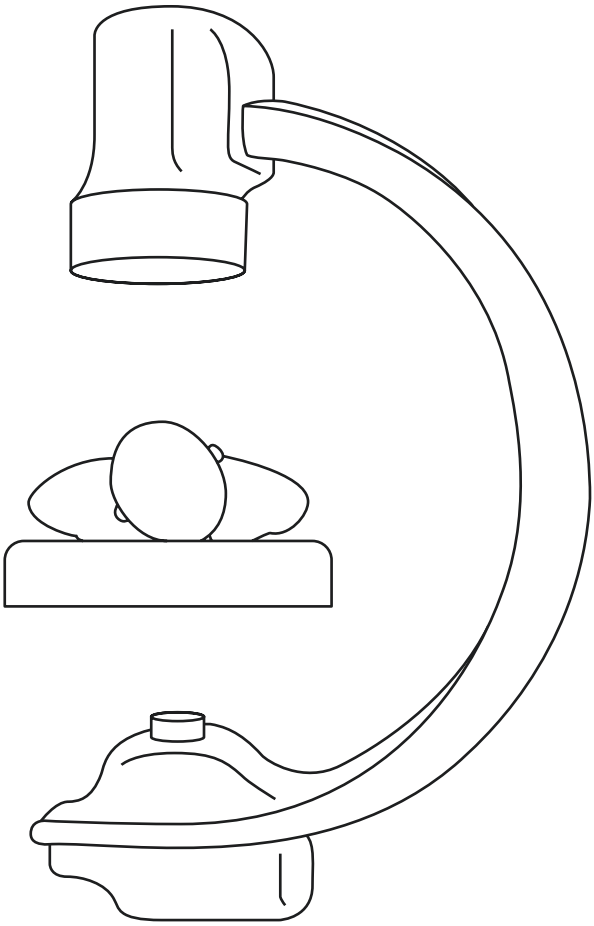


Fig. 12.3 The C-arm position is anterior-posterior to maintain needle position superficial to bony sacrum and coccyx

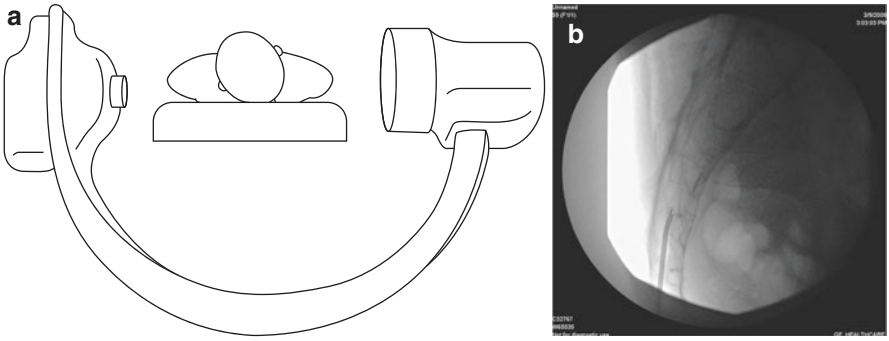


Fig. 12.4 (**a**) The C-arm is rotated to the lateral position to confirm placement. (**b**) The lateral image confirms placement

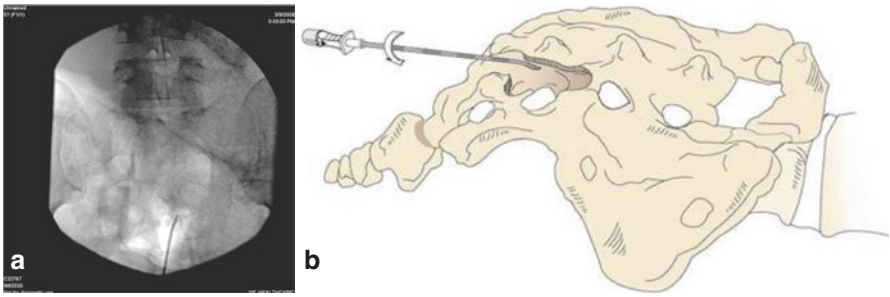


Fig. 12.5 (**a**) The RX- Coude needle is placed into the caudal canal and rotated toward the target. (**b**) Cutaway diagram of RX-Coude placement. The RX-Coude is rotated toward the painful side to facilitate catheter placement at the target area

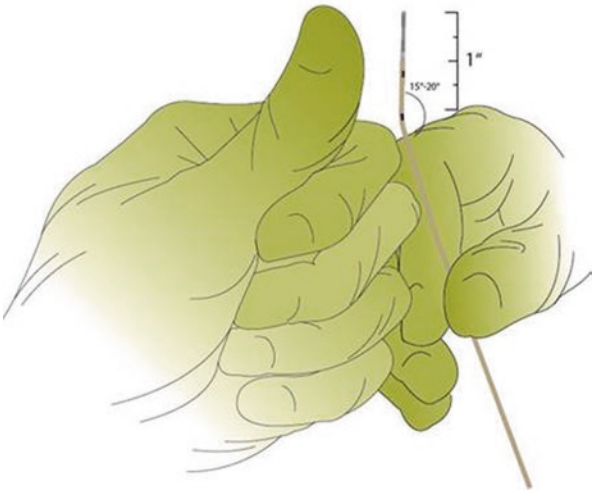


Fig. 12.6 A 15–20 degree bend is made at the insertion end of the catheter to facilitate steering

be steered anteriorly in the epidural space (Fig. 12.8a, b). The tip of the RX-Coude needle should be oriented toward the target (Fig. 12.9). The technique is described in more detail elsewhere [16]. Cases with long-term outcomes are also reported [17]. Recurrent scaring in the same area has been observed 22 years after an initial lysis procedure. Also, pain can occur months to years after a lysis procedure on the opposite side. The technique for securing the catheter is shown (Fig. 12.10).

Advanced Catheter Fastening Technique Steps

1. Make a full twist in the catheter to form a loop.
2. Place loop over the neck of the connector.
3. Pull catheter until securely around the connector body.
4. Use tape to secure the device.
5. Attach a bacterial 0.2-micron filter to maintain sterility.

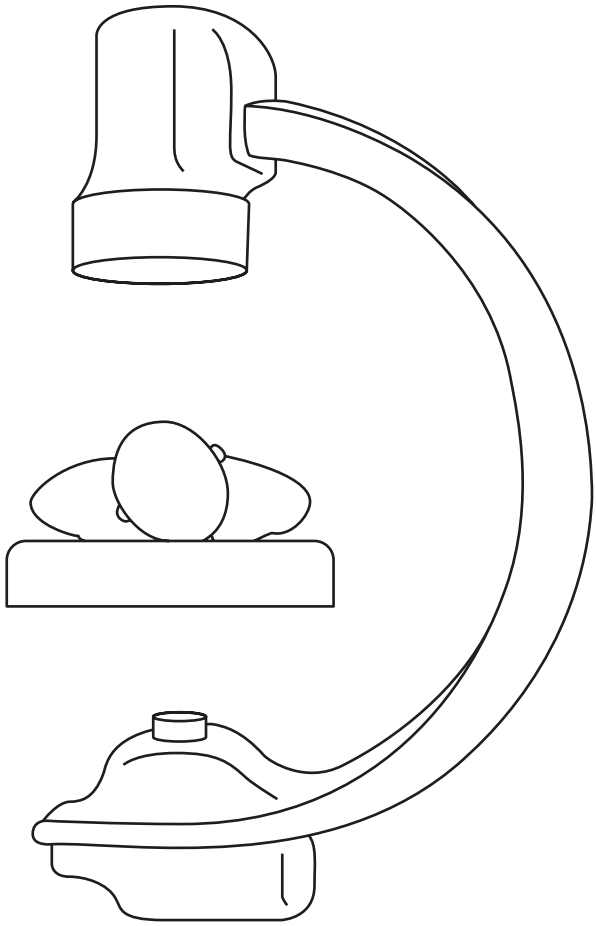


Fig. 12.7 The C-arm is rotated to anterior-posterior position

The connector is attached to the catheter for connection to a syringe (Fig. 12.11). The C-Arm is rotated to the lateral view (Fig. 12.12) Contrast injection is performed, showing contrast in the ventrolateral epidural space (Fig. 12.13). Subsequent injections result in the lysis of epidural adhesions (Fig. 12.14) Anterior-posterior shows contrast in the epidural space and contrast that has flowed out of the epidural space through the neural foramen (Fig. 12.15). Epimed's Stingray™ Connector design allows for a fastening technique that changes pulling force direction to prevent disconnects. The Stingray™ when compared to four other connectors for grip and strength was found to be the best; however, for repeat injections or prolonged use the following additional measures further enhance safety [10]. Using this technique, the force to separate the catheter is more than doubled. Bacterial filters are recommended in all instances when more than one injection is used or the catheter is left in place for prolonged period. Anytime there is a disconnection of the catheter and the connector the system should be removed from the patient. This is an essential

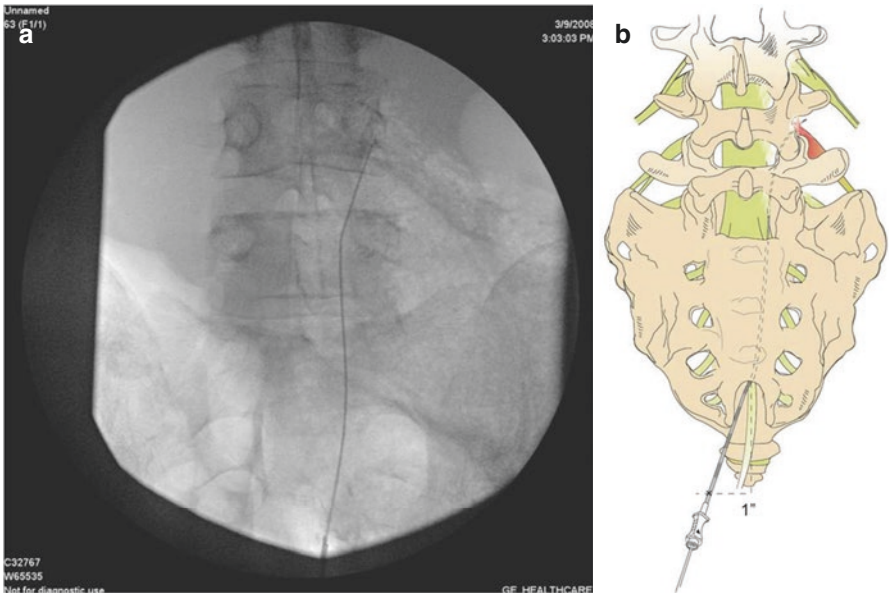


Fig. 12.8 (**a**) The catheter is passed to the target, L4 on the right, in this case. (**b**) The catheter is passed to the target, L4 on the right, in this case

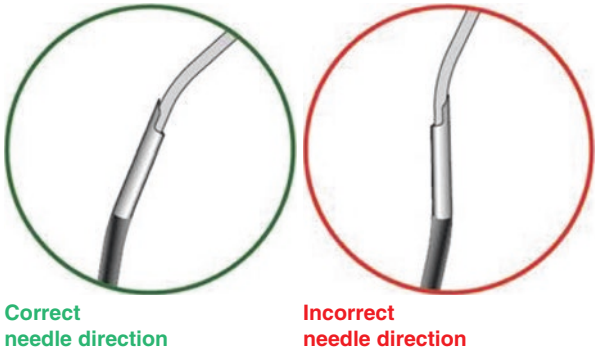


Fig. 12.9 The tip of the RX-Coude needle should be oriented toward the target to make placement easier. This also prevents shearing if the catheter needs to be withdrawn and redirected

precaution to prevent infection. After the catheter tip is placed in the proper location (ventral/lateral), attach Stingray™ Connector to inject the target site. Always use bacterial filter. Physicians use this technique during a three-day lysis series or post procedure injection of hypertonic saline in the recovery room for the one-day procedure. It is also useful when prolonged or postoperative infusion is utilized.

In order to simplify the procedure, several tips are offered:

1. Slow down.
2. Go near mid-sacral canal
3. Make a 15–20 degree bend at the 1-inch Racz® bend marker and steer only when the catheter is being advanced.



Fig. 12.10 Advanced catheter fastening technique steps. (1) Make a full twist in the catheter to form a loop. (2) Place loop over the neck of the connector. (3) Pull catheter until securely around the connector body. (4) Use tape to secure the device. (5) Attach a bacterial 0.2-micron filter to maintain sterility

Table 12.1 summarizes medications and doses for procedures (Table 12.2). Discharge criteria include ambulation and voiding. If patients have difficulty, they should be observed until recovery is complete. Spine surgery patients may have dural tears and need to be monitored for subdural blocks. Also, patients with dense scar may develop recurrent scarring within 3 months and lysis can be repeated in 1 month to prevent this from occurring. A variation of the series of 3 injections is the 1 day, single injection period, and technique.

The medications used are outlined below.

1 Day Lumbar Lysis

1. Diagnostic: 5–10 mL OMNIPAQUE™240* – outline filling defect and place catheter to target site
2. To show runoff and absence of loculation, contrast 4–5 mL OMNIPAQUE™ 240* injected through the catheter
3. 2–3 mL OMNIPAQUE™240* through catheter for verification of enzyme effectiveness
4. Spreading Factor: Hylenex® 150–300 units (human recombinant) diluted in 10 mL of preservative-free saline
5. Steroid Injection: 4 mg dexamethasone or 40 mg triamcinolone
6. Local Anesthetic: 10 mL 0.2% ropivacaine or 10 mL of 0.25% bupivacaine. Patients seem to respond to bupivacaine better than ropivacaine.
7. Depending on the physician's lysis technique, wait 20–30 min. Evaluate for motor block with a voluntary straight leg raise. If no motor block is present, with the patient's painful side down, inject 8–10 mL of 10% hypertonic saline over 20–30 minutes. If the patient experiences pain, inject 2–3 mL of local anesthetic.

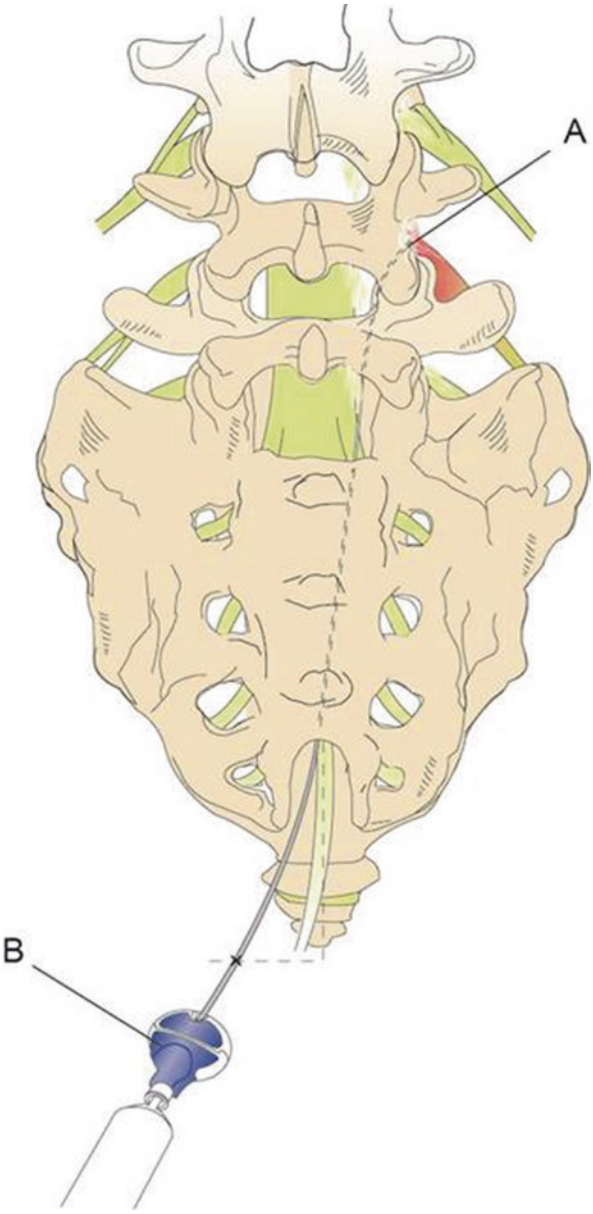


Fig. 12.11 The connector is attached to the catheter for a syringe. (**A**) Catheter tip position. (**B**) Stingray connector

After injections have been completed, the patient's motor function should be evaluated with by testing hip flexion with the knee extended. If there is a motor block, stop the procedure. Be sure to attach bacterial filter to the Stingray™ connector to guarantee sterility of the catheter. Wait 20–30 minutes. Place the patient with their painful side in the gravity–dependent position. Flush catheter with 2–3 ml normal saline solution. Once the nerve root is freed, the "neural flossing" exercises are started. Patient education about the exercises is an important component. Very commonly, patients also have facet joint arthropathy that requires additional treatment.

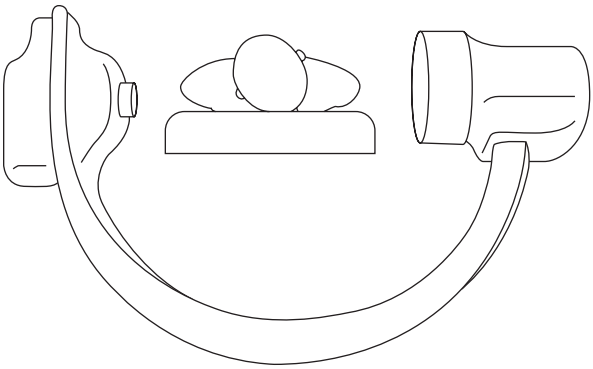


Fig. 12.12 The C-Arm is rotated to the lateral view

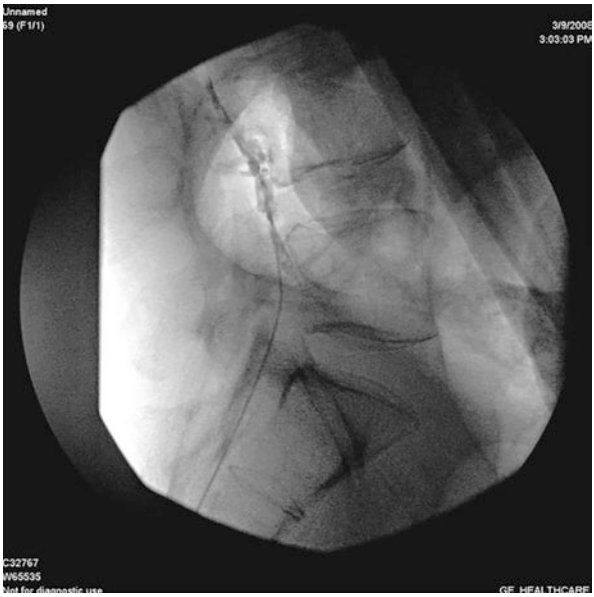


Fig. 12.13 Contrast injection shows placement in the ventrolateral epidural space

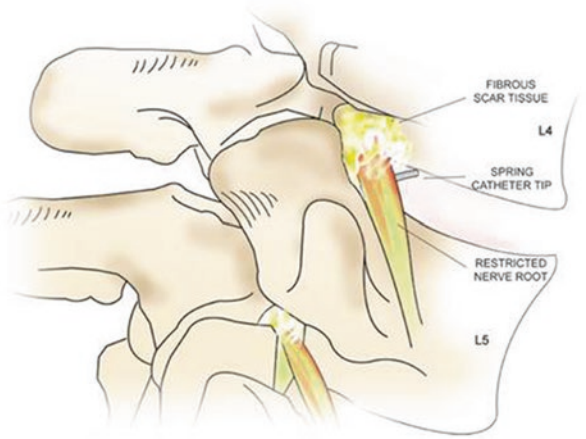


Fig. 12.14 Schematic of the lysis procedure. The catheter is placed in the ventrolateral epidural space and fluid is injected to open scar around the nerve root

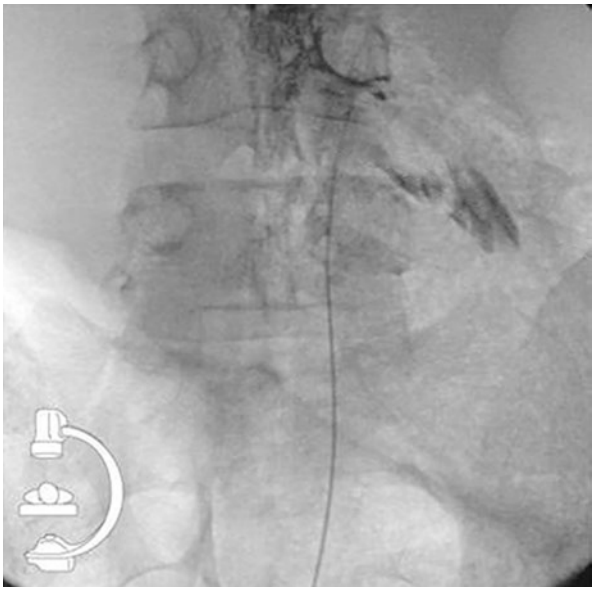


Fig. 12.15 Anterior-posterior image showing contrast in the epidural space and contrast that has flowed out of the epidural space through the neural foramen

Several tips are very useful. The catheter should be advanced with slow movements and rotation should be performed while advancing the catheter, not by twirling the catheter in place.

In order to make the catheter easier to steer for cervical and thoracic procedures, a Racz® bend is made at the 1/2 inch mark from the tip. For caudal procedures, the bend is made at the 1-inch mark. When the catheter is in the ventrolateral epidural space, the catheter will bend laterally. Most epidural scar formation occurs in the ventral and lateral recess. Fluid injection under pressure opens up the perineural space. The process is "compartmental filling"- where the injected fluid flows along the path of least resistance in the scar and then over flows into the adjoining "compartment." After the initial injections have been completed, the introducer needle must be withdrawn from the patient to allow for repeat bolus injections. Before removing the introducer needle, it is important to stabilize the catheter's position (Fig. 12.16a–c).

The sequence of steps is:

A. Stabilize catheter to prevent catheter tip displacement.
B. Withdraw introducer needle while holding catheter in place.
C. Remove introducer needle.

Once the introducer needle has been carefully extracted from the patient's body, secure the catheter body at the exit site. At this point, the introducer needle should be withdrawn completely from the catheter. Bacterial filters are recommended in all instances when more than one time injection is used or the catheter is left in place for prolonged period. Anytime there is a disconnection of the catheter and the connector the system should be removed from the patient. This is an essential precaution to prevent infection. The importance of securing an intact catheter cannot be overemphasized. The technique that has been developed is described next.

Table 12.1 Pain free hypertonic saline volumes and pharmacological adjustments

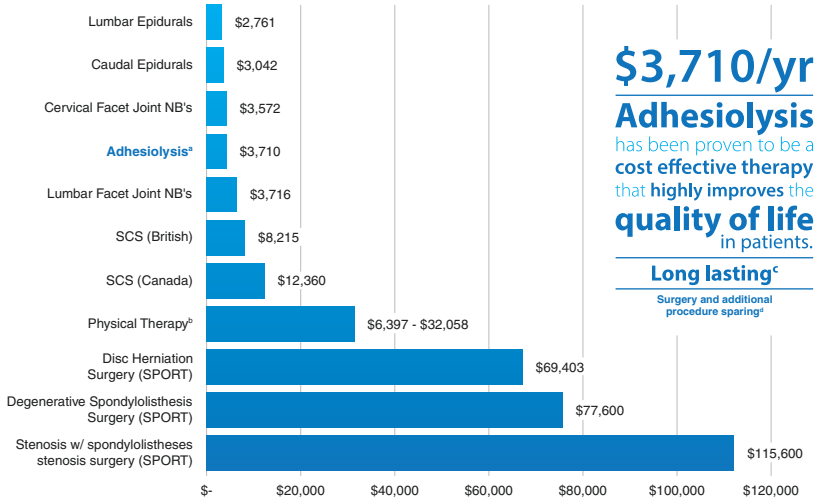
1st series injections	2nd series injections	3rd series injections
Caudal catheter (1) 10 mL caudal epidural omnipaque 240 contrast (2) 10 mL PF Normal Saline w/150 units Hyaluronidase (Hylenex – Human Recombinant) or 1500 units Hyaluronidase animal extract (3) 0.25% Bupivacaine with 40 mg Triamcinolone under fluoroscopic A/P and lateral observation. Lateral view [essential to rule out intravenous or subdural injection or spread through a partial surgical tear]. Observe patient for 20–30 minutes for delayed onset of motor block that would indicate subdural placement. [a]**Abandon procedure if motor block develops** Subdural motor block usually develops in 14–15 minutes later. Shorter Observations Not Recommended! Start Flexion-rotation exercises **New** – 20–30 minutes later. Lidocaine 1% 1.5 mL injection, followed by 2–3 minutes later 10 mL of 10% sodium chloride in 0.6% lidocaine, injected in 1mL increments fairly rapidly, over 3–5 minutes. The small volume, prehypertonic lidocaine, seem to cover the periphery of the injection site, therefore no pain from the hypertonic. Remember to flush at the end with 1mL PF saline. Frequent check for motor function – post operative observation requirements, maybe 2–4 hours. **Transforaminal catheter** Volumes are reduced to 5 mL. [Local and hypertonic]	**Caudal catheter** Usually same day, 4–6 hours later. (1) 10 mL of Bupivacaine 0.125% No Motor Block Wait 20–30 minutes If no motor block (2) 1.5 mL – 1% Lidocaine Wait 2–3 minutes (3) Inject 10 mL – (6 mL 1% Lidocaine, 4 mL of 23.4% NaCl) = 10% sodium chloride in 0.6% lidocaine, fairly rapidly, over 3–5 minutes Flush after observing patient for 30 minutes. **Transforaminal catheter** Volumes are reduced to 5 mL. [Local and hypertonic] Prehypertonic lidocaine: 1% 1 mL If there is no motor block and patient is able to ambulate, patient can go home in 45–60 minutes. Cost Saving	**Caudal catheter** Usually same day, 4–6 hours later. (1) 10 mL of Bupivacaine 0.125% No Motor Block Wait 20–30 minutes If no motor block (2) 1.5 mL – 1% Lidocaine Wait 2–3 minutes (3) Inject 10 mL – (6 mL 1% Lidocaine, 4 mL of 23.4% NaCl) = 10% sodium chloride in 0.6% lidocaine, fairly rapidly, over 3–5 minutes Flush after observing patient for 30 minutes. **Transforaminal catheter** Volumes are reduced to 5 mL. [Local and hypertonic] Prehypertonic lidocaine: 1% 1 mL If there is no motor block and patient is able to ambulate, patient can go home in 45–60 minutes. Cost Saving

Staff responsible for observation should be ACLS certifed

Techniques of Neurolysis - Chapter 7: Hypertonic Saline and Corticosteroid Injected Epidurally for Pain Control. (Racz, Heavner, Singleton and Caroline). Springer 1989

[a] Pain Free Hypertonic Volumes by Dr. Gabor B. Racz

Table 12.2 Cost effective per Quality-Adjusted Life Year (QALY)



Manchikanti et al. [42]

[a] Gerdesmeyer et al. [36]. This study strongly recommends Lysis be performed prior to spine surgery

[b] Veihelmann et al. [23]. Convincing superiority of Epidural Neuroplasty over Physical Therapy

[c] Racz and Noe [22]. Print

[d] Moon et al. [43]

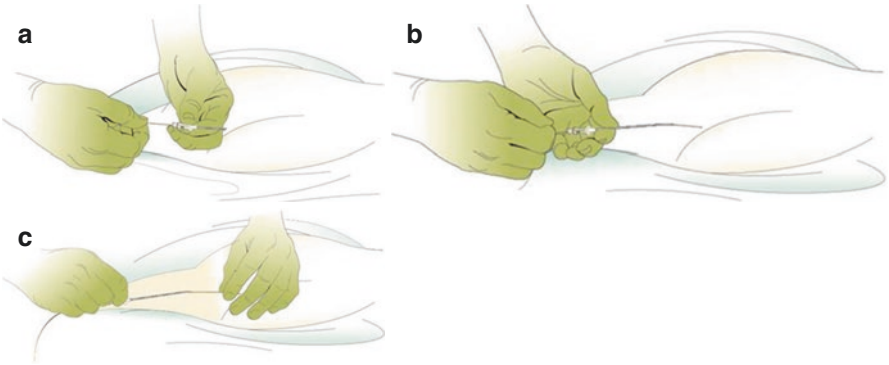


Fig. 12.16 A Needle removal without dislodging the catheter is critical. The sequence of steps is: (**a**) Stabilize catheter to prevent catheter tip displacement. (**b**) Withdraw introducer needle while holding catheter in place. (**c**) Remove introducer needle

Catheter Tape Down Technique

Place suture and tie loose loop. (Fig. 12.17)

Wrap around catheter two times and tie surgical knot. (Fig. 12.18)

Apply antibiotic ointment around skin entry and place two-split 2 × 2" gauze to keep antibiotic in place. (Fig. 12.19)

Apply adhesive i.e. tincture of benzoin around gauze. (Fig. 12.20)

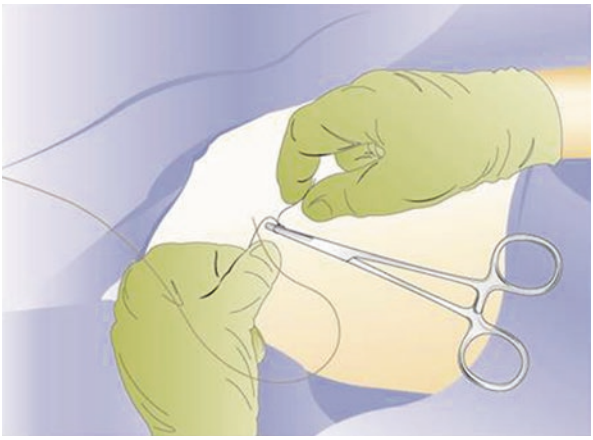


Fig. 12.17 Catheter tape down technique. Place suture and tie loose loop

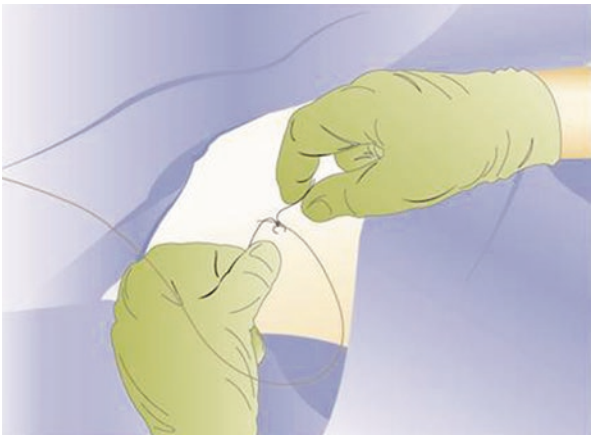


Fig. 12.18 Catheter tape down technique. Wrap around catheter two times and tie surgical knot



Fig. 12.19 Catheter tape down technique. Apply antibiotic ointment around skin entry and place two-split 2 × 2” gauze to keep antibiotic in place

Place one loop on catheter and transparent dressing i.e. opsite (Fig. 12.21)
Connect bacterial filter and four pieces of sweat resistant hypofix tape (Fig. 12.22).

After completion of the procedure cut suture and gently remove the catheter. If resistance is detected, don not forcibly pull on the catheter to avoid catheter shearing. Reposition the patient and repeat attempting to remove the catheter. Simultaneously pushing and twisting the catheter allows removal (Fig. 12.23). The use of wide-open R-X™ Coude® needles reduce the incidence of shearing. After injections have been completed, the patient's motor function should be evaluated by testing hip flexion with the knee in extension. If there is a motor block, stop the procedure. Be sure to attach bacterial filter to the Stingray™ connector to reduce the infection risk. Observe for 20–30 minutes. Place the patient with their painful side in the gravity dependent position.



Fig. 12.20 Catheter tape down technique. Apply adhesive i.e. tincture of benzoin around gauze

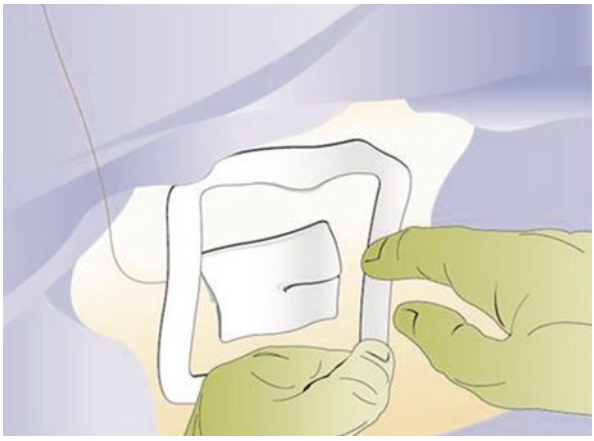


Fig. 12.21 Catheter tape down technique. Place one loop on catheter and transparent dressing i.e. opsite



Fig. 12.22 Catheter tape down technique. Connect bacterial filter and four pieces of sweat resistant hypofix tape

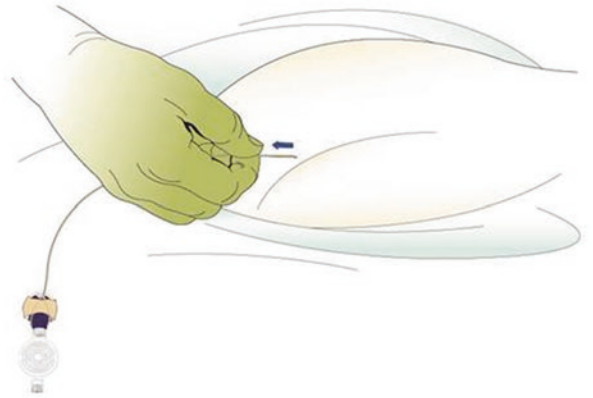


Fig. 12.23 Catheter removal with tip intact

Lumbar Transforaminal Catheter Placement

The R-X™ Coude® epidural needle and Racz® Catheter are used. Oblique lumbar anatomy is shown in Fig. 12.24. The C-Arm is rotated to the oblique position (Fig. 12.25). First, rotate the c-arm until the ipsilateral spinous process appears to move to the contralateral side of the spine. Second, adjust the cephalad caudal tilt until the superior pars is superimposed over the disc space. An approximately 30° oblique angle is used while viewing the (SAP) or the ear of the "Scottie Dog" as the needle target. Rotate the C-arm in the cephalad/caudal plane so that the ear of the "Scottie Dog" (SAP) is superimposed over the disk space. These two steps can be accomplished faster than the much lengthier process of trying to square the end plates. The R-X™ Coude® needle is steered to aim toward and come in bony contact with the tip of the superior pars (Fig. 12.26). Advance the RX™ Coude® needle until the tip comes in contact with the superior articular process (SAP). Once bony contact is made, rotate the RX™ Coude® needle 180° counter clockwise to orient

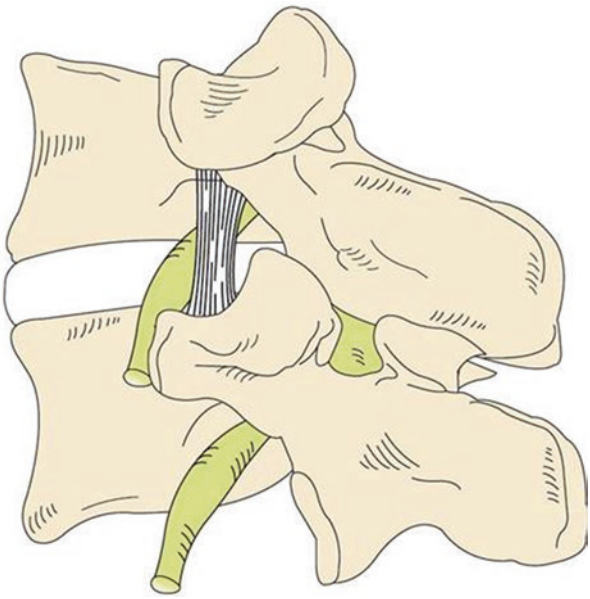


Fig. 12.24 Oblique lumbar spine anatomy

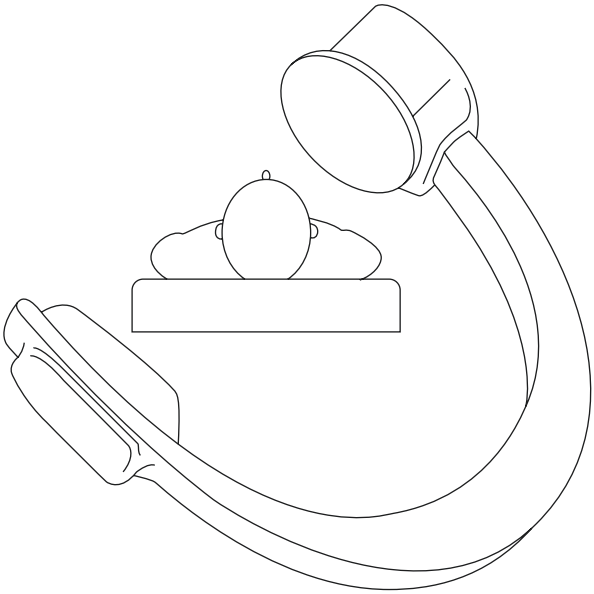


Fig. 12.25 C-arm is in oblique position

the needle laterally (Figs. 12.27 and 12.28). Rotate C-arm to give lateral view (Fig. 12.29). Using a lateral fluoroscopic view, the needle tip is navigated around the SAP (Fig. 12.30). Advance the needle to slide past the superior articular process. Change the C-arm to the lateral position and advance the RX™ Coude® needle until you feel it "pop" through the intertransverse ligament (Fig. 12.31). Rotate the

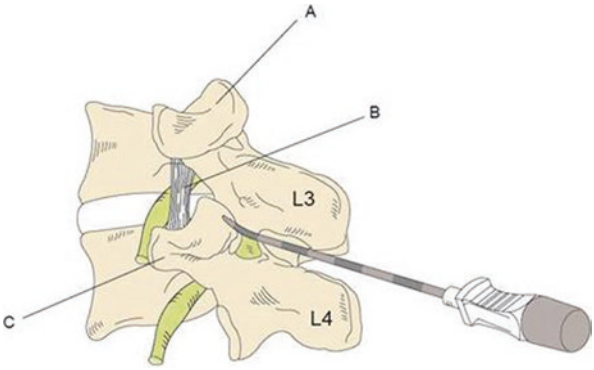


Fig. 12.26 The RX-Coude needle is advanced to make bony contact with the superior articular process posterior to the target foramen. (A) Superior articular process. (B) Intertransverse ligament. (C) Transverse process

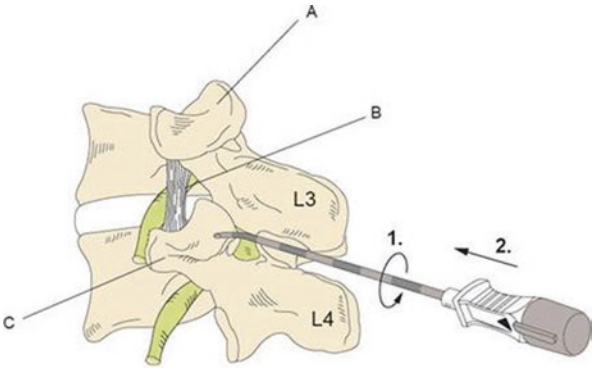


Fig. 12.27 The RX-Coude needle is rotated to a lateral orientation to allow navigation around the superior articular process. (A) Superior articular process. (B) Intertransverse ligament. (C) Transverse process

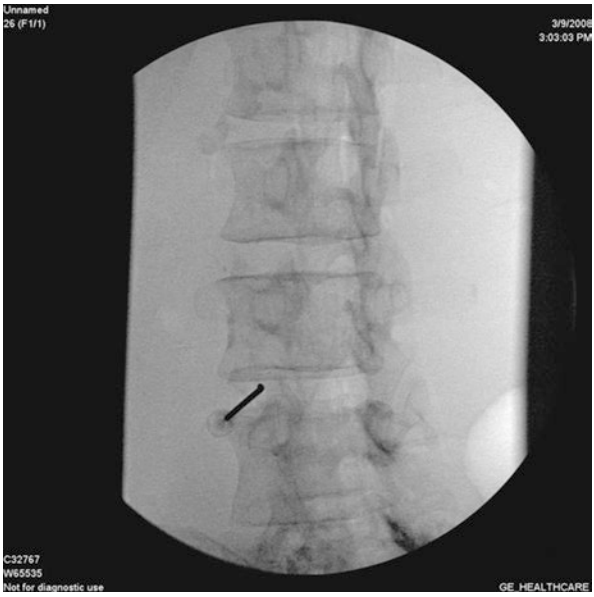


Fig. 12.28 The RX-Coude needle is rotated to a lateral orientation to allow navigation around the superior articular process

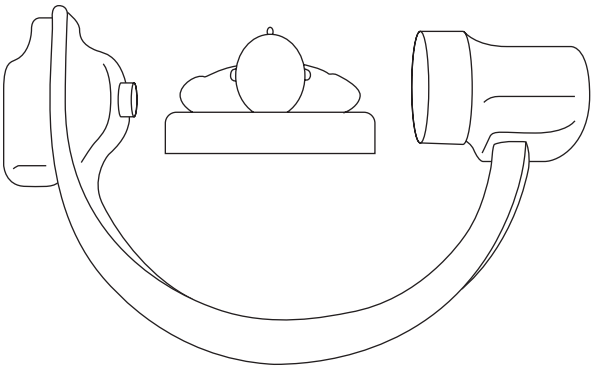


Fig. 12.29 The C-Arm is rotated to the lateral position



Fig. 12.30 The RX-Coude is advanced lateral and around the superior articular process

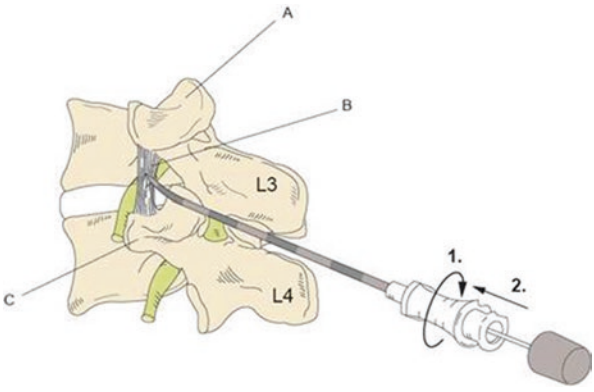


Fig. 12.31 The RX-Coude is advanced through the intertransverse ligament using the lateral view to avoid anterior placement. (A) Superior articular process. (B) Intertransverse ligament. (C) Transverse process

RX™ Coude® needle back 180° clockwise allowing the needle to curve back in the direction of the foramen (Fig. 12.32). The RX2™ Coude® needle has a second stylet that protrudes 1 mm beyond the needle tip to convert the needle to a blunt probe. Needle advancement is stopped at this point. The RX™ Coude® advancement should stop before the nerve root is reached. There should not be paresthesia or sharp nerve pain. When using the Versa-Kath® it is important to make a half inch 10°–15° bend in the catheter for optimum steering ability (Fig. 12.33). With the XL tip catheter, the stylet needs to be close to the tip for enhanced steering. If the bend is too short, the catheter tends to buckle. If the bend is too long, it is much harder to steer. The use of a wide-open RX™ Coude® needle reduces the incidence of



Fig. 12.32 The Rx-Coude needle is rotated to the medial orientation to facilitate catheter placement

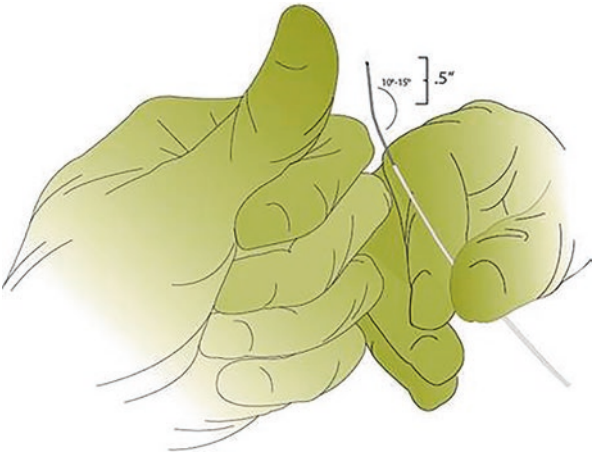


Fig. 12.33 A 10–15 degree bend is bend is made 1/2 inch from the catheter tip to facilitate steering

shearing. A Racz® catheter will readily be passed in the ventral epidural space to mid canal position (Figs. 12.34, 12.35, 12.36, and 12.37). A test dose of local anesthetic is administered and the patient is monitored for 15 minutes to ensure a subdural block is not produced. Using a sharp epidural needle for catheter placement may produce a puncture or laceration of the dural sleeve. Safely introduce a Racz® catheter to the ventral/lateral epidural space and halfway into the spinal canal. The optimal catheter placement should be halfway or less into the epidural space of the spinal canal, without crossing the midline. If perivenous counter spread (PVCS) is observed during injections, flexion and rotation exercises must be performed to reduce pressure by opening the neural foramina and allow fluid to escape through the foramina. Bacterial filters are recommended in all instances when more than one-time injection is used or the catheter is left in place for prolonged period. Anytime there is a disconnection of the catheter and the connector the system should

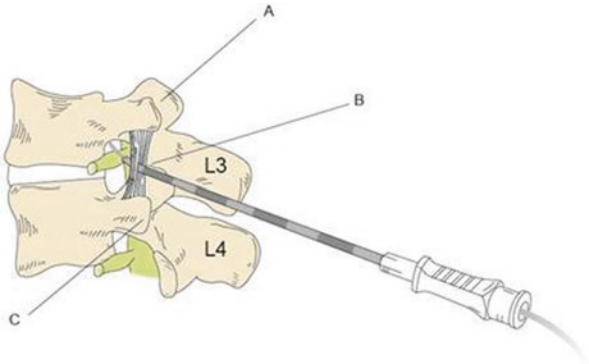


Fig. 12.34 Catheter placement, lateral anatomy. (A) Superior articular process. (B) Intertransverse ligament. (C) Transverse process



Fig. 12.35 Catheter placement in lateral image

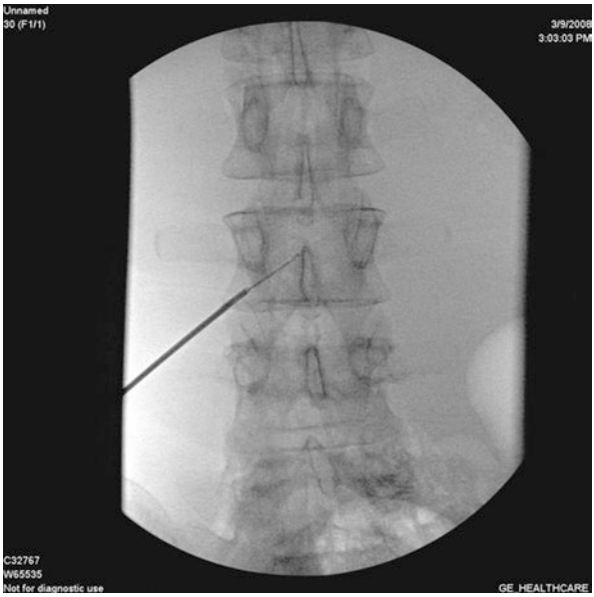


Fig. 12.36 Catheter placement in anterior-posterior image

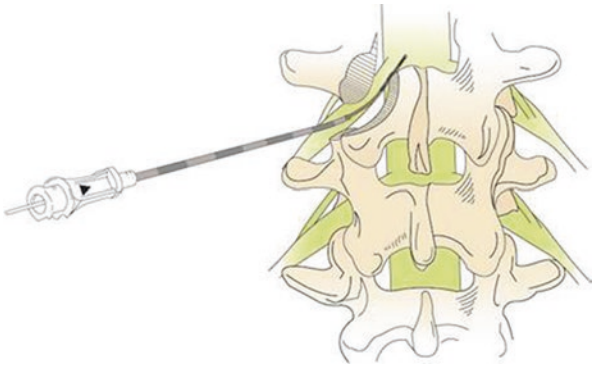


Fig. 12.37 Catheter placement in anterior-posterior cut away schematic drawing

be removed from the patient. This is an essential precaution to prevent infection. The catheter cannot be cleaned and reconnected if it becomes contaminated.

Steps to secure the catheter are:

1. Make a full twist in the catheter to form a loop.
2. Place loop over the neck of the connector.
3. Pull catheter until securely around the connector body.
4. Use tape to secure the device.
5. Attach a bacterial 0.2-micron filter to maintain sterility.

Epimed's Stingray™ Connector design allows for a fastening technique that changes pulling force direction to prevent disconnections. The Stingray™ Connector is designed to have more grip strength on the catheter. This is essential for repeat injections or prolonged use. Using this technique more than doubles the catheter

pull strength resistance. This technique is used for a three-day series of injections for lysis or post procedure injection of hypertonic saline in the recovery room for the one-day procedure. It is also useful when prolonged or postoperative infusion is utilized. Bacterial filters are recommended in all instances when more than one injection is used or the catheter is left in place for prolonged period. Anytime there is a disconnection of the catheter and the connector the system should be removed from the patient. This is an essential precaution to prevent infection. The catheter tape down technique, described previously, is used. After completion of the procedure cut the suture and gently remove the catheter. If resistance is detected, reposition the patient and repeat. At times, push and twist the catheter allows removal.

Sacral Foraminal Catheter Placement

The Scarring Triangle is a common location that requires lysis. A recent observation is that patients develop scaring in the L5-S1 dorsal root ganglion area that may be associated with ankle weakness or foot drop. This area is difficult to enter using a catheter. Teske described the space as the scaring triangle (Fig. 12.38) [18]. The space

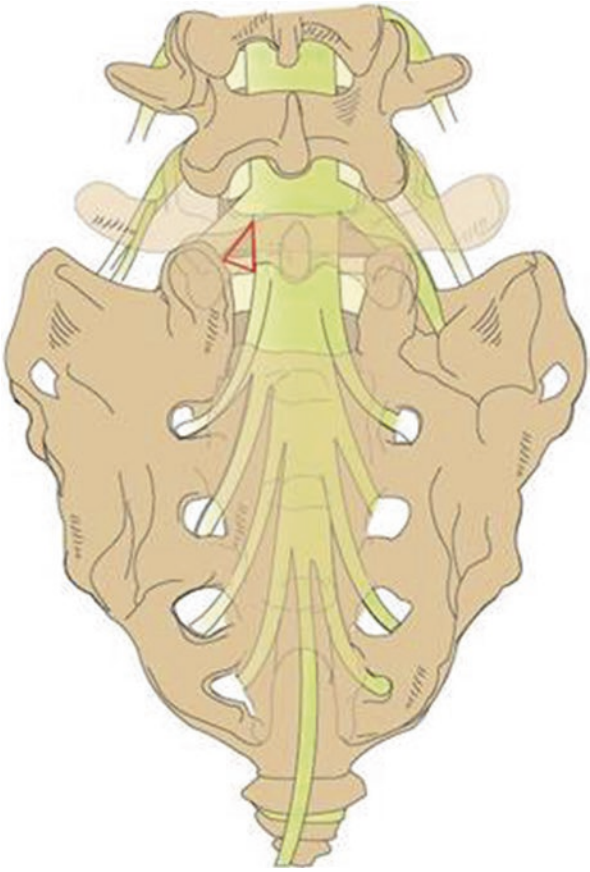


Fig. 12.38 Posterior lumbosacral anatomy, cutaway schematic showing the scarring triangle in red

measures 0.9–1.1 mL on each side. The boundaries are medial to the L5 nerve root, lateral to the S1 nerve root and the base of triangle is above the disc of L5- S1. This space is large enough to accept the average lose disc fragment. It tends to collect leaky disc material or the scars because of trauma and /or surgery. Due to the curvature of the sacrum and the formation of dense scarring, this area blocks catheters and scopes from entering into the ventral epidural space. Regular epidural catheters and scopes have not been able to enter this scarred area. Matsomuto described the use of a 21-gauge Versa-Kath® to successfully using a transforaminal approach [19]. Matsumoto realized that coming from the posterior S1 neural foramen with an 18 gauge RX-2™ Coudé® Needle and then rotating it, the curved tip allows ventral epidural projection of a 21 gauge VERSA-KATH®.2 The VERSA-KATH® is x-ray visible and steerable as long as rotation coincides with the advance of the catheter.

Place patient in prone position. The corresponding ventral and dorsal foramina are not at the same plane, but the posterior neural foramina are more proximal (Fig. 12.39). The 18-gauge needle has a curve near the tip, but one still needs a gentler angle to allow cephalad advancement of the catheter. The starting point will be the lateral side of the S2 posterior neural foramen (Fig. 12.40). Rotate the c-arm in a cephalad direction until the S1 ventral and dorsal neural foramina align. A slight lateral rotation helps separate the ventral and dorsal neural foramina. The needle entry point is from the S2 aiming towards the medial side of S1. Apply topical anesthesia and advance the needle through the skin. Curve the needle down to touch bone between the S1 and S2 on the sacrum (Figs. 12.41, 12.42, and 12.43). The Rx

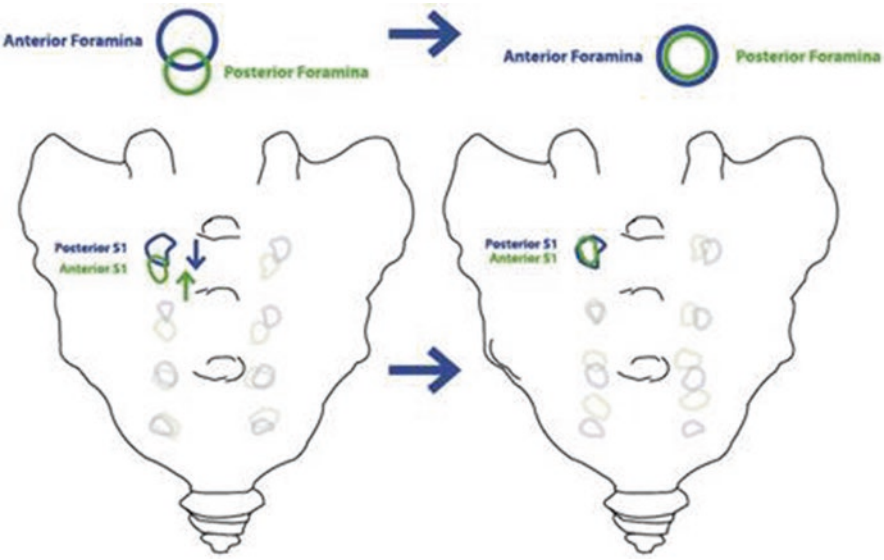


Fig. 12.39 The posterior and anterior foramina of the first sacral segment are not aligned in the anterior –posterior fluoroscopic image. In order to visualize the posterior foramen, it is helpful to rotate the C-arm in a cranial-caudal direction to facilitate foraminal entry

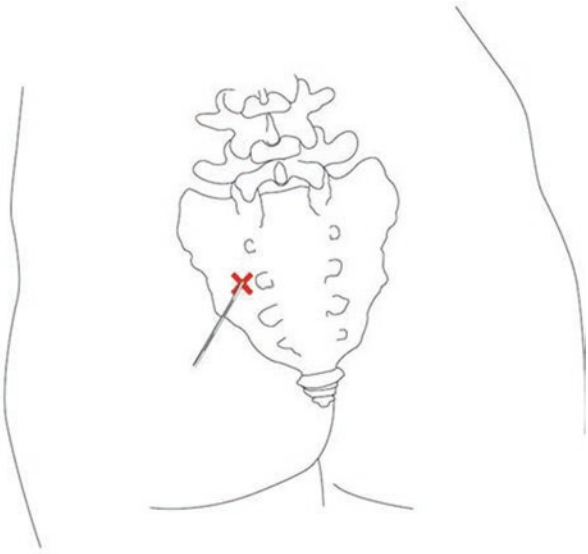


Fig. 12.40 The skin entry is inferior and lateral to the first sacral foramen at the level of the second sacral foramen

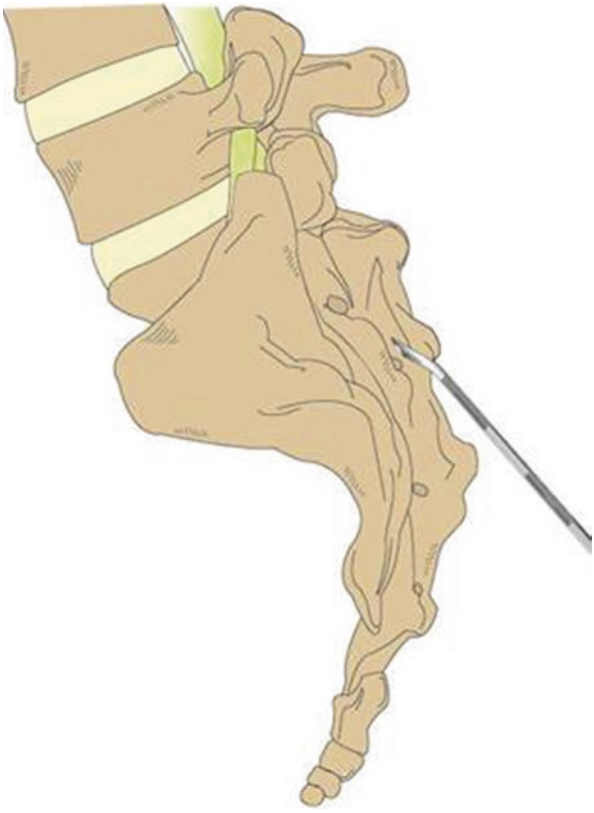


Fig. 12.41 The RX-Coude needle is advanced with the tip oriented in the anterior position to make bony contact between the first and second posterior sacral neural foramina

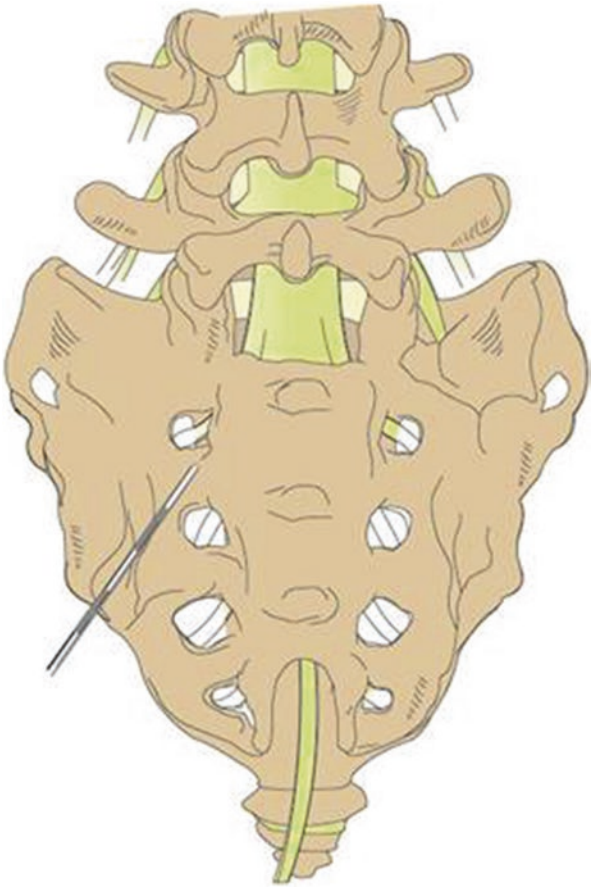


Fig. 12.42 The RX-Coude needle is advanced to make bony contact between the first and second posterior sacral neural foramina

Coude needle is rotated to orient the tip superiorly and the needle is advanced toward the first sacral foramen (Fig. 12.44a, b). Rotate the needle tip to navigate and become visible in the S1 fluoroscopic view (Fig. 12.45a, b).

Rotate the needle tip ventrally and elevate it while it is advancing to pop into the sacral canal (Fig. 12.46a, b) At this point, lateral fluoroscopic visualization will help advance the needle after rotation with the second stylet in place (Fig. 12.47). Rotate the c-arm into the anterior/posterior position with a cephalad tilt to avoid radiation exposure of the operator's hand. Only the needle tip is visible. With the stylet in place, advance the VERSA-KATH® within the sacral canal under fluoroscopic visualization. The catheter needs to cross the disc space and advance within the scar approximately near the top of the L5 neural foramen, not medial nor lateral in the imaginary triangle between L5 and S1 (Fig. 12.48a–d). It is possible to navigate the VERSA-KATH® by rotation during advancement.

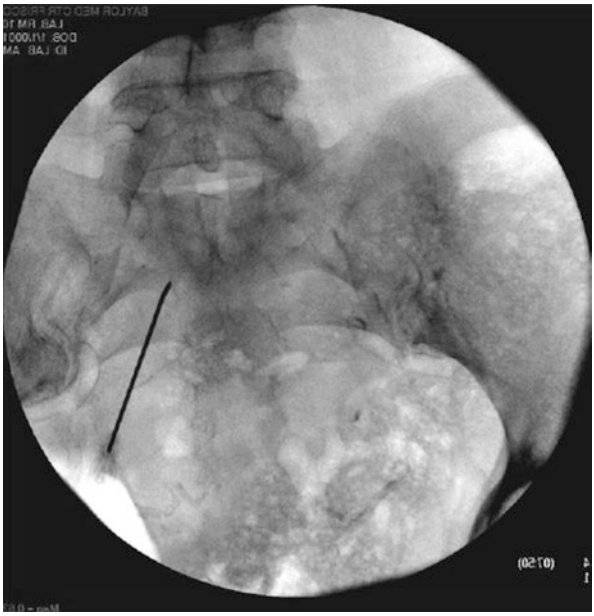


Fig. 12.43 The RX-Coude needle is advanced to make bony contact between the first and second posterior sacral neural foramina on anterior-posterior fluoroscopic imaging

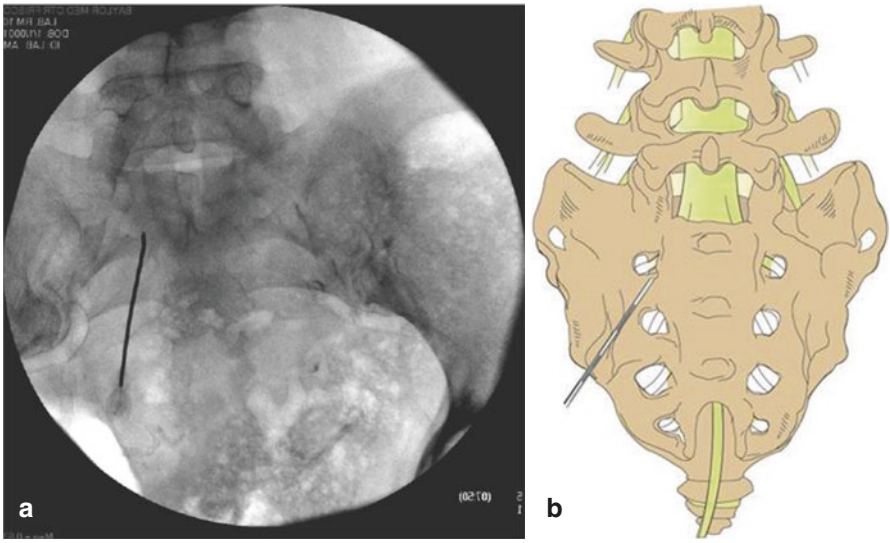


Fig. 12.44 (**a**) The Rx Coude needle is rotated to orient the tip superiorly and the needle is advanced toward the first sacral foramen. (**b**) The Rx Coude needle is rotated to orient the tip superiorly and the needle is advanced to the first sacral foramen

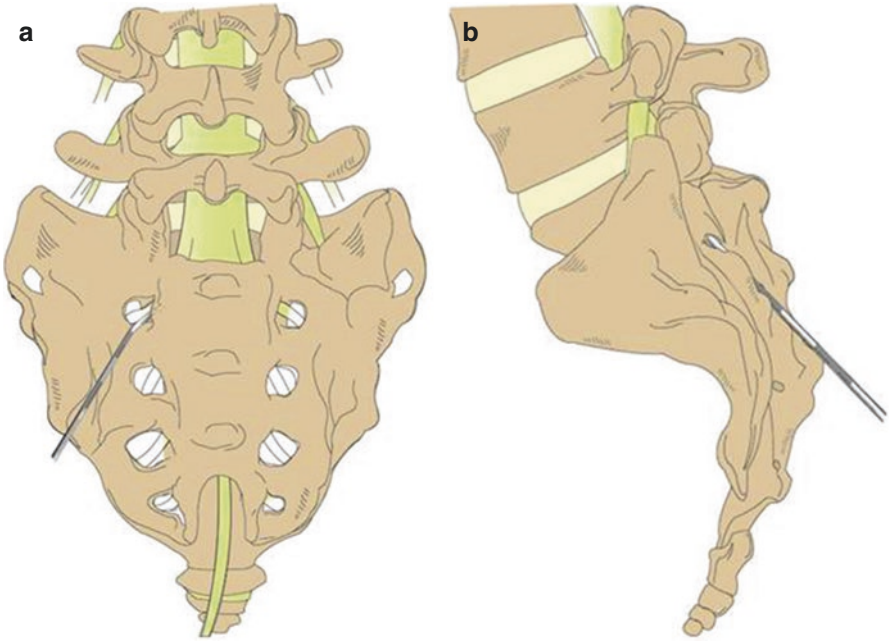


Fig. 12.45 (**a**) The RX-Coude needle is rotated to the medial tip orientation and advanced toward the medial foramen. (**b**) The RX-Coude needle is rotated to the medial tip orientation and advanced toward the medial foramen

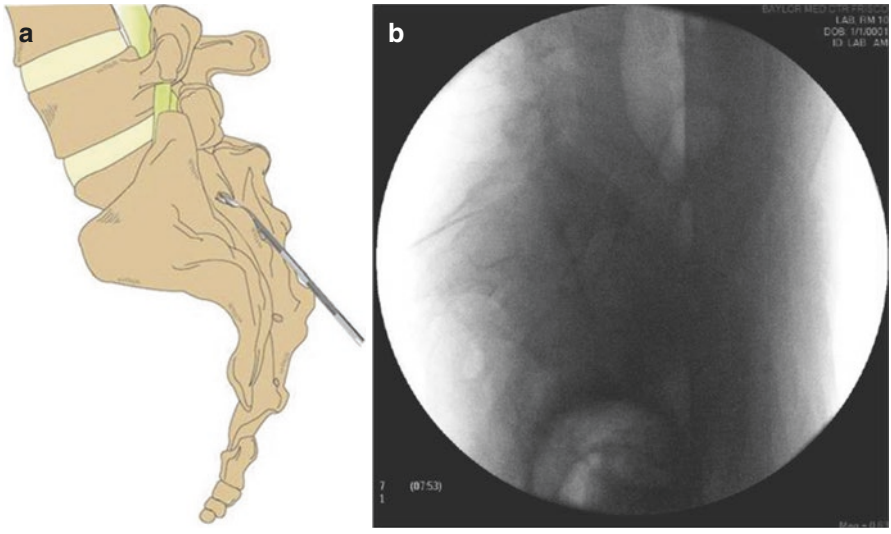


Fig. 12.46 (**a**) The Rx Coude needle is rotated to orient the tip anteriorly and the needle is advanced to the first sacral foramen. (**b**) The Rx Coude needle is rotated to orient the tip anteriorly to enter the posterior first sacral foramen

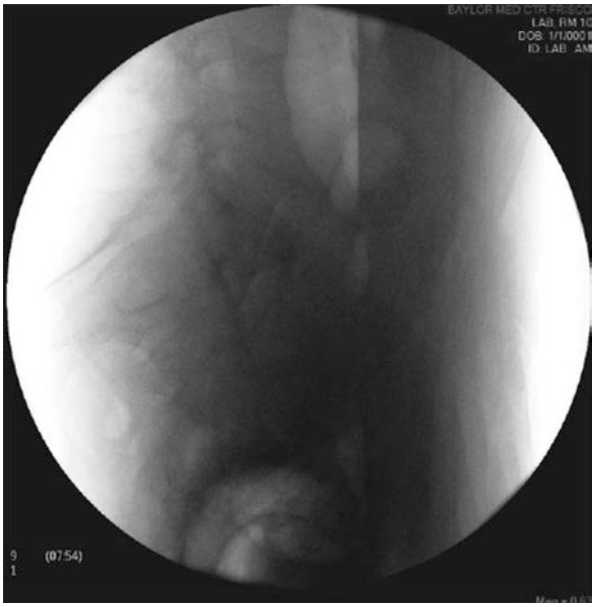


Fig. 12.47 The RX-Coude needle with the blunt stylet is rotated to the superior tip orientation and advanced into the epidural space

S1 Catheter Injections

1. Connect the Stingray® Connector and inject 10 cc of OMNIPAQUE™ 240 within the scarred area. Injection of contrast may require significant pressure for a complete spread due to its viscosity. It will open up the ventral epidural space, slowly crossing over, and spread from L4 down to S2 bilaterally.
2. Inject a mixture of 10 cc of preservative free saline and 150 units of Hylenex®; this will disperse the contrast. Carefully observe for a potential spread into the subdural and subarachnoid spaces, especially in failed surgery cases where the possibility of a dural tear may exist.
3. Slowly inject a mixture of 10 cc of 0.2% ropivacaine and 40 mg triamcinolone. Ask the patient to move their feet and to report any pain at any time other than during injection. Subdural injectate accumulation in the scarred area may produce bilateral pain and have atypical appearance. If subdural loculation occurs, it can be aspirated with an interlaminar needle placement.
4. After local anesthetic injection, observe the patient for 20–30 minutes and make sure they are able to perform a 90 degree straight leg raise without any evidence of motor block.
5. Infuse 10% NaCl over a 15-minute period. Then flush with local anesthetic or normal saline at completion.
6. If the patient develops a motor block, he or she may need to be admitted into the hospital for observation.

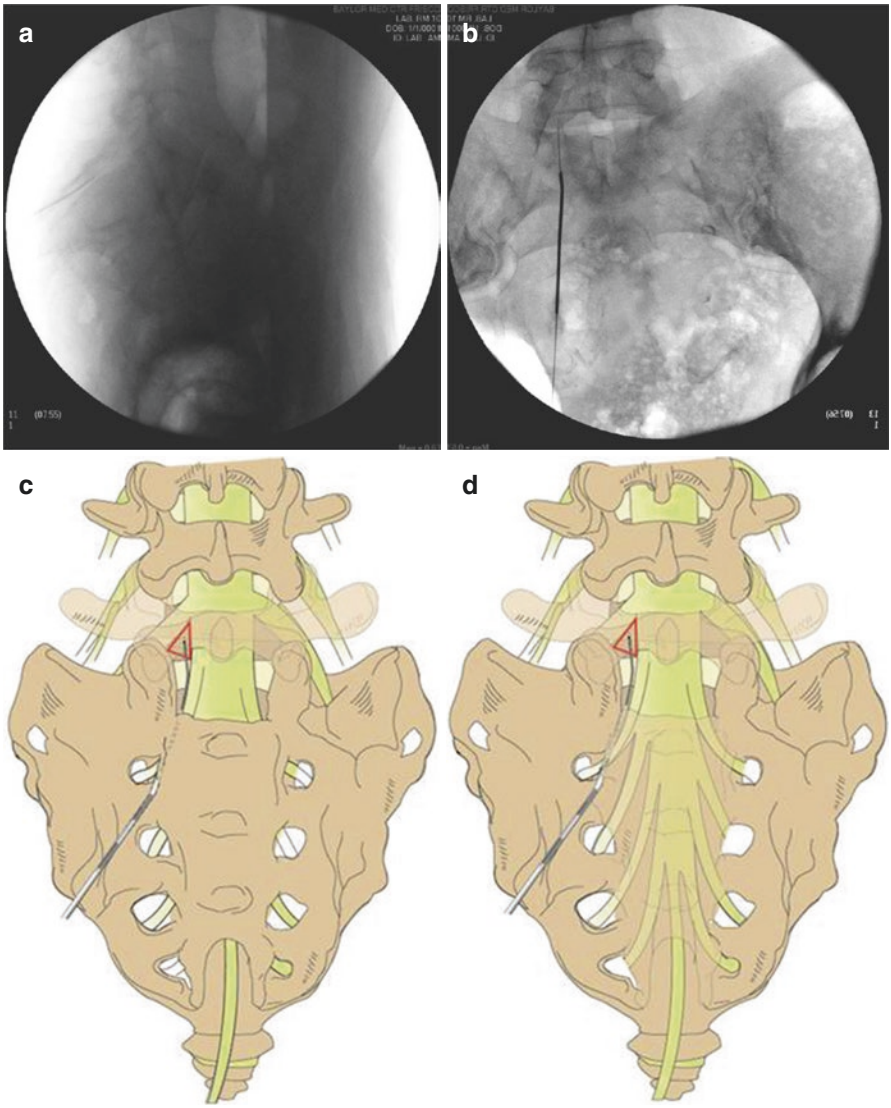


Fig. 12.48 (**a**) The Rx Coude needle tip is rotated to the superior orientation for catheter placement. (**b**) The Rx Coude needle tip is rotated to the superior orientation for catheter placement. (**c**) The Rx Coude needle tip is rotated to the superior orientation for catheter placement. The scarring triangle is indicated by the red triangle. (**d**) The Rx Coude needle tip is rotated to the superior orientation for catheter placement. The scarring triangle is indicated by the red triangle

7. A one-time injection into the scarring triangle is effective for a short period of time; however, three repeat injections, 6–8 hours apart, have been reported as more effective for many months to over a year.
8. Instruct the patient to perform neural flossing exercises to the patient for the sciatic area. There are also separate instructions for the upper lumbar area.

Apply the Stingray® Connector and inject 10 cc of OMNIPAQUE™ 240 within the scarred area. Contrast is viscous, sticky, and hard to inject. Injection of contrast requires significant pressure from the syringe. It will open up the ventral epidural space, gradually crossing over, and may spread from L4 all the way down to S2 bilaterally. Next, inject a mix of 10 mL of preservative-free saline and 150 units of Hylenex®. It will disperse the contrast. Carefully observe potential spread into the subdural and subarachnoid spaces, especially in failed surgery cases where the possibility of a dural tear exists. Slowly inject a mix of 10 cc of 0.2% Ropivacaine and 40 mg Triamcinolone. Ask the patient to move their feet bilaterally and report any continuous pain at times other than during injection. Subdural accumulation in the scarred area may produce bilateral pain and have atypical appearance. 3 If subdural loculation occurs, it can be aspirated with an interlaminar needle placement. So far, subdural spread has not been observed or reported except during a midline caudal catheter placement. Observe the patient for 20–30 minutes post local anesthetic injection and make sure they are able to do a 90-degree straight leg raise without any evidence of motor block. Infuse 10% NaCl over 15 minutes and flush with local anesthetic or saline at completion. Results appear significantly better when three repeat infusions are performed 6–8 hours apart. If the patient develops a motor block, they may need to be admitted into the hospital for observation. Indications include positive dural tug reproducing back pain and hip pain, L5-S1 radiculopathy, and foot drop. We have seen a bladder dysfunction recover following an unavoidable complication from a surgical laminectomy for spinal stenosis. Lower lumbosacral nerve root scarring and stretch injuries appeared responsible for foot drop after unsuccessful surgical procedures. Patients with spinal stenosis, in addition to the radiculopathy, also need a mid-canal transforaminal second catheter at the maximum stenotic area. In addition, each injected volume of contrast, hyaluronidase, and local anesthetic steroid, followed by 10% hypertonic saline are reduced to 5 mL. If pain is experienced during the hypertonic infusion, it may be necessary to top off 2–3 cc of 1% Lidocaine. A one-time injection to the scarring triangle is effective for a short period whereas three repeat injections have been reported to be more effective for many months to over a year. Finally, instruct and provide a handout to perform neural flossing exercises to the patient for the sciatic area. There are also separate instructions for the femoral upper lumbar area.

1. Start with the skin wheal needle technique to numb the entry point area of the introductory needle – RX™Coudé® Needle
2. Diagnostic: 5–10 mL OMNIPAQUE™240* – outline filling defect and place catheter to target site
3. To show runoff and absence of loculation, contrast 4–5 mL OMNIPAQUE™ 240* injected through the catheter
4. Spreading Factor: Hylenex® 150–300 units (human recombinant) diluted in 10 mL of preservative-free saline, or Hyaluronidase Bovine compounded 1500 units diluted in 10 mL preservative-free saline
5. Steroid Injection: 4 mg Dexamethasone or 40 mg Triamcinolone Local Anesthetic: 10 mL 0.2% Ropivacaine or 10 mL of 0.25% Bupivacaine

6. 2–3 mL OMNIPAQUE™240* through catheter
7. Depending on the physician's lysis technique, wait 20–30 min. Evaluate for motor block with a voluntary straight leg raise. If no motor block is present, with the patient's painful side down, inject 8–10 mL of 5–10% hypertonic saline over 20–30 minutes. If the patient experiences pain, inject 2–3 mL of local anesthetic.

Critical note: Make sure to use non-ionic water-soluble dye. Some physicians also use 5–10 mL of ISOVUE-M 200.

Cervical Interlaminar Epidural Catheter Placement

Cervical Interlaminar Epidural RX-2™ Coudé® Needle and Catheter Placement is an advanced technique for cervical radicular pain. The use of the RX™ Coudé® Needle with its second stylet allows atraumatic needle movement with reduced chance of complications. Any movement of a sharp needle in the epidural space has a hazard of cutting the dura or high-pressure veins. High pressure veins develop due to epidural adhesions restricting the veins. Decompressing these high pressure veins with the lysis of adhesions procedure and hydro-dissection is an important effect of the procedure to prevent bleeding. Hematoma formation is rare but must be kept in mind with needle placements into the upper thoracic epidural space. Symptoms can be delayed several hours later, which include back pain, bladder dysfunction, numbness, weakness, and paralysis. Immediate MRI followed by emergent surgical evacuation can prevent permanent cord injury. During the procedure, contrast injection though the catheter in the ventrolateral epidural space should flow out of the epidural space trough a neural foramen. If it doesn't contrast may flow to the opposite side of the epidural space and produce a mass effect and compress the cord. This phenomenon has been described as perivenous counter spread. It is treated with flexion rotation exercises to facilitate flow of injected fluid out of the epidural space by opening the neural foramen by these exercises. The cause of potential pressure build up in the absence of lateral runoff is perivenous counter spread (PVCS). Peri-Venous Counter Spread (PVCS) occurs in the presence of increased epidural pressure and becomes an indicator of possible spinal cord compression. If not sedated, the patient will complain of bilateral pain secondary to cord ischemia. During PVCS, the injected fluid spreads outside of the ventral epidural veins (perivenous) to the opposite side from the injection. If there is no lateral neural foraminal run off, epidural pressure can increase lateral to the cord, on the opposite side (Fig. 12.49). This leads to cord compression and is reported by the patient as bilateral arm and chest pain. Repetitive chin to shoulders flexion rotation enlarges the neural foramina, facilitates decompression, and allows lateral runoff. Recognition of PVCS is especially important if patient is given sedation and is unable to report pain from ischemia. Rotation of the head is safe when the catheter is in place as the needle entry site C7-T1, T1-T2, T2-T3 does not move during rotation. If not recognized and/or patient is sedated, the patient will have post-operative pain, weakness,

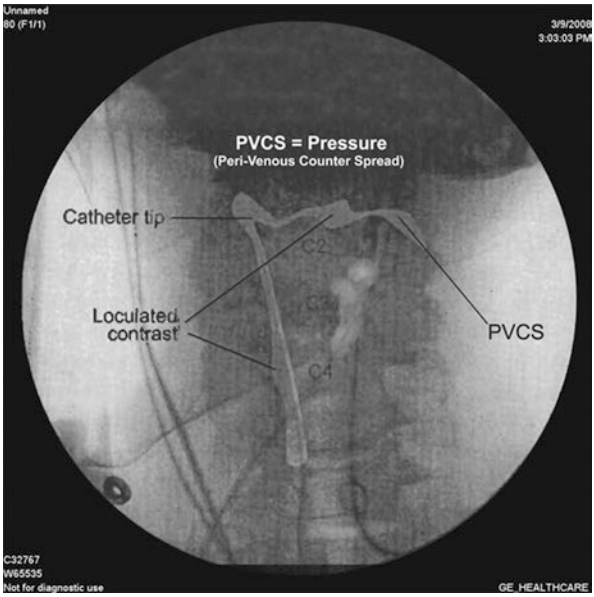


Fig. 12.49 Peri-venous counter spread can occur with cervical injections and requires prenvention and treatment with chin to shoulder maneuvers to open the cervical neural foramina and allow injected fluid to runoff through the foamen, thus decompresing the epidural space

bladder problems and paralysis. MRI does not detect the problem. Remember that cervical spine does not have fixed foramina diameters. They can be opened by flexion and rotation and pressure can be reduced by lateral runoff.

Enlarging of Neural Foramina by Flexion Rotation and Chin to Shoulder Maneuver

During flexion the inferior pars slides forward over the superior pars, making the neural foramen larger (Fig. 12.50). During extension, the inferior pars slides backwards over the superior pars, making the neural foramen smaller. During injection, the patient should flex and rotate the head from left to right to facilitate lateral runoff through the neural foramina. The opening and closing of the neural foramen will help in the assurance of fluid run off, decreasing the probability of increased pressure in the epidural space (PVCS). Flexion and lateral rotations of the spine will change the size of neural foramen, making lateral run off possible. This allows for reduced pressure created by fluid runoff, which will prevent loculation. The indication for pressure build up in the absence of lateral runoff is better known as a perivenous counter spread (PVCS). During flexion, the inferior pars slides anteriorly over the superior pars, making the neural foramen larger. During extension, the inferior pars slides posteriorly over the superior pars, making the neural foramen smaller [20]. During injection, the patient should flex and rotate the head from left to right. The opening and closing of the neural foramen will help in the assurance of fluid run off, decreasing the probability of increased pressure in the epidural space

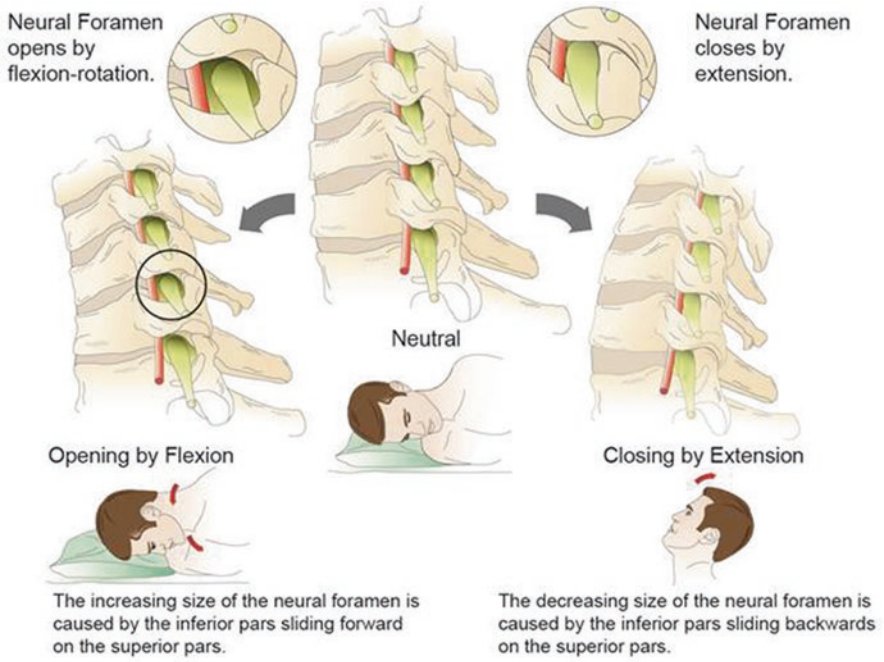


Fig. 12.50 Flexion widenes the neural foramen allowing pressure to be released by fluid flowing out of the foramen. Repeating the maneuver is important to prevent spinal cord compression

(PVCS). Flexion and lateral rotations of the spine will change the size of neural foramen, making lateral run off possible. The fluid runoff out the foramina results in reduced pressure for the loculation. The maneuvers should be continued until signs and symptoms resolve. Peri-Venous Counter Spread (PVCS) occurs in the presence of increased epidural pressure and becomes an indicator of possible spinal cord compression. If not sedated the patient will complain of pain secondary to ischemia. The fluid spreads outside the ventral epidural veins to the opposite side. If there is no lateral neural foraminal run off epidural pressure can also increase lateral to the cord. This leads to cord compression reported by the patient as bilateral arm and chest pain. Repetitive chin to shoulders flexion rotation enlarges the neural foramina, facilitates decompression, and allows lateral runoff. Recognition of PVCS is especially important if patient is given sedation and is unable to report pain from ischemia. Rotation of the head is safe as the needle entry site C7-T1, T1-T2, T2-T3 does not move during rotation. If not recognized and/or patient is sedated – patient will have post-operative pain, weakness, bladder problems – and paralysis. MRI will show nothing. Remember that cervical spine is not fixed, it is variable! The pressure can be reduced!

Cervical Interlaminar Epidural RX-2™ Coude® Needle and Catheter Placement

The posterior cervical anatomy is shown in Fig. 12.51. With the patient in the prone position with a pillow under the patient's chest with no head rest and the arms at the side with the shoulders relaxed anteriorly, the C-arm is rotated into the cephalad direction compensating for the patient's spinal kyphosis helping to optimize and enlarge the C7-T1 inter-laminar target site.

The C7-T1 interspace is used to avoid superior cervical levels where the ligamentum flavum is incomplete and also to avoid large venous plexuses at T1-T2 [21].

The Bromage grip should be used for needle advancement. The Bromage grip includes bracing the knuckles of the non-dominant hand against the patient's back or neck. The needle is advanced with the fingers so if the patient moves toward the needle, the hand and the needle move as well so that the needle does not penetrate deeper. The direction- depth -direction (3-D) technique is used to avoid subdural needle placement. AP and lateral fluoroscopic images are used in an alternating fashion to advance the needle. Following local anesthetic injection, the RX-2™ Coude® needle is introduced with the tip facing anterior medially. Using a paramedian approach allows smooth passage of the RX™ Coude® Needle to the midpoint of the interlaminar space. The point of entry is slightly medial to the pedicle at the level below the chosen interspace. The skin entry point is 1.5 segments below the target interspace and 0.5″ lateral from the spinous process (Fig. 12.52a, b). Orient the needle tip medially while crossing the interspace. Curving the needle medially crossing the interspace, rotate the needle tip down interiorly and bony contact is made with the lamina aiming towards the midpoint, the needle tip is steered until the

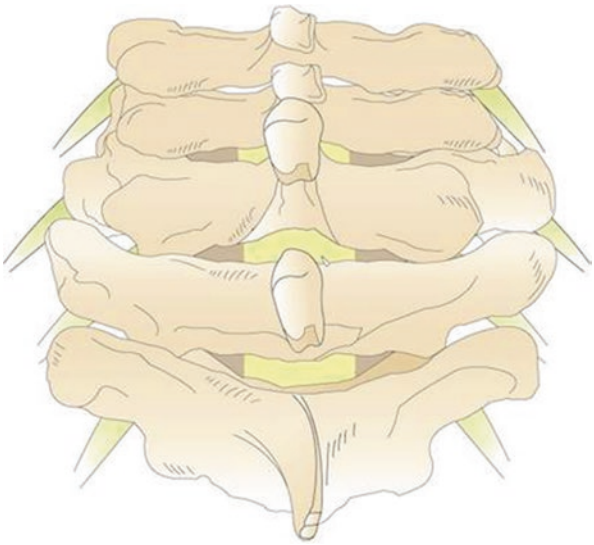


Fig. 12.51 Posterior cervical spine anatomy

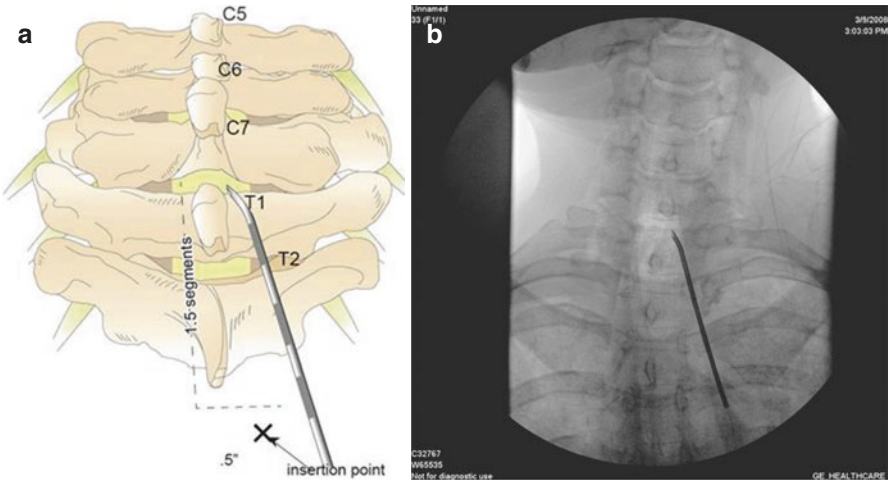


Fig. 12.52 (**a**) The skin entry point is 1.5 spinal segments inferior to the target interlaminar interspace. A paramedian approach is used with the RX-Coude needle tip in the medial orientation position. The seventh cervical – first thoracic interspace is used to avoid higher levels for needle puncture. (**b**) The skin entry point is 1.5 spinal segments inferior to the target interlaminar interspace

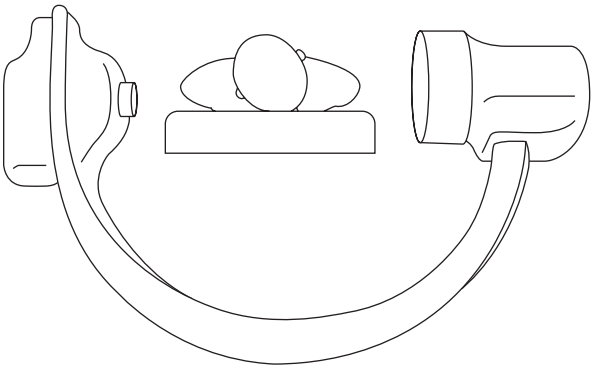


Fig. 12.53 The C-Arm is rotated to the lateral position

edge of the lamina is reached. When the tip of the needle crosses the proximal end of the T1 lamina, the C-arm is rotated to the lateral view (Fig. 12.53). (The base of the spinous processes form a straight line on fluoroscopic imaging) The ligamentum flavum is in direct extension between the straight lines. Rotate the needle anteriorly and advance to the ligamentum flavum (Fig. 12.54a, b). The needle is rotated so that the tip is now parallel with the ligamentum flavum. The lateral view is utilized on fluoroscopy. The needle is advanced to be in line with the "straight line". The straight line is the enhanced bony outline of the bony cortex of the inside and the outsides of the bifurcating lamina. The needle is positioned so that the bevel is parallel with the ligamentum flavum. The ligamentum flavum should be penetrated in the midline. The stylet should be removed and a (LOR) syringe is attached to the

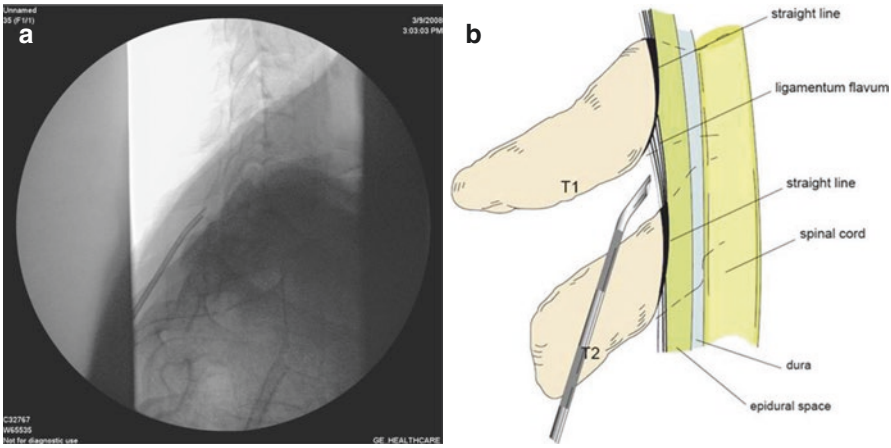


Fig. 12.54 (**a**) The RX-Coude needle is advanced using the lateral image. (**b**) The RX-Coude needle is advanced using the lateral image

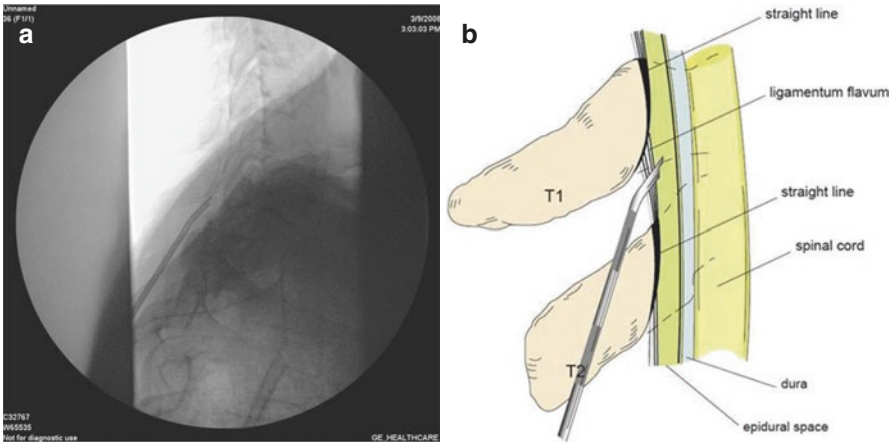


Fig. 12.55 (**a**) The loss of resistance technique is used to complement the lateral fluoroscopic image for identifying the epidural space. (**b**) The loss of resistance technique is used to complement the lateral fluoroscopic image for identifying the epidural space

hub of the needle. The needle is advanced with the "loss of resistance" or "loss of bounce" technique until a loss of resistance is felt which indicates entry into the epidural space. With this technique, the plane of the needle bevel tip is parallel to the plane of the dura, reducing the chance of penetration (Fig. 12.55a, b). The needle is advanced with the "loss of resistance" technique until a loss of resistance is felt which indicates entry into the epidural space. Small volumes of contrast injection can confirm epidural placement on lateral and AP view. The RX-2™ Coudé® Needle features an additional threaded interlocking blunt stylet that protrudes a short distance beyond the RX™ needle tip. The second stylet protrudes approx.

1 mm beyond the tip to convert the needle to a blunt probe. (This can also be used for tunneling a catheter for a stimulating catheter, for example) At this point, the second stylet is placed (Fig. 12.56a). The stylet should not be screwed in too tightly so it can be easily removed later for catheter placement. If the stylet is difficult to remove, the needle should be removed to avoid shearing the stylet. This allows the rotation of the curved needle toward the direction of the area where the catheter needs to be directed. The blunt tip safely pushes the dura away. The needle should only be rotated with the blunt protruding stylet in place. In this configuration, redirection (or rotation) of the needle tip is possible. Any needle directional rotation may cut the dura and lead to CSF leak and spinal headache. The RX-2™ protruding stylet prevents this. After the RX2™ Coude® needle has been rotated in the direction of the target, remove the extended blunt stylet (Fig. 12.56b). It is important to make a half inch 15° bend in the catheter for optimum steering ability (Fig. 12.57). With the XL tip catheter, the stylet needs to be close to the tip for enhanced steering. If the bend is too short, the catheter tends to buckle. If the bend is too long, it is much harder to steer. Insert the catheter into the RX-2™ needle that will safely place the catheter parallel to the dura. Following the RX-2™ Coude® entry into the epidural space, the soft tipped catheter is placed for a short 1/4″–1/3″ distance beyond the tip of the needle to push the dura away. This allows the rotation of the needle towards the intended target. This reduces the chance for the rotation of the needle tip from cutting the dura. When the RX-2™ Coude® needle is rotated in the direction of the target, the catheter is placed parallel to the dura. The RX-2™

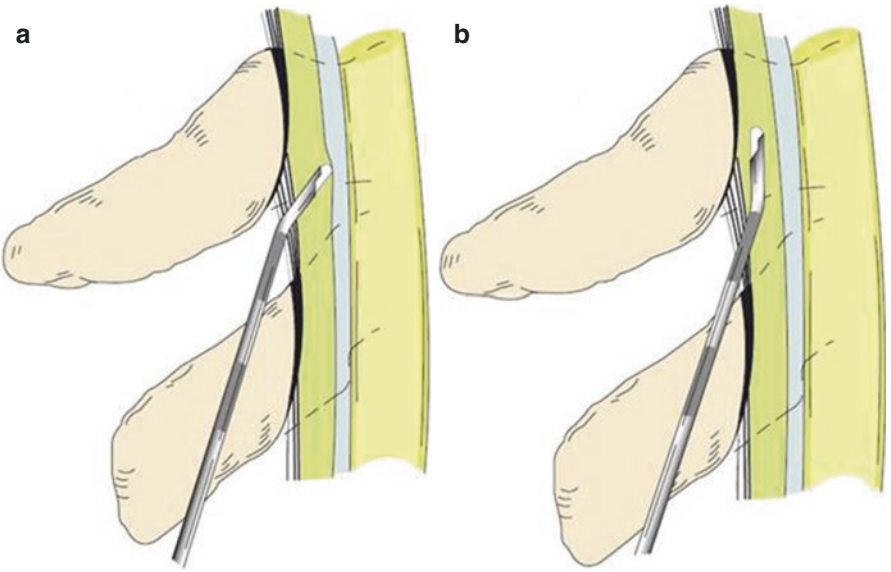


Fig. 12.56 (**a**) The blunt stylet is placed to prevent dural laceration or puncture. (**b**) The RX-Coude needle is rotated to the superior tip orientation with the blunt stylet in place to prevent dural or vascular laceration

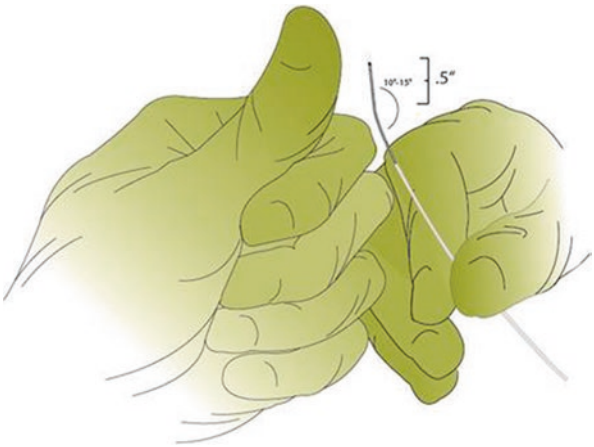


Fig. 12.57 A 10–15 degree bend is made one half inch from the catheter tip to facilitate steering

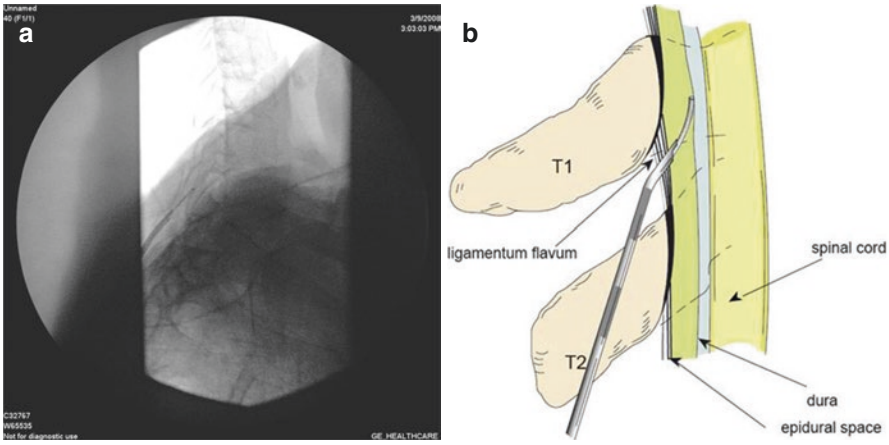


Fig. 12.58 (**a**) Incorrect needle position for catheter placement. The needle is in the anterior orientation position and so the catheter projects toward the dura. (**b**) Incorrect needle position for catheter placement. The needle is in the anterior orientation position and so the catheter projects toward the dura

Coudé® Needle should always point in the direction of the target. The incorrect needle orientation is shown in Fig. 12.58a, b. After rotation, the catheter or electrode becomes easier to direct and parallel to the plane of the dura (Fig. 12.59a, b). The C-Arm is rotated to the anterior –posterior position (Fig. 12.60). The catheter tip is placed towards the C6 ventral-lateral epidural space (Fig. 12.61a, b). Any movement of a sharp needle in the epidural space has a hazard of cutting the dura or high-pressure veins. Hematoma formation is rare but must be kept in mind with needle placements into the upper thoracic epidural space. Symptoms can come some hours later that include back pain, bladder dysfunction, numbness, weakness, and paralysis. Early MRI followed by surgical evacuation can prevent permanent

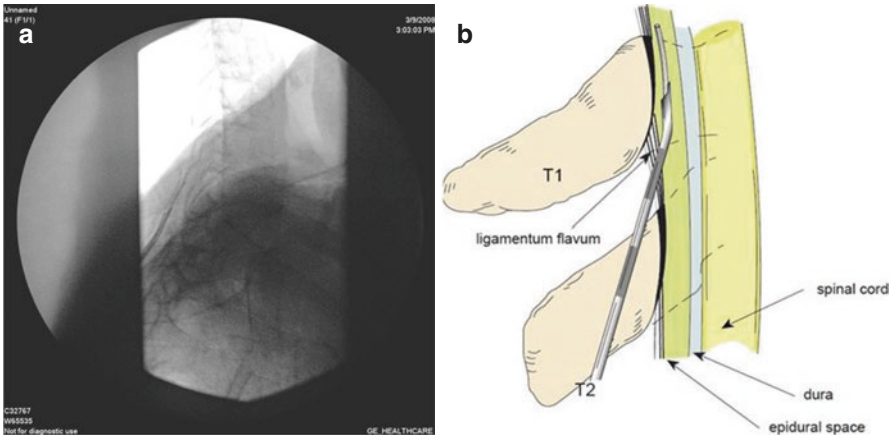


Fig. 12.59 (**a**) Correct needle orientation to allow catheter passage without dural compression. (**b**) Correct needle orientation to allow catheter passage without dural compression

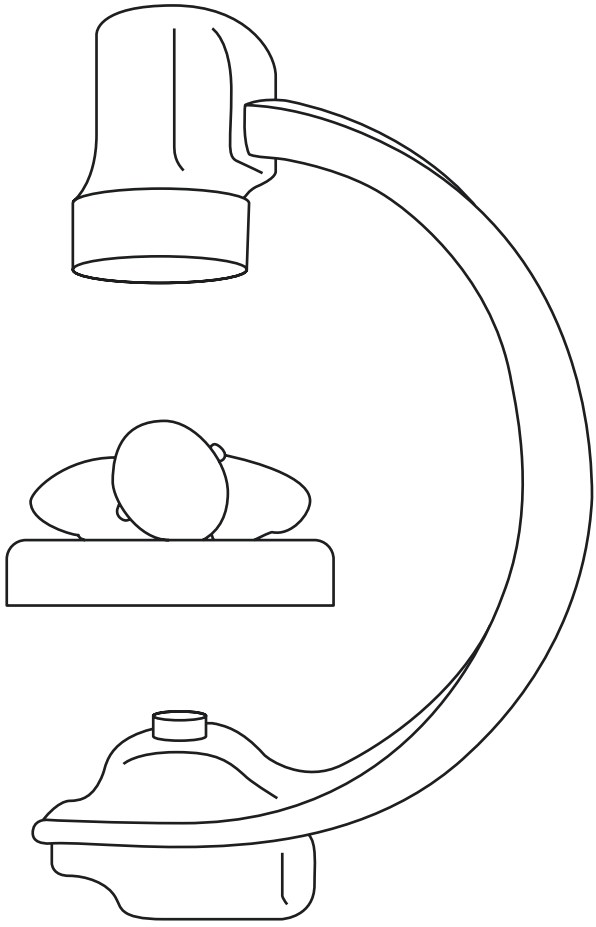


Fig. 12.60 The C-arm is rotated to the anterior –posterior position

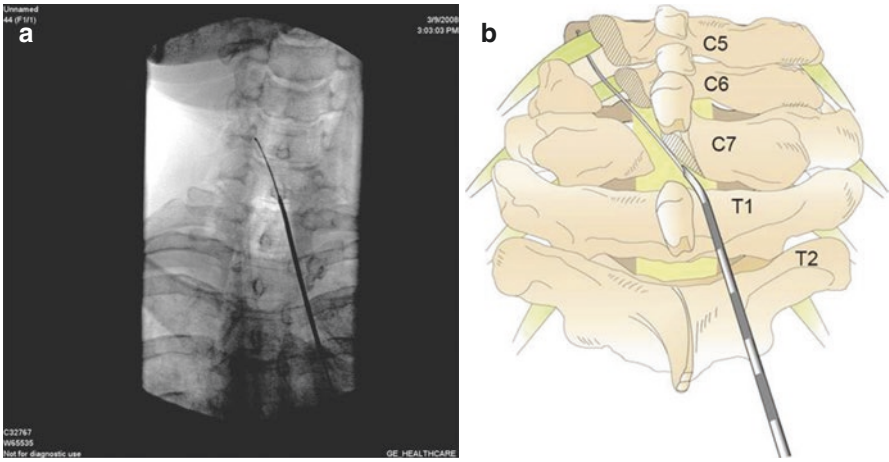


Fig. 12.61 (**a**) The catheter is placed to the target location. (**b**) Cutaway diagram – The catheter is placed to the target location

cord injury. The second stylet of the RX-2™ Coude® Needle makes needle movement atraumatic with reduced chance of above-mentioned complications. Bacterial filters are recommended in all instances when more than one-time injection is used or the catheter is left in place for prolonged period. Anytime there is a disconnection of the catheter and the connector the system should be removed from the patient. This is an essential precaution to prevent infection. Strongly recommend is the Advanced Catheter Fastening Technique as described next.

Advanced Catheter Fastening Technique

1. Make a full twist in the catheter to form a loop.
2. Place loop over the neck of the connector.
3. Pull catheter until securely around the connector body.
4. Use tape to secure the device.
5. Attach a bacterial 0.2-micron filter to maintain sterility.

Epimed's Stingray™ Connector design allows for a fastening technique that changes pulling force direction to prevent disconnects. The Stingray™ when compared to four other connectors for grip and strength was found to be the best; however, for repeat injections or prolonged use the following additional measures further enhance safety. Physicians have been known to use this technique during a three-day lysis series or post procedure injection of hypertonic saline in the recovery room for the one-day procedure. It is useful when prolonged or postoperative infusion is utilized. The force required to separate the catheter from the connector is more than double that of other connectors. Bacterial filters are recommended in all instances when more than one injection is used or the catheter is left in place for prolonged period. Anytime there is a disconnection of the catheter and the connector the system should be removed from the patient. This is an essential precaution to prevent infection.

Catheter Tape Down Technique

– Place suture and tie loose loop.
– Wrap around catheter two times and tie surgical knot.
– Apply antibiotic ointment around skin entry and place two-split 2 × 2" gauze to keep antibiotic in place.
– Apply adhesive i.e. tincture of benzoin around gauze.
– Place one loop on catheter and transparent dressing i.e. opsite
– Connect bacterial filter and four pieces of sweat resistant hypofix tape.

The 10 Step Approach to Safer Cervical Catheter Placement Using RX-2™ Coude Needle.

1. Point of entry is one and a half segment below the target 1/2 inch from mid line
2. Cross interspace- curving medially
3. Curve down to lamina to touch bone (lamina)
4. Curve medially and advance needle to edge of lamina in the mid line
5. Rotate C-arm to lateral view and look for the straight line
6. Advance needle to just below straight line and remove stylet
7. Use loss of resistance or loss of bounce technique to enter the epidural space
8. Reduce dura perforation from tip movement by either:
   A. advancing the catheter short distance beyond tip of needle to push dura free from tip of needle
   B. placing protruding RX-2™ stylet and make sure interlocking cap is rotated clockwise, pushing the dura from the tip of the needle
9. Rotate needle towards desired target side
10. Put 1/2 inch 15-degree bend at the Racz® bend mark on the distal tip of the catheter and thread the catheter to the lateral cervical epidural space at the target nerve root

Equipment Options

1. 18 Gauge RX-2™ Coude® needle and 21 Gauge Versa-Kath® with Stingray™ Connector and bacterial filter if multiple injections are anticipated.
   (a) Observe safety recommendations about taping, filter, and volumes of injections.
2. 15 or 16 Gauge RX-2™ Coude® needle and Brevi-XL™ with a similar 15 degree bend for one time use or Tun-L-XL™ 24 catheter if re-injections are anticipated.

1. Both catheters connect to the specific gauged Stingray™ Connector with or without the bacterial filter.

Cervical Injections

1. Diagnostic: 1–2 mL OMNIPAQUE™240* – outline filling defect and place catheter to target site
2. To show runoff and absence of loculation, contrast 0.5–1 mL OMNIPAQUE™ 240* injected through the catheter

3. 1–2 mL OMNIPAQUE™240* through catheter for verification of enzyme effectiveness
4. Spreading Factor: Hylenex® 150–300 units (human recombinant) diluted in 5 mL of preservative-free saline
5. Steroid Injection: 4 mg dexamethasone or 40 mg triamcinolone
6. Local Anesthetic: 6 mL 0.2% ropivacaine or 10 mL of 0.25% bupivacaine
7. Depending on the physician's lysis technique, wait 20–30 min. Evaluate for motor block. If no motor block is present, with the patient's painful side down, inject 5 mL of 10% hypertonic saline over 5–10 minutes. If the patient experiences pain, inject 2–3 mL of local anesthetic.

The 10 Step Approach for Safer Cervical Catheter Placement Using RX-2™ Coude® Needle.

1. Point of entry is one and a half segment below the target 1/2 inch from mid line
2. Cross interspace- curving medially
3. Curve down to lamina to touch bone (lamina)
4. Curve medially and advance needle to edge of lamina in the mid line
5. Rotate C-arm to lateral view and look for the straight line
6. Advance needle to just below straight line and remove stylet
7. Use loss of resistance or loss of bounce technique to enter the epidural space
8. Reduce dura perforation from tip movement by either:
   A. advancing the catheter short distance beyond tip of needle to push dura free from tip of needle
   B. placing protruding RX-2™ stylet and make sure interlocking cap is rotated clockwise, pushing the dura from the tip of the needle
9. Rotate needle towards desired target side
10. Put 1/2–3/4 inch 15-degree bend in catheter and thread the catheter to the lateral cervical epidural space at the target nerve root

Equipment Options

1. 18 Gauge RX-2™ Coude® needle and 21 Gauge Versa-Kath® with Stingray™ Connector and bacterial filter if multiple injections are anticipated.
   (a) Observe safety recommendations about taping, filter, and volumes of injections.
2. 15 or 16 Gauge RX-2™ Coude® needle and Brevi-XL™ with a similar 15 degree bend for one time use or Tun-L-XL™ 24 catheter if re-injections are anticipated.
3. Both catheters connect to the specific gauged Stingray™ Connector with or without the bacterial filter.

Epidural Lysis of Adhesions procedures are techniques for which there are CPT codes and virtually uniform reimbursement. Lysis is a technique that saves money, reduces the incidence of surgery, and even in the presence of failed back and neck surgery is much more beneficial and effective than repeated surgeries. It improves the effectiveness of spinal cord stimulation where the intensity of the pain may be too much for the spinal cord stimulator to reduce. We now see evidence of nerve function recovery following the use of the Lysis of Adhesions technique [22].

Neural Flossing Exercises

Before beginning the neural flossing exercises, the patient is given an instructional guide, available for both cervical and sciatic nerves. Femoral nerve stretch exercises are important for upper lumbar pathology. Experience supports sustained stretching, such as straight leg raises over a 20–30 second time span. This changes the stretching of the nerve to sliding of the nerve; thus regaining the mobility of the previously scarred nerve root. Clinical experiences show that physical therapy by itself does not free up a scarred nerve root very readily. Veihelman compared neuroplasty to physical therapy and reported that neuroplasty was more effective [23]. In failed neck surgery patients, patients respond to lysis of adhesions as manifested by reducing radiating pain. Continuation of cervical neural flossing exercises can result in complete resolution of pain and spasm up to 2 years after the lysis procedure (two to three times per day with 30 second sustained hold). Patient involvement is essential. The cervical exercises are shown as component moves in Figs. 12.62, 12.63 and 12.64. The combined exercises are shown in Fig. 12.65.

A recent observation after the lysis procedure is that patients may develop pain in the absence of positive straight leg raising. In these cases, the epidurogram shows scarring of the DRG (dorsal root ganglion) area. The nerve root has stretched and the ganglion and lateral recess area developed scarring. Movement related pain is not present. Repeat lysis, especially with hylenex, is effective and needs to be repeated. An important concept is the approach to treatment of scarred nerve roots. The problem begins with degenerative disc disease when the nucleus pulposus material leaks into the epidural space. This produces an inflammatory response leading to radiculitis secondary to inflammation and scar tissue, scarring of the

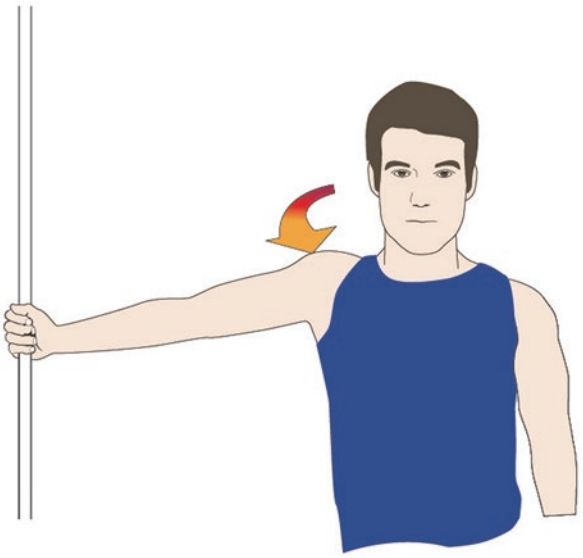


Fig. 12.62 Component move of cervical neural flossing 1

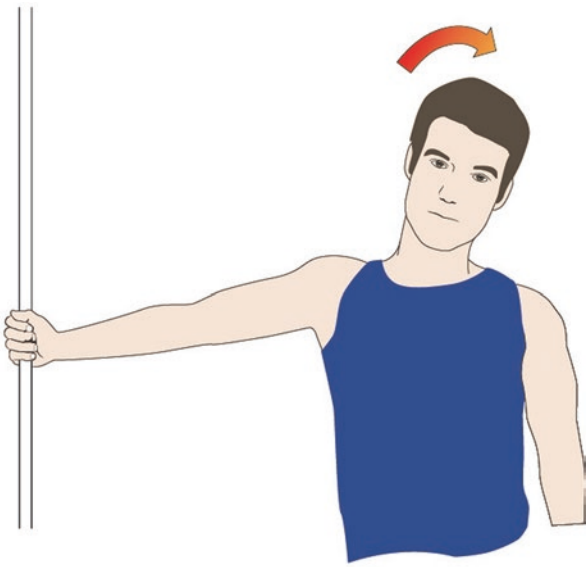


Fig. 12.63 Component move of cervical neural flossing 2

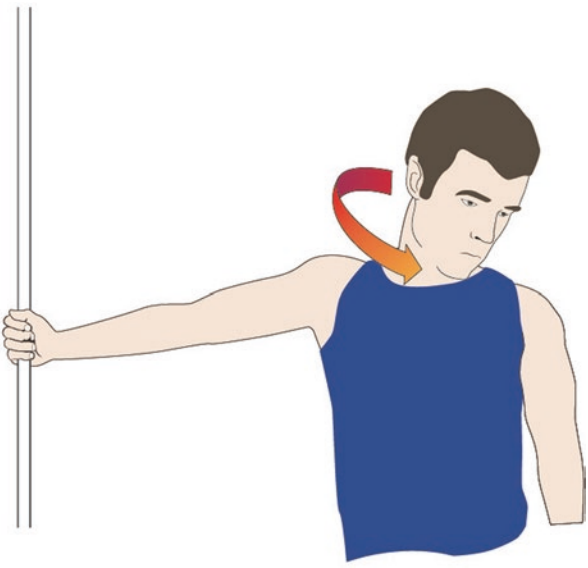


Fig. 12.64 Component move of cervical neural flossing 3

nerve root and movement related pain. The work of Indahl, et al., shows that discogenic impulses can lead to back spasms [24].

Alteration of the disc height and facet joint alignment is changed at the same time. Pain generation arises from the disc, nerve root, facets, and the muscles in the back and iliopsoas muscles. Patients suffering from upper lumbar back pain may develop significant back spasm and groin pain. To stretch out the back spasm, the patient may do this in a reclining position where curl up both knees and pull up to

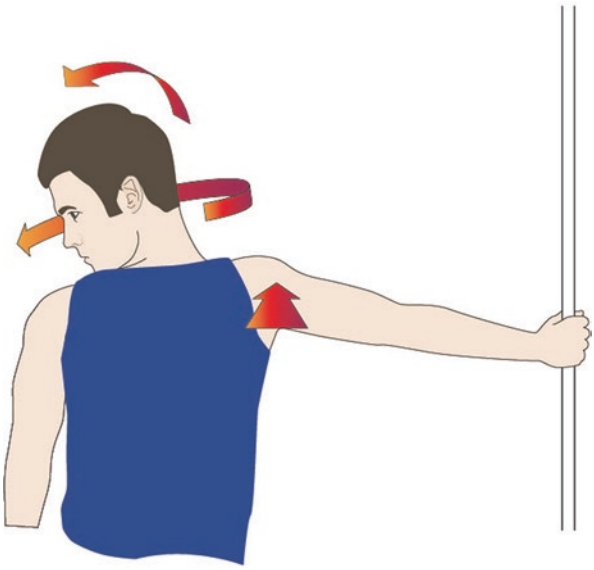


Fig. 12.65 Combined moves for chin to shoulder neural flossing maneuver

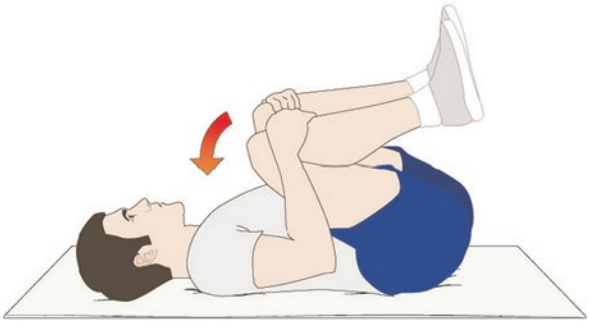


Fig. 12.66 Reclining lumbar neural flossing maneuver 1

the chest and holding the head in a straight position (Fig. 12.66). This can stretch the muscles that are in spasm. This stretching motion can be repeated multiple times during the day, but the crucial aspect it should be held for 22–30s duration. After a period of rest, it can be repeated several times. Additional exercises are also performed (Figs. 12.67 and 12.68). When there is femoral nerve involvement and back spasm as secondary feature, often there is a thigh pain. If the patient is unable to do the exercise in a standing position, the exercise can be performed on a comfortable mattress in the lateral position with the asymptomatic side in the dependent position. The ankle of the symptomatic leg is pulled to stretch the quadriceps femoris muscle as much as possible for 30 seconds. The neck and back should be extended to stretch the second and third lumbar nerve roots. This movement also loosens the large intra-abdominal psoas muscle that is involved in patients suffering from back pain from the upper lumbar nerve roots. These exercises should be performed 2–3 times a day and repetitions have been found to be helpful. Femoral Stretch exercises may be done standing. This exercise is primarily for patients

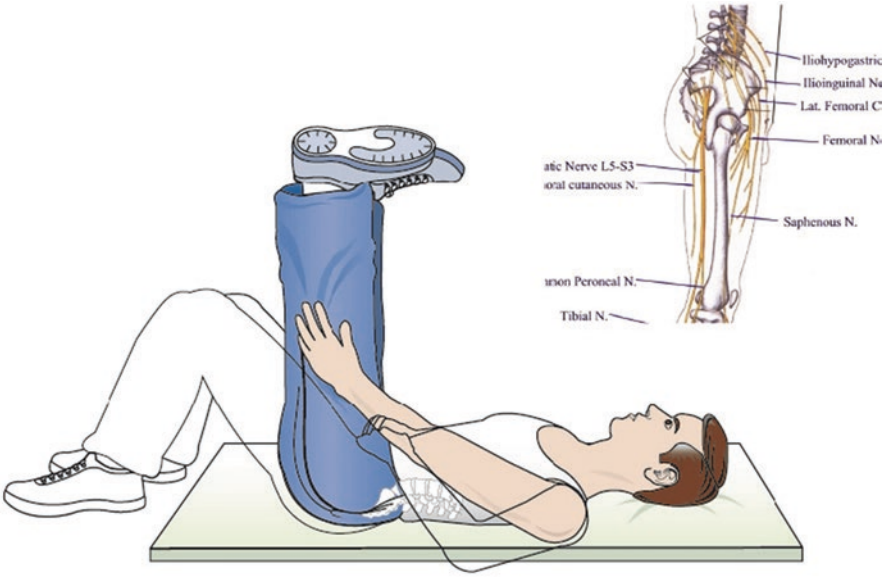


Fig. 12.67 Reclining lumbar neural flossing maneuver 2

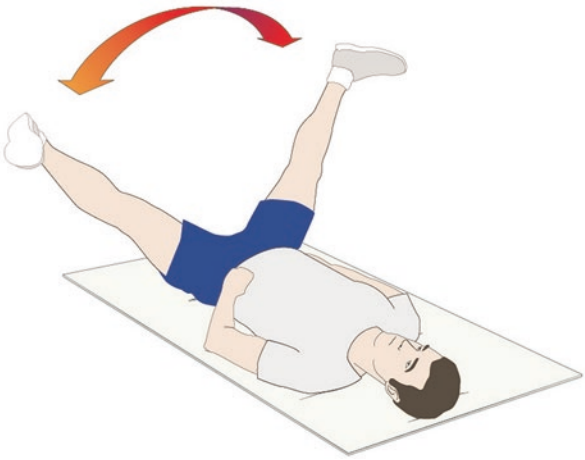


Fig. 12.68 Reclining lumbar neural flossing maneuver 3

suffering from upper lumbar back pain and radiating pain involving the lower extremity but specifically the anterior thigh area. The femoral nerve primarily comes from the second and third lumbar nerve roots and join together to form the femoral nerve. The femoral nerve exists through the front of the upper thigh from the abdomen and innervates the main muscles of the upper thigh and supplies sensory innervation down to the inside of the lower leg to the level of the inside or medial ankle. The patient suffering from back pain also develops severe muscle spasms in the iliopsoas muscle.

Patients that have hip replacement are at risk for subluxation of the hip joint. These patients may gently perform femoral stretch exercises in a standing position, leaning against a wall and extending at the hip to move the leg slowly back in a straight stretching position (Fig. 12.69). This will stretch the iliopsoas muscle as well as the femoral nerve roots and to mobilize the femoral nerve. Once the leg is moved back to maximally obtainable position, the stretch can be exaggerated by leaning and pushing backwards from the wall and maintaining this position for 22–30s duration, helps to reverse significant spasm. An alternate exercise for patients that have no hip joint issues is to stabilize against the wall hold the lower part of the foot. The leg may be forcibly pulled back while leaning backwards again achieving significant stretching of the muscle and spasm and help to mobilize the femoral nerve (Fig. 12.70). Again the stretching position should be maintained 20–30s and can be repeated many times whenever the pain is significant.

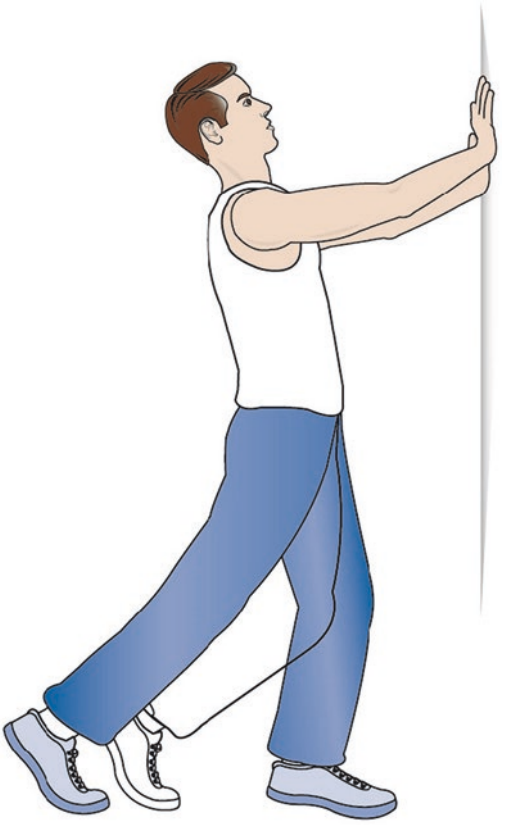


Fig. 12.69 Standing lumbar maneuver 1

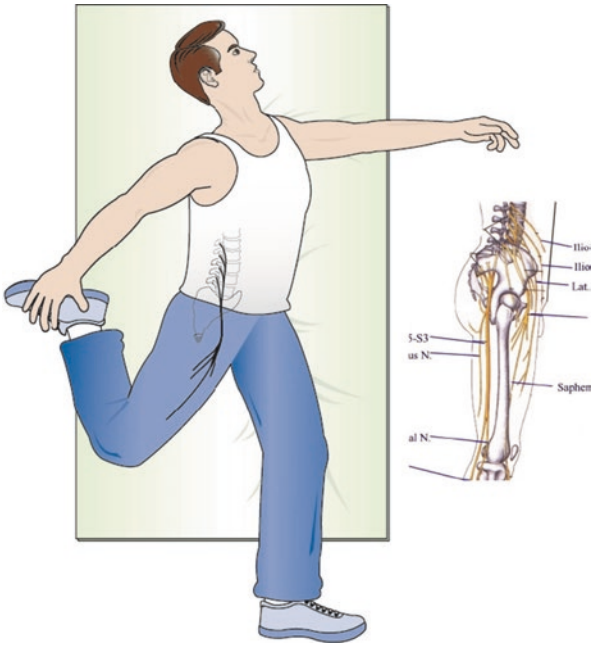


Fig. 12.70 Femoral stretch

Dural Flossing Exercises

Normally, the dura matter and posterior longitudinal ligament slide and glide over each other. The dura mater moves 10–15 mm left to right and superiorly and inferiorly. It may move irregularly if there is scarring that attaches it to the posterior longitudinal ligament or other structures. Dural flossing exercises are performed by the patient flexing the hips and extending the knee. Then the entire spine is flexed in order to creating shearing forces between the dura and posterior longitudinal ligament. These exercises maintain the separation between the dura and posterior longitudinal ligament that is created by the lysis of epidural adhesions procedure. These exercises should be performed 10 times per day after the lysis procedure to disrupt recurrent scar formation. Continuing exercises long term may help prevent recurrent scar formation and pain.

Medicolegal Concerns

Complications occur despite the best of training, technique and patient selection. The information we have gained regarding complications and disasters does not come from prospective randomized studies, but has come from the busy practitioner's daily work and experience in the medical/legal arena. Unfortunately, many of these complications have not been published because they have failed to become public information and either the patient or the physician did not give consent for

the information to become public. We need more evidence. We need more studies. However, one noteworthy aspect of published studies is the lack of reported complications. The studies do not teach us what we must avoid in order to spare our patients and ourselves the stresses and hazards of undesired outcomes and complications. However, this information must be shared. Denial of complications or pretending that complications can be avoided by following one's imaginary procedural guidelines amounts to misleading posture and wishful thinking. I am not aware of any physician who gets up first thing in the morning with the idea that "I am going to hurt somebody today." It is in the training, in the fiber, in the blood of the physician; the concept that has been around for centuries of "Primum non nocere" in other words "First, do no harm." The development of the techniques for these procedures has been evolving because there have been disasters, deaths and paralyzed patients. This is not due to the physician being careless, negligent, or failed to follow instructions. Having reviewed over 300 medical malpractice cases, one cannot miss when a similar scenario presents itself repeatedly. This pattern may mean that there is information not generally known for avoiding the development of this pattern that leads to the complication.

The ten-step approach for cervical needle and catheter placement described in this chapter will lead to significant reduction in the problems encountered where the patient may end up quadriplegic, paraplegic or suffer from hemiplegia or Brown-Sequard syndrome. The techniques described represent years of experience. Optimizing the speed in which procedures are carried out will dramatically reduce the complications that we are forced to defend in the medical/legal arena. The number of legal cases is increasing at a steady constant rate. Cases are related to loculation from an interlaminar single needle injection. Syrinx formation has occurred several weeks following a single shot epidural steroid injection, possibly due to loculation of injected fluid. The concept of treating peri-venous counter spread (PVCS) could possibly lead to prevention of permanent cord injury. The diameters of the cervical spinal canal and foramina are not static. Flexion and rotation of the cervical spine leads to enlargement of neural foramina and facilitates runoff from the cervical epidural space. This runoff may very well be lifesaving in that the pressure is reduced, blood supply is reestablished to the spinal cord, and a major disaster for the patient and a major lawsuit for the doctor may be averted. A number of cases of interlaminar, small gage Touhy needle injections have been associated with complications. The tip of the Touhy needle simultaneously ends up in the epidural and subdural space and local anesthetic injection leads to delayed cardiovascular collapse. Touhy needles in the cervical area can go through a gap in the ligamentum flavum and end up in the spinal cord. Scanlon et al., have recommended that the way to reduce cervical vascular injury and complications is to use blunt needles [25]. Similarly, using a spring tip catheter is less likely to penetrate or traumatize vascular or neural structures. The pattern of symptoms that should be recognized is bilateral arm pain, chest pain and even leg pain. Pain, numbness then weakness are the sequence of symptoms in PVCS [26]. Emergent communication with colleagues has been an effective way to help manage complications in this situation [27].

Informed Consent

Informed consent is very important as these procedures have significant risk and are often of limited efficacy. In Texas, state law requires specific language in written consent forms for neuroaxial procedures, peripheral and visceral nerve blocks and/or ablation, and implantation of pain control devices:

Neuroaxial procedures (injections into or around spine)
Failure to reduce pain or worsening of pain
Nerve damage including paralysis (inability to move)
Epidural hematoma (bleeding in or around spinal canal)
Infection
Seizure
Persistent leak of spinal fluid, which may require surgery
Breathing and/or heart problems including cardiac arrest (heart stops beating

Peripheral and visceral nerve blocks and/or ablation
Failure to reduce pain or worsen pain
Bleeding
Nerve damage including paralysis (inability to move)
Infection
Damage to nearby organ or structure seizure

Implantation of pain control devices
Failure to reduce pain or worsening of pain
Nerve damage including paralysis (inability to move)
Epidural hematoma (bleeding in or around spinal cord)
Infection
Persistent leak of spinal fluid which may require surgery

Complications

Adverse drug reactions include:
Allergic reactions or adverse drug reaction from antibiotics, iodine, contrast, disinfectants, local anesthetics, hyaluronidase, corticosteroid.
Headache
Cushingoid syndrome
Macular hemorrhage
Sensorimotor deficit
Bowel bladder and sexual dysfunction
Infection
Pain at site
Local anesthetic toxicity.

Procedure related problems include:
Pain at injection site
Inflammation

Pain from hypertonic saline
Barotrauma
Ischemia of spinal cord ot nerves
Retinal hemorrhage
Sterile technique
Catheter shearing
Misplaced catheter or needle
Wrong tissue plane or structure

The management of local anesthetic toxicity includes:

- discontinue all local anesthetic administration
- provide basic and advanced life support
- control airway with endotracheal intubation if indicated
- administer 100% oxygen, and ensure adequate pulmonary ventilation
- Intravenous access should be maintained
- Seizures should be treated with benzodiazepines

Intravenous lipid emulsion 20% is used to treat local anesthetic toxicity.

Initial intravenous bolus injection of 20% lipid emulsion 1.5 mL/kg over 1 minute and start an intravenous infusion of 20% lipid emulsion at 0.25 mL/kg/minute.

Hyaluronidase has been estimated to have an incidence of adverse reaction of 3% [28]. However, in practice, the incidence of adverse reactions to hyaluronidase is rare. Adrenal suppression, elevations of blood glucose, immunosuppression, osteoporosis, weight gain, fluid retention, cataract formation, mood changes and Cushing's syndrome are significant concern with corticosteroid use. Also steroids have multiple other effects that can be significant. However, most steroid related complications from single shot epidural steroid are associated with inadvertent intrathecal steroid injection [29].

Arachnoiditis and aseptic meningitis have been reported. However, this may be less common with the use of fluoroscopy, contrast injection to conform placement and test doses of local anesthetic. Complications are more likely in patients with arachnoiditis or severe scarring in the epidural space.

Barotrauma and spinal cord ischemia can occur from loculation of injectate in an epidural compartment. Rocco described the anatomy and flow of fluid in the compartments of the epidural space [30]. Injected fluid may accumulate in the compartment where it is injected but may move to another compartment when the pressure gradient reaches a point to produce flow. Hypertonic saline may flow from a compartment that is anesthetized into a compartment that is not. In this case, hypertonic injection needs to be stopped and additional local anesthetic given. Also, hypertonic saline draws water into the space where it is and increase the volume of fluid in the space. This can have an additional mass effect on the arteries and veins of the spinal cord and cause ischemia.

Pulmonary edema and cerebra infarction have been reported with the procedure. Following intrathecal hypertonic saline, paresis may occur and persist for hours. Transient hemiplegia, paresthesia lasting for weeks, permanent loss of sphincter

control and sacral anesthesia have been reported [31]. Vision impairment from retinal hemorrhage has been reported [29].

Misplaced needles and catheters can be a source of complications. Subdural injection of particulate steroid can result in vasospasm and cord ischemia. Using a loss of resistance technique and slow advancement of the needle can reduce the risk of puncturing the dura. Using an RK-2 needle with a blunt stylet can reduce the risk of dural puncture during needle rotation. Also using a spring tip catheter reduces the risk of catheter puncture of the dura. Aspiration tests and contrast injection helps to confirm epidural placement. A test dose of local anesthetic is used to ensure that a subdural block is not observed before therapeutic drugs are administered. Subdural contrast has a characteristic appearance as contrast flows circumferentially around the thecal sac. Contrast is most apparent on the tangential view and has been described as a railroad track appearance. The contrast will typically spread multiple segments and certainly more than would be anticipated with the same volume of contrast in the epidural space. Intrathecal contrast collects in the thecal sac and appears in the middle of the canal on the lateral fluoroscopic image as well as on the anterior-posterior view. The procedure should be aborted if the radiographic contrast does not appear to be clearly epidural. Patient can return several weeks later for an attempt with a different approach. Subdural aspiration with a separate needle can be effective at removing subdural injected hypertonic saline. Aspiration of a catheter is not very effective. The volume of the injected hypertonic saline can increase 11 fold and produce bowel and bladder denervation.

Trainees

Fellows are more likely to have problems within the first 4–6 months. The risk of catheter shearing or other complications necessitate close supervision for the first half of the fellowship year. Fellows should have graded independence as the fellowship progresses but each fellow progresses at their own pace. The requirements for the number of procedures in fellowship is below what is required for privileges at some hospitals so the goal should not be to meet the minimum number required. For the lysis procedure, 10 procedures should be performed as a trainee or under proctor by an experienced interventionist.

Prevention of Complications

Patient selection is one of the most important factors in avoiding complications. Having a proper indication for the procedure is critical. Procedure selection is important as well. Oftentimes, different procedures are reasonable options for patients with back and leg pain. For example, facet injections, medial branch blocks or radiofrequency denervation of the facet joints may be considered as a first step procedure. However, it is important to consider the procedure with the most likely chance of benefiting the patient as a first step procedure rather than performing multiple procedures on the same patient with the same complaint.

History of physical examination are important to identify risk factors, indications and contraindications for the procedure. Imaging should be performed to rule out conditions such as diskitis, osteomyelitis, neoplasm and other serious conditions. Multiple sclerosis and demyelinating diseases are relative contraindications. The presence of a syrinx is a relative contraindication. A syrinx may increase in size if epidural pressure if reduced. An increase in epidural pressure may reduce blood flow to tenuous areas of the cord. Nowadays many patients are taking prescription anticoagulants and many more take over the counter drugs or supplements that need to be stopped prior to a lysis of epidural adhesions procedure. Patients with mechanical heart valves must remain anticoagulated and are not candidates to the lysis procedure. Patients with recent coronary stints or deep venous thrombosis are high risk for holding anticoagulation and pain procedures are relatively contraindicated. Oftentimes the anticoagulant prescribing physician will tell patients to hold anticoagulation for a duration of time that is less than the guidelines recommend. The risk of bleeding is higher with a lysis procedure compared to a single shot injection and the balance between the risk of continuing or holding anticoagulation must be considered for all those caring for the patient. It is best to not perform the procedure if a disagreement about anticoagulation persists between the interventional pain physician and the anticoagulant prescribing physician.

Motivation to perform neural flossing exercises is an important factor. Patients should be given education about the exercises and asked to perform them to make sure the patient is doing the exercises correctly. A lack of motivation could contribute to a treatment failure. Obesity is a risk factor of increasing incidence. Fluoroscopic imaging quality goes down as obesity goes up. Also, obese patients may have a higher infection risk. Opioid use is another risk factor for poor outcomes. High doses and a history of multiple prescribers is associated with a higher risk of overdose and substance use disorder. Some patients will seek procedures in order to secure a new prescribing doctor. Psychological stability is associated with better surgical outcomes and this may be true for interventional pain procedures as well even though they are minimally invasive.

Smoking cessation should be encouraged in all patients as smoking is associated with back pain and degenerative disk disease. Diabetes is a risk factor due to the glycemic effect of corticosteroids. If patients have a history of hospitalization for diabetes, they may be poor candidates for the lysis procedure. However, it is possible to omit the corticosteroid from the procedure if necessary. Litigation or disability dispute can be a risk factor for poor outcomes. Any secondary gain issue can reduce patient's response to treatment.

Informed consent should be obtained before the patient is distracted by the preparation for the procedure. It should not be a rubber stamp process. Complications occur despite following proper procedure and patients need to understand this prior to the procedure. Infection is prevented by a surgical sterile prep. Sterile field preparation with chlorhexidine followed by alcohol is one recommended antiseptic. Caudal lysis procedures are the most concerning due to the proximity to the anus. A sterile field must be maintained during the procedure. A trigger point injection or peripheral nerve block may be performed with a simple prep but not a spine

procedure. Using prophylactic antibiotics consistent with the institutional policy for neurosurgical procedures is a good practice. Strict sterile technique is used and no on site sterilization of contaminated needles or catheters is allowed if they are dropped on the floor for example. A new needle or catheter should be used. Topical antibiotics are used at the puncture site. The catheter is tunneled in the caudal area and this provides more safety from spinal infection. If a catheter site develops redness or drainage or other signs of inflammation, it can be removed prematurely to prevent infection. Topical antibiotics are used at the catheter site. Oral antibiotics are used post procedure. The puncture site should be monitored for any redness, swelling, drainage or tenderness.

Epidural hematoma is a surgical emergency and an emergent MRI and spine surgeon consultation should be obtained within minutes, not hours. Patients with severe pain following a spine procedure with or without neurological deficits should be imaged emergently to rule out a hematoma. Patients with arteriovenous malformations (AVM) are at increased risk for hematoma formation. The pre procedure MRI findings may be subtle and noticed only after further review following a hematoma occurrence. Tortuosity of veins in the spinal canal may signify an AVM.

Arachnoiditis is a possible complication from epidural steroid injections. Taking measures to avoid subdural and intrathecal injections can reduce the possibility of arachnoiditis. However, patients who have had surgery with postoperative infection or hematoma may develop arachnoiditis that has a subtle appearance on imaging studies. This condition may worsen over time and seem to be related to an injection when in fact it is not.

Only myelogram grade contrast should be used for the procedure and limit dose to 300 mg/ml or 3060 mg iodine per single procedure of isohexol. For example, 17 ml is the upper limit for lumbar myelography. Iopamidol (Isovue-M 200) is limited to 15 ml for lumbar myelography. Renal toxicity is a limitation and patients with pre-existing renal disease should be given much smaller doses. CSF lavage has been recommended for intrathecal injection of contrast that is not myelogram grade contrast [32].

Many patients report a history of allergy to contrast. It is important to explore the allergy symptoms with the patient. Sometimes the symptoms experienced by the patient's where not allergy symptoms. Pretreatment with an H2 blocker, antihistamine and steroid is sometimes done to prevent allergy to contrast but it may be best to avoid the procedure if true allergy exists. Steroids may contain preservatives that are toxic. Alcohol free preparations are safe and are often diluted with normal saline to reduce the concentration of any additives. Particulate steroids are thought to be longer lasting but have been implicated in complications from transforaminal epidural injections. The theory is that particles block small arterioles if inadvertently injected into as segmental artery or vertebral artery.

Local anesthetics are toxic if injected intravascularly. Intralipid should be available in case of intravenous local anesthetic toxicity. Local anesthetic has the potential to produce a total spinal block associated with respiratory failure and shock. To prevent this, needles and catheters should be aspirated prior to injecting any drug. If blood or cerebrospinal fluid is aspirated, the needle or catheter needs to be

repositioned or removed. Local anesthetic can also produce a sympathetic block causing hypotension and shock.

Resuscitation personnel and equipment are required to be on hand when local anesthetic is administered in the spine. Respiratory failure from a block of the cervical levels and phrenic nerves is possible and personnel and equipment for airway management and ventilation is also required. Most of these procedures are performed in a licensed outpatient facility or hospital for these reasons. However, an anesthesiologist with the appropriate equipment and staff can safely perform these procedures in an advance clinic setting. Monitoring EKG for preexisting arrhythmias such as atrial fibrillation is important in case a patient develops a rapid ventricular response during the procedure. Patients should be monitored before any sedation is administered. EKG, pulse oximetry blood pressure and continuous observation by a trained nurse are basic monitoring needs. Intravenous access is needed for sedation and in case a vasovagal reaction occurs. Sedation may include small doses of midazolam and fentanyl. Vasovagal reactions are most common in young and middle aged males. Heart rate variability is a common antecedent. Glycopyrrolate 0.1 mg may be given to prevent a severe episode and repeated twice as needed. Severe vasovagal reactions are treated with atropine 0.5 mg intravenously. Patients having procedures in the afternoon may have been NPO for 24 hours and need IV fluid to help prevent severe vasovagal reactions. Patients should be monitored following procedures for vital signs, neurological symptoms and a physician should be available to evaluate unexpected numbness, weakness or other problems. General anesthesia is used for many interventional procedures but this practice is now discouraged by new guidelines [33].

General anesthesia is not indicated for lysis of adhesions but intravenous sedation is required.

Positioning is important to maximize the quality of the fluoroscopy, ease and safety of the procedure. The position for the caudal approach is with the patient prone with the hips on a pillow and legs internally rotated. Standard anterior-posterior and lateral images are usually adequate to perform the procedure. Sacral foraminal catheterization requires similar patient positioning but the fluoroscopy angle is slightly oblique to the ipsilateral side with some cranial tilt to visualize the posterior foramen. The anterior foramen may be seen as well and may confuse the imaging process. The lumbar transforaminal approach is performed with the patient in the prone position with a pillow under the lower abdomen. A slight oblique fluoroscopic image to the ipsilateral side is used for the approach. The cervical approach is with the patient in the prone position with a pillow under the chest and the neck flexed. These positioning steps facilitate the procedure and enhance the safety by improving image quality.

Patients, doctors, nurses and x-ray technicians should be shielded from radiation to the extent possible. The time of fluoroscopy for each procedure should be limited. The distance from the x-ray generator to the patient should be maximized. An x-ray table should be used for the procedure to maximize image quality. An x-ray technician should be available to operate the C-arm if necessary. Collimation is important to improve image quality. The physician should practice good radiation safety by

minimizing the time of exposure, increasing the distance between themselves and the radiation and finally by sophisticated shielding. One of the important views with fluoroscopy is the lateral view because it shows depth. This is especially true in the cervical levels. Oblique views have been used to improve image quality but image quality can be improved by positioning, collimation and boosting Kv. If patients are too large to be safely imaged, then they are too large for the procedure.

All drugs should be labeled in their syringes. Second and third injections in the series may be delegated to a trained nurse as long as the physician is in the building and immediately available. A test dose of local anesthetic is given prior to a therapeutic dose. If after a test dose of 3 ml of local anesthetic, no spinal block occurs after several minutes, an intrathecal injection is unlikely to have occurred. However, needles and catheters can move and puncture the dura and the dura mater can tear during the procedure. Constant monitoring and vigilance are necessary to recognize any signs of an intrathecal or subdural local anesthetic block that will require resuscitation. Incremental injections of local anesthetic rather than large single bolus injections are advised when using local anesthetic. Hyaluronidase allergy can occur and should be treated with diphenhydramine 12.5–25 mg intravenously and repeated as needed. Cortiocosteroid may also be given intravenously. Epinephrine 1 microgram (triple diluted 1 mg ampule in 10 ml syringe, 1 ml) may be used intravenously for severe reactions and this may be repeated while monitoring heart rate to avoid tachyarrhythmias.

Hypertonic saline is painful if injected into anaesthetized areas. Injection should be interrupted if pain develops. Additional local anesthetic should be administered prior to resuming the dose of hypertonic saline. Hypertonic saline 10% is tolerated in the epidural space but it is critical to administer local anesthetic at least 20 minutes before injecting hypertonic saline. A subdural block must be ruled out before hypertonic saline is used to prevent potentially serious neurological damage. Also, hypertonic saline injection is associated with severe pain that must be blocked by prior local anesthetic. Catheter placement and epidural contrast spread are not sufficient to ensure exclusive epidural placement of injectate.

Hypertonic saline is avoided in patients with multiple sclerosis or demyelinating diseases. Hypertonic saline injection should be aborted if patients experience severe pain, muscle cramps, hypertension, or arrhythmias. Intraneural injection can occur especially at the cervical and thoracic levels where the spinal cord is present. The spinal cord moves anteriorly in the prone position, creating some degree of safety but the cord does not move anteriorly as much as the lumbosacral nerve roots.

Preventing spinal cord puncture is prevented by using a straight lateral fluoroscopic image to help control needle depth. Using a modified Bromage grip helps to fix the needle, patient and physician's hand into one unit that moves together in the event that the patient jumps or moves suddenly. Holding the needle and syringe as if administering an intramuscular injection is avoided because if the patient raises from the prone position, the needle will be advanced deeper in an uncontrolled fashion. S5 nerve injury can occur with sacral canal penetration. This can be prevented by avoiding the extreme lateral area of the sacral hiatus and entering the sacral canal between the midline and the lateral one third of the foramen. Also, the catheter

placement can be verified with fluoroscopy and contrast injection. Venous runoff can be seen if contrast is injected under live fluoroscopy if the catheter is placed in a vein. Contrast should pool in the area of injection and not dissipate on a subsequent fluoroscopic image. Sheared catheters are caused by withdrawing catheters in order to reposition them near the target. This can be prevented by maintaining the needle opening in an orientation toward the target. Also the catheter should be withdrawn slowly and smoothly before redirecting. If the catheter meets resistance it can be advanced and rotated before attempting to withdraw again. If the catheter cannot be withdrawn past a point of resistance, the needle and can be withdrawn together as an intact unit. If the needle is removed but the catheter remains stuck, a needle can be replaced over the catheter and removal can be attempted again. Discharge criteria include independent ambulation and urinary voiding.

Complications can be avoided by using these steps:

A Loss of resistance technique using a modified bromage grip to prevent uncontrolled penetration.
Saline and air in LOR syringe to avoid pneumocephalus.
Abort the procedure if a wet tap is encountered
Using a blunt stylet to avoid dural laceration or puncture
Using an off midline technique to avoid loculation
Rotate RK needle tip toward target to avoid shearing if catheter is withdrawn to reposition
Check contrast in AP and lateral views to ensure epidural placement
Perform an aspiration test to avoid intravascular or intrathecal injection
Administer a test dose of local anesthetic to rule out a subdural catheter placement
Use local anesthetic to provide analgesia before administering hypertonic saline
Recognize perivenous counter spread and treat with flexion rotation exercises
Use flexion rotation exercises in patient who develop pain during or after injections to promote decompression by allowing injected fluid to exit the spinal canal via neural foramina

Outcome Studies and Conclusions

Selective nerve root blocks have been shown to reduce surgical rates in the short to intermediate term [34]. However, over time, the surgical rates between the selective nerve root block group and control converge [35]. No other single shot percutaneous procedure has shown better results with respect to surgical rates. Lysis of epidural adhesions is more involved and more expensive than a single shot procedure but it is also more effective and can reduce surgical rates long term. Caudal epidural lysis of adhesions had been studied in a randomized, sham controlled trial and the results show significant improvement of pain and function in the active treatment group compared to the sham group [36]. Long-term follow up of lumbar epidural lysis of adhesions shows maintenance of the analgesic effect and functional improvement 10 years after the procedure [37]. No long term problems were observed.

In a study of cervical percutaneous neuroplasty versus epidural steroid injections, the cervical percutaneous epidural neuroplasty group had better outcomes 6 months after treatment for cervical disk disease. [38] Importantly, contrast runoff through neural foramen correlates with improved outcome [39]. Achieving lateral runoff should be a goal of the procedure.

A systematic review and meta-analysis reported level 1 evidence for this procedure in the treatment of back and leg pain due to lumbar spine disease [40]. Finally, for patients with failed back surgery syndrome, epidural adhesiolysis is more effective than spinal cord stimulation which is more effective than epidural injections [41].

References

1. Kuslich SD, Ulstrom CL, Michael CJ. The tissue origin of low back pain and sciatica: a report of pain response to tissue stimulation during operations on the lumbar spine using local anesthesia. Orthop Clin North Am. 1991;22(2):181–7. PMID: 1826546.
2. Racz GB, Racz GJ, Racz TA. Interventional pain procedures. In: Noe C, editor. Pain management for clinicians: a guide to assessment and treatment. Springer; 2020. p. 523–654.
3. Oh CH, Ji GY, Cho PG, Choi WS, Shin DA, Kim KN, Kang HA. The catheter tip position and effects of percutaneous epidural neuroplasty in patients with lumbar disc disease during 6-months of follow-up. Pain Physician. 2014;17(5):E599–608. PMID: 25247909.
4. Cho PG, Ji GY, Yoon YS, Shin DA. Clinical effectiveness of percutaneous epidural neuroplasty according to type of single level lumbar disk herniation: a 12 month follow up study. J Korean Neurosurg. 2019;62:681.
5. Chun EH, Park HS. Effect of high-volume injectate in lumbar transforaminal epidural steroid injections: a randomized, active control tial. Pain Physician. 2015;18(6):519–25. PMID: 26606003.
6. Choi EJ, Yoo YJ, Lee PB, Kim YC, Lee SC, Moon JY. A retrospective study to evaluate the effect of concentration of hypertonic saline on efficacy and safety of epidural adhesiolysis. Anesth Analg. 2017;124(6):2021–9. https://doi.org/10.1213/ANE.0000000000001925. PMID: 28448392.
7. Racz GB, Heavner JE, Singleton W, Carline M. Hypertonic saline and corticosteroid injected epidurally for pain control. In: Racz GB, editor. Techniques of neurolysis. Klewer Academic Publisher; 1989. p. 73–86.
8. Hitchcock E. Hypothermic subarachnoid irrigation for intractable pain. Lancet. 1967;1(7500):1133–5.
9. Hitchcock E, Prandini MN. Hypertonic saline in management of intractable pain. Lancet. 1973;1:310–2.
10. Birkenmaier C, Redeker J, Sievers B, Melcher C, Jansson V, Mayer-Wagner S. An evaluation of medications commonly used for epidural neurolysis procedures in a human fibroblast cell culture model. Reg Anesth Pain Med. 2011;36:140–4.
11. Heavner J, Racz G, Raj P. Percutaneous epidural neuroplasty: prospective evaluation of 0.9% saline versus 10% saline with or without hyaluronidase. Reg Anesth Pain Med. 1999;24:202–7.
12. Arthur J, Racz G, Heinrich R, et al. Epidural space. Identification of filling defects and lysis of adhesions in the treatment of chronic painful conditions. In: 7th Word Congress on Pain. Paris, IASP Publications; 1993. p. 557. Abstracts.
13. Racz G, Day M, Heavner J, et al. Hyaluronidase: a review of approved formulations, indications and off-label use in chronic pain management. Expert Opin Biol Ther. 2010;10(1):127–31. 330 Pain and Treatment.
14. Hsu E, Atanelov L, Plinkett A, Chai N, Chen Y, Cohen SP. Epidural lysis of adhesions for failed back surgery and spinal stenosis: Factors associated with treatment outcome. Anesth Analg. 2014;118:215–24.

15. Bogduk N, Aprill C, Derby R. Lumbar discogenic pain: state-of-the-art review. Pain Med. 2013;14(6):813–36. https://doi.org/10.1111/pme.12082. Epub 2013 Apr 8. PMID: 23566298.
16. Racz GB, Heavner JE, Noe CE, Al-Kaisy A, Matsumoto T, Lee SC, Nagy L. Epidural lysis of adhesions and percutaneous neuroplasty. In: Racz GB, Noe CE, editors. Techniques of neurolysis. 2nd ed. Springer; 2016. p. 119–43. Copyright Holder Springer International Publishing Switzerland, eBook ISBN 978-3-319-27607-6. https://doi.org/10.1007/978-3-319-27607-6. Hardcover ISBN 978-3-319-27605-2.
17. Gabor Racz and James Heavner in Techniques of Neurolysis Edited by Gabor B. Racz and Carl E. Noe Copyright 2016 Publisher Springer International Publishing Copyright Holder Springer International Publishing Switzerland. eBook ISBN 978-3-319-27607-6. https://doi.org/10.1007/978-3-319-27607-6. Hardcover ISBN 978-3-319-27605-2 Edition Number 2, p. 1–12.
18. Teske W, Zirke S, Nottenkamper J, Lichtinger T, Theodoridis T, Kramer J, Schmidt K. Anatomical and surgical study of volume determination of the anterolateral epidural space nerve root L5/S1 under the aspect of epidural perineural injection in minimal invasive treatment of lumbar nerve root compression. Eur Spine J. 2011;20(4):537–41.
19. Matsumoto. Treatment of lower back and leg pain using the Racz Catheter-Matsu- moto way via S1 foramen. Maastricht: WIP World Congress; 2014.
20. Kitogana T, Fujiwara A, Kobayashi N, Saiki K, Tamai K, Saotome K. Morphologic changes in the cervical neural foramen due to flexion and extension (in vitro imaging study). Spine. 2004;29:2821–5.
21. Clemens HJ. Die Venensysteme Der Menschlichen Wirbelsaule. Berlin: Walter de Gruyter & Co; 1961.
22. Racz GB, Heavner JE. Introduction. In: Racz GB, Noe CE, editors. Techniques of neurolysis. 2nd ed. Springer; 2016. p. 1–12. Copyright holder Springer International Publishing Switzerland. eBook ISBN 978-3-319-27607-6. https://doi.org/10.1007/978-3-319-27607-6. Hardcover ISBN 978-3-319-27605-2.
23. Veihelmann A, Devens C, Trouiller H, et al. Epidural neuroplasty versus physio- therapy to relieve pain in patients with sciatica: a prospective randomized blinded clinical trial. J Orthop Sci. 2006;11(4):365–9.
24. Indahl A, Kailgle A, Reieras O, Holm S. Sacroiliac joint involvement in activation of the porcine spinal and gluteal musculature. J Spinal Disord. 1999;12(4):325–30.
25. Scanlon GC, Moeller-Betram T, Romanowsky SM, Wallace MS. Cervical transforaminal epidural steroid injections-more dangerous than we think? Spine. 32(11):1249–56.
26. Racz GB, Heavner JE. Cervical spinal canal loculation and secondary ischemic cord injury-PVCS-Perivenous Counter Spread-Danger Sign! Pain Pract. 2008;8(5):399–403.
27. Racz GB, Apicella E, Vohra P. Collegial communication and problem solving: intraspinal canal manipulation. Pain Pract. 2013;13(8):667–70.
28. Moore DC. The use of hyaluronidase in local and nerve block analgesia other than spinal block: 1520 cases. Anesthesiology. 1951;12(5):611–26. https://doi.org/10.1097/00000542-195109000-00009. PMID: 14868898.
29. Abram SE, O'Connor TC. Complications associated with epidural steroid injections. Reg Anesth. 1996;21(2):149–62. ISSN: 0146-521X.
30. Rocco AG, Philip JH, Boas RA, et al. Epidural space as a starling resistor and elevation of inflow resistance in a diseased epidural space. Reg Anesth. 1997;22:167–77.
31. Swerdlow M. Complications of neurolytic neural blockade. In: Cousins MJ, Bridenbaugh PO, editors. Neural blockade. Philadelphia, PA: Lippincott; 1980. p. 543–53.
32. Racz GB, Heavner JE. Complications associated with lysis of epidural adhesions and epiduroscopy. In: Rathmell J, editor. Complications in regional anesthesia & pain medicine. Elsevier; 2007. p. 301–11.
33. Van Boxem K, Rijsdijk M, Hans G, de Jong J, Kallewaard JW, Vissers K, van Kleef M, Rathmell JP, Van Zundert J. Safe use of epidural corticosteroid injections: recommendations of the WIP Benelux work group. Pain Pract. 2019;19:61–92. https://doi.org/10.1111/papr.12709.

34. Riew KD, Yin Y, Gilula L, Bridwell KH, Lenke LG, Lauryssen C, Goette K. The effect of nerve-root injections on the need for operative treatment of lumbar radicular pain. A prospective, randomized, controlled, double-blind study. J Bone Joint Surg Am. 2000;82-A(11):1589–93.
35. Daniel Riew K, Park J-B, Cho Y-S, Gilula L, Patel A, Lenke LG, Keith H. Bridwell nerve root blocks in the treatment of lumbar radicular pain. A minimum five-year follow-up. J Bone Joint Surg Am. 2006;88:1722–5. https://doi.org/10.2106/JBJS.E.00278.
36. Gerdesmeyer L, Wagenpfeil S, Birkenmaier C, Veihelmann A, Hauschild M, Wagner K, Muderis MA, Gollwitzer H, Diehl P, Toepfer A. Percutaneous epidural lysis of adhesions in chronic lumbar radicular pain: a randomized, double-blind, placebo-controlled trial. Pain Physician. 2013;16(3):185–96.
37. Epidural Neuroplasty – 10 year follow-up Ludger Gerdesmeyer, MD, PhD, FIPP. WIP 23rd Annual Budapest Conference & Workshop. August 27, 2018 - August 29, 2018.
38. Ji GY, Oh CH, Won KS, Han IB, Ha Y, Shin DA, Kim KN. Randomized controlled study of percutaneous epidural neuroplasty using Racz catheter and epidural steroid injection in cervical disc disease. Pain Physician. 2016 Feb.;19(2):39–48.
39. Contrast Runoff Correlates with the Clinical Outcome of Cervical Epidural Neuroplasty Using a Racz Catheter. Yun-Jong Han, MD, Myoung No Lee, MD, Min Ji Cho, MD, Hue Jung Park, MD, PhD, Dong Eon Moon, MD, PhD, and Young Hoon Kim, MD, PhD. Pain Physician 2016; 19: E1035-E1040 • ISSN 2150–1149.
40. Helm S 2nd, Racz GB, Gerdesmeyer L, et al. Percutaneous and endoscopic Adhesiolysis in managing low Back and lower extremity pain: a systematic review and meta-analysis. Pain Physician. 2016;19(2):E245–82.
41. Cho JH, Lee JH, Song KS, Hong J-Y, Lee D-H, Hwang CJ, Lee CS. Treatment outcomes for patients with failed back surgery. Pain Physician. 2017;20:E29–43.
42. Manchikanti L, et al. Cost utility analysis of percutaneous adhesiolysis in managing pain of post-lumbar surgery syndrome and lumbar central spinal stenosis. Pain Practice. 2015;15(5):414–22.
43. Moon DE, Park HJ, Kim YH. Assessment of clinical outcomes of cervical epidural neuroplasty using a Racz-Catheter and predictive factors of efficacy in patients with cervical spinal pain. Pain Physician. 2015;18:E163–70.

Pre-surgical Psychological Evaluation of Spine Surgery Candidates

13

Jeffrey Dodds

Introduction

Successful spine surgery and recovery requires a collaborative effort from patients, surgeons, healthcare teams, and insurance companies. There is a growing body of evidence that psychological and psychosocial factors can strongly influence the effectiveness of spine surgery and affect long-term outcomes after surgery [11, 13, 16, 20, 24]. Many medical treatment guidelines, including those of the International Society for Advancement of Spine Surgery, North American Spine Society, American College of Occupational and Environmental Medicine and the American College of Physicians, now contain specific recommendations for pre-surgical psychological evaluations [14]. Many payer policy statements, including those of the Centers for Medicare and Medicaid Services, Bluecross, Cigna, and United Healthcare, have adopted these recommendations as well and require pre-surgical psychological evaluations for spinal surgeries. These evaluations not only provide reliable, evidence-based recommendations to assist in the development of appropriate treatment plans for spine surgery patients, they also can be used to determine patients' outcome prognosis [7, 8]. Studies have shown that pre-surgical psychological evaluations are better predictors of spinal surgery outcome than MRIs [22, 23, 34]. The predictive power of these evaluations lies in the psychological and psychosocial information collected in the structured clinical interview, psychometric testing, and medical chart review [8, 9, 24, 30, 32].

J. Dodds (✉)
Division of Psychology, Department of Psychiatry, UT Southwestern Medical Center, Dallas, TX, USA
e-mail: Jeffrey.Dodds@UTSouthwestern.edu

© The Author(s), under exclusive license to Springer Nature Switzerland AG 2022

C. E. Noe (ed.), *Multidisciplinary Spine Care*,
https://doi.org/10.1007/978-3-031-04990-3_13

Guidelines for Presurgical Psychological Evaluations

Broadly speaking, there is no one standard presurgical psychological evaluation for spinal surgery candidates. However, there are extensive commonalities between the various approaches recommended by leading researchers in the field, including Andrew Block, JJ den Boer, and Daniel Bruns, just to name a few. The vast contributions to the guidelines for these evaluations made by these and other researchers over the past 25 years have resulted in what [14] has referred to as a converging set of criteria for the evaluation of candidates for invasive spine surgery. As a result, multiple states have now adopted specific guidelines and requirements for PPE. Colorado, Connecticut, Delaware, Montana, New York, Rhode Island, and Washington, are among the growing list of states whose medical treatment guidelines now require a pre-surgical psychological evaluation for spinal procedures [18]. Additionally, a carefully constructed guideline for presurgical psychological evaluations has been developed by the US Preventative Services Task Force [41].

Ultimately, the goals of presurgical evaluation are to optimize surgical outcomes and optimize patient satisfaction. The surgical outcomes can be assessed in a relatively straightforward manner with objective and biological measures. However, when measuring patient satisfaction, assessment must rely on subjective and behavioral variables [14]. These variables include the patient's reported pain level, satisfaction with care, reported level of functioning, reported/perceived quality of life, and ability and willingness to reduce opioid use. Subsequently, when we are defining surgical success, most surgical outcome variables have been found to be subjective and behavioral rather than biological and objective [21]. Of course, there are examples of surgeries largely based on biological variables and determined by biological criteria, such a surgical intervention for cauda equina. Yet in many cases the decision to perform surgery will be based on a number of behavioral variables and then the surgical outcome, in terms of success or failure, will be determined by the patient-reported behavioral variables noted above. Under these circumstances, many spine surgeries could be considered a behavioral treatment as much as a biological/medical treatment. This creates the possibility of an outcome paradox in that a procedure could be biologically successful but behaviorally a failure or vice versa [21]. So to the extent that a particular spine surgery is being performed as a behavioral intervention to the patient's satisfaction, behavioral tests actually have been found to be better outcome predictors than biological tests [22, 23]. As surgical outcomes are considered from this biopsychosocial perspective, the necessity of a collaborative evaluative process that includes both medical and psychological opinions becomes clear.

Domains of the Presurgical Psychological Evaluation

The clinical literature on presurgical psychological assessment consistently recommends that psychosocial and medical risk factors be assessed through multiple techniques. These techniques include, but are not limited to behavioral observation, a structured clinical interview, comprehensive review of the patient's medical records,

and administration of standardized psychometric assessments [7, 8, 14, 16, 26, 30]. Careful consideration and integration of the information gathered from these techniques and other sources allows the examiner to reach 4 very specific goals [8]. A primary goal of the evaluation process is to improve treatment outcomes for patients by identifying and, if necessary, arranging treatment for emotional and behavioral problems that could impede their recovery. The examiner can also identify patients who are likely to develop medication or compliance problems and recommend appropriate intervention. Another goal of the evaluative process is to improve overall treatment outcome by screening out patients with strong potential to experience poor outcome. Lastly, by adhering to this comprehensive process, the examiner can provide a strong and empirically validated rationale in cases where the surgeon may already feel uncomfortable about operating.

In the course of completing the medical chart review, structured clinical interview, and psychometric testing, the examiner will be looking for strengths as well as weaknesses on all three domains. The examiner will also be looking for both consistencies and inconsistencies in reporting, and attempting to clarify any identified inconsistencies as deemed necessary.

Medical Chart Review

The pre-surgical psychological assessment generally begins with a thorough medical chart review as this will establish the foundation for all other aspects of the assessment. In addition to providing basic information on the patient's medical status, the chart review can offer insight into other complex aspects of the patient's history that will be important to consider when determining their readiness for surgery. Behavioral risk factors such as medical compliance problems, current and past substance use history, medication seeking behavior, and psychiatric history all should be noted and then explored in detail during the clinical interview. Other more subtle behavioral risk factors indicative of recovery disincentive may be found during the chart review as well, including reports of excessive pain behaviors or disability seeking behaviors. The presence or absence of these types of risk factors should not be considered exclusionary factors in and of themselves. Rather, they should be considered in an overall assessment of the patient's ability to achieve pain relief and lifestyle improvement from a surgical procedure.

Equally worthy of consideration during the chart review are patients' strengths, those behavioral and biopsychosocial factors known to promote recovery. Block et al. [12] has identified a number of factors that may outweigh other risk factors identified in the evaluative process such as patient resilience, adaptive emotional responses, work ethic, avoidance of opioids, and most importantly, patient activation. Patient activation, defined as patients' engagement in maintaining their own health, may be determined by a history of such things as engaging in regular exercise, maintaining a healthy diet, and collaborating with their physicians on treatment plans [38]. Patient resilience may be evidenced by their reported ability to handle stress and "bounce back" after other challenges they may have faced. Other important strengths to consider in the evaluative process include evidence of

previous adaptive emotional responses to medical problems or significant stressors that may have occurred in their past.

Structured Clinical Interview

The clinical interview process provides means for identifying many of the risk factors associated with poor surgical outcomes as well as identifying strengths that may outweigh these risk factors. The clinical interview also provides opportunities to make more accurate interpretations of patients' distress and mood-related symptoms that may have been noted in their medical records or endorsed on psychological testing. Thoughtful follow-up questions in the clinical interview can help clarify if these symptoms are indicative of genuine psychiatric problems or symptoms directly related to their current physical condition.

Discussion of the location, severity, and chronicity of the pain the patient is experiencing is another important aspect of the interview process. This will provide the examiner with insight into the ways patients perceive and cope with pain, their opinions on the effectiveness of previous treatment for pain, and expectations for post-surgical results. In the course of this discussion, the examiner will also have the opportunity to ask about more general healthcare utilization, medical/surgical history, current medications, opioid use/abuse, and the adequacy of their social support [37]. There are three key aspects to conducting a successful clinical interview. First, it is important to normalize the evaluation process for patients by helping them understand it is a routine part of the work-up for surgery and not an implication that they were singled out for an assessment based on concerns that they have significant mental health issues [3]. It is not uncommon for patients to be somewhat suspicious of the evaluation process initially and view the psychological examiner as a gatekeeper in their journey to obtaining relief from chronic pain. Subsequently, a vitally important aspect of the interview process is establishing rapport with patients and helping them understand that the evaluation is being conducted with the goal of developing the best possible overall treatment plan for them. It is also important to structure the interview in a manner that will garner the information needed to make efficacious recommendations while still allowing for a certain level of flexibility in the process to provide patients with opportunities to discuss issues important to them [4]. Block and colleagues have stressed that when performing the clinical interview, a significant portion of the evaluation should be devoted to exploring and assessing the patients' views of their condition and the extent to which they believe the surgery will relieve their symptoms. Patients who express beliefs that they are unable to effectively change their conditions have been shown to show less improvement from spine surgery [4, 11]. That said, the examiner must be wary of over-pathologizing patients' worried or anxious presentations as this may be reflective of completely reasonable and understandable concerns about both medical and psychosocial aspects of the procedure. In addition to these entirely appropriate concerns about the procedure and post-procedural recovery, patients may have worries

about such things as the financial aspects of missed work or the possibility of overburdening members of their support system.

Psychometric Testing

An ambitious review of 125 meta-analyses concluded that psychometric tests can diagnose and predict outcomes as well as medical tests within their specific domains [34]. Other studies specifically focused on lumbar surgery outcomes found that psychological tests are actually better than MRI and other medical variables at predicting outcomes [2, 22, 23]. Psychometric testing can effectively identify several common risk factors for surgery while also providing the examiner with a relatively quick and concise picture of the patient's personality and emotional state [3]. Taken together, this data can be utilized as an additional check on the examer's perceptions of the patient from the clinical interview. Test results can also be used to predict treatment outcomes and tailor treatment recommendations to each individual [17, 42].

There is no consensus on the particular testing instruments to include in a pre-surgical assessment battery but any tests utilized must be peer-reviewed and have appropriate reliability, validity, and normative data. They also must specifically assess constructs relevant to selection criteria for the surgical candidates in this particular clinical setting [6]. Multiple studies support the inclusion of a standardized psychological assessment instrument such as the MMPI2-RF in the psychological evaluation of candidates for spine surgery and spinal cord stimulator implantation [5, 10–12, 33]. Extensive research on this particular testing instrument has consistently shown that patients with unresolved interpersonal problems, poor ability to cope with pain, high sensitivity to pain, or beliefs that they cannot get better, are far more likely to experience sub-optimal surgical outcomes [6, 12, 33, 38] Similarly, a comprehensive review of the literature conducted by Celestin et al. [24] found that pre-surgical somatization, depression, anxiety, and poor coping were predictive of poor outcome response to lumbar surgery and spinal cord stimulator implantation. Other important psychosocial risk factors to consider include the presence of fear/avoidance of injury, substance abuse history, and disincentives for recovery [31].

When administering psychological testing in this pre-surgical assessment setting, patients' underreporting on an instrument should not be interpreted in the same manner as it would be in other settings. Patients' under-reporting is not necessarily indicative of defensiveness, lying, or invalidity [6]. Both the examiner and the process of the exam itself may be viewed by patients as barriers to surgery rather than an element of integrated care. Additionally, when assessing medical patients with psychological invententories the examiner must be mindful of the tendencies for these inventories to score all physical symptoms as psychological syndromes [15]. So even in the testing domain of pre-surgical assessment, it is important to identify not only patients' risk factors but their protective factors as well.

As noted earlier, one of the most prominent protective factors is patient activation, a factor shown to be reliably measured by both the MMPI-2-RF and the Patient Activation Measure (PAM) [5, 6, 12] Patient activation has been shown to be associated with positive surgical outcomes in spite of the presence of other psychosocial risk factors [5]. Highly activated individuals experience better health outcomes than individuals who are less activated [44, 45]. More highly activated individuals also have been shown to report having more psychological resources when faced with adversity such as recovery from spine surgery [39].

Integration of Evaluation Data

There are multiple and sometimes complex considerations to be made when integrating the information collected during the presurgical evaluation. One important consideration is the possibility that patients suffering with chronic and severe pain may define their problems narrowly and either by choice or lack of insight, fail to recognize the role that mood symptoms and psychosocial factors are playing in their responses to treatment [37]. Frequently, patients will acknowledge the presence of depression and/or anxiety while expressing the belief that these mood symptoms are directly and solely related to the presence of chronic pain. While there certainly may be an element of truth in this belief, it is crucial that the examiner take time to consider the psychosocial and psychological factors that the patients themselves may not have taken into consideration. Multiple studies have shown that the presence of psychiatric conditions or history of psychological trauma can profoundly impact patients' perception of pain and ability to cope with pain [5, 11, 24, 28, 35, 43]. Studies have also shown that patients who somaticize or have increased sensitivity to pain tend not to do well after surgery [6, 12, 33].

A literature review conducted by Bruns and Disorbio [16] supports the utilization of a two-tiered approach to presurgical psychological evaluation that includes assessment of both primary and secondary psychosocial risk factors. The presence of primary psychosocial risk factors requires immediate intervention including but not limited to postponement or cancellation of the surgical candidate's procedure. Primary risk factors include suicidal ideation, homicidal ideation, psychosis, and acute intoxication. Psychological testing results often will provide reliable evidence of more serious psychopathology even when patients may have made efforts to deny or conceal them in visits with the surgeon or the pre-surgical examiner. Surgical candidates suffering with this level of psychological instability may be unable to cooperate and/or fully comply with the treatment recommendations of the surgical team. Under these circumstances, a feedback session will be required to provide the patient with recommendations for alternative noninvasive treatments such as a multidisciplinary chronic pain management program. The feedback session also will provide the examiner an opportunity to describe the significant risks of the more invasive procedures and explain to patients how these type procedures may actually worsen their pain and functional disability [4].

Secondary psychosocial risks are fairly common among candidates for elective surgery and include depression, anxiety, somatization, job satisfaction, and smoking. The presence of any of these secondary risk factors should not necessarily be viewed as exclusionary. However, research has consistently shown that as the number of secondary risk factors increases, so does the likelihood of poor prognosis. For spine patients in particular, the presence of depression and anxiety have been associated with impairment of health-related quality of life and poor long-term outcomes, such as higher levels of increased pain and perceived disability [1, 27, 36, 40]. Patients endorsing depression, smoking, and/or receiving disability benefits at the time of lumbar spine surgery have been found to have worse postoperative functional outcomes at 3 and 12 months after surgery. The presence of these three factors pre-surgically also have been associated with lower patient satisfaction after lumbar surgery [25]. Gatchel [29] found that the presence of 4 or more of these secondary psychosocial risk factors significantly increased the presence of psychopathology and doubled the odds that patients would not return to work after medical treatment.

When taking these secondary risk factors into account, the examiner must also consider to what degree socio-economic factors may be affecting patients' perception of their level of recovery and/or level of disability following surgery. For instance, a patient with a relatively low education level, limited job skills, and/or labor intensive occupation, may quite realistically fear their back problems will be permanently disabling to some degree even with successful surgery. Conversely, a patient with many transferable job skills and an advanced degree may view their back problems and pain issues as significant challenges but without the fear of an inability to maintain gainful employment. Information gathered in the clinical interview regarding the patient's educational background and work history can be utilized to better understand their perceptions of disability [37]. In most cases where a relatively low number secondary risk factors have been identified in the presence of protective factors such as high patient activation and resilience, patients can still be considered low risk and cleared psychiatrically for surgery. In cases where the risk factors have reached a moderate level, surgery can be delayed while patients participate in appropriate behavioral treatments such as smoking cessation, CBT for Chronic Pain, physical exercise programs, and medication reduction [8].

As with any psychological evaluation, there are multiple situational factors that must be taken into consideration in determining surgical candidates' readiness for surgery. Cultural issues, language barriers, and literacy issues may significantly impact the ways in which patients present and perceive their symptoms. Other formative experiences including those involving patients' age, sexuality, or race are also important details to explore in the evaluative process.

Psychological Clearance/Recommendations

Obtaining successful outcomes for spine patients requires a collaborative effort from patients, surgeons, healthcare teams, and insurance companies. Best practice guidelines suggest an interdisciplinary approach that will provide patients treatment

options from multiple disciplines for which there is good evidence of high efficacy, low risk, and low burden [14, 19]. Doing what is best for the patient means considering options from the least invasive interventions such as medication, physical therapies, and behavioral medicine to the more invasive options such as injections and surgery. It is at this stage of the treatment planning that the pre-surgical examiner's recommendations for pre-surgical treatment becomes vitally important. The recommendations included in the pre-surgical evaluation in conjunction with the patient's willingness and capacity to comply with these recommendations may determine if an elective surgery will ever be performed.

Determining the impact biopsychosocial factors could have on the surgical outcome and determining what if any, interventions might improve the surgical results are among the most important decisions made during the pre-surgical evaluation process. Careful integration and consideration of all the uniquely individual results of a pre-surgical evaluation allows the examiner to develop meaningful treatment recommendations for patients at any risk level [47]. Patients determined to be low to moderate risk surgical candidates can be provided with recommendations for facilitating in their surgical outcomes. And higher risk candidates who are not cleared psychiatrically for a surgical procedure can be provided with individually tailored interventions ranging from less invasive treatment alternatives to recommendations and referrals for appropriate psychiatric intervention. Frequently, the patients who comply with treatment recommendations made for undertreated or untreated mental health issues are able to resolve these issues to a degree that they can be cleared for a procedure at a later date.

Feedback Session

If, subsequent to a comprehensive pre-surgical psychological evaluation, the examiner has determined a surgical candidate is at moderate to high risk for suboptimal outcomes, a feedback session will be required. This will give the examiner the opportunity to clearly explain the results of the evaluation as well as explain any and all treatment recommendations that have been included [46]. The recommendations included in the evaluation should always be made within a framework of providing the highest efficacy, lowest risk, and lowest burden for the patients. Effectively conveying these recommendations to patients requires an amalgam of assessment and psychotherapeutic skills presented in a manner that often resembles a hybrid of testing and therapy.

The effectiveness of the feedback session should be measured primarily in terms of patient benefit. The examiner is essentially serving as a patient advocate at this stage of the evaluative process by emphasizing two simple goals; helping patients maximize benefit from the recommended evidence-based treatment and helping them avoid treatments that are likely to be unsuccessful. Much like the approach taken by an examiner in an assessment feedback session, the pre-surgical examiner

presents the results in plain language that can be easily understood and will not overwhelm patients with data. Feedback is then solicited and patients are encouraged to ask questions. And much like the approach taken in a therapy session, the examiner presents the results with honesty and empathy in an atmosphere of collaboration and support. The examiner also can increase the likelihood that patients will view them as an advocate rather than a gatekeeper to treatment by collaborating with surgeons in the assessment process. Surgeons can prepare their patients for the evaluation by conveying that it will help them better understand their condition and more accurately identify the best treatment for them [14].

Patients who have been informed that they have not been cleared psychiatrically for a surgical procedure initially may experience a range of negative emotions. Patients may feel anger toward the examiner based on a belief they are being denied access to treatment or experience a sense of fatalism or resignation about ever having their condition treated. It is not uncommon under these circumstances for patients to experience shame or self-blame related to psychiatric issues and/or lifestyle choices that may have played a role in their risk status. Considering the powerful impact that patient activation has been found to have on surgical outcomes, the feedback session can be a perfect opportunity for the examiner to encourage and foster increased activation with these particular patients. This can be accomplished through simple therapeutic techniques such as emphasizing patients' strengths, acknowledging what they may have already been doing or are planning to do, and reflecting on positive aspects of their current condition/circumstances. Conducting the feedback session in this hybrid form of assessment and psychotherapy has the potential to foster movement from blaming self or others to taking informed actions as well as movement from a sense of fatalism to a sense of autonomy.

Summary

A large and growing body of research has demonstrated that pre-surgical psychological evaluations can identify patients who will benefit most from treatment, identify patients at risk for poor outcome, and predict surgical outcome in a powerful, reliable, and systematic fashion. Thoughtful consideration of the data gathered in these evaluations allows the pre-surgical examiner to make meaningful, efficacious contributions to the surgical team. Patients with non-compliance issues and/or significant mental health issues can be identified and assisted in accessing appropriate treatment, thus improving the odds they will experience a positive outcome. Treatment outcomes are improved by screening out patients at high risk for poor outcome. Surgeons are provided with empirically-validated rationale in cases where they were already feeling uncomfortable about proceeding with surgery. And most importantly, the results of these evaluations when carefully integrated with contributions from patients, surgeons, healthcare teams, and insurance companies, helps ensure both surgical outcome and patient satisfaction is optimized.

References

1. Aalto TJ, Malmivaara A, Kovacs F, Herno A, Alen M, Salmi MA, Kroger H, Andrade J, Jimenez R, Tapaninaho A, Turunin V, Savolainen S, Airaksinen O. Preoperative predictors for postoperative clinical outcome in lumbar spinal stenosis: systematic review. Spine. 2006;31:E648–63.
2. Bigos SJ, Battie MC, Spengler DM, Fisher LD, Fordyce WE, Hansson TH, Nachemson AL, Wortley MD. A prospective study of work perceptions and psychosocial factors affecting the report of back injury. Spine. 1991;16(1):1–6.
3. Block AR, editor. Presurgical psychological screening in chronic pain syndromes: a guide for the behavioral health practitioner. New York: Phycology Press; 1996.
4. Block AR. Spine Surgery. In: Block AR, Sarwer DB, editors. Presurgical psychological screening: understanding patients, improving outcomes. Washington DC: APA Press; 2013. p. 43–60.
5. Block AR. Demoralization, patient activation, and outcome of spine surgery. Healthcare. 2016;4(1):11.
6. Block AR, Marek RJ. Presurgical psychological evaluation: risk factor identification and mitigation. J Clin Psychol Med Settings. 2020;27:396–405.
7. Block AR, Ohnmeiss DD, Guyer RD, Rashbaum RF, Hochschuler SH. The use of presurgical psychological screening to predict the outcome of spine surgery. Spine J. 2001;1:274–82. [PubMed: 14588332]
8. Block AR, Gatchel RJ, Deardorff WW, Guyer RD. The mind-body interface: establishing surgical prognosis. In: Block AR, Gatchel RJ, Deardorff WW, Guyer RD, editors. The psychology of spine surgery. Washington DC: American Psychological Association; 2003. p. 101–14.
9. Block AR, Ben-Porath YS, Ohnmeiss D, Burchett D. Pre-surgical psychological screening: a new algorithm, including the MMPI-2-RF, for predicting surgery results. Presented at the 26th annual meeting of the North American Spine Society, Chicago, IL, November 2011.
10. Block AR, Marek RJ, Ben-Porath YS, Ohnmeiss DD. Associations between Minnesota Multiphasic Personality Inventory-2-Restructured Form (MMPI-2-RF) scores, workers' compensation status, and spine surgery outcome. J Appl Biobehav Res. 2014;19:248–67.
11. Block AR, Marek RJ, Ben-Porath YS, Kukal D. Associations between pre-implant psychological factors and spinal cord stimulation outcome: evaluation using the MMPI2-RF. Assessment. 2017;24(1):60–70.
12. Block AR, Marek RJ, Ben-Porath YS. Patient activation mediates the association between psychosocial risk factors and spine surgery results. J Clin Psychol Med Settings. 2019;26(2):123–30.
13. Boersma K, Linton SJ. Screening to identify patients at risk: profiles of psychological risk factors for early intervention. Clin J Pain. 2005;21(1):38–43.
14. Bruns D. The presurgical evaluation: a perspective from guidelines and translational science. Presented at: North American Spine Society (NASS) 31 rd Annual Meeting. October 2016. Boston MA.
15. Bruns D, Disorbio JM. Battery for health improvement 2 manual. Minneapolis: Pearson; 2003.
16. Bruns D, Disorbio JM. Assessment of biopsychosocial risk factors for medical treatment: a collaborative approach. J Clin Psychol Med Settings. 2009;16:127–47. [PubMed: 19205851]
17. Bruns D, Disorbio JM. The psychological assessment of patients with chronic pain. In: Deer TR, editor. Comprehensive treatment of chronic pain: medical, interventional, and behavioral approaches. New York: Springer; 2013. p. 805–26.
18. Bruns D, Disorbio JM. Evaluating candidates for spinal cord stimulation, spinal surgery, and intrathecal pumps: a primer and glossary of term for psychologists. 2018 healthpsych.com.
19. Bruns D, Mueller K, Warren PA. Biopsychosocial law, health care reform, and the control of medical inflation in Colorado. Rehabil Psychol. 2012;57(2):81–97.
20. Campbell CM, Jamison RN, Edwards RR. Psychological screening/phenotyping as predictors for spinal cord stimulation. Curr Pain Headache Rep. 2013;17(1):307.
21. Carragee EJ. Minimal acceptable outcomes after lumbar spinal fusion. Spine J. 2010;10(4):313–20.

22. Carragee EJ, Barcohana B, Alamin T, van den Haak E. Perspective controlled study of the development of lower back pain in previously asymptomatic subjects undergoing experimental discography. Spine. 2004;29(10):1112–7. [PubMed: 15131439]
23. Carragee EJ, Alamin TF, Miller JL, Carragee JM. Discographic, MRI, and psychological determinants of low back pain disability and remission: a prospective study in subjects with benign persistent back pain. Spine J. 2005;4(1):24–35. [PubMed: 15653082]
24. Celestin J, Edwards RR, Jamison RN. Pretreatment psychosocial variables as predictors of outcomes following lumbar surgery and spinal cord stimulation: a systematic review and literature synthesis. Pain Med. 2009;10(4):639–53.
25. Chaplin L, Ward K, Ryken T. Preoperative depression, smoking, and employment status are significant factors in patient satisfaction after lumbar spine surgery. J Spine Disorders Tech. 2015;30:E725.
26. den Boer JJ, Oostendorp RA, Beems T, Munneke M, Oerlemans M, Evers AW. A systematic review of bio-psychosocial risk factors for an unfavorable outcome after lumbar disc surgery. Eur Spine J. 2006;15:527–36.
27. Epker J, Block AR. Presurgical psychological screening in back pain patients: a review. Clin J Pain. 2001;17:200–5.
28. Evers AW, Kraaimaat FW, van Riel PL, Bijlsma JW. Cognitive, behavioral and physiological reactivity to pain as a predictor of long-term pain in rheumatoid arthritis patients. Pain. 2001;93:139–46.
29. Gatchel RJ. Preventing chronic disability in patients with acute low back pain. Drug Benefit Trends. 2006;18:196–202.
30. Gatchel RJ, Mayer TG. Psychological evaluation of the spine patient. J Am Acad Orthop Surg. 2008;16:107–12.
31. Lieberman IH. Who should be referred for PPE and why? Presented at: North American Spine Society (NASS) 33 rd Annual Meeting. Sept 26, 2018. Los Angeles, CA.
32. Marek RJ, Ben-Porath YS. Using the MMPI-2-RF in medical settings. Presented at: APA Annual Convention. August 8, 2019. Chicago IL.
33. Marek RJ, Ben-Porath YS, Epker JT, Kreymer JK, Block AR. Reliability and validity of the Minnesota Multiphasic Personality Inventory-2-Restructured Form (MMPI-2-RF) in spine surgery and spinal cord stimulator samples. J Pers Assess. 2020;102(1):22–35.
34. Meyer GJ, Finn SE, Eyde LD, Kay GG, Moreland KL, Dies RR, et al. Psychological testing and psychological assessment. A review of evidence and issues. Am Psychol. 2001;56:128–65.
35. Schofferman J, Anderson D, Hines R, Smith G, White A. Childhood psychological trauma correlates with unsuccessful lumbar spine surgery. Spine. 1992;17(6):138–44.
36. Seebach CL, Kirkhart M, Lating JM, Wegener ST, Song Y, Riley LH, Archer KR. Examining the role of positive and negative affect in recovery from spine surgery. Pain. 2012;153:518–25.
37. Skarabis IM, Smith K. Psychological evaluation for those receiving devices for the treatment of pain. In: Deer's treatment of pain; 2019. p. 135–9.
38. Skolasky RL, MacKenzie EJ, Wegener ST, Riley LH. Patient activation and adherence to physical therapy in persons undergoing spine surgery. Spine. 2008;33(21):E784–91.
39. Skolasky RL, MacKenzie EJ, Riley LH, Wegener ST. Psychometric properties of the patient activation measure among individuals presenting for elective lumbar spine surgery. Qual Life Res. 2009;18(10):1357–66.
40. Slover J, Abdu WA, Hanscom B, Weinstein JN. The impact of comorbidities on the change of Short-Form 36 and Owestry scores following lumbar surgery. Spine. 2006;31:1974–80.
41. The Guide to Clinical Preventative Services 2012. www.USPreventativeServicesTaskForce.org
42. Turner JA, Jensen MP, Romano JM. Do beliefs, coping, and catastrophizing independently predict functioning in patients with chronic pain. Pain. 2000;85(1):115–25.
43. Velley AM, Mohit S. Epidemiology of pain and relation to psychiatric disorders. Prog Neuro-Psychopharmacol Biol Psychiatry. 2017;87:159.

44. Williams GC, McGregor HA, Zeldman A, Freedman ZR, Deci EL. Testing a self-determination theory process model for promoting glycemic control through diabetes self-management. Health Psychol. 2004;23(1):58–66.
45. Williams GC, McGregor HA, Zeldman A, Freedman ZR, Deci EL, Elder D. Promoting glycemic control through diabetes self-management; evaluating a patient activation intervention. Patient Educ Couns. 2005;56(1):28–34.
46. Wright JW. Providing feedback. Chapter 6. In: Conducting psychological assessment: a guide for practitioners; 2011.
47. Ziegelmann M, Kohler TS, Bailey GC, Miest T, Alom MM, Trost L. Surgical patient selection and counseling. Transl Androl Urol. 2017;6(4):609–16.

Pre and Postoperative Spine Rehabilitation

14

Nasser Ayyad and Denizen Kocak

Epidemiological Considerations

An appreciation for the total burden on spinal care is important to keep in mind when considering the possible economic and healthcare impacts of a prehabilitation program. Even small changes in the standard of practice can have a meaningful impact on an issue that is so prevalent in the United States.

Back pain is an extremely common healthcare complaint in the United States and is associated with significant reduction in quality of life and significant cost to the healthcare system and to work productivity. For adults in the U.S. between ages 20 and 69, the prevalence of chronic low back pain was estimated at 13.1% in 2009–2010, and it has been previously estimated that the lifetime prevalence of back pain may be as high as 84% [1]. Data from the 2009–2010 National Health and Nutrition Survey (NHANES) associated back pain increased with age, female gender, and Caucasian race. It also showed that US adults with chronic low back pain are often socioeconomically disadvantaged and were more likely to be unemployed, receive disability benefits, and have a household income of less than $20,000 [2]. The direct annual healthcare cost for persons with chronic low back pain was estimated to be roughly $8400 per person for those with chronic back pain versus $3600 for persons without chronic low back pain in 2005, with a total cost estimates of $102 billion to greater than 200 billion per year [3].

N. Ayyad (✉) · D. Kocak
Department of Physical Medicine and Rehabilitation, UT Southwestern Medical Center Dallas Texas, Dallas, TX, USA
e-mail: Nasser.Ayyad@UTSouthwestern.edu; Denizen.Kocak@UTSouthwestern.edu

© The Author(s), under exclusive license to Springer Nature Switzerland AG 2022

C. E. Noe (ed.), *Multidisciplinary Spine Care*,
https://doi.org/10.1007/978-3-031-04990-3_14

Additionally, patients with chronic back pain were more likely to have depression, sleep disturbances, and were more likely to smoke [2], all of which have downstream healthcare effects and costs. Indirect costs of low back pain are also high due to work absenteeism. Over a 6-month period, about 15% of patients with chronic low back pain will miss work and the median duration of missed workdays for an absence ranged from about 14–24 days in a meta-analysis [4].

In addition to the prevalence of back and neck pain the cost of spine surgery in the United States is also a significant factor on the economic burden of spine care. In 2011, spinal fusion surgery was the costliest operating room procedure in the United States, resulting in $12 billion in overall cost [5]. The average length of stay for lumbar fusion surgery is about three and a half days for an uneventful surgery with no complications and about 5 days when there is a complication [6].

Given the expected increase in absolute population and the shift towards an older population, it is likely that the costs associated with future spine care will have profound implications on the U.S. healthcare and labor systems. The U.S. Census Bureau has estimated that the population over the age of 65 in the United States will be 73 million by 2030 and 85 million by 2050 compared to just 49 million in 2016 [7]. As one might expect with an aging population, the number of spinal surgeries in the United States is also increasing. From 2004 to 2015, the number of elective lumbar fusion surgeries increased by about 62%, from 122,000 to nearly 200,000. Of note, the largest increase in rate of surgery was highest for the population age 65 and over, which increased 138% [8]. Lumbar discectomies are also a common intervention, with an estimated 480,000 lumbar discectomies performed annually in the United States [9]. Cervical spinal surgeries also increased significantly over a similar time period from 2002 to 2011 though the increase was not as dramatic [10]. There were approximately 27,400 cervical spine surgeries performed in 2002 with an increase to 36,400 in 2011, with the majority of these surgeries being anterior cervical fusions, which accounted for roughly 84% of cervical spine procedures. Over this same period, the mean age of patients undergoing surgery also increased from about 50–55 years old, and the overall number of comorbidities in the patients undergoing surgery also increased.

Now that we have a better understanding of the economic impact of a neck and back pain and spine interventions we will discuss how the preoperative spine patient and discuss how a prehabilitation program can improve outcomes and reduce costs associated with the surgical spine patient. Given that in one survey of spinal surgeons [11], less than 40% of surgeons provided written materials to patients preoperatively, there is certainly room for improvement.

Preoperative Spine Patient

Some patients will present with urgent and emergent needs for surgery and may have been near their functional baseline days to even hours before they undergo surgery, but this is not the typical picture. Surgical spine patients often have a

subacute to chronic presentation, with pathology combine with other comorbidities that has developed over years and can lead to a constellation of functional decline with pain, decreased mobility, difficulty completing activities of daily living, and muscular atrophy, among others. And any surgery, no matter how successful, and no matter how skillfully performed, presents the body with a physiologic stressor and can lead to an acute worsening of a patient's physical deficits.

In one cohort of 650 patients over 70 years old examined the association between back pain and decreased lower extremity physical function. They found that low back pain was independently associated with worsening rapid gait, chair stand, and foot tap. The threshold of this decline appeared to be four or more months of chronic pain. In this study chronic pain was also associated with increased comorbid anxiety and depression. Although the causal relationship is not clear, there is a clear association between the two, and there is some evidence that there is overlap in the central neuroplastic changes that occur in the two conditions [12].

Additionally, patients with depression, lower health-related quality of life, greater preoperative disability, and longer pain duration were more likely to have poor spine surgical outcomes. Notably, this same study suggested that patients who received preoperative physiotherapy or chiropractic care were less likely to report a poor outcome [13]. Although none of these concepts are particularly surprising, they serve to highlight specific areas which can be targeted with prior to surgery in order to improve outcomes.

What Is Prehabilitation?

Presurgical rehabilitation or prehabiliation, known more informally as "prehab" is a framework of care initiated before surgery, whereby patients' physical, nutritional, medical and physiological preparation, and caregiver involvement are strengthened while waiting for surgery in order to maximize physiologic reserve and enhance the patient's ability to maximize their recovery following surgery.

A prehabilitation program often consists of a preoperative baseline medical assessment together with evaluation of physical strength and flexibility, nutrition and mental health. A personalized program is then prepared based on medical, nutritional, and psychological optimization. The physical therapy component often includes strengthening, improving cardiovascular and pulmonary endurance, as well as stability and balance exercises.

Education

Patient education should be a cornerstone of preoperative preparation for the patient and sets the stage for patient expectations for surgery, recovery, and care at home. Ideally it should include those who will act as caregivers for the patient after discharge as they will also be instrumental in the recovery process.

Although research is generally heterogenous and no standard education protocol has been widely adopted, systematic reviews have been able to suggest that education, either as a stand-alone presurgical intervention, or as part of a multifaceted approach have beneficial effects. Most convincingly, there appears to be a positive effect on psychological outcome measures, with improved anxiety and depression, better feelings of preparedness, and better patient satisfaction. Evidence for pain, disability, and other performance-based outcomes did not show as convincing of a beneficial effect [14]. A lack of information, however, in the pre and perioperative phase has been associated with increases in anxiety and depression [15].

Delivery of information can be through a variety of means in terms of simple verbal discussion at the surgical consultation in office, written handouts, videos, or patient classes, and the optimal medium is still an area of active research. Although the method of delivery may vary, certain principle subjects for education should be addressed with the surgical spine patient:

- Diagnosis: Simply explain, by minimizing medical jargon and using common terms, to describe the patient's condition and purposed surgical procedure(s). Special care must be taken to ensure that materials are patient appropriate, with a targeted reading level of fifth or sixth grade. Often, even after removal of medical jargon, materials remain above this suggested reading level [16].
- Prognosis: This is an opportunity to set expectations for a patient regarding to acute, intermediate, and long-term outcomes for a particular condition and surgery. The patient's post-operative pain, function, as well as their rehabilitation process following surgery should be addressed by the physician as well as therapists. Patients with positive expectations may experience less pain and disability when compared to patients with negative expectations [17].

In one of the most aggressive approaches detailed, Fleege and colleagues in Germany described a four-hour interdisciplinary educational day where patients and family met with the entire care team including the surgeon, anesthesiologist, therapist, social workers, and patients who had previously undergone the procedure in the week leading up to the operation. Their program also included early post-operative intervention with a focus on mobilization so the true effect of education alone cannot be isolated, but it suggested greater patient satisfaction, >80% mobilization on day one, and a decreased length of stay of almost 4 hospital days. Despite the increased pre-operative costs which were about 30% higher, the overall cost was lower due to shortened hospital stays. Although this appears to be effective, it may not be applicable to the U.S. healthcare system and requires significant resource investment on the part of the institution. It does, however, highlight the positive impact a comprehensive surgical preparatory plan can make [18].

Reducing Fall Risk

Limiting fall risk post-operatively is an extremely important part of the pre-surgical rehab process. There are numerous different fall risk assessment tools available to the clinician including the Berg Balance scale score (score <50), the Timed Up and Go test (>12 seconds), and the Five Times Sit-to-Stand times (>12 seconds) appear to be the most effective assessment tools [19, 20]. Standard prehabilitation has unfortunately not consistently been shown to improve balance and strength post-operatively despite promising improvements in standing balance reported in one study following prehabilitation prior to total knee arthroplasty [21]. Based on clinical judgement, the physician and therapist can determine if an increased focus on balance and fall prevention training are indicated. Additional mind-body practices may be considered such as Tai Chi to improve balance. Tai Chi and other complementary therapeutic practices will be discussed later in the chapter.

Cardiovascular Conditioning

Exercise tolerance is also correlated with surgical outcomes. Preoperative cardiopulmonary exercise testing (CPET) acts as a measurement of exercise capacity, with an anaerobic threshold of less than 11 ml.O_2.kg being associated with increased risk of complications [22]. Systematic reviews have failed to consistently prove significant improvement in physiologic function or clinical outcomes, but these studies suffered from small sample sizes, poor adherence to the prehab programs, and heterogenous markers of outcomes. Logically, exercise would improve cardiovascular conditioning, but further research is required to demonstrate the efficacy of prehabilitation for cardiovascular conditioning [23].

Smoking Cessation

Smoking cessation only 4–6 weeks prior to surgery has been shown to have significant reductions in various postoperative complications including wound and bone healing, morbidity, and mortality. A systematic review of 21 studies showed that cessation of at least 4 weeks prior to a variety of surgical procedures appeared to be associated with a significant reduction in overall complications, wound complications, and pulmonary complications [24]. After spine procedures, smokers were less likely to return to work, less likely to have reached a clinically important difference in Oswestry Disability scores, more likely to require regular analgesics, and were less likely to be satisfied with the results of their operation [25].

Bone healing is a specific area of concern in the surgical spine patient and multiple studies have shown impaired bone healing in smokers. The etiology for this is multifactorial, but generally is theorized to be due to decreased bone mineral density, impaired osteoblastic cellular activity, and impaired blood flow and angiogenesis [25]. In lumbar spinal fusion surgeries, nonunion rates are significantly higher, and have been shown to be related to the overall number of cigarettes smoked per day. In patients undergoing two level lumbar fusions, nonunion rates appeared to be nearly 30% in the smoking group versus 11% in the nonsmoking group. For cervical intervention, the effect appears to be less marked still trends towards higher rates of pseudarthrosis. This again was more common in patients undergoing multilevel fusions and may also be more common in patients who undergo anterior cervical discectomy and fusion [25].

Literature has suggested a drop-off in the effectiveness of smoking cessation as an intervention if it is started less than 3–4 weeks preoperatively [26] and some studies have also suggested a nearly dose dependent relationship over the preoperative period where each additional week of smoking abstinence was associated with about a 19% increase in the magnitude of improvement in some postoperative complications [24]. Additionally, patients who were offered intensive interventions had higher rates of continued smoking cessation at 12 months post-operatively with patients being roughly three times more likely to remain abstinent at 12 months on pooled analysis of intensive smoking cessation interventions. Although the optimal timing of cessation is simple, the earlier the better, but at the minimum the provider should focus on cessation at least 1 month prior to intervention and be aware of cessation programs that are available.

Alcohol Cessation

Alcohol cessation is another important consideration in the preoperative optimization of surgical patients. Although we commonly consider alcohol related diseases like cirrhosis, pancreatitis, and withdrawals, we often fail to consider the effects on cardiac function, immunosuppression, hemostasis, and the overall ability to maintain homeostasis. In patients drinking three or more alcohol units on a daily basis (36 grams of alcohol, slightly less than three standard American drinks which is roughly 42 grams of alcohol [27], alcohol cessation interventions reduced post-operative complications when pooling all complications including wound related complications, need for secondary surgery, cardiac or pulmonary complications, and need for transfer to intensive care. It did not, however show a statistically significant effect on in hospital and 30-day mortality. Of note, both studies in the meta-analysis reporting these findings included pharmacotherapy for alcohol cessation, using either disulfiram or chlordiazepoxide [28].

Nutrition

Nutrition optimization in patients undergoing spine surgery is a critical variable of a prehabilitation program as improved outcomes and decreased complications are associated with enhanced perioperative nutritional status. As previously discussed, spine surgeries are expected to continue to increase in the U.S. and this holds particularly true for elderly patients. Older patients have an increased rate of metabolic disorders such as obesity, hypertriglyceridemia, and diabetes and subsequent nutritional deficits [29]. Other nutritional deficiencies may be secondary to conditions such as anorexia, malabsorption syndromes, metabolic imbalances that causes increased nutrient utilization, inflammatory bowel disease, psychiatric disorders, and neoplastic processes.

Patients undergoing elective spinal surgery should have a comprehensive nutritional assessment prior to surgery. Methods of optimization include preoperative screening with Nutritional Risk Score or other scoring systems, looking for changes in body mass index, detecting sarcopenia, and screening for metabolic abnormalities. Assessment of blood glucose, electrolytes, cholesterol, vitamin levels, water and fiber intake, visceral proteins, and lean body mass should also be done preoperatively, and close monitoring should be continued postoperatively. In patients with diabetes tight perioperative glucose control is important in order to decrease complications such as infection and deep venous thrombosis among other adverse outcomes [30].

Albumin is a nutritional marker that is often used to determine the health status of patients before surgery. An albumin level of 3.5 g/deciliter is a serological indicator of malnutrition [31]. Other serologic parameters of malnutrition that are often measured and tracked are total lymphocyte count, <1500 cells/mm^3, and transferrin levels, <200 mg/dL [32]. Pre-albumin is often ordered in conjunction with albumin as it is also an acute phase reactant and can be low in malnourished patients. The challenge with using pre-albumin as an indicator is that it can be increased in renal failure and hypothyroid disorders and decreased in liver disease and infections. A retrospective study indicated that a low prealbumin level is a possible risk factor for early-stage surgical site infection in spine surgery, though it was not statistically significant; operative time was the most important indicator of SSI on multivariate analysis [33].

Malnourished and volume depleted patients should be given balanced diets replenishing key nutrient deficits while monitoring glucose levels in diabetic patient. Postoperatively, patients should initiate a diet as soon as possible to decrease overall length of stay and complication rates, facilitating return to normal activities.

In addition to improving patient outcomes, studies have shown that nutritional intervention can be extremely cost effective, saving an estimated $52 for every dollar spent [34]. Again, the exact timing of nutritional intervention is not well studied, with 4 weeks before surgery as a minimum target [35].

Centralization of Pain and Psychologic Intervention

As previously mentioned, the perception of pain and anxiety/depression share many of the same neuroplastic characteristics, and both are common in the spine surgery population. Studies have reported that nearly 70% of patients who undergo lumbar spinal surgery have kinesophobia, and patients who catastrophize and display fear-avoidance behaviors tend to have worse surgical outcomes [36]. Although cognitive behavioral therapy and mindfulness have shown benefit in pain severity and functional limitations in chronic back pain [37], presurgical and post-surgical psychologic interventions are less robustly studied.

Preoperative pain neuroscience education has shown promise in decreasing healthcare utilization up to 1-year post-surgery but failed to show significant benefit in terms of pain and functional improvement [38]. Preoperative CBT has shown more rapid improvement in disability as defined by the Oswestry Disability Index (better at 3 months post-operatively compared to standard care) but has failed to show long term benefits in two studies which were reviewed by Lotzke et al. [36, 39].

Enhanced Recovery After Surgery Program

The largest semi-standardized prehabilitation program is the Enhanced Recovery After Surgery (ERAS) approach. It has been one of the more recent and effective movements to improve surgical care and improve postoperative recovery. It considers many of the previously discussed presurgical health variables and works to address them as part of a comprehensive and holistic plan to improve postoperative functional gains and to decrease costs, length of stay, and postoperative pain. It was first described in the colorectal surgery population with the first ERAS consensus paper published in 2005 [40].

Within the ERAS framework, there is traditionally the pre-hospital phase, the immediate preoperative phase, the intraoperative phase, the immediate postoperative phase, and the post-discharge phase which may include a variety of both at-home and inpatient options [41].

The most recent clinical guidelines from the ERAS society as of the writing of this book were released in 2018, with strong recommendations for smoking, alcohol and nutrition interventions and a weak recommendation for pre-surgical physical therapy interventions, presumably based on the low quality of existing evidence on postoperative clinical outcomes [42].

An Approach and Potential Model

The University of Texas Southwestern Spine Center ERAS program will be discussed here and in light of a lack of consensus guidelines on prehabilitation protocols may serve as a model for other practitioners or institutions who are planning to initiate an ERAS Spine program. The protocol is an active area of research at the institution, and we do not make claims that it is superior to other protocols.

Patients are screened for eligibility to participate in the ERAS program based on measures of frailty, likelihood of discharge to rehab or nursing facility, and measures of likely centrally sensitized pain. Frailty is measured by the FRAIL scale, which is a validated screening tool that includes fatigue, resistance, ambulation, illnesses, and loss of weight to predict future difficulties in ADLs and IADLs [43]. Likelihood of discharge to a rehabilitation hospital or nursing facility is estimated by the ACS NSQUIP (American College of Surgeons National Surgical Quality Improvement Program) "Discharge to Rehab or Nursing Facility" calculator. Risk of discharge to post-acute care varies for various specialties. Most relevant to spine surgery are the orthopedic and neurological surgery rates of admission to post-acute care, though note that these figures include all orthopedic and neurosurgical procedures. For orthopedic surgery, the overall rate of discharge to post-acute care was 24.5% and for neurosurgery it is 14.6% [44] and the ACS NSQUIP risk calculator can be used to compare risk of discharge to post-acute care for individualized patients. Centrally sensitized pain is measured by the Central Sensitization Inventory, and patients who have at least moderate centralized pain (a score of 40 or greater on a 100-point scale) meet criteria for eligibility [45].

The program itself includes a 4–8-week prehabilitation regimen with a minimum of five visits over this period. The goals of the program are to manage pain and pain avoidance behaviors through education, optimize body and gait mechanics including balance, improve strength and mobility, and increase cardiovascular conditioning.

Management of pain and activity/pain avoidance is a multifaceted educational approach on the benefits of movement for healing and the effects of movement on pain control with a targeted focus on cognitive behavioral therapy, pain neuroscience education, and overall wellness. Pain neuroscience education is a cognitive approach which educates patients on the concept of central sensitization to pain and shifts the focus from mechanical sources of pain to altered interpretation and sensation of pain. In combination with manual therapy, this can help to decrease pain and disability [46].

Improvement of body and gait mechanics includes work on positioning during sustained postures and a focus on a sustained daily walking program of at least 20 minutes in duration with an emphasis on education about gait mechanics with incorporation of an assistive device as needed. Strengthening occurs on a progression scheme with progression from isometric exercises to open and closed chain isotonic exercises with an additional focus on stretching and mobility exercises. Within this strengthening program, incorporation of functional mobility work include sit-to-stand exercises. In addition to this functional training, if there are expected post-operative weightbearing or range of motion restrictions, it may be beneficial to attempt to practice these preoperatively so that patients feel comfortable with the motions and have built up their stabilizer muscles prior to surgical intervention.

An additional focus should include cardiovascular conditioning in anticipation of deconditioning and additional exertion needed to maintain restrictions post-operatively. The program targets both time and intensity-based goals for aerobic exercise, with a goal of greater than 10 minutes of greater than 50% VO2max. VO2max is the maximum rate of oxygen consumption during increasingly intense exercise. Although one may have seen this measured in athletes with a fitted mask on a treadmill, there are various estimates of VO2max that are more practical and cost effective when applied to the prehabilitation population. There are estimates of VO2max that can be calculated based on the ratio of maximum heart rate to resting heart rate [47] and the Rockport fitness walking test which is a timed one-mile walk [48], among many other methods, including some that are now integrating common biosensors like watches that track movement and heart rate.

Often, this program will continue up until near the date of surgery, but there are discharge criteria by which patients may graduate from the program early. In order to be discharged from the program, patients must be independent in transfers and bed mobility, must be independent household ambulators, be independent in a home exercise program for continued presurgical optimization, be able to articulate post-operative expectations and discuss pain management strategies, and have met or exceeded the minimum measures of significant improvement in at least three of the six measured functional tests. After surgery, the same patients are enrolled in post-operative rehabilitation.

Postoperative Rehabilitation

As previously discussed, patients that undergo spine surgery have often experienced pain for months or even years prior to surgery. Others may develop new or worsened pain following spine surgery. A prospective cohort study revealed that lumbar fusion (one to two levels), lumber fusion (three or more levels) and complex spinal reconstruction were three of the six most painful procedures during the first postoperative

day [49]. Patients with pain naturally move less and may experience kinesiophobia, fear of worsening their pain with movement, which inhibits their activity levels. Decreased activity and mobility can set off a cascade of physical problems including deep vein thrombosis, depression, increased muscle stiffness, myofascial dysfunction, weakness, and increased pain [50]. Furthermore, decreased activity, even for a few weeks is directly link to weight gain and decreased fitness level which predisposes patients to further metabolic and cardiovascular health problems. Also, more invasive spine surgeries such as spinal fusion can disrupt the muscle structure of the abdomen or spine and may lend to acute post-operative weakness and biomechanical changes of the spine [51].

Early Mobilization (Day 0–2 Weeks)

Early mobilization following surgery is one of the pillars of ERAS programs and has shown benefits in reducing perioperative comorbidities and length of stay in other medical/surgical subspecialties. Recent systematic reviews have shown that early mobilization after spine surgery, while obeying precautions as described by the surgeon, has multiple benefits including increased control of pain, reduced morbidity (cardiopulmonary decompensation, urinary tract infections, deep vein thrombosis/pulmonary embolism, sepsis or infection), and limiting fatigue and atrophy of major muscles groups of the spine and extremities and thus improved mobility. Early mobilization also has consistently shown to decrease the risk of depression.

There is also evidence that early mobilization is superior to bed rest for performance-based and patient-reported outcomes following elective spinal surgery. In another review patients who underwent accelerated ambulation and rehabilitation also had a reduced length of hospital stay with no increased risk of complication or readmission [41].

Encouraging patients to mobilize soon after surgery makes physiological sense, however there are currently no guidelines or universally adopted procedure/surgery specific recommendations form mobilization following a specific spine surgery.

A 2019 review summarized the current practice for specific spine surgeries in the U.S. Per the review, on average, mobilization is encouraged within 1–2 hours following surgery for elective discectomies and microdiscectomies. Patients undergoing non-complex lumbar fusion (less than six levels) are often mobilized "right after surgery" and those undergoing multilevel fusion of the lumbar or thoracolumbar spine within 8 hours.

Following cervical laminectomies and foraminotomies and anterior cervical discectomy and fusion patients were mobilized within 6 hours following surgery. In the same study, mobilization for patients undergoing posterior cervical fusion were still advised to mobilize on the day of surgery but based on individual and clinical judgement [14]. Therefore, in most cases mobilization on the day of surgery and

ambulation from the first postoperative day is possible and should be the goal. Given the benefit of early mobilization the importance of safely developing mobilization protocols is clear. Moving forward the opportunity is to establish practice guidelines on the optimal type and timing of mobilization, and how this should be modified for different spinal procedures.

Timing and Frequency of Postoperative Rehabilitation

Despite the fact there are no definitive recommendation for the postoperative management after spinal surgery most patients are instructed by their surgeons to continue to ambulate but to avoid extensive bending, lifting, twisting, and to begin post-operative exercises programs 4–6 weeks after surgery. There are recent randomized controlled trials that suggest rehab exercises as early as 3 weeks, such as strength training, are safe and effective [52]. There are also no universal guidelines for post-operative exercise rehab programs for specific spinal surgery patients however the literature consistently shows that comprehensive physiotherapy commenced within the first 4 weeks after surgery does not increase the potential for an adverse event and leads to a moderate, statistically significant reduction in pain when compared with a control groups.

Postoperative Cardiovascular Exercise

Generalized, aerobic exercise is often a major element of postoperative rehabilitation. The most common form of cardiovascular activity recommended in the immediate postoperative period is walking. As previously noted, one of the primary indications of walking immediately following surgery is to prevent the formation of deep vein thrombosis. However, the benefits of walking extend much further for postoperative spine patient. Walking increases the flexibility and strength of the muscles and ligaments that support the spinal column such as the paraspinal muscles, abdominal muscles, lower extremities, and pelvic/gluteal musculature. It also promotes blood flow and oxygenation of these tissues. There is limited evidence to suggest that better perfusion of deconditioned muscle tissue facilitates the release of chronic low back pain by alleviating ischemia of tired and fatigued muscles The flexibility of your spinal ligaments and tendons is also increased, improving the overall range of motion in your cervical, thoracic, and lumbar spine. Walking may help with the diffusion of oxygen and glucose from the end plates of the vertebral bodies to the largely avascular spinal discs [53].

From a practical perspective, a recent prospective study showed that participants that walked more in the first post-operative week were more likely to have substantially improved function on the Oswestry Disability Questionnaire at 6-months [54].

Lastly, walking is a fundamental weight bearing activity that is an integral part of bone mineralization.

Aquatic Exercises

Aquatic or hydrotherapy is defined as exercise in warm water to improve muscle strength, flexibility, and cardiovascular fitness. Exercising in water can be used in conjunction with land-based exercises or can be the primary environment for exercises for the post-operative patient. Water's buoyancy decreases apparent body weight and lower limb internal joint forces thus limiting the stress that eccentric contractions place on landing and closed kinetic chain activities. For patients that cannot tolerate land based exercise due to comorbidities such as osteoarthritis of adjacent joints it may allow patients to walk and perform exercises earlier in the post-operative process in order to decrease muscle stiffness, regain mobility, improve strength, and reduce pain.

The benefits of water-based exercises were quantified in a 2018 systematic review that showed aquatic exercises significantly reduced pain and increased physical function in patients with low back pain. Although the authors noted that additional high-quality studies are needed to validate this form of therapy for chronic and post-operative low back pain and function [55]. In addition, a prospective comparative study suggested that aquatic backward locomotion exercises improved lumbar extension strength in patients who have undergone lumbar discectomy [56].

Most of the literature reviewed indicates that patients should wait until at least 2 weeks post-surgery to allow for wounds to heal prior to initiation of aquatic therapy. However, there is some emerging data that suggest that patients be allowed to submerge in water as early as 4 days postoperatively. A systematic review in 2013 showed that early aquatic therapy, less than 3 months after surgery, does not increase the risk of wound-related adverse events for adults after orthopedic surgery. Therefore, aquatic therapy should be considered as a safe and likely effective medium as a primary environment or as an extension to land based exercises for the post-operative spine patient. Additional research is needed to quantify the benefits for specific postoperative spine patient populations [57].

Strength Training

Another common component of spine rehabilitation for both nonoperative chronic neck and back pain as well as in the post-operative spine patient is strengthening of the trunk extensors and abdominal muscles, also known as motor control exercise. Theoretically, contracting muscles that surround the spine results in increased stiffness and stability and provides a splint effect that may be protective to the spine.

Conversely if the muscles around the low back are weak, the body relies more on passive structures such as ligaments and discs for stability. This may lead to accelerated degenerative changes and serve as a precursor to problems such as spinal stenosis.

Two trunk muscles seem to be particularly important to segmental spinal stabilization. First, the transverse abdominis (TA), located on each side on the torso, these muscles are sometimes referred to as the "corset muscles", and they play a key role in strengthening and stabilizing the core. The TA is the deepest of the abdominal muscles and it exerts a compressive force on the abdominal contents and pull on the thoracolumbar fascia. The TA is active with movement of the upper extremities and lower extremities as well as in flexion and extension of the trunk. Examples of TA exercises include hip bridges, planks, and set-ups. The second are the multifidus muscles that serve as the prime extensor muscles of the spine and stabilize the individual spinal segments and joints of the spine. The multifidus contract within the thoracolumbar fascia and exert a stabilizing effect on the spinal segment. Both the TA and multifidus have high concentrations of slow-twitch fibers and higher levels of oxidative enzymes, which implies that they are uniquely designed to provide prolonged tonic contractions and play a significant role in upright posture and stability.

A recent study examined the effectiveness and safety of isometric trunk exercises 3 weeks following lumbar fusion surgery. The exercises were focused on trunk extension, flexion, and lateral flexion muscles. The treatment group achieved earlier functional recovery than standard rehabilitation protocol and had no increased adverse events including no hardware loosening or failure [52].

A Cochrane Review in 2015 looked at the effectiveness of strength exercises for nonspecific low back pain and concluded that there is low to moderate quality evidence that strength training of abdominal and trunk extensors have a clinically important effect compared with a minimal intervention for chronic low back pain and low to moderate evidence that it provides similar outcomes to other forms of exercise. Some have argued that core strengthening is more important in the postoperative patient where studies have shown that lumbar fusion surgery negatively effects lumbar extensor muscle function and density. A prospective study examined the extent of atrophy of the lumbar paraspinal muscles after open lumbar interbody fusion. The results showed that there was a significant increase of electromyographic denervation activity and reduced recruitment of motor units 1-year following lumbar fusion. The paraspinal muscle volume also decreased from 67.8% to 60.4% after 1 year. Per the study results paraspinal muscle volume was significantly correlated with physical outcome, mental outcome, and pain after 1 year [58].

An extension of trunk strengthening is spinal motor control training. Spinal motor control is defined as coordinating the various components of the motor system to act in unison to produce loaded movements such as transfers, lifting, bending, and upright exercise. Proper neuromuscular reeducation for timing associated with muscle activation and increased intraabdominal pressures during these loaded

movements is also addressed during these movements. Spinal motor control is thought to be especially important following lumbar fusion of two or more consecutive lumbar spine units [59].

Neural Mobilization

Neural mobilization (NM), also known as neural dynamics or nerve gliding, is a movement-based intervention that is intended to facilitate neural function by restoring the homeostasis in and around the nervous system by mobilization of the nerve itself or the structures that surround it. For nerves to function properly they need to have the ability to slide and glide unhindered through different areas of the body. When spaces adjacent to nerves are damaged or compromised then irritation or increased pressure of an adjacent nerve may lead to painful symptoms.

NM has become a popular technique among therapist for patients with nerve related pain as is often the case in spine disorders such as stenosis or radiculopathy. They involve a sequence of maneuvers that lengthens the nerve at one joint or location while simultaneously reduces its length at an adjacent joint in order to produce sliding maneuver of neural structures relative to adjacent tissues. An example of a NM technique for low back with lower extremity radicular pain is placing the patient in the slump position (patient is placed in the seated position and slumps over by flexing their cervical and thoracic spine while extending their right or left knee) and then adding rotational, compressive, or traction forces to the spine.

Advocates of NM point to the basic science and physiology of pain medicine. Recent research using ultrasound have shown that nerves have both longitudinal and lateral movement capabilities. If blood flow is compromised due to a mechanical restriction of a nerve it can lead to a hypoxic state which can in turn lead to ischemic based pain. Furthermore, substances contained in the herniated disc can cause inflammation and radicular pain without compression of a nerve root. Studies have also show that proinflammatory chemicals such as phospholipase A1 (PLA2), is released in or around the intervertebral disc during a disc herniation, can cause damage to its protective myelin sheet and lead to peripheral sensitization of the nerve [60].

Theoretically if exercises and movements can utilize the inherent ability of a nerve to restore movement, improve blood flow, and limit the inflammatory response then it has the potential to decrease pain and improve function of the nervous system. As an extension to these results it has been suggested that neural mobilization should be part of a post-operative spine rehab program to prevent nerve root adhesions and improve outcome, but this has been largely anecdotal given the lack of studies in this population.

A systematic review of 40 studies in 2017 revealed that NM achieved statically significant improvement in disability and pain for upper and lower extremity

radicular pain. This was not replicated in individuals with carpal tunnel syndrome as NM was not effective in this population [61]. Conversely, a randomized control trial in 2001, where patients underwent NM following lumbar fusion, lumbar laminectomy, or lumbar discectomy showed no benefit for decreased pain or improved function at a 12-month follow-up [62]. It should be noted that this study included a heterogenous population and this may have affected the results. Also, the acute and subacute outcomes were not measured in this study.

Due to the scarcity of high-quality studies and conflicting results, there is insufficient evidence to make a strong argument for or against incorporating NM in the post- operative spine rehab plan. However, given its safety it's reasonable to consider adding neural tissue mobilization into the postoperative regiment, particularly for patients where there is suspicion of radicular pain secondary to scar or soft tissue adherence.

Soft-Tissue Mobilization/Manual Therapy

Manual therapy or soft tissue mobilization is a treatment used by therapists to treat musculoskeletal pain and disability; it mostly includes kneading and manipulation of muscles, joint mobilization and joint manipulation. Massage therapy is used postoperatively to reduce inflammation, decrease pain, and facilitate patient recovery. Other studies have reported that it reduces anxiety, fatigue, edema, and nausea. Pressure and touch are thought to facilitate these changes by restoring lymphatic drainage, improving blood circulation, lengthening short or tight connective tissue, relaxing tense muscles, and soothing the nervous system. The benefit of these modalities in the immediate post-operative period may be extended from other surgical specialties. Massage therapy administered post surgically has been shown to decrease patients' pain and need for analgesics after colectomy and cesarean section [63, 64]. Patients who received massage therapy after cardiovascular surgery also had significant decreases in postsurgical pain as well as a reduction in anxiety and tension compared with those who received standard care [65].

There are no significant studies that have shown conclusive evidence of the effectiveness of massage therapy in post-operative spine patient. A Cochrane Review in 2015 resulted in very little confidence for massage therapy as effective treatment for lumbar pain that had not undergone surgery [66]. This was partly due to the lack of high-quality studies. It revealed that acute, subacute, and chronic low back pain improved pain and functional improvement only in short term follow-up. It also showed that it was safe with no serious adverse effects due to massage in this population. There is insufficient evidence to advocate for or against soft-tissue mobilization's inclusion in postoperative spine rehabilitation programs. Manual therapy may be considered to provide a decrease in postsurgical pain and a reduction in anxiety and tension. If manual therapy is administered it is important to ensure no

significant stress is placed on the surgical location or adjacent levels in the acute post-operative period. More definitive trials with larger sample sizes are required to confirm the feasibility and potential therapeutic effectiveness of this approach.

Psychosocial Considerations

Over time episodic and chronic pain can influence a person's psychological state. Manifestations may include fear/avoidance behavior, anxiety, poor self-efficacy, catastrophizing, and depressed mood. Even psychologically healthy patients will endure psychological stress during the perioperative period as socioeconomic stressors associated with surgery such as unexpected medical bills or loss of income may affect their mood. A comparison analysis of the California Outcomes Database revealed that patients without a history of depression prior to spine surgery had an increased risk of post-operative depression relative to those who underwent other forms of major surgery or suffered from serious illnesses, including coronary artery bypass grafting (CABG) and congestive heart failure (CHF). The authors first identified patients without a prior history of depression who underwent spine surgery between the years of 2000 and 2010. They then identified patients with newly diagnosed depression within 5 years of undergoing spine surgery. Statistical analysis showed that the risk of post-operative depression for patients who underwent spinal surgery was approximately two-fold higher than for patients who underwent CABG or who have COPD, medical conditions known to be associated with postoperative depression [67].

Several types of psychotherapy have been utilized to help people with depression and pain in the post-operative process. One of the most practiced and studied forms of psychological therapy is cognitive behavior therapy (CBT). Cognitive therapy focuses on a patient's moods and thoughts. Behavioral therapy specifically targets actions and behaviors. CBT blends cognitive and behavioral therapy to modify thought patterns in order to change moods and behaviors. An RCT to determine the efficacy of a CBT based physical therapy program versus general education in post-operative lumbar patients revealed that the CBT group had significantly greater decrease in pain and disability and increase in general health and physical performance compared with the education group at the 3-month follow-up [68]. Another RCT evaluated the effectiveness of an exercise program for chronic low back pain that combined rehabilitation program with a CBT component that encouraged self-reliance and normal movement of the spine resulted in significantly greater improvements in disability and back pain scores at 1 year compared usual primary care management. The CBT cohort missed far less work and used fewer health care resources; the authors concluded that this approach was more cost-effective that the usual primary care [69]. Furthermore, a systematic review by the Agency for Healthcare Research and Quality in 2018 concluded that psychological therapies

were associated with slightly greater improvement than usual care to treat both function and pain at short term, intermediate-term, and long term follow up [70]. Overall, these findings point to the interdependent relationship of physical and psychological therapy during rehabilitative process and the importance of further high-quality studies to determine which therapies and techniques are most effectives in different post-operative populations.

Mind Body Exercises

Another strategy thought to improve psychological and physical well-being are mind-body activities. Mind-body interventions are increasingly used by people with chronic musculoskeletal pain and anxiety to help manage their symptoms and improve well-being. Examples of mind-body therapies include biofeedback, meditation, guided imagery, yoga, and Tai Chi. The Global Spine Care Initiative for chronic non-invasive management of back and neck pain recommend exercise, yoga, cognitive behavioral therapies, acupuncture, biofeedback, progressive relaxation therapy [71]. There are no significant studies that examine the efficacy of these exercises in the post-operative spine patient however they have been studied in the non-operative spine patient. An individualized approach should be taken when recommending these practices and modifications may be necessary to ensure that they do not worsen the patient's pain or cause complications in the poster operative spine patient. Two of the most practiced and studied mind-body practices for low back pain are yoga and Tai Chi.

- **Yoga:** There are various styles of yoga but in general they all incorporate various movements and poses with breath and meditation exercises. When practiced regularly it improves strength, flexibility, and balance. It is also has been associated with improved mood and decreased anxiety.

  A Cochrane Review in 2017 on yoga for chronic back pain concluded that it has low to moderate evidence to improve function but did not meet minimum clinical importance for improving pain compared to the non-exercise control. Yoga was associated with more adverse events that than the non-exercise control but did not pose more risk that other back focused exercises. The review did not find that yoga was not associated with serious adverse events [72].
- **Tai Chi:** Is an ancient Chinese art that involves a series of slow, flowing movements and stretches combine with breath work. There are various styles of Tai Chi, the most common being Tai Chi Chen. According to the principles of traditional Chinese medicine, Tai Chi's movements can help stimulate the flow of

vital energy, and promote healing for a variety of health conditions including stress, anxiety, and improving balance. Improvement of self-efficacy and improving balance is among many of the proposed benefits of Tai Chi [73, 74].

A systematic review and meta-analysis of 7 RCTS in 2019 concluded that Tai Chi alone or as adjunct therapy may decrease pain intensity and improve function for patients with non-specific low back pain [75]. A literature review that encompassed over 500 trials for a variety of health conditions reported that no serious adverse events have been associated with the practice of Tai Chi. Therefore, it can be considered as a safe practice that has the potential to improve low back pain, balance, function, and other physiological and physiological benefits.

Bibliography and Further Reading

1. Balagué F, Mannion AF, Pellisé F, Cedraschi C. Non-specific low back pain. Lancet. 2012;379(9814):482–91. https://doi.org/10.1016/S0140-6736(11)60610-7. Epub 2011 Oct 6. Review. PubMed PMID: 21982256.
2. Shmagel A, Foley R, Ibrahim H. Epidemiology of chronic low back pain in US adults: data from the 2009–2010 National Health and Nutrition Examination Survey. Arthritis Care Res (Hoboken). 2016;68(11):1688–94. https://doi.org/10.1002/acr.22890. PubMed PMID: 26991822; PubMed Central PMCID: PMC5027174.
3. Gore M, Sadosky A, Stacey BR, Tai KS, Leslie D. The burden of chronic low back pain: clinical comorbidities, treatment patterns, and health care costs in usual care settings. Spine (Phila Pa 1976). 2012;37(11):E668–77. https://doi.org/10.1097/BRS.0b013e318241e5de. PubMed PMID: 22146287.
4. Wynne-Jones G, Cowen J, Jordan JL, Uthman O, Main CJ, Glozier N, van der Windt D. Absence from work and return to work in people with back pain: a systematic review and meta-analysis. Occup Environ Med. 2014;71(6):448–56. https://doi.org/10.1136/oemed-2013-101571. Epub 2013 Nov 1. Review. PubMed PMID: 24186944; PubMed Central PMCID: PMC4033140.
5. Weiss AJ. (Truven Health Analytics), Elixhauser A (AHRQ). Trends in operating room procedures in U.S. Hospitals, 2001–2011. HCUP Statistical Brief #171. March 2014. Agency for Healthcare Research and Quality, Rockville, MD. http://www.hcup-us.ahrq.gov/reports/statbriefs/SB 171-Operating-Room-Procedure-Trends.pdf.
6. Shields LB, Clark L, Glassman SD, Shields CB. Decreasing hospital length of stay following lumbar fusion utilizing multidisciplinary committee meetings involving surgeons and other caretakers. Surg Neurol Int. 2017;8:5. https://doi.org/10.4103/2152-7806.198732. eCollection 2017. PubMed PMID: 28217384; PubMed Central PMCID: PMC5288986.
7. Vespa J, Medina L, Armstrong DM. "Demographic turning points for the United States: population projections for 2020 to 2060," current population reports, P25-1144. Washington, DC: U.S. Census Bureau; 2020.
8. Martin BI, Mirza SK, Spina N, Spiker WR, Lawrence B, Brodke DS. Trends in lumbar fusion procedure rates and associated hospital costs for degenerative spinal diseases in the United

States, 2004 to 2015. Spine (Phila Pa 1976). 2019;44(5):369–76. https://doi.org/10.1097/BRS.0000000000002822. PubMed PMID: 30074971.
9. Kosztowski T, Gokaslan ZL. Determining the extent of lumbar discectomy in patients with herniated lumbar discs. Insights Neurosurg. 2016;1(1):2.
10. Marquez-Lara A, Nandyala SV, Fineberg SJ, Singh K. Current trends in demographics, practice, and in-hospital outcomes in cervical spine surgery: a national database analysis between 2002 and 2011. Spine (Phila Pa 1976). 2014;39(6):476–81. https://doi.org/10.1097/BRS.0000000000000165. PubMed PMID: 24365907.
11. Rushton A, White L, Heap A, Heneghan N. Evaluation of current surgeon practice for patients undergoing lumbar spinal fusion surgery in the United Kingdom. World J Orthop. 2015;6(6):483–90. https://doi.org/10.5312/wjo.v6.i6.483. eCollection 2015 Jul 18. PubMed PMID: 26191495; PubMed Central PMCID: PMC4501934.
12. Sheng J, Liu S, Wang Y, Cui R, Zhang X. The link between depression and chronic pain: neural mechanisms in the brain. Neural Plast. 2017;2017:9724371. https://doi.org/10.1155/2017/9724371. Epub 2017 Jun 19. Review. PubMed PMID: 28706741; PubMed Central PMCID: PMC5494581.
13. Hébert JJ, Abraham E, Wedderkopp N, Bigney E, Richardson E, Darling M, Hall H, Fisher CG, Rampersaud YR, Thomas KC, Jacobs WB, Johnson M, Paquet J, Attabib N, Jarzem P, Wai EK, Rasoulinejad P, Ahn H, Nataraj A, Stratton A, Manson N. Preoperative factors predict postoperative trajectories of pain and disability following surgery for degenerative lumbar spinal stenosis. Spine (Phila Pa 1976). 2020;45(21):E1421–30. https://doi.org/10.1097/BRS.0000000000003587. PubMed PMID: 32541610; PubMed Central PMCID: PMC7547903.
14. Burgess LC, Arundel J, Wainwright TW. The effect of preoperative education on psychological, clinical and economic outcomes in elective spinal surgery: a systematic review. Healthcare (Basel). 2019;7(1). https://doi.org/10.3390/healthcare7010048. Review. PubMed PMID: 30901875; PubMed Central PMCID: PMC6473918.
15. Strøm J, Bjerrum MB, Nielsen CV, Thisted CN, Nielsen TL, Laursen M, Jørgensen LB. Anxiety and depression in spine surgery-a systematic integrative review. Spine J. 2018;18(7):1272–85. https://doi.org/10.1016/j.spinee.2018.03.017. Epub 2018 Apr 9. PubMed PMID: 29649613.
16. Sand-Jecklin K. The impact of medical terminology on readability of patient education materials. J Community Health Nurs. 2007;24(2):119–29. https://doi.org/10.1080/07370010701316254. PubMed PMID: 17563283.
17. Waljee J, McGlinn EP, Sears ED, Chung KC. Patient expectations and patient-reported outcomes in surgery: a systematic review. Surgery. 2014;155(5):799–808. https://doi.org/10.1016/j.surg.2013.12.015. Epub 2013 Dec 16. Review. PubMed PMID: 24787107; PubMed Central PMCID: PMC4170731.
18. Fleege C, Arabmotlagh M, Almajali A, Rauschmann M. Pre- and postoperative fast-track treatment concepts in spinal surgery: patient information and patient cooperation. Orthopade. 2014;43(12):1062–4, 1066–9. https://doi.org/10.1007/s00132-014-3040-5. PubMed PMID: 25387654.
19. Lusardi MM, Fritz S, Middleton A, Allison L, Wingood M, Phillips E, Criss M, Verma S, Osborne J, Chui KK. Determining risk of falls in community dwelling older adults: a systematic review and meta-analysis using posttest probability. J Geriatr Phys Ther. 2017;40(1):1–36. https://doi.org/10.1519/JPT.0000000000000099. Review. PubMed PMID: 27537070; PubMed Central PMCID: PMC5158094.

20. Park SH. Tools for assessing fall risk in the elderly: a systematic review and meta-analysis. Aging Clin Exp Res. 2018;30(1):1–16. https://doi.org/10.1007/s40520-017-0749-0. Epub 2017 Apr 3. Review. PubMed PMID: 28374345.
21. Peer MA, Rush R, Gallacher PD, Gleeson N. Pre-surgery exercise and post-operative physical function of people undergoing knee replacement surgery: a systematic review and meta-analysis of randomized controlled trials. J Rehabil Med. 2017;49(4):304–15. https://doi.org/10.2340/16501977-2210. Review. PubMed PMID: 28352936.
22. Whittle J, Wischmeyer PE, Grocott MPW, Miller TE. Surgical prehabilitation: nutrition and exercise. Anesthesiol Clin. 2018;36(4):567–80. https://doi.org/10.1016/j.anclin.2018.07.013. Review. PubMed PMID: 30390779.
23. Lemanu DP, Singh PP, MacCormick AD, Arroll B, Hill AG. Effect of preoperative exercise on cardiorespiratory function and recovery after surgery: a systematic review. World J Surg. 2013;37(4):711–20. https://doi.org/10.1007/s00268-012-1886-4. Review. PubMed PMID: 23292047.
24. Mills E, Eyawo O, Lockhart I, Kelly S, Wu P, Ebbert JO. Smoking cessation reduces postoperative complications: a systematic review and meta-analysis. Am J Med. 2011;124(2):144–154. e8. https://doi.org/10.1016/j.amjmed.2010.09.013. Review. PubMed PMID: 21295194.
25. Jackson KL 2nd, Devine JG. The effects of smoking and smoking cessation on spine surgery: a systematic review of the literature. Glob Spine J. 2016;6(7):695–701. https://doi.org/10.1055/s-0036-1571285. Epub 2016 Jan 15. Review. PubMed PMID: 27781190; PubMed Central PMCID: PMC5077710.
26. Truntzer J, Vopat B, Feldstein M, Matityahu A. Smoking cessation and bone healing: optimal cessation timing. Eur J Orthop Surg Traumatol. 2015;25(2):211–5. https://doi.org/10.1007/s00590-014-1488-y. Epub 2014 May 31. Review. PubMed PMID: 24879610.
27. Kalinowski A, Humphreys K. Governmental standard drink definitions and low-risk alcohol consumption guidelines in 37 countries. Addiction. 2016;111(7):1293–8. https://doi.org/10.1111/add.13341. Epub 2016 Apr 13. PubMed PMID: 27073140.
28. Oppedal K, Møller AM, Pedersen B, Tønnesen H. Preoperative alcohol cessation prior to elective surgery. Cochrane Database Syst Rev. 2012;(7):CD008343. https://doi.org/10.1002/14651858.CD008343.pub2. Review. PubMed PMID: 22786514.
29. Deyo RA, Mirza SK, Martin BI, Kreuter W, Goodman DC, Jarvik JG. Trends, major medical complications, and charges associated with surgery for lumbar spinal stenosis in older adults. JAMA. 2010;303(13):1259–65. https://doi.org/10.1001/jama.2010.338. PubMed PMID: 20371784; PubMed Central PMCID: PMC2885954.
30. Guzman JZ, Iatridis JC, Skovrlj B, et al. Outcomes and complications of diabetes mellitus on patients undergoing degenerative lumbar spine surgery. Spine (Phila Pa 1976). 2014;39(19):1596–604. https://doi.org/10.1097/BRS.0000000000000482.
31. Keller U. Nutritional laboratory markers in malnutrition. J Clin Med. 2019;8(6):775. Published 2019 May 31. https://doi.org/10.3390/jcm8060775.
32. Cross MB, Yi PH, Thomas CF, Garcia J, Della Valle CJ. Evaluation of malnutrition in orthopaedic surgery. J Am Acad Orthop Surg. 2014;22(3):193–9. https://doi.org/10.5435/JAAOS-22-03-193. Review. PubMed PMID: 24603829.
33. Kudo D, Miyakoshi N, Hongo M, Kasukawa Y, Ishikawa Y, Mizutani T, Shimada Y. Relationship between preoperative serum rapid turnover proteins and early-stage surgical wound infection after spine surgery. Eur Spine J. 2017;26(12):3156–61. https://doi.org/10.1007/s00586-016-4855-z. Epub 2016 Nov 10. PubMed PMID: 27832364.
34. West MA, Wischmeyer PE, Grocott MPW. Prehabilitation and nutritional support to improve perioperative outcomes. Curr Anesthesiol Rep. 2017;7(4):340–9. https://doi.org/10.1007/

s40140-017-0245-2. Epub 2017 Nov 7. Review. PubMed PMID: 29200973; PubMed Central PMCID: PMC5696441.
35. Borloni B, Huettner H, Schuerholz T. Preoperative nutritional conditioning: why, when and how. Visc Med. 2019;35(5):299–304. https://doi.org/10.1159/000503041. Epub 2019 Sep 10. Review. PubMed PMID: 31768393; PubMed Central PMCID: PMC6873024.
36. Lotzke H, Brisby H, Gutke A, Hägg O, Jakobsson M, Smeets R, Lundberg M. A person-centered prehabilitation program based on cognitive-behavioral physical therapy for patients scheduled for lumbar fusion surgery: a randomized controlled trial. Phys Ther. 2019;99(8):1069–88. https://doi.org/10.1093/ptj/pzz020. PubMed PMID: 30951604; PubMed Central PMCID: PMC6665875.
37. Cherkin DC, Sherman KJ, Balderson BH, Cook AJ, Anderson ML, Hawkes RJ, Hansen KE, Turner JA. Effect of mindfulness-based stress reduction vs cognitive behavioral therapy or usual care on back pain and functional limitations in adults with chronic low back pain: a randomized clinical trial. JAMA. 2016;315(12):1240–9. https://doi.org/10.1001/jama.2016.2323. PubMed PMID: 27002445; PubMed Central PMCID: PMC4914381.
38. Louw A, Diener I, Landers MR, Puentedura EJ. Preoperative pain neuroscience education for lumbar radiculopathy: a multicenter randomized controlled trial with 1-year follow-up. Spine (Phila Pa 1976). 2014;39(18):1449–57. https://doi.org/10.1097/BRS.0000000000000444. PubMed PMID: 24875964.
39. Lotzke H. Person-centred prehabilitation program to improve functioning in patients with severe low back pain planned for lumbar fusion surgery. Diss Univ Gothenbg. 2019:93–99.
40. Brindle M, Nelson G, Lobo DN, Ljungqvist O, Gustafsson UO. Recommendations from the ERAS® Society for standards for the development of enhanced recovery after surgery guidelines. BJS Open. 2020;4(1):157–63. https://doi.org/10.1002/bjs5.50238. Epub 2019 Dec 2. PubMed PMID: 32011810; PubMed Central PMCID: PMC6996628.
41. Elsarrag M, Soldozy S, Patel P, Norat P, Sokolowski JD, Park MS, Tvrdik P, Kalani MYS. Enhanced recovery after spine surgery: a systematic review. Neurosurg Focus. 2019;46(4):E3. https://doi.org/10.3171/2019.1.FOCUS18700. PubMed PMID: 30933920.
42. Gustafsson UO, Scott MJ, Hubner M, Nygren J, Demartines N, Francis N, Rockall TA, Young-Fadok TM, Hill AG, Soop M, de Boer HD, Urman RD, Chang GJ, Fichera A, Kessler H, Grass F, Whang EE, Fawcett WJ, Carli F, Lobo DN, Rollins KE, Balfour A, Baldini G, Riedel B, Ljungqvist O. Guidelines for perioperative care in elective colorectal surgery: enhanced recovery after surgery (ERAS®) Society recommendations: 2018. World J Surg. 2019;43(3):659–95. https://doi.org/10.1007/s00268-018-4844-y. Review. PubMed PMID: 30426190.
43. Morley JE, Malmstrom TK, Miller DK. A simple frailty questionnaire (FRAIL) predicts outcomes in middle aged African Americans. J Nutr Health Aging. 2012;16(7):601–8. https://doi.org/10.1007/s12603-012-0084-2. PubMed PMID: 22836700; PubMed Central PMCID: PMC4515112.
44. Mohanty S, Liu Y, Paruch JL, Kmiecik TE, Cohen ME, Ko CY, Bilimoria KY. Risk of discharge to postacute care: a patient-centered outcome for the American College of Surgeons National Surgical Quality Improvement Program surgical risk calculator. JAMA Surg. 2015;150(5):480–4. https://doi.org/10.1001/jamasurg.2014.3176. PubMed PMID: 25806660.
45. Cuesta-Vargas AI, Neblett R, Chiarotto A, Kregel J, Nijs J, van Wilgen CP, Pitance L, Knezevic A, Gatchel RJ, Mayer TG, Viti C, Roldan-Jiménez C, Testa M, Caumo W, Jeremic-Knezevic M, Luciano JV. Dimensionality and reliability of the central sensitization inventory in a pooled multicountry sample. J Pain. 2018;19(3):317–29. https://doi.org/10.1016/j.jpain.2017.11.006. Epub 2017 Dec 2. PubMed PMID: 29198933.
46. Puentedura EJ, Flynn T. Combining manual therapy with pain neuroscience education in the treatment of chronic low back pain: a narrative review of the literature. Physiother Theory Pract. 2016;32(5):408–14. https://doi.org/10.1080/09593985.2016.1194663. Epub 2016 Jun 30. Review. PubMed PMID: 27362980.

47. Uth N, Sørensen H, Overgaard K, Pedersen PK. Estimation of VO2max from the ratio between HRmax and HRrestDOUBLEHYPHEN—the Heart Rate Ratio Method. Eur J Appl Physiol 2004;91(1):111–115. doi: https://doi.org/10.1007/s00421-003-0988-y. Epub 2003 Nov 18. PubMed PMID: 14624296.
48. Kline GM, Porcari JP, Hintermeister R, Freedson PS, Ward A, McCarron RF, Ross J, Rippe JM. Estimation of VO2max from a one-mile track walk, gender, age, and body weight. Med Sci Sports Exerc. 1987;19(3):253–9. PubMed PMID: 3600239.
49. Gerbershagen HJ, Aduckathil S, van Wijck AJ, Peelen LM, Kalkman CJ, Meissner W. Pain intensity on the first day after surgery: a prospective cohort study comparing 179 surgical procedures. Anesthesiology. 2013;118(4):934–44. https://doi.org/10.1097/ALN.0b013e31828866b3. PubMed PMID: 23392233.
50. Antunes RS, de Macedo BG, Amaral Tda S, Gomes Hde A, Pereira LS, Rocha FL. Pain, kinesiophobia and quality of life in chronic low back pain and depression. Acta Ortop Bras. 2013;21(1):27–9. https://doi.org/10.1590/S1413-78522013000100005. PubMed PMID: 24453639; PubMed Central PMCID: PMC3862015.
51. Tarnanen SP, Neva MH, Häkkinen K, Kankaanpää M, Ylinen J, Kraemer WJ, Newton RU, Häkkinen A. Neutral spine control exercises in rehabilitation after lumbar spine fusion. J Strength Cond Res. 2014;28(7):2018–25. https://doi.org/10.1519/JSC.0000000000000334. PMID: 24343327
52. Kernc D, Strojnik V, Vengust R. Early initiation of a strength training based rehabilitation after lumbar spine fusion improves core muscle strength: a randomized controlled trial. J Orthop Surg Res. 2018;13:151. https://doi.org/10.1186/s13018-018-0853-7.
53. Valdivieso P, Franchi MV, Gerber C, Flück M. Does a better perfusion of deconditioned muscle tissue release chronic low Back pain? Front Med (Lausanne). 2018;5:77. https://doi.org/10.3389/fmed.2018.00077. eCollection 2018. PubMed PMID: 29616222; PubMed Central PMCID: PMC5869187.
54. Gilmore SJ, Hahne AJ, Davidson M, McClelland JA. Predictors of substantial improvement in physical function six months after lumbar surgery: is early post-operative walking important? A prospective cohort study. BMC Musculoskelet Disord. 2019;20(1):418. https://doi.org/10.1186/s12891-019-2806-7. PubMed PMID: 31506099; PubMed Central PMCID: PMC6737667.
55. Shi Z, Zhou H, Lu L, Pan B, Wei Z, Yao X, Kang Y, Liu L, Feng S. Aquatic exercises in the treatment of low back pain: a systematic review of the literature and meta-analysis of eight studies. Am J Phys Med Rehabil. 2018;97(2):116–22. https://doi.org/10.1097/PHM.0000000000000801. PubMed PMID: 28759476.
56. Kim YS, Park J, Shim JK. Effects of aquatic backward locomotion exercise and progressive resistance exercise on lumbar extension strength in patients who have undergone lumbar diskectomy. Arch Phys Med Rehabil. 2010;91(2):208–14. https://doi.org/10.1016/j.apmr.2009.10.014. PubMed PMID: 20159123.
57. Villalta EM, Peiris CL. Early aquatic physical therapy improves function and does not increase risk of wound-related adverse events for adults after orthopedic surgery: a systematic review and meta-analysis. Arch Phys Med Rehabil. 2013;94(1):138–48. https://doi.org/10.1016/j.apmr.2012.07.020. Epub 2012 Aug 7. Review. PubMed PMID.
58. Waschke A, Hartmann C, Walter J, Dünisch P, Wahnschaff F, Kalff R, Ewald C. Denervation and atrophy of paraspinal muscles after open lumbar interbody fusion is associated with clinical outcomeDOUBLEHYPHEN—electromyographic and CT-volumetric investigation of 30 patients. Acta Neurochir. 2014;156(2):235–44. https://doi.org/10.1007/s00701-013-1981-9. Epub 2014 Jan 3. PubMed PMID: 24384989.
59. Giangarra CE. Clinical orthopaedic rehabilitation. 4th ed. Philadelphia: Elsevier; 2018.
60. Piperno M, Hellio le Graverand MP, Reboul P, Mathieu P, Tron AM, Perrin G, Peschard MJ, Richard M, Vignon E. Phospholipase A2 activity in herniated lumbar discs. Clinical

correlations and inhibition by piroxicam. Spine (Phila Pa 1976). 1997;22(18):2061–5. https://doi.org/10.1097/00007632-199709150-00001. PubMed PMID: 9322315.
61. Basson A, Olivier B, Ellis R, Coppieters M, Stewart A, Mudzi W. The effectiveness of neural mobilization for neuromusculoskeletal conditions: a systematic review and meta-analysis. J Orthop Sports Phys Ther. 2017;47(9):593–615. https://doi.org/10.2519/jospt.2017.7117. Epub 2017 Jul 13. Review. PubMed PMID: 28704626.
62. Scrimshaw SV, Maher CG. Randomized controlled trial of neural mobilization after spinal surgery. Spine (Phila Pa 1976). 2001;26(24):2647–52. https://doi.org/10.1097/00007632-200112150-00002. PubMed PMID: 11740347.
63. Abbaspoor Z, Akbari M, Najar S. Effect of foot and hand massage in post-cesarean section pain control: a randomized control trial. Pain Manag Nurs. 2014;15(1):132–6. https://doi.org/10.1016/j.pmn.2012.07.008. Epub 2013 Jan 24. PubMed PMID: 23352729.
64. Le Blanc-Louvry I, Costaglioli B, Boulon C, Leroi AM, Ducrotte P. Does mechanical massage of the abdominal wall after colectomy reduce postoperative pain and shorten the duration of ileus? Results of a randomized study. J Gastrointest Surg. 2002;6(1):43–9. https://doi.org/10.1016/s1091-255x(01)00009-9. PubMed PMID: 11986017.
65. Bauer BA, Cutshall SM, Wentworth LJ, Engen D, Messner PK, Wood CM, Brekke KM, Kelly RF, Sundt TM 3rd. Effect of massage therapy on pain, anxiety, and tension after cardiac surgery: a randomized study. Complement Ther Clin Pract. 2010;16(2):70–5. https://doi.org/10.1016/j.ctcp.2009.06.012. Epub 2009 Jul 14. PubMed PMID: 20347836.
66. Furlan AD, Giraldo M, Baskwill A, Irvin E, Imamura M. Massage for low-back pain. Cochrane Database Syst Rev. 2015;(9):CD001929. https://doi.org/10.1002/14651858.CD001929.pub3. Review. PubMed PMID: 26329399.
67. Wilson BR, Tringale KR, Hirshman BR, Zhou T, Umlauf A, Taylor WR, Ciacci JD, Carter BS, Chen CC. Depression after spinal surgery: a comparative analysis of the California Outcomes Database. Mayo Clin Proc. 2017;92(1):88–97. https://doi.org/10.1016/j.mayocp.2016.06.030. Epub 2016 Nov 9. PubMed PMID: 27836112; PubMed Central PMCID: PMC5426910.
68. Archer KR, Devin CJ, Vanston SW, Koyama T, Phillips SE, Mathis SL, George SZ, McGirt MJ, Spengler DM, Aaronson OS, Cheng JS, Wegener ST. Cognitive-behavioral-based physical therapy for patients with chronic pain undergoing lumbar spine surgery: a randomized controlled trial. J Pain. 2016;17(1):76–89. https://doi.org/10.1016/j.jpain.2015.09.013. Epub 2015 Oct 23. PubMed PMID: 26476267; PubMed Central PMCID: PMC4709178.
69. Moffett JK, Torgerson D, Bell-Syer S, Jackson D, Llewlyn-Phillips H, Farrin A, Barber J. Randomised controlled trial of exercise for low back pain: clinical outcomes, costs, and preferences. BMJ. 1999;319(7205):279–83. https://doi.org/10.1136/bmj.319.7205.279. PubMed PMID: 10426734; PubMed Central PMCID: PMC28176
70. Skelly AC, Chou R, Dettori JR, Turner JA, Friedly JL, Rundell SD, Fu R, Brodt ED, Wasson N, Winter C, Ferguson AJR. Noninvasive nonpharmacological treatment for chronic pain: a systematic review [Internet]. Rockville (MD): Agency for Healthcare Research and Quality (US); 2018. Report No.: 18-EHC013-EF. PMID: 30179389
71. Chou R, Côté P, Randhawa K, Torres P, Yu H, Nordin M, Hurwitz EL, Haldeman S, Cedraschi C. The Global Spine Care Initiative: applying evidence-based guidelines on the non-invasive management of back and neck pain to lowand middle-income communities. Eur Spine J. 2018;27(Suppl 6):851–60. https://doi.org/10.1007/s00586-017-5433-8. Epub 2018 Feb 19. PMID: 29460009
72. Wieland LS, Skoetz N, Pilkington K, Vempati R, D'Adamo CR, Berman BM. Yoga treatment for chronic non-specific low back pain. Cochrane Database Syst Rev. 2017;1:CD010671. https://doi.org/10.1002/14651858.CD010671.pub2. Review. PubMed PMID: 28076926; PubMed Central PMCID: PMC5294833.
73. Tong Y, Chai L, Lei S, Liu M, Yang L. Effects of Tai Chi on self-efficacy: a systematic review. Evid Based Complement Alternat Med. 2018;2018:1701372. https://doi.org/10.1155/2018/1701372. eCollection 2018. Review. PubMed PMID: 30186352; PubMed Central PMCID: PMC6114250.

74. Zhong D, Xiao Q, Xiao X, Li Y, Ye J, Xia L, Zhang C, Li J, Zheng H, Jin R. Tai Chi for improving balance and reducing falls: An overview of 14 systematic reviews. Ann Phys Rehabil Med. 2020;63(6):505–17. https://doi.org/10.1016/j.rehab.2019.12.008. Epub 2020 Jan 22. Review. PubMed PMID: 31981834.
75. Qin J, Zhang Y, Wu L, He Z, Huang J, Tao J, Chen L. Effect of Tai Chi alone or as additional therapy on low back pain: systematic review and meta-analysis of randomized controlled trials. Medicine (Baltimore). 2019;98(37):e17099. https://doi.org/10.1097/MD.0000000000017099. PubMed PMID: 31517838; PubMed Central PMCID: PMC6750325.

15 Preoperative Optimization and Intraoperative Enhanced Recovery Principles for Patients Undergoing Spine Surgery

Megan G. Maxwell, Kehinde O. Odedosu, and Bryan T. Romito

Preoperative Assessment for Primary Care Providers

Advanced age, diabetes, heart disease, and lung disease have been identified as comorbidities that increase operative risk [1]. Once the patient and surgeon have agreed to pursue spine surgery, a multidisciplinary, systematic approach to assessing and optimizing these risk factors should occur in order to achieve a successful outcome. This often involves collaboration between the internist, consulting specialists, surgeons, and anesthesiologists. The preoperative medical optimization visit should not only include a thorough review of the patient's medical history, vital signs, and physical exam but should also include a discussion of functional status and new or concerning symptoms. It is imperative to focus on the stability and severity of chronic medical conditions, including a detailed current medication list and medication adherence. The goal of this visit is not to "clear" a patient for surgery but instead to optimize these complex patients while applying guideline-driven recommendations and testing when applicable. The following sections will review a system-based risk assessment for patients undergoing spine surgery.

M. G. Maxwell (✉)
University of Texas Southwestern Medical Center, Dallas, TX, USA
e-mail: Megan.maxwell@utsouthwestern.edu

K. O. Odedosu
University of Texas Southwestern Medical Center, Dallas, TX, USA

Parkland Health and Hospital Systems, Dallas, TX, USA
e-mail: kehinde.odedosu@utsouthwestern.edu

B. T. Romito
Department of Anesthesiology & Pain Management, University of Texas Southwestern Medical Center, Dallas, TX, USA
e-mail: bryan.romito@utsouthwestern.edu

© The Author(s), under exclusive license to Springer Nature Switzerland AG 2022

C. E. Noe (ed.), *Multidisciplinary Spine Care*,
https://doi.org/10.1007/978-3-031-04990-3_15

Cardiovascular Risk Assessment and Recommendations

Patients undergoing non-cardiac surgery are at risk for major adverse cardiac events (MACE) [2]. Therefore, a meticulous approach to preoperative cardiac evaluation should follow the 2014 American College of Cardiology (ACC) and American Heart Association (AHA) guidelines. In August 2014, the ACC/AHA updated their guidelines for cardiovascular evaluation and care of the patient undergoing non-cardiac surgery [3]. The guidelines provide a step-by-step approach to preoperative risk assessment and management, including a discussion surrounding several surgical risk calculators. Based on the guidelines, determination of the urgency and risk of surgery is a critical first step in the evaluation. Emergency or urgent procedures have been shown to increase risk of complications [4].

Procedure-Specific Risks

Fluid shifts, blood loss, and the degree of hemodynamic compromise have been reported to increase the risk of surgical complications [5]. While previous versions of guidelines classified the risk of the procedure as low, intermediate, or high, the current guidelines recommend stratifying risk into two simple categories: low and elevated risk [3, 6]. Calculation of this risk is based on a combination of both patient and surgical characteristics. Low-risk surgery is characterized as a <1% risk of MACE, while surgery with a risk of MACE of ≥1% is classified as elevated risk [3].

Patient-Specific Risks

The value of a comprehensive history and physical examination in the preoperative evaluation cannot be overstated. By understanding a patient's medical history, including identifying and assessing factors that can influence outcomes, the clinician can use evidence-based guidelines to guide decision surrounding preoperative testing and also to predict post-operative complications. The patient should ideally be evaluated within a month of the planned surgery. An assessment of functional capacity is a crucial aspect of this evaluation, with metabolic equivalents (METS) representing the primary method used to assess functional status. Functional status is defined as excellent (>10 METS), good (7–10 METS), moderate (4–6 METS), and poor (<4 METS). METS >4 has been considered adequate to proceed with surgery in the absence of active cardiac conditions or concerning clinical symptoms [3]. Recently published trials have found the Duke Activity Status Index (DASI), a questionnaire used to determine a patient's ability to achieve an appropriate METS, to be a more accurate measurement of a patient's actual functional capacity compared to a clinician's subjective assessment [7–9]. Moreover, DASI performed better when compared to stress testing and cardiac biomarkers like brain natriuretic peptide [8]. The DASI score is calculated by tallying the points of all performed activities, with higher numbers indicating a higher functional status. The score can

then be converted to METS. Based on a study from Wijeysundera et al., a DASI score of 34 or less means that the patient is at risk for MACE and post-operative complications [9].

Risk Calculators

Cardiac risk calculators have also been used to assess perioperative cardiac complications. The use of these calculators has evolved over time, and no single tool is perfect. The most cited and externally validated tool is the Revised Cardiac Risk Index (RCRI) [10]. The RCRI consists of six risk factors that increase a patient's risk of cardiac complications. These six risk factors include: history of congestive heart failure, history of cerebrovascular disease, history of ischemic heart disease, high-risk surgery, creatinine >2 mg/dl, and diabetes requiring insulin [10]. Per the 2014 ACC/AHA guidelines, a patient with 0 or 1 risk factor is considered low risk while ≥2 risk factors is considered elevated risk [3]. Other risk calculators include the Gupta Myocardial Infarction and Cardiac Arrest calculator and the American College of Surgeons National Surgical Quality Improvement Program (ACS-NSQIP) surgical risk calculator. Using the NSQIP database, Gupta et al. identified five unique predictors of MACE [11]. These include type of surgery, functional status, abnormal creatinine, age, and American Society of Anesthesiologists (ASA) physical status classification. The ACS-NSQIP calculator, although cumbersome, has garnered considerable attention as it is a more comprehensive risk calculator that enables procedure-specific risk assessment [12]. Both of these calculators are available online. Based on review of the evidence, these newer risk indices may perform better but should be applied in the setting in which they were studied [13].

Cardiovascular Clinical Risk Factors

Coronary Artery Disease

Livhits et al. observed that patients with a history of coronary artery disease (CAD) are at risk for cardiac complications, with recent acute coronary syndrome associated with an especially greater risk [14]. They also noted the postoperative myocardial infarction (MI) rate decreased as the length of time from MI to operation increased (0–30 days = 32.8%, 31–60 days = 18.7%, 61–90 days = 8.4%, 91–180 days = 5.9%), as did 30-day mortality (0–30 days = 14.2%, 31–60 days = 11.5%, 61–90 days = 10.5%, 91–180 days = 9.9%). The risk was modifiable by how long from the planned surgical date the myocardial infarction occurred, if revascularization was performed, and the type of revascularization [15]. Analyzing the data, surgery within 1 month of an acute coronary syndrome is associated with a significantly increased risk of MACE. The 2014 ACC/AHA guidelines recommend delaying elective surgery for at least 2 months following an MI [3]. Patients with stable CAD, without recent acute MI, red flags, or worrisome clinical symptoms should be able to proceed to surgery without any further risk stratification.

Valvular Heart Disease

Patients with valvular heart disease undergoing non-cardiac surgery have an increased risk of MACE in the postoperative period [16]. This risk is dependent on the valve, the severity of the disease, and the type of non-cardiac surgery. The 2014 ACC/AHA guidelines recommend an echocardiogram for moderate-to-severe valvular disease if an echocardiogram has not been performed within 1 year or there is a change in the clinical status or physical exam [3]. For patients with moderate-to-severe valvular disease, a multidisciplinary preoperative discussion with the cardiologist, surgeon, and anesthesiologist is crucial to develop a perioperative plan as additional hemodynamic monitoring may be needed. If indicated, valvular intervention prior to surgery can reduce risk [16].

Heart Failure

Heart failure is a significant cardiac risk factor [17]. Although evidence is scarce on how asymptomatic heart failure impacts postoperative outcomes, the literature and guidelines specify that patients with decompensated heart failure should have surgery delayed unless the procedure is emergent [3]. Several studies have shown that patients with a left ventricular ejection fraction of <35% may also have an increased risk of postoperative complications [18]. For this reason, close hemodynamic monitoring in the perioperative period may be warranted. Optimal goal directed medical therapy for the treatment of heart failure can mitigate some of the risk [19].

Pulmonary Risk Assessment and Recommendations

Pulmonary complications are an underappreciated cause of perioperative mortality and morbidity [20, 21]. A thorough history and physical examination, including evaluation of tobacco history, remains the mainstay of the preoperative pulmonary assessment. Pulmonary risk assessment can be further divided into patient-related risk factors and procedure-related risk factors. Patient-related factors include asthma, tobacco use, heart failure, chronic obstructive pulmonary disease (COPD), obesity, and obstructive sleep apnea (OSA). Procedure-related risk is largely determined by the surgical site, with surgery closer to the diaphragm associated with greater risk [20, 21].

Several risk indices have been developed to predict the risk of postoperative pulmonary complications, including pneumonia and respiratory failure [22, 23]. Of note, many of these pulmonary risk indices exclude OSA. Although many cases likely go undiagnosed, the prevalence of OSA is increasing in the population. Patients with OSA require close monitoring in the perioperative setting because of their increased risk for complications [24, 25]. Consequently, all patients should be screened for OSA preoperatively. The most widely used OSA screening tool is the STOP-BANG questionnaire. Chung et al. demonstrated that the STOP-BANG questionnaire is a reliable and easy tool to screen for OSA. Each positive risk factor is scored one point, with a total score ≥3 indicating an increased risk for OSA [24, 25].

Pulmonary Risk Reduction

Chronic pulmonary conditions such as asthma and COPD should be optimized prior to surgery. Recent pulmonary function tests (PFTs) are helpful in assessing the degree of obstructive or restrictive lung disease. Pulmonary risk reduction can occur in several stages during the course of care. Preoperatively, the internist or pulmonologist should focus on patient education, including awareness of OSA risk, tobacco cessation when applicable, optimization of underlying disease, and the use of bronchodilators and steroids when indicated. Postoperative risk reduction measures include continuation of bronchodilators, incentive spirometry, analgesia, and the use of continuous positive airway pressure as needed [26].

Hematologic Risk Assessment and Recommendations

Preoperative Anemia

Approximately one-third of patients evaluated during their preoperative assessment will be diagnosed with anemia [27]. As such, this represents a commonly-encountered and modifiable risk factor in the presurgical patient. Preoperative anemia is often attributed to iron-deficiency anemia and anemia of inflammation (also known as anemia of chronic disease) [28]. While anemia has been classically defined by the World Health Organization (WHO) as a hemoglobin <13 g/dL in men and <12 g/dL in women, a universal threshold of <13 g/dL in all perioperative patients may be considered [28, 29]. A retrospective analysis of 227,425 patients undergoing major noncardiac surgery showed an increased 30-day risk of mortality (odds ratio 1.42, confidence interval 1.31–1.54) and morbidity (odds ratio 1.35, confidence interval 1.3–1.4) in patients with preoperative anemia after controlling for other confounding variables [27]. As a first step in the risk assessment, consideration should be given to the cause of the anemia. This should include a thorough medical history and physical exam. Spine surgery may be postponed depending on the severity of anemia, diagnostic workup required, and urgency of the planned procedure.

Causes of Preoperative Anemia and Management

The management of preoperative anemia should employ a Patient Blood Management (PBM) approach [30–32]. The WHO defines PBM as "patient-focused, evidence-based and systematic approach to optimize the management of patients and transfusion of blood products for quality and effective patient care. It is designed to improve patient outcomes through the safe and rational use of blood and blood products and by minimizing unnecessary exposure to blood products" [32]. The core principles of PBM include: management of anemia, multimodal approach to blood conservation, optimize hemostasis, patient-centered care and decision making [30]. By adhering to these principles, providers can minimize postoperative

complications associated with anemia and the unnecessary use of blood transfusion. In most circumstances, anemia can be corrected with treatment prior to the planned surgery, which can also decrease the need for blood transfusion in the perioperative period [33, 34].

Iron Deficiency Anemia

Both oral and intravenous (IV) iron therapy are treatment options for iron deficiency anemia. Oral iron is often the first treatment choice provided there is sufficient time prior to surgery to replete iron stores and raise the hemoglobin value. Nonetheless, the efficacy of oral iron compared to IV iron in the presurgical patient with iron deficiency anemia has been debated [35]. Poor gastrointestinal absorption, patient intolerance, and an acute or chronic inflammatory state (referred to as anemia of chronic disease) may alter the efficacy of oral iron [34, 36]. Whichever approach is chosen, sufficient time must be given for either oral or IV iron to optimize hemoglobin and iron stores [33].

Anemia of Chronic Disease

Anemia of chronic disease, as seen in chronic renal insufficiency, is often driven by a decrease in erythropoietin production and iron metabolism [33, 34, 36]. Directed therapy involves not only addressing the chronic disease but also replenishing erythropoietin [36]. Erythropoiesis-stimulating agent (ESA) is the driver to produce red blood cells and works in synergy with iron, highlighting the importance of appropriate iron stores. There is growing evidence that the use of ESA in the preoperative management of anemia can decrease the need for allogenic blood transfusion [37–40]. However, the use of ESA in the presurgical patient has been a source of contention, particularly surrounding the matter of thrombosis. Evaluating the use of preoperative ESA in patients undergoing knee or hip arthroplasty, Alsaleh et al. found no significant difference in the risk of thromboembolism in patients who received ESA compared to patients who did not [37]. Additionally, the authors observed that there was a decreased rate of blood transfusion in the group of patients who received ESA. With appropriate patient selection, attention to the degree of blood loss for the planned surgery, and the use of a strong PBM program, ESA can decrease the need for blood transfusion in the perioperative period [32, 37–40].

Endocrine Risk Assessment and Recommendations

There is a significant amount of evidence linking hyperglycemia to poor outcomes in patients undergoing surgery [41, 42]. Poorly-controlled diabetes, regardless of the duration, can have detrimental effects in the postoperative period. Hyperglycemia is known to impair leukocyte function [43]. Unsurprisingly there is an association

between poorly-controlled diabetes with hyperglycemia and an increased risk of surgical site infection [43, 44]. When a patient arrives for a preoperative visit, it is important to determine their level of glycemic control, including their most recent hemoglobin A1C level. This value is not only useful for diagnosing diabetes (≥6.5% being diagnostic of diabetes) but is also an essential tool for assessing glycemic control. The ideal hemoglobin A1C for elective surgery has been a source of contention in the literature. Although there is no defined threshold preoperative hemoglobin A1C to proceed with surgery, a value <8% appears to correspond to a decrease risk of postoperative complications [45]. Achieving this goal requires timely referral to the managing clinician for optimization, which may include titration of existing medications or the addition of other agents. Additionally, the preoperative visit will include a discussion of the management of both oral and injectable diabetic medications on the day prior to surgery and the day of surgery. Although hemoglobin A1C and blood glucose have their limitations, they remain valuable indicators of a patient's glycemic control.

There is a growing body of literature describing the use of other markers that reflect glycemic control in the perioperative period [46]. One of these markers is serum fructosamine, which measures the level of glycated serum proteins like albumin. Fructosamine reflects mean glucose levels over 14–21 days and may be a better marker for poor glycemic control than hemoglobin A1C [46]. In a large prospective multicenter study, it was shown to be an excellent predictor of adverse outcomes in patients following total knee arthroplasty. In this same population, fructosamine better reflected glycemic control, possessed greater predictive power for complications, and responded faster to treatment compared to hemoglobin A1C [46].

Regardless of which screening tool is used, improving outcomes in the diabetic patient undergoing surgery starts with optimal glucose control. In general, it is recommended to stop oral hypoglycemic medications on the morning of surgery, stop prandial insulin, and continue basal insulin with dose adjustments based on individual patient needs.

Introduction to the Anesthesia Preoperative Evaluation

In addition to the preoperative assessment by primary care providers, further patient optimization and risk stratification can occur in the immediate preoperative setting by anesthesiology providers. Perioperative anesthesia evaluation and education of spine surgery patients not only helps to improve patient satisfaction but also allows the anesthesia team guide medical optimization in collaboration with patients' primary care providers, consulting specialists, and surgical team [47]. Furthermore, evaluation in an anesthesia preoperative clinic allows for a thorough medical assessment, laboratory and cardiac testing prior to the day of surgery, focused conversations regarding anesthetic risks, and an introduction to Enhanced Recovery after Surgery (ERAS) protocols.

The anesthesia preoperative clinic evaluation may begin by triaging patients based on complex vs simple spinal surgery and surgical urgency. Simple spine

surgery includes microdiscectomy and laminectomy for degenerative disease, while more complex surgery consists of spinal instrumentation, trauma, and tumor surgery [48]. While spinal surgeries are usually elective and thus allow time for adequate optimization, surgery for oncologic indications may be more time sensitive. Urgent procedures to address acute myelopathies or cauda equina syndrome mandate rapid evaluation [48].

System-Based Approach to the Preoperative Anesthesia Evaluation

Anesthetic preoperative evaluation usually follows a system-based approach. Special attention should be paid to preoperative vital signs to ensure not only that systemic blood pressure is optimized, but also well documented so that intraoperative hemodynamic goals can target 20% of the preoperative baseline. Even stricter blood pressure control may be required in patients with myelopathy or trauma to the spine [49]. The preoperative clinic is an ideal setting to discuss preoperative medication administration. Many clinics have established guidelines to instruct patients on which medications to continue perioperatively and which medications to hold. Depending on comorbidities, certain medications such as beta-blockers, pulmonary hypertension agents, and antiepileptics should be continued uninterrupted. Other medications like angiotensin converting enzyme (ACE) inhibitors and anticoagulants will likely need to be held. Discontinuation of anticoagulation or antiplatelet agents is advised only after consultation with the prescribing provider, especially in patients with recent cardiac stents or surgery.

Airway

A detailed airway evaluation is critical in spine surgery patients, especially those presenting for cervical or upper thoracic spine surgery. Careful documentation of the extent of mouth opening, neck range of motion, and symptoms such as pain or paresthesia elicited with neck movement should all be included. Many of these patients have disease pathology, such as rheumatoid arthritis or ankylosing spondylitis, that may limit neck mobility or distort airway anatomy. Preoperative airway evaluation will help the anesthesia team decide upon an appropriate intubation technique. For those patients with myelopathy or evidence of an unstable cervical spine, the anesthesiologist may choose to alter the intubation technique and perform an awake fiberoptic intubation [50].

Pulmonary

As mentioned above, chronic pulmonary conditions such as asthma or COPD should be optimized as much as possible prior to surgery. Recent PFTs are helpful

in assessing the degree of obstructive or restrictive lung disease. Patients presenting for spine surgery often have restrictive lung disease related to their spinal pathology or curvature, which can decrease their vital capacity and total lung capacity. Special consideration should be given to patients with severe restrictive lung disease, as this can progress to pulmonary hypertension [50]. When appropriate, the anesthesia preoperative clinic should work in conjunction with the patient's primary care provider and/or pulmonologist to optimize their pulmonary medication regimen and compliance prior to surgery. Major thoracic spine surgery may require one lung ventilation, and careful preoperative assessment and optimization is critical for these patients.

Cardiovascular

Approach to the preoperative cardiac evaluation should follow the updated ACC/AHA guidelines as discussed above. The decision to obtain a preoperative electrocardiogram (ECG), echocardiogram, stress testing, and even coronary angiography should be directed by evidenced-based protocols for non-cardiac surgery. However, specific to spine surgery, special consideration should be given to patients with significant restrictive lung disease as this may result in pulmonary hypertension or cor pulmonale. Preoperative assessment of functional activity may also be limited in these patients secondary to pain or myelopathy, therefore cardiac risk stratification based activity level (or METS achieved) may be more difficult to discern.

Spine surgery is usually performed in the prone position. This can lead to decreased venous return and left ventricular compliance, which can subsequently cause a reduction in cardiac output [50]. Therefore, a thorough documentation of prior cardiac history should be pursued, noting any structural or valvular defects. Prior ischemic heart disease and/or history of arrhythmias is also important to investigate. Many of these diagnoses have implications for perioperative anticoagulation, therefore a clear history should be obtained. Complex heart disease may also change the anesthetic plan, increasing the need for more invasive monitoring, vascular access, or even requiring the assistance of anesthesia teams specializing in cardiac anesthesia. Arranging this ahead of time, along with a thorough discussion of anesthetic risks can help ensure there is no day of surgery delay. Specific conditions, such as pulmonary hypertension and congestive heart failure can be especially associated with increased perioperative morbidity and mortality [51]. Gathering medical records and engaging with a patient's primary care provider, cardiologist, or pulmonologist preoperatively to ensure optimization is critical, especially for more complex or high-risk spine surgeries.

Neuromuscular

Meticulous evaluation of preexisting neuromuscular symptoms is important in the preoperative evaluation of the spine patient. Existing motor or sensory deficits

should be clearly documented. This ensures that care can be taken during operative positioning and that the postoperative exam can be compared to preoperative exam. The presence of existing motor deficits, such as weakness, immobility or paralysis may change the anesthetic plan. For example, the anesthesiologist may choose to avoid the use of succinylcholine as these patients may have upregulation of acetylcholine receptors. The use of succinylcholine may precipitate a dangerous episode of hyperkalemia.

Positioning Considerations

Most spinal procedures are performed in the prone position and as such, special considerations should be evaluated preoperatively. Attention to preoperative skin bruising or limited neck or extremity mobility should be noted so that care can be taken intraoperatively. The anesthesiologist performing the case may choose to provide extra padding to sensitive areas and avoid manipulation of the neck or extremities that may cause pain when patient is awake. For lumbar and lower thoracic surgery in the prone position, the arms may be tucked at the sides or placed in the "prone superman" position [50].

As above, prone positioning can also have effects on cardiovascular physiology. Abdominal compression can cause a decrease in venous return, resulting in decreased cardiac output and intraoperative hypotension. Preoperative screening should be sought to identify patients more at risk for intolerance of decreased venous return (e.g., those with valvular disorders, hypertrophic cardiomyopathy, etc.). Attention should also be given to patients with implantable devices such as pacemakers or implantable cardioverter defibrillators (ICDs). Depending on the underlying indication for placement, pacing dependency, and make/model, a coordinated plan should be developed with the patient's cardiologist. Magnet placement may be difficult or susceptible to malposition in the prone position. Preoperative reprogramming with a device representative may need to be coordinated prior to surgery. Prone positioning is also a risk factor for perioperative vision loss (POVL) [52]. This risk should be discussed with patients during their preoperative visit. An overview of the intraoperative considerations for POVL is provided later in this chapter.

Blood Bank Coordination

Complex spine surgery can be associated with significant blood loss. As previously mentioned, preoperative evaluation should include anemia screening and optimization with oral or IV iron as needed. For most spine surgery, a type and screen sample should be sent at their preoperative visit. If blood antibodies are identified during the preoperative type and screen, coordination with the blood bank should ensure that an adequate supply of type-specific blood is available. Certain types of spinal tumors are especially high risk for hemorrhage, (e.g., renal cell, melanoma, and sarcoma metastasis) and consideration should be given to preoperative tumor

embolization [48]. Cell saver may also be requested preoperatively. Preoperative autologous blood donation is not routine in all surgical centers; however, it may be considered for patients having complex spinal procedures where estimated blood loss is anticipated to be at least 500–1000 mL [53].

Pain Assessment

Attention should also be paid to chronic pain medication use and its effectiveness. Since spinal surgery can be associated with an increased need for postoperative analgesia, documenting baseline preoperative opioid use can be important in calculating perioperative opioid requirements. Evaluating the preoperative analgesic regimen effectiveness can also be done by using the Visual Analogue Score (VAS) Pain Assessment [54]. If ineffective pain control is noted, preoperative referral to a pain specialist may be helpful. For patients receiving chronic opioids, there should be consideration given to weaning and titration of non-opioid agents in conjunction with a pain specialist. If there is any concern for addiction, placing a preoperative referral to an addiction specialist is recommended.

Preoperative Testing

Preoperative testing should be deliberately ordered based on a patient's medical history, comorbidities, and the complexity of the planned surgery. Establishing a formalized set of guidelines or laboratory testing grid helps to ensure appropriate studies are performed and avoids the ordering of unnecessary tests [55]. Usually a baseline hemoglobin level, platelet count, and serum chemistry panel (including creatinine and electrolytes) are obtained on most spine surgery patients. Besides simple spine procedures, most spinal surgery requires a preoperative type and screen as well. As above, cardiac testing such as ECG, echocardiogram, and stress test should only be obtained based on the updated ACC/AHA guidelines. Women of childbearing age should be screened with a preoperative pregnancy test. An example of a preoperative testing order grid is shown in Table 15.1.

Informed Consent and Discussion of Anesthetic Risks

Discussion of the anesthetic plan and informed consent should be included in the preoperative clinic visit [48]. Patients should be informed of potential plans for extra vascular access, such as additional peripheral IVs, arterial lines, or possibly central venous catheters. The risks of anesthesia and spine surgery-specific risks should be discussed thoroughly. Albeit rare, prone positioning carries a unique set of risks including pressure or nerve-related injuries and POVL. The preoperative clinic visit is also an appropriate time to ensure the patient has capacity to consent and that advanced directives have been arranged [48].

Table 15.1 Sample preoperative testing order grid

	CBC	PTT/ PT/ INR	BMP	Heparin assay (UFH)	Type & screen	Preg test	ECG
Patient-specific factors							
Cardiovascular disease (other than well-controlled HTN) Poorly-controlled HTN is >140/90 OR <140/90 on ≥2 medications)	X		X				X
Pulmonary disease (other than mild-moderate asthma)	X		X				
Cerebrovascular disease (CVA, TIA)	X		X				X
History of bleeding disorders	X	X					
Diabetes mellitus (POC glucose always checked on DOS)			X				X
History of renal dysfunction/failure	X		X				
History of liver dysfunction/cirrhosis	X	X	X				
Pacemaker/defibrillator							X
AGE >65 for intermediate or high-risk procedure							X
Female pts ≤60 unless hysterectomy or post-menopausal for 1 year						X	
Medications							
Chemotherapy within last 6 months or any anticoagulant	X						
Use of diuretics, digoxin, potassium, ACEI or ARB			X				
Coumadin therapy (INR only, PTT not necessary)	X	X					
Heparin therapy (PTT no longer needed, heparin assay preferred)	X			X			
Procedure-specific factors							
Neurosurgery procedures-all except shunts, rhizotomy, DBS, intrathecal pumps	X		X		X		

Preoperative Introduction to Enhanced Recovery After Surgery

ERAS pathways are evidence-based, integrated, multidisciplinary protocols used to guide the perioperative management of surgical patients. Originally designed to speed recovery and minimize the surgical stress response in patients undergoing colorectal surgery, ERAS pathways have since been developed for several surgical specialties [56, 57]. The preoperative clinic visit is an excellent time to introduce the concept of ERAS. In accordance with these pathways, patients should be advised on smoking and alcohol cessation, postoperative pain expectations, and the overall pain management plan. Institutional preoperative fasting policies should also be discussed. Usually patients are asked to abstain from eating solids for 6–8 hours preoperatively; however, many ERAS protocols advocate for hydration and encourage consumption of a carbohydrate-loaded clear liquid 2–3 hours prior to surgery.

ERAS protocols often incorporate multimodal analgesia to minimize opioid requirements, and it is common to administer analgesics by mouth on the day of surgery. The preoperative clinic visit is an ideal time to discuss the preoperative administration of these agents and screen for any contraindications. Furthermore, providing patients with a written copy of the ERAS plan can improve compliance and satisfaction with the perioperative experience [47]. The remaining sections will review intraoperative anesthetic management principles for spine surgery in the context of ERAS.

Tenets of Anesthetic Intraoperative Management and Spine ERAS Pathways

Background

Despite a growing interest in enhanced recovery, the application of ERAS principles to spine surgery has only recently gained popularity. Given that spine surgery is often associated with a prolonged recovery period requiring intensive rehabilitation and pain management, the adoption of ERAS initiatives has the potential to improve outcomes and decrease rates of complications [58]. Because spine surgery includes procedures of varying degrees of complexity and invasiveness, there are different levels of surgical stress response activation and thus several options for surgical and anesthetic techniques. This heterogeneity has likely contributed to the delay in developing a "one size fits all" ERAS pathway for spine surgery [59]. Acknowledging this, there are some common intraoperative elements of ERAS pathways that can be applied to spine surgery. Namely attempts to reduce the surgical stress response with minimally invasive techniques, goal directed fluid management strategies, preservation of normothermia, and the use of multimodal analgesia including non-opioid agents [58, 60]. Separate from ERAS, there are unique considerations in spine surgery that require special attention from anesthesia providers. These include the choice of anesthetic technique, management of massive blood loss, and risk for POVL. The following sections will review the intraoperative components of ERAS relevant to spine surgery and highlight examples of published pathways.

Anesthetic Technique

There is no consensus on the optimal anesthetic technique for patients undergoing spine surgery. Available options include general anesthesia, monitored anesthesia care (MAC), or neuraxial (spinal or epidural) anesthesia. Each of these techniques is associated with advantages and disadvantages. While general anesthesia allows for a secure airway and motionless operating environment, it may be associated with more hemodynamic changes and higher rates of postoperative nausea and vomiting (PONV). Alternatively, MAC and neuraxial anesthesia can be performed

without manipulating the airway but may be associated with patient movement. Because of patient comfort, a neuraxial technique may be more appropriate in shorter, minimally invasive procedures. In high-risk patients undergoing lumbar spine surgery, performance of the procedure under spinal anesthesia is associated with better perioperative hemodynamic stability, shorter duration of surgery, and lower PONV rates than when performed under general anesthesia [61]. Similarly, a meta-analysis of randomized controlled trials comparing perioperative outcomes in lumbar spine surgery under spinal anesthesia versus general anesthesia concluded that spinal anesthesia offers several hemodynamic advantages in this patient population [62]. Despite these results, there remains heterogeneity in the anesthesia technique recommended in published ERAS pathways.

Fluid Management

The goal of intraoperative fluid management is maintenance of euvolemia. The application of goal directed fluid therapy strategies has been associated with improved perioperative outcomes [63–65]. While dynamic assessment of fluid responsiveness with stroke volume variation or pulse pressure variation may allow for an individualized fluid strategy, this has not been universally adopted. In general, it has been recommended to administer a maintenance rate of balanced crystalloid at 2–3 mL/kg/h with additional boluses of fluid as needed to treat hypovolemia [64]. A fluid strategy including both crystalloids and colloids may minimize the development of tissue edema.

Perioperative Vision Loss

POVL is a feared complication of spine surgery. It occurs with a frequency of 0.013–1% of cases [66]. The etiology of POVL is multifactorial and is variously attributed to ischemic optic neuropathy, central retinal artery occlusion, central retinal vein occlusion, cortical blindness, direct compression, and other causes [66]. Risk factors for the development of POVL include male gender, prolonged operating times, prone positioning, anemia, hypotension, obesity, use of the Wilson frame, and greater blood loss [66, 67]. To minimize the development of POVL, it is recommended to periodically monitor hemoglobin or hematocrit values in high-risk patients with substantial blood loss and transfuse as appropriate. If possible, high-risk patients should be positioned so that the head is level or higher than the rest of the body. Additionally, treatment of hypotension and evaluation of the patient's face and neck is warranted [68]. Direct pressure on the eyes should be avoided, and deliberate hypotension should be employed only if the anesthesiologist and surgeon agree that its use is essential.

Analgesia

Multimodal Analgesia

As introduced in the preoperative section, multimodal analgesia strategies are important components of many ERAS programs. The principle behind multimodal analgesia is achievement of pain management without a large reliance on opioids. This is often accomplished by administering several medications with different mechanisms of action and pharmacologic effects. In theory, such a strategy allows for effective analgesia and minimizes the negative effects of opioids, such as oversedation, ileus, nausea, respiratory depression, and addiction [69]. While a single best regimen has not been identified, acetaminophen, nonsteroidal anti-inflammatory drugs (NSAIDs), gabapentinoids, ketamine, muscle relaxants, local anesthetics, and neuraxial anesthetic techniques are often included in multimodal protocols [70–73]. These agents may be especially efficacious in patients with chronic pain who have previously been exposed to opioids.

Within spine surgery, acetaminophen, NSAIDs, gabapentinoids, neuraxial anesthesia, ketamine, and long-acting local anesthetics have all been found to reduce narcotic requirements and postoperative pain [70, 71]. A meta-analysis of 10 RCTs assessing the efficacy of preoperative gabapentin in spine surgery concluded that gabapentin was effective in reducing postoperative opioid consumption, VAS scores, and several postoperative side effects [74]. A meta-analysis of 14 randomized controlled trials concluded that supplemental perioperative ketamine reduces postoperative opioid consumption up to 24 hours following spine surgery [75]. Administration of a perioperative IV lidocaine infusion reduced pain scores and resulted in significantly improved quality of life scores at 1 and 3 months postoperatively in patients undergoing complex spine surgery [76]. Ketorolac is an NSAID that is commonly used to treat postoperative pain. As it has been implicated in inhibiting osteogenesis, the use of ketorolac in spine surgery is limited. Notably the results of a meta-analysis of five retrospective comparative studies concluded that short-term (<14 days) exposure to normal-dose ketorolac (<120 mg/day) was safe after spinal fusion while short-term exposure to high-dose ketorolac (>120 mg/day) increased the risk of nonunion [73, 77]. A large prospective randomized controlled trial designed to evaluate the effect of ketorolac on fusion rates is ongoing [78].

While there is high quality evidence that supports the administration of many of the individual medications included in multimodal regimens, there seems to be insufficient or conflicting evidence on the effectiveness of these medications when included within a multimodal pathway [70]. For example, Maheshwari et al. performed a randomized controlled trial to examine the effectiveness of a combination of four non-opioid analgesics versus placebo on Quality of Recovery scores, postoperative opioid consumption, and pain scores in adults undergoing multilevel spine surgery who were at high risk for postoperative pain [71]. In their study, an

analgesic pathway based on preoperative oral acetaminophen and gabapentin, combined with intraoperative infusions of lidocaine and ketamine did not improve day 3 Quality of Recovery scores, reduce pain scores, or reduce 48-hour opioid consumption. The results of this study suggest that further investigation into the effectiveness of multimodal analgesic strategies within spine surgery is needed.

Opioid-Free Analgesia

The complete elimination of opioids may represent the next frontier in spine surgery analgesia. Soffin et al. retrospectively evaluated an opioid-free analgesic regimen within an established ERAS pathway for lumbar decompressive surgery [79]. The authors compared perioperative opioid requirements in a matched cohort of patients managed with traditional analgesic regimens that included opioids. Their opioid-free regimen included preoperative oral acetaminophen, oral gabapentin, and midazolam. Intraoperatively, the patients received infusions of propofol, ketamine, and lidocaine in addition to inhalational anesthesia up to 0.5 minimum alveolar concentration (MAC). Dual antiemetic therapy with dexamethasone and ondansetron were administered, and ketorolac was given during surgical closure. All patients received subcutaneous infiltration with 10 mL of 0.25% bupivacaine following fascial closure and immediately prior to skin closure. Patients in the opioid-free analgesia group had a significant reduction in their total perioperative opioid consumption and did not have any adverse effects on postoperative pain scores, opioid requirements, or recovery [79].

Bleeding During Spine Surgery

Spine surgery has the potential for substantial blood loss. Effective planning and communication among all members of the care team can reduce perioperative bleeding, morbidity, and mortality [80]. Significant blood loss causes anemia, coagulopathy, hypotension, and organ dysfunction [81]. Furthermore, excessive bleeding requires allogenic blood transfusion, which has been associated with surgical site infections, lung injury, hypersensitivity reactions, immune modulation, and increased hospital length of stay (LOS) [80, 82, 83]. Perioperative bleeding in spine surgery increases the risk for spinal epidural hematoma formation, which can cause spinal cord compression [82]. Adopting a liberal perioperative blood transfusion strategy ($\geq$10 g/dL intraoperatively or $\geq$8 g/dL postoperatively) is associated with increased costs in patients undergoing spine surgery [84].

Monitoring for Blood Loss

Rotational thromboelastometry (ROTEM) is a rapid, real-time viscoelastometic method for hemostasis testing in whole blood. ROTEM allows for evaluation of the

interaction between multiple coagulation factors and cellular components during both the coagulation and lysis phases [85]. In this way, providers can identify the specific deficiency in the coagulation pathway and provide an individualized treatment [80]. In major spine surgery, the use of ROTEM-guided transfusion allows for standardization of transfusion practices [85]. Furthermore, the use of ROTEM during thoracolumbar deformity correction is associated with lower transfusion requirements [86].

Pharmacologic Agents to Mitigate Blood Loss

Excessive fibrinolysis has been implicated as a factor exacerbating blood loss in spine surgery. Antifibrinolytic agents work to decrease bleeding via inhibition of clot breakdown [87]. Antifibrinolytics such as aprotinin, tranexamic acid (TXA), and epsilon-aminocaproic acid have been shown to reduce perioperative blood loss and transfusion requirements in patients undergoing spine surgery [82, 88]. According to the results of a recently published meta-analysis of randomized control trials, TXA may be the most efficacious agent in reducing total blood loss, intraoperative blood loss, and blood transfusion [82]. Furthermore, there was no evidence from this analysis that the use of these agents was a risk factor for thromboembolism in spine surgery. While the optimal dosing and duration is still unclear, it is recommended that all patients undergoing major spine surgery receive a loading dose of TXA at incision followed by a maintenance infusion during the case [64, 88].

Hypotensive Anesthesia for Reducing Blood Loss

It is thought that controlled hypotension reduces blood extravasation and local wound blood flow [89]. While this technique may help reduce bleeding from soft tissues, both epidural venous plexus pressure and intraosseous pressure are more important determinants of blood loss during spine surgery, and these are both independent of arterial blood pressure [80, 89]. The major risks associated with controlled hypotension are impairing end-organ perfusion, especially the optic nerve and the spinal cord [80, 89]. This technique should only be performed if agreed upon by both the surgeon and anesthesiologist.

Bleeding and Temperature Management

Intraoperative hypothermia is a multifactorial clinical entity, caused by a low operating room (OR) temperature, administration of room-temperature IV fluids, evaporation from surgical wounds, and impaired thermoregulation from induction of general anesthesia [80]. Hypothermia is known to impair the function of platelets and enzymes of the coagulation cascade. Reductions in body temperature may also disrupt thrombin and fibrinogen synthesis [90]. In a pooled population of surgical

patients undergoing several procedures, even mild hypothermia (34–36 °C) significantly increased surgical blood loss and the relative risk for transfusion [91]. Despite this, the association between intraoperative hypothermia and increased bleeding in spine surgery is less clear. The results of some studies support this association while others do not [90, 92, 93]. Although further evaluation of this relationship is warranted, maintenance of normothermia is recommended as hypothermia is associated with an increased rate of mortality and complications in surgical patients [94].

Positioning Strategies for Minimization of Blood Loss

The prone position often required during spine surgery can be associated with increased bleeding. The epidural veins are connected to the inferior vena cava through a valveless venous system. When prone, intraabdominal pressure increases and causes compression of the vena cava. This will result in an increase in the epidural venous system pressure and increase the risk for intraoperative bleeding [89]. The reverse Trendelenburg position decreases central venous pressure (and subsequently epidural venous pressure) and can potentially reduce intraoperative blood loss [80]. In a study of 108 healthy patients undergoing elective prone spine surgery, the use of a Jackson table, compared with the Wilson frame or chest rolls, was associated with a significantly lower intraabdominal pressure [95]. In patients using a Wilson frame, both intraabdominal pressure and intraoperative blood loss were significantly less when using a wide pad support versus a narrow pad support [96].

Examples of Published Spine ERAS Protocols

As discussed previously, there is no generally accepted single ERAS pathway for spine surgery. Published protocols vary in their choice of anesthetic technique, multimodal analgesic regimen, and approach to fluid management. The following section highlights some of these pathways.

Dagal et al. developed an enhanced perioperative care (EPOC) pathway for patients undergoing major spine surgery [97]. Intraoperative anesthetic elements of their pathway included standardized OR temperature management, total intravenous anesthesia (TIVA) with propofol, remifentanil, and ketamine infusions, multimodal analgesia, goal directed fluid administration with stroke volume variation or pulse pressure variation-guided resuscitation, and routine administration of TXA. The establishment of their EPOC program was associated with a reduction in mean hospital LOS, intensive care unit LOS, and average cost. Wang et al. implemented a “fast track” program for patients undergoing minimally invasive transforaminal lumbar interbody fusion [98]. Intraoperative elements of this program included the use of endoscopic decompression, injections of liposomal bupivacaine for long-acting analgesia, and performing the surgery under sedation. Although supplemental oxygen was administered, patients’ airways were not manipulated. Patients were sedated with IV infusions of propofol and ketamine; no opioids were

administered. Compared with patients undergoing conventional minimally invasive transforaminal lumbar interbody fusion, patients in the ERAS group had less intraoperative blood loss, a shorter hospital LOS, and lower total cost for the acute care hospitalization. Ali et al. conducted a prospective cohort study comparing outcomes of patients undergoing elective spine or peripheral nerve surgery following implementation of an ERAS protocol compared to a historical control cohort [99]. Pathway elements include multimodal analgesia with gabapentin, acetaminophen, muscle relaxants, NSAIDS, infiltration of long-acting local anesthesia at the time of surgical closure, and minimization of opioids. In this study, the ERAS protocol improved postoperative mobilization and reduced opioid use in both the perioperative period and at 1-month after surgery.

Soffin et al. performed a retrospective cohort study examining the impact of an ERAS pathway on 61 patients presenting for microdiscectomy or lumbar laminectomy/laminectomy [59]. Patients received multimodal analgesia with acetaminophen and gabapentin. Although a TIVA technique using propofol and ketamine infusions was preferentially used, up to 0.5 MAC of inhaled agent was permitted to achieve the desired depth of anesthesia. Additional non-opioid analgesia with ketorolac and IV lidocaine was administered. The choice and dose of intraoperative opioids was left to the discretion of the anesthesiologist. Infiltration of the surgical incisions with local anesthesia was performed at the end of the procedure. Implementation of their pathway was associated with a short LOS, minimal complications, and no readmissions within 90 days of surgery. Grasu et al. reviewed the postoperative outcomes before and after the implementation of an enhanced recovery after oncologic spine surgery program [100]. Their pathway advocated for multimodal analgesia with acetaminophen, tramadol, and gabapentinoids along with a TIVA technique using infusions of propofol, lidocaine, ketamine, and dexmedetomidine, epidural analgesia or liposomal bupivacaine for surgical wound infiltration, goal directed fluid therapy, maintenance of normothermia, and a restrictive blood transfusion trigger with TXA administration. In this study, patients in the enhanced recovery group had a trend toward better pain scores and decreased opioid consumption compared with patients in the pre-enhanced recovery group.

In another study, Soffin et al. designed an enhanced recovery pathway for 1- and 2-level open lumbar fusion [101]. As with other pathways, patients received multimodal analgesia with oral gabapentin and acetaminophen. A TIVA technique using propofol, dexmedetomidine, and ketamine infusions was preferentially used, and up to 0.3 MAC of isoflurane was permitted to achieve the desired depth of anesthesia. Additional non-opioid analgesia with ketorolac, ketamine, and IV lidocaine was administered. Opioid administration was permitted at the discretion of the anesthesiologist, with a suggested limit of 2 mg of hydromorphone. This pathway did not include goal directed fluid administration or a formal assessment of volume status. Versus usual care, patients in the enhanced recovery group achieved statistically significant gains in early recovery, although a significant clinical impact was not demonstrated.

Finally, Smith et al. evaluated the impact of their ERAS program for 1–2 level lumbar spine fusion surgery [102]. In this pathway, patients received preoperative

acetaminophen and gabapentin and standard antiemetic prophylaxis intraoperatively. Patients with chronic pain or those receiving opioids received IV ketamine with induction of anesthesia; however, there were no specific guidelines for intraoperative opioid use. The protocol did not include intraoperative fluid or hemodynamic parameters. Authors found no impact on hospital LOS or postoperative pain scores but noted a significant decrease in the use of postoperative opioids and rescue antiemetics. Overall, while ERAS programs are being increasingly applied to spine surgery, further research is required to identify the optimal care pathway in this heterogenous patient population.

Conclusion

Patients undergoing spine surgery are at risk for significant morbidity and mortality. Providers must carefully consider both patient-specific and procedure-specific risk factors. A system-based approach to preoperative optimization is recommended. Although the widespread application of ERAS principles to spine surgery is in its early stages, these strategies have the potential to improve several clinical outcomes.

References

1. Schoenfeld AJ, Carey PA, Cleveland AW 3rd, Bader JO, Bono CM. Patient factors, comorbidities, and surgical characteristics that increase mortality and complication risk after spinal arthrodesis: a prognostic study based on 5,887 patients. Spine J. 2013;13(10):1171–9.
2. Devereaux PJ, Goldman L, Cook DJ, Gilbert K, Leslie K, Guyatt GH. Perioperative cardiac events in patients undergoing noncardiac surgery: a review of the magnitude of the problem, the pathophysiology of the events and methods to estimate and communicate risk. CMAJ. 2005;173(6):627–34.
3. Fleisher LA, Fleischmann KE, Auerbach AD, Barnason SA, Beckman JA, Bozkurt B, et al. 2014 ACC/AHA guideline on perioperative cardiovascular evaluation and management of patients undergoing noncardiac surgery: a report of the American College of Cardiology/American Heart Association Task Force on practice guidelines. J Am Coll Cardiol. 2014;64(22):e77–137.
4. Mullen MG, Michaels AD, Mehaffey JH, Guidry CA, Turrentine FE, Hedrick TL, et al. Risk associated with complications and mortality after urgent surgery vs elective and emergency surgery: implications for defining "quality" and reporting outcomes for urgent surgery. JAMA Surg. 2017;152(8):768–74.
5. Chappell D, Jacob M, Hofmann-Kiefer K, Conzen P, Rehm M. A rational approach to perioperative fluid management. Anesthesiology. 2008;109(4):723–40.
6. Eagle KA, Brundage BH, Chaitman BR, Ewy GA, Fleisher LA, Hertzer NR, et al. Guidelines for perioperative cardiovascular evaluation for noncardiac surgery. Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. Committee on Perioperative Cardiovascular Evaluation For Noncardiac Surgery. Circulation. 1996;93(6):1278–317.
7. Hlatky MA, Boineau RE, Higginbotham MB, Lee KL, Mark DB, Califf RM, et al. A brief self-administered questionnaire to determine functional capacity (the Duke Activity Status Index). Am J Cardiol. 1989;64(10):651–4.

8. Wijeysundera DN, Pearse RM, Shulman MA, Abbott TEF, Torres E, Ambosta A, et al. Assessment of functional capacity before major non-cardiac surgery: an international, prospective cohort study. Lancet. 2018;391(10140):2631–40.
9. Wijeysundera DN, Beattie WS, Hillis GS, Abbott TEF, Shulman MA, Ackland GL, et al. Integration of the Duke Activity Status Index into preoperative risk evaluation: a multicentre prospective cohort study. Br J Anaesth. 2020;124(3):261–70.
10. Lee TH, Marcantonio ER, Mangione CM, Thomas EJ, Polanczyk CA, Cook EF, et al. Derivation and prospective validation of a simple index for prediction of cardiac risk of major noncardiac surgery. Circulation. 1999;100(10):1043–9.
11. Gupta PK, Gupta H, Sundaram A, Kaushik M, Fang X, Miller WJ, et al. Development and validation of a risk calculator for prediction of cardiac risk after surgery. Circulation. 2011;124(4):381–7.
12. Bilimoria KY, Liu Y, Paruch JL, Zhou L, Kmiecik TE, Ko CY, et al. Development and evaluation of the universal ACS NSQIP surgical risk calculator: a decision aid and informed consent tool for patients and surgeons. J Am Coll Surg. 2013;217(5):833–42.e1–3.
13. Cohn SL, Fernandez Ros N. Comparison of 4 cardiac risk calculators in predicting postoperative cardiac complications after noncardiac operations. Am J Cardiol. 2018;121(1):125–30.
14. Livhits M, Ko CY, Leonardi MJ, Zingmond DS, Gibbons MM, de Virgilio C. Risk of surgery following recent myocardial infarction. Ann Surg. 2011;253(5):857–64.
15. Livhits M, Gibbons MM, de Virgilio C, O'Connell JB, Leonardi MJ, Ko CY, et al. Coronary revascularization after myocardial infarction can reduce risks of noncardiac surgery. J Am Coll Surg. 2011;212(6):1018–26.
16. Chaudhry W, Cohen MC. Cardiac screening in the noncardiac surgery patient. Surg Clin North Am. 2017;97(4):717–32.
17. Hammill BG, Curtis LH, Bennett-Guerrero E, O'Connor CM, Jollis JG, Schulman KA, et al. Impact of heart failure on patients undergoing major noncardiac surgery. Anesthesiology. 2008;108(4):559–67.
18. Healy KO, Waksmonski CA, Altman RK, Stetson PD, Reyentovich A, Maurer MS. Perioperative outcome and long-term mortality for heart failure patients undergoing intermediate- and high-risk noncardiac surgery: impact of left ventricular ejection fraction. Congest Heart Fail. 2010;16(2):45–9.
19. Yamaguchi T, Kitai T, Miyamoto T, Kagiyama N, Okumura T, Kida K, et al. Effect of optimizing guideline-directed medical therapy before discharge on mortality and heart failure readmission in patients hospitalized with heart failure with reduced ejection fraction. Am J Cardiol. 2018;121(8):969–74.
20. Smetana GW, Lawrence VA, Cornell JE. Preoperative pulmonary risk stratification for noncardiothoracic surgery: systematic review for the American College of Physicians. Ann Intern Med. 2006;144(8):581–95.
21. Gupta H, Ramanan B, Gupta PK, Fang X, Polich A, Modrykamien A, et al. Impact of COPD on postoperative outcomes: results from a national database. Chest. 2013;143(6):1599–606.
22. Arozullah AM, Khuri SF, Henderson WG, Daley J. Development and validation of a multifactorial risk index for predicting postoperative pneumonia after major noncardiac surgery. Ann Intern Med. 2001;135(10):847–57.
23. Canet J, Gallart L, Gomar C, Paluzie G, Vallès J, Castillo J, et al. Prediction of postoperative pulmonary complications in a population-based surgical cohort. Anesthesiology. 2010;113(6):1338–50.
24. Chung F, Memtsoudis SG, Ramachandran SK, Nagappa M, Opperer M, Cozowicz C, et al. Society of Anesthesia and Sleep Medicine guidelines on preoperative screening and assessment of adult patients with obstructive sleep apnea. Anesth Analg. 2016;123(2):452–73.
25. Chung F, Abdullah HR, Liao P. STOP-Bang Questionnaire: a practical approach to screen for obstructive sleep apnea. Chest. 2016;149(3):631–8.
26. Diaz-Fuentes G, Hashmi HR, Venkatram S. Perioperative evaluation of patients with pulmonary conditions undergoing non-cardiothoracic surgery. Health Serv Insights. 2016;9(Suppl 1):9–23.

27. Musallam KM, Tamim HM, Richards T, Spahn DR, Rosendaal FR, Habbal A, et al. Preoperative anaemia and postoperative outcomes in non-cardiac surgery: a retrospective cohort study. Lancet. 2011;378(9800):1396–407.
28. Seicean A, Seicean S, Alan N, Schiltz NK, Rosenbaum BP, Jones PK, et al. Preoperative anemia and perioperative outcomes in patients who undergo elective spine surgery. Spine (Phila Pa 1976). 2013;38(15):1331–41.
29. Nutritional anaemias. Report of a WHO Scientific Group. World Health Organ Tech Rep Ser. 1968;405:5–37.
30. (SABM) SftAoBM. Anemia in the pre-surgical patient. Recognition, diagnosis, and management. 2019. Available from: http://www.sabm.org/publications.
31. Franchini M, Marano G, Veropalumbo E, Masiello F, Pati I, Candura F, et al. Patient blood management: a revolutionary approach to transfusion medicine. Blood Transfus. 2019;17(3):191–5.
32. Mueller MM, Van Remoortel H, Meybohm P, Aranko K, Aubron C, Burger R, et al. Patient blood management: recommendations from the 2018 Frankfurt consensus conference. JAMA. 2019;321(10):983–97.
33. Warner MA, Shore-Lesserson L, Shander A, Patel SY, Perelman SI, Guinn NR. Perioperative anemia: prevention, diagnosis, and management throughout the spectrum of perioperative care. Anesth Analg. 2020;130(5):1364–80.
34. Ellermann I, Bueckmann A, Eveslage M, Buddendick H, Latal T, Niehoff D, et al. Treating anemia in the preanesthesia assessment clinic: results of a retrospective evaluation. Anesth Analg. 2018;127(5):1202–10.
35. Litton E, Xiao J, Ho KM. Safety and efficacy of intravenous iron therapy in reducing requirement for allogeneic blood transfusion: systematic review and meta-analysis of randomised clinical trials. BMJ. 2013;347:f4822.
36. Weiss G, Ganz T, Goodnough LT. Anemia of inflammation. Blood. 2019;133(1):40–50.
37. Alsaleh K, Alotaibi GS, Almodaimegh HS, Aleem AA, Kouroukis CT. The use of preoperative erythropoiesis-stimulating agents (ESAs) in patients who underwent knee or hip arthroplasty: a meta-analysis of randomized clinical trials. J Arthroplast. 2013;28(9):1463–72.
38. Kotzé A, Carter LA, Scally AJ. Effect of a patient blood management programme on preoperative anaemia, transfusion rate, and outcome after primary hip or knee arthroplasty: a quality improvement cycle. Br J Anaesth. 2012;108(6):943–52.
39. Cho BC, Serini J, Zorrilla-Vaca A, Scott MJ, Gehrie EA, Frank SM, et al. Impact of preoperative erythropoietin on allogeneic blood transfusions in surgical patients: results from a systematic review and meta-analysis. Anesth Analg. 2019;128(5):981–92.
40. Colomina MJ, Bagó J, Pellisé F, Godet C, Villanueva C. Preoperative erythropoietin in spine surgery. Eur Spine J. 2004;13(Suppl 1):S40–9.
41. Umpierrez GE, Hellman R, Korytkowski MT, Kosiborod M, Maynard GA, Montori VM, et al. Management of hyperglycemia in hospitalized patients in non-critical care setting: an endocrine society clinical practice guideline. J Clin Endocrinol Metab. 2012;97(1):16–38.
42. Frisch A, Chandra P, Smiley D, Peng L, Rizzo M, Gatcliffe C, et al. Prevalence and clinical outcome of hyperglycemia in the perioperative period in noncardiac surgery. Diabetes Care. 2010;33(8):1783–8.
43. Bagdade JD, Root RK, Bulger RJ. Impaired leukocyte function in patients with poorly controlled diabetes. Diabetes. 1974;23(1):9–15.
44. Martin ET, Kaye KS, Knott C, Nguyen H, Santarossa M, Evans R, et al. Diabetes and risk of surgical site infection: a systematic review and meta-analysis. Infect Control Hosp Epidemiol. 2016;37(1):88–99.
45. Underwood P, Askari R, Hurwitz S, Chamarthi B, Garg R. Preoperative A1C and clinical outcomes in patients with diabetes undergoing major noncardiac surgical procedures. Diabetes Care. 2014;37(3):611–6.
46. Shohat N, Tarabichi M, Tan TL, Goswami K, Kheir M, Malkani AL, et al. John Insall Award: fructosamine is a better glycaemic marker compared with glycated haemoglobin (HbA1C)

in predicting adverse outcomes following total knee arthroplasty: a prospective multicentre study. Bone Joint J. 2019;101-b(7_Supple_C):3–9.
47. Papanastassiou I, Anderson R, Barber N, Conover C, Castellvi AE. Effects of preoperative education on spinal surgery patients. SAS J. 2011;5(4):120–4.
48. Balint MA, Bapat S. Preoperative evaluation of neurosurgical patients. Anaesth Intensive Care Med. 2020;21(1):20–5.
49. Zheng K, Martin A. Guidelines for the intraoperative management of patients undergoing spine surgery. 2020. Available from: http://ether.stanford.edu/policies/spine_surgery.html.
50. Brown M. Anesthesia for elective spine surgery in adults. In: Post T, editor. UpToDate. UpToDate: Waltham. Accessed on 25 Dec 2020.
51. Memtsoudis SG, Vougioukas VI, Ma Y, Gaber-Baylis LK, Girardi FP. Perioperative morbidity and mortality after anterior, posterior, and anterior/posterior spine fusion surgery. Spine (Phila Pa 1976). 2011;36(22):1867–77.
52. Nowicki RWA. Anaesthesia for major spinal surgery. Contin Educ Anaesth Crit Care Pain. 2014;14(4):147–52.
53. Vanderlinde ES, Heal JM, Blumberg N. Autologous transfusion. BMJ. 2002;324(7340):772–5.
54. Haefeli M, Elfering A. Pain assessment. Eur Spine J. 2006;15(Suppl 1):S17–24.
55. Apfelbaum JL, Connis RT, Nickinovich DG, Pasternak LR, Arens JF, Caplan RA, et al. Practice advisory for preanesthesia evaluation: an updated report by the American Society of Anesthesiologists Task Force on Preanesthesia Evaluation. Anesthesiology. 2012;116(3):522–38.
56. Kehlet H. Multimodal approach to control postoperative pathophysiology and rehabilitation. Br J Anaesth. 1997;78(5):606–17.
57. Street AD, Elia JM, McBroom MM, Hamilton AJ, Grundt JE, Blackwell JN, et al. The impact of implementation of a hysterectomy enhanced recovery pathway on anesthetic medication costs. J Comp Eff Res. 2020;9(15):1067–77.
58. Dietz N, Sharma M, Adams S, Alhourani A, Ugiliweneza B, Wang D, et al. Enhanced recovery after surgery (ERAS) for spine surgery: a systematic review. World Neurosurg. 2019;130:415–26.
59. Soffin EM, Vaishnav AS, Wetmore DS, Barber L, Hill P, Gang CH, et al. Design and implementation of an enhanced recovery after surgery (ERAS) program for minimally invasive lumbar decompression spine surgery: initial experience. Spine (Phila Pa 1976). 2019;44(9):E561–e70.
60. Elsarrag M, Soldozy S, Patel P, Norat P, Sokolowski JD, Park MS, et al. Enhanced recovery after spine surgery: a systematic review. Neurosurg Focus. 2019;46(4):E3.
61. Finsterwald M, Muster M, Farshad M, Saporito A, Brada M, Aguirre JA. Spinal versus general anesthesia for lumbar spine surgery in high risk patients: perioperative hemodynamic stability, complications and costs. J Clin Anesth. 2018;46:3–7.
62. Meng T, Zhong Z, Meng L. Impact of spinal anaesthesia vs. general anaesthesia on perioperative outcome in lumbar spine surgery: a systematic review and meta-analysis of randomised, controlled trials. Anaesthesia. 2017;72(3):391–401.
63. Minto G, Scott MJ, Miller TE. Monitoring needs and goal-directed fluid therapy within an enhanced recovery program. Anesthesiol Clin. 2015;33(1):35–49.
64. Chakravarthy VB, Yokoi H, Coughlin DJ, Manlapaz MR, Krishnaney AA. Development and implementation of a comprehensive spine surgery enhanced recovery after surgery protocol: the Cleveland Clinic experience. Neurosurg Focus. 2019;46(4):E11.
65. Zhu AC, Agarwala A, Bao X. Perioperative fluid management in the enhanced recovery after surgery (ERAS) pathway. Clin Colon Rectal Surg. 2019;32(2):114–20.
66. Epstein NE. Perioperative visual loss following prone spinal surgery: a review. Surg Neurol Int. 2016;7(Suppl 13):S347–60.
67. Nickels TJ, Manlapaz MR, Farag E. Perioperative visual loss after spine surgery. World J Orthop. 2014;5(2):100–6.
68. Practice advisory for perioperative visual loss associated with spine surgery 2019: an updated report by the American Society of Anesthesiologists Task Force on Perioperative Visual Loss,

the North American Neuro-Ophthalmology Society, and the Society for Neuroscience in Anesthesiology and Critical Care. Anesthesiology. 2019;130(1):12–30.
69. Baldini A, Von Korff M, Lin EH. A review of potential adverse effects of long-term opioid therapy: a practitioner's guide. Prim Care Companion CNS Disord. 2012;14(3).
70. Devin CJ, McGirt MJ. Best evidence in multimodal pain management in spine surgery and means of assessing postoperative pain and functional outcomes. J Clin Neurosci. 2015;22(6):930–8.
71. Maheshwari K, Avitsian R, Sessler DI, Makarova N, Tanios M, Raza S, et al. Multimodal analgesic regimen for spine surgery: a randomized placebo-controlled trial. Anesthesiology. 2020;132(5):992–1002.
72. Practice guidelines for acute pain management in the perioperative setting: an updated report by the American Society of Anesthesiologists Task Force on Acute Pain Management. Anesthesiology. 2012;116(2):248–73.
73. Chakravarthy V, Yokoi H, Manlapaz MR, Krishnaney AA. Enhanced recovery in spine surgery and perioperative pain management. Neurosurg Clin N Am. 2020;31(1):81–91.
74. Han C, Kuang MJ, Ma JX, Ma XL. The efficacy of preoperative gabapentin in spinal surgery: a meta-analysis of randomized controlled trials. Pain Physician. 2017;20(7):649–61.
75. Pendi A, Field R, Farhan SD, Eichler M, Bederman SS. Perioperative ketamine for analgesia in spine surgery: a meta-analysis of randomized controlled trials. Spine (Phila Pa 1976). 2018;43(5):E299–e307.
76. Farag E, Ghobrial M, Sessler DI, Dalton JE, Liu J, Lee JH, et al. Effect of perioperative intravenous lidocaine administration on pain, opioid consumption, and quality of life after complex spine surgery. Anesthesiology. 2013;119(4):932–40.
77. Li Q, Zhang Z, Cai Z. High-dose ketorolac affects adult spinal fusion: a meta-analysis of the effect of perioperative nonsteroidal anti-inflammatory drugs on spinal fusion. Spine (Phila Pa 1976). 2011;36(7):E461–8.
78. Claus CF, Lytle E, Tong D, Sigler D, Lago D, Bahoura M, et al. The effect of ketorolac on posterior thoracolumbar spinal fusions: a prospective double-blinded randomised placebo-controlled trial protocol. BMJ Open. 2019;9(1):e025855.
79. Soffin EM, Wetmore DS, Beckman JD, Sheha ED, Vaishnav AS, Albert TJ, et al. Opioid-free anesthesia within an enhanced recovery after surgery pathway for minimally invasive lumbar spine surgery: a retrospective matched cohort study. Neurosurg Focus. 2019;46(4):E8.
80. Bible JE, Mirza M, Knaub MA. Blood-loss management in spine surgery. J Am Acad Orthop Surg. 2018;26(2):35–44.
81. Qureshi R, Puvanesarajah V, Jain A, Hassanzadeh H. Perioperative management of blood loss in spine surgery. Clin Spine Surg. 2017;30(9):383–8.
82. Li G, Sun TW, Luo G, Zhang C. Efficacy of antifibrinolytic agents on surgical bleeding and transfusion requirements in spine surgery: a meta-analysis. Eur Spine J. 2017;26(1):140–54.
83. Blackburn CW, Morrow KL, Tanenbaum JE, DeCaro JE, Gron JM, Steinmetz MP. Clinical outcomes associated with allogeneic red blood cell transfusions in spinal surgery: a systematic review. Glob Spine J. 2019;9(4):434–45.
84. Purvis TE, Goodwin CR, De la Garza-Ramos R, Ahmed AK, Lafage V, Neuman BJ, et al. Effect of liberal blood transfusion on clinical outcomes and cost in spine surgery patients. Spine J. 2017;17(9):1255–63.
85. Naik BI, Pajewski TN, Bogdonoff DI, Zuo Z, Clark P, Terkawi AS, et al. Rotational thromboelastometry-guided blood product management in major spine surgery. J Neurosurg Spine. 2015;23(2):239–49.
86. Guan J, Cole CD, Schmidt MH, Dailey AT. Utility of intraoperative rotational thromboelastometry in thoracolumbar deformity surgery. J Neurosurg Spine. 2017;27(5):528–33.
87. Eubanks JD. Antifibrinolytics in major orthopaedic surgery. J Am Acad Orthop Surg. 2010;18(3):132–8.
88. Cheriyan T, Maier SP 2nd, Bianco K, Slobodyanyuk K, Rattenni RN, Lafage V, et al. Efficacy of tranexamic acid on surgical bleeding in spine surgery: a meta-analysis. Spine J. 2015;15(4):752–61.

89. Tse EY, Cheung WY, Ng KF, Luk KD. Reducing perioperative blood loss and allogeneic blood transfusion in patients undergoing major spine surgery. J Bone Joint Surg Am. 2011;93(13):1268–77.
90. Tedesco NS, Korpi FP, Pazdernik VK, Cochran JM. Relationship between hypothermia and blood loss in adult patients undergoing open lumbar spine surgery. J Am Osteopath Assoc. 2014;114(11):828–38.
91. Rajagopalan S, Mascha E, Na J, Sessler DI. The effects of mild perioperative hypothermia on blood loss and transfusion requirement. Anesthesiology. 2008;108(1):71–7.
92. Guest JD, Vanni S, Silbert L. Mild hypothermia, blood loss and complications in elective spinal surgery. Spine J. 2004;4(2):130–7.
93. Lee HK, Jang YH, Choi KW, Lee JH. The effect of electrically heated humidifier on the body temperature and blood loss in spinal surgery under general anesthesia. Korean J Anesthesiol. 2011;61(2):112–6.
94. Billeter AT, Hohmann SF, Druen D, Cannon R, Polk HC Jr. Unintentional perioperative hypothermia is associated with severe complications and high mortality in elective operations. Surgery. 2014;156(5):1245–52.
95. Kim E, Kim HC, Lim YJ, Kim CH, Sohn S, Chung CK, et al. Comparison of intra-abdominal pressure among 3 prone positional apparatuses after changing from the supine to the prone position and applying positive end-expiratory pressure in healthy euvolemic patients: a prospective observational study. J Neurosurg Anesthesiol. 2017;29(1):14–20.
96. Park CK. The effect of patient positioning on intraabdominal pressure and blood loss in spinal surgery. Anesth Analg. 2000;91(3):552–7.
97. Dagal A, Bellabarba C, Bransford R, Zhang F, Chesnut RM, O'Keefe GE, et al. Enhanced perioperative care for major spine surgery. Spine (Phila Pa 1976). 2019;44(13):959–66.
98. Wang MY, Chang HK, Grossman J. Reduced acute care costs with the ERAS® minimally invasive transforaminal lumbar interbody fusion compared with conventional minimally invasive transforaminal lumbar interbody fusion. Neurosurgery. 2018;83(4):827–34.
99. Ali ZS, Flanders TM, Ozturk AK, Malhotra NR, Leszinsky L, McShane BJ, et al. Enhanced recovery after elective spinal and peripheral nerve surgery: pilot study from a single institution. J Neurosurg Spine. 2019:1–9.
100. Grasu RM, Cata JP, Dang AQ, Tatsui CE, Rhines LD, Hagan KB, et al. Implementation of an enhanced recovery after spine surgery program at a large cancer center: a preliminary analysis. J Neurosurg Spine. 2018;29(5):588–98.
101. Soffin EM, Beckman JD, Tseng A, Zhong H, Huang RC, Urban M, et al. Enhanced recovery after lumbar spine fusion: a randomized controlled trial to assess the quality of patient recovery. Anesthesiology. 2020;133(2):350–63.
102. Smith J, Probst S, Calandra C, Davis R, Sugimoto K, Nie L, et al. Enhanced recovery after surgery (ERAS) program for lumbar spine fusion. Perioper Med (Lond). 2019;8:4.

ICU Care for the Spine Patient

16

Jia W. Romito and Bryan T. Romito

Spine patients in the intensive care unit (ICU) generally include two groups of patients: postoperative patients after elective spine surgery and patients who have acute spinal cord injury (SCI). Patients are more likely to require critical care services after spine surgery if they have advanced age, increased comorbidity burden, increased surgical invasiveness, and development of postoperative complications [1]. Patients with SCI are more likely to need admission to the ICU if they have high cervical SCI, complete SCI, advanced age, history of cardiopulmonary disease, and need for significant respiratory support [2]. Every spine patient in the ICU has critical care needs specific to their individual comorbidities, their expected clinical course based on their admitting diagnosis, and their potential to develop complications. Comprehensive critical care is provided using a system-based approach. This chapter will discuss these systems in separate sections.

Neurological System

Neurological Exam

Spine patients in the critical care unit receive serial neurological evaluations. New-onset neurologic deficits require attention and further evaluation with imaging to rule out acute spinal cord compression or nerve root compression to assess if surgical intervention is needed [3, 4]. In the postoperative spine patient these evaluations

J. W. Romito (✉)
Departments of Anesthesiology & Pain Management, Neurological Surgery, and Neurology, University of Texas Southwestern Medical Center, Dallas, TX, USA
e-mail: Jia.Romito@utsouthwestern.edu

B. T. Romito
Department of Anesthesiology & Pain Management, University of Texas Southwestern Medical Center, Dallas, TX, USA
e-mail: Bryan.Romito@utsouthwestern.edu

© The Author(s), under exclusive license to Springer Nature Switzerland AG 2022

C. E. Noe (ed.), *Multidisciplinary Spine Care*,
https://doi.org/10.1007/978-3-031-04990-3_16

include level of consciousness, orientation, and bilateral upper and lower extremity motor/strength and sensation testing. In patients with SCI, the International Standards for Neurological Classification of Spinal Cord Injury represents the gold standard assessment for documentation of the level and severity of a spinal cord injury [5]. This was formulated as a result of collaboration between the International Standards Committee of the American Spinal Injury Association (ASIA) and the International Spinal Cord Society. The assessment is a detailed strength and sensory exam to evaluate the level of the neurological injury, complete or incomplete injury, the ASIA Impairment Scale (AIS) Grade, and the zone of partial preservation. A proper neurologic assessment always includes a rectal examination because sacral sparing has prognostic significance in the patients with SCI [6]. Acute cerebral ischemia and seizures can also occur in both groups of spine patients given the acute change in medical condition which requires intensive care in addition to individual predisposing comorbidities.

In patients with SCI, there are additional considerations for the neurological exam. Traumatic brain injury can occur concurrently with SCI. Spine immobilization should be maintained until definitive treatment. There is no evidence to recommend the routine use of steroids in order to improve functional recovery in this setting [4, 7].

Pain Management

Patients who undergo spine surgery typically require intensive pain management for acute postoperative pain in the setting of preexisting chronic pain. Patients who have preexisting chronic pain may have tolerance to narcotics and non-narcotic analgesics, complicating pain management. Immediately after spine surgery, there is often intense acute pain for the first three days. Adequate pain management in this period has been shown to correlate well with improved functional outcome, early ambulation, early discharge, and prevention of the development of chronic pain [8]. A multimodal pain regimen combining opioids and non-opioid analgesics can effectively control pain [8]. Opioids may be administered on a scheduled regimen, as needed, or utilizing a patient-controlled analgesia (PCA) pump. Common choices for non-opioid analgesics include muscle relaxants, nonsteroidal anti-inflammatory drugs (NSAIDs), acetaminophen, gabapentinoids (gabapentin/pregabalin), and ketamine. Continuous wound infiltration and/or epidural delivery of local anesthetics can also be utilized to reduce postoperative pain [8–10].

In addition to treatment of somatic and neuropathic pain with a multimodal regimen similar to that mentioned above, patients with SCI require treatment of spasticity. Oral baclofen and tizanidine are recommended as first treatments in SCI related spasticity, with intrathecal baclofen shown to also reduce autonomic dysreflexia [11, 12]. Orthoses, daily passive muscle stretching, and exercises to strength the muscle groups can aid in the management of spasticity, given the concern for spasticity to develop into chronic contractures [13].

Mobility, Therapy, and Rehabilitation

Physical and occupational therapists work with spine patients to evaluate mobility, balance, ambulation, and to assess for assistive device needs. Additionally, these therapists work with patients to perform their activities of daily living. Assessment of home layout and evaluation of caregiver support availability are completed prior to discharge from the hospital. Mobility during the daytime contributes to normal sleep-wake cycles, thus reducing delirium. It also decreases the risk of development of atelectasis, venous thromboembolic (VTE) disease, pressure sores, and musculoskeletal problems related to prolonged bedrest [13–16].

Delirium

Delirium has been noted to affect as many as 87% of patients in the ICU, and the incidence of postoperative delirium after spinal surgery ranges from 0.49% to 21% [17–19]. Predisposing patient factors for the development of delirium include older age, cognitive impairment, functional dependence, alcohol and drug abuse, and significant underlying comorbidities such as sensory impairment, diabetes, anemia, and malnutrition [17–21]. For those who undergo spine surgery, intraoperative factors include increased duration of surgery and the need for blood product transfusion [16, 17]. Furthermore, any acute change in medical status, especially one requiring ICU admission, can increase risk of developing delirium. Examples include hemodynamic instability, cardiopulmonary insufficiency, abnormal laboratory data, need for blood transfusion, and iatrogenic administration of certain medications such as opioids, benzodiazepines, and anticholinergics [16, 18, 19, 22]. Overall, early recognition of patients at an increased risk for delirium increases providers' ability to institute timely preventative and treatment strategies.

Effective treatment of delirium is mostly non-pharmacologic and includes maintaining a consistent family presence, decreasing polypharmacy, and encouraging a normal sleep-wake cycle. Patients should participate in activity during the day and minimize disruptions at night [16, 22, 23]. Correcting abnormal laboratory values and treating dehydration also reduces the incidence of delirium [19, 23]. In patients who require mechanical ventilation, daily spontaneous awakening trails, daily spontaneous breathing trials, and overall reduction in sedative use allows for early mobility with therapy services. This is associated with better clinical outcomes and a reduction in delirium [19, 24].

Cardiovascular System

Patients may have underlying comorbidities which predispose them to the development of arrhythmias or hemodynamic lability. Such baseline conditions include chronic hypertension, diabetes, coronary artery disease, congestive heart failure,

and chronic/paroxysmal arrhythmias. The physiological stress of surgery also increases the risk of hypotension, hypertension, new-onset arrhythmia, heart failure, and myocardial injury [25]. The goals of hemodynamic optimization are to preserve spinal cord perfusion, minimize postoperative surgical bleeding, and provide adequate end-organ perfusion. While hypertension can increase the risk of surgical site bleeding, it also increases myocardial demand and the risk for stroke. Conversely, hypotension increases the risk of ischemia to the spinal cord and end organs.

All patients in the critical care unit are monitored with continuous telemetry and serial blood pressure evaluations as the standard of care. Blood pressure is monitored either continuously using an arterial line or serially from a non-invasive blood pressure pneumatic cuff. Adequate perfusion is evaluated with serial neurological exams, serial laboratory tests, and frequent urine output. Further hemodynamic components that are routinely monitored and optimized include intravascular volume status, cardiac output, and global oxygen delivery.

Postoperative Cardiac Considerations After Spine Surgery

Protocol-based blood pressure goals have been shown to improve outcomes after elective spine surgery [25–27]. While there is not a consensus for an overarching post-spine surgery blood pressure target, the hemodynamic goals should take into consideration individual comorbidities, spinal cord perfusion, organ perfusion, and complications arising from both hypotension and hypertension [25].

Perioperative cardiac events are the leading cause of death following non-cardiac surgery. Major adverse cardiac events (MACEs) include myocardial infarction, cardiac arrhythmias, and cardiac arrest [28]. The postoperative median time period to develop myocardial infarction after elective spine surgery is approximately two days, affecting 1%–2% of patients [29]. Patients who are male with comorbidities of advanced age, obesity, diabetes mellitus, hypertension, anemia, chronic renal insufficiency, and a history of cardiac disorders are more likely to develop MACEs after spine surgery [30–32]. Intraoperative risk factors include larger surgeries with at least two levels of spinal fusion and intraoperative blood transfusion [30, 31]. MACEs can present with dyspnea and angina, abnormalities in continuous telemetry, acute electrocardiogram changes, and cardiac enzyme elevation. Transthoracic and transesophageal echocardiography can provide further information on the etiology and response to treatment. Although individual patient factors must be considered, the general treatment strategy for acute postoperative myocardial infarction includes administration of oxygen, nitroglycerin, morphine, beta blockade, and aspirin. The decision to proceed with emergent coronary revascularization with cardiac catheterization should take into consideration the need for periprocedural anticoagulation and postrevascularization antiplatelet therapy, both of which can increase the risk of postoperative bleeding [33].

Cardiac Considerations After Spinal Cord Injury

In order to ensure adequate spinal cord perfusion, current guidelines recommend maintaining a mean arterial blood pressure >85–90 mmHg for at least the first week after an acute SCI, past the time when spinal cord edema and vascular congestion are expected to be maximum [4, 34, 35]. Patients with SCI are at risk for spinal shock, neurogenic shock, and autonomic dysreflexia. Patients with SCI above the splanchnic sympathetic outflow (T5–T6) have pooling of blood within the venous system and decreased cardiac return, resulting in systemic hypotension. SCI above the level from which the cardiac accelerator nerves arise (T1-T4) directly contributes to decreased cardiac output and hypotension. Hypotension can also occur due to hemorrhage, pneumothorax, myocardial injury, pericardial tamponade, sepsis, abdominal injury, adrenal insufficiency, and other traumatic insults. These patients may present with bradycardia or arrhythmias due to direct loss of sympathetic input to the heart [4, 6]. In addition to managing the direct cause of hemodynamic instability, the Consortium for Spinal Cord Medicine guidelines suggest that cervical and thoracic injuries through T6 should be treated with vasoactive mediations that provide inotropy, chronotropy, and vasoconstriction. Either dopamine or norepinephrine are suggested as first-line vasoactive agents [4, 36, 37].

Orthostatic hypotension is common in patients with SCI and can be addressed with both nonpharmacologic and pharmacologic interventions. Nonpharmacologic treatment includes lower limb compression with graduated elastic stockings and elastic wraps, abdominal binders, adequate hydration, and gradual attainment of an upright position. Medication regimens can include fludrocortisones, midodrine, or ephedrine. Orthostatic hypotension should be aggressively managed as it impedes mobilization, rehabilitation, and recovery [6, 38].

Respiratory System

The respiratory system consists of the upper respiratory tract and the lower respiratory tract. The upper respiratory tract includes the airway structures of the nasal passages, oropharynx, and larynx above the vocal cords. The lower respiratory tract includes the larynx below the vocal cords, trachea, bronchi, bronchioles, and alveoli. Spine patients in the ICU can have difficulty with oxygenation and ventilation due to pathology in the upper respiratory tract, the lower respiratory tract, or both. Standard monitoring in the intensive care unit includes continuous pulse oximetry. To further evaluate the respiratory system, arterial blood gases and chest imaging are commonly utilized.

Airway Compromise After Spine Surgery

Patients can have baseline conditions that predispose them to upper respiratory tract complications after spine surgery. These include advanced age, morbid obesity,

prior smoking, obstructive sleep apnea, pulmonary disease, cervical myelopathy, and prior anterior cervical spine surgery [39, 40]. Intraoperative anesthetic and surgical events can increase the likelihood of postoperative upper respiratory tract complications. These include difficult airway, multiple intubation attempts, more than 300 mL of blood loss, prolonged procedures more than five hours, the type of surgical method, exposing more than three vertebral levels, and surgery that includes C2, C3, or C4 [39–41].

A rare but potentially lethal complication after anterior cervical spine surgery is respiratory compromise from airway obstruction. This is most likely to occur in the first 12–72 hours postoperatively. It may require emergent reintubation and has been reported to occur in 6.1% of patients following anterior cervical spine surgery [41]. Although airway obstruction is most likely due to the development of laryngopharyngeal edema, wound hematoma, abscess, cerebrospinal fluid collection, vocal cord dysfunction, and construct failure can also contribute. If there is concern for airway compromise, then an endotracheal tube cuff leak test or fiberoptic bronchoscopy can further assess airway patency prior to extubation. An endotracheal tube exchanger can be used as a placeholder in the trachea at the time of extubation to facilitate reintubation if necessary [39].

Lower Respiratory Tract Complications After Spine Surgery

Lower respiratory tract complications after spine surgery include acute respiratory failure, atelectasis, pleural effusion, pneumonia, pleural effusion, and pneumothorax [42–44]. Patients may have inherent comorbidities that predispose them to pulmonary complications in the lower respiratory tract after spine surgery. These comorbidities include advanced age, scoliosis causing restrictive lung disease, history of smoking, chronic obstructive pulmonary disease, asthma, pre-operative oxygen dependence, obstructive sleep apnea, poor baseline functional status, and diabetes [42, 43, 45]. There are procedure-related factors that may increase the risk of pulmonary complications following spine surgery. For example, a prolonged duration of surgery is associated with an increased risk. Furthermore, corrective surgery for spinal deformities can utilize a technical approach that invades the thoracic cavity. This invasion of the thoracic cavity can lead to lobar collapse. Diaphragmatic manipulation or irritation during surgery can cause postoperative pleural effusions. Intraoperative blood transfusions and aggressive volume resuscitation can lead to fluid shifts resulting in pulmonary edema. Prolonged surgical immobility and postoperative pain can increase atelectasis. The combined immobility from both the intraoperative period and the postoperative period increases the risk of pulmonary embolism (PE). Early mobilization and aggressive respiratory care can reduce several of these postoperative pulmonary complications [46–48].

Respiratory Care in Spinal Cord Injury

Patients with acute SCI require significant respiratory care and are at a high risk for respiratory complications. Baseline respiratory parameters should be obtained and monitored continuously. Standard respiratory care for patients with SCI includes frequent suctioning, using manually-assisted coughing to augment weak accessory muscles to expel retained secretions, pulmonary hygiene, and mechanical insufflation-exsufflation [4].

If intubation is necessary, then spine immobilization must be maintained if spinal cord instability is present [6]. Up to 100% of patients with cervical SCI require intubation and mechanical ventilation [6, 49]. Additional risk factors for intubation include advanced age, underlying respiratory pathology, and tachypnea on admission [2]. Weak or absent cough reflex, loss of accessory respiratory muscle function, diaphragmatic dysfunction, and weakness of abdominal muscles leads to aspiration, reduced tidal volumes, atelectasis, mucus plugging, and pneumonia. Additionally, patients with SCI can develop pleural effusions, pneumothorax, acute respiratory distress syndrome (ARDS), acute lung injury (ALI), transfusion-related acute lung injury, and pulmonary thromboembolism.

In patients without ALI or ARDS, larger tidal volumes may be utilized to titrate to a patient's respiratory mechanics, reduce atelectasis, and progress with weaning trials. Patients who develop ARDS or ALI should utilize mechanical ventilation with a lower tidal volume and a lower plateau pressure to reduce mortality [35, 50, 51]. Readiness for weaning from mechanical ventilation is indicated by sufficient vital capacity, improved secretions, ability to cooperate, upper airway patency, chest radiography without acute findings, and an improving mechanical ventilation support requirement [52]. Patients with complete SCI, especially cervical SCI, and those with significant concurrent injuries are more likely to undergo a tracheostomy given the increased likelihood of prolonged mechanical ventilation [35].

Renal System

Spine patients may have baseline comorbidities that mandate closer monitoring of their renal function, including advanced age, diabetes mellitus, hypertension, chronic kidney disease, and end-stage renal disease. During any surgical procedure, patients can develop hypotension from anesthetic medication effects, blood loss, and insensible free water losses. These can be exacerbated in prolonged spinal surgery procedures or those with significant intraoperative or postoperative bleeding. Accordingly, patients may present with acute kidney injury, urinary retention, and electrolyte derangements that require frequent serum chemistry evaluation and close monitoring of urine output.

Acute Kidney Injury

Acute kidney injury (AKI) is a common complication in hospitalized patients, with an incidence of 3–10% [53–57]. Patients who develop AKI have in-hospital mortality rates as high as 30–70% [54, 58–60]. In general, AKI can result from prerenal, intrinsic renal, and postrenal etiologies. The most common causes of these include renal hypoperfusion, administration of nephrotoxic medications, and urinary tract obstruction respectively. Close monitoring of urine output ensures a more appropriate volume status and has been associated with improved detection of AKI and reduced 30-day mortality in patients experiencing acute kidney injury [61]. Adequate urine output is typically defined as 0.5–1 ml/kg/hr to ensure perfusion, although each patient's comorbidities should be evaluated closely to individualize this target [62]. Over-resuscitation can result in volume overload, hypertension, hypoxemia, and exacerbation of congestive heart failure.

Electrolyte Disorders

Electrolyte disorders are common in patients in critical care and can cause increased morbidity and mortality [63, 64]. Patients are at an increased risk for electrolyte disorders if they have underlying diabetes, hypertension, and chronic renal insufficiency. Periprocedural blood loss requiring transfusion, various types of intravenous fluid resuscitation, and osmotic diuretics affect the physiologic electrolyte balance. Overall, electrolyte disorders increase the risk for the development of delirium, seizure, coma, cardiac arrhythmias, respiratory failure, gastrointestinal dysmotility, and cardiac arrest [63, 65, 66].

Urinary Retention

Postoperative urinary retention (POUR) is the most frequently reported genitourinary complication after spine surgery, with an incidence ranging from 5–38%. Patients are more likely to develop POUR if they are older, male, have a history of AKI, urinary tract infection (UTI), benign prostatic hypertrophy, hypertension, or diabetes mellitus. POUR can lead to the development of UTI, sepsis, and increased length of stay [67]. Indwelling urinary catheters can relieve bladder distention and are useful to closely monitor urinary output. Once close monitoring of the urine output is no longer necessary, the indwelling urinary catheter should be removed because their prolonged use can increase the risk of UTI. Intermittent catheterization of the bladder can be initiated if necessary following removal of the indwelling catheter.

Patients with SCI can have neurogenic bladder due to the loss of reflex activity in the lower genitourinary tract. This loss of ability to spontaneously void leads to urinary retention, with bladder distention being one of most common causes of autonomic dysreflexia [4, 68]. Similar to patients with POUR, the use of indwelling

urinary catheters can relieve bladder distention but should be removed when close monitoring of urinary output is no longer necessary [68].

Gastrointestinal System

Spine patients can have dysmotility in the upper and/or lower gastrointestinal tract. This commonly presents with nausea, vomiting, ileus, or diarrhea. Further evaluation may be necessary to rule out more significant causes for dysmotility such as infection, mechanical obstruction, acute colonic pseudo-obstruction, hemorrhage, and peritoneal wall rupture. Adequate nutrition and appropriate blood sugar control are vital to ensure wound healing and decreasing the risk of infection.

Dysphagia

Spine patients with dysphagia are at increased risk of pulmonary aspiration. These patients may be unable to swallow altogether or have trouble swallowing food and managing their secretions. Risk factors for spine patients to develop dysphagia include advanced age, weak or absent cough, anterior cervical spine surgery, cervical SCI, and prolonged respiratory failure [4, 69, 70]. It is imperative to identify dysphagia early in order to initiate timely evaluation and intervention by speech pathologists. These efforts can reduce the risks of aspiration, chemical pneumonitis, and pneumonia [4, 69]. Speech therapists can evaluate for dysphagia with a bedside swallowing examination, videofluoroscopic swallowing study, and fiberoptic endoscopic examination of swallowing.

Postoperative Ileus

Ileus is a relatively common postoperative finding after elective spine surgery, occurring in approximately 3.5% of patients undergoing lumbar spine surgery [46, 71]. Preoperative risk factors include chronic anemia, alcohol abuse, chronic lung disease, fluid/electrolyte disorders, and recent weight loss [71]. Intraoperative surgical factors that increase the risk and duration of postoperative ileus include multiple fusion levels, increased surgical blood loss, and increased intestinal manipulation from an anterior approach [71, 72]. In addition, all types of anesthesia have an effect on bowel motility [73, 74]. The use of postoperative opioids for analgesia has an inhibitory effect on gastric motility [74]. Providing medications for symptom management, correcting abnormal serum electrolytes, and minimizing agents that exacerbate symptoms can prevent or reduce the more significant complications of aspiration, underfeeding, and decreased mobility [75]. Early ambulation has been postulated to have a prokinetic effect on the gastrointestinal system, and this may also help to reduce rates of delirium, atelectasis, and VTE [13–16, 22, 23, 46, 76].

Neurogenic Bowel Dysfunction

Patients with SCI commonly have loss of colonic motility control. The presence or absence of the bulbocavernosus reflex indicates upper motor neuron versus lower motor neuron bowel dysfunction. Early in the treatment course, a bowel regimen should be created and titrated to the needs of each patient depending on the presentation of constipation or diarrhea. The goal is to have one bowel movement per day, with a combined regimen of oral medications, suppositories, and digital stimulation [4, 77]. Constipation and impaction are some of most common causes of autonomic dysreflexia in patients with SCI at level T6 or above. If a patient presents with diarrhea, a rectal tube may be a useful temporary measure while the etiology is evaluated, which may include medication effect, electrolyte abnormality, or infection [4].

Glycemic Control and Nutrition

Hyperglycemia, diabetes, and malnutrition have been associated with adverse outcomes in both patients after elective spine surgery and those with SCI. Patients with diabetes have been reported to have an increased risk of infection, including surgical site infections, ventilator associated pneumonia, and catheter-related bacteremia. There is increase in adverse complications especially in patients who have serum blood sugars >200 mg/dL [4, 78–81]. The goal of glycemic management is euglycemia, targeting blood glucose values <180 mg/dL. Early enteral nutrition should be initiated in both patients with SCI and those after elective spine surgery to reduce complications and improve outcomes [4, 82–84]. In patients with SCI, this has been associated with an improvement in wound healing, reductions in infectious complications and gastric stress ulceration, and a lower incidence of hyperglycemia [4, 84].

Hematologic System

In spine patients, there is a need to balance pathologic bleeding and increased thromboembolic risk, both of which are associated with morbidity and mortality. Bleeding can cause or exacerbate neurological deficits, MACEs, hemodynamic instability, and progressive coagulopathy. VTE disease can cause morbidity from post-thrombotic syndrome involving local tissues from deep venous thrombosis (DVT) or increase mortality rates from PE.

Hematologic Considerations After Spine Surgery

The incidence of VTE after spine surgery has been reported to occur in up to 31% of patients, with the rate of DVT ranging from 0.3–15.5% and the rate of PE ranging from 0.06–18%. Fatal PEs represent about 6% of all PEs that occur after spine surgery [85–87]. Patients are at an increased risk of VTE if they are male, older, obese,

have baseline dependent functional status, or have a history of malignancy, hypertension, transient ischemic attack, or stroke. Longer operative time, perioperative blood transfusions, and lumbar spine surgery further increase the risk of postoperative VTE [88–92]. Prolonged postoperative immobilization increases the risk of VTE, while early ambulation can decrease the incidence of VTE [46, 88]. Mechanical and chemical thromboprophylaxis interventions include the use of intraoperative and postoperative elastic stockings on the legs, intermittent pneumatic compression devices, early mobility, and administration of unfractionated heparin or low molecular weight heparin [93].

Conversely, patients who undergo spine surgery can experience morbidity and mortality due to acute anemia blood loss, coagulopathy, hemodynamic instability, and MACEs. Formation of an epidural hematoma can require emergent surgical evacuation and risks permanent neurological damage. For these reasons, the risk of bleeding should be balanced with the risk of VTE for each patient's comorbidities, surgical procedure, and postoperative needs.

Hematologic Considerations in Spinal Cord Injury

Patients with SCI are at a high risk of developing VTE within the first week of injury. Advanced age, malignancy, and concurrent injuries increase the risk of developing VTE [35]. Early administration of thromboprophylaxis within 72 hours of the initial injury is recommended after SCI if there are no contraindications. This includes mechanical and chemical thromboprophylaxis interventions such as intermittent pneumatic compression and low molecular weight heparin or unfractionated heparin [4, 6].

Potential contraindications to the administration of chemical thromboprophylaxis include inadequate hemostasis, intracranial bleeding, neuraxial hematoma, or hemothorax. If patients have active bleeding for more than 72 hours, then inferior vena cava filter placement may be needed. Once hemostasis is achieved, then chemical thromboprophylaxis should be initiated as soon as possible [4]. Inferior vena cava filters are not recommended as a routine prophylactic measure in SCI patients. These filters can be utilized in patients who have VTE despite anticoagulation and for those with contraindications to mechanical and chemical thromboprophylaxis [6].

Infectious Disease Considerations

Spine patients in the ICU can have infectious disease considerations affecting the spine as well as other organ systems. The former includes incisional and soft tissue infections, deep infections, vertebral osteomyelitis and discitis, central nervous system abscesses, and spinal hardware infections. The latter includes pneumonia, urinary tract infections, line-associated infections, bacteremia, infectious colitis, and pressure ulcers.

All spine surgical procedures carry a risk of post-procedural infection. Postoperative spine infection rates have been reported to be up to 20% of spine surgeries. Patients with advanced age, diabetes, developmental delay, immunosuppression, obesity, smoking, and malnutrition are at an increased risk of infection. Spine surgeries for trauma/SCI, instrumentation placement, increased blood loss, and a prolonged operative time further increase the risk of infection [78, 94–97].

Patients with an infection at the site of the surgical procedure will present with persistent, progressive back pain out of proportion to the physical findings. The pain may radiate to the buttock, thigh, leg, groin, perineum, or abdomen. Constitutional symptoms are not consistently present. Superficial, skin, and soft tissue infections may present with redness, swelling, or purulent drainage at the incision site, however less than 10% of surgical incisions will present in this manner [78, 94]. The presence of a neurologic deficit should raise suspicion for an epidural abscess. Postoperative infections after anterior cervical procedures may present with painful swallowing due to development of a retropharyngeal abscess [78]. Further evaluation including laboratory, imaging, and surgical exploration may be necessary to delineate between the different differential diagnoses [94].

Infectious Disease Evaluation

Laboratory evaluation often includes a complete blood count with differential, erythrocyte sedimentation rate, C-reactive protein, and blood cultures. Serial assessment of these parameters may help providers identify an evolving infection and monitor the effectiveness of their treatment. Blood culture data can help direct appropriate antimicrobial therapy based on local infectious patterns [78, 97, 98].

Imaging modalities include plain radiographs, computerized tomography (CT) scans, and magnetic resonance imaging (MRI). Negative plain radiographic findings do not exclude infection [78]. Spine CT can show bony destruction, soft tissue collections, erosive changes at the endplates, and loss of intervertebral disc space [78, 94]. MRI with gadolinium enhancement is the preferred imaging modality to visualize postprocedural spine infections and is able to clearly delineate epidural, subfascial, and subcutaneous abscesses [78, 94, 96–98].

Infectious Disease Treatment Principles

Effective treatment of post-neurosurgical infections requires that antimicrobials penetrate the blood-brain and blood-CSF barriers. Certain antimicrobials are reportedly effective in the in vitro setting but should not be used in practice as they do not adequately penetrate the CNS. Additionally, biofilms on hardware are more difficult for antimicrobial medications to penetrate [94]. Tissue biopsy or needle aspiration of purulent material can aid in providing a microbiological diagnosis to direct antimicrobial therapy [78, 94]. Treatment length is based on clinical, laboratory, and radiographic responses.

As discussed previously, other infectious disease considerations can include pneumonia, UTIs, line-associated infections, bacteremia, and pressure ulcers. The risk of pneumonia can be reduced with pulmonary toilet, respiratory therapy, monitoring for aspiration, and early mobilization [42–44, 46–48]. The majority of UTIs occur in the setting of POUR and indwelling urinary catheters [67]. Removal of lines and catheters as soon as possible can reduce the risk of UTIs and other line-associated infections. Pressure ulcers and foot drop injuries can be mitigated with frequent turning, orthoses, and early mobility [13]. Finally, early administration of appropriate antibiotics, infectious source control, and intravascular volume resuscitation are crucial to improve morbidity and mortality in patients with sepsis and septic shock [99].

Conclusion

Overall, spine patients in the intensive care unit represent a complex, heterogeneous patient population. Diligent monitoring, early recognition, and timely management of complications are essential. Providers should consider a systematic evaluation of patients' care needs given the potential for morbidity and mortality.

References

1. Memtsoudis SG, Stundner O, Sun X, Chiu Y-L, Ma Y, Fleischut P, et al. Critical care in patients undergoing lumbar spine fusion: a population-based study. J Intensive Care Med. 2014;29(5):275–84.
2. Casha S, Christie S. A systematic review of intensive cardiopulmonary management after spinal cord injury. J Neurotrauma. 2011;28(8):1479–95.
3. Street JT, Lenehan BJ, DiPaola CP, Boyd MD, Kwon BK, Paquette SJ, et al. Morbidity and mortality of major adult spinal surgery. A prospective cohort analysis of 942 consecutive patients. Spine J. 2012;12(1):22–34.
4. Early acute management in adults with spinal cord injury: a clinical practice guideline for health-care professionals. J Spinal Cord Med. 2008;31(4):403–79.
5. International standards for neurological classification of spinal cord injury. Richmond, VA: American Spinal Injury Association; 2019.
6. Schuster JM, Syre P. 30 - Spine trauma and spinal cord injury. In: Kumar M, Kofke WA, Levine JM, Schuster J, editors. Neurocritical care management of the neurosurgical patient. London: Elsevier; 2018. p. 303–14.
7. Shank CD, Walters BC, Hadley MN. Current topics in the management of acute traumatic spinal cord injury. Neurocrit Care. 2019;30(2):261–71.
8. Bajwa SJ, Haldar R. Pain management following spinal surgeries: an appraisal of the available options. J Craniovertebr Junction Spine. 2015;6(3):105–10.
9. Terracina S, Robba C, Prete A, Sergi PG, Bilotta F. Prevention and treatment of postoperative pain after lumbar spine procedures: a systematic review. Pain Pract. 2018;18(7):925–45.
10. Soffin EM, Wetmore DS, Beckman JD, Sheha ED, Vaishnav AS, Albert TJ, et al. Opioid-free anesthesia within an enhanced recovery after surgery pathway for minimally invasive lumbar spine surgery: a retrospective matched cohort study. Neurosurg Focus. 2019;46(4):E8.
11. Del Fabro AS, Mejia M, Nemunaitis G. An investigation of the relationship between autonomic dysreflexia and intrathecal baclofen in patients with spinal cord injury. J Spinal Cord Med. 2018;41(1):102–5.

12. Yelnik AP, Simon O, Bensmail D, Chaleat-Valayer E, Decq P, Dehail P, et al. Drug treatments for spasticity. Ann Phys Rehabil Med. 2009;52(10):746–56.
13. Elbasiouny SM, Moroz D, Bakr MM, Mushahwar VK. Management of spasticity after spinal cord injury: current techniques and future directions. Neurorehabil Neural Repair. 2010;24(1):23–33.
14. Madera M, Brady J, Deily S, McGinty T, Moroz L, Singh D, et al. The role of physical therapy and rehabilitation after lumbar fusion surgery for degenerative disease: a systematic review. J Neurosurg Spine. 2017;26(6):694–704.
15. Parry SM, Puthucheary ZA. The impact of extended bed rest on the musculoskeletal system in the critical care environment. Extrem Physiol Med. 2015;4:16.
16. Inouye SK. Delirium in older persons. N Engl J Med. 2006;354(11):1157–65.
17. Kang T, Park SY, Lee JH, Lee SH, Park JH, Kim SK, et al. Incidence & risk factors of postoperative delirium after spinal surgery in older patients. Sci Rep. 2020;10(1):9232.
18. Cavallazzi R, Saad M, Marik PE. Delirium in the ICU: an overview. Ann Intensive Care. 2012;2(1):49.
19. Arumugam S, El-Menyar A, Al-Hassani A, Strandvik G, Asim M, Mekkodithal A, et al. Delirium in the intensive care unit. J Emerg Trauma Shock. 2017;10(1):37–46.
20. Ushida T, Yokoyama T, Kishida Y, Hosokawa M, Taniguchi S, Inoue S, et al. Incidence and risk factors of postoperative delirium in cervical spine surgery. Spine (Phila Pa 1976). 2009;34(23):2500–4.
21. Gao R, Yang ZZ, Li M, Shi ZC, Fu Q. Probable risk factors for postoperative delirium in patients undergoing spinal surgery. Eur Spine J. 2008;17(11):1531–7.
22. Kawaguchi Y, Kanamori M, Ishihara H, Abe Y, Nobukiyo M, Sigeta T, et al. Postoperative delirium in spine surgery. Spine J. 2006;6(2):164–9.
23. Inouye SK, Baker DI, Fugal P, Bradley EH. Dissemination of the hospital elder life program: implementation, adaptation, and successes. J Am Geriatr Soc. 2006;54(10):1492–9.
24. Marra A, Ely EW, Pandharipande PP, Patel MB. The ABCDEF bundle in critical care. Crit Care Clin. 2017;33(2):225–43.
25. Meng L, Yu W, Wang T, Zhang L, Heerdt PM, Gelb AW. Blood pressure targets in perioperative care. Hypertension. 2018;72(4):806–17.
26. Sessler DI, Meyhoff CS, Zimmerman NM, Mao G, Leslie K, Vásquez SM, et al. Period-dependent associations between hypotension during and for four days after noncardiac surgery and a composite of myocardial infarction and death: a substudy of the POISE-2 trial. Anesthesiology. 2018;128(2):317–27.
27. Futier E, Lefrant JY, Guinot PG, Godet T, Lorne E, Cuvillon P, et al. Effect of individualized vs standard blood pressure management strategies on postoperative organ dysfunction among high-risk patients undergoing major surgery: a randomized clinical trial. JAMA. 2017;318(14):1346–57.
28. Smith JS, Saulle D, Chen CJ, Lenke LG, Polly DW Jr, Kasliwal MK, et al. Rates and causes of mortality associated with spine surgery based on 108,419 procedures: a review of the Scoliosis Research Society Morbidity and Mortality Database. Spine (Phila Pa 1976). 2012;37(23):1975–82.
29. Bohl DD, Webb ML, Lukasiewicz AM, Samuel AM, Basques BA, Ahn J, et al. Timing of complications after spinal fusion surgery. Spine (Phila Pa 1976). 2015;40(19):1527–35.
30. Harwin B, Formanek B, Spoonamore M, Robertson D, Buser Z, Wang JC. The incidence of myocardial infarction after lumbar spine surgery. Eur Spine J. 2019;28(9):2070–6.
31. Wang TY, Martin JR, Loriaux DB, Desai R, Shammas RL, Adogwa O, et al. Risk assessment and characterization of 30-day perioperative myocardial infarction following spine surgery: a retrospective analysis of 1346 consecutive adult patients. Spine (Phila Pa 1976). 2016;41(5):438–44.
32. Bovonratwet P, Bohl DD, Malpani R, Haynes MS, Rubio DR, Ondeck NT, et al. Cardiac complications related to spine surgery: timing, risk factors, and clinical effect. J Am Acad Orthop Surg. 2019;27(7):256–63.

33. Fleisher LA, Beckman JA, Brown KA, Calkins H, Chaikof E, Fleischmann KE, et al. ACC/AHA 2007 guidelines on perioperative cardiovascular evaluation and care for noncardiac surgery: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to revise the 2002 guidelines on perioperative cardiovascular evaluation for noncardiac surgery): developed in Collaboration with the American Society of Echocardiography, American Society of Nuclear Cardiology, Heart Rhythm Society, Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, Society for Vascular Medicine and Biology, and Society for Vascular Surgery. Circulation. 2007;116(17):1971–96.
34. Ryken TC, Hurlbert RJ, Hadley MN, Aarabi B, Dhall SS, Gelb DE, et al. The acute cardiopulmonary management of patients with cervical spinal cord injuries. Neurosurgery. 2013;72(Suppl 2):84–92.
35. Rogers WK, Todd M. Acute spinal cord injury. Best Pract Res Clin Anaesthesiol. 2016;30(1):27–39.
36. Inoue T, Manley GT, Patel N, Whetstone WD. Medical and surgical management after spinal cord injury: vasopressor usage, early surgerys, and complications. J Neurotrauma. 2014;31(3):284–91.
37. Romito B, Meltzer JS. Tachycardia and Bradycardia. In: Vincent JL, Abraham EMF, Kochanek PM, Fink MP, editors. Textbook of critical care. Philadelphia: Elsevier Inc.; 2017. p. 27–9.
38. Grigorean VT, Sandu AM, Popescu M, Iacobini MA, Stoian R, Neascu C, et al. Cardiac dysfunctions following spinal cord injury. J Med Life. 2009;2(2):133–45.
39. Palumbo MA, Aidlen JP, Daniels AH, Bianco A, Caiati JM. Airway compromise due to laryngopharyngeal edema after anterior cervical spine surgery. J Clin Anesth. 2013;25(1):66–72.
40. Li H, Huang Y, Shen B, Ba Z, Wu D. Multivariate analysis of airway obstruction and reintubation after anterior cervical surgery: a Retrospective Cohort Study of 774 patients. Int J Surg. 2017;41:28–33.
41. Sagi HC, Beutler W, Carroll E, Connolly PJ. Airway complications associated with surgery on the anterior cervical spine. Spine (Phila Pa 1976). 2002;27(9):949–53.
42. Jules-Elysee K, Urban MK, Urquhart BL, Susman MH, Brown AC, Kelsey WT. Pulmonary complications in anterior-posterior thoracic lumbar fusions. Spine J. 2004;4(3):312–6.
43. Murgai R, D'Oro A, Heindel P, Schoell K, Barkoh K, Buser Z, et al. Incidence of respiratory complications following lumbar spine surgery. Int J Spine Surg. 2018;12(6):718–24.
44. Gabel BC, Schnell EC, Dettori JR, Jeyamohan S, Oskouian R. Pulmonary complications following thoracic spinal surgery: a systematic review. Global Spine J. 2016;6(3):296–303.
45. Chughtai M, Gwam CU, Mohamed N, Khlopas A, Newman JM, Khan R, et al. The epidemiology and risk factors for postoperative pneumonia. J Clin Med Res. 2017;9(6):466–75.
46. Daniels AH, Ritterman SA, Rubin LE. Paralytic ileus in the orthopaedic patient. J Am Acad Orthop Surg. 2015;23(6):365–72.
47. Adogwa O, Elsamadicy AA, Fialkoff J, Cheng J, Karikari IO, Bagley C. Early ambulation decreases length of hospital stay, perioperative complications and improves functional outcomes in elderly patients undergoing surgery for correction of adult degenerative scoliosis. Spine (Phila Pa 1976). 2017;42(18):1420–5.
48. Epstein NE. A review article on the benefits of early mobilization following spinal surgery and other medical/surgical procedures. Surg Neurol Int. 2014;5(Suppl 3):S66–73.
49. Como JJ, Sutton ER, McCunn M, Dutton RP, Johnson SB, Aarabi B, et al. Characterizing the need for mechanical ventilation following cervical spinal cord injury with neurologic deficit. J Trauma. 2005;59(4):912–6. discussion 6
50. Petrucci N, De Feo C. Lung protective ventilation strategy for the acute respiratory distress syndrome. Cochrane Database Syst Rev. 2013;2013(2):Cd003844.
51. Nathens AB, Johnson JL, Minei JP, Moore EE, Shapiro M, Bankey P, et al. Inflammation and the Host Response to Injury, a large-scale collaborative project: Patient-Oriented Research Core--standard operating procedures for clinical care. I. Guidelines for mechanical ventilation of the trauma patient. J Trauma. 2005;59(3):764–9.

52. Berlowitz DJ, Wadsworth B, Ross J. Respiratory problems and management in people with spinal cord injury. Breathe (Sheff). 2016;12(4):328–40.
53. Metnitz PG, Krenn CG, Steltzer H, Lang T, Ploder J, Lenz K, et al. Effect of acute renal failure requiring renal replacement therapy on outcome in critically ill patients. Crit Care Med. 2002;30(9):2051–8.
54. Liu KD, Matthay MA, Chertow GM. Evolving practices in critical care and potential implications for management of acute kidney injury. Clin J Am Soc Nephrol. 2006;1(4):869–73.
55. Nolan CR, Anderson RJ. Hospital-acquired acute renal failure. J Am Soc Nephrol. 1998;9(4):710–8.
56. Liaño F, Pascual J. Outcomes in acute renal failure. Semin Nephrol. 1998;18(5):541–50.
57. Brivet FG, Kleinknecht DJ, Loirat P, Landais PJ. Acute renal failure in intensive care units--causes, outcome, and prognostic factors of hospital mortality; a prospective, multicenter study. French Study Group on Acute Renal Failure. Crit Care Med. 1996;24(2):192–8.
58. Uchino S, Kellum JA, Bellomo R, Doig GS, Morimatsu H, Morgera S, et al. Acute renal failure in critically ill patients: a multinational, multicenter study. JAMA. 2005;294(7):813–8.
59. Ympa YP, Sakr Y, Reinhart K, Vincent JL. Has mortality from acute renal failure decreased? A systematic review of the literature. Am J Med. 2005;118(8):827–32.
60. Mehta RL, Pascual MT, Soroko S, Savage BR, Himmelfarb J, Ikizler TA, et al. Spectrum of acute renal failure in the intensive care unit: the PICARD experience. Kidney Int. 2004;66(4):1613–21.
61. Jin K, Murugan R, Sileanu FE, Foldes E, Priyanka P, Clermont G, et al. Intensive monitoring of urine output is associated with increased detection of acute kidney injury and improved outcomes. Chest. 2017;152(5):972–9.
62. Naik BI, Colquhoun DA, McKinney WE, Smith AB, Titus B, McMurry TL, et al. Incidence and risk factors for acute kidney injury after spine surgery using the RIFLE classification. J Neurosurg Spine. 2014;20(5):505–11.
63. Lee JW. Fluid and electrolyte disturbances in critically ill patients. Electrolyte Blood Press. 2010;8(2):72–81.
64. Kraft MD, Btaiche IF, Sacks GS, Kudsk KA. Treatment of electrolyte disorders in adult patients in the intensive care unit. Am J Health Syst Pharm. 2005;62(16):1663–82.
65. Wang LH, Xu DJ, Wei XJ, Chang HT, Xu GH. Electrolyte disorders and aging: risk factors for delirium in patients undergoing orthopedic surgeries. BMC Psychiatry. 2016;16(1):418.
66. Romito B, Dhillon A. Hyperkalemia and hypokalemia. In: Vincent JL, Abraham EMF, Kochanek PM, Fink MP, editors. Textbook of critical care. Philadelphia: Elsevier Inc.; 2017. p. 52–5.
67. Golubovsky JL, Ilyas H, Chen J, Tanenbaum JE, Mroz TE, Steinmetz MP. Risk factors and associated complications for postoperative urinary retention after lumbar surgery for lumbar spinal stenosis. Spine J. 2018;18(9):1533–9.
68. Bladder management for adults with spinal cord injury: a clinical practice guideline for healthcare providers. J Spinal Cord Med. 2006;29(5):527–73.
69. Chaw E, Shem K, Castillo K, Wong SL, Chang J. Dysphagia and associated respiratory considerations in cervical spinal cord injury. Top Spinal Cord Inj Rehabil. 2012;18(4):291–9.
70. Anderson KK, Arnold PM. Oropharyngeal Dysphagia after anterior cervical spine surgery: a review. Global Spine J. 2013;3(4):273–86.
71. Fineberg SJ, Nandyala SV, Kurd MF, Marquez-Lara A, Noureldin M, Sankaranarayanan S, et al. Incidence and risk factors for postoperative ileus following anterior, posterior, and circumferential lumbar fusion. Spine J. 2014;14(8):1680–5.
72. Artinyan A, Nunoo-Mensah JW, Balasubramaniam S, Gauderman J, Essani R, Gonzalez-Ruiz C, et al. Prolonged postoperative ileus-definition, risk factors, and predictors after surgery. World J Surg. 2008;32(7):1495–500.
73. Ogilvy AJ, Smith G. The gastrointestinal tract after anaesthesia. Eur J Anaesthesiol Suppl. 1995;10:35–42.
74. Luckey A, Livingston E, Taché Y. Mechanisms and treatment of postoperative ileus. Arch Surg. 2003;138(2):206–14.

75. Vazquez-Sandoval A, Ghamande S, Surani S. Critically ill patients and gut motility: are we addressing it? World J Gastrointest Pharmacol Ther. 2017;8(3):174–9.
76. Waldhausen JH, Schirmer BD. The effect of ambulation on recovery from postoperative ileus. Ann Surg. 1990;212(6):671–7.
77. Emmanuel A. Neurogenic bowel dysfunction. F1000Res. 2019;8:F1000 Faculty-Rev1800.
78. Chaudhary SB, Vives MJ, Basra SK, Reiter MF. Postoperative spinal wound infections and postprocedural diskitis. J Spinal Cord Med. 2007;30(5):441–51.
79. Olsen MA, Mayfield J, Lauryssen C, Polish LB, Jones M, Vest J, et al. Risk factors for surgical site infection in spinal surgery. J Neurosurg. 2003;98(2 Suppl):149–55.
80. Nasser R, Kosty JA, Shah S, Wang J, Cheng J. Risk factors and prevention of surgical site infections following spinal procedures. Global Spine J. 2018;8(4 Suppl):44s–8s.
81. Klein JD, Hey LA, Yu CS, Klein BB, Coufal FJ, Young EP, et al. Perioperative nutrition and postoperative complications in patients undergoing spinal surgery. Spine (Phila Pa 1976). 1996;21(22):2676–82.
82. Wainwright TW, Immins T, Middleton RG. Enhanced recovery after surgery (ERAS) and its applicability for major spine surgery. Best Pract Res Clin Anaesthesiol. 2016;30(1):91–102.
83. Qureshi R, Rasool M, Puvanesarajah V, Hassanzadeh H. Perioperative nutritional optimization in spine surgery. Clin Spine Surg. 2018;31(3):103–7.
84. Thibault-Halman G, Casha S, Singer S, Christie S. Acute management of nutritional demands after spinal cord injury. J Neurotrauma. 2011;28(8):1497–507.
85. Cox JB, Weaver KJ, Neal DW, Jacob RP, Hoh DJ. Decreased incidence of venous thromboembolism after spine surgery with early multimodal prophylaxis: clinical article. J Neurosurg Spine. 2014;21(4):677–84.
86. Inoue H, Watanabe H, Okami H, Kimura A, Takeshita K. The rate of venous thromboembolism before and after spine surgery as determined with indirect multidetector CT. JB JS Open Access. 2018;3(3):e0015.
87. Mosenthal WP, Landy DC, Boyajian HH, Idowu OA, Shi LL, Ramos E, et al. Thromboprophylaxis in spinal surgery. Spine (Phila Pa 1976). 2018;43(8):E474–e81.
88. Buchanan IA, Lin M, Donoho DA, Ding L, Giannotta SL, Attenello F, et al. Venous thromboembolism after degenerative spine surgery: a Nationwide Readmissions Database Analysis. World Neurosurg. 2019;125:e165–e74.
89. Schoenfeld AJ, Herzog JP, Dunn JC, Bader JO, Belmont PJ Jr. Patient-based and surgical characteristics associated with the acute development of deep venous thrombosis and pulmonary embolism after spine surgery. Spine (Phila Pa 1976). 2013;38(21):1892–8.
90. Piper K, Algattas H, DeAndrea-Lazarus IA, Kimmell KT, Li YM, Walter KA, et al. Risk factors associated with venous thromboembolism in patients undergoing spine surgery. J Neurosurg Spine. 2017;26(1):90–6.
91. Kelly M, Speciale A, Patton C, Turcotte J. A predictive model of preoperative risk factors for venous thromboembolism after fusion for degenerative conditions of the thoracolumbar spine. J Orthop. 2020;22:598–601.
92. McLynn RP, Diaz-Collado PJ, Ottesen TD, Ondeck NT, Cui JJ, Bovonratwet P, et al. Risk factors and pharmacologic prophylaxis for venous thromboembolism in elective spine surgery. Spine J. 2018;18(6):970–8.
93. Alvarado AM, Porto GBF, Wessell J, Buchholz AL, Arnold PM. Venous Thromboprophylaxis in spine surgery. Global Spine J. 2020;10(1 Suppl):65s–70s.
94. Bhimraj A. Postoperative Neurosurgical Infections. 2018. p. 509–517.
95. Epstein NE. How much medicine do spine surgeons need to know to better select and care for patients? Surg Neurol Int. 2012;3(Suppl 5):S329–49.
96. Di Martino A, Papalia R, Albo E, Diaz L, Denaro L, Denaro V. Infection after spinal surgery and procedures. Eur Rev Med Pharmacol Sci. 2019;23(2 Suppl):173–8.
97. Kalfas F, Severi P, Scudieri C. Infection with spinal instrumentation: a 20-year, single-institution experience with review of pathogenesis, diagnosis, prevention, and management. Asian J Neurosurg. 2019;14(4):1181–9.

98. Kasliwal MK, Tan LA, Traynelis VC. Infection with spinal instrumentation: review of pathogenesis, diagnosis, prevention, and management. Surg Neurol Int. 2013;4(Suppl 5):S392–403.
99. Rhodes A, Evans LE, Alhazzani W, Levy MM, Antonelli M, Ferrer R, et al. Surviving sepsis campaign: international guidelines for management of sepsis and septic shock: 2016. Intensive Care Med. 2017;43(3):304–77.

Postoperative Care of the Spine Surgery Patient

17

Lori A. Tappen

Post-operative care of the surgical spine patient starts before the surgery has ever taken place and varies with the type of surgery, extent of surgery, the comorbid conditions of the patient and the patient's willingness to be an active participant in their own care. Without prior planning, education, and setting of expectations, even the most simple day surgery procedure can turn in to a fiasco.

Successful surgical outcomes depend on meeting or exceeding patient, surgeon and hospital expectations. These expectations need to be clear, well defined and understood prior to the surgery. In order to create a predicable or near predictable outcome for the patient, prior planning and education is a must. Generally speaking there are three basic groups of non-trauma spine surgery: degenerative, deformity and spinal tumors. Patient care is tailored to meet the patient's needs to ensure optimal outcomes. Realistic expectations and goals need to be set along with expectations of functional outcomes, postoperative care, medication usage postoperatively, and the length of stay in the hospital. In the following chapter the postoperative care of the surgical patient will be broken down by minimally invasive surgeries of the degenerative nature which require no to one inpatient day stay to the more complicated and invasive deformity surgeries and spinal tumor surgeries that require several days' inpatient stay and more intensive inpatient care.

Preparation for Surgery and Postoperative Care

Patient preparation for surgery is more than getting that pre op clearance and making sure that they arrive at the hospital on time the day of surgery. As with any procedure, education is paramount [3]. The educational process not only includes the patient, but also their caregivers and the nursing staff that will be caring for

L. A. Tappen (✉)
Department of Orthopedic Surgery, UT Southwestern Medical Center, Dallas, TX, USA
e-mail: Lori.Tappen@utsouthwestern.edu

© The Author(s), under exclusive license to Springer Nature Switzerland AG 2022

C. E. Noe (ed.), *Multidisciplinary Spine Care*,
https://doi.org/10.1007/978-3-031-04990-3_17

them. Proper preparation in the beginning will prevent serious downfalls later and will also provide a better patient experience and outcome. Knowing what the patient expects is extremely important. Setting realistic expectations in the beginning with the understanding of what the functional capacity should be post operatively, as well as what the surgery, hospitalization, post-operative care and medication usage will look like will provide a more predictable outcome and improved patient satisfaction.

Expectations

Will the surgery cover the symptoms, all of them, some of them? How much pain should I expect postoperatively? What is the procedure and how do you do it? How many days will I be in the hospital? What medications do I go home on? Will I be normal after this? How long is the recovery? What are my restrictions? The list is extensive and the more detailed the plan and the setting of honest and accurate expectations in the beginning will prevent a lot of hard work later. Most patients do not realize that some surgeries may leave them in increased pain afterwards for a few days to weeks. Make sure that the expectation is not to awaken in PACU pain free. Post-operative pain can be the same, less, more or even different- all of which can be normal for the spine surgery patient. Reassure the patient that the pain will be helped with mediations in the postoperative phase.

Education

Describing the process from the moment the patient agrees to surgery to the last expected post op appointment will give the patient a sense of control. Having the patient become an active participant in their own care helps to solidify the patient's commitment to the success of the surgery. The more actively the patient engages in the process the better the outcome [7]. If the patient is reluctant to actively engage in their care, there are programs available to help promote patient buy in. Sometimes psychiatric evaluations and counseling are beneficial especially for the more invasive deformity surgeries. There are programs designed to prepare the patient for the pre and post-operative phases of surgery such as the ERAS (Enhanced Recovery after Surgery) programs [6] or POSH (Perioperative Optimization of Senior Health) programs. POSH / ERAS program protocols can be designed and made specific for any specialty for any institution using evidence based medicine. The institutions work to design a program that will provide step by step instructions on what to expect the day of surgery and walk the patient through the check in process, the preoperative day surgery area, the intubation, the actual surgical procedure and the post anesthesia care unit as well as post-operative home care–all of which helps to alleviate patient anxiety. Whether using a formal program, packets of information or face to face education; the better prepared the patient the better the outcome.

Aftercare

Before the surgery has been done surgical after care needs to be discussed. The limitations for recovery need to be evaluated and preparations need to be made. Limitations include patient perception of surgical outcomes as well as the social setting around the patient and their comorbid health issues. If the patient requires a prolonged admission in the hospital, a skilled nursing unit, in patient or outpatient rehab, or home health; decisions need to be made and facilities notified as there are criteria that have to be met by many insurance carriers prior to the admission or discharge to a facility. Unfortunately in today's environment, insurance carriers control much of patient care. Insurance carriers now determine which surgeries will be able to have the patient admitted into the hospital and which will not. There are also rules regarding a patient being admitted and having to meet requirements of length of stay or physical ability before transfer. Up front planning and familiarity with the insurance carriers and their requirements will help for a smooth transfer of postoperative care if needed.

The degenerative surgical conditions rarely require an overnight stay in the hospital and most are considered outpatient surgeries by the insurance carriers. Occasionally, the day surgery procedures may require a patient to be kept overnight for observation for pain, limited mobility, lack of support at home, age of patient, or medical health issues that occur during the perioperative and immediate post-operative period. Making nursing staff familiar with Orthopaedic or neurosurgery spine surgery post-operative recovery is imperative. Other than the standard admit orders and medications, the nursing staff need to know what an emergent situation is and what is considered a normal postoperative issue. Sudden changes in sensation, inability to palpate distal pulses, inability to urinate, breathing difficulties, marked bleeding and pain control are frequent issues in the post-operative spine patient. The deformity and more invasive surgeries may also require transfusion protocols and need the patient to be evaluated by Occupational and Physical therapy as well as a social worker for inpatient and discharge care. Detailed in patient instructions and orders for the postoperative spine patient will be addressed in future sections.

General Care Instructions

Inpatient Nursing

Pain is expected postoperatively. The nursing staff need to be aware of what type or intensity of pain is normal. Often spine surgery can leave the patient in more pain than what was present previous to the surgery. Recognizing the intensity and nature of the pain as normal or abnormal is essential. Post operatively the pain can be present as it was prior to the surgical procedure, worse than prior to the procedure and even in a different pattern than prior to the procedure. Sometimes numbness and

tingling can be present as well. Educating the patient and the nursing staff on what to expect will make the immediate postoperative time period smoother and less anxious for the staff and the patient. Controlling this pain and keeping the levels tolerable is important. Medication needs to be given in a manner that will cover the pain symptoms but will not cause the patient to go into respiratory distress. Pain medication in the in-patient setting are usually given orally, by injection or via a PCA pump. The medications that are used intravenously through a PCA are dosed in such a way to cause the patient to fall asleep before they can over self-medicate. Dosing is limited and the machine will lock the ability to deliver further doses of pain medication until a certain time period has elapsed. All patients who receive pain medications via the PCA pump need to be on monitoring for oxygen saturation. In many facilities it is a requirement to also have the patient on 2L oxygen by nasal cannula when on a pain pump. Family members should never push the PCA pump for the patient as they risk causing an overdose of pain medications which can lead to respiratory arrest. Do not keep the patient on PCA for a prolonged amount of time. Patients should be moved to medications by mouth for pain as soon as possible. It is generally thought that post op day 1 or 2 transitioning to PO medications for pain should begin. Educating the patient prior to the surgical procedure and setting definite boundaries of length of time for PCA type medications and then pain medications post op will help to guide the patient and family towards a more favorable experience and outcome.

Neuroleptics, anticonvulsants, some antiarrhythmic medications, steroids, antidepressants, Acetaminophen and Nonsteroidal anti-inflammatory medications (NSAID's) are often used in place of or with opiod pain medications. Caution is needed with administration of these medications with preexisting liver or kidney disease. In the older or geriatric patient the effects of some of the medications can be more intense and lead to confusion or severe sedation. It is helpful with the inpatient admission of the elderly or geriatric patient to get a geriatric medication consult.

Side effect profiles, medications interactions, and the type of surgery the patient has undergone are all pertinent to the patient's medication regimen. Anticonvulsants are used for neuropathic pain and they suppress nerve activity and firing. Anticonvulsants can be very sedating and cause slowed mentation. Patients are often started on these medications at bedtime and the dose is slowly increased over a few days to help build tolerance to the side effects. Examples of anticonvulsants used for neuropathic pain are: carbamazepine, gabapentin, oxcarbazepine, and pregabalin,

It is thought that antidepressant medications may increase neurotransmitters in the spinal cord that reduce pain signals [4]. Common antidepressants used for pain are tricyclic antidepressants such as amitriptyline, nortriptyline, protriptyline, doxepin, imipramine, clomipramine and desipramine or serotonin and norepinephrine reuptake inhibitors such as venlafaxine, duloxetine, milnacipran, and desvenlafaxine. The mechanism of action is not understood fully and they do not work immediately. Because of the delay onset of action, many providers reserve these for the chronic pain management patients.

When prescribing steroids to help with nerve pain from swelling and inflammation caution is needed for the patients who are immunocompromised, have diabetes, TB, ongoing infections, glaucoma, cataracts, osteoporosis, Addison's syndrome, and stomach / duodenal ulcers to note a few. Patients need to be instructed to complete the entire prescription or blister pack of steroids in order to avoid potential problems with the adrenal glands, which in some cases can be fatal. Tylenol, which is used for fever and as a pain medication alternate is not safe in patients with liver disease or liver failure. The NSAID group of medications, which also help with pain management and inflammation should be used with caution in diabetics, renal failure or chronic kidney disease, peptic ulcer disease, and in fusion surgeries or large deformity surgeries. In surgeries where the goal is to grow bone, the surgeon may not want to expose the patient to NSAIDs in the first 3 months of recovery. NSAIDs are believed to delay or even stop bone formation. In lesser deformity surgeries the NSAIDs are used in conjunction with the pain medications to synergistically help to decrease pain and pain medication use [5]. New studies are also indicating that the effects of extra strength Tylenol taken in conjunction with Advil have the same or better pain relief profile as Percocet without the addiction potential. Nonsteroidal anti-inflammatory use should always involve warning the patient to take food with the medications and to be alert for warning signs of gastric irritation or ulcerations.

At all times, the patients cognitive abilities can be affected by any or all of the medications. Elderly patients are also more susceptible to confusion, hallucinations, and to sun downing. The presence of infection can also alter mentation. Assessment of mentation needs to be done at regular intervals. If there is altered mental status the etiology needs to be determined. Infection work up and medication adjustment is to be made based on the mentation assessment.

The taking of the vital signs should be appropriate for the type of surgery. Degenerative cases that are being observed over night the usual every 4 hours is adequate. If the patient has had an event on the operating room table, a complication immediately post op, or has had a significant amount of blood loss- the vitals would be more frequent. During these assessments your nursing staff should be making notes of the patient's mental status, alertness, and the Oxygen saturation levels (every patient on opioid medications should always be on O2 saturation monitoring). Patients that are over medicated will usually have poor inspiratory effort, they will be lethargic and difficult to arouse or engage in conversation. Further evaluation will be needed to rule out more serious issues such as pulmonary embolism and stroke.

It is rare for a degenerative spine surgery patient to require a postoperative drain, however in cases where the patient has a bleeding disorder or is on blood thinning medications a drain may be necessary. Some surgeons use drains on one level lumbar fusion for degenerative cases even if the drain is pulled the same day in order to avoid post op blood accumulation around the surgical site. An accumulation of blood or fluid within the surgical site can be painful. It becomes worrisome especially in cases where there has been a large area of decompression as the fluid can cause a mass effect on the spinal cord or exiting nerves. In the anterior cervical spine surgery bleeding can cause enough compression to compromise patient

breathing. In the smaller surgeries some fluid collection is normal and can often be seen on a post op MRI. Usually these small seromas will be reabsorbed by the body without intervention. If there is concern of neurologic injury or damage, or a compromise in the ability to breathe a patient can be brought back into surgery and have the seroma or hematoma washed out. Cervical hematomas are considered to be an emergent issue whereas lumbar are more urgent than emergent. Prolonged compression can cause permanent injury to the nerve or in the case of a large compressing cervical hematoma, death. If at the bedside and the patient begins to have severely compromised breathing from the anterior cervical bleeding, removal of the surgical sutures allowing the immediate drainage of the hematoma can be done at the bedside.

Fever is usually not seen immediately post op. If there is a fever immediately post op or in the first day or so after the surgery concern usually falls to the lungs as a cause. Pneumonia, aspiration and pulmonary embolisms are common causes. One of the most common causes of fever in the first 48 hours post-surgery is atelectasis. The further out from surgery other causes are more common. Urinary tract infections, in and out catheter related infections or infections from indwelling catheters can appear around post op day 3. Generally abscesses, wound infections, cellulitis, phlebitis, and blood stream infections occur a little later around post op day 5 on. At any time patients can run fever from medications cellulitis, blood stream infections, or blood products from transfusions. Of the causes of fever, atelectasis may be the easiest to prevent and if present treat. Patients should be instructed on the use of incentive spirometry. Encouraging the patient to breathe deeply and frequently until they are able to be up and ambulate regular is important. Failure to treat atelectasis can result in more serious lung infections such as bronchitis or pneumonia. Plenty of fluids and Tylenol along with the increased use of incentive spirometry will usually relieve the fever with in a day or so. Patients need to be educated that the opioid medications also contribute to poor respiratory effort and use should be limited as much as possible. Smoking and inactivity along with pain medications are a perfect storm scenario for atelectasis. The utilization of respiratory therapy for the inpatient setting can also be of benefit.

Postoperative activity for the inpatient and outpatient setting can be determined prior to the surgery with a Fall Assessment Risk. The assessment needs to be made again once the patient has recovered from the anesthesia. There are many factors that contribute to falls within the hospital and community setting and most are preventable or at the least reducible [2]. On the hospital floor, prior to a physical therapy or occupational therapy evaluation the patient needs to be evaluated medically by the admitting doctor and the nursing staff. Lingering effects of anesthesia, impaired mentation, over medication / sedation, blood loss, pain medications, low blood pressure and possible new weakness or paresthesia from the surgery can cause a patient to fall. Recognizing these elements will prevent fall injuries. The post-operative activity level can also be changed depending on circumstances during the surgical procedure. If a patient should have a Dural tear or nerve injury the surgeon may require that the patient remain in bed, sometimes lying flat or sometimes at a head elevation of 30 degrees. When this occurs the patient will need an

indwelling Foley placed. This time period can range from 24–72 hours depending on the severity of the injury to the dura or nerve. Once the patient is able to ambulate the Foley can be removed.

Anesthesia, medications, inactivity, prostatic hypertrophy, cauda equine syndrome and other comorbid conditions can cause postoperative complications of bladder or bowel incontinence, constipation, obstipation, and urinary retention. When these situations occur postoperatively the etiology will often dictate the treatment plan and options. For most men with prostatic hypertrophy the addition of an alpha-blocker (a medication that relaxes the muscles in the prostate and bladder neck, making it easier to urinate) is an option. With the addition of the medication, the wearing off of the anesthesia effect, decreasing the narcotic pain medications and increasing activity help to make this a temporary condition. If a patient is having difficulty urinating immediately post op, doing an in and out catheter up to two times can be an option. If there is still an issue with urination, an indwelling Foley catheter may be placed for a few days. The patient should be instructed to follow up with urology for evaluation and removal of the Foley after discharge from the hospital. Examples of alpha blockers used for this purpose are tamulosin and alfuzosin. Anticholinergic agents are used to treat over active bladders, urge incontinence or incontinence. These medications work by relaxing the bladder muscle allowing the bladder to hold more urine. With urination there is a more complete emptying of the bladder. An example of this medication would be oxybutynin. Over sedated patients often have loss of bladder control as they are unable to awaken sufficiently to get to the bathroom prior to losing control of the bladder. This is different from incontinence and can be corrected with decreasing the pain medications and / or sedating medications. As the patient becomes more awake, they will become more aware of bladder fullness in time to make it to the bathroom. To the other extreme is constipation and obstipation. Anesthesia and pain medications combined with inactivity slow down the gut. If a patient is to be on opiod medications or already has issues with constipation before anesthesia it would be best to start them on a stool softener. If the condition worsens after surgery laxatives and enemas may be used with caution. These medications work best with plenty of water or fluid intake. Orders should be available to the nursing staff to assist the patient with these issues on admission.

Surgical sites should be examined by the nursing staff on arrival to the floor and the next morning. The most common issue with the surgical site immediately post op is bleeding. Compression bandages are usually placed immediately post op and can usually be removed that evening or the next morning by the nursing staff. If there is an excessive amount of bleeding or discharge the dressings may be changed and compression reapplied. Limiting activity and icing the surgical site combined with the compression will help to decrease bleeding. If the patient has torn their internal sutures, external interrupted sutures can be placed to help control the extra bleeding. Rarely, the patient may need to be taken back into the operating room to repair the area of concern.

Tegaderm coverings are common over surgical sites and allow the patient to shower in the postoperative phase. This product is water resistant but not waterproof. Patients are usually allowed to shower the next day after lumbar surgery with

the water hitting them anteriorly and only rinsing posteriorly. Cervical surgery patients are instructed to have the water hit at a level below the incision. Patient's should not soak the area or allow the shower to pour directly over the tegaderm. There are many product type available including different types of skin glues for closure, but the care of the surgical site is essentially the same. Once, the patient has cleaned themselves, the surgical area is patted dry and then bracing if ordered may be reapplied. The tegaderm and skin glues are clear and allows the patient or the caretaker to exam the incision on a daily regular basis to inspect for bleeding, infection, irritation, or other concerning issues. This examination will provide early warning of concerning issues and allow for a more timely treatment window.

DVT Prophylaxis

The risk of a deep venous thrombosis is not common, but can be a possible complication of spine surgery. Inactivity combined with the comorbidities of the patient contributes to the risk of DVT. Most hospitals have a DVT prophylaxis protocol in place. Compression devices, ted hose, early ambulation, and drug therapies such as warfarin help to prevent DVTs. Patients are kept under the DVT prophylaxis protocols until they are up and ambulating on a regular basis. Patients and nursing staff need to be aware of symptoms of shortness of breath, chest pain, or painful swelling, erythema, and heat of the calves, as this could be a sign of DVT. Patients exhibiting these signs need to be evaluated quickly. Patients are usually allowed to resume their home anticoagulant medications post op day three, sometimes bridging with alternate medications are done if there is a concern of bleeding at the surgical site.

Bracing

Types of bracing and prescribed length of time of use for cervical and lumbar spine patient differ. Cervical spine patients that have had an anterior cervical fusion (one to three levels) or a total disc replacement (up to two levels) will often be placed in a neck brace immediately postop to prevent accidental injury to the patient's cervical spine when transferring the patient from the operating table to the day surgery stretcher. Once the patient is fully awake the brace may be removed. Guidelines for length of time to wear cervical bracing for anterior cervical fusions vary from 2 to 6 weeks, based on each surgeon's preference regarding patient bone quality and other risk factors. Data has shown that the bracing of an anterior cervical fusion does not improve fusion rates and can be associated with other complications with extended wear times [1]. Posterior cervical fusions are thought to need bracing to protect the hard ware from becoming loose from stain of movement. With surgeries for degenerative conditions, the anterior cervical fusion patients are usually instructed to wear the brace in a moving vehicle only to prevent injury from whiplash. The brace is usually discarded after a 2–4 week period. Constant wearing of the neck brace is usually discouraged.

Lumbar bracing varies with the intensity and type of surgery. In the degenerative cases such as decompression laminectomies, discectomies and lumbar fusions (posterior/ anterior/ lateral / interbody) of 1–3 levels a hard brace is not always used. Deformity or tumor surgeries typically use hard bracing. These braces may cover from the lumbosacral spine all the way up to the thoracic spine. Length of use also varies depending on the severity and levels of surgery. Hard bracing can be used for 6 weeks to 3 months depending on surgeon preference and number of levels of surgery. Soft elastic binders with Velcro closures are often used in lieu of the hard brace for simple or degenerative cases. In this case, the concern is more about the hoop stress of the abdomen on the skin of the back. This hoop stress pulls on the surgical incision affecting the ability to heal the skin. A soft elastic wrap around binder that uses Velcro closure can provide additional support to the skin. Patients will like the binder as it does take pressure off of the low back. Patients are instructed to wear the binder 24 hours a day, 7 days a week for 3 weeks. They are allowed to remove the binder to shower or to have the binder cleaned. Once the patient is seen at follow up and the incision is healing, the binder may be discontinued. Prolonged wearing of either type of binder past the prescribed time frame is discouraged as it will weaken or decondition the muscles of the back, causing the patient to ache more once it has been discontinued. Most binders are now latex free, however there are some patients that are sensitive to wearing binders. For the sensitive skin patient it is advised that they wear a T-shirt or cotton shirt under the binder to avoid contact with the skin.

Discharge

Prior to discharge, patients need to be assessed by Occupational Therapy, Physical Therapy, and in some cases by a Social Worker. Home care and safety assessment need to be made. Patients with no home help or support may need home health visits or admittance to rehabilitation or nursing care facility. Prior planning before the surgery will help to make the transfer more seamless. Due to insurance carrier criteria, some prerequisites need to be met. Length of stay, and physical limitations and abilities are some of the factors to determine if a patient can be admitted into a transitional facility. Whether the patient is transitioning to home or to another care facility the patient and the family are to be given discharge instructions on what to expect during the postoperative phase, follow up appointments, incisional / wound care instructions, physical restrictions, and warning signs of possible complications. If the patient is transferring to another facility a copy of these discharge instructions need to be included. Consultation with an inpatient physical medicine and rehabilitation team can also give reliable answer on where a patient would best recover after acute hospitalization. Recommendations could be any of the following skilled nursing, inpatient rehab, home health therapies and other services, and home with no additional care needed. Care coordinators are usually located on the floor can be a very useful tool for proper transition of care to facilities or to ensure that proper home care has been setup for patient.

Activity After Discharge

In degenerative surgeries the patients are usually able to be up and ambulating the day of surgery once the effects of the anesthesia have been cleared. Patient's need to be cautioned to resume their activity slowly and to avoid strenuous activity for the next 6 weeks. Walking is usually the best form of exercise and is best done with gradually increasing the distance, time and intensity over a few weeks. As the patient experiences less pain and starts to gain more strength and endurance they can slowly increase the amount and distance of their walking. Even pavement or walking inside a building or mall is usually best. There will be days when the patient feels that they can increase the activity dramatically. Patient needs to understand that in the postoperative period of the next four months their activity can be increased on good days, but not excessively. Over activity on a good day will lend several painful bad days later. Each day will be different; the main idea is to adjust their activity accordingly. On bad days back off of the activity and on good days increase the activity but in a limited manner. The further out from the surgery, there will be less variation in the symptoms and abilities. Usually by four months postop most patients are doing quite well.

Neuropathic pain usually does not always resolve quickly. It can take a nerve up to 120 days to start the healing process once it has been relieved of pressure. Occasionally the patient may have the neuropathic pain resolve only to have it return. When this occurs without history of trauma, a good history of patient activity and home care is needed. Often, the patient has been over active and is not icing the incision. Deep tissue swelling can also cause pressure on the site of surgery causing the nerves or tissues to believe that they are being compressed much as they were with the pre surgery compression caused by disc, ligamentum flavum, bone, etc. The body senses post-operative deep tissue swelling and physical pressure from issues with the anatomy in the same way. Icing the skin 20 minutes every hour helps decrease the swelling of the deep tissue and helps to decrease the symptoms. When a patient ices the incision they need to be made aware to wrap the ice in a towel to protect the skin from damage. The skin requires time between icings to recover. Usually 45 minutes to an hour will be enough. This practice should continue at home on discharge for the first few weeks. If appropriate, anticonvulsant medications can help with the neuropathic symptoms.

Lifting restrictions are commonly put in place to prevent injury to the newly forming bone herniated disc, or surrounding tissue and skin. Cervical fusion surgeries need to avoid lifting greater than 10 pounds and overhead lifting for the 6 weeks period. Lumbar fusions, decompressions and discectomies are limited to lifting of no more than 10–20 pounds for the 6 weeks period. Caution should be used with twisting and bending as these activities put undue stress on the skin incision and can cause the incision to dehisce, cause sutures to be broken, and there can be bleeding. Open incisions can lead to infections. Twisting, bending and lifting can also cause a risk of re-herniating the disc if too much stress is placed on the disc in the post-operative period. In fusions, the new bone growth is sensitive to extreme stress. The newly forming bone can be fractured stopping the fusion process causing a pseudo arthrosis.

The incision should be inspected daily by the patient or care giver. In surgeries the incision is typically covered with steri strips and then Tegaderm. The Tegaderm helps to protect the incision from infection and allows the patient to shower without fear of compromising the surgical site. Patients need to understand that this product is water resistant but not water proof. Showers are encouraged but absolutely no soaking in a tub or immersion in water. The Tegaderm is removed 7 days post-surgery or sooner if there is evidence of water leakage or large amounts of bleeding underneath. In some patients, skin sensitivity to the Tegaderm can develop. Those patients are instructed to remove the Tegaderm covering as soon as they have itching, redness, or blistering. Patients are generally instructed to not immerse the surgical site in water until instructed otherwise by the provider. Two to three weeks post op, the patient is seen in the office to evaluate the healing of the surgical incision and for removal of external sutures or staples if there are any. If external sutures or staples are removed, the patient should not immerse the incision for at least 24 hours. If there is scabbing or incomplete healing this will delay immersion longer. It is not uncommon for patients who are obese, immunocompromised, or who have poor nutrition to develop eschar or wet scar on the incision. Eschar healing is slower, there can be drainage and if the healing is delayed enough the incision can be pulled opened causing dehiscence. Patient will need to be trained to keep the area clean and dry, and understand that this is a delayed healing that now needs to be monitored more diligently. Risk of infection is now greater as is the risk of fully dehiscing the incision. Some practitioners will monitor the incision, keeping it clean and dry. Some prophylactically will cover the patient with antibiotics depending on their health status and the overall look of the incision. At this time, reviewing dietary needs such as sufficient amounts of daily protein intake are essential. There are many types of dressings and knowing the manufactures recommendations are important. Referral to a wound care clinic or specialist can also be helpful especially in the more difficult to heal cases. Occasionally, the patient may need to be brought back into surgery to debride and reclose the incision.

Over activity in addition to possibly causing the incision to dehisce can also cause bleeding. Small fluid collections (seromas) deep in the surgical site can leak to the surface of the incision. Patients have also been known to break their deep sutures and cause hematomas. Seromas and hematomas if large enough can cause increased pain as well as an opportunity for infection to occur. If the drainage is significant the patient needs to be examined in the office. The exact area of leakage can be determined by having a patient lay on a clean Abd pad for 5–10 minutes. At the end of the time, the pad can be examined for the exact area of concerned. Adding additional external sutures is an option. If the drainage is small enough, having the patient restrict activity, apply pressure to the incision along with icing the area will help to stop the bleeding. Often these patients are placed on prophylactic antibiotics. If there is any question of infection- fever, feeling ill, erythema and heat from the incision, purulent drainage, etc. the patient should have blood markers taken as a base line. These labs should include but are not limited to Complete blood count, Sedimentation rate and C reactive protein. If an infection is suspected and the patient has had hardware implanted at the surgical site, a return to the OR to debride

and clean may be needed. If the infection has reached the level of the hardware, a consult to Infectious Disease is indicated and possibly removal of the infected hardware.

Driving a vehicle should never be done while taking pain medications. Reaction time, muscle strength and coordination need to be normal before attempting to drive. A common rule to prevent injury to the surgical area and to keep the patient safe is no driving for 1–2 weeks post op, depending on the type and intensity of the surgery. The no driving period may be extended even further out for the patients who have had tumor or deformity surgery.

Pain Management and Poly Pharmacy

As previously discussed pain medications and pain management need to be addressed prior to the surgery and expectations need to be made clear. To avoid potential opioid abuse patients should be instructed to be using only one pharmacy for their prescriptions. Many states have implemented secure sites to review a patient's medication refill history. There are federal and state laws regarding the prescribing and refilling of opioid medications. Strict guidelines have been imposed to protect the patient and the practitioner from becoming entangled in an opioid abuse situation. Concurrent use of alternate non-opioid medications outlined in the previous section with continued use after discontinuing the opioid medications will help to transition the patient to non-opioid pain control. Patients should be made aware that the plan is to stop the opioid medication as soon as is reasonably possible. Prolonged use of opioid medications will not be prescribed and that the expectation should be to have the opioid medications discontinued within a few days to a few weeks. The patient should be aware that some pain might continue to be present, but that pain will be treated with other modalities until they have healed. Make the patient aware that some postoperative pain is normal and should be expected. With tincture of time they will soon be returning to their normal base line -which could be from a few weeks to a few months. For the more invasive surgeries recovery can often take up to a year or more. Physical therapy can be utilized at different stages of healing, providing techniques on daily activities of living, stretching and strengthening to more aggressive work conditioning programs when the patient is ready. As the patient progresses in their care, the level of pain will change. Medication management needs to be adjusted and decreased as appropriate, with the goal of stopping all medications.

Many patients fear that the pain will be intolerable and so intense that they will not be able to cope. Not addressing these fears will increase the patient's reluctance to reduce or stop narcotic medications. Fear of not getting a refill of narcotic medications will often lead to patient stock piling their medications in anticipation that they would be unable to get any further refills. Honest and direct communication is the only key to preventing these types of behaviors. If the patient exhibits intense concern about pain management it is prudent to get a psychological / behavioral

consult prior to proceeding with the surgery. The same plan would also apply to patients that are already on opioid medications and have been treated for pain for some time. Chronic pain management patients can be "detoxed" or weaned off of or decrease the use of their opioid medications prior to the surgical procedure. Decreasing these medications will reset the patients' opioid level of tolerance and allow the practitioner better control of the opioid needs postoperatively.

Icing, early ambulation and proper body mechanics will help the body return to its baseline. Gradually increasing activity over time, limiting lifting, bending and twisting for several weeks postoperatively will allow proper healing and eventually decreased pain with increased activity or abilities.

Early on patients need to be educated on their physical limitations immediately postop and for the next few months. The goal is to prevent injury to the patient and to facilitate healing of the surgical site. Although a patient may be experiencing pain, they still need to become mobile. Lifting restrictions are usually placed on the patient to prevent injury to the healing surgical site. Cervical patients are usually limited to lifting up to 10 pounds with no overhead lifting. Lumbar patients are usually limited to 10–20 pounds lifting. These restrictions generally last for the first 6 weeks of the recovery phase, but depending on the severity of the surgery or the physical condition of the patient, these restrictions may need to be in place longer. As the patient heals, the ability to lift and be more active can resume slowly. Educating patients on a slow return to normal physical activity will prevent re-injury. In some case physical therapy or work conditioning may be necessary. With physical therapy, age appropriate care should be taken. The expectation is not to make every patient young again; it is to get them back to being active to where they can resume their normal activities of daily living. If one is not familiar with the physical therapist caring for their patient, and there is a concern of over doing therapy; try to pair older patients with older physical therapists or trainers and younger patients with younger therapists or trainers. In pairing this way, there is less likelihood of not having the patient injured from over an aggressive physical therapist.

Complications and Emergencies After Discharge

Once the patient has been discharged home, the complications and emergency situations change from that of the inpatient status. Risk of DVT or PE is decreased but still present. Fevers, infections, pain control, mobility, self-care and medication issues become more common. Shortness of breath, chest pain, swelling of the extremities, difficulty swallowing, fever greater than 100 degrees Fahrenheit, incisional changes, drainage, or acute changes in strength or mobility are emergencies that need to be evaluated. Providing the patient and their caregiver a list of the general complications and emergencies with a way to contact the provider or their office is important. If the situation is severe or the patient believes that there is a possibility of loss of life, patients should be instructed to call 911 for emergency services and transport to a hospital first.

References

1. Camara R, Ajayi OO, Asgarzadie F. Are external cervical orthoses necessary after anterior cervical discectomy and fusion: a review of the literature. Cureus. 2016;8:e688.
2. Ganz DA, Latham NK. Prevention of falls in community-dwelling older adults. N Engl J Med. 2020;382:734–43.
3. Donna Eastwood RMO, et al. Improving postoperative patient reported benefits and satisaction following spinal fusion with a single preoperative education session. Spine J. 2019;840(19):840–5.
4. Ivan Urits, J. P. M. S. O. J. W. R. P. V. O. A. D. K. O. V.. Off-label antidepressant Use for Treatment and Management of Chronic Pain:Evolving Understanding and Comprehensive Review, s.l.: Current Pain and Headache Reports, 2019.
5. Lumawig JMT, Yamazaki A, Wantanabe K. Dose-dependent inhibition of diclofenac sodium on posterior lumbar interbody fusion rates. Spine Jouirnal. 2009;9(5):343–9.
6. Megan Melnyk MM, Casey RG, Black P, Koupparis AJ. Enhanced recovery after surgery (ERAS) protocold: time to change practice? CAUJ/ JAUC. 2011;5(5):342–8.
7. Skolasky RL, et al. Functional recovery in lumbar spine surgery: a controlled trial of health behavior change counseling to improve outcomes. Contemp Clin Trials. 2013;36(1):207–17.

The Neurosurgical Management of Pain

18

Eric Montgomery, Cody Wolfe, Jeffrey Traylor, Salah G. Aoun, and Mazin Al Tamimi

Introduction

Pain is a complex human experience informed by both its psychosocial components and the fundamental neurobiology of nociception. Although primarily treated pharmacologically, many advances in neurosurgery provide treatment options to alleviate refractory pain. To best understand these options, pain should be classified by its pathologic origin. Nociceptive pain results from activation of the A-delta and C fiber peripheral pain receptors to noxious stimuli. Neuropathic pain, on the other hand, is due to the disproportionate representation in the CNS of pain signaling, often due to injury of the somatosensory nervous system. Nociplastic pain is a recently defined, intermediary category of pain that involves altered nociceptive signaling, without activation of nociceptive receptors or injury to the somatosensory nervous system. Pain may also be categorized as primary or secondary. These encompass musculoskeletal diseases that may or may not stimulate nociceptors. Examples of primary pain conditions, which lack nociceptive signaling, include fibromyalgia, complex regional pain syndrome, and non-specific low back pain. Whereas secondary pain involves nociceptive signaling and may be due to chronic inflammation from crystal deposition, structural damage such as osteoarthritis, or diseases of the nervous system such as Parkinson's disease or multiple sclerosis. Cancer-related pain falls under the purview of mixed pain as it frequently involves nociceptive and/or neuropathic pain depending on the location.

E. Montgomery · C. Wolfe · J. Traylor
Department of Neurological Surgery and Spine Clinic, UT Southwestern Medical Center, Dallas, TX, USA
e-mail: Eric.Montgomery@UTSouthwestern.edu; Cody.Wolfe@phhs.org; Jeffrey.Traylor@UTSouthwestern.edu

S. G. Aoun · M. Al Tamimi (✉)
Department of Neurological Surgery and Spine Center, UT Southwestern Medical Center, Dallas, TX, USA
e-mail: Salah.Aoun@UTSouthwestern.edu; Mazin.AlTamimi@UTSouthwestern.edu

© The Author(s), under exclusive license to Springer Nature Switzerland AG 2022

C. E. Noe (ed.), *Multidisciplinary Spine Care*,
https://doi.org/10.1007/978-3-031-04990-3_18

The neurosurgical treatment of pain is divided into two primary strategies: ablation and neuromodulation. Historically, ablative procedures involve the destruction of a neural structure via coagulation or radiofrequency methods. Ablation targets are often inflamed or injured from neighboring structures, resulting in hyperactive nociceptive stimuli. Neuromodulation techniques are more recent and provide stimulatory input via an electrode to modulate the transmission of nociceptive stimuli anywhere from the periphery to the brain. Spinal cord stimulation (SCS), deep brain stimulation (DBS), and peripheral nerve stimulation (PNS) are neuromodulation procedures that hold great potential for analgesic therapy in the future. Additionally, direct administration of analgesic drugs into the intrathecal space provides effective relief for many patients and bypasses the compendium of systemic side effects accompanying these medications. Lastly, we will cover two classic pain conditions, trigeminal neuralgia and occipital neuralgia, whose neurosurgical treatment has been extensively studied.

Spinal Ablative Procedures

Ablative procedures represent the first attempts to modulate the phenomenon of pain. Spiller and Martin reported in 1912 their employment of sectioning the anterolateral tract of the spinal cord to treat lower body pain [1]. Since then, sectioning has been attempted in a multitude of locations and with numerous methods, including knives, radioactive strontium needle, and electric current or radiofrequency electrodes among others [2, 3]. The revelations by Melzack and Wall in 1965 represented a watershed moment in the understanding of nociception, and although most of the initial techniques are not used today due to lack of empirical support, some neuro-destructive procedures remain indicated for patients with refractory pain conditions. Their use, however, is relatively limited compared to neurostimulatory procedures and intrathecal analgesia injection due to the lack of titratability and reversibility of the treatment. Nevertheless, the procedures covered below require an intimate and comprehensive knowledge of neuroanatomy to understand how a lesion at each specific loci affects the transmission of nociception.

Anterolateral Cordotomy

Overview

The target of an anterolateral cordotomy is the anterolateral spinothalamic tract (STT), whose fibers carry pain and temperature information from the contralateral half of the body. The function of sectioning these pathways is to control pain in all regions below the lesion on the opposite half of the body. An important anatomical nuance of the STT is that the primary afferent fibers, after entering the spinal cord via the Lissauer tract, ascend ipsilaterally for a few vertebral levels before forming synapses with second-order neurons in the substantia gelantosa or nucleus proprius, before crossing the anterior white commissure. Clinically, this means that a C1/2 cordotomy will modulate pain sensation at C5 or below. Thus, the ideal candidate

for this procedure exhibits refractory cancer-related pain of the lower trunk and/or extremity [4]. Patients with non-cancer-related pain are not considered ideal candidates due to their propensity for pain recurrence or emergence of neuropathic pain [4]. One contraindication unique to cordotomy surgery is poor respiratory function [5]. The spinal nuclei regulating autonomic breathing are adjacent to the STT in the craniocervical region and require great caution not to compromise during cervical cordotomies. A one-second forced vital capacity of at least 12 mL/kg is recommended before undergoing surgery. Other contraindications for cordotomy when using the percutaneous technique include the inability to lie still for the entirety of the procedure or to accurately communicate changes in sensation.

Percutaneous Versus Open Surgery

The operation can be carried out percutaneously or via open surgery, depending on the analgesic target region. Open cordotomies are approached via full or hemilaminectomy, and after dural opening, the dentate ligament is sectioned at the target spinal level to allow for manipulation of the cord [5]. The medial pia must not be disrupted to ensure maintenance of the anterior spinal artery. Comparatively, percutaneous cordotomy utilizes radiofrequency ablation and allows for the operation to occur while the patient is awake under local anesthetic. It is typically performed at the C1/2 interspace under the guidance of fluoroscopic or CT imaging, the latter of which has greatly reduced complication rates. After injecting intrathecal contrast dye via lumbar puncture, the needle is advanced to the spinal target before inserting a radiofrequency electrode. Procession of the electrode into the cord is charted via impedance mapping, with an increase in impedance from 300 ohms in the CSF to 700 signaling entry into the spinal cord [6]. Local spinal cord mapping is carried out via low- and high-frequency stimulation, where low-frequency stimulation determines the motor threshold that reflects the distance from the corticospinal tract. High-frequency stimulation yields sensory changes in the contralateral half of the body and dictates position in the STT, at which point the needle is heated to 80 °C for 60 s [5].

Complications

Cordotomy historically has a low complication rate at 5%, especially with the move away from open procedures [4]. The complication that surgeons most hope to avoid with cordotomy is pain re-emergence. Despite percutaneous and open cordotomy achieving pain relief in 90% and 77% of patients immediately after surgery, respectively, only half of the patients maintain that relief after 1 year [4]. This is the main reason that patient selection is key. Cancer-related pain purports better results here in part due to the shortened life expectancy of the patients and thus less time for the pain to re-emerge. Even so, patients that do experience re-emergence of their pain still report that the procedure reduced their dependence on analgesic medications such as opiates [4]. Mortality in cordotomy patients (3%) is mostly a consequence of respiratory suppression (aka Ondine's curse) due to the aforementioned proximity to the autonomic nuclei regulating breathing [4].

The most common complications observed reflect the functions of the neuroanatomical structures being manipulated. Compromised spinocerebellar tract or

corticospinal tract function results in ataxia or paresis, respectively, and fortunately, these impairments resolve rapidly in most patients (2.9–100%) [7]. However, a minority of patients may have lasting functional deficits (1–20%) [7]. Urinary retention or incontinence and permanent dysesthesias have also been reported in 11–33% and 7–11% of open cordotomy patients, respectively [4]. In percutaneous cordotomy patients, bladder dysfunction and temporary ipsilateral weakness are the most common complications, both occurring in 7.6% of patients [4]. Other reported complications are likely due to lesioning of adjacent neuroanatomical structures, which may result in acquired sleep apnea, sympathetic dysfunction, and Horner syndrome among others [5]. Another interesting complication is mirror pain in open thoracic cordotomy patients, where the patient reports the same pain but opposite the side of the original pain within weeks or months of the operation.

Dorsal Root Entry Zone (DREZ) Lesioning

The Dorsal Root Entry Zone (DREZ) is the region adjacent to the spinal cord where first-order neurons carrying nociceptive information either form synapses in the dorsal horn (at Rexed lamina I–V) or pass through and rostrally ascend via the Lissauer tract before synapsing. In plexus avulsion, the pain generator is focused on the nucleus proprius (lamina III–IV), which are known as the "wide dynamic range" neurons that transmit both non-painful and nociceptive signals. DREZ lesion surgery is best suited for patients with pain distinct to the unique pattern of fibers as they pass through the DREZ, commonly manifesting as pain in a unilateral limb or pain limited to the brachial plexus [4]. Such pain is often described as prickly or electric, shooting pain, loss of sensation without loss of motor function in the affected limb, or hyperpathia between normal sensation and loss of sensation associated with spinal cord injury [4]. Ideal DREZ surgical candidates include patients with traumatic brachial plexus injury, segmental pain at the same vertebral level as their spinal cord injury (potentially due to a pseudomeningocele), or any lesion in the thoracic apex that compresses the inferior brachial plexus (e.g. Pancoast tumor) [4]. Findings that portend poor outcomes with DREZ lesioning include constant burning limb pain, herpetic neuropathy-related pain, pain that extends medially from the limb into the trunk or pelvis, or burning pain in lower extremities that does not correspond to the level of the spinal cord injury [4].

Spinal DREZ

Spinal DREZ procedures are approached via hemilaminectomy for unilateral and laminectomy for bilateral operations, at least two vertebral levels above and below the lesion location [4]. Remembering the orientation of the nociceptive fibers as they enter the dorsal root, the ideal location for electrode placement is along the lateral edge of fibers as they enter the cord. Impedance mapping can help navigate the healthy and normal fibers, where values of 500–1000 ohms indicate an injured region and 1200–2000 ohms represent normal neural matter. Once in the ideal location, radiofrequency ablation is carried out at 75 °C for 15–20 s. As the surgeon

proceeds to make a series of small lesions to the DREZ, they must remain focused on maintaining the electrodes position parallel to the dorsal lateral sulcus rootlets [4]. The operation is complete upon reaching one or two vertebral levels above the site of injury or when impedance mapping has normalized.

The best outcomes in DREZ operations are seen in brachial plexus injury patients, with 54–91% of patients expressing immediate improvement of their pain and at least 50% maintaining those results after 5 years [4]. For spinal cord injury patients, better results are seen for end-zone pain (78%) rather than diffuse distal pain (20%). Multiple groups have suggested that intraoperative impedance mapping improves treatment effectiveness by revealing DREZ regions of hyperactivity that would not have been anticipated preoperatively [8, 9]. Friedman and Bullitt found in their series on postherpetic neuralgia patients that almost all patients reported positive initial results (29/32) but only eight expressed lasting pain relief after 1 year [10]. The most common complications result from mechanical error in lesioning adjacent structures. Since 1990, 3–14% of patients experience ipsilateral weakness due to injury to the corticospinal tract, which runs lateral to the site of lesion [4]. Thoracic DREZ lesions are at the greatest risk for this complication, as the dorsal horn is thinnest in this region. Sensory loss, such as ipsilateral proprioception, light touch, and vibration due to injury to the dorsal columns medially, is reported in 2–70% of studies [4]. The broad range may be explained in part by small sample sizes or non-representative sample populations.

Nucleus Caudalis DREZ

Alternatively, DREZ lesions may aim to ablate the spinal trigeminal nucleus pars caudalis within the cervicomedullary junction. Destruction of this structure was initially pursued as treatment for pain conditions of the craniofacial region, including trigeminal deafferentation pain, trigeminal neuropathic pain, and glossopharyngeal or occipital pain [11]. With the unique somatotopic coverage allowed for by targeting the nucleus caudalis, reported results have been encouraging. In a recent series of 16 nucleus caudalis DREZ patients, Chivukula et al. described a mean pain reduction of 58.3% lasting for an average of 4.3 years [12]. Complications included ataxia (2/16) and neuro/radiculopathy (2/16).

Punctate Midline Myelotomy

Midline myelotomy (MM) is the lesion of the anterior commissure that projects the second-order neurons of the STT contralateral from their origin. Indications for MM are similar to those of cordotomy, with the exception that MM is advantageous for patients with bilateral, instead of unilateral, extremity pain by reducing the risk of duplicitous lesioning procedures. Additionally, the approach allows for the midline division of the dorsal columns, which carries some visceral pain information. The operation can be carried out by one of three techniques, including open limited myelotomy, percutaneous radiofrequency myelotomy, or percutaneous mechanical myelotomy. The approach is carried out via midline laminectomy and durotomy,

and the lesion is continued until the anterior pia is detected, indicating complete division. A limited MM has become popular in recent years to specifically treat visceral pain, where the lesion is only carried out in the dorsal columns. The surgeon must recall that thoracic spinal targets are several levels above their corresponding vertebral bodies, so for example, T3–4 is the common target for upper abdominal pain and T6–7 is used for perineal pain [13].

Besides being a good surgical candidate with a life expectancy of greater than 3 months, indications for MM include patients with bilateral lower extremity or abdominal/pelvic organ pain refractory to opiates [14]. The studies reporting outcomes are limited but over half of patients report good or excellent results postoperatively [4]. In on recent series, Vedantam et al. reported better outcomes for patients undergoing open limited myelotomy, compared to percutaneous radiofrequency lesioning or percutaneous mechanical lesioning [13]. Complications include leg weakness (27%), dysesthesias causing gait disturbance or burning sensation (9%), or impaired proprioception due to manipulation of the dorsal columns [4]. However, results from these studies must be taken in the context of the patient population, who are often terminal cancer patients ridden with progressive malignancy.

Neurotomy

Neurotomy or neurectomy is a well-described procedure that seeks to address focal regions of painful dysesthesia by lesioning the corresponding sensory nerves. Candidate nerves include the saphenous nerve, sural nerve, superficial radial nerve, and the cutaneous nerves of the trunk and upper/lower extremities. Neuromas, the proliferative growth of axons and Schwann cells without end innervation, are a common source of such pain syndromes and require resection. The surgery can be carried out under general anesthesia or simply with peripheral nerve blocks. The culprit nerve is transected and implanted into the proximal muscle, enabling the facilitation of axonal growth [15]. Neurolysis is employed to relive compressive injury to peripheral nerves, most commonly the median nerve in the carpal tunnel and the ulnar nerve in the cubital tunnel. Compression not only hinders axonal transport, but it also interrupts microvascular supply, causing ischemia and edema along the downstream nerve. The procedure involves first mobilizing the proximal and distal ends of the nerve before localizing to the entrapped nerve. The nerve is then freed circumferentially from the interfering structure, which in some cases may require interfascicular dissection to delineate pathologic nerve from healthy nerve.

Outcomes are generally positive for these procedures. Meta-analyses for neurolysis of the median nerve, ulnar nerve, and lateral femoral cutaneous nerve have described pain relief in 87%, 70%, and 88% of patients, respectively [15]. A recent area of study, one that remains controversial despite some positive evidence, is multi-tunnel decompression for diabetic neuropathy patients. Meta-analyses for neurotomy studies have reported excellent outcomes for meralgia paresthetica, with pain relief in 85–95% of patients [15, 16]. Positive results for neuroma patients can be expected in 68% (95% CI: 51–84%) of patients after neurolysis and 74% (95%

CI: 66–82%) of patients [17]. Complication rates are overall quite low for these procedures, at less than 5% [15–17]. Patient selection and patient understanding of treatment goals are key to preventing complications. Some patients report neuropathic pain in the immediate postoperative period, which should resolve in the subsequent days to weeks [15]. In the case of neuromas, recurrence is inevitable, so the goal of treatment is to prevent painful neuromas from forming, not to eliminate the neuroma altogether.

Dorsal Root Ganglionectomy

Dorsal root ganglionectomy (DRGectomy) was initially proposed as a superior alternative to neurotomy, focusing on the primary afferent cell bodies to prevent peripheral nerve regeneration, remove the ventral root afferents arising from the DRG, and eliminate any spontaneous nociceptive activity in the DRG [18]. Initial studies described positive outcomes for failed back surgery cohorts, [19] but longitudinal outcomes were overall disappointing [20]. Not only did pain recur, but it tended to spread across dermatomes that were not initially involved [21]. Recent efforts have examined its efficacy in treating occipital neuralgia via the C2/3 DRG. However, its results demonstrate consistent pain recurrence with inferior results to neuromodulatory treatments [22–24].

Intracranial Ablative Procedures

Bilateral Anterior Cingulotomy

The cingulate gyrus is a central structure inferomedial to the cerebral hemispheres that serves as a transmission point of multiple core cerebral functions, including cognition, motor, emotional, pain, and visuospatial. It is the anterior cingulate cortex (ACC) that is the focus of emotion and pain modulation. Neurosurgical ablation of the ACC has been explored in neuropathic pain conditions as well as obsessive-compulsive disorder (OCD). The lesions are carried out via stereotactic thermocoagulation under the guidance of intraoperative neurophysiological recordings. Retrospective studies have reported positive outcomes in 45–70% of OCD patients with ablation of the dorsal ACC [25]. Systematic reviews of intractable cancer-related pain patients found reports of pain relief in 32–83% of patients [26, 27]. Complications are rare at less than 5% in reported series and include seizures, hemiparesis, and mood change or apathy.

Medial Thalamotomy

Ablation of the medial thalamus, specifically the nociception-transmitting mediodorsal, centromedian, intralaminar, and parafascicularis nuclei, is carried out via

stereotactic radiosurgery (SRS). Focal radiation doses of 140–180 Gy are delivered to the nuclei and provide generally positive outcomes, depending on the pathologic origin of the pain [28–31]. Indications arise after the failure of multiple non-surgical interventions and include malignancy-related pain, trigeminal neuralgia, postherpetic neuralgia, trigeminal neuropathic pain, thalamic pain, and facial pain among others [31]. In one of the most recent case series on trigeminal neuralgia patients, Gallay et al. found that 63% of patients had pain relief at 3 months and 88% at 1-year postop, without any serious adverse events [28]. Conversely, Roberts and Pouratian reported in their systematic review only 23% pain relief in thalamotomy SRS patients [30]. Nevertheless, these patients suffer from chronic refractory pain conditions, where any pain relief is clinically beneficial. Complications are infrequent but may arise due to imprecise or excessive radiation, including radionecrosis and collateral damage to adjacent structures.

Mesencephalotomy

Mesencephalotomy is the stereotactic ablation of the spinothalamic, trigeminothalamic, or spinoreticular tracts as they pass through the midbrain. The first two tracts transmit peripheral nociceptive signals, while the latter is involved in the emotional response to pain. Specifically, the lesion targets the inferior colliculus, splitting the difference between the lateral edge of the aqueduct and the lateral edge of the midbrain. The procedure is reserved for cancer patients with limited life expectancy and unilateral nociceptive pain that has failed previous therapy, particularly of the head or neck region [32, 33]. An older case series found no clinical benefit for deafferentation pain [34]. Although there are few recent case series, complication rates have improved with stereotactic guidance, but the few reported complications include dysphagia, dysarthria, upward gaze paralysis, ocular convergence defects, skew deviation, miotic pupils, weakness/paresis, painful dysesthesias, and altered mental status [32–35].

Hypophysectomy

Hypophysectomy is one of the less common examples of a procedure that has fallen out of favor, with the advent of neuromodulation techniques, despite historical reports of positive outcomes. Previous studies indicate pain relief in 70–75% of patients, with initial postop results reaching as high as 87% [30, 36, 37]. Focal radiation doses of 150–160 Gy are most commonly used now, targeting the superior portion at the junction of the stalk and neurohypophysis [37]. Evidence is limited to low-quality studies at the moment, but the available case series report excellent consistency in pain relief [37]. Complications are reduced as well with the latest treatment paradigms, with only 1 of 16 patients who received 150–160 Gy experiencing an adverse endocrinological event [37]. Notably, there are two clinical trials

underway to methodically address the efficacy of hypophysectomy, whose results the authors greatly look forward to (NCT03377517, NCT02637479).

Deep Brain Stimulation/Motor Cortex Stimulation

Overview

Before its modern-day application in movement disorders, Deep Brain Stimulation (DBS) was first attempted in the treatment of pain. Observations of "stimulus-produced analgesia" in rodent models led to the early studies on the analgesic effects of periaqueductal gray (PAG) and periventricular gray (PVG) stimulation in humans [38–40]. Despite these early reports, the field did not advance substantially in the subsequent decades due to poorly designed studies with either a small sample size or an overall lack of a methodical experimental approach when testing the location of stimulation or the indication for treatment. Similarly, motor cortex stimulation (MCS) generated excitement at first, especially in the treatment of trigeminal neuralgia-related pain, but studies over the years have found its long-term results underwhelming. Neurosurgery, however, has not given up on analgesic DBS just yet. Current applications being explored include stimulation of the hypothalamus for cluster headaches and of multiple regions for poststroke pain.

Procedure

DBS for pain treatment remains off-label due to the lack of prospective data, but it has been shown to be most beneficial for medically intractable, nociceptive pain patients, as well as phantom limb pain, complex regional pain syndrome type 1, poststroke pain, and cluster headache [41, 42]. After the surgeon has deemed the patient an ideal candidate for surgery, the patient is placed in a stereotactic frame and sent to obtain a stereotactic MRI or CT imaging. The stereotactic coordinates guide the placement of the electrode intraoperatively. In the operating room, the patient is kept awake to enable real-time feedback and a small parasagittal frontal burr hole allows access for the electrode to the target. Microelectrode recordings, micro-stimulation, and macro-stimulation enable the surgeon to functionally define the target area before final electrode placement. Postoperatively, the surgeon obtains MRI or CT to confirm placement and to determine whether any intracranial complications occurred. The electrodes are externalized for a 1-week trial period, where combinations of stimulation inputs are tested, before being connected to an implanted pulse generator [42, 43].

MCS follows a similar procedure, with some differences that we will note. Indications for MCS are medically-unresponsive central and neuropathic pain syndromes, including trigeminal nerve injury of the root, ganglion, or peripheral branches, postherpetic facial neuralgia, central pain after thalamic or lateral

medullary stroke, phantom limb pain, brachial plexus avulsion, and neuropathic spinal cord injury pain [44]. A functional MRI (fMRI) is more commonly utilized to initially identify the motor cortex and image-guided neuronavigation is subsequently used intraoperatively. A linear skin incision is first made along the motor cortex, about 10 cm long, before making a circular craniotomy of 5 cm in diameter. Once exposed, electrophysiologic monitoring is used to functionally define the target of stimulation. Electrode strips are then placed epidurally based on the homuncular location of the pain. The electrodes are externalized for a 1-week trial period, and if sufficient pain relief is achieved, the electrode is connected to an implanted pulse generator that is placed subcutaneously [42].

Outcomes

Consensus on the benefit of DBS for the treatment of pain is far from certain due to the heterogeneity of studies. A meta-analysis in 2003 found that 50% (561/1114) of patients had long-term pain relief with DBS, ranging from 19% to 79% in the included studies [45]. When subdivided based on the type of pain, nociceptive pain patients were found to only express successful analgesia when stimulated in the PAG/PVG (59%), whereas none of the patients stimulated in the VPL/VPM had long-term success. Conversely for neuropathic pain patients, stimulation in the VPL/VPM resulted in long-term success for 56% (228/409) of patients, while PAG/PVG stimulation yielded 23% (35/155). A similar meta-analysis in 2005 found that overall DBS had greater efficacy for nociceptive pain than neuropathic pain [46]. They also found that 79% of patients expressed long-term alleviation of pain when stimulated in the PAG/PVG, with an increase to 87% when the VPL/VPM or internal capsule was also stimulated. Certain conditions were found to be most responsive to DBS, including low back pain, phantom limb pain, and neuropathies.

Long-term success in the first paper was limited to >50% reduction in pain or continued use of the stimulator at 1 year, whereas in the second paper the authors used the definition established by each primary study [45, 46]. With this broad definition of success and that most of the studies analyzed were from the 1970s and 80s, it is important to note the overall lack of updated quality data supporting specific indications for DBS in pain. In a more recent series of studies, Boccard et al. reported that 66% (39/59) of implanted, neuropathic pain patients expressed clinical benefit when stimulation was in the PAG or ventral posterior nuclei of the thalamus (VPL/VPM) and 83.3% (10/12) when in the anterior cingulate cortex [47, 48]. In a recent randomized, double-blind crossover trial, Lempka et al. found significant improvements in multiple outcomes measures when poststroke pain patients received stimulation to the ventral striatum/anterior limb of the internal capsule [49]. Fontaine et al. found in their double-blind, randomized control trial that 54.5% (6/11) patients with refractory cluster headache experienced >50% reduction in their frequency of attacks when stimulating the posterior hypothalamus [50]. Although beneficial for some patients, future studies must methodically test the

location of stimulation and differentiate based on pain type to accurately define the indications for DBS in pain patients.

Outcomes for MCS are similarly mixed, thus it not being covered by insurance or Medicare in the US. In a 2005 prospective observational study on ten patients with trigeminal neuropathic pain, Brown and Pilitsis reported 88% immediate pain relief and 75% relief at 10 months postop [44]. Mo et al. reported in their systematic review refractory pain improvement rates of 35.2% in poststroke pain, 46.5% in trigeminal neuropathic pain, 29.8% of brachial plexus avulsion pain, 34.1% in phantom pain, and 65.1% in post-radicular pain [51]. In another systematic review, Henssen et al. found that 43.7% of studies reported pain reduction of 70–100%, while 31.1% of studies reported 0–40% pain reduction [52]. However, other studies have brought into question the sustainability of the analgesic effect, such as Sachs et al. who found only 2 of 14 patients had lasting pain improvement at 55 months postop [53]. With a mean follow-up on the order of a few months to multiple years in the aforementioned systematic reviews, it is evident that outcomes data for MCS fall prey to the same issues as DBS, with high levels of heterogeneity and an overall lack of substantive empirical guidance.

Complications

Complications are relatively infrequent but have been reported for both DBS and MCS. Besides electrode fracture, the complication that surgeons should be most wary of for DBS is intracranial hemorrhage, which can occur upon insertion or removal of the electrode [42]. Mortality is exceedingly rare in DBS, but three of the four reported cases were a consequence of intracranial hemorrhage. Infectious complications occur in 3.3–13.3% of cases. Headache is reported in upwards of half of cases, although most resolve by the time the patient is discharged from the hospital. Other potential complications reflect the focus of stimulation, including ophthalmologic disturbances such as diplopia, vertical gaze palsies, or horizontal nystagmus when stimulating the PAG/PVG. Complications of MCS are similar to those of DBS. Seizure induction has also been reported multiple times, with at least one case of severe epilepsy after long-term MCS treatment [54].

Spinal Cord Stimulation

Overview

Spinal cord stimulation (SCS) for pain control was first attempted by Dr. Norman Shealy and colleagues in 1967, shortly after Melzack and Wall's seminal publication on the gating theory of pain [55]. Based upon the new model, Shealy et al. surmised that stimulation of large A-beta fibers would inhibit, or "close the gate" of smaller pain-invoking, A-delta and C fibers [56]. Their observations proved

prescient, and although the exact mechanism has yet to be fully elucidated, SCS is indicated for multiple intractable pain conditions involving chronic neuropathic pain of spinal origin, as well as vascular insufficiency pain and anginal pain [57]. Other common indications include failed back surgery syndrome (FBSS), complex regional pain syndrome (CRPS), and peripheral neuropathy. Compared to DBS, the evidence supporting SCS has been more consistent over the decades. Yet, it still falls prey to the same lack of high-quality evidence, and recent studies continue to refine the level of specificity between the loci or pattern of stimulation and clinical indication.

Conventional treatment applies 40–60 Hz stimulation to induce paresthesia over the affected area. SCS induces an inhibitory effect via release of neurotransmitters such as GABA, Substance P, and Serotonin [57]. The primary hypotheses of SCS's mechanism are through stimulation of the dorsal column fibers, the most proximal aspect of the spinal cord to the electrodes, and/or the dorsal root fibers, with their lower stimulation threshold for recruitment [58]. Each hypothesis has its merits, as well as its incongruencies with clinical observations. Midline stimulation of the dorsal columns should theoretically first elicit paresthesia in the legs via the gracile fasciculus, followed by the arms via the more lateral cuneate fasciculus. However, midline cervical stimulation first generates paresthesia in the arms, before involving the legs at higher stimulation amplitudes. In rodent models, severance of the dorsal columns rostral to the site of stimulation greatly reduces, but does not eliminate, SCS effectiveness, suggesting that other fibers are sufficient for its mechanism [59]. For the dorsal root hypothesis, one would expect stimulation to elicit responses from other similarly low-threshold fibers, such as Ia muscle spindle afferents, causing muscle contractions and a proprioceptive response, which are not typically seen. Also, by this hypothesis, focal stimulation should not elicit multi-dermatomal paresthesia, which it commonly does [58].

Patient Selection

The first key step to effective treatment in SCS is patient selection. SCS treatment has only proven effective in 40–50% of refractory pain patients, with imprecise adherence to patient selection guidelines being a major factor in treatment failure. Inclusion criteria for SCS take into consideration of a multi-factorial array of factors that influence outcomes and include: appendicular pain after previous spine surgery, pain of at least 6-month duration, the lack of chronic or recurring pain above the T10 dermatome, leg pain that radiates below the knee greater than back pain, and proper psychological evaluation [60]. Furthermore, surgeons must be intimately aware of the specific indications for the various treatment loci and stimulation patterns. Exclusion criteria include surgical procedure within 6 months before screening, active psychiatric disorder, age less than 18 years old, no previous attempt at non-surgical therapy for the condition, or previous failed SCS trial. SCS is also not recommended for those with cancer-related pain or those with limited life

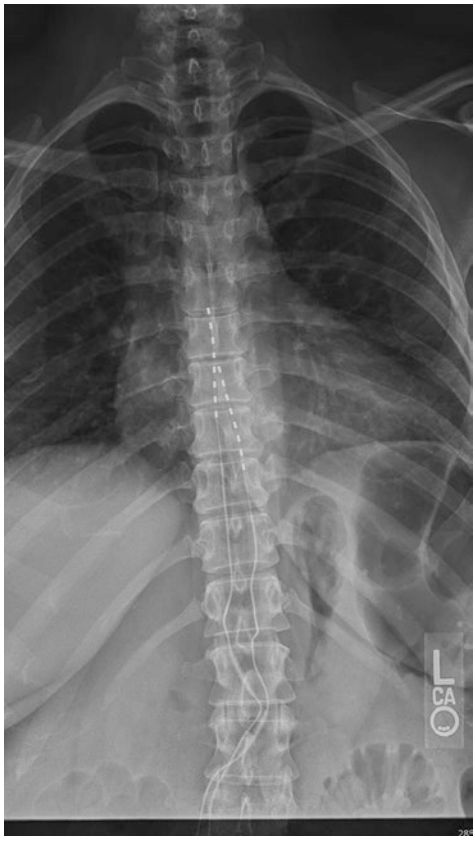


Fig. 18.1 Anterior/posterior radiograph demonstrating electrode leads of a spinal cord stimulator within the spinal canal

expectancy [60]. Figure 18.1 shows an anterior/posterior radiograph demonstrating electrode leads of a spinal cord stimulator within the spinal canal.

If the above criteria are met, the patient may proceed to a screening trial of 1 week for treatment efficacy with an external transmitter. Electrodes are placed epidurally along the section of the spine corresponding to the target of paresthesia by one of two techniques: (1) Paddle lead via laminotomy or (2) Percutaneous electrode via Tuohy needle [57]. The former provides greater longitudinal stability, but the latter is less invasive and better for trial implants. If only unilateral stimulation is required, then a hemilaminectomy is utilized. Whereas if bilateral stimulation is required, then the electrodes are centered along the midline. Ideal candidates that proceed to permanent receiver implantation exhibit at least a 50% reduction in pain (based on pre- and post-treatment VAS scores), functional improvement (as determined by functional outcome evaluation in clinic), and induced paresthesia that is concordant with the region of pain and not undesirable (as expressed by the patient) [60].

Outcome

Duarte et al. found in their meta-analysis of randomized controlled trials (RCT) comparing SCS versus placebo or sham surgery that pain scores (scale 1–10) declined −1.15 (95% CI: −1.75 to −0.55) compared to the control groups [61]. Multiple trials reported at least a 10% greater patient satisfaction with SCS compared to sham surgery. A systematic review on SCS for axial low back pain found that 79% of patients (95% CI: 70–87%) expressed >50% reduction in pain [62]. In their RCT for FBSS, North et al. described that 47.4% (9/19) of SCS patients expressed >50% reduction in pain, whereas only 11.5% (3/26) re-operation patients had improvement [63]. Two meta-analyses of RCTs on SCS for refractory angina found that SCS reduced angina frequency and nitroglycerin consumption, as well as improving several measures of health-related quality of life [64, 65].

Complications

The complication rate in SCS is reported to be around 30–40% [57, 66], and the complications can be generally understood as hardware- or biologically-related. The most common hardware malfunctions include lead migration, lead connection failure, or lead breakage [57]. Lead migration is one of the most common complications overall (15.5%, 95% CI: 9.21–21.77%) but can be mitigated by the stimulation technique [66]. Percutaneous leads, despite their convenience, are far more likely to migrate compared to paddle leads. Biological complications include pain at the hardware site (6.15%, 95% CI: 0.97–11.33%) or infection (4.89%, 95% CI: 3.38–6.39%) [66]. Rare but serious complications to watch out for are neurologic damage due to intraoperative root or spinal cord injury, or even epidural hematoma. Overall, complications can be minimized with the proper experience and expertise.

Figure 18.2 shows a lateral radiograph demonstrating paddle leads of a spinal cord stimulator within the spinal canal.

Figure 18.3 shows an anterior/posterior radiograph directed more inferiorly in the same patient revealing the spinal cord stimulator generator and a separate intrathecal baclofen pump system.

Direct Drug Administration Into CNS

Overview

Yaksh and Rudy first demonstrated in 1976 the analgesic potential for direct injection of opioids into the subarachnoid space [67]. Since then, direct drug administration, whether into the subarachnoid, epidural, or intraventricular spaces, has remained a mainstay in the analgesic regimens of nociceptive cancer-related pain patients and nociceptive “mechanical” spine disorders. Although historically the efficacy of intrathecal drug delivery has been limited for neuropathic pain patients, the Polyanalgesic Consensus Conference (PACC) recommended in 2017 to no



Fig. 18.2 Lateral radiograph demonstrating paddle leads of a spinal cord stimulator within the spinal canal

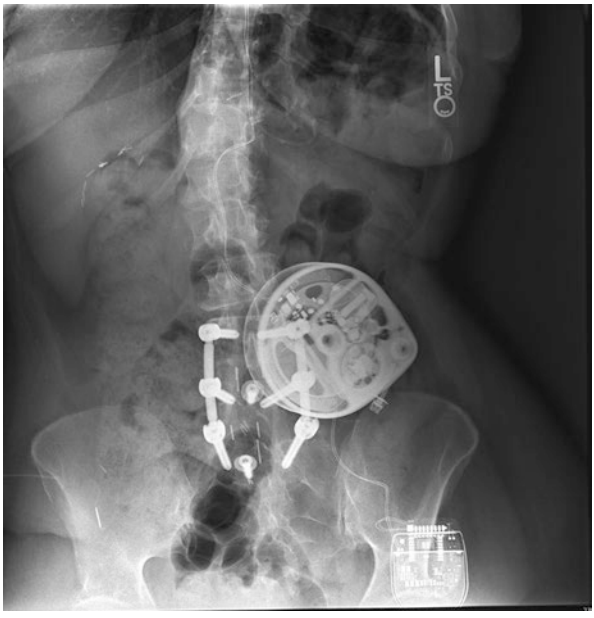


Fig. 18.3 Anterior/posterior radiograph directed more inferiorly in the same patient revealing the spinal cord stimulator generator and a separate intrathecal baclofen pump system

longer discriminate treatment plans based on this categorization of pain. These modalities are most used in the setting of fetus delivery, as a one-time dose administered to the epidural space surrounding the lumbar spine. The primary advantage

of direct administration is that it allows the drug to bypass the two main barriers to reaching the CNS from the circulatory system, the blood-brain barrier, and the liver. Not only does this increase the dose arriving in the CNS, but it also reduces the systemic interactions of the drugs that mediate their side effects.

The main indications for direct intrathecal treatment are cancer-related pain, post-laminectomy syndrome, chronic compression fractures, spinal stenosis, spondylosis, spondylolisthesis, complex regional pain syndrome, neuropathies, rheumatoid arthritis, and chronic pancreatitis, among other non-cancer-related pain conditions [68]. Contraindications include conditions of diffuse multifocal pain, headache or facial pain, ischemic heart disease, heart failure, cerebrovascular disease, or conditions with less than 3 months of life expectancy [68]. It is not necessary for the patient to have failed a trial of oral opiates or neurostimulation before consideration for intrathecal analgesia. There was a period when intracerebroventricular injection was more commonly used, but currently, it is almost solely used for terminal cancer patients with craniofacial pain [69]. Complications have been reported in 10.5% of patients, and they include catheter kinking, disconnection or displacement, pump failure, or CSF leak [70]. Surgical complications such as bleeding and infection are more rare.

Procedure

The first step is to determine the ideal analgesic and its effective dose via direct injection [18]. Trials of morphine can be done via bolus injection, continuous epidural infusion, or continuous intrathecal infusion, whereas trials of ziconotide cannot be done epidurally. It is important to always remember that "success" in the case of intrathecal therapy trials is patient-specific and focuses on achieving a state deemed tolerable by the patient, but not necessarily completely lacking of pain. Of note, oral opiates may impair the efficacy of intrathecal opiate efficacy and thus should be reduced or ceased altogether before trialing [71, 72]. Once the ideal balance of analgesia and side effects is established, the catheter is implanted percutaneously with a Tuohy needle or directly via a hemilaminectomy. The micromotor-driven drug pump is implanted in a subcutaneous pocket under general anesthesia or local anesthesia in patients with certain medical conditions and then connected to the catheter. Pump reservoirs will need to be refilled based on vessel capacity and rate of drug administration.

Medication Options

The only two FDA-approved (USA) medications for chronic pain are morphine (μ opioid receptor agonist) and ziconotide (calcium channel blocker). While both may be considered first-line for cancer-related and non-cancer-related pain, the evidence is weaker for using morphine in non-cancer-related pain [73]. Combination therapy with non-FDA-approved therapies continues to be explored, including with

baclofen, clonidine, octreotide, dextromethorphan, benzodiazepines, nitric oxide synthetase inhibitors, tricyclic antidepressants, and lidocaine [18]. Contraindications to morphine include patients whose pain is refractory to previous oral opiate prescriptions or those with substance abuse, pulmonary disease, or sleep apnea (central or obstructive) [68]. Besides the general risks associated with opioid usage such as tolerance and opioid-induced hyperalgesia, other potentially serious adverse events associated with morphine are granuloma formation and myoclonus [68]. Physicians should order imaging if the patient presents with focal neurologic deficits. Ziconotide does not come with the same risks of cardiopulmonary depression as morphine, but it is contraindicated in those with a past history of psychosis and requires careful monitoring if the patient is taking another CNS medication (i.e. antiepileptics, neuroleptics, sedatives) [68]. Physicians should also monitor serum creatine kinase (CK) levels, as ziconotide has been shown to cause elevated CK in upwards of 40% of patients, usually within the first 2 months of treatment. Ziconotide is not associated with any withdrawal symptoms upon treatment discontinuation.

The 2016 PACC guidelines recommend starting continuous dosing for opioid-naïve patients at 0.1–0.5 mg/d for morphine and 0.5–1.2 mcg/d for ziconotide [73]. The range is broader in those patients that have developed tolerance to oral opioids, ranging from 1–10 mg/d, but the physician should always consider that opioid dose and the frequency of adverse events are directly related. If the desired effect has not been produced after escalating dosage to 50% of the PACC recommended maximum limit (i.e. 20 mg/mL or 15 mg/d), then the physician should check the integrity of the pump. Despite its relative lack of side effects, ziconotide has a narrower therapeutic window and its side effects more closely correlate with the rate of administration than absolute dosage [68]. Its notorious cognitive side effects arise more frequently with high initial doses and rapid titration increments and tend to have a delayed onset, anywhere from 10 days to weeks after initial treatment [68]. Thus, dosing of ziconotide should begin low (<2.4 mcg/d) and titrate slowly (increments of <2.4 mcg/d every 2–4 days, maximum dosage of 19.2 mcg/d). Tolerability may be enhanced by a more conservative starting dose and titration schedule, as well as altering the flow rate or concentration in the pump reservoir. Two recently described paradigms found that nighttime bolus dosing or patient-controlled administration of ziconotide may further improve outcomes [74, 75].

Classic Examples of Pain Conditions Treated by Surgery

Trigeminal Neuralgia

Trigeminal Neuralgia (TN) is a debilitating condition characterized by unilateral, paroxysmal pain in the somatotopic pattern of one of the main trigeminal nerve subdivisions. The pain is classically described as shock- or lightning-like. Unlike many of the pain conditions discussed in this chapter, the diagnosis and treatment paradigms are extensively studied and well-supported. TN is often the result of an aberrant vessel, most commonly the superior cerebellar artery, compressing the

trigeminal nerve DREZ in the prepontine cistern [18]. Secondary TN can be due to multiple etiologies, including multiple sclerosis, tumor, vascular malformation, or herpes zoster infection. Therefore, an MRI is first required to rule out secondary etiology. First-line treatment is carbamazepine or oxcarbazepine, followed by second-line baclofen or lamotrigine. Although other antiepileptic drugs are currently being explored for refractory TN, surgery provides an effective option for medically refractory patients. When not secondary to neurovascular compression, TN may be effectively treated with ablation of the culprit nerve. Elderly patients were once thought to not be ideal surgical candidates for TN microvascular decompression, but studies have no found little difference in the outcomes and complication rates compared to younger cohorts [76].

The terminology used for the diagnosis of TN is highly similar, but each indicates a unique etiology. According to Dr. Kim Burchiel, TN1 is idiopathic TN that presents as the classic electric shock-like episodes of pain, whereas TN2 is idiopathic TN of a more burning or aching type of pain for greater than 50% of the time [77, 78]. She and co-authors have surmised in the past that TN1 progresses to TN2 if left untreated, signifying neural injury, and TN2 is more likely to be due to an intracranial mass, thus requiring imaging for diagnosis [77, 78]. Trigeminal neuropathic pain is usually secondary to trauma (e.g. skull base, oral or ENT surgery, stroke, etc) and is described as constant burning or throbbing pain. Trigeminal deafferentation pain involves patients subject to intentional injury to their trigeminal system (e.g. neurectomy, gangliolysis, rhizotomy, nucleotomy, tractotomy, etc) and is described as crawling or itching pain. Symptomatic TN refers to someone with multiple sclerosis. About 1% of TN patients have MS and around 1/3 of MS patients have TN, so any patient presenting with TN and a sensorimotor deficit should be evaluated for MS [77, 78]. Postherpetic TN follows a breakout of facial herpes zoster, usually in the V1 nerve pattern. Patients describe the development of allodynia with a burning dysesthesia. Lastly, atypical facial pain should only be used to describe patients with facial pain in the context of a somatoform pain disorder. The pain is classically described as bilateral and spreading outside of the trigeminal somatotopy and is usually concomitant with other somatic issues. Diagnoses to consider include fibromyalgia and chronic fatigue syndrome, among others.

Anesthesia dolorosa (AD) is a particularly severe consequence of surgery for TN and results from deafferentation injury to the first order trigeminal nerve, releasing inhibitory signaling on the second order nociceptive trigeminal neurons [79]. It is characterized by the co-occurrence of numbness and severe levels of pain. Due in part to its rarity, there is little empirical basis for treatment. First-line therapy includes medications such as gabapentin, which often fails [80]. Surgical treatment may then be pursued, with at least one report of lasting positive outcomes after DREZ of the nucleus caudalis [79].

Microvascular Decompression

Microvascular decompression (MVD) is considered the gold standard treatment option for TN due to neurovascular compression. Indications for MVD include drug-resistant idiopathic TN (or drug-responsive when the side effects do not

subside), MRI-confirmed neurovascular etiology, and sufficient condition for surgery [81]. Contraindications are atypical or secondary TN, MRI-confirmed etiology that is not neurovascular compression, or poor surgical candidate. Old age was once considered a contraindication, but new evidence has called that into question [76, 82]. MVD is less effective for patients where the underlying etiology is not due to neurovascular compression (e.g. in the setting of multiple sclerosis) or where the nerve damage may be more extensive (e.g. TN2). The procedure is a retrosigmoid approach. Upon reaching the lateral brainstem, the surgeon identifies the trigeminal nerve and traces along the DREZ for the culprit vessel. The vessel is then dissected and mobilized from the nerve, with Telfa or Teflon™ pledgets placed between the nerve and vessel to prevent recurrence.

Studies have found the MVD reduces pain immediately postop in 80–95% of patients [83]. One recent meta-analysis found that 76.0% of patients (n = 3897) reported a lack of pain with an average follow-up of 1.7 years (SD: 1.3) [84]. The authors analyzed predictors of favorable outcomes via random effects modeling and found that disease duration <5 years, arterial compression (rather than venous), involvement of the superior cerebellar artery, and Burchiel type 1 TN all predicted favorable outcomes with MVD. One long-term study found that at 15 years postop, 73.4% of patients remained pain-free [85]. MVD is the most invasive surgical option for TN, and thus carries the greatest risk of morbidity. Nevertheless, serious complications are rare. The most commonly reported adverse events include hyponatremia (5.6%), CSF fistula (4.2%), and hearing loss (0.9–1.9%), depending on the study analyzed [84]. Other common complaints include headaches (3.6%), facial dysesthesia(2.7–5.7%), or numbness in the trigeminal distribution (0.9–13.9%) [84]. The risk of mortality is exceedingly low, at less than 1% [83].

Percutaneous Treatments

Percutaneous procedures for TN are considered if no neurovascular compression is observed on imaging or if the patient is a less than ideal surgical candidate. They consist of three techniques: glycerol rhizotomy, balloon decompression, and thermocoagulation. All are performed under general anesthesia with a needle advanced through the medial foramen ovale advancing towards the clivus [86]. The anesthesia is then reversed, and the patient is brought into a seated position. For glycerol rhizolysis, the contrast is slowly replaced with glycerol (0.36 mL—the volume of Meckel's cave), before withdrawing the needle and maintaining the patient's position for at least 2 h. In thermocoagulation, the needle tip contains an electrode that is aimed slightly more laterally towards the trigeminal nerve ganglion. The patient is awakened to confirm location via physiologic stimulation testing before resedating and ablating the nerve by delivering 65–70 °C for 60 s. The balloon technique follows the same process, followed by contrast-mediated test inflation to ensure correct position. Ideal compression aims to achieve a pear shape, usually entailing 0.35–0.5 mL of contrast. Meta-analytic review demonstrates that, compared to glycerol rhizotomy, thermocoagulation results in greater immediate pain relief (OR: 2.65, 95% CI: 1.29–5.44), and balloon decompression has a lower risk of mastication weakness (OR: 9.29, 95% CI: 2.71–31.86) and diplopia (OR: 6.31,

95% CI: 1.70–23.33) [87]. One recent study found that percutaneous balloon decompression and thermocoagulation were more effective than glycerol rhizotomy in multiple sclerosis-related TN patients [88].

Stereotactic Radiosurgery

Stereotactic radiosurgery (SRS) is an advantageous option for TN patients in certain circumstances. Multiple large studies have shown that SRS is inferior to microvascular decompression in both its propensity for pain relief and duration of effects [89, 90]. However, it may be an ideal option for patients who are poor surgical candidates and those that do not demonstrate neurovascular compression on imaging. In one recent large study, SRS resulted in immediate pain relief for 75% of patients, compared to 96% in microvascular decompression cases [89]. The main superiority of SRS is that it resulted in zero complications, compared to 3.9% CSF leak and 3.3% pseudomeningocele in surgical patients [89]. Similarly, one meta-analysis found that failed SRS TN patients had better outcomes with microvascular decompression rather than repeat SRS [90, 91]. Meta-analysis of multiple sclerosis-related TN found that 83% (95% CI: 74–90%) of patients had initial pain relief but only 47% (95% CI: 33–60%) had relief at the end of follow-up [92].

Occipital Neuralgia

Occipital neuralgia (ON) is a chronic pain condition characterized by unilateral or bilateral, paroxysmal or shooting pain that radiates from the base of the skull along the projections of the greater, lesser, or third occipital nerves. Its trigger point is the superior nuchal line, and it may be accompanied by dysesthesia. Besides the characteristic presentation, diagnosis may also test whether occipital nerve block temporarily resolves the pain. The etiology of ON is complex and often involves trauma or nerve entrapment. Treatment begins with first-line pharmacologic therapies, such as antiepileptics or anti-depressants, and refractory cases may pursue nerve block, steroids, or botulin toxin [93]. Surgical interventions are only considered for cases that remain refractory to non-invasive treatments. However, the low overall incidence of ON, and particularly refractory ON, is a severe hindrance to developing robust empirical support for surgical treatment. Numerous techniques have been attempted, including ganglionectomy, occipital nerve decompression, neurolysis, radiofrequency ablation, without lasting success [94]. A recent systematic review carried out by the Congress of Neurological Surgeons found that occipital nerve stimulation was a viable and potentially efficacious treatment modality in these patients [93]. Figure 18.4 shows an occipital stimulator on a lateral x-ray view.

Occipital Nerve Decompression

There are several potential points of compression along the path of the occipital nerve. Thus, surgical decompression of the nerve, most often the greater occipital



Fig. 18.4 Occipital stimulator on a lateral x-ray view

nerve, is a common treatment option for refractory ON. The five most frequent sites of compression include (1) C2 root compression in the cervical spine, (2) within the semispinalis capitis, (3) within the inferior obliques, (4) within the trapezial tunnel, and (5) any intersection with arteries, veins, or lymphatics along its course [95]. Multiple techniques can be utilized, but overall, there does not exist enough evidence to definitively support one approach. A recent prospective study found that 9 of 11 patients expressed complete or significant reduction in pain with 12.45-month follow-up (SD 1.29) [96]. The only complication reported was surgical site paresthesia, but all cases were temporary. Other options, especially for decompression-refractory patients, include occipital nerve excision, cervical dorsal rhizotomy, or C2 dorsal root ganglionectomy.

Occipital Nerve Stimulation

As with other applications of neuromodulation, occipital nerve stimulation demonstrates significant promise but is still lacking robust empirical support beyond retrospective studies and case series [95]. Nevertheless, these data were sufficient for the Congress of Neurological Surgeons to publish a systematic review that found

occipital nerve stimulation to be an effective treatment option for refractory ON patients [93]. Side effects were infrequent and involve mostly infection or lead migration, as well as one report of worsening cervical pain. Occipital nerve stimulation has been more thoroughly studied in cluster headaches and other chronic headache disorders, with excellent results [95]. Occipital nerve stimulation is advantageous among surgical techniques for ON patients due to its minimal invasiveness, reversibility, and titratability. The procedure is approached via an open cervical incision, and a Tuohy needle is inserted through the incisions, projecting towards the mastoid process [97]. The stylet is removed, and the electrode is then proceeded through the Tuohy needle. Once in place, the Tuohy needle is removed, and the electrode is secured to the fascia and connected to the impulse generator. Similar to other neuromodulatory procedures, complications to be wary of include infections, lead migration, and hardware malfunction [97].

Conclusions

Pain remains a complex clinical burden whose neurobiological basis has yet to be fully elucidated. Surgical intervention provides an effective treatment option for many medically refractory patients. Ablative procedures were the first to be tested and remain especially beneficial for patients with focal peripheral nerve injuries. Although more recent, neuromodulation has already exhibited great potential to alleviate pain, without the irreversibility of ablative procedures. Direct intrathecal administration of analgesics can significantly improve the quality of life for indicated patients, such as those suffering from chronic cancer pain, and allows for finer control of drugs that often have undesirable side effects. Ongoing research will only further enhance the capacity of neurosurgeons to treat pain by improving the efficacy of current paradigms, expanding the indications for the described procedures, and continuing to innovate technologically.

References

1. Spiller WG, Martin E. The treatment of persistent pain of organic origin in the lower part of the body by division of the anterolateral column of the spinal cord. J Am Med Assoc. 1912;LVIII(20):1489–90.
2. Raslan AM, McCartney S, Burchiel KJ. Management of chronic severe pain: spinal neuromodulatory and neuroablative approaches. Acta Neurochir Suppl. 2007;97(Pt 1):33–41.
3. Raslan AM, McCartney S, Burchiel KJ. Management of chronic severe pain: cerebral neuromodulatory and neuroablative approaches. Acta Neurochir Suppl. 2007;97(Pt 2):17–26.
4. Konrad P. Dorsal root entry zone lesion, midline myelotomy and anterolateral cordotomy. Neurosurg Clin N Am. 2014;25(4):699–722.
5. Javed S, Viswanathan A, Abdi S. Cordotomy for intractable cancer pain: a narrative review. Pain Physician. 2020;23(3):283–92.
6. Gildenberg PL, et al. Impedance measuring device for detection of penetration of the spinal cord in anterior percutaneous cervical cordotomy. Technical note. J Neurosurg. 1969;30(1):87–92.

7. Raslan AM. Percutaneous computed tomography-guided radiofrequency ablation of upper spinal cord pain pathways for cancer-related pain. Neurosurgery. 2008;62(3 Suppl 1):226–33; discussion 233–4.
8. Falci S, et al. Dorsal root entry zone microcoagulation for spinal cord injury-related central pain: operative intramedullary electrophysiological guidance and clinical outcome. J Neurosurg. 2002;97(2 Suppl):193–200.
9. Haninec P, et al. Usefulness of screening tools in the evaluation of long-term effectiveness of DREZ lesioning in the treatment of neuropathic pain after brachial plexus injury. BMC Neurol. 2014;14:225.
10. Friedman AH, Bullitt E. Dorsal root entry zone lesions in the treatment of pain following brachial plexus avulsion, spinal cord injury and herpes zoster. Appl Neurophysiol. 1988;51(2–5):164–9.
11. Kanpolat Y, et al. Spinal and nucleus caudalis dorsal root entry zone operations for chronic pain. Neurosurgery. 2008;62(3 Suppl 1):235–42; discussion 242–4.
12. Chivukula S, et al. Spinal and nucleus caudalis dorsal root entry zone lesioning for chronic pain: efficacy and outcomes. World Neurosurg. 2015;84(2):494–504.
13. Vedantam A, et al. Limited midline myelotomy for intractable visceral pain: surgical techniques and outcomes. Neurosurgery. 2018;83(4):783–9.
14. Hong D, Andren-Sandberg A. Punctate midline myelotomy: a minimally invasive procedure for the treatment of pain in inextirpable abdominal and pelvic cancer. J Pain Symptom Manag. 2007;33(1):99–109.
15. Lipinski LJ, Spinner RJ. Neurolysis, neurectomy, and nerve repair/reconstruction for chronic pain. Neurosurg Clin N Am. 2014;25(4):777–87.
16. Lu VM, et al. Meralgia paresthetica treated by injection, decompression, and neurectomy: a systematic review and meta-analysis of pain and operative outcomes. J Neurosurg. 2021;135:912–22.
17. Lu VM, et al. Treating Morton's neuroma by injection, neurolysis, or neurectomy: a systematic review and meta-analysis of pain and satisfaction outcomes. Acta Neurochir (Wien). 2021;163(2):531–43.
18. Burchiel KJ, Raslan AM. Contemporary concepts of pain surgery. J Neurosurg. 2019;130(4):1039–49.
19. North RB, et al. Dorsal root ganglionectomy for failed back surgery syndrome: a 5-year follow-up study. J Neurosurg. 1991;74(2):236–42.
20. Taub A, Robinson F, Taub E. Dorsal root ganglionectomy for intractable monoradicular sciatica. A series of 61 patients. Stereotact Funct Neurosurg. 1995;65(1–4):106–10.
21. Weigel R, et al. Selective thoracic ganglionectomy for the treatment of segmental neuropathic pain. Eur J Pain. 2012;16(10):1398–402.
22. Khan FR, Henderson JM. Does ganglionectomy still have a role in the era of neuromodulation? World Neurosurg. 2012;77(2):280–2.
23. Acar F, et al. Pain relief after cervical ganglionectomy (C2 and C3) for the treatment of medically intractable occipital neuralgia. Stereotact Funct Neurosurg. 2008;86(2):106–12.
24. Pisapia JM, et al. Salvage C2 ganglionectomy after C2 nerve root decompression provides similar pain relief as a single surgical procedure for intractable occipital neuralgia. World Neurosurg. 2012;77(2):362–9.
25. Banks GP, et al. Neuroanatomical characteristics associated with response to dorsal anterior cingulotomy for obsessive-compulsive disorder. JAMA Psychiat. 2015;72(2):127–35.
26. Viswanathan A, et al. Cingulotomy for medically refractory cancer pain. Neurosurg Focus. 2013;35(3):E1.
27. Sharim J, Pouratian N. Anterior cingulotomy for the treatment of chronic intractable pain: a systematic review. Pain Physician. 2016;19(8):537–50.
28. Gallay MN, Moser D, Jeanmonod D. MR-guided focused ultrasound central lateral thalamotomy for trigeminal neuralgia. Single center experience. Front Neurol. 2020;11:271.

29. Jeanmonod D, et al. Transcranial magnetic resonance imaging-guided focused ultrasound: noninvasive central lateral thalamotomy for chronic neuropathic pain. Neurosurg Focus. 2012;32(1):E1.
30. Roberts DG, Pouratian N. Stereotactic radiosurgery for the treatment of chronic intractable pain: a systematic review. Oper Neurosurg (Hagerstown). 2017;13(5):543–51.
31. Urgosik D, Liscak R. Medial Gamma Knife thalamotomy for intractable pain. J Neurosurg. 2018;129(Suppl1):72–6.
32. Kim DR, Lee SW, Son BC. Stereotactic mesencephalotomy for cancer – related facial pain. J Korean Neurosurg Soc. 2014;56(1):71–4.
33. Ivanishvili Z, et al. Stereotactic mesencephalotomy for palliative care pain control: a case report, literature review and plea to rediscover this operation. Br J Neurosurg. 2016;30(4):444–7.
34. Bosch DA. Stereotactic rostral mesencephalotomy in cancer pain and deafferentation pain. A series of 40 cases with follow-up results. J Neurosurg. 1991;75(5):747–51.
35. Amano K, et al. Stereotactic mesencephalotomy for pain relief. A plea for stereotactic surgery. Stereotact Funct Neurosurg. 1992;59(1–4):25–32.
36. Ramirez LF, Levin AB. Pain relief after hypophysectomy. Neurosurgery. 1984;14(4):499–504.
37. Larkin MB, et al. Stereotactic radiosurgery hypophysectomy for palliative treatment of refractory cancer pain: a historical review and update. Front Oncol. 2020;10:572557.
38. Richardson DE, Akil H. Pain reduction by electrical brain stimulation in man. Part 1: Acute administration in periaqueductal and periventricular sites. J Neurosurg. 1977;47(2):178–83.
39. Richardson DE, Akil H. Pain reduction by electrical brain stimulation in man. Part 2: Chronic self-administration in the periventricular gray matter. J Neurosurg. 1977;47(2):184–94.
40. Hosobuchi Y, Adams JE, Linchitz R. Pain relief by electrical stimulation of the central gray matter in humans and its reversal by naloxone. Science. 1977;197(4299):183–6.
41. Parmar VK, et al. Supraspinal stimulation for treatment of refractory pain. Clin Neurol Neurosurg. 2014;123:155–63.
42. Levy R, Deer TR, Henderson J. Intracranial neurostimulation for pain control: a review. Pain Physician. 2010;13(2):157–65.
43. Falowski SM. Deep brain stimulation for chronic pain. Curr Pain Headache Rep. 2015;19(7):27.
44. Brown JA, Pilitsis JG. Motor cortex stimulation for central and neuropathic facial pain: a prospective study of 10 patients and observations of enhanced sensory and motor function during stimulation. Neurosurgery. 2005;56(2):290–7; discussion 290–7.
45. Levy RM. Deep brain stimulation for the treatment of intractable pain. Neurosurg Clin N Am. 2003;14(3):389–99, vi.
46. Bittar RG, et al. Deep brain stimulation for pain relief: a meta-analysis. J Clin Neurosci. 2005;12(5):515–9.
47. Boccard SG, et al. Long-term outcomes of deep brain stimulation for neuropathic pain. Neurosurgery. 2013;72(2):221–30; discussion 231.
48. Boccard SGJ, et al. Long-term results of deep brain stimulation of the anterior cingulate cortex for neuropathic pain. World Neurosurg. 2017;106:625–37.
49. Lempka SF, et al. Randomized clinical trial of deep brain stimulation for poststroke pain. Ann Neurol. 2017;81(5):653–63.
50. Fontaine D, et al. Safety and efficacy of deep brain stimulation in refractory cluster headache: a randomized placebo-controlled double-blind trial followed by a 1-year open extension. J Headache Pain. 2010;11(1):23–31.
51. Mo JJ, et al. Motor cortex stimulation: a systematic literature-based analysis of effectiveness and case series experience. BMC Neurol. 2019;19(1):48.
52. Henssen D, et al. Motor cortex stimulation in chronic neuropathic orofacial pain syndromes: a systematic review and meta-analysis. Sci Rep. 2020;10(1):7195.
53. Sachs AJ, et al. Lack of efficacy of motor cortex stimulation for the treatment of neuropathic pain in 14 patients. Neuromodulation. 2014;17(4):303–10; discussion 310–11.
54. Henderson JM, Heit G, Fisher RS. Recurrent seizures related to motor cortex stimulator programming. Neuromodulation. 2010;13(1):37–43.
55. Melzack R, Wall PD. Pain mechanisms: a new theory. Science. 1965;150(3699):971–9.

56. Shealy CN, Mortimer JT, Hagfors NR. Dorsal column electroanalgesia. J Neurosurg. 1970;32(5):560–4.
57. Jeon YH. Spinal cord stimulation in pain management: a review. Korean J Pain. 2012;25(3):143–50.
58. Jensen MP, Brownstone RM. Mechanisms of spinal cord stimulation for the treatment of pain: still in the dark after 50 years. Eur J Pain. 2019;23(4):652–9.
59. Barchini J, et al. Spinal segmental and supraspinal mechanisms underlying the pain-relieving effects of spinal cord stimulation: an experimental study in a rat model of neuropathy. Neuroscience. 2012;215:196–208.
60. Oakley JC. Spinal cord stimulation: patient selection, technique, and outcomes. Neurosurg Clin N Am. 2003;14(3):365–80, vi.
61. Duarte RV, et al. Systematic review and meta-analysis of placebo/sham controlled randomised trials of spinal cord stimulation for neuropathic pain. Pain. 2020;161(1):24–35.
62. Conger A, et al. The effectiveness of spinal cord stimulation for the treatment of axial low back pain: a systematic review with narrative synthesis. Pain Med. 2020;21(11):2699–712.
63. North RB, et al. Spinal cord stimulation versus repeated lumbosacral spine surgery for chronic pain: a randomized, controlled trial. Neurosurgery. 2005;56(1):98–106; discussion 106–7.
64. Wang S, et al. Spinal cord stimulation versus other therapies in patients with refractory angina: a meta-analysis. Transl Perioper Pain Med. 2017;2(1):31–41.
65. Pan X, et al. Spinal cord stimulation for refractory angina pectoris: a systematic review and meta-analysis. Clin J Pain. 2017;33(6):543–51.
66. Eldabe S, Buchser E, Duarte RV. Complications of spinal cord stimulation and peripheral nerve stimulation techniques: a review of the literature. Pain Med. 2016;17(2):325–36.
67. Yaksh TL, Rudy TA. Analgesia mediated by a direct spinal action of narcotics. Science. 1976;192(4246):1357–8.
68. Deer TR, et al. Intrathecal therapy for chronic pain: a review of morphine and ziconotide as firstline optionsin. Pain Med. 2019;20(4):784–98.
69. Raffa RB, Pergolizzi JV Jr. Intracerebroventricular opioids for intractable pain. Br J Clin Pharmacol. 2012;74(1):34–41.
70. Bottros MM, Christo PJ. Current perspectives on intrathecal drug delivery. J Pain Res. 2014;7:615–26.
71. Grider JS, et al. Trialing and maintenance dosing using a low-dose intrathecal opioid method for chronic nonmalignant pain: a prospective 36-month study. Neuromodulation. 2016;19(2):206–19.
72. Grider JS, Harned ME, Etscheidt MA. Patient selection and outcomes using a low-dose intrathecal opioid trialing method for chronic nonmalignant pain. Pain Physician. 2011;14(4):343–51.
73. Deer TR, et al. The polyanalgesic consensus conference (PACC): recommendations for trialing of intrathecal drug delivery infusion therapy. Neuromodulation. 2017;20(2):133–54.
74. McDowell GC 2nd, Pope JE. Intrathecal ziconotide: dosing and administration strategies in patients with refractory chronic pain. Neuromodulation. 2016;19(5):522–32.
75. Pope JE, Deer TR. Intrathecal pharmacology update: novel dosing strategy for intrathecal monotherapy ziconotide on efficacy and sustainability. Neuromodulation. 2015;18(5):414–20.
76. Ferroli P, et al. Advanced age as a contraindication to microvascular decompression for drug-resistant trigeminal neuralgia: evidence of prejudice? Neurol Sci. 2010;31(1):23–8.
77. Burchiel KJ. A new classification for facial pain. Neurosurgery. 2003;53(5):1164–6; discussion 1166–7.
78. Eller JL, Raslan AM, Burchiel KJ. Trigeminal neuralgia: definition and classification. Neurosurg Focus. 2005;18(5):E3.
79. Sandwell SE, El-Naggar AO. Nucleus caudalis dorsal root entry zone lesioning for the treatment of anesthesia dolorosa. J Neurosurg. 2013;118(3):534–8.
80. Zakrzewska JM. Medical management of trigeminal neuropathic pains. Expert Opin Pharmacother. 2010;11(8):1239–54.
81. Broggi G, et al. Surgical technique for trigeminal microvascular decompression. Acta Neurochir (Wien). 2012;154(6):1089–95.

82. Bick SK, et al. Older patients have better pain outcomes following microvascular decompression for trigeminal neuralgia. Neurosurgery. 2019;84(1):116–22.
83. Bick SKB, Eskandar EN. Surgical treatment of trigeminal neuralgia. Neurosurg Clin N Am. 2017;28(3):429–38.
84. Holste K, et al. Pain outcomes following microvascular decompression for drug-resistant trigeminal neuralgia: a systematic review and meta-analysis. Neurosurgery. 2020;86(2):182–90.
85. Sindou M, et al. Microvascular decompression for primary trigeminal neuralgia: long-term effectiveness and prognostic factors in a series of 362 consecutive patients with clear-cut neurovascular conflicts who underwent pure decompression. J Neurosurg. 2007;107(6):1144–53.
86. Noorani I, et al. Comparison of first-time microvascular decompression with percutaneous surgery for trigeminal neuralgia: long-term outcomes and prognostic factors. Acta Neurochir (Wien). 2021;163(6):1623–34.
87. Texakalidis P, et al. Comparative safety and efficacy of percutaneous approaches for the treatment of trigeminal neuralgia: a systematic review and meta-analysis. Clin Neurol Neurosurg. 2019;182:112–22.
88. Noorani I, et al. The effectiveness of percutaneous balloon compression, thermocoagulation, and glycerol rhizolysis for trigeminal neuralgia in multiple sclerosis. Neurosurgery. 2019;85(4):E684–92.
89. Wang DD, et al. Prospective comparison of long-term pain relief rates after first-time microvascular decompression and stereotactic radiosurgery for trigeminal neuralgia. J Neurosurg. 2018;128(1):68–77.
90. Raygor KP, et al. Long-term pain outcomes for recurrent idiopathic trigeminal neuralgia after stereotactic radiosurgery: a prospective comparison of first-time microvascular decompression and repeat stereotactic radiosurgery. J Neurosurg. 2018;131:1207–15.
91. Patra DP, et al. Repeat Gamma Knife radiosurgery versus microvascular decompression following failure of GKRS in trigeminal neuralgia: a systematic review and meta-analysis. J Neurosurg. 2018;131:1197–206.
92. Spina A, et al. Efficacy of Gamma Knife radiosurgery in the management of multiple sclerosis-related trigeminal neuralgia: a systematic review and meta-analysis. Neurosurg Rev. 2021;44(6):3069–77.
93. Sweet JA, et al. Occipital nerve stimulation for the treatment of patients with medically refractory occipital neuralgia: Congress of Neurological Surgeons systematic review and evidence-based guideline. Neurosurgery. 2015;77(3):332–41.
94. Salmasi V, et al. Peripheral nerve stimulation for occipital neuralgia. Pain Med. 2020;21(Suppl 1):S13–7.
95. Urits I, et al. A review of the recent findings in minimally invasive treatment options for the management of occipital neuralgia. Neurol Ther. 2020;9(2):229–41.
96. Jose A, et al. Greater occipital nerve decompression for occipital neuralgia. J Craniofac Surg. 2018;29(5):e518–21.
97. Keifer OP Jr, et al. Occipital nerve stimulation for the treatment of refractory occipital neuralgia: a case series. World Neurosurg. 2017;105:599–604.

Endoscopic Spine Surgery

19

Omar Akbik, Peter Shin, and Mazin Al Tamimi

Introduction

History

Having shown promising results in rabbit models with dissolution of the nucleus pulposus by direct injection of chymopapain, in 1964, Lyman Smith published promising results in human clinical trials pushing forward the concept of indirect spinal canal decompression via chemoneucleolysis causing decreased intra-discal pressure [1].

Separately, Kambin [2] and Hijikata [3] expanded on the concept of indirect decompression by using a cannula in a posterolateral approach to perform a non-visualized percutaneous nucleotomy.

In 1988, Kambin would later introduce the endoscope for visualized nucleotomy publishing the first intraoperative diskoscopic views of herniated nucleusos pulposus [4]. Schreiber and Suezawa [5] would go on to inject indigo carmine dye into the disk space in order to stain and identify abnormal nucleus and annular fissures.

In 1990, Kambin defined a triangular safety zone of entry into the disk utilizing the foramen. 'Kambin's triangle' is bounded anteriorly by the exiting nerve root, inferiorly by the end plate of the lower lumbar vertebra, and posteriorly by the

O. Akbik
Department of Neurological Surgery, UT Southwestern Medical Center, Dallas, TX, USA

P. Shin
Department of Neurological Surgery, University of New Mexico Hospital, Albuquerque, NM, USA

M. Al Tamimi (✉)
Department of Neurological Surgery and Spine Center, UT Southwestern Medical Center, Dallas, TX, USA
e-mail: Mazin.AlTamimi@UTSouthwestern.edu

© The Author(s), under exclusive license to Springer Nature Switzerland AG 2022

C. E. Noe (ed.), *Multidisciplinary Spine Care*,
https://doi.org/10.1007/978-3-031-04990-3_19

superior articular process of the inferior vertebra. Shifting attention away from percutaneous nucleotomies, Kambin's triangle afforded the operator a larger channel to explore the foramen with direct decompression of the exiting and traversing nerves.

Endoscopic Lumbar Spine

The two endoscopic approaches to the lumbar spine include the transforaminal and interlaminar approach. Traditional indications for endoscopic spine surgery was for a soft lumbar disk herniation that is associated with chronic lumbar radiculopathy that has failed conservative measures [6]. However, the applications are growing specifically in the treatment of spinal stenosis, central and foraminal, secondary to ligamentous hypertrophy or boney overgrowth.

Percutaneous Transforaminal Endoscopic Discectomy

Anatomy

The transforaminal approach is performed through the intervertebral foramen which is bounded by the following:

1. Superiorly – inferior vertebral notch of the pedicle above
2. Inferiorly – superior vertebral notch of the pedicle below
3. Posteriorly – facet joint comprised of the SAP and IAP of the respective vertebra
4. Anteriorly – lower posterior lateral aspect of the vertebral body and intervertebral disk
5. Medially – thecal sac
6. Laterally the psoas muscle and fascial sheath

The contents of the intervertebral foramen include the exiting nerve root including the dorsal and ventral roots enclosed by the dural sheath, lymphatics, spinal branch of segmental artery, communicating veins between internal and external venous plexus, sinuvertebral nerves, and surrounding fat [7]. The transforaminal approach is based upon working within the confines of the Kambin's triangle.

Technique

There are several variations on the transforaminal approach used to target a variety of pathologies causing extra-foraminal herniations, foraminal stenosis, lateral recess stenosis, and central spinal stenosis. The intra-discal approach popularized by Dr. Yueng [8, 9] was based upon Dr. Kambin's arthroscopic discectomy techniques [10]. The extra-discal approach described by Schubert and Hoogland [11] employs an initial removal of the ventral/lateral portion of the superior articular process

allowing access to the ventral portion of the canal. The extreme far lateral approach described by Dr. Ruetten also describes a transforaminal approach that allows access to the ventral spinal canal for decompression of broad based disks herniations [12].

Outcomes

Yeung et al. published on their 1-year outcomes in 307 cases of percutaneous transforaminal endoscopic discectomy. With a 91% response rate to questionnaire, they reported 90.7% were satisfied with their surgical outcome and would undergo the same endoscopic procedure again. The reoperation rate was 5% with an average follow up of 19 months [13]. Using pre and post-operative VAS scores, Gu et al. reported on their 2 year outcomes for percutaneous transforaminal endoscopic discectomy in 209 cases. VAS scores of leg pain dropped from an average 9 to 1 immediately after surgery and remained at went to 0 at 2 years after operation with 95.7% of patients reporting excellent or good outcomes at 2 years based on the MacNab classification [14]. When looking at the addition of chymopapain, a proteolytic enzyme intended to dissolve part of the disk, injection into the disk space, Hoogland et al. reported 85.4% excellent or good result at 2 years in the group 1 (PTED alone) vs 93.3% excellent or good results in the group 2 (PTED with chymopapain injection) using the MacNab classification. Recurrence rates were 6.9% and 1.6% at 1 year for both groups respectively [15].

Re-herniation

When looking at risk factors for recurrence, Park et al. reviewed 1900 patients who underwent PTED and reported a recurrence rate of 11% with most recurrences occurring between 2 and 30 days. Recurrence was not related to BMI, DM, HTN, smoking status, or the presence of spondylolisthesis. They found that in those that did recur, the smaller sized disk herniation tended to recur earlier [16]. Recurrence rates for PTED have been reported with a variety of definitions but when defining it as a re-appearance of a symptomatic lumbar disc herniation at the same level after at least a month of no pain symptoms, the median recurrence rat was 1.7% with a range of 0–12% [17, 18].

PTED Versus Microdiscectomy

In 2018, Zhang et al. published a meta-analysis comparing PTED versus conventional microdiscectomy [19]. Analyzing five randomized control trials [20–24] and four retrospective reviews [25–28], they concluded that PTED was superior to open microdiscectomy in length of hospital stay but had no advantage in leg pain, functional recovery, and incidence of complications. Future studies would find in

subgroup analysis of far lateral herniation, patients had significantly less improvement in ODI scores at 3, 6, and 12 months [29]. A 5 year follow up study on a comparative cohort of PTED versus open lumbar microdiscectomy would show comparable results in regard to outcome but would again show that hospital stay as well as time to return to work were significantly shorter for the PTED group [30].

Other

The transforaminal approach in the lumbar spine is one that has been traditionally used for the treatment of a herniated disk. However, as the technology improves, the application has been expanded to several pathologies. There are case reports on the use of an endoscopic transforaminal approach for the resection of a migrated disk [31], lumbar perineural cyst [32], and synovial cyst [33, 34]. One of the major advantages of the transforaminal approach is its ability to reduce destabilization by

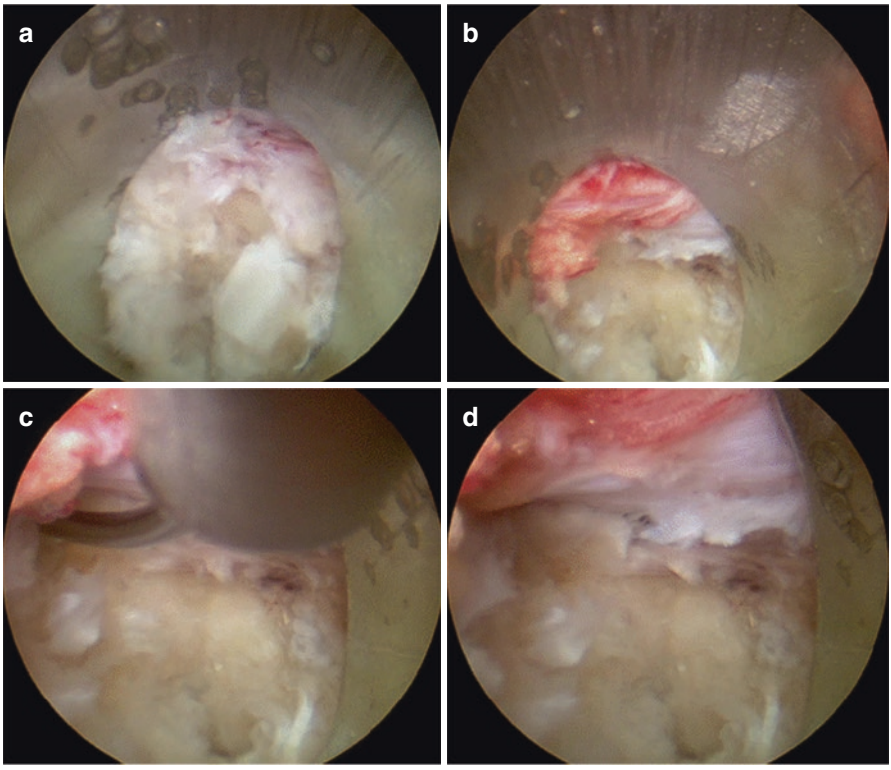


Fig. 19.1 Endoscopic transforaminal lumbar discectomy. (**a**) View of herniated disk with lateral thecal sac slightly visualized. (**b**) As discectomy is performed, epidural fat is seen to protrude down. (**c**) Pointer instrument is placed under the thecal sac to help with any adhesion the disk may have and can be placed in the lateral recess to help with orientation. (**d**) Better visualization of the lateral thecal sac and traversing nerve

reducing the amount of facetectomy typically seen in traditional discectomies. Small case series exist on the use of the endoscopic transforaminal approach to treat radiculopathy in the setting of adjacent segment disease after instrumented fusion [35] as well as in the setting of lateral lumbar spondylolisthesis secondary to adult degenerative scoliosis [36].

Figure 19.1 shows a sequence of images during a transforaminal endoscopic lumbar discectomy.

Percutaneous Endoscopic Interlaminar Discectomy

Outcomes

The interlaminar approach has been found to be a safe and effective treatment strategy for lumbar disk herniation. A 2018 meta-analysis of interlaminar approach for central or lateral recess stenosis treatment showed an average decrease of 5.95 and 4.22 in VAS leg and back scores at 12 month follow up [37]. A year later, Wasinpongwanich et al. would report on their experience with 545 patients undergoing endoscopic interlaminar discectomy. Preoperative VAS back and leg scores were 4.99 and 5.70 respectively and decreased to 2.19 and 1.19 at 4 year follow up which would echo previous studies with a larger improvement in VAS leg scores as compared to VAS back scores. Similar results were shown when using the interlaminar approach for the treatment of lumbar degenerative central spinal stenosis. At 2 year follow up, Komp et al. reported that 71% of patients had no leg pain, 22% had great reduction in leg pain, and 7% experienced essentially no improvement [38].

Re-herniation

Recurrence rates have been reported to be anywhere between 1.9% and 12.1% [39–41] with an average time to recurrence occurring at 46.3 months [41]. However with a steep learning curve, studies have shown a decrease in complication rates and even recurrent disk herniation with time. In regards to the interlaminar approach, when looking at the first 100 cases, recurrent herniation was 6% which went down to 1.3% for the next 378 cases [39].

Interlaminar Versus Microdiscectomy

Ruetten et al. performed a randomized prospective controlled study with patients being randomized to endoscopic interlaminar approach vs microdiscectomy for the treatment of lateral recess stenosis. With a 2 year follow up, they found clinical results to be the same in each group with a significantly shorter hospital duration for the endoscopic arm [22]. In regards to the treatment of lumbar disk herniation, a

5 year retrospective showed similar satisfactory results between endoscopic interlaminar approach vs traditional microdiscectomy with the former having shorter hospital stays and faster postoperative recovery [40].

Interlaminar Versus Transforaminal Approach

Numerous studies have looked at the difference in outcomes between a transforaminal approach and interlaminar approach to an endoscopic lumbar discectomy. Following the PRISMA checklist, a 2019 meta-analysis by Chen et al. compared the outcomes endoscopic lumbar discectomies via transforaminal vs interlaminar approach [42]. Twenty-six publications following 3294 patients were included in the final analysis. Short- and long-term VAS scores were significantly lower with PETD vs PEID although recurrence rates were found to be significantly higher in PETD vs PEID. While some reports indicate higher rates of dural tears in the PEID approach [43], Chen et al. reported a similar complication rate in their review. Ultimately the authors concluded that while PETD had a longer operative time, outcomes were better for the PETD approach as compared to PEID approach as reflected by lower VAS scores as well as shorter hospital stay postoperatively.

Lumbar Five – Sacral One Disc Herniation

In the world of endoscopic procedures, the L5–S1 disc space represents unique challenges. Figure 19.2a shows sagittal and Fig. 19.2b axial MRI images of a right

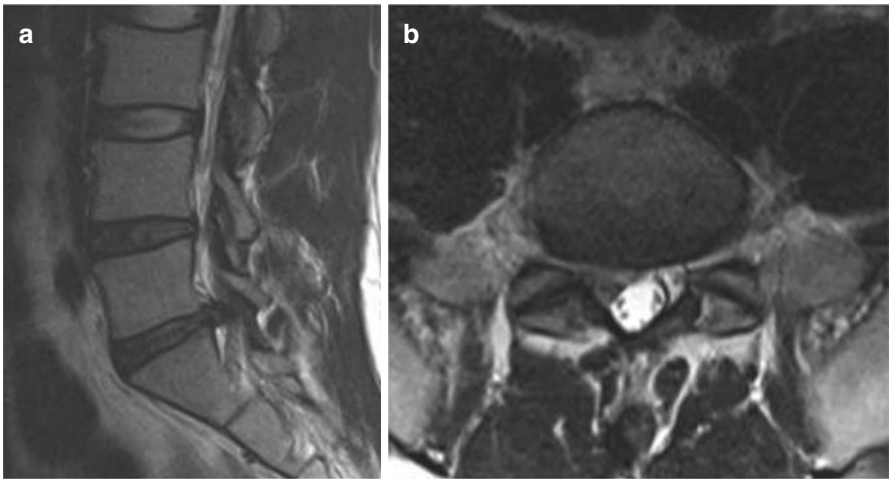


Fig. 19.2 (**a**) Preoperative MRI L spine T2 sagittal shows a disk herniation at the L5–S1 level. (**b**) MRI L spine T2 axial view shows a right lateralized disk herniation with effacement of the thecal sac and resultant foraminal stenosis

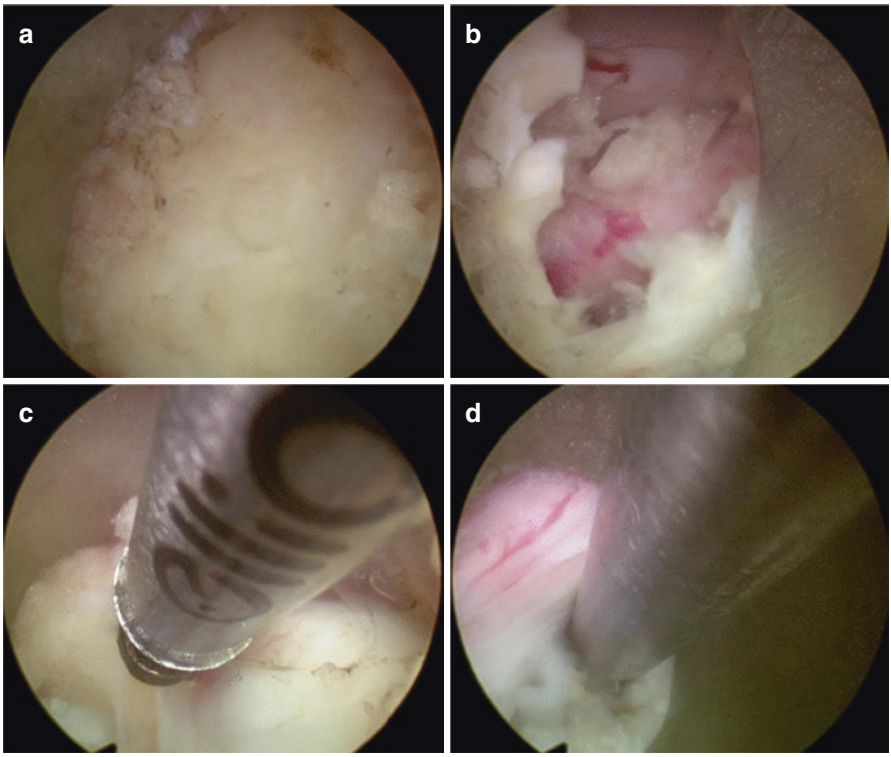


Fig. 19.3 Endoscopic interlaminar lumbar discectomy at L5–S1. (**a**) Drilling of the right inferior L5 lamina and superior S1 lamina reveals the ligamentum flavum. (**b**) Resection of the ligamentum flavum reveals epidural fat and the thecal sac. (**c**) Rotation of the endoscopic tube moves the thecal sac medially revealing a disk herniation which is coagulated. (**d**) Discectomy is performed with the forceps within the herniated disk and the thecal sac located medially

lateral disk herniation. There is typically a larger interlaminar space at this level and the large facet joint provides a narrower foramen. Access to the canal via the foramen can be affected by the location of iliac bone. Several studies have looked outcomes in this specific subset of patients when using an endoscopic interlaminar vs transforaminal approach for lumbar discectomy. Meta-analysis of nine studies looking at PETD vs PEID for the treatment of LDH at the L5–S1 level reported that PTED was found to have significantly longer operative times and greater fluoroscopy times but that the PEID approach was associated with a higher incidence of dural tears. However, in contrast to treatment at other lumbar disk herniation levels, no difference in outcome scores as measured by VAS scores was found between these two approaches at the L5–S1 level [44].

Figure 19.3 shows a sequence of images during an endoscopic interlaminar lumbar discectomy at L5 S1.

Endoscopic Transforaminal Lumbar Interbody Fusion

The transforaminal lumbar interbody fusion (TLIF) is considered to be a standard technique providing decompression of neural elements as well as fusion [45, 46]. As minimally invasive techniques became more popular with less tissue destruction and adequate fusion rates [47], the next natural outgrowth of the endoscopic transforaminal approach was the endoscopic TLIF. Most of the literature at this time consists of retrospective case series and as such, there is still significant research needed to prove its effectiveness as compared to other techniques.

Outcome

There have been a number of retrospective studies published documenting improvements in VAS neck/leg as well as ODI [48–51]. Jin et al. reported on their 2 year follow up on 39 patients undergoing ETLIF with improvements in VAS scores for back and leg as well as ODI of 89.5%, 95%, and 71.2% respectively [52]. In a 100 patients with at least 1 year follow up, Kolcun et al. showed significant sustained reduction in ODI scores from 29.6 to 17.2 [53].

The advantages of ETLIF included less blood loss, shorter hospital stays, and being able to perform a fusion under procedural sedation and local anesthetics. However, there is a significant learning curve associated with this technique. It is important to note that several steps of the ETLIF require fluoroscopic guidance increasing radiation exposure and complications of ETLIF can include cage subsidence/migration as well as non-fusion.

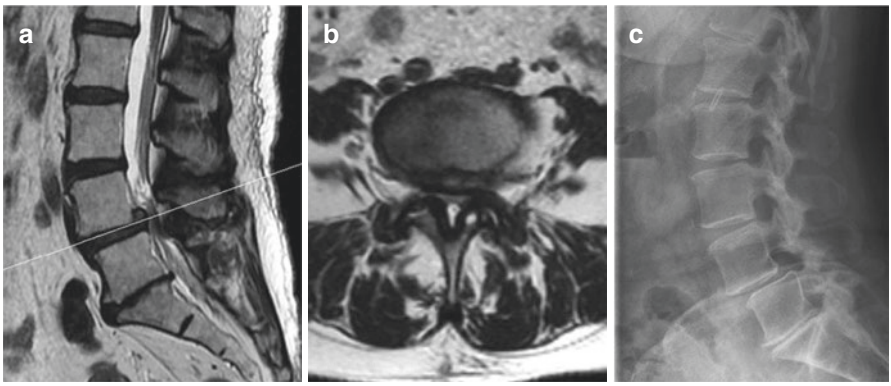


Fig. 19.4 Spondylolisthesis at Lumbar 4–5 with spinal stenosis. (**a**) MRI Lumbar spine T2 sagittal shows grade 1 spondylolisthesis with disk herniation causing spinal stenosis at the L4–5 level. (**b**) MRI Lumbar spine T2 axial shows a broad-based disk with resultant severe lumbar spinal stenosis. (**c**) Standing plain film X-Ray shows dynamic instability with increase in spondylolisthesis at that level

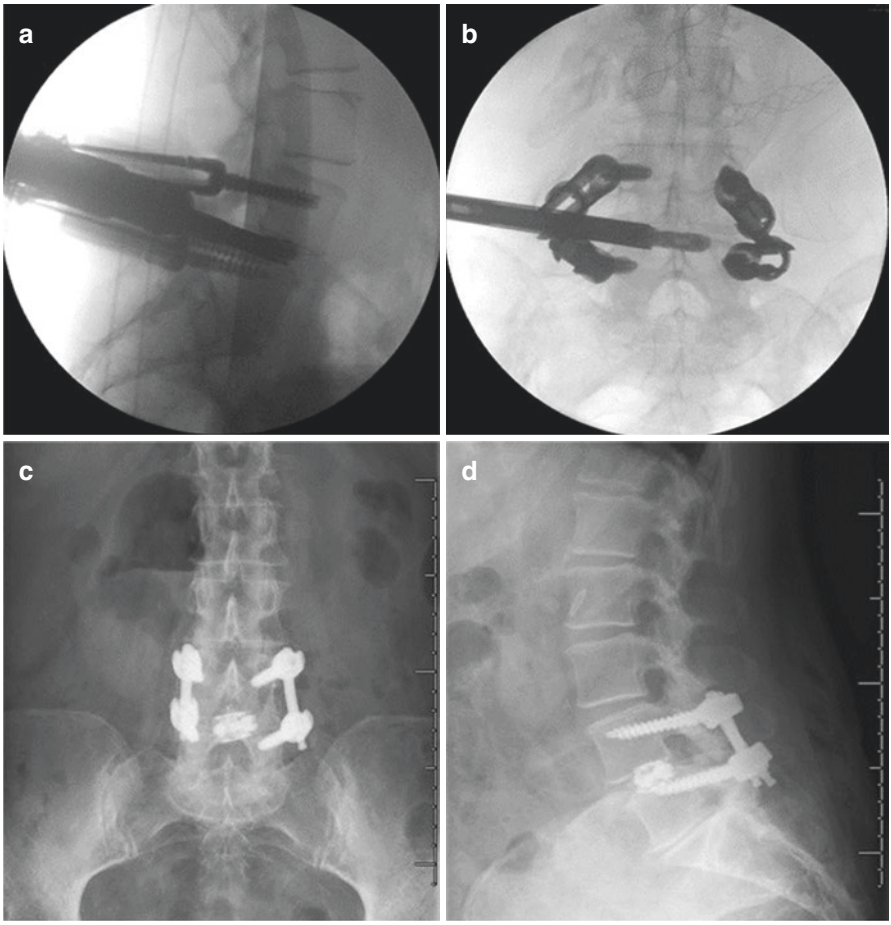


Fig. 19.5 Endoscopic transforaminal lumbar interbody fusion. (**a**) Intraoperative lateral fluoroscopy of transforaminal placed introducer at the anterior boundary of the L4–5 disk space. (**b**) Intraoperative anterior posterior fluoroscopy of transforaminal place tunneller with interbody being deployed, crossing midline. (**c**) Postoperative anterior posterior plain film displaying percutaneous placed pedicle screws and rods at L4–5 with an expanded interbody. (**d**) Postoperative lateral plain film showing instrumentation and correction of spondylolisthesis while standing

Liu et al. compared 184 patients undergoing ETLIF vs 176 patients undergoing traditional MIS-TLIF and reported lower satisfaction of 86.3% vs 92.2% respectively. While ETLIF had a lower rate of adjacent segment disease as compared to MIS TILF (0% vs 2.87%), ETLIF had a higher rate of postoperative back pain as compared to MIS TLIF [54].

Figure 19.4 shows sagittal and axial MRI images and standing lateral plain film x-ray in a patient with grade 1 spondylolisthesis with disk herniation causing spinal stenosis at the L4–5 level.

Figure 19.5 shows intraoperative fluoroscopic images of transforaminal placed introducer at the anterior boundary of the L4–5 disk space and postoperative

anterior–posterior and lateral plain films showing instrumentation and correction of spondylolisthesis while standing.

Endoscopic Posterior Cervical Foraminotomy

Indications

Indications for endoscopic posterior cervical foraminotomy include chronic radicular pain and/or neurologic deficits secondary to cervical disk herniation. This also includes other causes of cervical nerve root compression such as foraminal stenosis due to boney overgrowth, synovial cyst, or abscess. Contraindications would include neck pain without radicular symptoms, significant central stenosis with associated myelopathy, or deformity/instability [55].

Outcomes

There is a need for more studies on endoscopic posterior cervical foraminotomy in the treatment of cervical radiculopathy secondary to compression. Zheng et al. reports on one of the largest series with a report on 249 patients undergoing endoscopic posterior cervical foraminotomy and discectomy for cervical radiculopathy secondary to soft disk herniation and/or foraminal stenosis [56]. At final follow up, 86.7% (216 of 249 patients) had no or minimal arm pain i.e. excellent or good outcome according to the Macnab criteria. Thirty-three (13.3%) had occasional pain which is classified as a fair outcome by Macnab criteria.

Endoscopic Posterior Cervical Foraminotomy Versus Anterior Cervical Discectomy and Fusion (ACDF)

In one of the few prospective studies, Ruetten et al. reported on a randomized controlled trial comparing the efficacy of endoscopic cervical posterior foraminotomy vs ACDF for the treatment of lateral disk herniation [57]. Patients experience a significant reduction in radicular arm pain at 3 month, 6 month, 12 month, and 2 year follow up in both groups with no significant difference in outcomes between the two interventions. There was no significant difference in complication rates between the two groups; however, postoperative pain was significantly reduced in the EPCF arm. Postoperative work disability was also shorter in the EPCF group (19 days) versus the ACDF group (34 days). Similar return to work results seen in a cohort of 100 patients reported by Adamson et al. with an average return to work of 1.9 weeks with 60% returning back to normal activity within 1 week [58].

Figure 19.6 shows sagittal and axial MRI images of a patient with a right C5–6 disk herniation.

Figure 19.7 shows images from an endoscopic posterior cervical approach.

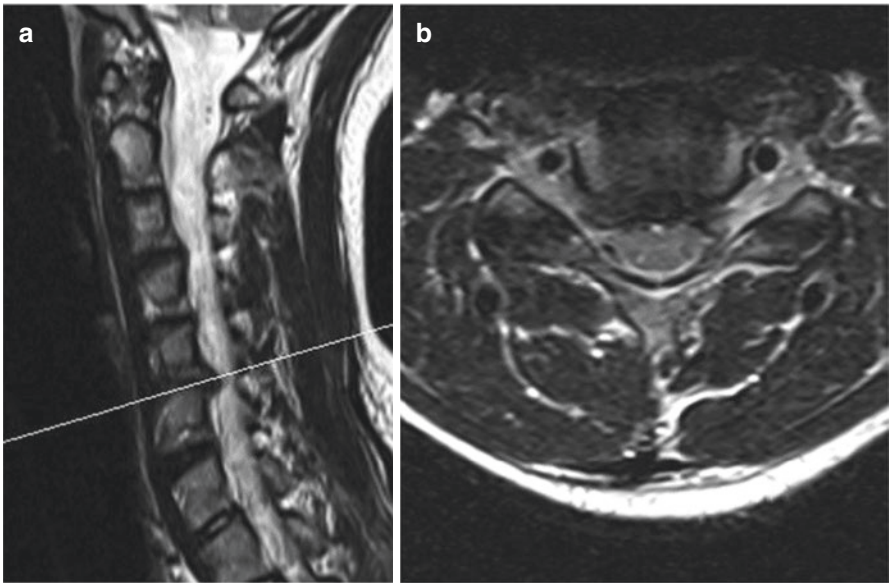


Fig. 19.6 Right lateralized Cervical 5–6 herniated disk. (**a**) Preoperative MRI cervical spine T2 sagittal shows a herniated disk at the C5–6 level lateralized to the right causing radiculopathy. (**b**) MRI cervical spine T2 axial shows right sided foraminal stenosis with nerve root compression secondary to disk herniation at the C5–6 neural foramen

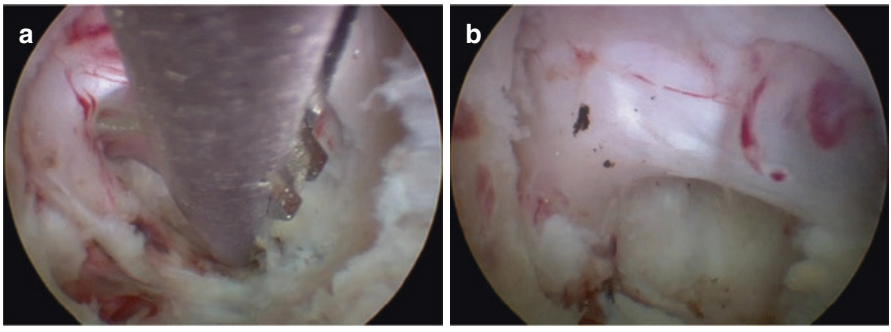


Fig. 19.7 Endoscopic posterior cervical foraminotomy. (**a**) View from an endoscopic posterior cervical approach with the drilling of the lamina and medial facet with exposure of the exiting C6 nerve root and forceps grasping the herniated disk. (**b**) Removal of the disk herniation with a relaxed exiting nerve root laterally and medially thecal sac

Endoscopic Thoracic Discectomy

The interlaminar and extraforaminal approaches to the thoracic spine are similar to previous descriptions in this chapter to the lumbar and cervical spine. However, owing to the unique anatomy of the lungs and pleural space, a third approach exists,

namely the transthoracic retropleural approach. The retropleural space is dissected with or without deflation of the lung, and the rib head is resected to expose the pedicle at the level of the disc herniation. Ruetten et al. describes their experiences with all three approaches using the interlaminar for posterior pathologies, transforaminal for lateralized disk herniations, and transthoracic for medial disk herniations [59]. With thoracic disk herniations constituting up to 4% of all spine disk herniations, no literature exists comparing endoscopic thoracic approaches to traditional approaches.

Endoscopic Treatment of Spinal Infections

Endoscopic treatment of spinal infections has a small body of evidence but is continuing to grow. The smaller incision and decreased tissue disruption provide less potential for iatrogenic seeding of the infection especially in patients who may already be immunocompromised with poor wound healing capabilities. Patients who are high risk for general anesthesia may find an alternative option with an endoscopic approach performed under local anesthetics. Choi et al. reported on their experience with various endoscopic approaches to treat a variety of lumbar spine infections including facet joint abscess, diskitis with extension to the psoas, and lumbar epidural abscess [60]. Typically, in thoracic spine infections, an open surgery may entail a transpedicular approach to reach the ventral epidural space or disk space. This approach creates a discussion on the need for an instrumented fusion in the setting of known infection. A thoracic endoscopic transforaminal approach avoiding removal of the pedicle, may be of value for those patients without spinal instability or deformity secondary to their infectious process [61, 62]. A systematic review of endoscopic treatment of spinal infections reported a reoperation rate of 21% [63].

Conclusion

Endoscopic spine surgery has been evolving for over three decades. Improvements in the endoscope and instrumentation have pushed the boundaries of endoscopic spine procedures. The endoscopic discectomy has a significant amount of literature showing efficacy and typically shorter hospital stays than traditional open procedures. The use of the endoscope and orientation of the anatomy especially for the transforaminal approach involves a steep learning curve. However, proficiency with this tool can allow for novel treatments of pathologies that otherwise involved larger open decompressions and fusions such as with thoracic disks herniation and foraminal stenosis in adult degenerative scoliosis. Endoscopic interbody fusion is an exciting area of endoscopic spine that warrants large prospective trials and long term follow up for fusion rates in order to truly evaluate its utility as compared to standard and minimally invasive transforaminal lumbar interbody fusion techniques.

References

1. Smith L. Enzyme dissolution of the nucleus pulposus in humans. JAMA. 1964;187:137–40. https://doi.org/10.1001/jama.1964.03060150061016.
2. Kambin P. Arthroscopic microdiscectomy: minimal intervention in spinal surgery. Urban & Schwarzenberg; 1991. p. 147.
3. Hijikata S, Yamagshi M, Nakayma T, Oomori K. Percutaneous discectomy: a new treatment method for lumbar disc herniation. J Toden Hosp. 1975;5:5–13.
4. Kambin P, Nixon JE, Chait A, Schaffer JL. Annular protrusion: pathophysiology and roentgenographic appearance. Spine (Phila Pa 1976). 1988;13(6):671–5.
5. Schreiber A, Suezawa Y, Leu H. Does percutaneous nucleotomy with discoscopy replace conventional discectomy? Eight years of experience and results in treatment of herniated lumbar disc. Clin Orthop Relat Res. 1989;(238):35–42.
6. Ahn Y. Endoscopic spine discectomy: indications and outcomes. Int Orthop. 2019;43(4):909–16. https://doi.org/10.1007/s00264-018-04283-w.
7. Choi G, Pophale CS, Patel B, Uniyal P. Endoscopic spine surgery. J Korean Neurosurg Soc. 2017;60(5):485–97. https://doi.org/10.3340/jkns.2017.0203.004.
8. Yeung AT. The evolution and advancement of endoscopic foraminal surgery: one surgeon's experience incorporating adjunctive techologies. SAS J. 2007;1(3):108–17. https://doi.org/10.1016/SASJ-2006-0014-RR.
9. Yeung AT. The evolution of percutaneous spinal endoscopy and discectomy: state of the art. Mt Sinai J Med. 2000;67(4):327–32.
10. Kambin P. Arthroscopic microdiscectomy of the lumbar spine. Clin Sports Med. 1993;12(1):143–50.
11. Schubert M, Hoogland T. Endoscopic transforaminal nucleotomy with foraminoplasty for lumbar disk herniation. Oper Orthop Traumatol. 2005;17(6):641–61. https://doi.org/10.1007/s00064-005-1156-9.
12. Ruetten S, Komp M, Godolias G. An extreme lateral access for the surgery of lumbar disc herniations inside the spinal canal using the full-endoscopic uniportal transforaminal approach-technique and prospective results of 463 patients. Spine (Phila Pa 1976). 2005;30(22):2570–8. https://doi.org/10.1097/01.brs.0000186327.21435.cc.
13. Yeung AT, Tsou PM. Posterolateral endoscopic excision for lumbar disc herniation: surgical technique, outcome, and complications in 307 consecutive cases. Spine (Phila Pa 1976). 2002;27(7):722–31. https://doi.org/10.1097/00007632-200204010-00009.
14. Gu YT, Cui Z, Shao HW, Ye Y, Gu AQ. Percutaneous transforaminal endoscopic surgery (PTES) for symptomatic lumbar disc herniation: a surgical technique, outcome, and complications in 209 consecutive cases. J Orthop Surg Res. 2017;12(1):25. https://doi.org/10.1186/s13018-017-0524-0.
15. Hoogland T, Schubert M, Miklitz B, Ramirez A. Transforaminal posterolateral endoscopic discectomy with or without the combination of a low-dose chymopapain: a prospective randomized study in 280 consecutive cases. Spine (Phila Pa 1976). 2006;31(24):E890–7. https://doi.org/10.1097/01.brs.0000245955.22358.3a.
16. Park CH, Park ES, Lee SH, et al. Risk factors for early recurrence after transforaminal endoscopic lumbar disc decompression. Pain Physician. 2019;22(2):E133–8.
17. Nellensteijn J, Ostelo R, Bartels R, Peul W, van Royen B, van Tulder M. Transforaminal endoscopic surgery for symptomatic lumbar disc herniations: a systematic review of the literature. Eur Spine J. 2010;19(2):181–204. https://doi.org/10.1007/s00586-009-1155-x.
18. Kosztowski TA, Choi D, Fridley J, et al. Lumbar disc reherniation after transforaminal lumbar endoscopic discectomy. Ann Transl Med. 2018;6(6):106. https://doi.org/10.21037/atm.2018.02.26.
19. Zhang B, Liu S, Liu J, et al. Transforaminal endoscopic discectomy versus conventional microdiscectomy for lumbar discherniation: a systematic review and meta-analysis. J Orthop Surg Res. 2018;13(1):169. https://doi.org/10.1186/s13018-018-0868-0.

20. Hermantin FU, Peters T, Quartararo L, Kambin P. A prospective, randomized study comparing the results of open discectomy with those of video-assisted arthroscopic microdiscectomy. J Bone Joint Surg Am. 1999;81(7):958–65. https://doi.org/10.2106/00004623-199907000-00008.
21. Mayer HM, Brock M. Percutaneous endoscopic discectomy: surgical technique and preliminary results compared to microsurgical discectomy. J Neurosurg. 1993;78(2):216–25. https://doi.org/10.3171/jns.1993.78.2.0216.
22. Ruetten S, Komp M, Merk H, Godolias G. Full-endoscopic interlaminar and transforaminal lumbar discectomy versus conventional microsurgical technique: a prospective, randomized, controlled study. Spine (Phila Pa 1976). 2008;33(9):931–9. https://doi.org/10.1097/BRS.0b013e31816c8af7.
23. Gibson JNA, Subramanian AS, Scott CEH. A randomised controlled trial of transforaminal endoscopic discectomy vs microdiscectomy. Eur Spine J. 2017;26(3):847–56. https://doi.org/10.1007/s00586-016-4885-6.
24. Akcakaya MO, Yorukoglu AG, Aydoseli A, et al. Serum creatine phosphokinase levels as an indicator of muscle injury following lumbar disc surgery: comparison of fully endoscopic discectomy and microdiscectomy. Clin Neurol Neurosurg. 2016;145:74–8. https://doi.org/10.1016/j.clineuro.2016.04.004.
25. Hsu HT, Chang SJ, Yang SS, Chai CL. Learning curve of full-endoscopic lumbar discectomy. Eur Spine J. 2013;22(4):727–33. https://doi.org/10.1007/s00586-012-2540-4.
26. Ahn SS, Kim SH, Kim DW, Lee BH. Comparison of outcomes of percutaneous endoscopic lumbar discectomy and open lumbar microdiscectomy for young adults: a retrospective matched cohort study. World Neurosurg. 2016;86:250–8. https://doi.org/10.1016/j.wneu.2015.09.047.
27. Lee SH, Chung SE, Ahn Y, Kim TH, Park JY, Shin SW. Comparative radiologic evaluation of percutaneous endoscopic lumbar discectomy and open microdiscectomy: a matched cohort analysis. Mt Sinai J Med. 2006;73(5):795–801.
28. Kim MJ, Lee SH, Jung ES, et al. Targeted percutaneous transforaminal endoscopic diskectomy in 295 patients: comparison with results of microscopic diskectomy. Surg Neurol. 2007;68(6):623–31. https://doi.org/10.1016/j.surneu.2006.12.051.
29. Chen Z, Zhang L, Dong J, et al. Percutaneous transforaminal endoscopic discectomy compared with microendoscopic discectomy for lumbar disc herniation: 1-year results of an ongoing randomized controlled trial. J Neurosurg Spine. 2018;28(3):300–10. https://doi.org/10.3171/2017.7.SPINE161434.
30. Ahn Y, Lee SG, Son S, Keum HJ. Transforaminal endoscopic lumbar discectomy versus open lumbar microdiscectomy: a comparative cohort study with a 5-year follow-up. Pain Physician. 2019;22(3):295–304.
31. Ahn Y, Jang IT, Kim WK. Transforaminal percutaneous endoscopic lumbar discectomy for very high-grade migrated disc herniation. Clin Neurol Neurosurg. 2016;147:11–7. https://doi.org/10.1016/j.clineuro.2016.05.016.
32. Telfeian AE, Doberstein C, Oyelese AA, Fridley J, Donahue JE, Gokaslan Z. A transforaminal endoscopic approach for treatment of a lumbar perineural cyst: technical note. World Neurosurg. 2019;127:85–91. https://doi.org/10.1016/j.wneu.2019.02.237.
33. Telfeian AE, Oyelese A, Fridley J, Moldovan K, Gokaslan ZL. Transforaminal endoscopic approach for lumbar extraforaminal synovial cysts: technical note. World Neurosurg. 2020;134:415–9. https://doi.org/10.1016/j.wneu.2019.11.033.
34. Kim HS, Sharma SB, Raorane HD. How I do it? Transforaminal endoscopic decompression of intraspinal facet cyst. Acta Neurochir (Wien). 2019;161(9):1895–900. https://doi.org/10.1007/s00701-019-03995-1.
35. Telfeian AE. Transforaminal endoscopic surgery for adjacent segment disease after lumbar fusion. World Neurosurg. 2017;97:231–5. https://doi.org/10.1016/j.wneu.2016.09.099.
36. Telfeian AE, Oyelese A, Fridley J, Gokaslan ZL. Transforaminal endoscopic decompression in the setting of lateral lumbar spondylolisthesis. World Neurosurg. 2018;117:321–5. https://doi.org/10.1016/j.wneu.2018.06.106.
37. Lee CH, Choi M, Ryu DS, et al. Efficacy and safety of full-endoscopic decompression via interlaminar approach for central or lateral recess spinal stenosis of the lumbar spine:

a meta-analysis. Spine (Phila Pa 1976). 2018;43(24):1756–64. https://doi.org/10.1097/BRS.0000000000002708.
38. Blanco G, Coulton GR, Biggin A, et al. The kyphoscoliosis (ky) mouse is deficient in hypertrophic responses and is caused by a mutation in a novel muscle-specific protein. Hum Mol Genet. 2001;10(1):9–16. https://doi.org/10.1093/hmg/10.1.9.
39. Xie TH, Zeng JC, Li ZH, et al. Complications of lumbar disc herniation following full-endoscopic interlaminar lumbar discectomy: a large, single-center, retrospective study. Pain Physician. 2017;20(3):E379–87.
40. Tu Z, Li YW, Wang B, et al. Clinical outcome of full-endoscopic interlaminar discectomy for single-level lumbar disc herniation: a minimum of 5-year follow-up. Pain Physician. 2017;20(3):E425–30.
41. Wasinpongwanich K, Pongpirul K, Lwin KMM, Kesornsak W, Kuansongtham V, Ruetten S. Full-endoscopic interlaminar lumbar discectomy: retrospective review of clinical results and complications in 545 international patients. World Neurosurg. 2019;132:e922–8. https://doi.org/10.1016/j.wneu.2019.07.101.
42. Chen P, Hu Y, Li Z. Percutaneous endoscopic transforaminal discectomy precedes interlaminar discectomy in the efficacy and safety for lumbar disc herniation. Biosci Rep. 2019;39(2):BSR20181866. https://doi.org/10.1042/BSR20181866.
43. Yin J, Jiang Y, Nong L. Transforaminal approach versus interlaminar approach: a meta-analysis of operative complication of percutaneous endoscopic lumbar discectomy. Medicine (Baltimore). 2020;99(25):e20709. https://doi.org/10.1097/MD.0000000000020709.
44. Chen J, Jing X, Li C, Jiang Y, Cheng S, Ma J. Percutaneous endoscopic lumbar discectomy for L5S1 lumbar disc herniation using a transforaminal approach versus an interlaminar approach: a systematic review and meta-analysis. World Neurosurg. 2018;116:412–420.e2. https://doi.org/10.1016/j.wneu.2018.05.075.
45. Rosenberg WS, Mummaneni PV. Transforaminal lumbar interbody fusion: technique, complications, and early results. Neurosurgery. 2001;48(3):569–74; discussion 574–5. https://doi.org/10.1097/00006123-200103000-00022.
46. Hee HT, Castro FP Jr, Majd ME, Holt RT, Myers L. Anterior/posterior lumbar fusion versus transforaminal lumbar interbody fusion: analysis of complications and predictive factors. J Spinal Disord. 2001;14(6):533–40. https://doi.org/10.1097/00002517-200112000-00013.
47. Schwender JD, Holly LT, Rouben DP, Foley KT. Minimally invasive transforaminal lumbar interbody fusion (TLIF): technical feasibility and initial results. J Spinal Disord Tech. 2005;18 Suppl:S1–6. https://doi.org/10.1097/01.bsd.0000132291.50455.d0.
48. Osman SG. Endoscopic transforaminal decompression, interbody fusion, and percutaneous pedicle screw implantation of the lumbar spine: a case series report. Int J Spine Surg. 2012;6:157–66. https://doi.org/10.1016/j.ijsp.2012.04.001.
49. Jacquot F, Gastambide D. Percutaneous endoscopic transforaminal lumbar interbody fusion: is it worth it? Int Orthop. 2013;37(8):1507–10. https://doi.org/10.1007/s00264-013-1905-6.
50. Lee SH, Erken HY, Bae J. Percutaneous transforaminal endoscopic lumbar interbody fusion: clinical and radiological results of mean 46-month follow-up. Biomed Res Int. 2017;2017:3731983. https://doi.org/10.1155/2017/3731983.
51. Kamson S, Lu D, Sampson PD, Zhang Y. Full-endoscopic lumbar fusion outcomes in patients with minimal deformities: a retrospective study of data collected between 2011 and 2015. Pain Physician. 2019;22(1):75–88.
52. Jin MR, Zhang J, Shao HY, Liu JW, Huang YZ. Percutaneous transforaminal endoscopic lumbar interbody fusion for degenerative lumbar diseases: a consecutive case series with mean 2-year follow-up. Pain Physician. 2020;23(2):165–74.
53. Kolcun JPG, Brusko GD, Basil GW, Epstein R, Wang MY. Endoscopic transforaminal lumbar interbody fusion without general anesthesia: operative and clinical outcomes in 100 consecutive patients with a minimum 1-year follow-up. Neurosurg Focus. 2019;46(4):E14. https://doi.org/10.3171/2018.12.FOCUS18701.

54. Liu C, Zhou Y. Percutaneous endoscopic lumbar discectomy and minimally invasive transforaminal lumbar interbody fusion for massive lumbar disc herniation. Clin Neurol Neurosurg. 2019;176:19–24. https://doi.org/10.1016/j.clineuro.2018.10.017.
55. Komp M, Oezdemir S, Hahn P, Ruetten S. Full-endoscopic posterior foraminotomy surgery for cervical disc herniations. Oper Orthop Traumatol. 2018;30(1):13–24. Vollendoskopische dorsale Foraminotomie zur Operation des zervikalen Bandscheibenvorfalls. https://doi.org/10.1007/s00064-017-0529-1.
56. Zheng C, Huang X, Yu J, Ye X. Posterior percutaneous endoscopic cervical diskectomy: a single-center experience of 252 cases. World Neurosurg. 2018;120:e63–7. https://doi.org/10.1016/j.wneu.2018.07.141.
57. Ruetten S, Komp M, Merk H, Godolias G. Full-endoscopic cervical posterior foraminotomy for the operation of lateral disc herniations using 5.9-mm endoscopes: a prospective, randomized, controlled study. Spine (Phila Pa 1976). 2008;33(9):940–8. https://doi.org/10.1097/BRS.0b013e31816c8b67.
58. Adamson TE. Microendoscopic posterior cervical laminoforaminotomy for unilateral radiculopathy: results of a new technique in 100 cases. J Neurosurg. 2001;95(1 Suppl):51–7. https://doi.org/10.3171/spi.2001.95.1.0051.
59. Ruetten S, Hahn P, Oezdemir S, et al. Full-endoscopic uniportal decompression in disc herniations and stenosis of the thoracic spine using the interlaminar, extraforaminal, or transthoracic retropleural approach. J Neurosurg Spine. 2018;29(2):157–68. https://doi.org/10.3171/2017.12.SPINE171096.
60. Choi EJ, Kim SY, Kim HG, Shon HS, Kim TK, Kim KH. Percutaneous endoscopic debridement and drainage with four different approach methods for the treatment of spinal infection. Pain Physician. 2017;20(6):E933–40.
61. Akbik OS, Shin PC. Endoscopic transforaminal drainage of a ventrally located thoracic epidural abscess. World Neurosurg. 2020;139:268–73. https://doi.org/10.1016/j.wneu.2020.03.193.
62. Xu Z, Zheng Y. Percutaneous endoscopic debridement and irrigation for thoracic infections. Rev Assoc Med Bras (1992). 2018;64(6):518–24. https://doi.org/10.1590/1806-9282.64.06.518.
63. Mao Y, Li Y, Cui X. Percutaneous endoscopic debridement and drainage for spinal infection: systemic review and meta-analysis. Pain Physician. 2019;22(4):323–30.

Minimally Invasive Spine Surgery

20

Carl Youssef, Salah G. Aoun, and Mazin Al Tamimi

Minimally Invasive Thoracolumbar Spine Surgery

The first report of microdiscectomy utilizing the microscope was in 1967, which still involved open dissection of the paraspinal musculature and laminae [2]. In 1969, injection of the proteolytic enzyme chymopapain into the disc was used in a technique referred to as chemonucleolysis, resulting in the breakdown of macromolecules in the nucleus pulposus [3]. This was considered the first minimally invasive spine procedure, although it was not popularized at the time due to several reports of arachnoiditis and chemical discitis, resulting in several months of low back pain. In 1975, small self-retaining soft tissues retractors were introduced, allowing performance of microdiscectomy through a smaller window [4]. The use of laser technology in spine surgery was first reported in 1978 when it was used to excise spinal cord tumors, but it was not until 1984 that it was first used to treat lumbar disc disease [5].

A major milestone in the history of MISS was the development of tubular access and retractor systems. The first rudimentary application of this system was in 1991 [6]. Under biplanar fluoroscopic guidance, a cannula with a guide wire followed by a working sleeve with an outer diameter of 5.4 mm were introduced into the affected disc. The guide wire was then removed and "nucleus forceps" and high vacuum and irrigation were used to remove the disc material. This procedure was performed under local anesthesia, and usually took about 20 minutes.

C. Youssef
Department of Neurological Surgery, UT Southwestern Medical Center, Dallas, TX, USA
e-mail: Carl.Youssef@phhs.org

S. G. Aoun · M. Al Tamimi (✉)
Department of Neurological Surgery and Spine Center, UT Southwestern Medical Center, Dallas, TX, USA
e-mail: Salah.Aoun@utsouthwestern.edu; Mazin.AlTamimi@utsouthwestern.edu

© The Author(s), under exclusive license to Springer Nature Switzerland AG 2022
C. E. Noe (ed.), *Multidisciplinary Spine Care*,
https://doi.org/10.1007/978-3-031-04990-3_20

The tubular approach was further refined in 1997 with the introduction of the microendoscopic discectomy (MED) system, in which serial dilators were used to introduce a bigger tubular retractor, to which an endoscope is attached [7]. This technique did not gain immediate popularity initially, primarily due to the steep learning curve and surgeon unfamiliarity with the endoscope, which resulted in relatively high rate of unintended dural tears. With more experience, however, it was shown to be a reliable minimally invasive approach to microdiscectomies, even for large disc herniations. When compared with open microdiscectomy, the MED approach had equivalent long-term improvement in pain and disability but with less morbidity [8–11].

In the early 2000's, the MED system evolved into the Microscopic Endoscopic Tubular Retractor System (METRx; developed by Medtronic Sofamor DaneK, Memphis, TN). Like MED, the METRx system also consists of a series of dilators and tubular retractors. One of the distinguishing features between the two systems is the incorporation of the operative microscope. The first application of the METRx technique was in lumbar microdiscectomies. Initial experience followed by several studies demonstrated excellent clinical results and cost effectiveness with this approach, particularly in terms of decreased blood loss, less tissue trauma, less post-operative pain, lower rates of surgical site infections, shorter hospital stays, and faster return to work [12–14]. This system was also found to be favorable in obese patients, which is a patient population that typically requires larger incisions and is more prone to post-operative infections [15].

Figure 20.1a–e show microdiscectomy using the METRx system. Figure 20.1a shows the paramedian approach to the lumbar spine using the tubular retractor. Figure 20.1b shows fluoroscopic confirmation of the tubular retractor position over the intended disc space. Figure 20.1c shows the view through the tubular retractor with the operative microscope. Figure 20.1d shows the small size of the incision needed for the procedure. Figure 20.1e shows the extracted large disc fragment.

The use of MISS techniques expanded to decompressive laminectomies, with the ability to perform bilateral decompression via a unilateral approach [16, 17]. Perhaps the most notable benefit of the minimally invasive approach to decompression is the preservation of the supporting structures in the lumbar spine, which has been shown to minimize post-operative instability and the need for fusion [18]. This is particularly important in patients with degenerative spondylolisthesis, in which minimally invasive decompression resulted in less progression of the slip and lower reoperation rates for secondary fusion [19].

The application of minimally invasive techniques to instrumentation represents the next major step in the evolution of MISS. In contemporary spine surgery, pedicle screw fixation has become the standard technique for instrumentation in the thoracolumbar spine. To expose the entry point of pedicle screws via an open approach, the the multifidus muscle has to be elevated and retracted off the laminae and facet joints. This results in atrophy of the muscles due to denervation from damage to the medial branch of the posterior rami as well as ischemic necrosis from prolonged retraction. Functionally, this is associated with increased post-operative pain and decreased truncal extensor muscle strength [20, 21].

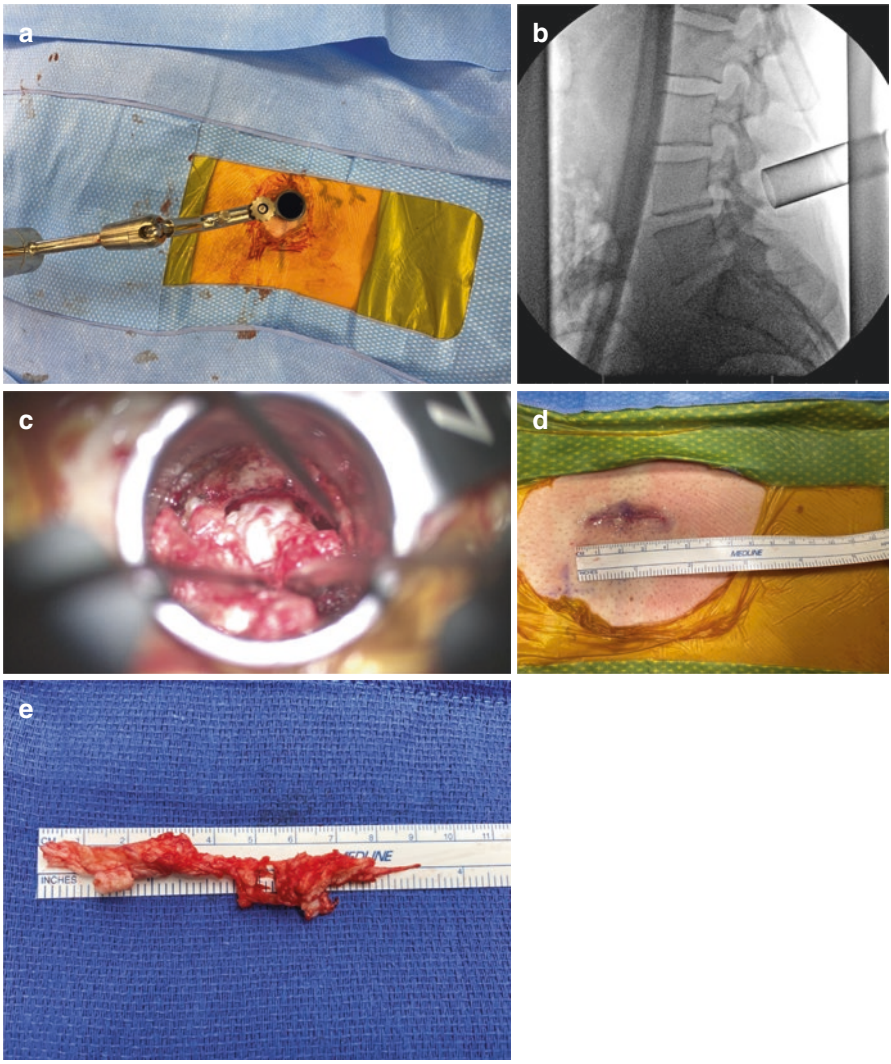


Fig. 20.1 (**a**) Paramedian approach to the lumbar spine using the tubular retractor. (**b**) Fluoroscopic confirmation of the tubular retractor position over the intended disc space. (**c**) View through the tubular retractor with the operative microscope. (**d**) Demonstration of the small size of the incision needed. (**e**) Extracted large disc fragment

To minimize soft tissue damage, percutaneous pedicle screw fixation was introduced in the early 2000's [22, 23]. Using this technique, the pedicle is cannulated percutaneously via a small stab incision through the skin and fascia, leaving the paraspinal musculature essentially intact. Rods are then fitted onto the screws in a subfascial fashion using one of several different systems. In thoracolumbar trauma, this technique was shown to be a feasible alternative to open fusion, with lower operative time, perioperative blood loss, surgical site infection, and pain [24, 25].

In addition to tubular retractors, percutaneous pedicle screw fixation has revolutionarized the field MISS, particularly for degenerative conditions. In 2002, the first minimally invasive posterior lumbar interbody fusion was reported, showing feasibility of achieving wide decompression with interbody fusion while minimizing iatrogenic damage [26]. In 2005, several studies reported success with minimally invasive transforaminal interbody fusion [27–29]. These early positive results were confirmed by several recent systematic analyses, demonstrating efficacy, safety, and cost effectiveness [30–33].

The next phase of MISS came in the form of anterolateral lumbar interbody fusion techniques. These techniques include the transposas (e.g. lateral), prepsoas (e.g. oblique), and anterior approaches [34]. The detailed differences among these approaches are beyond the scope of this chapter, but these approaches offer several advantages worth noting here. Since anterolateral approaches can be used as a standlone arthrodesis technique of the anterior lumbar spine, one of the major advantages is the complete avoidance of violating the paraspinal musculature, facet joints, and other posterior supporting ligaments, thus maintaining structural integrity of the lumbar spine. Even when posterior instrumentation is required, it is often achieved with percuatneous techniques that maintain the minimally invasive nature of the procedure. In patients with prior fusions presenting with adjacent segment disease requiring revision, an anterolateral approach can be utilized to treat that adjacent segment, thus avoiding re-opening the posterior incision and the morbidity associated with revision surgery [35, 36]. Another major advantage of minimally invasive anterolateral approaches is the ability to provide indirect decompression in patients with central or foraminal stenosis. By removing the collapsed disc and placing an interbody cage, the disc height is restored, which in turns increases foraminal height and minimizes “buckling” of the ligamentum flavum postrerior to the thecal sac [37–40].

Minimally Invasive Thoracic Spine Surgery

The thoracic spine is the most structuraly stable segment of the mobile spine because of the added support by the ribcage [41]. As a result, degenrative conditions are not as common in this region as they are in the cervical or lumbar spine. Nonetheless, several conditions, such as deformity, tumors, trauma, and infections, can affect the thoracic spine and necessitate surgery. From the late nineteenth century to the early twentieth century, surgery on the thoracic spine has predominantly consisted of dorsal decompression via laminectomy. The main limitation of that approach is the inability to achieve ventral decompression of the thecal sac or reach lesions involving the anterial thoracic spine due to the presence of the spinal cord [42]. To address that limitation, several posterolateral techniques were introduced as early as 1894 when the costotransversectomy approach was decribed to drain tuberculous paraspinal abscesses in patients with Pott’s disease [42]. In 1956, the anterolateral approach via thoracotomy was introduced to provide wide multilevel exposure to

the anterior thoracic spine, which is sometimes necessary for correction of kyphoscoliotic defomities and tumor resections [43].

The posterolateral and transthoracic approaches to the thoracic spine have allowed for much better access to the ventral thoracic spine. However, as one can imagine, these can be very invasive procedures and can be associated with significant morbity. The reported complication rate for the transthoracic approach is as high as 39% whereas the complication rate for posterolateral approaches ranges between 15% and 17% [44]. Thus, the need for the incorporation of MISS techniques to this challenging region of the spine has become apparent.

One of the major advances in minimally invasive thoracic spine surgery is the incorporation of video-assisted thoracoscopic surgery (VATS) technology. This techonology was developed by cardiothoracic surgery in the early 1990's to supplant the traditional thoracotomy approaches to several intrathoracic pathologies [45]. The advantages of VATS over open thoracotomy were readily apparent—smaller incision, less acute and chronic pain, reduced length of hospital stay, and faster return to normal activities. Since 1991, the utility of VATS has been successfully demonstrated in treating thoracic disc herniations, anterior release for deformity corrections, corpectomies, and drainage of spinal abscesses, without the high morbidity associated with the traditional thoracotomy approach [46]. This procedure, however, is associated with a steep learning curve and requires specialized training and collaboration with thoracic surgeons [47].

With regard to posterolateral approaches, advances in minimally invasive techniques to the thoracic spine were developed in parallel with those employed in the lumbar spine. Rather than prolonged immobilization or open instrumented fusion, the percutaneous pedicle screw fixation technique has been successfully applied to internally stabilize fractures [48]. Similarly,the use of tubular retractor systems has made it possible to transform invasive procedures requiring long incisions and extensive dissection into much less invasive ones [49, 50].

Minimally Invasive Cervical Spine Surgery

Surgical approaches to the cervical spine have evolved significantly over the past few decades. Disorders of the cervical spine can be treated via an anterior approach as well as a posterior one. Anteriorly, disc herniations, traumatic injuries, and neoplasms involving the vertebral bodies have been treated with anterior cervical discectomy and fusion (ACDF). First introduced in 1955 and refined in 1958, ACDF offers a relatively minimally invasive approach to the anterior cervical spine [51]. It is performed through a small incision and without much iatrogenic tissue disruption as it takes advantage of the normal tissue planes in the neck.

Variations of the ACDF approach have been developed over the past two decades to make the procedure even less invasive. Cervical disc arthroplasty is an example of such variation which was popularized in the early 2000's [52]. It involves removal of the diseased disc and replacing it with an artificial disc implant that preserves

segmental motion at that level. This procedure does not require placement of screws or plates and does not require the aggressive preparation of the endplates needed to promote arthrodesis. Furthermore, becasuse of the motion preservation and the minimal disruption to normal cervical spine biomechanics, some studies reported better long term outcomes compared to ACDF in terms of improved pain and lower incidence of reoperation for adjacent segment disease [53].

Similar to decompression of the thoracic and lumbar spine, laminectomy has been the gold standard for dorsal decompression of the neural elements in the cervical spine. Traditionally, open decompression and/or stabilization with screws/rods involve extensive muscular dissection and retraction, which has negative impact on the structral integrety of the spine. Postlaminectomy kyphosis is a well-documented long-term consequence of the disruption of the posterior supporting bony, ligamentous, and muscular structures, and is particularly improtant in patients with multilevel decompression and baseline reversal of normal cervical lordosis [54]. To minimze collateral iatrogenic damage, minimally invasive approaches to the posterior cervical spine were developed, the most prevalent of which is tubular microscopic or endoscopic laminoforaminotomy [55]. This procedure allows for decompression of the lateral thecal sac and exiting nerve root in patients with radiculopathy with minimal trauma to the posterior paraspinal musculature, and has been shown to reduce post-operative analgesic medication usage, intra-operative blood loss, and length of hospital stay when compared with the open approach [56]. Additionally, when compared with ACDF, minimally invasive laminoforamintomy was shown to be at least as efficacious as ACDF in treating radiculopathy while still maintaining a lower complication profile and reoperation rate [57].

With regard to fusion procedures, open approaches have remained the gold standard for instrumented posterolateral fixation of the axial and subaxial cervical spine. Nonetheless, few minimally invasive posterior fusion techniques are described in the literature. One example is C1–C2 instrumented fixation using tubular retractors [58]. The procedure is performed through bilateral 2 cm incisions that are 2 cm off the midline, and fluoroscopy is used for screw placement. Similarly, multilevel lateral mass screws can be placed using specialized tubular retractors with deep tissue expanders called “skirts” [59]. These procedures, however, are technically challenging and requires normal unaltered anatomy, comfort with open instrumentation and general minimally invasive techniques, and excellent fluoroscopic visualization.

Percutaneous facet joint instrumentation is another interesting example. The facetal distraction-fixation procedure was first reported in 2004 as an adjunct to screw/rod fixation for atlantoaxial instability [60]. It has then evolved to treat instability and degenerative pathologies in the axial and subaxial spine by “jamming” a metallic cage implant in the distracted joint either as a percutaneous standalone fixation technique or in combination with open lateral mass screw/rod systems [61]. This facetal distraction-fixation technique provides indirect decompression of the nerve root and confers segmental stability by promoting arthrodesis. Indeed, the fusion rate of the standalone technique after 2 years is up to 98.1%, with no segmental kyphosis, device failures, or reoperations [62]. Contraindications to this procedure are infections, tumors affecting the facet joint, traumatic facet injuries, and high grade listhesis [63].

Miscellaneous

There are other notable examples of MISS that do not fit within any of the above sections. One such example is sacroiliac (SI) joint fusion. The prevalence of sacroiliitis in patients with chronic low back pain is reported to be up to 30% [64]. Nonetheless, it has remained an under-recognized problem in patients presenting with low back or buttock pain due to the significant overlap of symptoms and the lack of specific diagnostic tests or reliable physical exam findings [65]. Once the diagnosis is established, usually by a constellation of exam findings and diagnostic injections, surgical treatment can be offered to stabilize the joint if the patient fails a trial of therapeutic injections and/or radiofrequency denervation. Different surgical approaches to the SI joint have been described. The intra-pelvic anterior approach to the SI joint over the pelvic brim is one of the earliest approaches described in the literature, but it is an invasive procedure and access to the joint is limited by the iliac vessels and the S1 and S2 nerve roots [66]. To avoid the morbidity of the anterior approach, an open lateral trans-iliac subgluteal approach was developed, which minimized the possibility of direct injury to the major vessels and nerve roots [67]. Still, this also constituted an invasive approach, requiring dissection of the gluteal muscles and drilling a bony window in the iliac bone, entailing the possibility of indirect neurovasular injury with misguided screws or dowels across the ventromedial aspect of the joint.

Beginning in the early 2000's, minimally invasive SI joint fusion techniques have been introduced, utilizing fluoroscopic guidance to percutaneously place triangular or cylindrical implants across the joint through either a lateral transarticular approach or a posterior intraarticular approach [68, 69]. When compared with their open counterparts, minimnally invasive techniques demonstrate superior pain relief and decreased perioperative morbidity [70]. When compared to nonoperative management, SI joint fusion undoubtedly provides excellent long term outcomes in terms of improvement in pain, decreased opioid consumption, faster return to work, and improved quality of life [71, 72]. Currently, as progress is made in the diagnosis and treatment of sacroilitis, minimally invasive SI joint fusion is increasingly becoming an integral component in managing patients who have failed a trial of conservative management.

Vertebroplasty and kyphoplasty represent another major form of MISS. This procedure was initially described in 1987 in France [73]. In the mid 1990's, the procedure gained popularity in the United States, and its use has expanded to encompass osteoporotic fractures, pathologic fractures, and augmentation of weak vertebrae prior to surgery [74]. It is a minimally invasive procedure that is performed percutaneously under fluoroscopic guidance by inflating a balloon to restore height and injecting methyl methacrylate cement into the vertebral body through a transpedicular or parapedicular needle [75]. The most common indication for the procedure is osteoporotic compression fracture refractory to conservative management for at least 2 weeks. Another common indication is the treatment of metastases with or without adjucant surgery or radiation to not only relieve pain but also to maintain structural integrity in the setting of lytic vertebral body lesions. The procedure is very effective, with significant short and long term improvement in mobility, analgesic usage, pain at rest, and pain with activity [54].

Technological Advances in Minimally Invasive Spine Surgery

Image-guided surgery (IGS) has had a tremendous impact in the development and expansion of the field of MISS. Intra-operative imaging evolved from two-dimentional (2-D) fluoroscopy and plain films to more advanced three-dimentional (3-D) intra-operative navigation systems. The first application of a 3-D navigation system in spine surgery was reported in 1996 when a cranial neurosurgery navigation system utilizing pre-operative CT images was adapted to spine surgery [76]. This interactive navigation system demonstrated improved instrumentation accuracy and better intraoperative localization of important anatomic structures compared to traditional 2-D imaging methods. Building upon that technology, fluoroscopy-based navigation systems were developed, with the main advantage of offering "real time" intra-operative images rather than using images obtained pre-operatively [77–79]. Further advances led to the development of intra-operative CT-guided navigation systems (e.g. O-Arm, Medtronic Inc., Louisville, Colorado, USA), which currently remain the gold standard in intra-operative navigation in spine surgery. The newer low-dose CT-based systems allow for the rapid acquisition of optimal intra-operative imaging and precise navigated instrumentation, while still decreasing overall radiation exposure to surgical staff and decrreasing operative time in certain situations [80–82].

Another exciting example of the influx of technology into the field of spine surgery is the incorporation of robotic technology. Surgical robotic technology in general is divided into two categories: telesurgical robotic systems and robotic-assisted navigation (RAN) [83]. An example of the former is the Da Vinci robotic system (Intuitive Surgical Inc., Sunnyvale, CA, USA), through which the surgeon is able to perform the surgery from a command station with the robot handling all the instruments. This system is most commonly utilized in general surgical specialties; it is not FDA-approved for spine surgery and its role in spine surgery to date has been limited to few reports describing its usage for anterior exposure of the lumbar spine.

The latter category of robotic surgery is more relevant to the field of MISS. In RAN, the role of the robot is to provide guidance to the surgeon in placement of instrumentation utilizing pre- or intra-operatively obtained imaging. The first RAN system was developed in 2004 and later obtained FDA aproval for use in spine surgery [84]. The initial prototype utilized pre-operative CT scans merged with intra-operative fluoroscopy. It demonstrated high accuracy in pedicle screw placement and significantly reduced radiation exposure when compared to fluoroscopy-guided instrumentation [85]. As with any new technology, however, initial experience revealed a steep learning curve and occasional issues with accuracy due to issues with registration or excessive pressure from soft tissues or the surgeon on the robotic arm resulting in deviation from planned trajectory. Newer iterations of RAN improved upon the initial prototype, producing smaller robotic systems that are able to process information seamlessly, plan multiple trajectories simultaneously, detect drill skiving, and compensate for patient movement. With these recent refinements, accuracy of pedicle screw placement was as high as 99% and with minimal need to return to the operating room for malpositioned screws [86]. Nonetheless, robotic

technology in spine surgery remains in its infancy, with ongoing studies about long term outcomes and cost-effectiveness compared to the more established technologies [87, 88].

Lastly, we will conclude this section with a discussion about augmented reality (AR) surgical navigation technology in spine surgery. With this technology, the surgeon, via wearable heads up display or the operative microscope, is able to have "x-ray" vision by superimposing a virtual picture onto the patient's physical anatomy. This technology has been applied not only in pedicle screw placement but also in other procedures such as tumor resections, deformity corrections, and vertebroplasty/kyphoplasty [89]. One advantage of AR over prior methods of IGS is the ability of the surgeon to maintain field of vision over the patient rather looking away from the surgical field onto a screen. Furthermore, AR provides an excellent educational tool outside of the operating room, allowing trainees to place virtual pedicle screws with haptic feedback [90]. Again, as is the case with robotics, AR still remains in a very early stage in its clinical application to spine surgery, and further studies are needed to validate its outcomes and cost-effectiveness.

Conclusion

Tremendous advances have been made in the field of minimally invasive spine surgery. With growing technology, spine surgery is gradually transforming away from the traditional open approaches that usually result in extensive collateral iatrogenic to more sleek approaches utilizing an armamentarium of new imaging and instrumentation tools. The overall end result of this paradim shift is less acute and chronic pain, minimal blood loss, shorter hospital stay, less radiation exposure, and faster return to normal function. The future of MISS is promising as current technologies are constantly being refined and newer advances are continuously being implemented and validated.

References

1. Tsui C, Klein R, Garabrant M. Minimally invasive surgery: national trends in adoption and future directions for hospital strategy. Surg Endosc. 2013;27(7):2253–7.
2. Yasargil MG. Microsurgical operation of herniated lumbar disc. In: Lumbar disc adult hydrocephalus. Advances in neurosurgery, vol. 4. Berlin, Heidelberg: Springer; 1977.
3. Smith L. Chemonucleolysis. Clin Orthop Relat Res. 1969;67:72–80.
4. Williams RW. Microlumbar discectomy: a conservative surgical approach to the virgin herniated lumbar disc. Spine (Phila Pa 1976). 1978;3(2):175–82.
5. Ascher PW, Heppner F. CO2-laser in neurosurgery. Neurosurg Rev. 1984;7(2–3):123–33.
6. Faubert C, Caspar W. Lumbar percutaneous discectomy. Initial experience in 28 cases. Neuroradiology. 1991;33(5):407–10.
7. Perez-Cruet MJ, et al. Microendoscopic lumbar discectomy: technical note. Neurosurgery. 2002;51(5 Suppl):S129–36.
8. Wu X, et al. Microendoscopic discectomy for lumbar disc herniation: surgical technique and outcome in 873 consecutive cases. Spine (Phila Pa 1976). 2006;31(23):2689–94.

9. Righesso O, Falavigna A, Avanzi O. Comparison of open discectomy with microendoscopic discectomy in lumbar disc herniations: results of a randomized controlled trial. Neurosurgery. 2007;61(3):545–9. discussion 549
10. Schizas C, Tsiridis E, Saksena J. Microendoscopic discectomy compared with standard microsurgical discectomy for treatment of uncontained or large contained disc herniations. Neurosurgery. 2005;57(4 Suppl):357–60; discussion 357–60.
11. Hussein M, Abdeldayem A, Mattar MM. Surgical technique and effectiveness of microendoscopic discectomy for large uncontained lumbar disc herniations: a prospective, randomized, controlled study with 8 years of follow-up. Eur Spine J. 2014;23(9):1992–9.
12. Palmer S. Use of a tubular retractor system in microscopic lumbar discectomy: 1 year prospective results in 135 patients. Neurosurg Focus. 2002;13(2):E5.
13. O'Toole JE, Eichholz KM, Fessler RG. Surgical site infection rates after minimally invasive spinal surgery. J Neurosurg Spine. 2009;11(4):471–6.
14. Harrington JF, French P. Open versus minimally invasive lumbar microdiscectomy: comparison of operative times, length of hospital stay, narcotic use and complications. Minim Invasive Neurosurg. 2008;51(1):30–5.
15. Cole JS 4th, Jackson TR. Minimally invasive lumbar discectomy in obese patients. Neurosurgery. 2007;61(3):539–44; discussion 544.
16. Phan K, Mobbs RJ. Minimally invasive versus open laminectomy for lumbar stenosis: a systematic review and meta-analysis. Spine (Phila Pa 1976). 2016;41(2):E91–E100.
17. Mobbs RJ, et al. Outcomes after decompressive laminectomy for lumbar spinal stenosis: comparison between minimally invasive unilateral laminectomy for bilateral decompression and open laminectomy: clinical article. J Neurosurg Spine. 2014;21(2):179–86.
18. Minamide A, et al. Minimally invasive spinal decompression for degenerative lumbar spondylolisthesis and stenosis maintains stability and may avoid the need for fusion. Bone Joint J. 2018;100-B(4):499–506.
19. Scholler K, et al. Lumbar spinal stenosis associated with degenerative lumbar spondylolisthesis: a systematic review and meta-analysis of secondary fusion rates following open vs minimally invasive decompression. Neurosurgery. 2017;80(3):355–67.
20. Kim DY, et al. Comparison of multifidus muscle atrophy and trunk extension muscle strength: percutaneous versus open pedicle screw fixation. Spine (Phila Pa 1976). 2005;30(1):123–9.
21. Regev GJ, et al. Nerve injury to the posterior rami medial branch during the insertion of pedicle screws: comparison of mini-open versus percutaneous pedicle screw insertion techniques. Spine (Phila Pa 1976). 2009;34(11):1239–42.
22. Wiesner L, et al. Clinical evaluation and computed tomography scan analysis of screw tracts after percutaneous insertion of pedicle screws in the lumbar spine. Spine (Phila Pa 1976). 2000;25(5):615–21.
23. Foley KT, Gupta SK. Percutaneous pedicle screw fixation of the lumbar spine: preliminary clinical results. J Neurosurg. 2002;97(1 Suppl):7–12.
24. Phan K, Rao PJ, Mobbs RJ. Percutaneous versus open pedicle screw fixation for treatment of thoracolumbar fractures: systematic review and meta-analysis of comparative studies. Clin Neurol Neurosurg. 2015;135:85–92.
25. Ni WF, et al. Percutaneous pedicle screw fixation for neurologic intact thoracolumbar burst fractures. J Spinal Disord Tech. 2010;23(8):530–7.
26. Khoo LT, et al. Minimally invasive percutaneous posterior lumbar interbody fusion. Neurosurgery. 2002;51(5 Suppl):S166–81.
27. Isaacs RE, et al. Minimally invasive microendoscopy-assisted transforaminal lumbar interbody fusion with instrumentation. J Neurosurg Spine. 2005;3(2):98–105.
28. Ozgur BM, et al. Minimally-invasive technique for transforaminal lumbar interbody fusion (TLIF). Eur Spine J. 2005;14(9):887–94.
29. Schwender JD, et al. Minimally invasive transforaminal lumbar interbody fusion (TLIF): technical feasibility and initial results. J Spinal Disord Tech. 2005;18 Suppl:S1–6.

30. Khan NR, et al. Surgical outcomes for minimally invasive vs open transforaminal lumbar interbody fusion: an updated systematic review and meta-analysis. Neurosurgery. 2015;77(6):847–74; discussion 874.
31. Phan K, et al. Minimally invasive versus open transforaminal lumbar interbody fusion for treatment of degenerative lumbar disease: systematic review and meta-analysis. Eur Spine J. 2015;24(5):1017–30.
32. Hu W, et al. Minimally invasive versus open transforaminal lumbar fusion: a systematic review of complications. Int Orthop. 2016;40(9):1883–90.
33. Phan K, Hogan JA, Mobbs RJ. Cost-utility of minimally invasive versus open transforaminal lumbar interbody fusion: systematic review and economic evaluation. Eur Spine J. 2015;24(11):2503–13.
34. Xu DS, et al. Minimally invasive anterior, lateral, and oblique lumbar interbody fusion: a literature review. Ann Transl Med. 2018;6(6):104.
35. Tu Z, et al. Stand-alone anterolateral interbody fusion versus extended posterior fusion for symptomatic adjacent-segment degeneration: a retrospective study of 2 years' follow-up. World Neurosurg. 2018;115:e748–55.
36. Wang MY, Vasudevan R, Mindea SA. Minimally invasive lateral interbody fusion for the treatment of rostral adjacent-segment lumbar degenerative stenosis without supplemental pedicle screw fixation. J Neurosurg Spine. 2014;21(6):861–6.
37. Elowitz EH, et al. Evaluation of indirect decompression of the lumbar spinal canal following minimally invasive lateral transpsoas interbody fusion: radiographic and outcome analysis. Minim Invasive Neurosurg. 2011;54(5–6):201–6.
38. Lin GX, et al. Clinical and radiologic outcomes of direct versus indirect decompression with lumbar interbody fusion: a matched-pair comparison analysis. World Neurosurg. 2018;119:e898–909.
39. Fujibayashi S, et al. Effect of indirect neural decompression through oblique lateral interbody fusion for degenerative lumbar disease. Spine (Phila Pa 1976). 2015;40(3):E175–82.
40. Limthongkul W, et al. Indirect decompression effect to central canal and ligamentum flavum after extreme lateral lumbar interbody fusion and oblique lumbar interbody fusion. Spine (Phila Pa 1976). 2020;45(17):E1077–84.
41. Watkins R 4th, et al. Stability provided by the sternum and rib cage in the thoracic spine. Spine (Phila Pa 1976). 2005;30(11):1283–6.
42. Dohn DF. Thoracic spinal cord decompression: alternative surgical approaches and basis of choice. Clin Neurosurg. 1980;27:611–23.
43. Hodgson AR, Stock FE. Anterior spinal fusion a preliminary communication on the radical treatment of Pott's disease and Pott's paraplegia. Br J Surg. 1956;44(185):266–75.
44. Lubelski D, et al. Lateral extracavitary, costotransversectomy, and transthoracic thoracotomy approaches to the thoracic spine: review of techniques and complications. J Spinal Disord Tech. 2013;26(4):222–32.
45. Mineo TC, Ambrogi V. A glance at the history of uniportal video-assisted thoracic surgery. J Vis Surg. 2017;3:157.
46. Mack MJ, et al. Video-assisted thoracic surgery for the anterior approach to the thoracic spine. Ann Thorac Surg. 1995;59(5):1100–6.
47. Visocchi M, et al. Thoracoscopic approaches to the thoracic spine. Acta Neurochir. 1998;140(8):737–43; discussion 743–4.
48. Anderson DG, et al. Percutaneous instrumentation of the thoracic and lumbar spine. Orthop Clin North Am. 2007;38(3):401–8; abstract vii.
49. Kim DH, et al. Minimally invasive posterolateral thoracic corpectomy: cadaveric feasibility study and report of four clinical cases. Neurosurgery. 2009;64(4):746–52; discussion 752–3.
50. Fessler RG, et al. Current advances and evidence in minimally invasive spine surgery. Minim Invasive Surg. 2012;2012:508415.
51. Cloward RB. The anterior approach for removal of ruptured cervical disks. J Neurosurg. 1958;15(6):602–17.

52. Le H, Thongtrangan I, Kim DH. Historical review of cervical arthroplasty. Neurosurg Focus. 2004;17(3):E1.
53. Wang QL, et al. Long-term results comparing cervical disc arthroplasty to anterior cervical discectomy and fusion: a systematic review and meta-analysis of randomized controlled trials. Orthop Surg. 2020;12(1):16–30.
54. Albert TJ, Vacarro A. Postlaminectomy kyphosis. Spine (Phila Pa 1976). 1998;23(24):2738–45.
55. Gala VC, et al. Posterior minimally invasive approaches for the cervical spine. Orthop Clin North Am. 2007;38(3):339–49; abstract v.
56. Winder MJ, Thomas KC. Minimally invasive versus open approach for cervical laminoforaminotomy. Can J Neurol Sci. 2011;38(2):262–7.
57. Sahai N, et al. Minimally invasive posterior cervical foraminotomy as an alternative to anterior cervical discectomy and fusion for unilateral cervical radiculopathy: a systematic review and meta-analysis. Spine (Phila Pa 1976). 2019;44(24):1731–9.
58. Holly LT, Isaacs RE, Frempong-Boadu AK. Minimally invasive atlantoaxial fusion. Neurosurgery. 2010;66(3 Suppl):193–7.
59. Mikhael MM, et al. Minimally invasive cervical spine foraminotomy and lateral mass screw placement. Spine (Phila Pa 1976). 2012;37(5):E318–22.
60. Goel A. Treatment of basilar invagination by atlantoaxial joint distraction and direct lateral mass fixation. J Neurosurg Spine. 2004;1(3):281–6.
61. Goel A. Interfacetal intra-articular spacers: emergence of a concept. J Craniovertebr Junction Spine. 2016;7(2):72–4.
62. Siemionow K, et al. Clinical and radiographic results of indirect decompression and posterior cervical fusion for single-level cervical radiculopathy using an expandable implant with 2-year follow-up. J Neurol Surg A Cent Eur Neurosurg. 2016;77(6):482–8.
63. McCormack BM, et al. Percutaneous posterior cervical fusion with the DTRAX Facet System for single-level radiculopathy: results in 60 patients. J Neurosurg Spine. 2013;18(3):245–54.
64. Schwarzer AC, Aprill CN, Bogduk N. The sacroiliac joint in chronic low back pain. Spine (Phila Pa 1976). 1995;20(1):31–7.
65. Cohen SP, Chen Y, Neufeld NJ. Sacroiliac joint pain: a comprehensive review of epidemiology, diagnosis and treatment. Expert Rev Neurother. 2013;13(1):99–116.
66. Rand JA. Anterior sacro-iliac arthrodesis for post-traumatic sacro-iliac arthritis. A case report. J Bone Joint Surg Am. 1985;67(1):157–9.
67. Smith-Petersen M. Arthrodesis of the sacroiliac joint. A new method of approach. J Bone Joint Surg. 1921;3(8):400–5.
68. Wise CL, Dall BE. Minimally invasive sacroiliac arthrodesis: outcomes of a new technique. J Spinal Disord Tech. 2008;21(8):579–84.
69. Rudolf L. Sacroiliac joint arthrodesis-MIS technique with titanium implants: report of the first 50 patients and outcomes. Open Orthop J. 2012;6:495–502.
70. Smith AG, et al. Open versus minimally invasive sacroiliac joint fusion: a multi-center comparison of perioperative measures and clinical outcomes. Ann Surg Innov Res. 2013;7(1):14.
71. Vanaclocha V, et al. Minimally invasive sacroiliac joint fusion, radiofrequency denervation, and conservative management for sacroiliac joint pain: 6-year comparative case series. Neurosurgery. 2018;82(1):48–55.
72. Dengler J, et al. Randomized trial of sacroiliac joint arthrodesis compared with conservative management for chronic low back pain attributed to the sacroiliac joint. J Bone Joint Surg Am. 2019;101(5):400–11.
73. Galibert P, et al. Preliminary note on the treatment of vertebral angioma by percutaneous acrylic vertebroplasty. Neurochirurgie. 1987;33(2):166–8.
74. Cotten A, et al. Percutaneous vertebroplasty: state of the art. Radiographics. 1998;18(2):311–20; discussion 320–3.
75. Predey TA, Sewall LE, Smith SJ. Percutaneous vertebroplasty: new treatment for vertebral compression fractures. Am Fam Physician. 2002;66(4):611–5.
76. Foley KT, Smith MM. Image-guided spine surgery. Neurosurg Clin N Am. 1996;7(2):171–86.

77. Villavicencio AT, et al. Utility of computerized isocentric fluoroscopy for minimally invasive spinal surgical techniques. J Spinal Disord Tech. 2005;18(4):369–75.
78. Foley KT, Simon DA, Rampersaud YR. Virtual fluoroscopy: computer-assisted fluoroscopic navigation. Spine (Phila Pa 1976). 2001;26(4):347–51.
79. Nolte LP, et al. A new approach to computer-aided spine surgery: fluoroscopy-based surgical navigation. Eur Spine J. 2000;9(Suppl 1):S78–88.
80. Kim TT, et al. Minimally invasive spinal surgery with intraoperative image-guided navigation. Biomed Res Int. 2016;2016:5716235.
81. Feng W, et al. O-arm navigation versus C-arm guidance for pedicle screw placement in spine surgery: a systematic review and meta-analysis. Int Orthop. 2020;44(5):919–26.
82. Costa F, et al. Radiation exposure in spine surgery using an image-guided system based on intraoperative cone-beam computed tomography: analysis of 107 consecutive cases. J Neurosurg Spine. 2016;25(5):654–9.
83. Vadala G, et al. Robotic spine surgery and augmented reality systems: a state of the art. Neurospine. 2020;17(1):88–100.
84. Barzilay Y, et al. Miniature robotic guidance for spine surgery--introduction of a novel system and analysis of challenges encountered during the clinical development phase at two spine centres. Int J Med Robot. 2006;2(2):146–53.
85. van Dijk JD, et al. Clinical pedicle screw accuracy and deviation from planning in robot-guided spine surgery: robot-guided pedicle screw accuracy. Spine (Phila Pa 1976). 2015;40(17):E986–91.
86. Huntsman KT, et al. Robotic-assisted navigated minimally invasive pedicle screw placement in the first 100 cases at a single institution. J Robot Surg. 2020;14(1):199–203.
87. Menger RP, et al. A cost-effectiveness analysis of the integration of robotic spine technology in spine surgery. Neurospine. 2018;15(3):216–24.
88. Fiani B, et al. Impact of robot-assisted spine surgery on health care quality and neurosurgical economics: a systemic review. Neurosurg Rev. 2020;43(1):17–25.
89. Elmi-Terander A, et al. Pedicle screw placement using augmented reality surgical navigation with intraoperative 3D imaging: a first in-human prospective cohort study. Spine (Phila Pa 1976). 2019;44(7):517–25.
90. Luciano CJ, et al. Learning retention of thoracic pedicle screw placement using a high-resolution augmented reality simulator with haptic feedback. Neurosurgery. 2011;69(1 Suppl Operative):ons14–9; discussion ons19.

Cervical Spine Problems

21

Michael Van Hal

The cervical spine is a critical structure for both maintaining and protecting the structural integrity of the cervical spinal cord and nerve roots as well as providing the structural support for the head. The cervical spinal cord itself functions as an information superhighway that carries information to and from the arms, thorax, and lower body to the brain and back in a variety of complex efferent and afferent pathways which are beyond the scope of this chapter. These pathways can be disrupted in a variety of ways such as tumors, trauma, infections, medical diseases, toxins, or degenerative conditions. While tumor, trauma and infectious etiologies will be discussed in other chapters of this book, this chapter will deal with the variety of ways that this complex structure can develop degenerative and inflammatory disease and the surgical options to address these disorders.

We are going to look at a variety of ways the cervical spine degenerates and the clinical scenarios such as axial neck pain, disc herniation, spondylosis, and spondylotic myelopathy. We will briefly discuss the medical causes of myelopathy and myelitis that should be considered prior to surgical intervention. Then finally we will consider inflammatory conditions with special consideration for the pathology that is specific to patients with rheumatoid arthritis particularly at the upper cervical spine.

The distinction between normal aging and pathological conditions is blurred. There is not always a distinguishable level of degeneration that correlates with a pathological state versus the normal aging process. While individual discs can show degenerative changes if they are traumatized, more often discs degenerate more symmetrically which shares many of the same characteristics of aging discs [1]. Neck pain is nearly universal for all populations for short periods of time and spondylotic changes are nearly universal but the prevalence of these changes increases with increasing age [2]. There is relatively poor correlation between the severity of

M. Van Hal (✉)
Department of Orthopaedic Surgery, UT Southwestern Medical Center, Dallas, TX, USA
e-mail: Michael.Vanhal@UTSouthwestern.edu

© The Author(s), under exclusive license to Springer Nature Switzerland AG 2022

C. E. Noe (ed.), *Multidisciplinary Spine Care*,
https://doi.org/10.1007/978-3-031-04990-3_21

spondylotic changes on imaging with the degree of symptoms. Thus, both the art and science of medicine must be used to evaluate patients' complaints and imaging findings to correlate the two sources of information before intervention can be recommended.

While in the healthy state, the disc, which includes the nucleus pulposus and outer annulus fibrosis, functions in concert with the ligamentous complex in the posterior portion of the spine. This ligamentous complex is composed of multiple individual ligaments and the facet capsules which function in concert to create a tension band that restrains the kyphotic forces on the spine. This complex configuration provides stability with the bone structure to protect the neural structures within the spinal canal and to support loads placed upon the spine. The flexibility of the disc and ligaments allows motion to occur despite this need for stability and protection. However, this motion does make the spine susceptible to degenerative changes [3].

Even with "normal physiologic aging" the disc undergoes a variety of changes with a relative loss of water content [1]. The glycosaminoglycans (GAGs) begin to change in amount and type. The highly hydrophilic GAG aggrecan is lost and the subsequent water turgor pressure seen in the young healthy disc is lost [1].

The degeneration of the disc creates a posterior bulge. The healthy disc is normally higher anteriorly than posteriorly (lordotic alignment of the disc). However, when it degenerates it loses this lordotic alignment. A degenerative disc creates a relative kyphotic level which further exacerbates the posterior loading of the disc [4]. The loss of disc height can also cause buckling of the ligamentum flavum on the posterior aspect of the canal. This can lead to further narrowing of the canal in addition to the posterior disc bulging. There is an ensuing increase in the subsequent force that is placed on the endplates in the cervical spine. This increased physiological load leads to the formation of osteophytes in the body's attempt to distribute this increased load. This is seen especially at the facets, uncovertebral joints, and the margins of the disc. Osteophytes at any of these locations can lead to neurological symptoms if they irritate/impinge the neural elements leading to clinical symptoms characteristic of radiculopathy and myelopathy. This cascade can lead to a vicious cycle of disc degeneration, kyphotic alignment, facet loading and subsequent narrowing of the foramen and the more central aspects of the canal.

This simplified background is reviewed here to develop a framework of understanding the cause of degenerative changes in the neck and it is useful in educating patients. Two of the more common questions asked in practice: "How did this happen?" and "How do I slow or reverse it?". These questions are frustrating to clinicians as well since we are forced to admit that despite our understanding of the process, other than avoiding exacerbating factors, we do not know how to forestall or reverse this process.

Therefore, much of the excitement regarding regenerative medicine holds such promise in that it might be able to repair or reverse the process that leads to many of these degenerative clinical conditions. However, in its current state the scientific support of most regenerative medicine is poorly substantiated.

In such a background of disc degeneration and spondylosis, we will now consider axial neck pain, acute disc herniations, cervical spondylosis that can lead to radiculopathy and or myelopathy. We will consider these all from a surgical perspective. As we approach each clinical entity, we will consider their clinical presentation, initial management and surgical options and surgical approaches. Of note, every person's clinical presentation needs individual correlation. For example, disc herniations in a large, capacious canal may produce minimal symptoms other than neck pain, while a similarly sized disc herniation in a patient with congenital stenosis may be severely affected with myeloradiculopathy. Thus, each patient's care must be individualized within his or her entire clinical scenario.

Axial neck pain is a common clinical scenario, in fact the incidence is about 10% per year [5, 6]. Moreover, a majority of people will experience it at some point in time [2, 7]. While neck pain can be due to a multitude of causes, the majority are due to either muscular strains or ligament sprains [5]. However, there are many other causes that can lead to neck pain such as stress or referred pain from the temporomandibular joint or the shoulder. There is evidence that discs and facet joints themselves can be a source of pain via the sinuvertebral nerve and the dorsal primary rami respectively which can transmit pain [8]. Degenerative changes in these areas do appear to correlate with patient's pain albeit imperfectly and these areas can be targeted for pain alleviation. Yet, other confounding sources of pain must be properly considered and ruled out.

While axial neck pain is common, it is usually self-limited in its natural history and as such the mainstay of treatments is aimed at symptom control while the natural history takes its course. Most care includes a large amount of reassurance. There is often much fear that the pain will progressively worsen or that something serious is happening which can exacerbate the symptoms [9]. A minority of patients that do not improve should be more closely evaluated for subtle treatable causes of neck pain. Treating neck pain with an operation to fuse the cervical spine is extremely controversial and it has not been shown to be effective [10]. Furthermore, there are radicular patterns that can cause pain in the neck which will be addressed. These radicular type neck pain patterns form a subset of neck pain patients that may respond to surgical treatment of the radiculopathy that manifests primarily with neck pain, but these are a separate entity from a majority of true axial neck pain patients.

Non-surgical management of axial neck pain should include education which can be sufficiently therapeutic in some patients [11]. This has been more effective than most other treatments when combined with manual therapy and exercise. A short course of intermittent soft collar is frequently helpful in the short term. This allows the muscles to rest and reduces secondary neck muscle spasms without using pharmacological agents. Intermittent use is recommended to avoid muscle atrophy in the neck.

Additionally, a short trial of non-steroidal anti-inflammatory drugs should be considered as these have shown to have good efficacy in treating axial neck pain. Whether these medications change the natural history to shorten the course of an

acute axial neck pain episode is unclear. Regardless they do help effectively manage the pain and either the natural history or the medicine itself will help control the symptoms in a majority of cases. Muscle relaxants are another short-term medicinal option for treating neck pain. This muscular pain can either be primary or secondary to other causes of acute neck pain as the neck attempts to restrict movement. Oral steroids are another medical option but there is little clinical data for steroids although they are frequently used for acute neck pain and acute onset radiculopathy as well. Antidepressants or anticonvulsants are another option although their use in acute neck pain is poorly substantiated by any rigorous studies [12].

Physical therapy and gentle exercise and/or traction may be helpful for symptom control. While more effective than most other treatments it has poor evidence that it actually changes the natural history of acute axial type neck pain or for acute cervical radicular syndromes [11].

Cervical manipulation is frequently promoted as a treatment for neck pain. Patients frequently ask if its use is recommended or scientifically supported. Currently, there is no established science to support this treatment for neck pain despite multiple theories. Additionally, there are multiple reports of catastrophic cases where manipulation was performed [13–16]. Given the questionable benefits gained by this procedure, it is not recommended.

Steroid injections as a treatment for axial neck pain is dependent on the ability to localize a pain generator. These interventions may also have serious and even devastating complications. These are thankfully rare but serious ones can range from minor infections, dural punctures, epidural hematomas, to even spinal cord injuries [17, 18]. They do have potential therapeutic benefit although there is conflicting information in the literature [19]. Furthermore, they can potentially provide diagnostic benefit by being able to help confirm a pain generator. This can be especially true in diagnostically unclear situations such as multilevel degenerative disease or in the more cephalad level cervical disc herniations without more distinctly associated dermatomes. In an increasingly cost-conscious healthcare system and with increasing push for quality health care, steroid injections for neck and low back radiculopathy have come under increasing scrutiny. While there have been studies that these injections do not provide long term relief therapeutically, they still appear to be useful diagnostically [20].

Cervical disc herniations occur when the nucleus pulposus is no longer contained by the annulus fibrosis and it protrudes partially or fully through the annulus fibrosus. This can be due to chronic degenerative changes or acutely due to a traumatic event. There is some evidence that many times this may in fact be a spectrum of the same disease: degeneration and subsequent loads exceeding the degenerative disc's ability to handle such loads even in the physiological range or due to some level of trauma—a supraphysiologic load. Recently there is even some thought that there may be a subclinical infection that weakens discs and makes them susceptible to herniations and degenerative changes [21]. Regardless of the precise precipitating event, the acute nature of the response sets off a standard inflammatory response with pro-inflammatory cytokines and phagocytic cell recruitment to the disc herniation site.

An acute herniated disc usually has a relatively benign natural history much like that of axial neck pain episodes. Given the benign natural history, it can usually be managed non-operatively either entirely or initially [19]. Much of the initial management for the first 6 weeks is similar to that described previously for axial neck pain treatment. However, the differences lie in how severe the symptoms are. If significant radicular symptoms such as weakness are noted, then more aggressive intervention may be necessary. Epidural steroid injections or selective nerve root blocks have not been definitively shown to change the natural history of acute cervical radiculopathy, though they may make the natural history more bearable [22]. They may be able to avoid surgery in a small percentage of cases, but this is controversial [23]. While acute soft disc herniations usually have a benign natural history, those that become chronic can develop "hard discs" which become calcified and osteophytes which are likely less amenable to injections. Surgical intervention for radiculopathy is usually reserved for refractory symptoms, progressive symptoms, or significant disability (such as significant motor weakness). For those patients that fail conservative treatment, surgery is typically very effective in preserving function and restoring function and those benefits are maintained long-term [24, 25].

Cervical radiculopathy as a clinical entity is fairly straightforward. Each nerve in the cervical spine C5 through T1 has a characteristic dermatome, myotome. Furthermore, C5, C6, C7 and C8 all have reflexes that can be used to help identify which nerve is affected. (See Table 21.1 Cervical myotomes, dermatomes, and reflexes).

The nerve root irritation can cause depolarization and irritation of the nerve which can cause pain, numbness, tingling, paresthesia in the distribution of the nerve. Diagnosing radiculopathy in nerve roots above C5 is more challenging since these nerve roots do not have a characteristic myotome and there is more overlap in the dermatomes to include the axial neck region. C2 radiculopathy can present as an occipital headache or numbness, while C3 radiculopathy can appear as subaxial pain in the neck. When C4 radiculopathy is present, the neck pain is typically located more distally. Thus, while C3 and C4 do not have a specific myotome, radiculopathy from either level can be misdiagnosed as axial neck pain.

Table 21.1 Cervical nerve roots and their respective dermatomes, myotomes, and reflexes if present

Cervical nerve root	Cervical dermatomes	Cervical myotomes (Primary ones)	Reflex (If present)
C2	Occipital	None	None
C3	Upper cervical neck	None	None
C4	Mid neck	None	None
C5	Upper neck to shoulder/ lateral arm	Deltoid/biceps	Biceps
C6	Lateral forearm	Wrist extension	Brachioradialis
C7	Middle finger	Triceps	Triceps
C8	Ring/small finger	Finger flexion	None
T1	Ulnar forearm	Finger abduction	None

One useful way to distinguish cervical radiculopathy from non-specific axial neck pain is nerve tension signs. The shoulder abduction test is one such test which is positive when the patient's pain is relieved by raising the arm over the head. This decreases the tension on the nerve from the weight of the arm itself. This improvement in symptoms is usually the opposite pattern from shoulder pathology which can mimic pain in the neck. However, shoulder pathology pain is usually exacerbated when lifting the arm. While this can be seen in cervical radiculopathy at all levels in the cervical spine, it is especially useful for C3 and C4 radiculopathies given the relatively generic symptoms and non-specific findings associated with these particular nerve roots.

Electromyography and nerve conduction studies are frequently not helpful diagnostically in these upper cervical spine radiculopathy scenarios since there is not a specific myotome and there is no peripheral nerve easily accessed. However, diagnostic shots can be very helpful in addition to a thorough history and excellent physical examination. This is especially true in the multidisciplinary spine center setting which fosters close association with non-operative spine specialists. These specialists can provide an accurate localization of the pain source. Clear communication as well as the ability to review the imaging of the injection's location can help increase the information available to surgeons. This can be especially useful for counseling patients. Utilizing providers within one system and maintaining very subspecialized roles within that system increases accuracy and communication and facilitates patient outcomes and fewer fusions [26]. Furthermore, higher volume centers and high-volume surgeons appear to be associated with better outcomes [27, 28]. Muscle testing for each nerve root myotome is challenging with significant overlap between muscle groups even in the lower myotome-associated levels of the cervical spine. Additionally, there are frequent anatomic variations which can further cloud the diagnostic accuracy. For the purpose of training, testing, and evaluations, the ASIA worksheet shows classic myotomes and dermatomes. This can be seen in Fig. 21.1.

Unfortunately, non-operative care is usually compared to operative interventions, but the comparison is rarely homogenous. The non-operative care can vary widely between non-operative patients.

The surgical options for an acute disc herniation causing radicular findings include one of three standard surgical approaches: foraminotomy with or without disc fragmentectomy, complete discectomy and fusion, and complete discectomy and an artificial disc replacement. Unlike disc herniations in the lumbar spine, in which the surgeon can mobilize the cauda equina nerve roots to access the disc herniation in the canal, the cervical spinal cord cannot be mobilized and as such the disc herniation must be delicately handled to ensure no spinal cord damage is inflicted while mobilizing/removing the herniated portion. This leaves two relative approaches: either anterior with removal of the offending pathology or a posterior approach that accesses the disc and nerve root lateral to the spinal cord. If the disc herniation is lateral to the spinal cord, then mobilization of the nerve root alone and foraminotomy of the cervical nerve root is reasonable and should yield excellent results in a safe manner [29]. However, more often the disc herniation is not

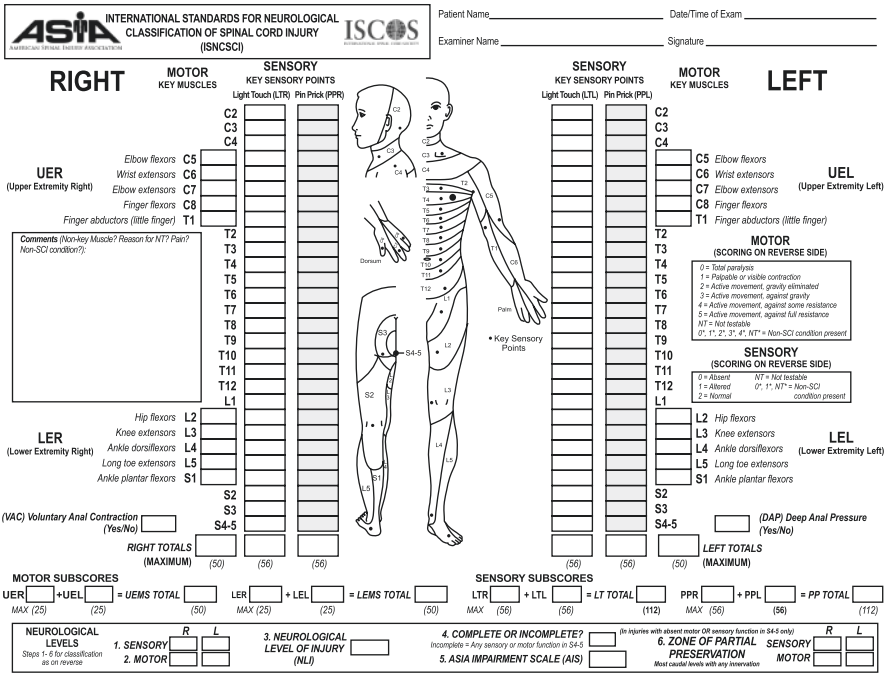


Fig. 21.1 American Spinal Injury Association Worksheet for Key Muscle and Sensory Points

localized to only the lateral aspect of the canal and the disc is not just in the foramina but it is more broadly based with both more central canal and lateral portion. If the disc herniation is in the lateral aspect of the canal and causes stenosis of only the nerve root and the neuroforamen then mobilization of the nerve root and subsequent discectomy is not only less technically difficult but also less risky to the patient. Also compared to anterior cervical discectomy and fusion, posterior foraminotomy preserves a motion segment which can be especially advantageous in the young, non-spondylotic patient.

Anterior approach surgery for the cervical spine is a great option as it is much less disruptive since it can utilize the normal tissue planes with just manipulation of potential spaces. This allows the patient to have a less painful recovery and usually a lower wound complication rate [30]. However, violating the disc from the front requires removing the entire disc. Violating the disc even with a needle leads to a progressive disc degeneration [31]. As such, replacing the disc with material such as bone for fusion or an artificial disc replacement is the standard of care. There have been discectomies from the front without fusion or disc replacement but this is not done frequently due to return of symptoms and development of local kyphosis although this is controversial [32].

While anterior cervical discectomies and subsequent fusions are generally a very well tolerated procedure and the outcomes are excellent, the concern for adjacent segment degeneration, and preservation of motion prompted the development of

cervical disc replacement (CDR) as a surgical option. This has proven to yield excellent results. While CDR is a relatively newer technology, the long-term results to date for appropriately selected patients reveal very encouraging outcomes [33, 34]. The amount of degenerative changes and facet arthropathy that is amenable to disc replacement is still controversial. Currently, significant degenerative changes in the cervical spine are a relative contraindication for the use of a cervical disc replacement.

While a disc herniation can occur in the relatively young and healthy appearing spine, the aging spine frequently shows spondylosis at multiple levels. This cervical spondylosis can also lead to pain and dysfunction in the axial spine. Surgical treatment of axial neck pain as stated before is controversial as it is frequently difficult to find a localized source of the pain.

However, the degenerative changes may progress and may lead to a decreased cross-sectional area in the canal or the foramen leading to distinct clinical syndromes: cervical myelopathy or cervical radiculopathy respectively. These usually have a different natural history than the acute soft disc herniation which is more frequently benign and more commonly associated with radiculopathy primarily.

When spondylotic changes narrow the foramen, this narrowing may subsequently lead to radicular symptoms if the nerve is impinged. However, these symptoms present similarly to those of an acute disc herniations in the neck. However, these spondylotic radicular patients often have a more insidious onset of symptoms with long standing neck pain that can slowly progress to persistent arm symptoms as well. These patients are successfully treated with an anterior discectomy and fusion similar to the surgery for a disc herniation and foraminal narrowing around the nerve. While posterior foraminotomies are an option, given the spondylotic changes that usually accompany the uncovertebral joints, and the posterior facet hypertrophy, a foraminotomy alone can be difficult to fully address the foraminal stenosis. A complete discectomy anteriorly can remove not only the disc but also resect the osteophytes from the uncovertebral joint which are a frequent source of compression for the exiting nerve roots. Additionally, the fusion can help to alleviate motion through the facet indirectly alleviating the arthritic pain preventing the development of further facet hypertrophy that can cause nerve root compression and re-stenosis. Finally, placing a solid material in the disc space can help indirectly open the foramen by restoring the disc space height to normal. This allows the foramen to enlarge in the superior/inferior dimension which leads to indirect decompression. As stated previously, cervical disc replacements are controversial when the degenerative changes become more extensive, but this is an evolving opinion since it is a relatively new technology.

Facet arthropathy is frequently seen in a degenerative cervical spine as well. While facet arthropathy can be a frequent source of pain, facet arthropathy alone, is not a reason to perform surgery as these patients have inconsistent and variable results with surgical intervention, namely, cervical fusions. Cervical fusions are controversial in the absence of radicular or myelopathic clinical features. Fusions to treat axial neck pain even in the setting of severe spondylolitc changes is extremely controversial as the outcomes are less reliable [35].

Rarely however, the arthritic cervical facet joint can develop facet cysts. These are due to degeneration of the facet joints and a relative weakness in the facet capsule. This allows the development of a cyst adjacent to the facet joint. These are relatively rare but given the variability in the size of the cyst, they can cause a variety of symptoms. Treatment for these is usually conservative to a point but then surgery can be very successful to remove the cyst. Some people argue these should be fused to prevent recurrence of the cyst by removing the joint motion and thus the causative agent of the cyst creation. However, this is controversial. These cysts are rare enough that the best treatment and natural history is still as yet unclear [36]. One option is to remove the cyst, which would require a posterior approach given the location of the cyst originating from the facet which is clearly posterior to the spinal cord. However, one option is to not resect the cyst and simply fuse it anteriorly. This does not directly remove the cyst, but it can stop the motion at the facet and allow the facet cyst to reabsorb over time with the lack of motion at this level, which has been shown in the lumbar spine [37].

As spondylotic changes progress, the central canal may be compromised leading to compression and dysfunction of the spinal cord itself and not just the exiting nerves.

Cervical spondylotic myelopathy as a clinical scenario is most often considered a surgical diagnosis. While there have been some studies that show some mild cases do not progress, the majority do. However, there are no clear natural history studies that clearly demonstrate the course without intervention. There is a wide variation in the clinical presentation, as well as the rate of progression. In a majority of cases, there is relatively insidious onset of hand clumsiness which is often attributed to comorbidities such as arthritis of the hands. As the myelopathy progresses it can lead to more significant upper motor neuron dysfunction such as weakness, ataxia, clumsiness that progresses to weakness, hand atrophy, and even lower extremity symptoms as well as bowel and bladder dysfunction. In a progressive, untreated case, the patient frequently goes from walking unassisted, to walking with assistive devices, and then to being wheelchair dependent. While the pathology can be addressed at any point, doing it when dysfunction is mild is highly recommended [38, 39]. Cervical spine surgery is much more predictable at preserving function of the spinal cord than restoring it.

When a patient with spondylolytic changes also has symptoms and signs of myelopathy, it is important to consider and rule out other mimickers of cervical spondylotic myelopathy (CSM). This includes other medical causes of myelopathy such as cerebral dysfunction like Parkinson's disease, cerebellar dysfunction, and cerebrovascular accidents. The other mimickers of cervical myelopathy due to spondylosis can be intrinsic spinal cord dysfunction. Examples of these include: amyotrophic lateral sclerosis (ALS), multiple sclerosis, (MS) and subacute combined degeneration of the spinal cord (B12 deficiency). While the differential can be extensive, these can usually be ruled out with a thorough history. Probably the most common mimicker of CSM would be MS or transverse myelitis. In fact, transverse myelitis can be the initial symptom in MS in many cases (13%) [40]. Amyotrophic lateral sclerosis characteristically affects the anterior horn cells. As such it is

sensory sparing but can show mixed upper and lower motor neuron signs which can make it similar to CSM.

Cervical spondylotic myelopathy (CSM) also may have mixed upper and lower motor neuron signs. Furthermore, the physical examination frequently shows upper motor neuron signs that localize to the cervical spine. These include but are not limited to the Hoffman sign, Babinski sign, and inverted brachioradialis reflex. Of note, the mandibular reflex should not be present and if it is then it would argue that there may be an intra-cranial pathology that is causing upper motor neuron signs.

In general, the surgical goal for to CSM) patients is to decompress the canal. A posterior laminectomy/laminoplasty will allow the cervical spinal canal to increase in diameter and allow the spinal cord to expand and thus hopefully recover some function or at least halt the progression of the dysfunction. A posterior approach for laminectomy or laminoplasty does require a neutral or lordotic alignment of the spine. This can be an issue when the spondylotic changes are extensive which have a tendency to create either locally kyphotic segments or a globally kyphotic cervical spine. This is due to degeneration of the disc, which is normally higher anteriorly than posteriorly, which when it degenerates it shortens, effectively causing kyphosis the cervical spine. If this is the case, then anterior and posterior approaches to restore the alignment anteriorly and improve the canal diameter posteriorly can be utilized for a complete spine reconstruction. While the canal could be fully decompressed from the front with a corpectomy (removal of the cervical vertebral body), generally this requires stabilization from a posterior approach as well if it is more than a single level corpectomy [3, 41]. Corpectomies can be necessary for severely kyphotic necks or for severely damaged vertebral bodies or if the pathology is posterior to the vertebral bodies such as in ossification of the posterior longitudinal ligament.

However, generally if the pathology is extensive and the alignment is neutral or lordotic, a posterior laminectomy or laminoplasty is the preferred approach to make more room for the spinal cord. Except in extremely low demand patients or near ankylosed spines, a fusion is generally added to laminectomies to enhance stability and prevent post-laminectomy kyphosis which can occur in about 20% [42]. Posterior cervical fusions have been performed by getting the posterior facets to fuse. This can be accomplished by bone grafting into the facet joints. Furthermore, the joints can be stabilized with posterior lateral mass screws. These implants, while very useful for stabilizing the lateral masses, do add significantly to the overall surgical cost.

Determining the best posterior approach option between laminectomy and fusion versus a laminoplasty is controversial. In general, both achieve a larger canal. Laminoplasty has the advantage of decreased operating time and costs. Laminoplasty does preserve motion but this may be associated with increased neck pain. So, significant preoperative neck pain is a relative contraindication to laminoplasty. A fusion does require longer operative times, more blood loss, higher implant cost, but it may be associated with less post-operative neck pain. More extensive foraminotomies can be performed given the increased stability with the fusion rather than a laminoplasty.

Another disease that can lead to cervical myelopathy which is not necessarily associated with spondylotic changes include the entity of ossified posterior longitudinal ligament (OPLL). This is a clinical syndrome which was first described in the Japanese population where it can be found in approximately 2–4% of the population [43]. However, the exact disease mechanism is still not completely understood. There appears to be a significant genetic component although it is multifactorial in its development [43]. Laminoplasty was originally developed to address OPLL since it is seen more frequently in younger patients. A posterior approach for OPLL spines with myelopathy is usually recommended due to the higher chance of a durotomy with anterior procedures. This anterior durotomy can be extremely challenging to repair. Thus, posterior approaches are usually preferred to avoid this potential complication. However, the OPLL is still present in cases of posterior approaches. Thus, there is the potential for continued growth of the OPLL post operatively. While this OPLL progression may be seen regardless of the surgical approach, it may be lower in fusion cases [44].

Other diseases besides spondylosis (degenerative arthrosis) can affect the cervical spine. Inflammatory conditions may also affect the cervical spine and lead to instability and/or neurological compromise. A complete discussion of the pathogenesis, patterns and treatments of these is well beyond this chapter's scope as these are whole body diagnoses that also affect the cervical spine. A quintessential example would be rheumatoid arthritis (RA) as many as 80% of these patients have some involvement of the cervical spine [45].

There are other conditions such as psoriatic arthritis, reactive arthritis, ankylosing spondylitis and diffuse idiopathic skeletal hyperostosis (DISH) all of which can affect the cervical spine. Ankylosing spondylitis and DISH are important for cervical spine disease as they can have progressive deformities and can fracture more easily. However, they will be discussed in the trauma and deformity chapters in more detail.

Ankylosing spondylitis patients can have C1–2 joint erosion with synovial tissue around the odontoid. This erosion around the odontoid can erode and create instability at the atlas and the odontoid. In addition to this, the dens can migrate into the foramen magnum and may require stabilization of the occiput and cervical spine to prevent progressive migration/deformity and cervical spine instability. Rather than discuss how each individual disease can affect the cervical spine, such as ankylosing spondylitis. We will focus on one disease, rheumatoid arthritis, as a prototype of these diseases.

Rheumatoid patients frequently have progressive joint destruction and inflammatory conditions that can lead to cervical spine joint destruction, cervical spine instability, and neurological compromise.

Since rheumatoid arthritis is an inflammatory condition, it can affect nearly any synovial joint. As such, even joints that typically are not commonly affected as severely by degenerative changes can be involved. This includes the atlantoaxial articulation at C1–2, occipital cervical settling with subsequent basilar invagination, and finally the subaxial spine degenerative changes and subluxation.

A complete review of the pathophysiology of rheumatoid arthritis is beyond the scope of this chapter. However, a brief review of the changes that occur is useful to design treatments aimed at specific pathologies encountered in the rheumatic patient. Rheumatoid arthritis is due to the immune system that becomes activated against the host's own tissues. This is usually seen early with attacks on the soft tissues but in later stages can advance to involve both cartilage and bone. There is a pathoanatomical progression of antibodies and antigen-antibody reactions which can be seen in rheumatoid factor in the blood. This is an IgM antibody against an IgG antibody. These antibody complexes can be deposited in tissues and contribute to end-organ dysfunction. These antibody complexes can then cause microvascular proliferation and obstruction, which may lead to synovial pannus formation and intimal hyperplasia seen on histological sections [46]. This pannus formation can lead to joint subluxation from destruction of the ligaments and subsequent deformities. Since the advent of disease modifying antirheumatic drugs (DMARDs), the severity of rheumatoid disease progression has greatly decreased and the subsequent deformities greatly reduced as the deformities are associated with more severe disease [45, 46]. However, deformities are still present in those patients who do not respond to these drugs or those patients who cannot tolerate these drugs due to side effects, or those patients who do not have access to the DMARDs. Fortunately, these deformities occur at a lower incidence than in the past.

The C1–2 articulation is a complex interaction of three joints: the paired facet joints and the pivot type joint with the anterior articulation of the odontoid/dens of C2 and anterior arch of C1 which is held in proximity chiefly by the transverse ligament of C1. Rheumatoid patients can develop erosion of this transverse ligament of C1 and thus develop C1–2 instability. This can be seen on flexion and extension radiographs such that if the interval between the odontoid and the anterior atlas ring known as the AADI (atlanto-dens interval) is greater than 10 mm surgery is indicated even without neurological symptoms. If neurological symptoms are present, then surgery to stabilize/fuse this level is indicated.

The options to stabilize the AADI are limited. In the short term a halo can be used, but it is not a long-term solution. While a cervical collar can be useful in immobilizing the subaxial spine, the upper cervical spine is not well immobilized with just a cervical collar.

Surgical options are usually a fusion operation. Historically wiring of C1–C2 was the primary method to achieve a fusion. However, screw fixation has largely replaced wiring. This is mostly often accomplished with C1 lateral mass screws and C2 pars or pedicle screws attached with posterior rods (Harms technique) [47]. There is another option to pass a single screw from the inferior portion of C2 into C1 lateral mass, across the C1–C2 facet joint. This is dependent on vertebral artery anatomy and up to one out of five patients may not be a candidate for this transarticular screw due to an inadequate corridor [48].

Rheumatoid necks can also have progressive superior migration of the odontoid due to erosion and bone loss from C1 and C2. There are a variety of measurements that predict impending neurological impairment. However, surgical indications include progressive neurological compromise or progressive cranial migration of

the dens. This superior migration of the odontoid is typically treated with an occipital cervical fusion usually to C2. Often there is such advanced erosion that the subluxed C1–2 is fixed and a cervical decompression of the posterior arch of C1 is also often indicated to decompress the subluxed and secondarily narrowed canal.

While subaxial subluxations do occur, these are treated more similarly to anterior or posterior fusions described previously. Four or more millimeters of dynamic subluxation is considered unstable, and stabilization with either anterior or posterior fusion is likely indicated. Anterior versus posterior approach is chosen based on the severity of the compression as well as patient factors, such a prior surgery and overall cervical alignment. Fusions are generally recommended due to the progressive bone loss and joint destruction in these rheumatoid cases.

In conclusion, surgery for cervical spine pathology has many different approaches and options. Like many surgical options for the spine the outcomes are directly related to the indication for the surgery as well as the patient's overall health. While the surgical technique itself is vital, the patient's overall health both mentally and physically are also critically important. Choosing the right patient, and the right indication and the right approach is a delicate balance of the art and science of surgery which can lead to excellent clinical outcomes when employed skillfully.

References

1. Vo NV, et al. Molecular mechanisms of biological aging in intervertebral discs. J Orthop Res. 2016;34(8):1289–306.
2. Rao R. Neck pain, cervical radiculopathy, and cervical myelopathy: pathophysiology, natural history, and clinical evaluation. J Bone Joint Surg Am. 2002;84(10):1872–81.
3. White AA 3rd, et al. Biomechanical analysis of clinical stability in the cervical spine. Clin Orthop Relat Res. 1975;109:85–96.
4. Ferrara LA. The biomechanics of cervical spondylosis. Adv Orthop. 2012;2012:1–5.
5. Binder AI. Neck pain. BMJ Clin Evid. 2008;2008:1103.
6. Cote P, Cassidy JD, Carroll L. The Saskatchewan Health and Back Pain Survey. The prevalence of neck pain and related disability in Saskatchewan adults. Spine (Phila Pa 1976). 1998;23(15):1689–98.
7. Cohen SP. Epidemiology, diagnosis, and treatment of neck pain. Mayo Clin Proc. 2015;90(2):284–99.
8. Cloward RB. The clinical significance of the sinu-vertebral nerve of the cervical spine in relation to the cervical disk syndrome. J Neurol Neurosurg Psychiatry. 1960;23:321–6.
9. Vlaeyen JW, Linton SJ. Fear-avoidance model of chronic musculoskeletal pain: 12 years on. Pain. 2012;153(6):1144–7.
10. Carragee EJ, et al. Treatment of neck pain: injections and surgical interventions: results of the Bone and Joint Decade 2000–2010 Task Force on Neck Pain and Its Associated Disorders. J Manipulative Physiol Ther. 2009;32(2 Suppl):S176–93.
11. Hurwitz EL, et al. Treatment of neck pain: noninvasive interventions: results of the Bone and Joint Decade 2000–2010 Task Force on Neck Pain and Its Associated Disorders. J Manipulative Physiol Ther. 2009;32(2 Suppl):S141–75.
12. Kremer M, et al. The antiallodynic action of pregabalin in neuropathic pain is independent from the opioid system. Mol Pain. 2016;12:1744806916633477.
13. Biller J, et al. Cervical arterial dissections and association with cervical manipulative therapy: a statement for healthcare professionals from the American Heart Association/American Stroke Association. Stroke. 2014;45(10):3155–74.

14. Gross AR, et al. A Cochrane review of manipulation and mobilization for mechanical neck disorders. Spine (Phila Pa 1976). 2004;29(14):1541–8.
15. Stevinson C, Ernst E. Risks associated with spinal manipulation. Am J Med. 2002;112(7):566–71.
16. Jeret JS, Bluth M. Stroke following chiropractic manipulation. Report of 3 cases and review of the literature. Cerebrovasc Dis. 2002;13(3):210–3.
17. Abbasi A, et al. Complications of interlaminar cervical epidural steroid injections: a review of the literature. Spine (Phila Pa 1976). 2007;32(19):2144–51.
18. Epstein NE. Major risks and complications of cervical epidural steroid injections: an updated review. Surg Neurol Int. 2018;9:86.
19. Bono CM, et al. An evidence-based clinical guideline for the diagnosis and treatment of cervical radiculopathy from degenerative disorders. Spine J. 2011;11(1):64–72.
20. Sasso RC, et al. Selective nerve root injections can predict surgical outcome for lumbar and cervical radiculopathy: comparison to magnetic resonance imaging. J Spinal Disord Tech. 2005;18(6):471–8.
21. Shan Z, et al. The influence of direct inoculation of propionibacterium acnes on modic changes in the spine: evidence from a rabbit model. J Bone Joint Surg Am. 2017;99(6):472–81.
22. Manchikanti L, et al. Do cervical epidural injections provide long-term relief in neck and upper extremity pain? A systematic review. Pain Physician. 2015;18(1):39–60.
23. Bicket MC, et al. Epidural injections in prevention of surgery for spinal pain: systematic review and meta-analysis of randomized controlled trials. Spine J. 2015;15(2):348–62.
24. Buttermann GR. Anterior cervical discectomy and fusion outcomes over 10 years: a prospective study. Spine (Phila Pa 1976). 2018;43(3):207–14.
25. Yue WM, Brodner W, Highland TR. Long-term results after anterior cervical discectomy and fusion with allograft and plating: a 5- to 11-year radiologic and clinical follow-up study. Spine (Phila Pa 1976). 2005;30(19):2138–44.
26. Yanamadala V, et al. Multidisciplinary evaluation leads to the decreased utilization of lumbar spine fusion: an observational cohort pilot study. Spine (Phila Pa 1976). 2017;42(17):E1016–23.
27. Adkins ZB, et al. Does hospital volume affect outcomes in spine surgeries? A systematic review. Clin Spine Surg. 2019;32(7):285–94.
28. Malik AT, et al. The impact of surgeon volume on patient outcome in spine surgery: a systematic review. Eur Spine J. 2018;27(3):530–42.
29. Sahai N, et al. Minimally invasive posterior cervical foraminotomy as an alternative to anterior cervical discectomy and fusion for unilateral cervical radiculopathy: a systematic review and meta-analysis. Spine (Phila Pa 1976). 2019;44(24):1731–9.
30. Fang W, et al. Anterior cervical discectomy and fusion versus posterior cervical foraminotomy for the treatment of single-level unilateral cervical radiculopathy: a meta-analysis. J Orthop Surg Res. 2020;15(1):202.
31. Nassr A, et al. Does incorrect level needle localization during anterior cervical discectomy and fusion lead to accelerated disc degeneration? Spine (Phila Pa 1976). 2009;34(2):189–92.
32. Xie JC, Hurlbert RJ. Discectomy versus discectomy with fusion versus discectomy with fusion and instrumentation: a prospective randomized study. Neurosurgery. 2007;61(1):107–16; discussion 116–7.
33. Radcliff K, et al. Long-term evaluation of cervical disc arthroplasty with the Mobi-C(c) cervical disc: a randomized, prospective, multicenter clinical trial with seven-year follow-up. Int J Spine Surg. 2017;11:31.
34. Zou S, et al. Anterior cervical discectomy and fusion (ACDF) versus cervical disc arthroplasty (CDA) for two contiguous levels cervical disc degenerative disease: a meta-analysis of randomized controlled trials. Eur Spine J. 2017;26(4):985–97.
35. Riew KD, Ecker E, Dettori JR. Anterior cervical discectomy and fusion for the management of axial neck pain in the absence of radiculopathy or myelopathy. Evid Based Spine Care J. 2010;1(3):45–50.
36. Costa F, et al. Cervical synovial cyst: case report and review of literature. Eur Spine J. 2010;19(Suppl 2):S100–2.

37. Massey GM, et al. Lumbar facet cyst resolution following anterior interbody fusion. J Clin Neurosci. 2013;20(12):1771–3.
38. Fehlings MG, et al. Efficacy and safety of surgical decompression in patients with cervical spondylotic myelopathy: results of the AOSpine North America prospective multi-center study. J Bone Joint Surg Am. 2013;95(18):1651–8.
39. Lindsey RW. Surgery for cervical spondylotic myelopathy: right evidence or evidence right now?: commentary on an article by Michael G. Fehlings, MD, PhD, et al.: "Efficacy and safety of surgical decompression in patients with cervical spondylotic myelopathy. Results of the AOSpine North America prospective multi-center study". J Bone Joint Surg Am. 2013;95(18):e138.
40. Cobo Calvo A, et al. Idiopathic acute transverse myelitis: outcome and conversion to multiple sclerosis in a large series. BMC Neurol. 2013;13:135.
41. Bayerl SH, et al. Two-level cervical corpectomy-long-term follow-up reveals the high rate of material failure in patients, who received an anterior approach only. Neurosurg Rev. 2019;42(2):511–8.
42. Kaptain GJ, et al. Incidence and outcome of kyphotic deformity following laminectomy for cervical spondylotic myelopathy. J Neurosurg. 2000;93(2 Suppl):199–204.
43. Stapleton CJ, et al. Ossification of the posterior longitudinal ligament: genetics and pathophysiology. Neurosurg Focus. 2011;30(3):E6.
44. Lee CH, et al. Expansive laminoplasty versus laminectomy alone versus laminectomy and fusion for cervical ossification of the posterior longitudinal ligament: is there a difference in the clinical outcome and sagittal alignment? Clin Spine Surg. 2016;29(1):E9–15.
45. Gillick JL, Wainwright J, Das K. Rheumatoid arthritis and the cervical spine: a review on the role of surgery. Int J Rheumatol. 2015;2015:252456.
46. Guo Q, et al. Rheumatoid arthritis: pathological mechanisms and modern pharmacologic therapies. Bone Res. 2018;6:15.
47. Harms J, Melcher RP. Posterior C1–C2 fusion with polyaxial screw and rod fixation. Spine (Phila Pa 1976). 2001;26(22):2467–71.
48. Paramore CG, Dickman CA, Sonntag VK. The anatomical suitability of the C1–2 complex for transarticular screw fixation. J Neurosurg. 1996;85(2):221–4.

Thoracic Degenerative Conditions

22

Ammar Adenwalla, Olatunde Badejo, Kylan Larsen, Nisha Reddy, Neha Mulpuri, and Shaleen Vira

Introduction

Thoracic degenerative disease refers to conditions that result in a loss of normal structure and function of the thoracic spine. The use of the term degenerative indicates that the pathological basis for these conditions typically arises from age-related deterioration and is generally not due to trauma, malignancy, congenital deformity, or an infectious process. Although degenerative conditions of the thoracic spine are less common than cervical or lumbar degenerative conditions, they still represent a significant cause of back pain, leg pain, sensorimotor deficit, and bowel or bladder dysfunction. First-line treatment for these conditions is conservative management, but surgical management is indicated if symptoms continue to persist or worsen. The unique anatomical and biomechanical features of the thoracic spine affect surgical treatment strategies of thoracic degenerative diseases.

Anatomy and Biomechanics of Thoracic Spine

The human spine is classically divided into cervical, thoracic, lumbar, sacral, and coccygeal sections. Each region of the spine has unique defining characteristics, however, there is a gradual transition within the spine as the anatomy adjusts to meet the biomechanical requirements of each section. The Thoracic spine consists of 12 vertebrae numbered T1–T12 and serves as a rigid stabilizing zone between the more mobile cervical and lumbar divisions. The shared features of most vertebrae are also present in the thoracic spine, with each vertebra containing a vertebral body,

A. Adenwalla · O. Badejo · K. Larsen · N. Reddy · N. Mulpuri · S. Vira (✉)
Department of Orthopaedic Surgery, UT Southwestern Medical Center, Dallas, TX, USA
e-mail: ammar.adenwalla@utsouthwestern.edu; olatunde.badejo@utsouthwestern.edu; kylan.larsen@utsouthwestern.edu; neha.mulpuri@utsouthwestern.edu; shaleen.vira@utsouthwestern.edu

© The Author(s), under exclusive license to Springer Nature Switzerland AG 2022

C. E. Noe (ed.), *Multidisciplinary Spine Care*,
https://doi.org/10.1007/978-3-031-04990-3_22

spinous process, lamina, pedicle, and articular processes. Unique characteristics of these shared features in the thoracic spine include a changing morphology of the spinous processes as they progress caudally and increasing surface area of the vertebral bodies to account for an increased load as more body weight is added caudally.

A key distinguishing feature of thoracic vertebral anatomy is the presence of costal facets where the ribs articulate with the vertebrae. The ribs extend to the ventral portion of the body where they articulate with the sternum, either directly or indirectly via the costal cartilages. This closed ring adds a level of stability and rigidity to the thoracic spine that is not present in the other spinal sections. The existence of these articulations leads to the characteristic rigidity of the thoracic spine, contributing as much as 40% to the stability of the region [42]. The orientation of the facet joints in the thoracic spine also plays a role in the increased stability of the region. The facet joints in the thoracic spine are oriented in the coronal plane, while the cervical facet joints are more axially oriented, and the lumbar facet joints more sagittally oriented. Due to this orientation thoracic vertebrae are much less likely to dislocate without the presence of a fracture, and greater force is required to generate instability in the thoracic spine [28]. Consistent with the reduced flexibility of the thoracic region, the spinal canal also narrows considerably in the thoracic spine, reaching a cross-sectional area of 198 mm^2 at its narrowest compared to 280 mm^2 at its widest [30]. As the spine progresses through its different regions, there are key transitional areas where the biomechanical differences of the regions meet, and this leads to an increased likelihood of pathology. The cervical to thoracic transition zone as well as the thoracic to lumbar transition zone are areas where the difference in flexibility and stability of the thoracic spine contributes to an increased incidence of pathology.

An illustrative example of how an understanding of biomechanical and anatomical factors has influenced the treatment of thoracic degenerative conditions is how the surgical management of thoracic disc herniations has changed over time. Prior to the 1960s, a decompressive laminectomy was the treatment of choice for thoracic disc herniations [2, 6]. These surgeries frequently resulted in inadequate decompression, but no reasonable alternative existed. Studies have shown that decompressive procedures such as posterior laminectomies do not alter the biomechanical stability of the thoracic spine [13, 21]. The underlying reason for the failure of these posterior laminectomies relates to the anatomy of the thoracic region. Due to the narrowing of the spinal canal, surgeons found it extremely difficult to properly manipulate the spinal cord in the posterior approach [8], and thus morbidity and mortality from the procedure were substantial. Over time, novel approaches were utilized in the treatment of thoracic disc herniations, and the posterior laminectomy was abandoned for approaches that better considered the anatomic limitations of the region.

The unique anatomy and biomechanical properties of the thoracic spine have led to the creation of numerous surgical approaches and techniques to deal with pathologies arising in this region of the spine. The surgical management of thoracic degenerative conditions requires a critical understanding of anatomic and biomechanical principles in order to select the best approach and surgical technique to provide relief to the patient.

General Surgical Approaches to the Thoracic Spine

The surgical treatment of thoracic degenerative conditions requires the surgeon to have excellent knowledge of the relevant patient anatomy, pathology of the condition, and patient characteristics that may favor certain procedures and approaches over others (Fig. 22.1). Selection of the proper approach to correct the pathology is paramount to gaining adequate exposure and achieving a successful outcome. There are numerous surgical approaches to the thoracic spine, each with its benefits and drawbacks. The specific procedural details of each of the approaches described below are beyond the scope of this section, instead, a brief overview along with the advantages and disadvantages of each approach will be discussed.

Dorsal Approaches

Dorsal approaches to thoracic spine pathology represent a relatively straightforward option to gain access to the entire spinal column. These approaches utilize a midline incision to expose the paraspinal muscles, which are ultimately dissected away to gain visualization of the spinous processes and lamina. These bony landmarks may then be excised via a laminectomy, transpedicular approach, or other novel technique. Benefits

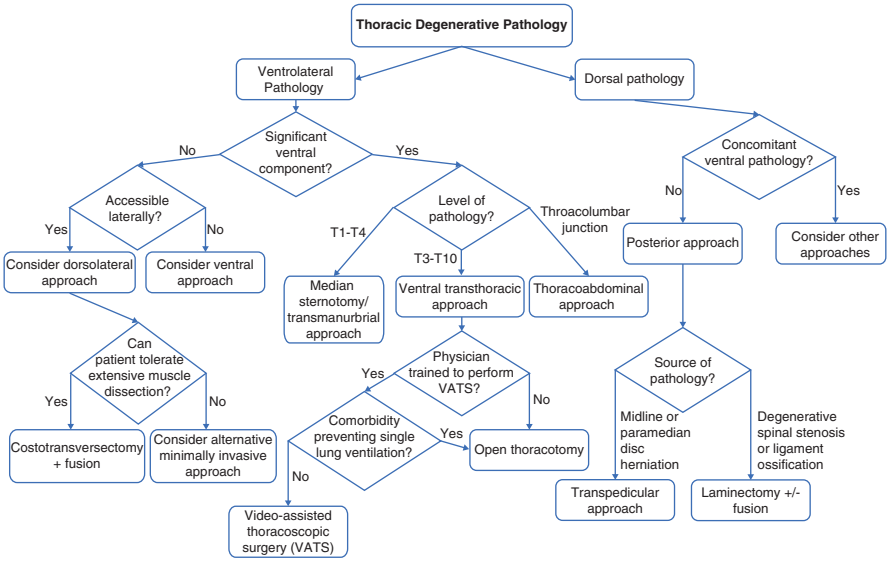


Fig. 22.1 Decision tree for selecting the appropriate surgical approach for managing thoracic degenerative pathologies. Several factors play a role in determining the most optimal surgical approach, including the location of the pathology, the comorbidities of the patient, and the technical skill of the surgeon. While this diagram provides a high-level overview of the decision-making process, every patient presents with a nuanced set of circumstances. Therefore, it is ultimately up to the physician to decide upon the most appropriate course of action on a case-by-case basis

of the dorsal approach include overall smaller operating times when compared to other approaches, the ease of performing concurrent instrumentation, and the ability to repair dural tears that may arise. Drawbacks to this approach include the previously mentioned issues with access to the pathology and the inability to manipulate the neural elements in the same manner as what is allowed in the lumbar spine.

Ventral Approaches

Ventral approaches are used for a wide array of thoracic spinal pathologies, including degenerative conditions, as these approaches allow for direct and exquisite visualization of the thoracic vertebral bodies. The specific ventral approach selected depends on the level of the thoracic spine involved. Pathologies at the cervicothoracic junction or high in the thoracic spine (T1–T3) are usually treated with a standard ventral incision with median sternotomy or a transmanubrial approach if the pathology extends to slightly lower levels (T4). Advantages of the ventral incision with median sternotomy or transmanubrial approach include direct visualization of the vertebral bodies, the ease of performing instrumentation, and the ability to gain ventral exposure of the dura via corpectomy. Disadvantages are related to the limited levels of exposure and potential damage to surrounding structures such as the recurrent laryngeal nerve. Another option to treat high thoracic pathologies is the transaxillary approach. As part of this approach the ipsilateral lung must be deflated, so patients with severely diminished respiratory capacity are not good candidates for the procedure. The other main drawback is the potential for damage to the sympathetic chain.

The ventral transthoracic approach utilizes a thoracotomy to gain access to a wide range of the thoracic spine (generally from T3 to T10). The thoracotomy can be performed on either side, and the incision should be placed as close to the level of pathology as possible. The thoracotomy can be performed with different techniques including the transpleural thoracotomy or the retropleural thoracotomy. The ventral transthoracic approach can be performed with rib resection or rib sparing techniques. Disadvantages of this approach include the need to deflate the lung ipsilateral to the incision, the need to retract the diaphragm in order to access lower thoracic levels, the potential for the development of a pneumothorax, pneumonia, or pleural effusion, and injury to the blood vessels that traverse the thorax in the indicated region.

For pathologies located at the junction of the lower thoracic and upper lumbar regions, the ventral thoracoabdominal approach can be utilized. This approach can be performed on either the right or left side, but the left side is generally preferred to avoid the liver and inferior vena cava. The disadvantages of this approach include having to enter the peritoneum, the potential development of diaphragmatic hernias, and damage to major blood vessels or the thoracic duct.

Dorsolateral Approaches

Dorsolateral approaches allow for visualization of the posterolateral aspects of the thoracic spine and easy visualization of lateral neural components of the

spinal cord. These approaches also have the benefit of avoiding entrance into the pleural or abdominal cavities. The costotranversectomy is an approach where the rib and portions of the transverse process of the vertebrae are resected in order to gain visualization of the pedicle and vertebral body. The main disadvantage of the costotranversectomy is that it requires extensive muscle dissection which can significantly prolong recovery times. Nevertheless, it is the workhorse of complex spine surgery in the thoracic spine with wide applications ranging from degenerative conditions to clinical scenarios involving complex deformity or tumors. The lateral extracavitary approach is a similar technique used in large deformity corrections or tumor care and is discussed in those chapters.

Minimally Invasive Surgery

Minimally invasive approaches aim to have the same or improved post-operative success rate as their open surgical counterparts while minimizing surgical exposure and tissue disruption. These approaches have been gaining popularity in spine surgery and are now used to treat pathologies at all levels of the spinal column. In the thoracic spine, one notable minimally invasive approach is the thoracoscopic assisted approach. In this multidisciplinary technique, a thoracic surgeon provides exposure of the thoracic cavity via an endoscope and a series of small incisions for instruments. From there the spinal column can be exposed and the pathology treated. The main benefit of this technique is the reduction of approach-related morbidity, as well as the ability to directly visualize and access the pathology with a high definition endoscope. The disadvantages include the need for single lung ventilation and the high level of technical skill required for mastery of the procedure. Previous literature has demonstrated that a substantial learning curve exists for spine surgeons in mastering minimally invasive techniques [9].

Overall, selection of the proper approach to surgically treat thoracic degenerative conditions depends on the thoracic levels involved, the laterality of the pathology, patient characteristics and operative history, and the desire for a minimally invasive or open technique. Knowledge of all the approaches utilized to treat pathologies of the thoracic spine is essential for the surgeon to be able to offer adequate surgical treatment for the wide range of thoracic degenerative conditions that may arise in patients.

Degenerative Diseases of the Thoracic Spine

Degenerative diseases that impact the thoracic spine can be broken down into three broad categories depending on the affected location: the intervertebral disc, the osseous canal, and the ligaments within the spinal canal. All of these degenerative changes eventually lead to spinal stenosis or compression of other neural elements, manifesting as a combination of pain, radiculopathy, or myelopathy.

Disc Degeneration and Herniation

Pathophysiology

Degeneration of the intervertebral discs gradually occurs with aging, and several factors contribute to it, including cell death and microenvironment changes [46]. Disc degeneration begins with subtle changes to the disc matrix as the balance shifts towards catabolic proteolytic activity [15]. Proinflammatory cytokine signaling mediates this matrix breakdown, and these cytokines as well as the byproducts of the breakdown lead to the perception of pain [15]. With moderate to advanced degeneration, the discs become stiffer and weaker due to fibrosis and cracks or tears develop [15]. Although there is reduced mechanical stress on the thoracic spine, it is still subject to compressive stress [40]. If there is disc herniation, patients may develop neurological symptoms associated with the compression of the spinal cord or nerve root by the disc. A majority of thoracic disk herniations occur between the T8 and L1 levels [41].

Epidemiology

A study of 90 asymptomatic individuals found that 73% had some degenerative anatomical changes in the thoracic spine, with 37% having disc herniation, 53% having bulging of a disc, and 58% having an annular tear based on magnetic resonance imaging [43]. Symptomatic degenerative disc disease is much less common in the thoracic spine compared to the cervical and lumbar regions, and it is estimated that thoracic disc herniation occurs in only one patient per million population per year [4, 41]. Symptomatic thoracic disc degeneration most frequently occurs between the fourth and sixth decades of life, and there is a slight male predominance [41].

Physical Exam Findings

There are three major manifestations of thoracic disc degeneration. The first is mild to moderate localized axial pain in the thoracic region, which is the most common presenting symptom [41]. The second constellation of symptoms involves radicular pain and is often described as a bandlike discomfort along a dermatomal distribution, and it most commonly occurs with upper thoracic lesions and lateral disc herniations [41]. The third manifestation is myelopathy, characterized by muscle weakness, mild paraparesis, abnormal gait, and bowel/bladder incontinence, and this presentation requires the most serious attention [41]. Palpating or percussing the thoracic spine can help to localize the pain and recreate radicular symptoms in order to elucidate the region where the disc herniation has occurred [41].

Imaging Findings

Generally, imaging begins with plain radiographs to rule out other common diagnoses such as acute fractures or neoplasms. However, MRI is ideal for evaluating thoracic disc disease since it is highly sensitive and can be used to determine the

morphology and location of the disc herniation noninvasively [41]. Herniations appear as an intermediate intensity signal on T1-weighted images and as an area of low signal density on T2-weighted images [41]. CT with myelography provides the additional benefit of clearly illustrating osseous anatomy in relation to the soft- or hard-tissue disk herniation [41]. In addition to identifying the level of the herniation, imaging helps to classify the herniation based on midline, paramedian, or lateral positions, with a majority of herniations occurring in either midline or paramedian positions [41].

Initial Management

Most cases of thoracic disc degeneration can be managed with conservative therapy. In patients without significant acute or progressive neurological symptoms, treatment usually begins with nonsteroidal anti-inflammatory drugs, lifestyle modifications, and physical therapy [41]. Conservative management is typically sufficient for most cases of symptomatic thoracic disc herniation, and if the patient experiences extreme discomfort, narcotics and muscle relaxers can be used temporarily [3, 41]. Symptoms related to radiculopathy can also be treated medically with the addition of corticosteroid injections into the intercostal nerves [41]. If nonoperative management yields insignificant improvement or worsening symptoms after 4–6 weeks or the patient presents with myelopathy or progressive neurological deficits, operative intervention should be considered [41].

Surgical Management

With advances in surgical techniques, decompressive laminectomy is no longer used for treating thoracic disc herniation [33, 41]. Instead, anterior, posterior, and lateral approaches are used depending on the morphology and location of the disc herniation [41]. Transthoracic anterior approaches are most frequently used, and they provide excellent exposure for herniations occurring from T2 to T10 and do not affect posterior column stability, but they require a thoracotomy so it may not be ideal for high-risk patients [41]. Lateral approaches are best for lateral disc herniations and may avoid thoracotomy, but the limitations of this approach include increased operative time and the need for significant bone resection which can introduce morbidity and potentially instability [41]. Posterior approaches such as pediculofacetectomy are best for upper thoracic herniations occurring from T2 to T4 and for high-risk patients who may be unable to undergo thoracotomy, but these procedures may cause spinal instability and incomplete resolution of the herniation [41].

Recently, minimally invasive procedures such as video-assisted thoracoscopic surgery (VATS) have been gaining popularity due to their significantly reduced postoperative recovery period and ability to be performed on high-risk patients [41]. However, since these procedures are technically challenging and require a steep learning curve, it has taken some time for them to be universally adopted [7, 41]. In general, VATS has better outcomes when compared to traditional open approaches for removing herniated discs in the thoracic region [32, 35, 41].

Osseous Degenerative Changes

Thoracic degenerative conditions can also be caused by changes to the bony morphology of the spine. These changes are not caused by inflammatory processes as in other conditions, such as ankylosing spondylitis, but are the result of age and use related degeneration. The conditions described below all involve changes to the bone structure of the thoracic spine, and they do not primarily involve the spinal ligaments or intervertebral disks. These conditions often manifest as back pain, and if left undiagnosed and untreated can progress to worsening myelopathy or radiculopathy.

Pathophysiology

In these osseous degenerative changes, the underlying cause of degeneration is simply use-related stress that is correlated to age. Due to the previously described biomechanical stability of the thoracic spine, many of these conditions are uncommon in the thoracic region versus the more mobile cervical and lumbar spine. With thoracic vertebral spondylosis, the majority of cases afflicted the lower thoracic segment [37]. The stress experienced by the bone in these conditions can cause morphological changes producing outgrowths such as osteophytes or syndesmophytes. These bony morphological changes may progress to symptomatic myelopathy, radiculopathy, claudication, or pain if the bony deformities begin to compress on the spinal cord or impinge on other neural structures [5].

Epidemiology

Osteoarthritis of the spine involves the facet joints and is most commonly linked to those levels of the spine frequently afflicted by degenerative disc disease, as the disc, facet joint, and spinal ligaments form a complex where changes in one can affect the others. This leads to a decreased incidence of facet joint osteoarthritis in the thoracic spine as conditions affecting the intervertebral disc and spinal ligaments are less common in the thoracic region. No definitive study has been conducted to evaluate the presence of facet joint osteoarthritis of the thoracic spine specifically, however other studies have demonstrated the widespread nature of facet joint osteoarthritis in other spine regions, with up to 57% of adults over 65 demonstrating radiological evidence of the condition [11, 25, 39].

Facet Joint hypertrophy is an additional pathology involving the joints of the spine that can cause radicular symptoms or myelopathy if the growth begins to impinge on the spinal cord [20]. This condition is also rare in the thoracic spine with no studies showing broader population-level frequency and the literature consisting mainly of individual case reports and case series.

Physical Exam Findings

The physical exam findings in these conditions are related to the pathophysiology. Pain is a common finding either as a direct result of the degeneration and bony changes or due to compression of neural elements [11]. Symptoms of radiculopathy and myelopathy in the thoracic region such as paresthesia, weakness, incontinence,

difficulty walking, and pain may also arise. A full neurologic exam is pertinent to discovering the symptoms, with reports of deteriorating condition raising the index of suspicion. In rare instances the formation of osteophytes has damaged structures surrounding the spinal cord, such as the esophagus, causing dysphagia and other symptoms [31].

Imaging Findings

Imaging studies commonly used to detect these degenerative changes are computed tomography (CT) and magnetic resonance imaging (MRI). There is an ongoing debate about whether findings on imaging are predictive of or even correlated to clinical symptoms and findings [16, 19, 36]. Typically back pain alone is not an indication for further imaging, however, if worrisome symptoms such as persistent radiculopathy, myelopathy, incontinence, or urinary retention arise, then imaging and further workup is indicated. T2 weighted imaging in particular would show cord and/or neuroforamina compression. The bony outgrowths may be visible on CT or MRI but their presence does not always correlate to symptoms [19].

Management

First-line treatment for these conditions involves medical management with nonsteroidal anti-inflammatory drugs, neuropathic pain medication, or physical therapy [11]. Epidural or facet joint steroid injections may also provide some benefit. Surgical treatment is typically reserved for patients with spinal instability, and worsening symptoms of myelopathy or radiculopathy. Surgical management involves decompression of the spinal canal most often through a posterior laminectomy [20]. Concurrent fusion may be done to correct any instability that may be present.

Ligament Ossification

Pathophysiology

Ossification of the posterior longitudinal ligament (OPLL) and ossification of the ligamentum flavum (OLF) can narrow the spinal canal in the thoracic region, manifesting as a wide range of neurological deficits depending on the severity. OPLL and OLF can occur individually or can be found simultaneously in patients [22]. Although the pathogenesis of these diseases is poorly understood, it is thought that genetics, hormonal, and biomechanical factors all contribute to ossification formation and progression [1, 27, 34]. Additionally, given the kyphosis and limited blood supply of the thoracic spine, the spinal cord in this region is naturally vulnerable to pressure being applied from the ventral side, which is the case in OPLL [22].

Epidemiology

OPLL has an incidence of 2.4% in the Asian population compared to just 0.16% in non-Asian populations, and it is most commonly seen in Japan [34]. However, thoracic OPLL occurs less frequently than cervical OPLL and a majority of thoracic

OPLL occurs in tandem with cervical OPLL [22]. Thoracic OPLL commonly affects the upper and middle regions of the thoracic spine with the average ossification having an apex at T5 and spanning 4.8 intervertebral segments [22, 24]. Thoracic OPLL appears predominantly in females who are older than 40 years of age [24]. OPLL has been associated with other musculoskeletal diseases such as DISH, ankylosing spondylitis, and other spondyloarthropathies [34].

Thoracic OLF most commonly affects the lower thoracic spine and involvement of the upper thoracic spine is also common, but OLF rarely occurs in the middle thoracic spine [14, 22, 45]. This is likely because the lower thoracic spine has more mobility, so the ligamentum flavum in this region is subject to greater mechanical stress as a result [22]. The most common variation of OLF begins with the ossification of the capsular portion of the ligamentum flavum and extends medially and progresses to the interlaminar portion [1, 22]. OLF occurs more frequently in men than women, and although the paucity of data makes it difficult to estimate the true prevalence, asymptomatic OLF may not be that rare in individuals over the age of 30 [22]. OLF also frequently occurs in the Japanese population, but the extent to which it impacts other populations has yet to be elucidated [12].

Physical Exam Findings

Patients with thoracic OPLL are generally asymptomatic until the ossification begins to compress the spinal cord [22]. Patients may start with pain in the back corresponding to the level of spinal compression before the appearance of symptoms of myelopathy such as pain or numbness in the lower extremities, difficulty walking, and stiffness in the trunk and lower limbs [22]. Neurologic exam findings for thoracic OPLL usually include hyperreflexia of the lower extremities, gait disturbances, and sensory disturbances below the dermatome corresponding to the level of the compression [22]. Other symptoms include thoracic radiculopathy, urinary retention, and bladder/bowel incontinence [24]. Once symptoms of myelopathy appear, patients usually experience gradual and steady neurological deterioration, but there have been cases where patients have been unable to walk within a short period of time [22].

The clinical presentation of thoracic OLF depends on the severity of spinal cord compression, but it typically involves local thoracic pain and progressive thoracic myelopathy [22]. Early on, posterior cord compression develops since the ossification begins on the posterior side of the spinal cord, and lateral corticospinal tracts are compressed as the lesion progresses, eventually leading to loss of sensation [1]. Since OLF frequently occurs in the lower thoracic region, neurologic symptoms can mimic those caused by lumbar spinal disease or peripheral neuropathy [22]. Patients with lower thoracic OLF may have weakness and atrophy of the leg muscles along with deep tendon reflex abnormalities [22]. Patients usually also have symptoms of leg numbness, gait disturbances, and bowel/bladder incontinence [22, 45]. There can be asymmetry in neurological symptoms attributed to a difference in thickness of the ossification between the left and right sides [22].

Imaging Findings

In order to evaluate thoracic OPLL, radiographic imaging can be used to determine the severity and location of the ossification. The lesion typically appears as a mushroom or hill shape with a sharp radiolucent line between the posterior vertebral body margin and the ossified ligament on an axial CT [38]. While OPLL can be detected using plain films, computed tomography and/or myelography provide better accuracy for locating OPLL ossification [34]. Additionally, CT myelography or MR imaging is useful for determining the degree of stenosis [24]. Imaging studies are also useful for identifying signs of dural penetration, cord signal change, and if the ossification extends to other parts of the spine or involves other ligaments [24].

Thoracic OLF is difficult to diagnose with simple radiography since the upper and lower thoracic spine are often hidden by overlapping structures. Therefore, MRI is used to evaluate signal changes in the spinal cord, while CT helps distinguish between calcification and hypertrophy [1]. An ossified mass which encompasses more than 2/3 of the dura matter and a low-signal line between the parallel bone plates on CT are two findings that are indicative of dural involvement [1]. Imaging also provides a way to better characterize the lesion, such as categorizing OLF into fused type versus non-fused type as well as beak type versus round type [18, 45].

Initial Management

Treatment of OPLL and OLF generally requires surgical intervention because the ossification is progressive with little response to conservative therapies [1, 22, 45]. Immediate surgical intervention is important for improving the functional outcomes of patients [1, 45]. Medical treatments are currently limited to relieving symptoms rather than halting or reversing the ossification, and they include topical agents, anti-inflammatory drugs, antidepressants, anticonvulsants, and opioids [34].

Surgical Management

The main goal of surgical intervention is to decompress the spinal cord in order to alleviate neurological symptoms. This can be accomplished via a variety of surgical techniques including posterior decompressive laminectomy, posterior decompressive laminectomy with fusion, anterior decompression through an anterior or lateral approach, anterior decompression through a posterior approach, posterior decompression with fusion followed by anterior or lateral decompression, and laminoplasty [24]. Although there is no standard protocol for how to surgically treat thoracic OPLL, both the extent and location of the ossification can be used to determine the optimal surgical approach. In addition to decompression, stabilizing the thoracic spine with posterior segmental instrumentation or laminectomy with fusion helps prevent worsening kyphosis and reduces the compressive force on the spinal cord [24].

Posterior decompression via laminectomy with or without fusion or laminoplasty is frequently used to treat OPLL. While laminoplasty has favorable outcomes for OPLL lesions in the upper thoracic spine, instrumentation should be considered

when dealing with OPLL in the middle or lower thoracic spine due to the physiological kyphosis creating additional pressure on the spinal cord [23, 24]. Moreover, posterior instrumented fusion reduces spinal mobility, leading to improved neurological recovery [24, 44].

Advances in surgical techniques have enabled anterior decompression via an anterior, posterior, or lateral approach to be performed at all levels of the thoracic spine [24]. Anterior decompression is ideal for treating myelopathy caused by OPLL, but outcomes are poorer when it is used to treat worsening myelopathy after laminectomy has already been performed [10, 24, 29]. A transsternal approach allows access to lesions in the upper thoracic vertebra, while a transthoracic anterior approach through thoracotomy is optimal for pathology below T4 [24, 29]. For the most severe cases, combined anterior-posterior decompression can be performed by a posterior decompression and stabilization with an anterior decompression via thoracotomy with interbody fusion [17]. Anterior decompression for OPLL has a relatively high risk of cerebrospinal fluid (CSF) leakage and accidental damage to the spinal cord or nerve roots [24, 26].

Similarly, a common approach to thoracic OLF is posterior decompression because it provides excellent visualization and access to completely resect the ossified ligament [45]. If the ossified ligament is attached to the dura, care must be taken to avoid complications such as CSF leakage and intraoperative cord damage [1, 45]. In this case, the outer layer of the dura is also excised, and a graft is used to repair the dural defect [1, 45].

Conclusion

Thoracic degenerative conditions may be caused by a wide range of pathologies affecting the thoracic spine. Symptomatic presentation of these conditions is uncommon compared to their cervical and lumbar counterparts due to the inherent anatomical and biomechanical stability present in the thoracic spine. Surgery is indicated for these conditions if neurologic symptoms manifest acutely or continue to worsen, or if instability is evident. The anatomical and biomechanical differences in the thoracic spine result in a unique set of conditions that can cause symptomatic degenerative disease, and these differences, when compared to other spinal regions, must be taken into account when selecting appropriate surgical therapy to provide symptomatic relief. The choice of which approach and operative technique to use involves carefully considering the specific pathology, level of the degeneration, and patient characteristics.

References

1. Ahn DK, Lee S, Moon SH, Boo KH, Chang BK, Lee JI. Ossification of the ligamentum flavum. Asian Spine J. 2014;8:89–96.
2. Arce CA, Dohrmann GJ. Herniated thoracic disks. Neurol Clin. 1985;3:383–92.
3. Brown CW, Deffer JP, Akmakjian J, Donaldson DH, Brugman JL. The natural history of thoracic disc herniation. Spine. 1992;17:S97–102.

4. Carson J, Gumpert J, Jefferson A. Diagnosis and treatment of thoracic intervertebral disc protrusions. J Neurol Neurosurg Psychiatry. 1971;34:68–77.
5. Chana J, Afshar F. Thoracic spondylosis presenting with spastic paraparesis. Postgrad Med J. 1996;72:243–4.
6. Court C, Mansour E, Bouthors C. Thoracic disc herniation: surgical treatment. Orthop Traumatol Surg Res. 2018;104:S31–s40.
7. Dahdaleh NS. Minimally invasive treatment of thoracic disc herniations. In: Minimally invasive spine surgery, an issue of neurosurgery clinics of North America, E-Book, Vol. 25. 2014. p. 271.
8. El-Kalliny M, Tew JM, van Loveren H, Dunsker S. Surgical approaches to thoracic disc herniations. Acta Neurochir. 1991;111:22–32.
9. Epstein NE. Learning curves for minimally invasive spine surgeries: are they worth it? Surg Neurol Int. 2017;8:61.
10. Fujimura Y, Nishi Y, Nakamura M, Toyama Y, Suzuki N. Long-term follow-up study of anterior decompression and fusion for thoracic myelopathy resulting from ossification of the posterior longitudinal ligament. Spine. 1997;22:305–11.
11. Gellhorn AC, Katz JN, Suri P. Osteoarthritis of the spine: the facet joints. Nat Rev Rheumatol. 2013;9:216–24.
12. Guo JJ, Luk KD, Karppinen J, Yang H, Cheung KM. Prevalence, distribution, and morphology of ossification of the ligamentum flavum: a population study of one thousand seven hundred thirty-six magnetic resonance imaging scans. Spine. 2010;35:51–6.
13. Healy AT, Lubelski D, Mageswaran P, Bhowmick DA, Bartsch AJ, Benzel EC, et al. Biomechanical analysis of the upper thoracic spine after decompressive procedures. Spine J. 2014;14:1010–6.
14. Hur H, Lee J-K, Lee J-H, Kim J-H, Kim S-H. Thoracic myelopathy caused by ossification of the ligamentum flavum. J Korean Neurosurg Soc. 2009;46:189.
15. Ito K, Creemers L. Mechanisms of intervertebral disk degeneration/injury and pain: a review. Global Spine J. 2013;3:145–52.
16. Kalichman L, Li L, Kim D, Guermazi A, Berkin V, O'Donnell CJ, et al. Facet joint osteoarthritis and low back pain in the community-based population. Spine. 2008;33:2560.
17. Kawahara N, Tomita K, Murakami H, Hato T, Demura S, Sekino Y, et al. Circumspinal decompression with dekyphosis stabilization for thoracic myelopathy due to ossification of the posterior longitudinal ligament. Spine. 2008;33:39–46.
18. Kuh SU, Kim YS, Cho YE, Jin BH, Kim KS, Yoon YS, et al. Contributing factors affecting the prognosis surgical outcome for thoracic OLF. Eur Spine J. 2006;15:485–91.
19. Lewinnek GE, Warfield CA. Facet joint degeneration as a cause of low back pain. Clin Orthop Relat Res. 1986;(213):216–22.
20. Lim A, D'Urso P. Single-level bilateral facet joint hypertrophy causing thoracic spinal canal stenosis. J Clin Neurosci. 2009;16:1363–5.
21. Lubelski D, Healy AT, Mageswaran P, Benzel EC, Mroz TE. Biomechanics of the lower thoracic spine after decompression and fusion: a cadaveric analysis. Spine J. 2014;14:2216–23.
22. Matsumoto M, Chiba K, Toyama Y. Clinical manifestations of thoracic OPLL and OLF. In: OPLL. Springer; 2006. p. 121–5.
23. Matsumoto M, Chiba K, Toyama Y, Takeshita K, Seichi A, Nakamura K, et al. Surgical results and related factors for ossification of posterior longitudinal ligament of the thoracic spine: a multi-institutional retrospective study. Spine. 2008;33:1034–41.
24. McClendon J Jr, Sugrue PA, Ganju A, Koski TR, Liu JC. Management of ossification of the posterior longitudinal ligament of the thoracic spine. Neurosurg Focus. 2011;30:E16.
25. Mikkelsen WM, Duff I, Dodge HJ. Age-sex specific prevalence of radiographic abnormalities of the joints of the hands, wrists and cervical spine of adult residents of the Tecumseh, Michigan, Community Health Study area, 1962–1965. J Chronic Dis. 1970;23:151–9.
26. Min J-H, Jang J-S, Lee S-H. Significance of the double-layer and single-layer signs in the ossification of the posterior longitudinal ligament of the cervical spine. J Neurosurg Spine. 2007;6:309–12.

27. Nakajima M, Takahashi A, Tsuji T, Karasugi T, Baba H, Uchida K, et al. A genome-wide association study identifies susceptibility loci for ossification of the posterior longitudinal ligament of the spine. Nat Genet. 2014;46:1012–6.
28. Oda I, Abumi K, Lü D, Shono Y, Kaneda K. Biomechanical role of the posterior elements, costovertebral joints, and rib cage in the stability of the thoracic spine. Spine (Phila Pa 1976). 1996;21:1423–9.
29. Ohtani K, Nakai S, Fujimura Y, Manzoku S, Shibasaki K. Anterior surgical decompression for thoracic myelopathy as a result of ossification of the posterior longitudinal ligament. Clin Orthop Relat Res. 1982;(166):82–8.
30. Panjabi MM, Takata K, Goel V, Federico D, Oxland T, Duranceau J, et al. Thoracic human vertebrae. Quantitative three-dimensional anatomy. Spine (Phila Pa 1976). 1991;16:888–901.
31. Rathinam S, Makarawo T, Norton R, Collins F. Thoracic osteophyte: rare cause of esophageal perforation. Dis Esophagus. 2010;23:E5–8.
32. Regan JJ. Percutaneous endoscopic thoracic discectomy. Neurosurg Clin N Am. 1996;7:87–98.
33. Russo A, Balamurali G, Nowicki R, Boszczyk BM. Anterior thoracic foraminotomy through mini-thoracotomy for the treatment of giant thoracic disc herniations. Eur Spine J. 2012;21:212–20.
34. Saetia K, Cho D, Lee S, Kim DH, Kim SD. Ossification of the posterior longitudinal ligament: a review. Neurosurg Focus. 2011;30:E1.
35. Sasani M, Ozer F, Oktenoglu T, Kaner T, Solmaz B, Canbulat N, et al. Thoracoscopic surgery for thoracic disc herniation. J Neurosurg Sci. 2011;55:391–5.
36. Schwarzer AC, Wang SC, O'Driscoll D, Harrington T, Bogduk N, Laurent R. The ability of computed tomography to identify a painful zygapophysial joint in patients with chronic low back pain. Spine. 1995;20:907–12.
37. Smith DE, Godersky JC. Thoracic spondylosis: an unusual cause of myelopathy. Neurosurgery. 1987;20:589–93.
38. Soo M, Rajaratnam S. Symptomatic ossification of the posterior longitudinal ligament of the cervical spine: pictorial essay. Australas Radiol. 2000;44:14–8.
39. Suri P, Miyakoshi A, Hunter DJ, Jarvik JG, Rainville J, Guermazi A, et al. Does lumbar spinal degeneration begin with the anterior structures? A study of the observed epidemiology in a community-based population. BMC Musculoskelet Disord. 2011;12:202.
40. Teraguchi M, Yoshimura N, Hashizume H, Muraki S, Yamada H, Minamide A, et al. Prevalence and distribution of intervertebral disc degeneration over the entire spine in a population-based cohort: the Wakayama Spine Study. Osteoarthr Cartil. 2014;22:104–10.
41. Vanichkachorn JS, Vaccaro AR. Thoracic disk disease: diagnosis and treatment. J Am Acad Orthop Surg. 2000;8:159–69.
42. Watkins R, Watkins R 3rd, Williams L, Ahlbrand S, Garcia R, Karamanian A, et al. Stability provided by the sternum and rib cage in the thoracic spine. Spine (Phila Pa 1976). 2005;30:1283–6.
43. Wood KB, Garvey TA, Gundry C, Heithoff KB. Magnetic resonance imaging of the thoracic spine. Evaluation of asymptomatic individuals. J Bone Joint Surg Am. 1995;77:1631–8.
44. Yamazaki M, Okawa A, Fujiyoshi T, Furuya T, Koda M. Posterior decompression with instrumented fusion for thoracic myelopathy caused by ossification of the posterior longitudinal ligament. Eur Spine J. 2010;19:691–8.
45. Yu S, Wu D, Li F, Hou T. Surgical results and prognostic factors for thoracic myelopathy caused by ossification of ligamentum flavum: posterior surgery by laminectomy. Acta Neurochir. 2013;155:1169–77.
46. Zhang F, Zhao X, Shen H, Zhang C. Molecular mechanisms of cell death in intervertebral disc degeneration. Int J Mol Med. 2016;37:1439–48.

Lumbar Spinal Stenosis

23

Carl Youssef, Sai Chilakapati, and Owoicho Adogwa

Definition, Classification, and Grading

Lumbar spinal stenosis is a broad term that describes the narrowing of the spinal canal and compression of the neural structures. Several classifications exist based on etiology, location, and radiographic features [1]. In general, there are two main types of LSS: congenital and acquired. In congenital, or primary, LSS, the spinal canal is narrowed due to short pedicles and/or a decreased vertebral body width. Acquired, or secondary, LSS, on the other hand, is caused by various conditions that result in compression of the thecal sac. Degenerative disease is the most common cause of acquired LSS and will be the focus of this chapter. Other causes of acquired LSS include fractures (e.g., compression fracture with retropulsion), epidural hematoma, infections, and tumors.

LSS can also be classified based on the location of the stenosis. Central stenosis involves compression of the thecal sac in the middle of the spinal canal, although it is usually circumferential in cases of degenerative LSS. Lateral recess stenosis refers to compression of the lateral spinal canal with resultant impingement of the traversing nerve root(s); this can be caused by a paracentral disc herniation or facet joint hypertrophy with ligamentous thickening. Lastly, foraminal or extraforaminal stenosis refers to compression of the nerve root as it exits through the intervertebral foramen and is often caused by a foraminal or "far lateral" disc herniation. Additionally, three spinal canal shapes have been described: round, ovoid, and trefoil, with the trefoil-shaped spinal canals being associated with the smallest cross-sectional area [2].

Several radiographic grading systems have been published to assess the severity of LSS, using varying definitions of LSS and with mixed inter- and intra-rater

C. Youssef · S. Chilakapati · O. Adogwa (✉)
Department of Neurological Surgery, UT Southwestern Medical Center, Dallas, TX, USA
e-mail: Carl.Youssef@utsouthwestern.edu; Sai.Chilakapati@utsouthwestern.edu; Owoicho.Adogwa@UTSouthwestern.edu

© The Author(s), under exclusive license to Springer Nature Switzerland AG 2022

C. E. Noe (ed.), *Multidisciplinary Spine Care*,
https://doi.org/10.1007/978-3-031-04990-3_23

reliability [3]. For example, Schizas and colleagues published a grading system based on the morphology of the thecal sac and nerve roots on an axial T2-weighted MRI [4]. According to this classification, Grade A and its subtypes correspond to little or no stenosis, with the rootlets visible surrounded by cerebrospinal fluid (CSF). Grade B corresponds to moderate stenosis in which the rootlets are still identifiable, although with a scant amount of surrounding CSF. Grade C corresponds to severe stenosis in which no CSF is seen in the presence of epidural fat. Lastly, Grade D corresponds to severe stenosis in which no CSF or epidural fat is seen.

Relevant Anatomy and Pathophysiology

As with any spinal disorder, knowledge of anatomy is the foundation of understanding the disease's pathogenesis. Anteriorly, the spinal canal is bordered by the vertebral bodies and intervertebral discs, with the posterior longitudinal ligament running on their dorsal surface. Posteriorly, the spinal canal is bordered by the laminae with the underlying ligamentum flavum. The pedicles define the lateral border, and the facet joints and capsules define the posterolateral border.

Degenerative LSS usually manifests as a constellation of spondylotic or "wear-and-tear" changes within the lumbar spine that result in progressive narrowing of the spinal canal. These changes were described in detail by Farfan and Sullivan. Kirkaldy and colleagues introduced the concept of the "three-joint complex" at each level formed by the intervertebral disc anteriorly and the two facet joints posteriorly [5, 6]. They postulated that two types of micro traumatic injuries occur in degenerative LSS: recurrent rotational strain and repeated compressive injury. Anteriorly, the disc goes through several stages of degeneration, the earliest of which is annular tears. Further trauma to the disc leads to the disc material's internal disruption, resorption, and eventual loss of disc height. Posteriorly, the facet joints also undergo several degenerative changes that include synovitis, degeneration, and irregularity of the articular cartilage, osteophyte formation, and laxity of the joint capsule.

This combined effect of the degenerative disc disease and facet joint arthropathy results in micro instability due to structural compromise that further contributes to the progression of the degenerative process at the index level and adjacent segments. The ligamentum flavum becomes thickened due to the added mechanical stress, which narrows the canal medially and laterally, where the ligament attaches to the anterior aspect of the facet joint forming the anterior capsule. Micro instability also causes degenerative spondylolisthesis, which occurs when there is subluxation (typically in the anteroposterior plane but can be lateral and/or rotational) of one vertebra upon another [7]. When coexistent with LSS, it results in a further decrease in the spinal canal diameter and contributes to foraminal stenosis. Similarly, degenerative LSS can also be accompanied by scoliotic deformity when several levels are affected, further contributing to instability, canal compromise, and neural compression [8].

Physiologically, chronic compression results in several changes to the nerve roots. In an experimental study, dogs with induced compression of their nerve roots were found to have venous congestion of the root and nerve root ganglion, arterial narrowing, and changes in cortical evoked potential [9]. Histologically, compressed nerve roots showed edema, loss of myelin, blockade of axoplasmic flow, and Wallerian degeneration.

Epidemiology

LSS is a ubiquitous disease affecting millions of people around the globe. In the United States, the prevalence of acquired radiographic and symptomatic LSS is 29.7% and 22.5%, respectively, based on an ancillary population sample of the Framingham Heart Study [10]. A similar cross-sectional population study in Japan reported that the prevalence of radiographic and symptomatic acquired LSS is 76.5% and 9.3%, respectively [11]. The drastic difference in the prevalence of radiographic LSS between those two studies is likely related to imaging modality differences (CT versus MRI).

Another study of the Framingham population investigated the association between patient demographics and LSS [12]. Interestingly, it showed no statistically significant association between LSS and age, sex, and BMI. However, age was significantly associated with degenerative disc disease, facet osteoarthritis, and degenerative spondylolisthesis. Female sex was also significantly associated with degenerative spondylolisthesis. There was no significant association between BMI and any degenerative radiographic changes.

Clinical Presentation

A review of the literature reveals the poor predictability of symptomatic LSS based on radiographic findings. For example, in the Japanese Wakayama Spine Study, only 17.5% of participants with severe radiographic LSS were symptomatic [13]. Another study of asymptomatic patients who underwent MRI of the lumbar spine revealed that 1% of those younger than 60 years old and 21% of those older than 60 years old had lumbar stenosis [14].

Neurogenic claudication is one of the most common and classic symptoms of LSS [15]. It refers to pain or discomfort in the buttocks, groins, thighs, calves, and/or feet that occurs with standing and walking, and is relieved with resting. Patients typically report decreased walking tolerance due to weakness or "heaviness" of the lower extremities, often described as "knees buckling" or "legs giving out." Patients with LSS usually walk "stooped forward," such as when leaning on a shopping cart. A distinguishing feature between neurogenic and vascular etiologies is cycling tolerance— patients with neurogenic claudication usually have no symptoms while cycling due to the lumbar spine being flexed. In contrast, patients with peripheral

vascular disease will have claudicatory symptoms regardless of their posture. On examination, patients presenting with neurogenic claudication usually have no focal neurologic deficits. The lumbar spine range of motion may be decreased, particularly with extension. The straight leg raise test is negative and a "Simian stance" is typically observed when standing, with posterior pelvic tilt and flexion of the hips and knees.

Low back pain (LBP) is also a common and nonspecific symptom. In the Framingham study mentioned earlier, radiographic LSS was significantly associated with LBP, with an odds ratio of 3.16 [10]. The back pain is often nonspecific, and patients may have point tenderness at multiple levels. However, in patients with superimposed spondylolisthesis, the back pain can have a mechanical component, indicative of instability.

Patients with LSS can also have radiculopathy, usually manifesting as unilateral pain or dermatomal numbness radiating down an extremity, in contrast to neurogenic claudication, which is bilateral. The straight leg raise test may be positive in patients with radiculopathy. Radiculopathy can also be associated with a motor deficit such as a hip flexor weakness or foot drop.

Rarely, due to its chronic nature, severe LSS can result in conus medullaris or, more commonly, cauda equina syndromes, depending on the level of compression. Conus medullaris syndrome (CMS) occurs if the conus medullaris extends down into the upper lumbar spine (which in most adults is at the level of L1–2). In contrast, the more common cauda equina syndrome (CES) occurs when the nerve roots within the thecal sac below the conus medullaris are compressed. Both syndromes are characterized by lower extremity weakness, saddle anesthesia, urinary retention, and bowel incontinence. A clinical distinction can be made between the two syndromes. CMS patients have mixed upper and lower motor neuron signs (e.g., hyperreflexia, spasticity), bilateral and symmetrical symptoms, and mild-moderate back pain. CES patients, in contrast, typically have diminished reflexes, asymmetrical symptoms, and severe back pain.

Management Options

In the absence of objective neurologic deficits (e.g., weakness, bowel/bladder incontinence) or intractable symptoms, conservative management should always be the first line of treatment in patients with symptomatic LSS. Several conservative modalities exist that may offer symptomatic relief, including medications, physical therapy (PT), epidural injections, and chiropractic treatments. Surgery is reserved for patients who have persistent symptoms despite maximal conservative management.

The commonly used medications in patients with symptomatic LSS are nonsteroidal anti-inflammatory medications, acetaminophen, opioids, neuropathic pain medications (e.g., gabapentin and pregabalin), and muscle relaxants. However, surgeons must exercise caution when prescribing those medications to patients with LSS, since most of the patients are elderly and are at risk for polypharmacy.

PT represents a crucial component of the conservative management strategy. The average duration of therapy is usually 6 weeks. Several techniques are employed,

including lumbar flexion exercises, treadmill walking programs, ultrasound tissue therapy, and manual PT maneuvers. These PT maneuvers include thrust and non-thrust manipulation of the spine and lower extremity joints, manual stretching, and muscle strengthening exercises [16]. The long term efficacy of PT remains unclear in the literature. A systematic review concluded that PT "modalities have no additional effect to exercise." However, the review was admittedly limited due to the small number and low quality of studies, as well as the heterogeneity in treatments and outcome measures [17]. A similar literature review of chiropractic treatments concluded no definitive benefit, although they showed promise in some patients [18].

Epidural injections represent another major nonsurgical treatment modality in patients with LSS. A randomized, double-blind controlled trial compared epidural injections with only local anesthetic versus local anesthetic and steroids [19]. At the end of the study's 2-year follow-up, 51% of patients with an average of 5.1 procedures in the anesthetic only group versus 57% of patients with an average of 4.5 procedures in the anesthetic and steroids group reported significant pain relief and improvement in functional status. As such, the study found a similar efficacy with or without steroids. In a retrospective review of patients that received epidural steroids injections, only 32% of patients reported pain relief lasting more than 2 months, 39% reported pain relief lasting less than 2 months, and 29% reported no relief at all [20]. Additionally, a systematic review analyzing the efficacy of the different epidural injection approaches concluded that there is Level II evidence for the long-term efficacy of caudal and interlaminar injections, compared with Level III evidence for transforaminal injections [21].

For patients who fail a trial of conservative treatment, surgery can provide durable symptomatic relief. The Spine Patient Outcomes Research Trial (SPORT) investigators compared decompressive laminectomy against "usual nonsurgical care." They found surgery to be superior to nonsurgical care at 3 months and 2 years [22]. Another prospective study of patients undergoing decompression alone versus decompression and fusion demonstrated that patients in both groups had substantial improvement in outcomes at 1-year, with more improvement noted in the fusion group [23]. The Lumbar Stenosis Outcome Study (LSOS) in Europe reported that surgical and nonsurgical LSS patients had a similar health-related quality of life at 1-year. However, the study pointed out that conservative management was associated with higher costs due to repeated injections and/or patients eventually requiring surgery [24]. This finding is similar to that reported by another study in the United States evaluating the long term cost-effectiveness of maximal conservative management in patients that ultimately required surgery [25].

Summary

LSS is a complex disease that affects millions of patients. The diagnosis is made by a thorough evaluation of advanced imaging and the clinical presentation. The treatment can range from non-invasive modalities, such as medications and exercise, to more invasive methods, such as epidural injections, to the most invasive form of treatment, comprised of decompression with or without fusion. However, there still

exists a lack of robust, high quality, prospective controlled trials to definitively ascertain the effectiveness, risks, and cost-effectiveness of the available treatment methods. With an aging population and in an era of greater emphasis on improving outcomes while decreasing costs, LSS will continue to be a significant disease and an active research area.

References

1. Schroeder GD, Kurd MF, Vaccaro AR. Lumbar spinal stenosis: how is it classified? J Am Acad Orthop Surg. 2016;24(12):843–52.
2. Hilibrand AS, Rand N. Degenerative lumbar stenosis: diagnosis and management. J Am Acad Orthop Surg. 1999;7(4):239–49.
3. Andreisek G, et al. A systematic review of semiquantitative and qualitative radiologic criteria for the diagnosis of lumbar spinal stenosis. AJR Am J Roentgenol. 2013;201(5):W735–46.
4. Schizas C, et al. Qualitative grading of severity of lumbar spinal stenosis based on the morphology of the dural sac on magnetic resonance images. Spine (Phila Pa 1976). 2010;35(21):1919–24.
5. Kirkaldy-Willis WH, et al. Pathology and pathogenesis of lumbar spondylosis and stenosis. Spine (Phila Pa 1976). 1978;3(4):319–28.
6. Farfan HF, Sullivan JD. The relation of facet orientation to intervertebral disc failure. Can J Surg. 1967;10(2):179–85.
7. Farfan HF. The pathological anatomy of degenerative spondylolisthesis. A cadaver study. Spine (Phila Pa 1976). 1980;5(5):412–8.
8. Ploumis A, Transfledt EE, Denis F. Degenerative lumbar scoliosis associated with spinal stenosis. Spine J. 2007;7(4):428–36.
9. Delamarter RB, et al. Experimental lumbar spinal stenosis. Analysis of the cortical evoked potentials, microvasculature, and histopathology. J Bone Joint Surg Am. 1990;72(1):110–20.
10. Kalichman L, et al. Spinal stenosis prevalence and association with symptoms: the Framingham Study. Spine J. 2009;9(7):545–50.
11. Ishimoto Y, et al. Prevalence of symptomatic lumbar spinal stenosis and its association with physical performance in a population-based cohort in Japan: the Wakayama Spine Study. Osteoarthr Cartil. 2012;20(10):1103–8.
12. Kalichman L, et al. Association between age, sex, BMI and CT-evaluated spinal degeneration features. J Back Musculoskelet Rehabil. 2009;22(4):189–95.
13. Ishimoto Y, et al. Associations between radiographic lumbar spinal stenosis and clinical symptoms in the general population: the Wakayama Spine Study. Osteoarthr Cartil. 2013;21(6):783–8.
14. Boden SD, et al. Abnormal magnetic-resonance scans of the lumbar spine in asymptomatic subjects. A prospective investigation. J Bone Joint Surg Am. 1990;72(3):403–8.
15. Porter RW. Spinal stenosis and neurogenic claudication. Spine (Phila Pa 1976). 1996;21(17):2046–52.
16. Whitman JM, et al. A comparison between two physical therapy treatment programs for patients with lumbar spinal stenosis: a randomized clinical trial. Spine (Phila Pa 1976). 2006;31(22):2541–9.
17. Macedo LG, et al. Physical therapy interventions for degenerative lumbar spinal stenosis: a systematic review. Phys Ther. 2013;93(12):1646–60.
18. Stuber K, Sajko S, Kristmanson K. Chiropractic treatment of lumbar spinal stenosis: a review of the literature. J Chiropr Med. 2009;8(2):77–85.
19. Manchikanti L, et al. Results of 2-year follow-up of a randomized, double-blind, controlled trial of fluoroscopic caudal epidural injections in central spinal stenosis. Pain Physician. 2012;15(5):371–84.

20. Delport EG, et al. Treatment of lumbar spinal stenosis with epidural steroid injections: a retrospective outcome study. Arch Phys Med Rehabil. 2004;85(3):479–84.
21. Manchikanti L, et al. Efficacy of epidural injections in the treatment of lumbar central spinal stenosis: a systematic review. Anesth Pain Med. 2015;5(1):e23139.
22. Weinstein JN, et al. Surgical versus nonsurgical therapy for lumbar spinal stenosis. N Engl J Med. 2008;358(8):794–810.
23. Ghogawala Z, et al. Prospective outcomes evaluation after decompression with or without instrumented fusion for lumbar stenosis and degenerative Grade I spondylolisthesis. J Neurosurg Spine. 2004;1(3):267–72.
24. Aichmair A, et al. Cost-effectiveness of conservative versus surgical treatment strategies of lumbar spinal stenosis in the Swiss setting: analysis of the prospective multicenter Lumbar Stenosis Outcome Study (LSOS). Eur Spine J. 2017;26(2):501–9.
25. Adogwa O, et al. Long-term costs of maximum nonoperative treatments in patients with symptomatic lumbar stenosis or spondylolisthesis that ultimately required surgery: a 5-year cost analysis. Spine (Phila Pa 1976). 2019;44(6):424–30.

Spine Vascular Lesions of the Bone and the Epidural Space

24

James P. Caruso, Salah G. Aoun, and Kevin C. Morrill

Vertebtal Hemangiomas

Vertebral hemangiomas are among the most common vascular lesions of the spine. They have a prevalence of up to 28% in adults, and comparable incidence in men and women. They tend to increase in frequency with age, and are seen more commonly in the elderly population [1]. Macroscopically, hemangiomas have well-defined borders and appear dark red with honeycombed segmentations which are created by cavitation of blood products surrounded by trabecular bone [2]. Microscopically, they can be divided into cavernous, capillary, and mixed (cavernous-capillary) subtypes. Cavernous hemangiomas have thin-walled large vessels surrounded by adipocytes and interstitial edema without intervening normal bone. Capillary hemangiomas consist of thin-walled small-diameter vessels separated by normal bone. Mixed hemangiomas demonstrate a combination of capillary and cavernous features [3, 4]. Radiographically, hemangiomas can be classified as typical, atypical and aggressive [5], according to the proportions of fatty and vascular components visible on imaging, the degree of cortical involvement, and the degree of extension beyond the vertebral body [6]. Invasive lesions that expand beyond bone typically have a lower concentration of adipocytes and appear isointense (dark) on T1 MRI sequences and hyperintense (bright) on T2 MRI sequences [2]. In contrast, less invasive lesions have a higher concentration of adipocytes and appear hyperintense on T1 and T2 [2]. On CT, vertebral hemangiomas can demonstrate alternating foci of diminished and heightened

J. P. Caruso · K. C. Morrill (✉)
The University of Texas Southwestern, Department of Neurological Surgery, Dallas, TX, USA
e-mail: James.Caruso@utsouthwestern.edu; Kevin.Morrill@UTSouthwestenr.edu

S. G. Aoun
Department of Neurological Surgery and Spine Center, UT Southwestern Medical Center, Dallas, TX, USA
e-mail: Salah.Aoun@utsouthwestern.edu

© The Author(s), under exclusive license to Springer Nature Switzerland AG 2022
C. E. Noe (ed.), *Multidisciplinary Spine Care*,
https://doi.org/10.1007/978-3-031-04990-3_24

trabecular density within the vertebral body, which is termed a "honeycomb," "jail bar," or "corduroy cloth' pattern [7]. Additionally, vertebral hemangiomas frequently demonstrate a "polka-dotted" appearance on CT and MRI, due to the contrasting hyperdense trabeculae and hypodense stroma [8].

Vertebral hemangiomas are typically asymptomatic and discovered incidentally, but they can present with pain, radiculopathy, and spinal cord compression resulting in myelopathy in approximately 1% of cases [2, 9, 10]. Neural element compression arises from lesion expansion beyond the vertebral body into the posterior elements and epidural space, and it occasionally leads to vertebral body collapse. Symptomatic hemangiomas typically occur in younger patients and more commonly demonstrate heterogeneous imaging features, such as irregular honeycombing and lytic foci of varying sizes [2, 10, 11]. Pregnancy can also perpetuate vertebral hemangioma enlargement. Elevated serum progesterone can lead to hemangioma expansion, and uterine compression of the inferior vena cava perpetuates paravertebral venous plexus hypertension and promotes intravascular filling of the lesion [12, 13].

Asymptomatic vertebral hemangiomas can typically be managed conservatively, with regular interval surveillance imaging if the lesion is extensive or has shown evidence of growth. However, symptomatic lesions can warrant interventional treatment. The ideal treatment method varies with symptom severity, rate of progression, imaging findings, lesion location, and the patient's ability to tolerate invasive procedures. For patients who suffer pathologic fractures as a result of vertebral hemangiomas, vertebroplasty can provide pain relief, hemostasis, and structural stability [10, 14]. However, this approach has an increased risk of cement intravasation into the spinal canal with recurrence and progression of neurologic symptoms, and can also embolize into the veins draining the lesion, and lead to pulmonary embolism which can be life-threatening [15, 16]. Generally, surgical treatment is warranted if the patient develops progressive neurologic symptoms, and is in need of a structural decompression [17]. Favorable rates of local control after gross total resection can be achieved without total resection [18], but select case series have shown that en-bloc approaches are associated with less blood loss compared to piecemeal removal [19]. However, en-bloc resection is technically demanding and carries a 36% complication rate [20]. When gross total resection is not feasible, subtotal resection can also result in acceptable disease control, provided that it is accompanied by adjuvant radiation therapy [21–23]. In patients presenting with pain, or slowly progressing neurologic changes that do not constitute a neurologic emergency, radiation therapy alone has led to clinical improvement and low rates of recurrence on follow-up [23]. Smaller case series have demonstrated symptomatic control and quality of life benefit after radiation [24], and a dose-dependent effect on symptom relief has been observed [25]. Endovascular embolization has also been suggested as a primary and adjunct treatment option, since vertebral hemangiomas are highly vascular and surgical resection risks significant blood loss. Data is inconsistent regarding the efficacy of embolization alone. A minority of patients report symptom improvement [26, 27], but some case reports have shown effective relief of neurologic symptoms when embolization is performed in combination with radiation therapy [28]. However, embolization is better characterized as an adjunct to surgical resection for aggressive symptomatic lesions. Retrospective data suggests that preoperative endovascular embolization with

subsequent gross total resection leads to favorable outcomes and minimal risk of recurrence in series with long-term follow-up [29]. Pre-operative angiography and embolization also allow for accurate pre-operative treatment planning and characterization of vascular anatomy, which can lead to reduced blood loss during surgical resection [30]. For lesions not suitable for complete resection, such as those with multiple large lytic foci that intimately involve the neural elements, combination therapy with surgical decompression, embolization, and radiation, can result in prolonged relief of symptoms and favorable long-term outcomes [22, 31, 32].

Aneurysmal Bone Cysts

Aneurysmal bone cysts (ABCs) are benign lesions that are most common in children and young adults [33]. The true epidemiologic burden of ABCs is difficult to define, since these lesions are often incidentally discovered and can spontaneously regress [34]. Full-body MRI screening data suggests that 0.7% of patients with back and neck pain have ABCs [1]. The term "aneurysmal bone cyst" originated in 1942 from a radiographic description of the lesion as a "blown out distention with blood filled cavities." [35] While ABCs appear as blood-filled intraosseous cavities that take the shape of a "cyst" or an "aneurysm," neither term is technically accurate. Rather, they are generated by osteoclasts within bone and fibrous tissue, leading to the appearance of expanding blood-filled cavities interspersed within the trabeculae [36]. It is not clear what mechanism propagates ABC formation, but some hypotheses suggest a post-traumatic reactive process or rapid bony erosion and remodeling of a pre-existing vascular lesion, leading to a thin-shelled, cystic-appearing intraosseous cavity [37–40]. Macroscopically, the vascular tissue appears spongy, and the posterior elements and vertebral bodies often demonstrate cavities of non-clotted blood lined by thin rims of fragile cortical and subperiosteal bone [39]. Microscopically, the blood-filled cavities lack surrounding endothelial cells and smooth muscle characteristic of normal vessels, or aneurysms [39]. The fibrous tissue is well differentiated and benign-appearing, but mitotic figures may be observed in more aggressive lesions that enlarge and invade adjacent bony structures [39, 41]. On CT, ABCs typically appear lytic, centrally located, and locally aggressive [33, 39]. Typical descriptors include "blown-out" or "ballooned" lesions with "egg-shelled" foci of cortical bone and fluid-fluid levels [36, 42]. Fluid-fluid levels and multi-loculated fluid collections within bone are also present) on MRI, and blood of varying ages causes the fluid collections to demonstrate a heterogeneous intensity [39, 43].

If ABCs are asymptomatic, discovered incidentally, and do not demonstrate aggressive features or neural element compromise on imaging, then they can be managed safely with judicious follow-up imaging. Symptomatic lesions typically present with back or neck pain, but radicular and myelopathic symptoms can also occur, and a number of therapeutic options exist for expansile ABCs that produce neurologic symptoms or compromise the structural integrity of the spinal column [44]. The overall recurrence rate after treatment is 12.8%, and recurrence rates even after complete resection can be as high as 8.2%. Intraoperative blood loss can be reduced with the use of en-bloc resection techniques that do not breach the lesion's

intra-trabecular cavities [45]. Incomplete resection techniques, such as curettage and posterior decompression, have higher rates of recurrence [45]. Therefore, en-bloc resection is often preferred to ensure favorable extent of resection and minimize the risk of recurrence, since more generous doses of radiation can be administered once the neural elements are decompressed, and instrumentation is used to mitigate post-radiation vertebral collapse [46]. However, aggressive resections should be considered with care, since they are associated with greater morbidity and risk of neurologic injury compared with minimalistic surgical approaches [47].

When compared with surgical approaches, the risks and benefits of endovascular embolization are not well-established. However, some case series suggest that it is an effective first-line treatment in patients who do not demonstrate neurologic symptoms or have an elevated risk of pathologic fracture [35, 48]. While recurrence rates after embolization are often comparable to those after surgical excision [45, 46], patients often require multiple rounds of embolization to fully obliterate the lesion. It remains unclear whether aggressive endovascular treatment is necessary, because of the frequent post-procedural angiographic persistence of feeding vessels, and or the delayed development of collateral circulation pathways. The clinical impact of these angiographic recurrences is still unknown [46]. However, performing neo-adjuvant endovascular embolization prior to complete surgical resection can further reduce the risk of intraoperative bleeding and recurrence [45]. Pre-operative embolization should be performed judiciously or avoided in emergent scenarios, since it may delay emergent surgical decompression in patients who present with rapid neurologic decline [40]. In patients who cannot tolerate surgery, endovascular embolization may serve as the primary treatment modality, provided that the lesion does cause high risk features, such as spinal instability or deformity, acute neurologic compromise, or pathologic fractures [39]. Radiation has also been proposed as a primary and adjunct therapy, although existing data in the literature presents controversial results. Radiation is traditionally reserved for refractory cases unresponsive to surgery or embolization, and is associated with a host of complications including radiation-induced myelopathy and development of secondary bone tumors [36, 40, 49, 50]. However, it remains a potential treatment option for patients with inoperable lesions or who are at high risk for embolization or surgery [39]. Other less invasive techniques, such as sodium tetradecyl sulfate (STS) or doxycycline sclerotherapy have shown promising results in patients with recurrent disease after prior resection, but larger analyses to validate these techniques are necessary [39, 51, 52]. An evolving body of literature advocates for combination therapy, beginning with arterial embolization in select cases, followed by en-bloc excision if the lesion involves the posterior elements, and complete curettage for anteriorly located lesions with neurologic compromise or in cases associated with vertebral fractures [35].

Spinal Arteriovenous Lesions with Extradural Involvement

Myriad classification systems exist for spinal arteriovenous lesions, and our discussion will follow designations proposed by Spetzler and Kim, which are most commonly referenced [53]. In brief, spinal arteriovenous fistulae (AVFs) are classified

as extradural, intradural – dorsal, or intradural – ventral, and spinal arteriovenous malformations (AVMs) are classified as extradural – intradural, intramedullary, or conus medullaris. Since this chapter focuses on bony and epidural vascular pathology of the spine, we will limit our discussion to extradural AVFs and extradural-intradural AVMs. The remaining classifications of arteriovenous lesions will be discussed in separate sections of this book.

Purely extradural spinal AVFs) are rare lesions that typically originate from a fistulous connection between an epidural radicular artery and the epidural venous plexus, and they generally present with slow progression of radicular or myelopathic symptoms related to vascular steal or compression [53–55]. Since existing literature is limited mainly to small case series, established management paradigms are not well-defined. Angiographic lesion features should be incorporated into treatment planning, including the identification of the principal feeding artery, evaluation of extradural venous plexus enlargement, and identification of intradural retrograde venous drainage [56]. In patients with an enlarged epidural venous plexus and associated compressive myelopathy, endovascular embolization may effectively reduce blood supply to the lesion with low intraprocedural rupture risk, while microsurgery may be necessary if the lesion is particularly large or has multiple feeding vessels [56]. However, in lesions with intradural retrograde venous drainage, complete occlusion of the proximal intradural vein is required [56]. This can be performed with endovascular techniques, but microsurgical occlusion may reduce the risk of ischemic complications [56].

Extradural – intradural AVMs (also termed metameric AVMs) are extensive high flow lesions that encompass bone, soft tissue, skin, and neural tissue [53]. Like extradural AVFs, these lesions are very rare [57]. They can present with progressive myelopathic symptoms due to vascular steal, pain and radicular symptoms from compression, and hemorrhage [54]. These lesions can be rapidly progressive and debilitating. Palliation is often the primary treatment goal, and treatment usually requires a combination of surgical and endovascular embolization strategies [58, 59]. Treatment typically targets the spinal component of the lesion, as opposed to the soft tissue components [57]. A pooled analysis of existing case series revealed a 2.1% annual risk of hemorrhage, with comparatively low rates of complete obliteration compared to other spinal arteriovenous lesions [60]. Embolization alone resulted in a 29% obliteration rate, with 71% of patients improved at follow-up, while surgery (with and without pre-procedural embolization) resulted in a 36% obliteration rate with 77% of patients improving at follow-up [60]. However, these differences were not statistically significant, and these figures may be partially influenced by positive reporting bias from individual case reports.

Case Presentation

A 40-year-old man presented with worsening right chest wall man and bilateral lower extremity ataxia. Figure 24.1 shows a spinal angiogram revealing a spinal dural arteriovenous fistula arising from the right T5 intercostal artery with a radicular branch entering the right T5–T6 foramen (Yellow arrow, Fig. 24.1).

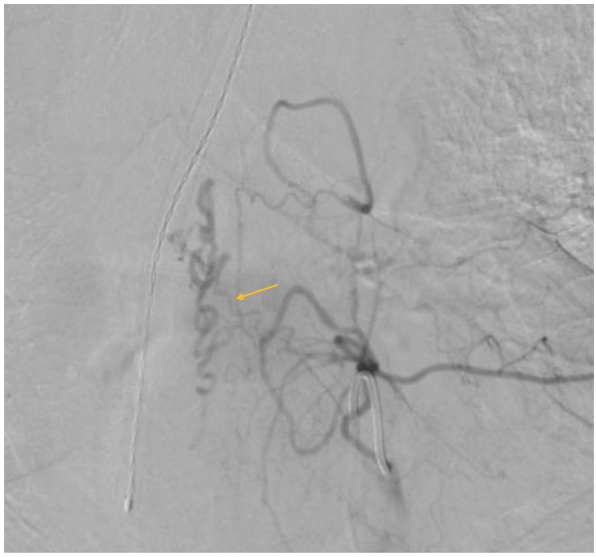


Fig. 24.1 Spinal dural arteriovenous fistula arising from the right T5 intercostal artery



Fig. 24.2 Filling present throughout the lesion

Endovascular embolization was performed, but the patient's symptoms persisted, and filling was present throughout the lesion (Fig. 24.2). Figure 24.2 shows filling throughout the lesion.

Surgical treatment was undertaken, and the arterial feeder was obliterated with clip ligation. Figure 24.3 shows a postoperative CT angiogram scan axial image that shows the T5 laminectomy, residual embolization material from prior endovascular

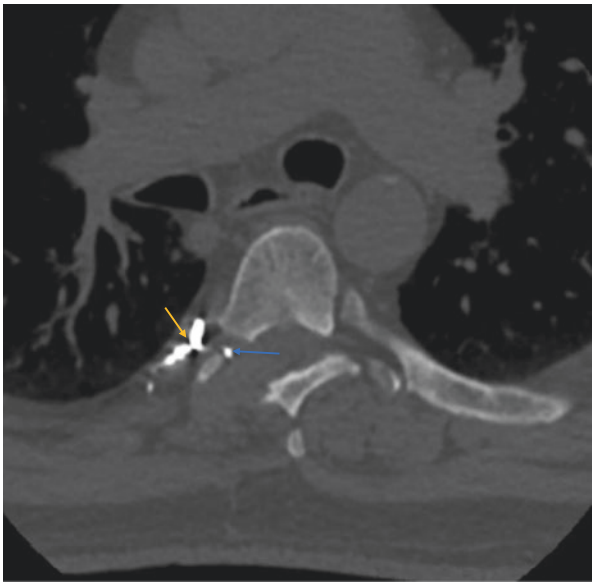


Fig. 24.3 Post-operative CT angiogram

procedure (Yellow arrow), and the clip ligation of the fistulous vessel involving the dura rostral to the T5–6 foramen (blue arrow). A postoperative angiogram demonstrated no residual vascular lesion.

References

1. Kasch R, Scheele J, Hancock M, et al. Prevalence of benign osseous lesions of the spine and association with spinal pain in the general population in whole body MRI. PLoS One. 2019;14(9):e0219846.
2. Gaudino S, Martucci M, Colantonio R, et al. A systematic approach to vertebral hemangioma. Skelet Radiol. 2015;44(1):25–36.
3. Baudrez V, Galant C, Vande Berg BC. Benign vertebral hemangioma: MR-histological correlation. Skelet Radiol. 2001;30(8):442–6.
4. Pastushyn AI, Slin'ko EI, Mirzoyeva GM. Vertebral hemangiomas: diagnosis, management, natural history and clinicopathological correlates in 86 patients. Surg Neurol. 1998;50(6):535–47.
5. Cross JJ, Antoun NM, Laing RJ, Xuereb J. Imaging of compressive vertebral haemangiomas. Eur Radiol. 2000;10(6):997–1002.
6. Laredo JD, Assouline E, Gelbert F, Wybier M, Merland JJ, Tubiana JM. Vertebral hemangiomas: fat content as a sign of aggressiveness. Radiology. 1990;177(2):467–72.
7. Ross JS, Masaryk TJ, Modic MT, Carter JR, Mapstone T, Dengel FH. Vertebral hemangiomas: MR imaging. Radiology. 1987;165(1):165–9.
8. Persaud T. The polka-dot sign. Radiology. 2008;246(3):980–1.
9. Acosta FL Jr, Sanai N, Chi JH, et al. Comprehensive management of symptomatic and aggressive vertebral hemangiomas. Neurosurg Clin N Am. 2008;19(1):17–29.
10. Vasudeva VS, Chi JH, Groff MW. Surgical treatment of aggressive vertebral hemangiomas. Neurosurg Focus. 2016;41(2):E7.
11. Laredo JD, Reizine D, Bard M, Merland JJ. Vertebral hemangiomas: radiologic evaluation. Radiology. 1986;161(1):183–9.

12. Lavi E, Jamieson DG, Granat M. Epidural haemangiomas during pregnancy. J Neurol Neurosurg Psychiatry. 1986;49(6):709–12.
13. Wang GX, Mu YD, Che JY, Zhang GF, Jiang G, Gao CP. Compressive myelopathy and compression fracture of aggressive vertebral hemangioma after parturition: a case report and review of literature. Medicine (Baltimore). 2019;98(50):e18285.
14. Hao J, Hu Z. Percutaneous cement vertebroplasty in the treatment of symptomatic vertebral hemangiomas. Pain Physician. 2012;15(1):43–9.
15. Evangelopoulos DS, Kontovazenitis P, Kokkinis K, Glynos M, Korres DS, Sapkas G. Cement leakage in a symptomatic vertebral hemangioma: a case report and review of the literature. Cases J. 2009;2:7148.
16. Deramond H, Depriester C, Galibert P, Le Gars D. Percutaneous vertebroplasty with polymethylmethacrylate. Technique, indications, and results. Radiol Clin N Am. 1998;36(3):533–46.
17. Nguyen JP, Djindjian M, Gaston A, et al. Vertebral hemangiomas presenting with neurologic symptoms. Surg Neurol. 1987;27(4):391–7.
18. Goldstein CL, Varga PP, Gokaslan ZL, et al. Spinal hemangiomas: results of surgical management for local recurrence and mortality in a multicenter study. Spine (Phila Pa 1976). 2015;40(9):656–64.
19. Kato S, Kawahara N, Murakami H, et al. Surgical management of aggressive vertebral hemangiomas causing spinal cord compression: long-term clinical follow-up of five cases. J Orthop Sci. 2010;15(3):350–6.
20. Cloyd JM, Acosta FL Jr, Polley MY, Ames CP. En bloc resection for primary and metastatic tumors of the spine: a systematic review of the literature. Neurosurgery. 2010;67(2):435–44; discussion 444–5.
21. Bremnes RM, Hauge HN, Sagsveen R. Radiotherapy in the treatment of symptomatic vertebral hemangiomas: technical case report. Neurosurgery. 1996;39(5):1054–8.
22. Fox MW, Onofrio BM. The natural history and management of symptomatic and asymptomatic vertebral hemangiomas. J Neurosurg. 1993;78(1):36–45.
23. Jiang L, Liu XG, Yuan HS, et al. Diagnosis and treatment of vertebral hemangiomas with neurologic deficit: a report of 29 cases and literature review. Spine J. 2014;14(6):944–54.
24. Aich RK, Deb AR, Banerjee A, Karim R, Gupta P. Symptomatic vertebral hemangioma: treatment with radiotherapy. J Cancer Res Ther. 2010;6(2):199–203.
25. Rades D, Bajrovic A, Alberti W, Rudat V. Is there a dose-effect relationship for the treatment of symptomatic vertebral hemangioma? Int J Radiat Oncol Biol Phys. 2003;55(1):178–81.
26. Raco A, Ciappetta P, Artico M, Salvati M, Guidetti G, Guglielmi G. Vertebral hemangiomas with cord compression: the role of embolization in five cases. Surg Neurol. 1990;34(3):164–8.
27. Smith TP, Koci T, Mehringer CM, et al. Transarterial embolization of vertebral hemangioma. J Vasc Interv Radiol. 1993;4(5):681–5.
28. Hekster RE, Endtz LJ. Spinal-cord compression caused by vertebral haemangioma relieved by percutaneous catheter embolisation: 15 years later. Neuroradiology. 1987;29(1):101.
29. Handa M, Demura S, Kato S, et al. Long-term outcomes of excision surgery for aggressive vertebral hemangiomas. World Neurosurg. 2020;142:e474–80.
30. Yu B, Wu D, Shen B, et al. Noncontiguous lumbar vertebral hemangiomas treated by posterior decompression, intraoperative kyphoplasty, and segmental fixation. J Neurosurg Spine. 2014;20(1):60–6.
31. Djindjian M, Nguyen JP, Gaston A, Pavlovitch JM, Poirier J, Awad IA. Multiple vertebral hemangiomas with neurological signs. Case report. J Neurosurg. 1992;76(6):1025–8.
32. Jayakumar PN, Vasudev MK, Srikanth SG. Symptomatic vertebral haemangioma: endovascular treatment of 12 patients. Spinal Cord. 1997;35(9):624–8.
33. Grahneis F, Klein A, Baur-Melnyk A, et al. Aneurysmal bone cyst: a review of 65 patients. J Bone Oncol. 2019;18:100255.
34. Sağlik Y, Kapicioğlu MI, Güzel B. Spontaneous regression of aneurysmal bone cyst. A case report. Arch Orthop Trauma Surg. 1993;112(4):203–4.
35. Boriani S, De Iure F, Campanacci L, et al. Aneurysmal bone cyst of the mobile spine: report on 41 cases. Spine (Phila Pa 1976). 2001;26(1):27–35.

36. Burch S, Hu S, Berven S. Aneurysmal bone cysts of the spine. Neurosurg Clin N Am. 2008;19(1):41–7.
37. Ameli NO, Abbassioun K, Saleh H, Eslamdoost A. Aneurysmal bone cysts of the spine. Report of 17 cases. J Neurosurg. 1985;63(5):685–90.
38. Lichtenstein L. Aneurysmal bone cyst; observations on fifty cases. J Bone Joint Surg Am. 1957;39-a(4):873–82.
39. Liu JK, Brockmeyer DL, Dailey AT, Schmidt MH. Surgical management of aneurysmal bone cysts of the spine. Neurosurg Focus. 2003;15(5):E4.
40. Papagelopoulos PJ, Currier BL, Shaughnessy WJ, et al. Aneurysmal bone cyst of the spine. Management and outcome. Spine (Phila Pa 1976). 1998;23(5):621–8.
41. Hay MC, Paterson D, Taylor TK. Aneurysmal bone cysts of the spine. J Bone Joint Surg Br. 1978;60-b(3):406–11.
42. Keenan S, Bui-Mansfield LT. Musculoskeletal lesions with fluid-fluid level: a pictorial essay. J Comput Assist Tomogr. 2006;30(3):517–24.
43. Chan MS, Wong YC, Yuen MK, Lam D. Spinal aneurysmal bone cyst causing acute cord compression without vertebral collapse: CT and MRI findings. Pediatr Radiol. 2002;32(8):601–4.
44. Zileli M, Isik HS, Ogut FE, Is M, Cagli S, Calli C. Aneurysmal bone cysts of the spine. Eur Spine J. 2013;22(3):593–601.
45. Parker J, Soltani S, Boissiere L, Obeid I, Gille O, Kieser DC. Spinal aneurysmal bone cysts (ABCs): optimal management. Orthop Res Rev. 2019;11:159–66.
46. Boriani S, Lo SF, Puvanesarajah V, et al. Aneurysmal bone cysts of the spine: treatment options and considerations. J Neuro-Oncol. 2014;120(1):171–8.
47. Boriani S, Bandiera S, Donthineni R, et al. Morbidity of en bloc resections in the spine. Eur Spine J. 2010;19(2):231–41.
48. Amendola L, Simonetti L, Simoes CE, Bandiera S, De Iure F, Boriani S. Aneurysmal bone cyst of the mobile spine: the therapeutic role of embolization. Eur Spine J. 2013;22(3):533–41.
49. Capanna R, Bertoni F, Bettelli G, et al. Aneurysmal bone cysts of the pelvis. Arch Orthop Trauma Surg. 1986;105(5):279–84.
50. Maccarty CS, Dahlin DC, Doyle JB Jr, Lipscomb PR, Pugh DG. Aneurysmal bone cysts of the neural axis. J Neurosurg. 1961;18:671–7.
51. Desai SB, O'Brien C, Shaikh R, et al. Multidisciplinary management of spinal aneurysmal bone cysts: a single-center experience. Interv Neuroradiol. 2019;25(5):564–9.
52. Liu X, Han SB, Si G, et al. Percutaneous albumin/doxycycline injection versus open surgery for aneurysmal bone cysts in the mobile spine. Eur Spine J. 2019;28(6):1529–36.
53. Kim LJ, Spetzler RF. Classification and surgical management of spinal arteriovenous lesions: arteriovenous fistulae and arteriovenous malformations. Neurosurgery. 2006;59(5 Suppl 3):S195–201; discussion S3–13.
54. McDougall CG, Deshmukh VR, Fiorella DJ, Albuquerque FC, Spetzler RF. Endovascular techniques for vascular malformations of the spinal axis. Neurosurg Clin N Am. 2005;16(2):395–410, x–xi.
55. Kiyosue H, Matsumaru Y, Niimi Y, et al. Angiographic and clinical characteristics of thoracolumbar spinal epidural and dural arteriovenous fistulas. Stroke. 2017;48(12):3215–22.
56. Takai K, Shojima M, Imai H, Saito N, Taniguchi M. Microsurgical and endovascular treatments of spinal extradural arteriovenous fistulas with or without intradural venous drainage. World Neurosurg. 2018;111:e819–29.
57. Brinjikji W, Lanzino G. Endovascular treatment of spinal arteriovenous malformations. Handb Clin Neurol. 2017;143:161–74.
58. Veznedaroglu E, Nelson PK, Jabbour PM, Rosenwasser RH. Endovascular treatment of spinal cord arteriovenous malformations. Neurosurgery. 2006;59(5 Suppl 3):S202–9; discussion S203–13.
59. Fairhall JM, Reddy R, Sears W, Wenderoth JD, Stoodley MA. Successful endovascular and surgical treatment of spinal extradural metameric arteriovenous malformation. Case report. J Neurosurg Spine. 2010;13(6):784–8.
60. Gross BA, Du R. Spinal juvenile (Type III) extradural-intradural arteriovenous malformations. J Neurosurg Spine. 2014;20(4):452–8.

Trauma

25

Douglas Dickson

Traumatic spine fractures may result from high-speed injuries which includes motor vehicle collisions, fall from heights and blunt trauma to the head. The cervical spine has a unique anatomic makeup divided into the upper cervical spine and subaxial cervical spine.

Cervical spine injuries remain a significant problem in our present society, over one million acute spine injuries occur in the United States and one third of the spinal cord injuries occur in the cervical spine.

Cervical Spine Anatomy

It is composed of seven vertebrae. The atlas C1 is a ring which articulates with the occiput. It is important to note that the C1 has no body or spinous processes. The axis C2 is so named because it pivots around the atlas turning to rotate the head. The atlas has a vertical extension, the dens, which articulates with C1. There is a canal for the vertebral arteries are located bilaterally.

The upper cervical spine is from occiput to C2 and the subaxial cervical spine is from C3 to C7. Most traumatic cervical spine injury occur in the subaxial region of the cervical spine between C3 and C7.The minority of cervical spine injuries that occur in the upper cervical spine carries with it a high rate of mortality and most people who experience such type of injury expire. Any blunt trauma to the head should raised a high suspicion for at least a cervical spine injury.

D. Dickson (✉)
Department of Orthopaedic Surgery, UT Southwestern Medical Center, Dallas, TX, USA
e-mail: Douglas.Dickson@UTSouthwestern.edu

© The Author(s), under exclusive license to Springer Nature Switzerland AG 2022

C. E. Noe (ed.), *Multidisciplinary Spine Care*,
https://doi.org/10.1007/978-3-031-04990-3_25

Evaluation

In patients who are suspected to have a cervical spine injury a three view cervical spine X-rays are normally required which includes anteroposterior, lateral and odontoid views, also CT scans have been used more frequently as it provides a better detail of the bony element. MRI is warranted to evaluate the neural elements and the posteroligamentous complex.

A thorough physical examination is of utmost importance documenting all the neurologic findings in detail assessing the motor, sensory and all other reflexes including pathologic reflexes.

There are several types of cervical spine fractures; Jefferson fractures are caused by compression of the base of the skull against C1 resulting in the cracking of the ring of C1. This injury is best identified on the open mouth odontoid x-ray and widening of the lateral masses of C1 away from the dens due to disruption of the C1 ring. Most of these injuries can be treated non surgically in a hard cervical collar.

C2 fractures are usually caused by hyper flexion or hyper extension injuries. They comprise 8% of all the injuries associated with C1 fractures. C2 fractures often present as dens fractures resultant of hyper flexion injuries or Hangman fractures resultant of hyperextension injuries which manifest with bilateral fractures the pedicles of C2. Fractures above C4 can be associated with paralysis of muscles of respiration. The diaphragm is innervated by the C3–C5 nerve roots. Fractures of the mid cervical region are associated with dysfunction of the upper extremities more often than the lower extremities.

Surgical Indications

Mechanical instability, neurologic demise and compression of the neural elements are the main indications for surgical intervention.

There are several classification systems available to classify cervical spine fractures and help with treatment. The goal of the classification system is to aid in identification of injury pattern, communication between physicians including injury mechanism, injury morphology and aid in treatment. One classification system that seems to try and address all these qualities is the cervical spine injury classification severity score (CSISS) which groups the cervical spine segment into columns [1]. The anterior column, posterior column and the right and left pillars. The anterior column consists of the vertebral body the disc and the posterior longitudinal ligaments, the posterior column consists of the lamina, posterior ligamentous complex, and the pillars consists of the lateral masses and pedicle. Each column is graded from 0 to 5, with 0 being nondisplaced and 5 being maximum displacement or worst possible injury. The CSISS causes the surgeon to evaluate all the critical components and columns of the injury. Patients with a score of 7 or greater most likely underwent surgery compared to those with a score below 7.

The Subaxial cervical injury Classifications described by Vaccaro focuses on the subaxial spine (C3–C7) [2]. It consists of three main categories: injury

morphology, discoligamentous status and neurologic status. Morphology is grouped into for subtypes: compression, which is assigned a numerical value of 1, burst fracture (2), Distraction injury (3), and Rotational injury (4). For the discoligamentous complex (DLC) there are three subcategories: Intact (0), Indeterminant (1) and disrupted (2) and the neurologic status if there is no neurologic change, we assign a numeric grade of 0, a nerve root injury is assigned 1 complete cord injury 2, incomplete cord injury 3 and continuous cord compression in the setting of a neurologic deficit get an additional 1 point. A total score less than or equal to 3 warrants non operative treatment, Total score of 4 either treatment Non operative versus surgical treatment can be chosen and total numeric score of five or greater warrants surgical intervention.

The goal of surgery is to stabilize the spinal column in the midst of instability and decompress the neural elements in the midst of compression. Timely decompression and stabilization of the spinal column when medically feasible in an unstable cervical spine fracture is highly encouraged. The surgical approaches could be anteriorly or from a posterior approach or a combination depending on the complexity of the injury.

Thoracolumbar Spine Injuries

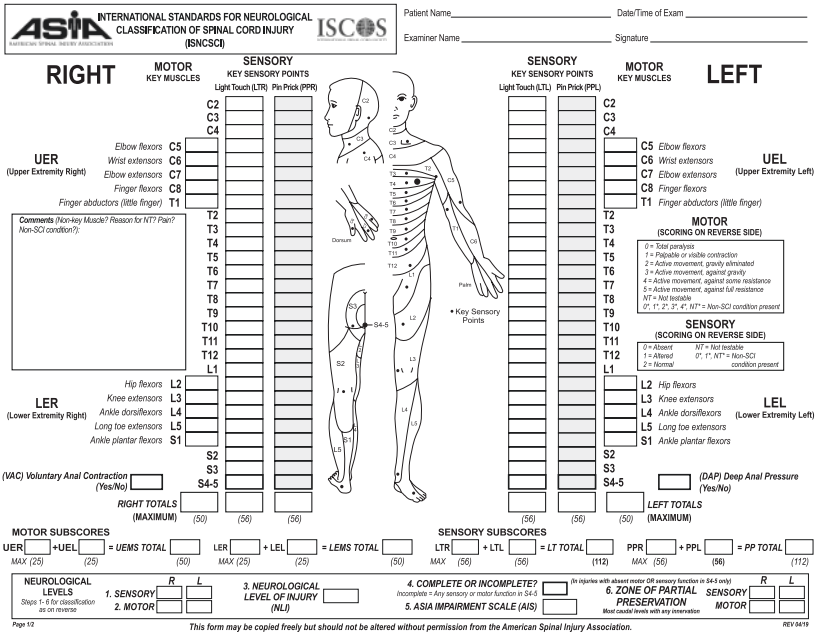
The thoracolumbar spine lends itself with a unique anatomic make up. We have the rigid thoracic spinal column made up of 12 vertebral segments in a kyphotic orientation and the lumbar spine made up of 5 vertebral segments, mobile and lordotic. Most thoracolumbar fractures occur from fall from heights and high energy trauma situations including motor vehicle collisions for the younger patients and all from standing for the older patients most likely due to their inherently less optimal bone quality.

Evaluation of patients with suspected thoracolumbar fractures requires detailed neurologic examination which includes motor sensory and reflex functions of the neural axis. Muscle and sensory grading in addition using the ASIA impairment scale to assess neurologic status is paramount [3] (Fig. 25.1). Figure 25.1a, b summarize the American Spinal Injury Association Standard Neurological Classification of Spinal Cord Injury.

Radiographic Evaluation to assess for instability is required to aid with surgical decision making. Anteroposterior and lateral imaging of the segment and also all contiguous segments of the spine is recommended us there are times in which there may be multiple fractures in different segments of the spine. Imaging of the whole spine is recommended in high energy traumas. CT scan and MRIs are also needed to help classify and assess stability. Immediate mechanical stability can be assessed by looking at the injury morphology, integrity of the posteroligamentous complex assesses the long-term stability and neurologic status assesses the neurologic state.

There have been various classifications systems developed to help classify these fractures and aid with treatment. The thoracolumbar injury classification system (TLICS) is more commonly used due to its comparable simplicity and

a



b

Muscle Function Grading

0 = Total paralysis

1 = Palpable or visible contraction

2 = Active movement, full range of motion (ROM) with gravity eliminated

3 = Active movement, full ROM against gravity

4 = Active movement, full ROM against gravity and moderate resistance in a muscle specific position

5 = (Normal) active movement, full ROM against gravity and full resistance in a functional muscle position expected from an otherwise unimpaired person

NT = Not testable (i.e. due to immobilization, severe pain such that the patient cannot be graded, amputation of limb, or contracture of > 50% of the normal ROM)

0*, 1*, 2*, 3*, 4*, NT* = Non-SCI condition present [a]

Sensory Grading

0 = Absent **1** = Altered, either decreased/impaired sensation or hypersensitivity

2 = Normal **NT** = Not testable

0*, 1*, NT* = Non-SCI condition present [a]

[a] Note: Abnormal motor and sensory scores should be tagged with a '*' to indicate an impairment due to a non-SCI condition. The non-SCI condition should be explained in the comments box together with information about how the score is rated for classification purposes (at least normal / not normal for classification).

When to Test Non-Key Muscles:

In a patient with an apparent AIS B classification, non-key muscle functions more than 3 levels below the motor level on each side should be tested to most accurately classify the injury (differentiate between AIS B and C).

Movement	Root level
Shoulder: Flexion, extension, adbuction, adduction, internal and external rotation **Elbow:** Supination	C5
Elbow: Pronation **Wrist:** Flexion	C6
Finger: Flexion at proximal joint, extension **Thumb:** Flexion, extension and abduction in plane of thumb	C7
Finger: Flexion at MCP joint **Thumb:** Opposition, adduction and abduction perpendicular to palm	C8
Finger: Abduction of the index finger	T1
Hip: Adduction	L2
Hip: External rotation	L3
Hip: Extension, abduction, internal rotation **Knee:** Flexion **Ankle:** Inversion and eversion **Toe:** MP and IP extension	L4
Hallux and Toe: DIP and PIP flexion and abduction	L5
Hallux: Adduction	S1

ASIA Impairment Scale (AIS)

A = Complete. No sensory or motor function is preserved in the sacral segments S4-5.

B = Sensory Incomplete. Sensory but not motor function is preserved below the neurological level and includes the sacral segments S4-5 (light touch or pin prick at S4-5 or deep anal pressure) AND no motor function is preserved more than three levels below the motor level on either side of the body.

C = Motor Incomplete. Motor function is preserved at the most caudal sacral segments for voluntary anal contraction (VAC) OR the patient meets the criteria for sensory incomplete status (sensory function preserved at the most caudal sacral segments S4-5 by LT, PP or DAP), and has some sparing of motor function more than three levels below the ipsilateral motor level on either side of the body. (This includes key or non-key muscle functions to determine motor incomplete status.) For AIS C – less than half of key muscle functions below the single NLI have a muscle grade ≥ 3.

D = Motor Incomplete. Motor incomplete status as defined above, with at least half (half or more) of key muscle functions below the single NLI having a muscle grade ≥ 3.

E = Normal. If sensation and motor function as tested with the ISNCSCI are graded as normal in all segments, and the patient had prior deficits, then the AIS grade is E. Someone without an initial SCI does not receive an AIS grade.

Using ND: To document the sensory, motor and NLI levels, the ASIA Impairment Scale grade, and/or the zone of partial preservation (ZPP) when they are unable to be determined based on the examination results.

AMERICAN SPINAL INJURY ASSOCIATION

INTERNATIONAL STANDARDS FOR NEUROLOGICAL CLASSIFICATION OF SPINAL CORD INJURY

ISCOS

Page 2/2

Steps in Classification

The following order is recommended for determining the classification of individuals with SCI.

1. Determine sensory levels for right and left sides.
The sensory level is the most caudal, intact dermatome for both pin prick and light touch sensation.

2. Determine motor levels for right and left sides.
Defined by the lowest key muscle function that has a grade of at least 3 (on supine testing), providing the key muscle functions represented by segments above that level are judged to be intact (graded as a 5).
Note: in regions where there is no myotome to test, the motor level is presumed to be the same as the sensory level, if testable motor function above that level is also normal.

3. Determine the neurological level of injury (NLI).
This refers to the most caudal segment of the cord with intact sensation and antigravity (3 or more) muscle function strength, provided that there is normal (intact) sensory and motor function rostrally respectively.
The NLI is the most cephalad of the sensory and motor levels determined in steps 1 and 2.

4. Determine whether the injury is Complete or Incomplete.
(i.e. absence or presence of sacral sparing)
*If voluntary anal contraction = **No** AND all S4-5 sensory scores = **0** AND deep anal pressure = **No**, then injury is **Complete.***
*Otherwise, injury is **Incomplete**.*

5. Determine ASIA Impairment Scale (AIS) Grade.
Is injury Complete? If YES, AIS=A
NO
Is injury Motor Complete? If YES, AIS=B
NO (No=voluntary anal contraction OR motor function more than three levels below the motor level on a given side, if the patient has sensory incomplete classification)
Are at least half (half or more) of the key muscles below the neurological level of injury graded 3 or better?
NO AIS=C **YES** AIS=D
If sensation and motor function is normal in all segments, AIS=E
Note: AIS E is used in follow-up testing when an individual with a documented SCI has recovered normal function. If at initial testing no deficits are found, the individual is neurologically intact and the ASIA Impairment Scale does not apply.

6. Determine the zone of partial preservation (ZPP).
The ZPP is used only in injuries with absent motor (no VAC) OR sensory function (no DAP, no LT and no PP sensation) in the lowest sacral segments S4-5, and refers to those dermatomes and myotomes caudal to the sensory and motor levels that remain partially innervated. With sacral sparing of sensory function, the sensory ZPP is not applicable and therefore "NA" is recorded in the block of the worksheet. Accordingly, if VAC is present, the motor ZPP is not applicable and is noted as "NA".

Fig. 25.1 (**a**) American Spinal Injury Association Standard Neurological Classification of Spinal Cord Injury. (**b**) American Spinal Injury Association Standard Neurological Classification of Spinal Cord Injury

reproducibility [4]. The TLICS system looks at three critical aspects of the injury: injury morphology, integrity of posteroligamentous complex and neurologic status of the patient.

Injury morphology is grouped into four main subtypes: 1: Compression fracture, which the fracture involves the anterior column of the vertebral body, burst fracture involves the anterior and middle column of the vertebral body, translational/rotational injury and flexion distraction injury. The compression fracture is assigned a numerical value of 1, burst 2, translational/rotational injury 3 and flexion distraction injury (4) (Fig. 25.2). Figure 25.2 is a classification of the main subtypes of spine fractures.

Integrity of the posteroligamentous complex (PLC) is better visualized with the MRI. An intact PLC is given a numerical value of (1), suspected/indeterminant injury (2), and disrupted PLC (3) (Fig. 25.3). Figure 25.3 is a classification of posterior ligamentous complex injury classification.

The thoracolumbar injury classification and severity score (TLICS) is also used to guide surgical decision making.

With the neurologic status, a patient with no neurologic deficit is assigned a numerical value of zero (0), nerve root injury (2), complete cord injury (2) and an incomplete cord injury and cauda equina injury (3).

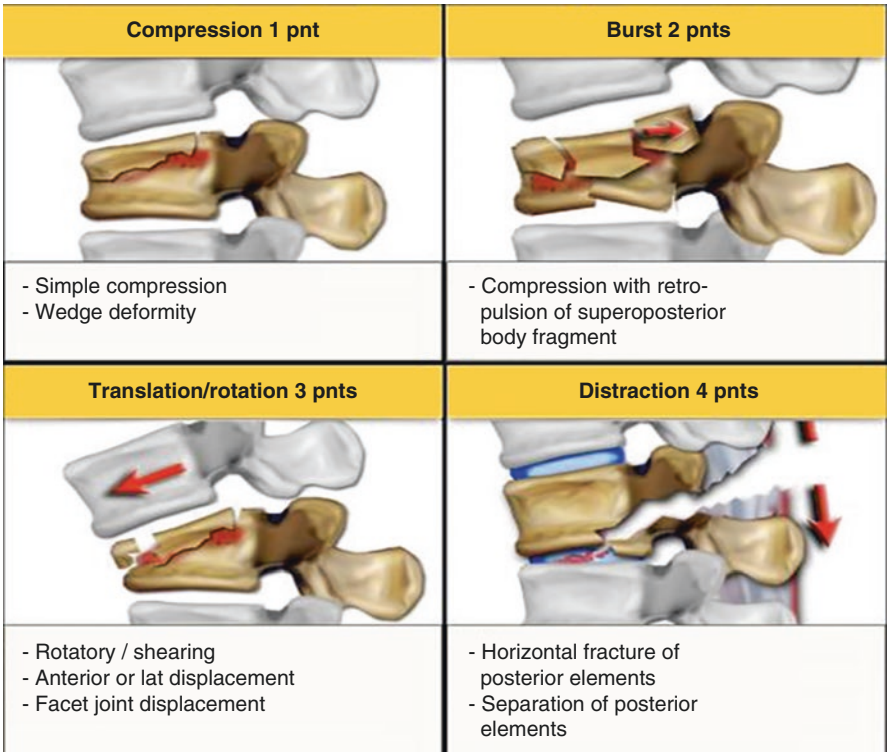


Fig. 25.2 Four main subtypes of spine fractures

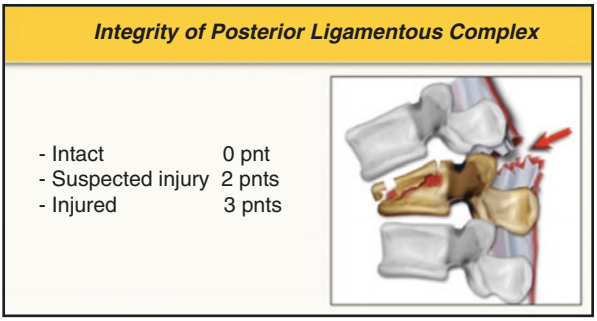


Fig. 25.3 Posterior ligamentous complex injury classification

TLICS 3 independent predictors				
1	**Morphology** immediate stability	- Compression - Burst - Translation/rotation - Distraction	1 2 3 4	- Radiographs - CT
2	**Integrity of PLC** longterm stability	- Intact - Suspected - Injured	0 2 3	- MRI
3	**Neurological status**	- Intact - Nerve root - Complete cord - Incomplete cord - Cauda equina	0 2 2 3 3	- Physical examination
Predicts		**- Need for surgery**	0 – 3 4 > 4	- nonsurgical - surgeon's choice - surgical

Fig. 25.4 Thoracolumbar injury classification and severity score. https://radiologyassistant.nl/musculoskeletal/spine/tlics-classification

Patient with a total score of 3 or less are treated non operatively, A score of 4 patient could be treated non operatively or surgically. A score of 5 or greater warrants surgical intervention. Figure 25.4 is a thoracolumbar injury classification and severity score.

The TLICS system helps in surgical decision making and has been validated by various surgeons due to the scoring system being reliable and reproducible. It takes into consideration all the important segments of the injury and helps the treating physician to consider all the critical aspects of the injury.

References

1. Anderson PA, Moore TA, Davis KW, Molinari RW, Resnick DK, Vaccaro AR, Bono CM, Dimar JR 2nd, Aarabi B, Leverson G, Spinal Trauma Study Group. Cervical spine injury severity score. Assessment of reliability. J Bone Joint Surg Am. 2007;89(5):1057–65. https://doi.org/10.2106/JBJS.F.00684. PMID: 17473144.

2. Vaccaro AR, Hulbert RJ, Patel AA, Fisher C, Dvorak M, Lehman RA Jr, Anderson P, Harrop J, Oner FC, Arnold P, Fehlings M, Hedlund R, Madrazo I, Rechtine G, Aarabi B, Shainline M, Spine Trauma Study Group. The subaxial cervical spine injury classification system: a novel approach to recognize the importance of morphology, neurology, and integrity of the disco-ligamentous complex. Spine (Phila Pa 1976). 2007;32(21):2365–74. https://doi.org/10.1097/BRS.0b013e3181557b92. PMID: 17906580.
3. ASIA and ISCoS International Standards Committee. The 2019 revision of the International Standards for Neurological Classification of Spinal Cord Injury (ISNCSCI)-What's new? Spinal Cord. 2019;57(10):815–7.
4. Vaccaro AR, Lehman RA Jr, Hurlbert RJ, et al. A new classification of thoracolumbar injuries: the importance of injury morphology, the integrity of the posterior ligamentous complex, and neurologic status. Spine (Phila Pa 1976). 2005;30:2325–33.

Post Surgical Spinal Deformities

26

Sai Chilakapati, James P. Caruso, Zachary Johnson, and Owoicho Adogwa

Introduction

Any patient who has undergone spinal fusion or decompression surgery is at risk for developing post-surgical spinal deformity. The etiologies of these deformities are diverse and include flatback syndrome, junctional kyphosis, post-laminectomy deformities, adjacent-level degeneration, post-operative instability and pseudarthrosis. Additionally, factors such as advancing age compromise bony structures and soft tissue, making these problems particularly challenging for the spine surgeon. While postsurgical spinal deformities may be a consequence of the unavoidable natural history of spinal fusion or decompression, the majority of these deformities may be prevented by a fundamental understanding of sagittal balance parameters and surgical techniques to preserve spinal stability intraoperatively and to ensure proper alignment.

Maintenance of spinal stability and horizontal gaze are important to humans. Panjabi et al., defined spinal stability as the ability of the spine to maintain its pattern of displacement such that no neurological deficit, major deformity or incapacitating pain occurs under physiologic loads [1]. Stability is maintained through a complex multi-articular system and is necessary for the efficient transfer of forces between the upper and lower limbs, as well as to prevent mechanical deterioration that occurs as a result of local compensatory mechanisms. Loss of spinal stability manifests as an inability of the spine to maintain an adequate load-bearing capacity.

S. Chilakapati · J. P. Caruso · Z. Johnson · O. Adogwa (✉)
Department of Neurological Surgery, UT Southwestern Medical Center, Dallas, TX, USA
e-mail: Sai.Chilakapati@UTSouthwestern.edu; James.Caruso@phhs.org; Zachary.Johnson@phhs.org; owoicho.adogwa@UTsouthwestern.edu

© The Author(s), under exclusive license to Springer Nature Switzerland AG 2022

C. E. Noe (ed.), *Multidisciplinary Spine Care*,
https://doi.org/10.1007/978-3-031-04990-3_26

Malalignment of the spine is a common manifestation of spinal instability and the hallmark of spinal deformity. Neglected spinal malalignment often progresses to marked functional limitation.

Similarly, severe sagittal imbalance may render one socially and functionally debilitated, as it compromises maintenance of horizontal gaze. Thus, performing activities of daily living, including driving, swallowing, speaking, and upkeep of personal hygiene may become more difficult with worsening kyphosis. Glassman and colleagues demonstrated that quality of life is negatively affected by mild sagittal imbalance and is significantly compromised as sagittal imbalance increases [2]. Progressive kyphosis is better tolerated in the thoracic spine and poorly tolerated in the cervical and lumbar spine [2–4]. In addition to pain and poor general health, postoperative spinal deformity may result in acute or progressive neurologic injury [5, 6]. For example, Watanabe and colleagues reported that the neurologic status of 20% of patients who sustained an acute proximal junctional fracture deteriorated from Frankel E to Frankel B [6]. After proximal extension of the instrumentation and revision arthrodesis, these patients recovered to Frankel D [6]. Thus, all surgical strategies should be utilized intraoperatively to both avoid destabilizing the spine and avoid instrumenting and fusing the spine in a misaligned position. These surgical strategies and the etiologies of post-surgical spinal deformities are addressed in detail in this chapter.

Decompression-Related Postoperative Spinal Deformities

Posterior Decompression

Many cases of post-surgical deformity develop as a result of destabilization following multilevel laminectomies in the cervical, thoracic, and lumbar spine. While this approach is generally efficacious and safe in relieving neural element compression, it hinges on removing an appropriate amount of lamina and compressive pathology within the lateral recess. Inadequate bony decompression can result in persistent pain and disability, while aggressive extended facetectomy and pars resection can jeopardize spinal column stability. Most commonly, this manifests as progressive kyphosis in the cervical spine and accelerated segmental degeneration and worsening spondylolisthesis in the lumbar spine.

In the cervical spine, one of the most encountered postoperative deformity is kyphosis. Kyphosis is the forward angulation of the spine that occurs secondary to decompensation in the sagittal plane and is the most common naturally occurring deformity in elderly patients because of disc degeneration. Anatomically, intervertebral disc have greater height anteriorly versus posteriorly, contributing to the lordortic shape of the cervical spine. Cervical kyphosis occurs with aging and progressive disc desiccation due to asymmetric disc height loss (anterior > posterior). Additionally, the cervical spine is intrinsically unstable. This relative instability is biologically advantageous as it facilitates greater mobility in frontal, coronal and sagittal planes than other anatomical regions of the spine; however, this relative

instability renders the cervical spine highly dependent on its soft tissue envelop for stability. Posterior decompression disrupts these structures, such that removal of as little as 25% of the bilateral facets can predispose patients to kyphotic deformity. Because of this small margin of error, prophylactic instrumentation and fusion should follow multilevel cervical laminectomy to decrease the risk of post-laminectomy kyphosis [7, 8].

While the lumbar spine can better tolerate extended facetectomy without developing acute instability [9, 10], facet integrity in the lumbar spine remains important for rotational and axial stability. In a finite element model amount, Zander et al. demonstrated that unilateral hemifacetectomy led to increased intersegmental rotation during axial rotation [10]. Lumbar facet joint compromise also results in greater load transmission to the intervertebral disc annulus and anterior longitudinal ligament during axial loading [9, 11]. While this may not produce acute instability, it accelerates segmental degeneration and will often require posterior instrumentation and fusion [9]. In addition to adequate facet preservation, maintaining integrity of the lateral pars interarticularis and true pars in the lumbar spine is important for spinal stability. When performing a lumbar laminectomy, the lateral pars should be exposed to determine its width so as to avoid over resection. Care should also be taken when resecting the superior edge of the distal vertebra's lamina during decompression. An overly aggressive resection at this site can also lead to an iatrogenic pars fracture. Unlike facet destruction, pars fractures can lead to acute postoperative instability and thus require posterior instrumentation and fusion.

Clinical Presentation

Post-surgical deformities have diverse and complex clinical presentations. The majority of patients present with mechanical pain with or without neurological deficits, months after the primary surgery. It is not uncommon for patient to feel well after the index surgery and with an increase in physical activity, increasing pain amount, neurological deficits or deformity are observed. Initially, the pain is mechanical, worse with loading and improved with unloading; however, later in the disease course, the pain from compression of the neural structures predominate. Self-imposed conservative treatments are initiated and other forms of nonprescription care, such as home traction devices, chiropractic care, use of over-the-counter prescription medications and muscle strengthening. In a subset of patients, these conservative measures can be successful, however, with increasing instability, deformity and neurological deficits, repeat decompression with instrumented fusion is usually necessary to address mechanical pain.

Instrumentation-Related Postoperative Deformities

The major goal of posterior spinal instrumentation and fusion is to restore spinal stability and maintain or improve spinal alignment. Unfortunately, spinal instrumentation can easily result in iatrogenic spinal deformity, particularly iatrogenic loss of lumbar lordosis, or "flatback", or "fixed sagittal imbalance". This

phenomenon was first described in 1973 by Doherty et al. [12], in patients with scoliosis treated with Harrington instrumentation. While this distraction-based system arrested progression of the coronal deformity, it caused or contributed to sagittal plane malalignment and iatrogenic flatback. Booth et al. classified iatrogenic flatback into two categories: Type 1—segmental loss of lumbar lordosis or lumbar kyphosis with preservation of normal sagittal balance; and Type 2—global flatback syndrome with fixed positive sagittal imbalance [13]. In type 1, patients can compensate with hyperextending the thoracic spine (thoracic compensation) and pelvic retroversion. These compensatory mechanisms are usually effective; however, with progressive paraspinal muscle fatigue, maintaining horizontal gaze and sagittal balance requires increasing pelvic retroversion and hip extension. Since pelvic retroversion occurs mainly in patients with high pelvic incidence, patients with low pelvic incidence have limited ability to retrovert their pelvis and are less able to functionally tolerate a flatback. A wide range of pathologies are implicated in the formation of flatback syndrome. These include placement of distraction instrumentation in the lower lumbar spine or sacrum, fixed thoracic hypokyphosis, or pseudarthrosis resulting in loss of sagittal plane correction. Placement of distraction instrumentation into the lower lumbar spine or sacrum is the most common reported cause of flatback syndrome. Although modern posterior instrumentation improves sagittal alignment correction, it can also result in iatrogenic flatback if implants are misused or meticulous attention to lumbar lordosis is not maintained or improved at the time of operation. Blondel et al. [14], emphasized the importance of adequate restoration of thoracic kyphosis for patients undergoing surgical correction of adolescent idiopathic scoliosis. Similarly, Trobisch et al. [15], noted that in patients undergoing selective thoracic fusion with pedicle screw constructs, high preoperative lumbar lordosis and surgical decrease in thoracic kyphosis were risk factors for postoperative loss of lumbar lordosis.

Clinically, patients with a flatback deformity present with stance and gait abnormalities with increasing pain and fatigue. When standing upright patients compensate with hyperextension of unfused segments in the thoracic and lumbar spine along with hip extension. In severe cases, where compensatory mechanisms have been exhausted, knee flexion occurs to bring the torso over the center of gravity.

An appreciation of the various compensatory mechanisms deployed to maintain balance and horizontal gaze is of utmost importance and necessitates a comprehensive assessment of regional and global balance of any patient undergoing spinal surgery. Radiologic assessment should include a standing full-length (36-inch) lateral and anteroposterior film to allow for assessment of sagittal vertical axis (SVA), thoracic kyphosis (TK) and spinopelvic parameters (pelvic incidence (PI), sacral slope (SS) and pelvic tilt (PT)). Intraoperatively, meticulous attention to appropriate positioning of the hip is critical to ensure adequate lumbar lordosis and minimize the risk of inducing iatrogenic flatback [16]. In the setting of a posterior lumbar fusion, patients should be placed prone with their hips extended to maximize lumbar lordosis [16, 17]. In several clinical studies, intraoperative hip flexion has been

found to decrease lumbar lordosis by 26–67% [17–20]. This is in contrast to hip extension, which consistently has been shown to increase lumbar lordosis [17–20]. In addition to patient positioning, global and segmental angulation of the lumbar spine should be analyzed carefully. The average lumbar lordosis is approximately 60° [21]. Particular attention should be paid to the segmental angulation between L4 and S1 because these two segments account for nearly 70% of total lordosis and the apex of lumbar lordosis is located, on average, at the L4 vertebral body [21]. As hypolordosis of instrumented L4–S1 segments results in increased loading of the posterior column of the adjacent segments [22], straight rods from L4 to S1 should be avoided and rod contouring should focus on adequate L4–S1 lordosis. In addition to ensuring optimal lumbar lordosis intraoperatively, providing sufficient distal fixation is also important to minimize the risk of postoperative complications and spinal decompensation. Fixation to S1 is sufficiently robust in short posterior constructs that terminate at L3 or below. However, in longer posterior lumbar fusions and instrumentations that extend proximal to L3 for adult spinal deformities, only instrumenting and fusing to S1 is not recommended because it is associated with high rates of complications, such as pseudarthrosis, sagittal deformity, sacral insufficiency fractures and instrumentation failure [23–26]. These complications are presumed to be due to inadequate bone stock of the sacrum, a large number of segments requiring arthrodesis, and unfavorable biomechanics due to a long lever arm at the lumbosacral junction [25]. Enhancing distal fixation may be accomplished with extension to the S2 pedicle; however, McCord and colleagues demonstrated that the most biomechanically stable fixation of the lumbosacral joint included fixation of the ilium bilaterally combined with bilateral S1 fixation [27], as this provides 4 points of fixation of the sacrum and pelvis. Although this construct is biomechanically superior to S1-only fixation, iliac screws are not without complications. With a minimum of 5-year follow-up data, Tsuchiya and coworkers demonstrated that symptomatic iliac screw prominence and the need for screw removal is common (23 of 67 patients; 34.3%) [28]. Other potential difficulties associated with iliac screws are SI joint pain and gait abnormalities, including a short step or "waddle." [25] A technique designed to decrease iliac screw prominence is the S2 alar-iliac screw that has its entry site between the S1 and S2 foramen, which is 15 mm deeper than the posterior superior iliac spine entry point of traditional iliac wing screws [29–31]. In addition to being low-profile, S2 alar-iliac screws provide another point for distal lumbosacral fixation, which makes them ideal in revision surgeries for lumbosacral pseudarthrosis or instability [32]. Although iliac fixation has evolved, the important fundamental concept is that sacral fixation at S1 should consistently be supplemented with either unilateral or bilateral iliac fixation for long posterior lumbar fusions. Additionally, interbody grafting of L4–L5 and L5–S1 should be used in this setting to minimize stress of the lumbosacral instrumentation. With all the aforementioned intraoperative techniques in mind, one may minimize the chances of producing an iatrogenic flatback and decrease the risk of postoperative pseudarthrosis, instrumentation failures, and significant spinal imbalance postoperatively.

Adjacent Segment Disease

The proximal and distal extents of posterior fusions are all considered "at risk" for failure as a result of increased loads and motion between the last instrumented vertebrae and unfused adjacent segment [6, 19, 33–35]. Any pathology that occurs at the proximal or distal aspects of a posterior fusion is referred to as adjacent segment degeneration (ASD) and is classified based on the time at which it occurs postoperatively. Acute complications at the junctions of a posterior construct are commonly a result of a fracture at the proximal vertebrae, instrumentation pullout, or failure of the soft-tissue posterior ligamentous structures [5, 36–41]. These primarily occur at the proximal junction of a thoracolumbar fusion extending from the pelvis into the upper lumbar or thoracic spine with spondylolisthesis occurring more commonly at the upper thoracic spine and vertebral body fractures occurring more commonly at the lower thoracic spine.

Risk factors for acute adjacent segment degeneration include, but are not limited to, osteopenia, age greater than 55 years, obesity, and severe global sagittal imbalance [5, 6, 39, 40, 42]. In the cervical spine, adjacent segment degeneration occurs most commonly in the subaxial spine. The unique anatomy of the cervical spine coupled with reliance on the soft tissue envelope explain the difference in the development of adjacent segment degeneration in this region as opposed to the lumbosacral region. Radiographically, the prevalence of adjacent segment degeneration ranges from 8.5% to 20%. Herkowitz et al. [43], in a randomized surgical trial of anterior cervical discectomy and fusion versus posterior cervical foraminotomy without fusion, noted ASD rate of 41% for the ACDF cohort and 50% for the posterior cervical foraminotomy cohort. Gore et al. [44, 45], in a 5-year prospective study observed new spondylosis in 25% of patients and progression of preexisting spondylosis in another 25%. Bohlman et al. [46], in a 6-year prospective study observed an ASD rate of 9% in patients that underwent ACDF. Risk factors after cervical spine surgery for development of ASD include sagittal malalignment, existence of neural compression at the adjacent level at the time of the index surgery, and surgery in the lower subaxial spine (C5–C6 and C6–C7).

In the lumbar spine, adjacent segment degeneration occurs most commonly after lumbar fusions. Lehman et al. [47], in a retrospective series of 62 patients followed for 33 months reported adjacent segmental instability in 45% of patients. Etebar et al. [48], observed adjacent segment degeneration rates of 14% of patients following lumbar fusions, the majority (83%) of whom were postmenopausal females. An understanding of sagittal balance parameters at the index surgery is necessary to decrease the risk of adjacent segment degeneration. Although spinal and pelvic parameters are well known by spine surgeons, their application in surgical planning and strategy during treatment of spinal disorders is not well understood, leading to a common source of mistakes. Current teaching and application of spinal and pelvic parameters has simplified our understanding; such that within the ideal sagittal balance, adaptable reciprocal value of pelvic incidence minus lumbar lordosis should be almost constant. As a result, surgical strategy has focused on restoration of this ideal angular value of lumbar lordosis as determined by the pelvic incidence;

however, this theory although simple to understand is based on the presumption that only a single geometric spinal shape exists. Roussouly et al. [21], recently proposed a classification of spinopelvic morphotypes that accounts for both angular measurements as well as geometric shape. The four subgroups are—Type 1 characterized by low PI and low SS (<35°) with apex of LL in the center of the L5 vertebral body; Type 2—physiologic flatback characterized by low PI and low SS (<35°) with apex of LL in the center of the base of L4 vertebral body; Type 3--characterized by PI between 50° and 60°, 35° < SS < 45° with apex of LL in the center of the L4 vertebral body; and Type 4-- characterized by high PI and SS > 45° with apex of LL at the base of the L3 vertebral body. The length of the lower arch of lumbar lordosis, measured as the distance from the apex to the sacral plateau increases as one moves from types 1 through type 4. Ignoring the shape of the spine in surgical planning leads to various technical errors, frequently encountered after short segment interbody fusions where improper placement of the interbody spacer and inadequate rod bending results in loss of lordosis and adjacent compensation of the first nonfused level. Biomechanically, lumbar fusions alter the kinematics of the adjacent segments, redistributing mobility to the juxtafused segments and cause loading of unfused segments beyond their physiological limits. In in-vitro studies, Weinhoffer et al. demonstrated a correlation between number of fusion levels and increased intradiscal pressures. Similarly, Chow et al. [49], noted increased intradiscal pressures and hypermobility at adjacent segments following lumbar fusions. Take the following case example (Fig. 26.1). A 55-year-old male with degenerative lumbar

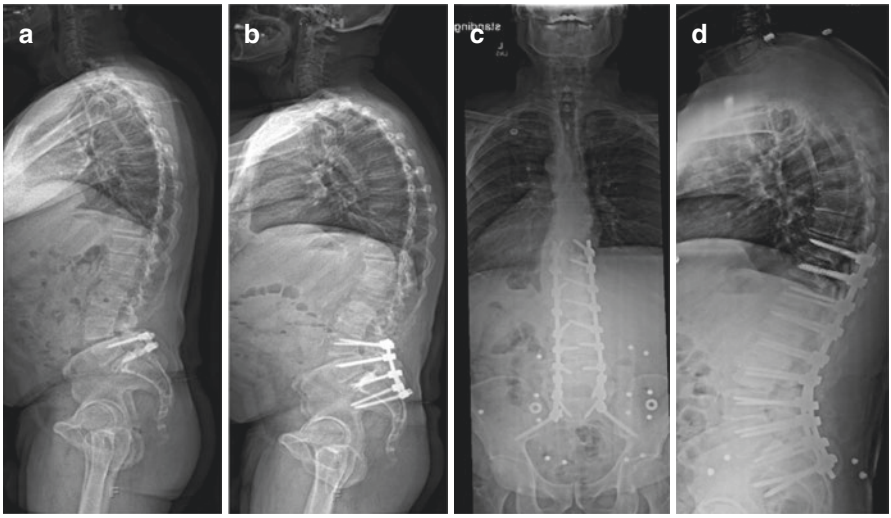


Fig. 26.1 A 55-year-old male who underwent an L5–S1 fusion for lumbar stenosis and spondylolisthesis. (**a**) 56 year old patient with progressive adjacent segment disease corrected with a T10 – sacrum fusion with Iliac fixation. (**b**) represented with adjacent segment disease and underwent a decompression with extension of fusion to L4 and subsequently developed symptomatic adjacent segment disease with progressive deformity. (**c**, **d**), decompression, correction of deformity and stabilization with restoration of sagittal alignment parameters

stenosis and spondylolisthesis at L5–S1 who underwent a L5 decompressive laminectomy with L5 – sacrum fusion at an outside hospital. He represented a few months later with neurogenic claudication and mechanical low back pain secondary to adjacent segment degeneration and subsequently underwent a decompressive laminectomy at L4–L5 with extension of fusion to L4. However, 12 months postoperatively, the patient reported worsening back and bilateral leg pain with progressively worsening difficulty with ambulation. Standing radiographs demonstrated adjacent segment degeneration with development of degenerative lumbar scoliosis (see Fig. 26.1). This was presumed to be secondary to inadequate restoration of sagittal balance and geometric shape based on the Roussouly classification. He was subsequently transferred to our center where he underwent decompression, deformity correction and stabilization. First, with a PI of 65° and pelvic tilt of 30°, he was classified as a Roussouly false type 2. These patients tend to have a high thoracolumbar inflection point with an apex of lumbar lordosis located the base of the L3 vertebral body. A revision T10 – sacrum fusion was performed with generous facet releases at T11 – L3 and Smith-Peterson osteotomy at L2–L3 and L3–L4. Postoperatively, regional and global sagittal and coronal alignment parameters were restored. Two-years postoperatively, adequate alignment was maintained. This scenario exemplifies the sequalae of inattention to sagittal alignment parameters and restoration of geometric shape when performing short segment fusions. Prevention and treatment of adjacent segment degeneration is complicated and controversial. The argument for prophylactically including a degenerative disc above a segment requiring fusion to prevent adjacent segment degeneration and postoperative pain is not uniformly accepted. Furthermore, given the fact that only a subset of patients with radiographic evidence of adjacent segment degeneration have associated clinical symptoms supports the argument against prophylactic fusion. However, there is agreement on when prophylactic adjacent segment fusion might be beneficial. In patients with sagittal malalignment, instability or discography proven axial pain above the segment requiring fusion, inclusion of the adjacent level may play a role in averting adjacent segment degeneration, as well as being vital to obtaining a successful outcome.

Proximal Junctional Failures

Proximal junctional kyphosis (PJK) is a recognized complications in patients undergoing segmental instrumented fusion for spinal deformities. PJK is defined as a 10° or greater increase in kyphosis at the proximal junction measured as the angle from the caudal endplate of the uppermost instrumented vertebrae to the cephalad endplate of the vertebrae two segments cranial to the UIV. PJK is associated with structural failure at the UIV. Structural failure is considered a vertebral body fracture, disruption of the posterior ligamentous complex, or both. Fractures are commonly atraumatic and are the most common of the three aforementioned acute junctional complications with a reported incidence reaching 62% [42]. Fractures result from a failure of the anterior column and occur in two forms: one at the most proximal

instrumented level (UIV) and the other at the vertebra one level proximal to the most proximal instrumented level (UIV+1) [6]. Fractures of UIV are presumed to be a result of acute concentration of mechanical stress on the UIV after correction of considerable sagittal imbalance with rigid pedicle screw constructs, whereas fractures of UIV+1 are theorized to be the result of stress concentration on unfused adjacent segments [6]. In contrast to proximal junctional fractures that occur more commonly at the proximal junction of a fusion extending from the pelvis to the upper lumbar or lower thoracic spine, posterior soft tissue disruption and subsequent spondylolisthesis occur more commonly at the proximal extent of a thoracolumbar fusion extending from the pelvis to the upper thoracic spine. As acute junctional failures often result in an acute destabilization of the spine, neurologic injury is a significant risk [5, 6]. Therefore, prevention of these complications is important. Take the following case example (Fig. 26.2). Another example of adjacent segment degeneration. A 54 year old female degenerative stenosis and spondylolisthesis who underwent an L4–S1 decompression and fusion at an outside hospital. Two-years after her index surgery, she presented with mechanical back pain, radiculopathy and adjacent segment disease and underwent an extension of fusion proximally to L2. However, 2 months post-operative, she developed proximal junctional failure and presented with severe mechanical back pain and a 60° focal kyphotic deformity. She subsequently underwent an extension of fusion proximally to T10, and again developed proximal junctional failure. She was transferred to our center where she underwent decompression, stabilization and deformity correction. Similar to the example provided above, she had a pelvic incidence of 53°. Classified as a Roussouly type 3. Due to progressive deformity and pelvic retroversion, she presented to our center as a Roussouly false type 2 (Pelvic incidence 53°,

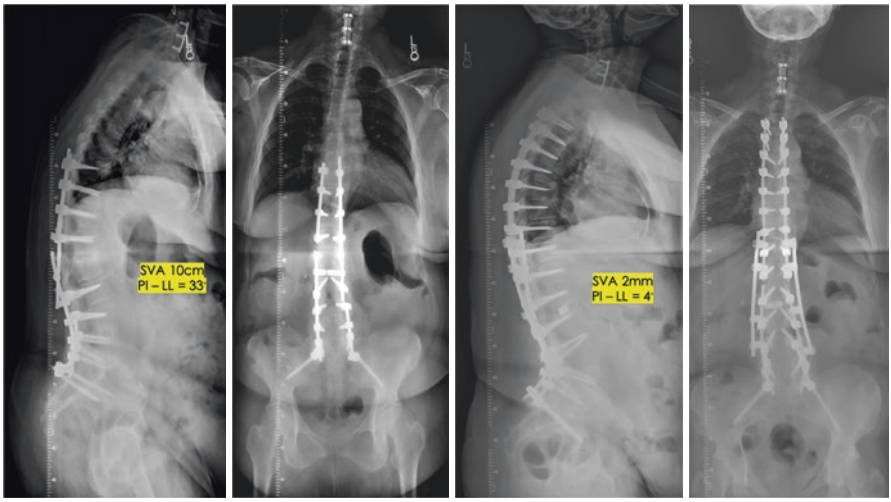


Fig. 26.2 A 54 year old presented after five prior spine surgeries with severe pain (inadequately controlled with opioids), proximal junctional kyphosis, rod fractures, and sagittal plane malalignment. She underwent a 3-column osteotomy at L4 for deformity correction

pelvic tile 22°, lumbar lordosis 30°). A revision T5 to the sacrum with iliac fixation was performed with an L4 pedicle subtraction osteotomy. Postoperatively, regional and global sagittal and coronal alignment parameters were restored. Similar to the aforementioned example, this case exemplifies the sequalae of inattention to sagittal alignment parameters and restoration of geometric shape when performing short segment fusions.

Strategies to prevent acute junctional failures are aimed at augmenting the anterior column's load-bearing capacity or the posterior ligamentous structures. To strengthen the anterior column, injection of cement via a vertebroplasty or kyphoplasty technique has been proposed and demonstrated to be safe and efficacious for preventing acute junctional fractures [37, 50]. In a clinical analysis, Hart and colleagues found that proximal junctional fractures after thoracolumbar fusions from the lumbosacral junction to the lower thoracic spine occurred in no adults treated with cement augmentation of the proximal vertebrae and in 15% of patients not treated with cement augmentation [37]. It appears that cement augmentation of both the UIV and the UIV+1 may provide a stronger construct and thus be better for fracture prevention than no cement or only one-level cement augmentation of the UIV or UIV+1 [50]. In a biomechanical study on cadaveric spines instrumented from T10 to L5, Kebaish et al. observed that only 17% of cadavers with two-level cement augmentation (UIV and UIV+1) had a proximal junctional fracture, whereas 67% of specimens with one-level vertebroplasty and 100% of specimens without cement augmentation had a proximal junctional fracture [50]. Clinically, Martin and coworkers found a 13% rate of proximal junctional pathology after long thoracolumbar fusions with two-level proximal junctional cement augmentation, which is a lower incidence of proximal junctional pathology relative to historic data [51]. In addition to augmenting the anterior column, strategies focused on augmenting the posterior tension band and providing a gradual transition at the junctions are also proposed to decrease junctional failures [52, 53].

Proposed posterior-based strategies to decrease the risk of proximal junctional failures include using transverse process hooks at the most proximal vertebrae in the upper thoracic spine instead of pedicle screws and transitional posterior dynamic stabilization devices [52, 53]. Biomechanically, both strategies have been demonstrated to provide a more gradual transition to normal motion compared with pedicle screws in long posterior spinal fusion constructs [52, 53]. In a porcine spine, the use of transverse process hooks compared to pedicle screws at the UIV of long fusions terminating in the midthoracic spine was found to decrease stiffness between the UIV and UIV+1 and provide a more harmonious range of motion from the distal segments to proximal segments [52, 53]. However, a separate biomechanical study in osteoporotic human cadaveric specimens did not find a difference in the incidence of proximal junctional fractures between constructs that ended proximally with transverse process hooks or pedicle screws [54]. In regard to posterior dynamic stabilization devices, few studies have evaluated their efficacy in the setting of long thoracolumbar fusions. Nonetheless, in cadaveric spines instrumented from T7 to L3, Durrani and colleagues demonstrated that a dynamic stabilization device placed at the most distal instrumented level was able to lessen hypermobile conditions of

the adjacent noninstrumented level caused by rigid fixation in flexion-extension, lateral bending, and axial rotation [52]. Despite promising results from in vitro investigations, the efficacy of these techniques in preventing acute junctional failures in the clinical setting remains unknown. Until further clinical studies are performed, preservation of the posterior soft tissue elements (i.e., facet joint capsules and posterior ligamentous complex) remains the major strategy to minimize the risk of developing acute proximal and distal junctional failures.

Surgical Correction of Postsurgical Deformity

Surgical strategies to address postsurgical spinal deformity are dictated by patient age, medical comorbidities, degree of debilitation, and the unique characteristics of each deformity. Regional and global spinal deformity radiographic measurements, including spinopelvic parameters, and recognition of deviations from these parameters [55] are important to understand when assessing each deformity. All patients with postoperative spinal deformity should have a diagnostic evaluation for metabolic bone disease, which includes a dual-energy x-ray absorptiometry (DEXA) scan and assessment of levels of vitamin D, calcium, and parathyroid hormone. Appropriate pharmacologic treatment of osteoporosis and other documented metabolic deficiencies should be initiated before proceeding with surgery [56]. Additionally, radiographic evaluation should always include full-length, standing lateral, and anteroposterior spinal radiographs. Advanced imaging, including a computed tomography (CT) myelogram, should be obtained preoperatively to evaluate the location and etiology of a radiculopathy or a neurologic deficit. A magnetic resonance image (MRI) may also be obtained to evaluate for compressive pathology in the revision setting, particularly if the location of the purported pathology is distant to the previous instrumentation. The ideal radiographic goals of deformity correction are to bring the pelvic incidence (a fixed value) and lumbar lordosis (a dynamic value) to within 10° of one another, to improve pelvic tilt to less than 20°, and to achieve less than 4 cm of coronal and sagittal imbalance. Also important is the appreciation of the lordotic shape variation according to pelvic incidence. This classification described by Roussouly et al [21] emphasizes not only angular measurements, but also lordosis curve organization and distribution of lumbar lordosis and thoracic kyphosis. To achieve these goals, a thoughtful and integrated evaluation of the following should be assessed: spinal mobility, the deformity's apex, previous levels of fusions, decompressions, or instrumentation, junctional pathology, neurologic status and need for decompression, and planned levels of instrumentation.

Assessing the mobility of curves cranial and caudal to previous instrumentations/fusions assists in planning the extent of fusion/instrumentation and determining the need for osteotomies to mobilize the spine. This may be accomplished by obtaining preoperative flexion-extension radiographs or lateral bending radiographs. Supine full-length x-rays also provide useful information on the flexibility of the deformity. As is the case in all deformity operations, planned

instrumentation/fusions should include the entirety of a structural coronal curve (i.e., terminate at the end vertebra) and should not end at the apex of a sagittal deformity. Evaluating disc segments contained within the entire planned fusion construct is also necessary. For example, if neither disc nor facets are fused then a deep posterior based release with total articular process resection plus interlaminar release is sufficient. If there are mobile disc motion segments within the planned fusion construct with total posterior fusion, then multiple posterior-based osteotomies, including Ponte or Smith-Petersen osteotomies, may be employed to improve alignment [57–59]. The Ponte osteotomy has been reported to allow for 5–15° of angular correction per level [59], whereas the Smith-Petersen osteotomy has been reported to allow for 10° of correction per spinal level [58]. In contrast, solid arthrodesis and severe deformity that does not include mobile motion segments may require more acute angular correction using three-column osteotomies, including a pedicle subtraction osteotomy or a vertebral column resection [57, 59, 60]. The level of the PSO is also very important. A L3 PSO has historically been performed because of ease of accessibility and low rate of neurological complications. However, recent studies have demonstrated a relationship between apex of lumbar lordosis and development of proximal junctional kyphosis: the higher the PSO level, the greater the risk of PJK. PSO's should be performed at L4 or L5 to reconstruct the inferior arch of lumbar lordosis. The pedicle subtraction osteotomy has been reported to provide an average of approximately 30° of correction [61]. Vertebral column resection allows for greater correction of both sagittal and coronal plane deformity as well as restoration of the anterior column's height and load-bearing capacity with a cage or strut osseous graft. The anterior column may also be reconstructed via traditional or minimally invasive anterior approaches. Although spinal deformity correction with the aforementioned techniques are focused at the apex of deformity, an overall harmonious deformity correction that closely matches normal anatomy is the ideal goal. This type of correction theoretically has anatomic advantages related to minimizing proximal junctional disease over the creation of an acute angular correction that may result in a reciprocal kyphosis proximal to the deformity correction. Additionally, a more harmonious deformity correction does not require extravagant bends in one's spinal rods. This is important as the biomaterials used in spinal deformity operations all have unique intrinsic properties that relate to their elastic and plastic deformations. Compared to a rod that is significantly bent, a rod with a less acute bend has a lower chance of being manipulated past the zone of elastic deformation into the zone of plastic deformation where it is permanently weakened. The use of multiple rod constructs in the setting of deformity correction has gained popularity, as multiple rods facilitate less rod manipulation and minimize the risks of plastic deformation. Although modifications in spinal constructs are important to improve outcomes, a comprehensive analysis of the nuances of each deformity, a firm working knowledge of spinal deformity surgical principles, and a careful understanding of patient expectations are the keys to treating postsurgical deformity.

Conclusion

Postoperative deformity is common in patients who have undergone prior spinal surgery. It results in an imbalance of the spine and can lead to progressively worsening disability. The etiology can be diverse, however, proper patient positioning during surgery, minimizing bony removal during decompression and adequate restoration of geometric shape can minimize the risk of postoperative deformity.

References

1. Panjabi MM. The stabilizing system of the spine. Part I. Function, dysfunction, adaptation, and enhancement. J Spinal Disord. 1992;5(4):383–9; discussion 397.
2. Glassman SD, Bridwell K, Dimar JR, Horton W, Berven S, Schwab F. The impact of positive sagittal balance in adult spinal deformity. Spine (Phila Pa 1976). 2005;30(18):2024–9.
3. Tang JA, Scheer JK, Smith JS, et al. The impact of standing regional cervical sagittal alignment on outcomes in posterior cervical fusion surgery. Neurosurgery. 2012;71(3):662–9; discussion 669.
4. Villavicencio AT, Babuska JM, Ashton A, et al. Prospective, randomized, double-blind clinical study evaluating the correlation of clinical outcomes and cervical sagittal alignment. Neurosurgery. 2011;68(5):1309–16; discussion 1316.
5. O'Leary PT, Bridwell KH, Lenke LG, et al. Risk factors and outcomes for catastrophic failures at the top of long pedicle screw constructs: a matched cohort analysis performed at a single center. Spine (Phila Pa 1976). 2009;34(20):2134–9.
6. Watanabe K, Lenke LG, Bridwell KH, Kim YJ, Koester L, Hensley M. Proximal junctional vertebral fracture in adults after spinal deformity surgery using pedicle screw constructs: analysis of morphological features. Spine (Phila Pa 1976). 2010;35(2):138–45.
7. Albert TJ, Vacarro A. Postlaminectomy kyphosis. Spine (Phila Pa 1976). 1998;23(24):2738–45.
8. Nowinski GP, Visarius H, Nolte LP, Herkowitz HN. A biomechanical comparison of cervical laminaplasty and cervical laminectomy with progressive facetectomy. Spine (Phila Pa 1976). 1993;18(14):1995–2004.
9. Haher TR, O'Brien M, Dryer JW, Nucci R, Zipnick R, Leone DJ. The role of the lumbar facet joints in spinal stability. Identification of alternative paths of loading. Spine (Phila Pa 1976). 1994;19(23):2667–70; discussion 2671.
10. Zander T, Rohlmann A, Klockner C, Bergmann G. Influence of graded facetectomy and laminectomy on spinal biomechanics. Eur Spine J. 2003;12(4):427–34.
11. Lee KK, Teo EC. Effects of laminectomy and facetectomy on the stability of the lumbar motion segment. Med Eng Phys. 2004;26(3):183–92.
12. Doherty J. Complications of fusion in lumbar scoliosis: proceedings of the Scoliosis Research Society. J Bone Joint Surg Am. 1973;55:438.
13. Booth KC, Bridwell KH, Lenke LG, Baldus CR, Blanke KM. Complications and predictive factors for the successful treatment of flatback deformity (fixed sagittal imbalance). Spine (Phila Pa 1976). 1999;24(16):1712–20.
14. Blondel B, Jouve JL, Panuel M, et al. Pelvic incidence reliability in spine sagittal balance. Rev Chir Orthop Reparatrice Appar Mot. 2008;94(4):321–6.
15. Trobisch PD, Samdani AF, Betz RR, Bastrom T, Pahys JM, Cahill PJ. Analysis of risk factors for loss of lumbar lordosis in patients who had surgical treatment with segmental instrumentation for adolescent idiopathic scoliosis. Eur Spine J. 2013;22(6):1312–6.
16. Potter BK, Lenke LG, Kuklo TR. Prevention and management of iatrogenic flatback deformity. J Bone Joint Surg Am. 2004;86(8):1793–808.

17. Benfanti PL, Geissele AE. The effect of intraoperative hip position on maintenance of lumbar lordosis: a radiographic study of anesthetized patients and unanesthetized volunteers on the Wilson frame. Spine (Phila Pa 1976). 1997;22(19):2299–303.
18. Guanciale AF, Dinsay JM, Watkins RG. Lumbar lordosis in spinal fusion. A comparison of intraoperative results of patient positioning on two different operative table frame types. Spine (Phila Pa 1976). 1996;21(8):964–9.
19. Shono Y, Kaneda K, Abumi K, McAfee PC, Cunningham BW. Stability of posterior spinal instrumentation and its effects on adjacent motion segments in the lumbosacral spine. Spine (Phila Pa 1976). 1998;23(14):1550–8.
20. Stephens GC, Yoo JU, Wilbur G. Comparison of lumbar sagittal alignment produced by different operative positions. Spine (Phila Pa 1976). 1996;21(15):1802–6; discussion 1807.
21. Roussouly P, Gollogly S, Berthonnaud E, Dimnet J. Classification of the normal variation in the sagittal alignment of the human lumbar spine and pelvis in the standing position. Spine (Phila Pa 1976). 2005;30(3):346–53.
22. Umehara S, Zindrick MR, Patwardhan AG, et al. The biomechanical effect of postoperative hypolordosis in instrumented lumbar fusion on instrumented and adjacent spinal segments. Spine (Phila Pa 1976). 2000;25(13):1617–24.
23. Balderston RA, Winter RB, Moe JH, Bradford DS, Lonstein JE. Fusion to the sacrum for nonparalytic scoliosis in the adult. Spine (Phila Pa 1976). 1986;11(8):824–9.
24. Devlin VJ, Boachie-Adjei O, Bradford DS, Ogilvie JW, Transfeldt EE. Treatment of adult spinal deformity with fusion to the sacrum using CD instrumentation. J Spinal Disord. 1991;4(1):1–14.
25. Emami A, Deviren V, Berven S, Smith JA, Hu SS, Bradford DS. Outcome and complications of long fusions to the sacrum in adult spine deformity: Luque-Galveston, combined iliac and sacral screws, and sacral fixation. Spine (Phila Pa 1976). 2002;27(7):776–86.
26. Kostuik JP, Hall BB. Spinal fusions to the sacrum in adults with scoliosis. Spine (Phila Pa 1976). 1983;8(5):489–500.
27. McCord DH, Cunningham BW, Shono Y, Myers JJ, McAfee PC. Biomechanical analysis of lumbosacral fixation. Spine (Phila Pa 1976). 1992;17(8 Suppl):S235–43.
28. Tsuchiya K, Bridwell KH, Kuklo TR, Lenke LG, Baldus C. Minimum 5-year analysis of L5-S1 fusion using sacropelvic fixation (bilateral S1 and iliac screws) for spinal deformity. Spine (Phila Pa 1976). 2006;31(3):303–8.
29. Chang TL, Sponseller PD, Kebaish KM, Fishman EK. Low profile pelvic fixation: anatomic parameters for sacral alar-iliac fixation versus traditional iliac fixation. Spine (Phila Pa 1976). 2009;34(5):436–40.
30. Nottmeier EW, Pirris SM, Balseiro S, Fenton D. Three-dimensional image-guided placement of S2 alar screws to adjunct or salvage lumbosacral fixation. Spine J. 2010;10(7):595–601.
31. O'Brien JR, Yu WD, Bhatnagar R, Sponseller P, Kebaish KM. An anatomic study of the S2 iliac technique for lumbopelvic screw placement. Spine (Phila Pa 1976). 2009;34(12):E439–42.
32. Mattei TA, Fassett DR. Combined S-1 and S-2 sacral alar-iliac screws as a salvage technique for pelvic fixation after pseudarthrosis and lumbosacropelvic instability: technical note. J Neurosurg Spine. 2013;19(3):321–30.
33. Bastian L, Lange U, Knop C, Tusch G, Blauth M. Evaluation of the mobility of adjacent segments after posterior thoracolumbar fixation: a biomechanical study. Eur Spine J. 2001;10(4):295–300.
34. Park P, Garton HJ, Gala VC, Hoff JT, McGillicuddy JE. Adjacent segment disease after lumbar or lumbosacral fusion: review of the literature. Spine (Phila Pa 1976). 2004;29(17):1938–44.
35. Untch C, Liu Q, Hart R. Segmental motion adjacent to an instrumented lumbar fusion: the effect of extension of fusion to the sacrum. Spine (Phila Pa 1976). 2004;29(21):2376–81.
36. DeWald CJ, Stanley T. Instrumentation-related complications of multilevel fusions for adult spinal deformity patients over age 65: surgical considerations and treatment options in patients with poor bone quality. Spine (Phila Pa 1976). 2006;31(19 Suppl):S144–51.
37. Hart RA, McCarthy I, Ames CP, Shaffrey CI, Hamilton DK, Hostin R. Proximal junctional kyphosis and proximal junctional failure. Neurosurg Clin N Am. 2013;24(2):213–8.

38. Hostin R, McCarthy I, O'Brien M, et al. Incidence, mode, and location of acute proximal junctional failures after surgical treatment of adult spinal deformity. Spine (Phila Pa 1976). 2013;38(12):1008–15.
39. Kim YJ, Bridwell KH, Lenke LG, Rhim S, Cheh G. Sagittal thoracic decompensation following long adult lumbar spinal instrumentation and fusion to L5 or S1: causes, prevalence, and risk factor analysis. Spine (Phila Pa 1976). 2006;31(20):2359–66.
40. Lewis SJ, Abbas H, Chua S, et al. Upper instrumented vertebral fractures in long lumbar fusions: what are the associated risk factors? Spine (Phila Pa 1976). 2012;37(16):1407–14.
41. Yasuhara T, Takahashi Y, Kumamoto S, et al. Proximal vertebral body fracture after 4-level fusion using l1 as the upper instrumented vertebra for lumbar degenerative disease: report of 2 cases with literature review. Acta Med Okayama. 2013;67(3):197–202.
42. Yagi M, Akilah KB, Boachie-Adjei O. Incidence, risk factors and classification of proximal junctional kyphosis: surgical outcomes review of adult idiopathic scoliosis. Spine (Phila Pa 1976). 2011;36(1):E60–8.
43. Zigler J, Delamarter R, Spivak JM, et al. Results of the prospective, randomized, multicenter Food and Drug Administration investigational device exemption study of the ProDisc-L total disc replacement versus circumferential fusion for the treatment of 1-level degenerative disc disease. Spine (Phila Pa 1976). 2007;32(11):1155–62; discussion 1163.
44. Gore DR, Gardner GM, Sepic SB, Murray MP. Roentgenographic findings following anterior cervical fusion. Skelet Radiol. 1986;15(7):556–9.
45. Gore DR, Sepic SB. Anterior cervical fusion for degenerated or protruded discs. A review of one hundred forty-six patients. Spine (Phila Pa 1976). 1984;9(7):667–71.
46. Hilibrand AS, Yoo JU, Carlson GD, Bohlman HH. The success of anterior cervical arthrodesis adjacent to a previous fusion. Spine (Phila Pa 1976). 1997;22(14):1574–9.
47. Cardoso MJ, Dmitriev AE, Helgeson M, Lehman RA, Kuklo TR, Rosner MK. Does superior-segment facet violation or laminectomy destabilize the adjacent level in lumbar transpedicular fixation? An in vitro human cadaveric assessment. Spine (Phila Pa 1976). 2008;33(26):2868–73.
48. Etebar S, Cahill DW. Risk factors for adjacent-segment failure following lumbar fixation with rigid instrumentation for degenerative instability. J Neurosurg. 1999;90(2 Suppl):163–9.
49. Chow DH, Luk KD, Evans JH, Leong JC. Effects of short anterior lumbar interbody fusion on biomechanics of neighboring unfused segments. Spine (Phila Pa 1976). 1996;21(5):549–55.
50. Kebaish KM, Martin CT, O'Brien JR, LaMotta IE, Voros GD, Belkoff SM. Use of vertebroplasty to prevent proximal junctional fractures in adult deformity surgery: a biomechanical cadaveric study. Spine J. 2013;13(12):1897–903.
51. Martin CT, Skolasky RL, Mohamed AS, Kebaish KM. Preliminary results of the effect of prophylactic vertebroplasty on the incidence of proximal junctional complications after posterior spinal fusion to the low thoracic spine. Spine Deform. 2013;1(2):132–8.
52. Durrani A, Jain V, Desai R, et al. Could junctional problems at the end of a long construct be addressed by providing a graduated reduction in stiffness? A biomechanical investigation. Spine (Phila Pa 1976). 2012;37(1):E16–22.
53. Thawrani DP, Glos DL, Coombs MT, Bylski-Austrow DI, Sturm PF. Transverse process hooks at upper instrumented vertebra provide more gradual motion transition than pedicle screws. Spine (Phila Pa 1976). 2014;39(14):E826–32.
54. Mac-Thiong JM, Levasseur A, Parent S, Petit Y. The influence of proximal anchors on the risk of proximal junctional fracture in the osteoporotic spine: biomechanical comparison between pedicle screws and transverse process hooks. J Spinal Disord Tech. 2014;27(2):E49–54.
55. Legaye J, Duval-Beaupere G, Hecquet J, Marty C. Pelvic incidence: a fundamental pelvic parameter for three-dimensional regulation of spinal sagittal curves. Eur Spine J. 1998;7(2):99–103.
56. Mankin HJ, Mankin CJ. Metabolic bone disease: a review and update. Instr Course Lect. 2008;57:575–93.
57. Bridwell KH. Decision making regarding Smith-Petersen vs. pedicle subtraction osteotomy vs. vertebral column resection for spinal deformity. Spine (Phila Pa 1976). 2006;31(19 Suppl):S171–8.

58. Burton DC. Smith-Petersen osteotomy of the spine. Instr Course Lect. 2006;55:577–82.
59. Geck MJ, Macagno A, Ponte A, Shufflebarger HL. The Ponte procedure: posterior only treatment of Scheuermann's kyphosis using segmental posterior shortening and pedicle screw instrumentation. J Spinal Disord Tech. 2007;20(8):586–93.
60. Cho KJ, Bridwell KH, Lenke LG, Berra A, Baldus C. Comparison of Smith-Petersen versus pedicle subtraction osteotomy for the correction of fixed sagittal imbalance. Spine (Phila Pa 1976). 2005;30(18):2030–7; discussion 2038.
61. Bridwell KH, Lewis SJ, Rinella A, Lenke LG, Baldus C, Blanke K. Pedicle subtraction osteotomy for the treatment of fixed sagittal imbalance. Surgical technique. J Bone Joint Surg Am. 2004;86-A Suppl 1:44–50.

Patient Reported Outcomes in Spine Surgery

27

Jose Marin Sanchez, Salah G. Aoun, Kristen Hall, and Carlos A. Bagley

Introduction

In recent years, there has been a gradual movement in healthcare towards increasing the value of the care provided to patients. To this end, the importance of patient reported outcome measurement has gained in prevalence and in importance in the emerging reimbursement model that hinges more on the quality of the care provided, rather than the amount or volume of care [10, 11]. This patient-centered system focuses on the value that an individual receives from a surgical intervention as defined as the quality of care and patient experience per unit of cost [10]. Quality measurement value is an inherently difficult task as this

J. M. Sanchez
University of Texas Southwestern Medical Center, Department of Neurological Surgery, Dallas, TX, USA
e-mail: Jose.Sanchez@utsouthwestern.edu

S. G. Aoun
Department of Neurological Surgery and Spine Center, UT Southwestern Medical Center, Dallas, TX, USA
e-mail: Salah.Aoun@utsouthwestern.edu

K. Hall
University of Texas Southwestern Medical Center, Spine Center, Dallas, TX, USA
e-mail: Kristen.Hall@utsouthwestern.edu

C. A. Bagley (✉)
University of Texas Southwestern Medical Center, Department of Neurological Surgery, Dallas, TX, USA

University of Texas Southwestern Medical Center, Spine Center, Dallas, TX, USA

University of Texas Southwestern Medical Center, Department of Orthopedic Surgery, Dallas, TX, USA
e-mail: Carlos.Bagley@utsouthwestern.edu

© The Author(s), under exclusive license to Springer Nature Switzerland AG 2022

C. E. Noe (ed.), *Multidisciplinary Spine Care*,
https://doi.org/10.1007/978-3-031-04990-3_27

definition may vary based on the perspective taken (i.e. patient, hospital, physician, etc.). PRO have the objective of focusing on what patients find valuable when undergoing an intervention rather than focusing on what clinicians determine to be favorable outcomes. PRO also aim to quantify health status from the patient's viewpoint without interpretation of responses by the clinician [10]. These tools can be extremely valuable when measuring patient's progress as they focus on the aspects that motivated a patient to pursue an intervention. PRO may also be analyzed in conjunction with clinical parameters in order to provide a more wholistic view of the quality of the services rendered [10]. In this manner, the clinician can evaluate the quality of care that is being offered to the patient. These tools are usually divided into categories such as general quality of life, functioning, pain, and disease specific outcomes and the goal is to objectify measures that do not align with traditional outcomes measures [1]. This chapter focuses on PRO used in the field of spine surgery.

History

The use of PRO is a practice that is becoming more popular in modern medical literature. The steady increase in the use of PRO has been reported to be as dramatic as a fivefold increase per decade [2]. Some of the first forms of PRO can be traced back to the early 1900s. Some of the more popular PRO tools that can be found in modern literature of spine surgery include the Oswestry disability index (ODI), Visual Analog Scale, European Quality of Life five-dimension (EuroQoL-5D), "Short Form Survey (36, 12, 22), Scoliosis Research Society (SRS), Numeric Rating Scale (NRS) among others [1]. Additionally, we have also seen a dramatic increase in the creation and use of disease specific tools such as the neck disability index (NDI) and Myelopathy disability index (MDI) as well as the use of interdisciplinary care models such as Enhanced Recovery After Surgery Pathways (ERAS) that attempt to exploit the value of PRO by using a comprehensive multisystem approach [1].

General Health Measures

Some of the general health assessment tools that have been regarded as highly valuable in the assessment of general health measures in patients undergoing spine surgery include the EuroQoL-5D and the Short Form Questionnaires, particularly the SF-36 as it provides a more comprehensive evaluation compared to the SF-12 and SF-6 [9]. Additionally, there are tools such as the McGill Pain Questionnaire (MPQ), NRS and the visual analogue scale (VAS) that, although not comprehensive or disease specific, they report relevant information that combined with other tools help evaluate the patient undergoing spine surgery in a general and comprehensive manner [9].

European Quality of Life Five-Dimension (EuroQoL-5D)

The EuroQoL-5D is a popular and well accepted tool published in the 1990s and was designed to provide a simple and generic measure of general health [5]. This tool is dived into five "dimensions" that address mobility, self-care, usual activities, pain (or discomfort), anxiety and/or depression [5]. This survey has been translated into more than 150 languages and is commonly used worldwide in numerous specialties [5].

Short Form Questionnaire

The medical Outcomes Study Short Form Questionnaire, particularly the SF-36, is the most used general health outcome measure [10]. It is a consolidation of the original RAND Corporation's 1989 Medical Outcomes Study which was a 116-item questionnaire with 8 different domains [10]. Results of the SF-36 are generally reported in the 8 separate domain scores and/or two summary scores ranging from 0 to 100 [10]. These forms have been validated to use across a broad spectrum of diseases and is powerful enough to assess and compare the overall health status of patients suffering completely different pathologies. One of the major disadvantages is that it takes considerably more time and effort to complete compared to the EuroQoL-5D [10].

Numeric Rating Scale

The NRS for pain is a numeric, one-dimensional scale that is useful for rating pain across multiple conditions and diseases [4]. Most commonly, it is used as a scale from 0 to 10 where 0 denotes no pain and 10 denotes the worst possible imaginable pain [4]. The patient then picks a discrete number in the scale to rate his or her pain. This scale has been validated across multiple conditions and can be a tool used to compare pain across multiple conditions that produce somatic symptoms as well as conditions that result primarily in emotional distress [4].

Visual Analog Scale

Like the NRS, VAS is another one-dimensional scale for rating pain or emotional distress [4]. The VAS was traditionally presented as a 10 cm scale on which the patient can mark any point on the scale [4]. Then, the results are interpreted based on preestablished cutoffs. For example, 0–4 mm can be interpreted as no pain, 5–44 mm as mild pain and so on [10]. This tool has also been validated across multiple conditions and can also be used to compare pain or distress between two completely different processes but the highly subjective nature of these scales causes significant variability and limits comparison across a range of patients or even

between multiple assessments in the same patient. For example, lower scores have been reported for horizontally oriented VAS compared to vertical ones [8].

McGill Pain Questionnaire

The MPQ was introduced in 1975 but was later replaced with the shorter version SF-MPQ and most recently by the SF-MPQ-2 [10]. The SF-MPQ-2 is composed of a list of pain descriptors and incorporates the principles of the NRS and the VAS. In this tool, the patient selects a value from 0 to 10 presented as a visual scale for each pain descriptor [13]. These values can be summed to report a total pain score from 0 to 220 or can be used individually for each component [13]. This in turn allows the clinician to provide targeted treatments for the areas that produce the most distress while keeping in mind the multifactorial nature of pain.

Disease-Specific Measures

Multiple disease-specific PRO tools exist. Here we will focus on measures designed to assess functional impairment in patients with spine related pathology.

Cervical Spine

The use of PRO tools have gained popularity as a way to quantify patient outcomes, however, the number of tools available to assess disability of the cervical spine remains limited and the correlation between certain clinical parameters such as sagittal balance with disability in the cervical spine remains modest [19]. Furthermore, neck pain is often multifactorial. Specifically, neck pain may be caused by primary cervical deformity which could be the result of degenerative causes, congenital causes, trauma or iatrogenic causes [14]. Cervical deformity can lead to a wide variety of symptoms including gait disturbance, pain, cervical myelopathy, dysphagia, and many others in the more extreme presentations such as the chin-on-chest deformity [14]. In the setting of trauma, injuries can result in a myriad of symptoms and conditions including pain, spinal cord injuries and deformities that whether these are corrected or not, can impair a patient's ability to perform everyday activities. For all these reasons, capturing disability from cervical deformity and trauma has been difficult [22]. Although some tools designed to evaluate PRO in patients with cervical myelopathy investigate some of the symptoms and complications that are shared between degenerative spine disease, deformity and trauma, there is no PRO tool that successfully measures disability in patients with cervical spine deformity or trauma in a targeted manner [20]. There are, however, tools that assess the disability caused by cervical spine pathology as a broad category. A clear and well validated example is the NDI.

The NDI was published in 1991 and it is one of the most validated disease-specific tools in spine surgery [21]. This PRO is composed of ten questions that assess how neck pain has affected an individual's ability to manage in everyday life [21]. This tool includes questions related to pain intensity, personal care, lifting,

reading, headaches, concentration, work, driving, sleeping and recreation [21]. Each question is graded in a scale of 0–5 for a total of 50 points. The score is then divided by the total possible score depending on the number of questions answered by the individual and reported as a percentage [21]. The NDI has been validated and found to meet the responsiveness required to detect when a true change has occurred [21]. Furthermore, the NDI showed a fair test-retest reliability in patients with disability secondary to neck pain or other cervical spine pathology [21].

The Cervical Spine Outcomes Questionnaire (CSOQ) was originally described in 2002 and consists of 35 questions divided into 6 different categories that measure physical symptoms, psychological distress, neck pain, arm pain, physical disability, and healthcare utilization [17]. This tool has been validated and has been found to have good correlation with other standard outcome tools.

Cervical myelopathy is a subcategory of cervical spine pathology for which PRO tools have been developed. Examples include the Modified Japanese Orthopaedic Association Scale (JOA) and the Myelopathy Disability Index (MDI) [8]. Similar to the ODI and the NDI, the MDI is a 10-question tool that is scored on a scale from 0 to 3 with a total possible score of 30 [8]. This tool was initially developed with the aim of objectively measure disability in patients with rheumatoid arthritis complicated by cervical myelopathy [3]. This tool has been found to be responsive and sensitive to detect changes on patients undergoing surgical intervention for cervical myelopathy [3].

The modified JOA, is another tool that was developed with the goal of assessing patients with cervical spondylotic myelopathy and resultant disability [8]. Although validation of this tool is still in its early stages, the mJOA has been widely used across the world, particularly in Japan [8]. This tool consists of six categories: motor function of the upper extremity, motor function of the lower extremity, sensory function of the upper extremity, sensory function of the trunk, sensory function of the lower extremity, and bladder function [8]. This tool grants a higher point score to patients with motor dysfunction, suggesting that patients with motor dysfunction suffer from greater disability.

Capturing disability from cervical pathology can be a difficult task. Cervical pathology can lead to a diverse range of symptoms such as myelopathy, radiculopathy, dysphagia, neck pain, gait disturbance, gaze impairment [2]. Current PRO tools are able to look at disability from neck pathology as a broad category which perhaps could be sufficient to detect meaningful changes in a simple and efficient manner in patients undergoing surgery of the cervical spine [9]. However, the number of tools that look at specific pathology remain scarce. While it is important to keep PRO tools simple and user friendly so they can be effectively used in the timeframe of an office visit by a wide variety of patients including the elderly, the development of PRO tools for specific pathologies could prove useful for clinicians and researchers that wish to understand the value of a surgical intervention.

Thoracolumbar Spine

Several PRO tools have been developed to assess disability due to thoracolumbar spine pathology. Although many of the questions overlap with other general health

and spinal disease measures, these tools tend to focus on symptoms that are more prevalent in thoracolumbar spine disease such as low back pain (LBP), lumbar function and walking. Notable tools include the Oswestry Disability Index (ODI), Roland-Morris Disability Questionnaire (RMDQ), Quebec Back Pain Disability Scale (QBPDS), and the Japanese Orthopedic Association Back Pain Evaluation Questionnaire (JOABPEQ). Like the tools available for cervical spine, thoracolumbar disability can be the result of multiple processes such as deformity, degenerative spine disease, and trauma among others so many of these tools capture disability from thoracolumbar spine disease as a generalized entity and do not distinguish between different causes of disability [1].

The ODI was first published in 1980 and it was widely disseminated at the International Society of the Study of the Lumbar Spine in 1981 [6]. The ODI is a questionnaire that contains 10 categories each one graded from 0 to 5. Like the NDI, the score is then added up and divided by the total possible score and it's converted into a percentage. The score is typically classified into different degrees of disability but in simple terms, the higher the percentage, the greater the disability [18]. The advantages of the ODI include that it can be completed just within a couple of minutes, it has higher sensitivity for higher levels of disability, and it was been validated by numerous authors [7].

The RMDQ was developed in 1993 and contrary to the ODI, is more sensitive in patients with milder symptoms of back pain [15]. This tool was designed for use in research for clinical trials, but it has also been found to be useful in clinical settings. The questionnaire consists of 24 "because of my pain" statements that give a short description of the disability suffered by patients with back pain. In this questionnaire, the responses are binary and consist of "yes" or "no" [15]. Patients receive 1 point for every answered with a "yes" for a total possible score of 24. Higher scores indicate higher disability. One of the disadvantages of the RMDQ) is that it does not assess psychological and psychosocial disability therefore, it correlates less with measures of psychosocial disability [16]. Nonetheless, the RMDQ has been extensively validated and reviewed and it has been found to have a high degree of internal consistency as well as good correlation with other measures of disability [16].

Similar to the limitations of PRO tools for cervical spine, tools designed for the thoracolumbar spine do not discriminate between different causes of thoracolumbar disability. These tools look at thoracolumbar disease as a single entity. Although most are simple enough to be administered in a regular office visit, many of these tools often fail to capture the disability of the patient in a comprehensive manner. This in turn, limits the usefulness of these tools for clinical and research purposes.

Discussion

The investigation of surgical outcomes is an important practice that allows us to make improvements in clinical care delivery. Traditionally, the focus of these outcomes has been what practicing clinicians determine to be important. In recent years, however, there has been an increasing trend in the PRO tools cited in spine

literature [1]. This is particularly important as the number of patient's seeking spine care continues to increase and places tremendous economic strain on the entire healthcare system [12]. While a large portion of the spine literature has focused on operative metrics, the data does not always consistently align with better functional outcomes and patient satisfaction with care. Examples of success that this type of patient centric, PRO measurement focused approach includes the reduction of opioid use and readmission rates in patients undergoing thoracolumbar fusions for degenerative deformity through a multidisciplinary ERAS at a large urban tertiary health system [1]. One of the interesting characteristics of this program is that patients received preoperative care that included behavioral health and psychology referrals with the goal to understand which symptoms motivate individual patients to pursue a surgical intervention and to manage patient's expectations. This is just one example of the potential to improve health care quality and outcomes by shifting to a patient centered approach. PRO measurement will become an integral component of outcomes evaluations and future research. In an ideal scenario, a clinician should be able to assess each condition with a disease specific tool. This tool should be comprehensive and include patient-centered questions that assess characteristics that clinicians have traditionally assessed subjectively in addition to the more traditional characteristics assessed in the past. The primary criticism of currently available measurement tools is that many of these tools fail to capture the true disability of patients and can mislead providers regarding patient outcomes. Clinicians and researchers should also be careful to maintain a low administrative burden as long surveys have been shown to have a lower completion rate. Additionally, it should be noted that some of the PRO instruments currently used have not been well validated which highlights the need for disease-specific instruments for common subsets of spine disease. Regardless, the use of PRO instruments should not be discouraged by the lack of disease-specific tools as it is known that general health measures correlate with disease-specific measures [20]. Ultimately, more research is needed to investigate whether this approach will produce useful data regarding the value of a surgical intervention for spine disease.

Conflict of Interest Statement The authors have no conflict of interest to declare.

References

1. Algattas H, et al. Trends in the use of patient-reported outcome instruments in neurosurgical adult thoracolumbar deformity and degenerative disease literature. J Craniovertebr Junction Spine. 2017;8(2):103. https://doi.org/10.4103/jcvjs.jcvjs_29_17.
2. Azimi P, Shahzadi S, Benzel EC, Montazari A. Updated measuring motor, sensory and sphincter dysfunctions in patients with cervical myelopathy using the modified Japanese Orthopedic Association (mJOA) score: a validation study. J Inj Violence Res. 2012;4(3 Suppl 1):41.
3. Casey AT, et al. Development of a functional scoring system for rheumatoid arthritis patients with cervical myelopathy. Ann Rheum Dis. 1996;55(12):901–6. https://doi.org/10.1136/ard.55.12.901.
4. Downie WW, Leatham PA, Rhind VM, Wright V, Branco JA, Anderson JA. Studies with pain rating scales. Ann Rheum Dis. 1978;37:378–81; 62.

5. EuroQol Group. EuroQol – a new facility for the measurement of health-related quality of life. Health Policy. 1990;16(3):199–208. https://doi.org/10.1016/0168-8510(90)90421-9.
6. Fairbank JC, et al. Oswestry low back pain disability questionnaire. Physiotherapy. 1980;66:271–3. https://doi.org/10.1037/t04205-000.
7. Fairbank JC, Pynsent PB. The Oswestry Disability Index. Spine. 2000;25(22):2940–53. https://doi.org/10.1097/00007632-200011150-00017.
8. Furlan JC, Catharine Craven B. Psychometric analysis and critical appraisal of the original, revised, and modified versions of the Japanese Orthopaedic Association score in the assessment of patients with cervical spondylotic myelopathy. Neurosurg Focus. 2016;40(6). https://doi.org/10.3171/2016.3.focus1648.
9. Kopjar B, et al. Psychometric properties of the modified Japanese Orthopaedic Association Scale in patients with cervical spondylotic myelopathy. Spine. 2015;40(1):E23–8. https://doi.org/10.1097/brs.0000000000000648.
10. Huskisson EC. Measurement of pain. Lancet. 1974;2(7889):1127–31.
11. Malhotra NR, et al. Tracking patient-reported outcomes in spinal disorders. Surg Neurol Int. 2015;6(20):490. https://doi.org/10.4103/2152-7806.166892.
12. McCormick JD, et al. Patient-reported outcome measures in spine surgery. J Am Acad Orthop Surg. 2013;21(2):99–107. https://doi.org/10.5435/jaaos-21-02-99.
13. Ostelo RW, Deyo RA, Stratford P, Waddell G, Croft P, Von Korff M, editors. Interpreting change scores for pain and functional status in low back pain: towards international consensus regarding minimal important change. Spine (Phila Pa 1976). 2008;33:90–4.
14. Protopsaltis T, et al. T1 slope minus cervical lordosis (TS-CL), the cervical answer to PI-LL, defines cervical sagittal deformity in patients undergoing thoracolumbar osteotomy. Int J Spine Surg. 2018;12(3):362–70. https://doi.org/10.14444/5042.
15. Roland M, Morris R. A study of the natural history of back pain. Part I: development of a reliable and sensitive measure of disability in low-back pain. Spine (Phila Pa 1976). 1983;8:141–4.
16. Roland M, Morris R. A study of the natural history of low-back pain. Part II: development of guidelines for trials of treatment in primary care. Spine (Phila Pa 1976). 1983;8:145–50.
17. Skolasky RL, et al. Psychometric properties of the cervical spine outcomes questionnaire and its relationship to standard assessment tools used in spine research. Spine J. 2007;7(2):174–9. https://doi.org/10.1016/j.spinee.2006.07.005.
18. Strong J, et al. Function and the patient with chronic low back pain. Clin J Pain. 1994;10(3):191–6. https://doi.org/10.1097/00002508-199409000-00004.
19. Tang JA, et al. The impact of standing regional cervical sagittal alignment on outcomes in posterior cervical fusion surgery. Neurosurgery. 2012;76(Suppl_1):S14–21. https://doi.org/10.1227/01.neu.0000462074.66077.2b.
20. Vaishnav AS, et al. Correlation between NDI, PROMIS and SF-12 in cervical spine surgery. Spine J. 2020;20(3):409–16. https://doi.org/10.1016/j.spinee.2019.10.017.
21. Vernon H. The neck disability index: state-of-the-art, 1991–2008. J Manip Physiol Ther. 2008;31(7):491–502. https://doi.org/10.1016/j.jmpt.2008.08.006.
22. Villavicencio AT, et al. Prospective, randomized, double-blind clinical study evaluating the correlation of clinical outcomes and cervical sagittal alignment. Neurosurgery. 2011;68(5):1309–16. https://doi.org/10.1227/neu.0b013e31820b51f3.

Scoliosis in Children

28

Karl Rathjen

Definition

Scoliosis is a three-dimensional deformity of the spine. The term *scoliosis*, first used by Galen (131–201 AD), is derived from the Greek word meaning "crooked" [535]. Today, scoliosis is defined as lateral deviation of the normal vertical line of the spine, which when measured on a coronal or anterior-posterior radiograph, is greater than 10° (Fig. 28.1).

Because the lateral curvature of the spine is associated with rotation of the vertebrae within the curve, a three-dimensional deformity occurs with the most significant abnormality located in the apical region. As the deformity worsens, structural changes develop in the vertebrae and rib cage. Although relationships between intrathoracic and abdominal organs may be distorted if the deformity becomes severe, pulmonary function is the only organ system adversely effected by scoliosis.

K. Rathjen (✉)
Department of Orthopaedics, UT Southwestern Medical School and Scottish Rite for Children, Dallas, TX, USA
e-mail: Karl.Rathjen@tsrh.org

© The Author(s), under exclusive license to Springer Nature Switzerland AG 2022

C. E. Noe (ed.), *Multidisciplinary Spine Care*,
https://doi.org/10.1007/978-3-031-04990-3_28

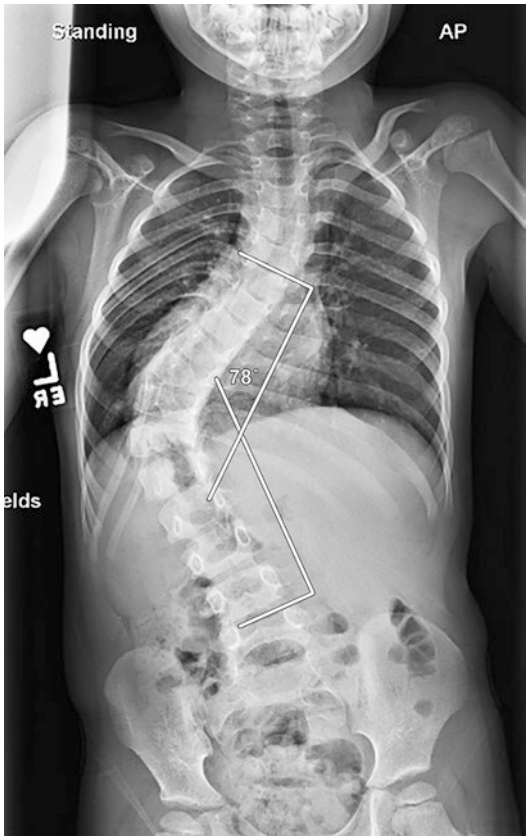


Fig. 28.1 Cobb angle is measured on an AP radiograph by measuring the angle between the top of the cephalad most angled vertebrae and the bottom of the caudal most angled vertebrae

Classification of Scoliotic Curves

A variety of terms are used to describe the different types of scoliotic curves. Table 28.1 provides definitions for the most common ones.

Idiopathic Scoliosis

Idiopathic scoliosis, for which a definitive cause of the deformity has not been established, is the most common type of scoliosis. The diagnosis of idiopathic scoliosis can be made only after a thorough physical and radiographic examination has ruled out neurologic causes, syndromes, and congenital anomalies. Idiopathic scoliosis may have its onset at any age during growth, but three fairly well defined peak periods are accepted: (1) in the first year of life, (2) at 5–6 years of age, and (3) after 11 years of age to the end of skeletal growth. This third peak period is the time when most idiopathic scoliosis patients are diagnosed.

Table 28.1 Types of scoliosis and scoliotic curves

Adult scoliosis: Spinal curvature present after skeletal maturity as a result of any cause.
Cervicothoracic curve: Any spinal curvature in which the apex is at C7 to T1.
Compensatory curve: Secondary curve located above or below the structural component that develops to maintain normal body alignment.
Congenital scoliosis: Scoliosis caused by bone abnormalities of the spine that are present at birth. The anomalies are classified as failure of vertebral formation or failure of segmentation.
Double curve: Scoliosis in which two lateral curves are present in the same section of the spine.
Double major curve: Scoliosis in which two structural curves, usually of similar size and rotation, are present.
Double thoracic curve: Scoliosis with a structural upper thoracic curve; a larger, more deforming lower thoracic curve; and a relatively nonstructural lumbar curve.
Hysterical scoliosis: Nonstructural deformity of the spine that is a manifestation of the psychological disorder.
Idiopathic scoliosis: Structural spinal curvature, the cause of which has not been definitely established.
Kyphoscoliosis: Seen as an increased round back on a lateral radiograph, this condition my represent a true kyphotic deformity (as occurs in some pathologic condition), or it may represent such excessive rotation of the spine that a lateral radiograph is actually reflecting the scoliotic deformity. (In idiopathic scoliosis, true kyphotic deformity does not occur.)
Lordoscoliosis: Structural scoliosis associated with increased swayback or loss of normal kyphosis within the measured curve; it is nearly always present in idiopathic scoliosis.
Lumbar curve: Spinal curvature in which the apex is between L1 and L4.
Lumbosacral curve: Spinal curvature in which the apex is at L5 or below.
Neuromuscular scoliosis: Scoliosis caused by a neurologic disorder of the central nervous system or muscle.
Nonstructural (functional) curve: Curvature that does not have a fixed deformity and may be compensatory in nature. The curve may be a result of leg length discrepancy (in which case it disappears when the patient is supine), poor posture, muscle spasm, or some other cause.
Primary curve: The first or earliest curve present.
Structural curve: Segment of the spine that has a fixed lateral curvature.
Thoracic curve: Spinal curvature in which the apex is between T2 and T11.
Thoracolumbar curve: Spinal curvature in which the apex is at T12, L1, or the T12-L1 interspace.

From Tachdijain's Pediatric Orthopaedics 6th Edition

The term *adolescent idiopathic scoliosis* (AIS) is used when the deformity is recognized after the child has reached 10 years of age but before skeletal maturity, although it is typically noted before the onset of puberty. *Infantile idiopathic* (younger than 3 years) and *juvenile idiopathic scoliosis* (3–9 years old) are now included within "early-onset" scoliosis, a group that includes any type of scoliosis diagnosed before the age of 10 years.

Adolescent Idiopathic Scoliosis

Prevalence

In children over the age of ten, the prevalence of radiographic curves measuring at least 10° ranges from 1.5% to 3.0%, that of curves exceeding 20° is between 0.3% and 0.5%, and that of curves exceeding 30° is between 0.2% and 0.3%.

A definite relationship between idiopathic scoliosis and gender has been noted, particularly as the magnitude of the curve increases. The ratio of affected females to males has been reported to be 1:1 for curves between 6° and 10°, 1.4:1 for curves between 11° and 20°, 5.4:1 for curves exceeding 21° but not requiring treatment, and 7.2:1 for curves requiring treatment [658]. The gender prevalence in adolescent idiopathic scoliosis—that is, an equal prevalence between the sexes for small curves (<10°), with increasing female prevalence for larger and progressive curves has been well documented [35, 162, 456, 658].

Natural History After Skeletal Maturity

In general, the rate of progression of scoliosis in adults is much slower than that in adolescence and depends on the size of the curve once skeletal maturity has been reached. Regardless of the curve pattern, curves of less than 30° in a mature individual are unlikely to progress. Larger curves, however, may progress. Recent reviews examining long-term outcomes in patients with adolescent idiopathic scoliosis who received no treatment showed that single thoracic curves of 50–75° progress 0.73°/year over a 40-year period [36, 161]. This progression does not result in increased mortality, but pulmonary symptoms may be associated with larger curves. Although no long-term studies using modern quality of life questionnaires exist, AIS does not alter social function, childbearing, and marriage in adults. Most individuals with AIS and moderate curve size around maturity can be expected to function well and lead a normal life in terms of work and family. Pain is common in adults with scoliosis, although it is not related to the size or location of the curvature [150, 555, 844]. The pain does not usually interfere with the patient's ability to work or perform daily activities. Despite outwardly apparent deformities because of long-standing untreated scoliosis, most adults have no significant psychological difficulties when compared with persons without scoliosis [845].

In summary, thoracic scoliosis of greater than 50–60° in adulthood may progressively worsen and potentially reduce pulmonary function. Lumbar curves, especially those greater than 45–50°, are also likely to progress in adulthood and may lead to osteoarthritis.

Natural History Before Skeletal Maturity

Children with curves of less than 20° are at relatively low risk for progression, particularly as they approach skeletal maturity [457]. For larger curves the factors which are important in determining risk of progression include gender, growth remaining, curve magnitude, and curve pattern [98, 457, 556].

Gender

As previously mentioned, the majority of patients whose curves progress and ultimately require treatment are female [35, 162, 456, 658]. Although the exact reason for this phenomenon remains unknown, hormonal influences have been proposed [11, 283, 726].

Remaining Growth

Patients with adolescent idiopathic scoliosis who have curves that progress, have the most progression during the adolescent growth spurt. Thus, understanding a patient's potential for remaining growth is vital in determining the risk for potential progression. Growth potential can be assessed using a variety of maturity indices. The most commonly used include: the Risser sign (a skeletal marker of the pelvis), the Sanders stage (based on hand and wrist skeletal maturity [683, 722, 820]), the proximal humeral ossification system [175, 437, 438], the elbow's olecranon skeletal maturity [128], peak height velocity (PHV), and in females, menarchal status.

The Risser sign is a radiographic measurement based on ossification of the iliac apophysis, which is divided into four quadrants [651], beginning on the lateral aspect of the iliac apophysis and progressing medially. The Risser sign proceeds from grade 0, no ossification, to grade 4, in which all four quadrants of the apophysis show ossification ("capping"). When the ossified apophysis has fused completely to the ilium (Risser grade 5), the patient is fully skeletally mature.

Sanders developed a simplified skeletal maturity scoring system for AIS that uses radiographs of the hand that is increasingly utilized to determine the remaining skeletal growth and subsequent risk for progression (Fig. 28.2) [683, 825].

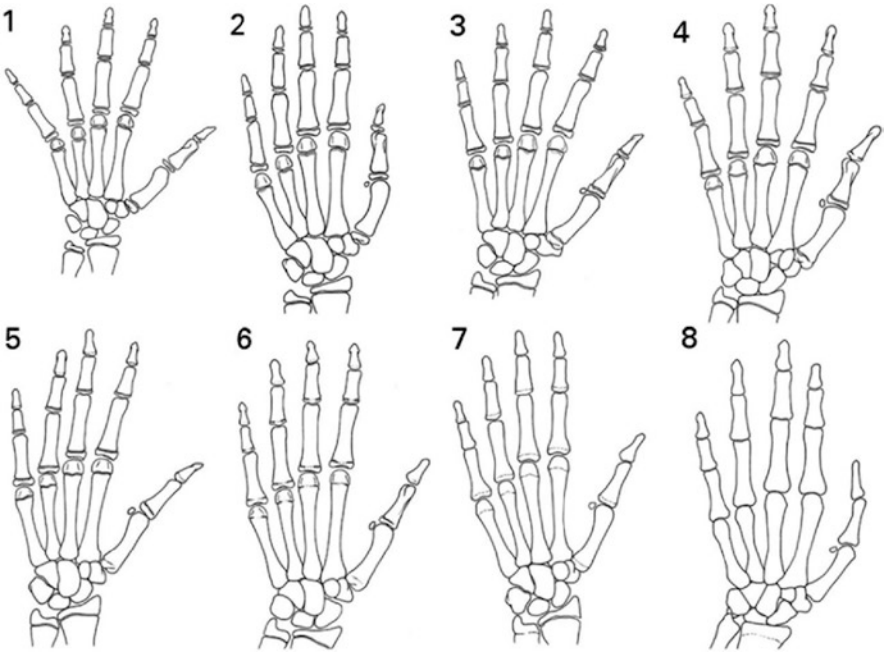


Fig. 28.2 Sanders' simplified skeletal maturity scoring system for AIS. (From Beauchamp et al. [902])

This system which is based on the radiographic appearance of the epiphyses of the phalanges and the distal radius correlates more strongly with the behavior of idiopathic scoliosis than does the Risser sign.

The Proximal Humeral Ossification System (PHOS) is another recently developed radiographic assessment that has been shown to correlate with AIS progression more significantly than the Risser stage. Unlike the Sanders stage, which requires a separate radiograph of the hand, the PHOS can be scored on an AP or PA x-ray of the spine; as long as the upper extremities are appropriately positioned [175, 437, 438].

PHV is a measurement of the maximal skeletal growth that occurs during the adolescent growth spurt [447, 684, 736]. Calculated from changes in a patient's height measurements over time, PHV is fairly consistent in the published literature and is reported to be about 8.0 cm/yr for girls and 9.5 cm/yr for boys [94, 218, 783]. For PHV to be clinically useful, serial height measurements must be obtained. Six-month intervals are preferred because shorter intervals may result in significant measurement error. The information can often be obtained from the family, school, or pediatrician. Although PHV requires analysis of serial height measurements collected over time, it is the earliest and best index available to demonstrate that growth is slowing and the risk for curve progression is diminishing.

Diméglio correlated PHV with the elbow radiograph's olecranon stages of skeletal maturation and noted that curves greater than 30° prior to the onset of pubertal growth have a 100% risk of progressing over 45° ($P < 0.0001$) and curves 21–30° have a progression risk of 72.5% ($P = 0.0034$). Thus, plotting curve magnitude against height measurements and the stages of olecranon maturation offers a reliable prediction of curve progression risk in Risser 0 patients with AIS [128].

Menarchal status is a clinical measurement applicable only to females. A premenarchal girl is still in the active growth period. After menarche, she enters the deceleration phase of growth, and the likelihood of curve progression lessens.

Curve Magnitude

The size of the existing curve when scoliosis is recognized is helpful in predicting curve progression. The combination of this factor and assessment of remaining growth is used to predict the natural history in young patients with scoliosis. Immature patients (premenarchal, Risser grade 0) with curves greater than 20° are at substantial risk for progression of spinal deformity (Table 28.2) [98, 265, 453, 454, 556, 658].

For immature patients with curves exceeding 25°, the risk for curve progression is believed to be significant enough to recommend orthotic management at the time

Table 28.2 Incidence of curve progression based on curve magnitude and Risser grade

	Percentage of curves that progress	
Risser grade	Curves 5–19°	Curves 20–29°
0 or 1	22	68
2, 3, or 4	1.6	23

From Tachdijain's Pediatric Orthopaedics 6th Edition

Table 28.3 Curve magnitude and skeletal maturity and treatment recommendations

Curve magnitude (degrees)	Risser sign		
	Grade 0/premenarchal	Grade 1 or 2	Grade 3, 4, or 5
<25	Observation	Observation	Observation
30–40	Brace therapy (begin when the curve is >25°)	Brace therapy	Observation
>45	Surgery	Surgery	Surgery (when the curve is >50°)

From Tachdijain's Pediatric Orthopaedics 6th Edition

of initial evaluation [366, 459, 556, 624, 746, 803]. Currently, consideration is given to initiate orthotic management in immature patients with curves as low as 20° because of the finding that curves exceeding 30° at the onset of the pubertal growth spurt have a high risk of progression to a surgical magnitude (≥45°) [128, 447].

Table 28.3 summarizes treatment recommendations based on curve magnitude and skeletal maturity.

Scoliosis Screening

Scoliosis screening is a controversial topic, particularly when discussing school screening programs. Because of the growing body of evidence that bracing is effective in limiting progression – particularly when started in younger patients with smaller curves – several professional organizations continue to advocate for scoliosis screening; including the American Academy of Pediatrics (AAP) the American Academy of Orthopaedic Surgeons (AAOS), the Scoliosis Research Society (SRS), the Pediatric Orthopaedic Society of North America (POSNA). In a 2016 statement, these organizations supported incorporation of screening of children for AIS by knowledgeable health care providers as part of preventive services visit for females at age 10 and 12 years (grades 5 and 7), and for males, once, at age 13 or 14 years (grades 8 or 9) [317].

Several clinical signs are indicative of possible scoliosis and are frequently used in screening programs, including shoulder asymmetry, unequal scapular prominence, appearance of an elevated or prominent hip, and a positive Adams forward-bending test. The Adams test is performed by having the child bend forward until the spine is horizontal and, while examining the patient from the rear, noting whether one side of the back appears higher than the other (Fig. 28.3).

This test is the most common noninvasive clinical method for evaluating scoliosis [10, 97, 152, 545]. In an effort to quantitatively assess the asymmetry and thus establish an appropriate degree of deformity that justifies referral for medical evaluation, Bunnell introduced the scoliometer in 1984 (Fig. 28.4) [97].

Current recommendations are for radiographic assessment of children who have a 7° or greater angle of trunk rotation [99]. With this criterion the chance of missing a curve greater than 30° (the curve magnitude at which bracing is usually initiated) is low. When this approach is used, the referral rate is approximately 3% of persons screened, with a 95% detection rate of curves requiring brace treatment. An exception to this 7° rotation recommendation is found in obese patients. In this instance,



Fig. 28.3 Adams test. Clinical photographs of a patient with surgical magnitude adolescent idiopathic scoliosis. (**a**) Note trunk shift to right and scapular and shoulder height asymmetry. (**b**) Adams Forward bending test. Note the thoracic rotation

their body mass alters scoliometer measurements leading to an underestimation of curve size. Obese patients should be referred at a scoliometer angle of 5° [487].

Etiology

Adolescent idiopathic scoliosis is most likely a multifactorial condition. Research on the etiology of scoliosis has focused on neurologic factors, connective tissue differences and genetics. Historically, biochemical and nutritional deficiency [292, 340, 874], structural defects [108], and endocrine abnormalities have also been investigated [726].



Fig. 28.4 Bunnell scoliometer. Scoliometer demonstrating (shown analog and with a mobile app) quantification of thoracic rotation

Neurologic Factors

Responses to vibratory stimuli are reportedly reduced significantly and asymmetric between the left and right sides in scoliotic patients when compared with controls [50, 589, 875]. In addition to abnormalities in the sensory pathways, motor dysfunction has been reported, thus suggesting that the organization of the entire brain is asymmetric in individuals with scoliosis [264]. Regional differences in brain volume have also been reported in patients with AIS [448].

The pineal gland and melatonin's role in scoliosis have also been studied extensively. Experiments on pinealectomized chickens revealed that melatonin deficiency contributes to the development of scoliosis in this model, probably by interfering with the normal symmetric growth of the proprioceptive system involving the paraspinal muscles and the spine [45, 67, 136, 330, 474–479, 889]. However, melatonin therapy after pinealectomy in chickens had no effect on the development or progression of scoliosis, thus raising doubts about its role [44, 811]. Lower melatonin levels and impaired melatonin signaling have been reported in patients with scoliosis versus controls [477, 544, 835], but other investigators have refuted this finding [7, 91, 214, 269, 308, 893]. Casting further doubt on the role of meatonin is the finding that mutations in the gene coding for human melatonin receptor are not associated with scoliosis [543, 564, 714].

Connective Tissue Abnormalities

A number of investigations have identified abnormalities in the connective tissue associated with the spine in patients with scoliosis [134, 463, 464, 878]. Differences in collagen have been found between normal individuals and those with AIS; however, this finding is not universal [819]. In histologic studies of the ligamentum flavum in scoliotic patients, the elastic fiber system was found to have disarranged fibers, a marked decrease in fiber density, and a non-uniform distribution of fibers throughout the ligament [282]. The paravertebral musculature in patients with scoliosis may exhibit abnormalities in the muscle spindle [461], in individual muscle fiber morphology [319, 665, 898], in histochemistry [580, 738], and on electromyography [21, 641]. Abnormal platelet structure and function have been reported in patients with scoliosis [228, 384, 440, 463, 550, 611, 664]. Platelet calmodulin levels in adolescents with progressive scoliosis are significantly higher than those in normal individuals, in patients with stable curves, and in those whose progressive curves were stabilized by bracing or spinal fusion [7, 384, 463]. Reduction in the serum levels of Vitamin D3 and calcitonin has been found in AIS females when compared to normal females [270].

Genetic Factors

Several extensive clinical studies of affected families conducted in the 1960s and 1970s revealed a high prevalence of familial scoliosis (6.9–11.1% of first-degree relatives) [153, 650, 876]. More recent literature has shown evidence of a strong genetic tendency in some families of patients with AIS [52, 53, 125, 353, 373, 523, 524, 668, 832]. In a meta-analysis of scoliosis in twins, monozygous twins had a significantly higher rate of concordance than did dizygous twins, and the curves in monozygous twins developed and progressed together [373].

Studies are now under way to identify the genes that cause scoliosis and its progression [248, 278, 707, 870]. Significant evidence has recently been found for association of the LBX1 locus with AIS [418]. The LBX1 protein is involved in proper migration of muscle precursor cells, specification of cardiac neural crest cells, and neuronal determination in developing neural tubes. Findings have also been reported that involve chromosomes 3p26.3 single nucleotide polymorphism [707], 6 [523], 8 [52, 53], 9 [523], 10 [869], 16 [523], 17 [523, 668], 18q [278], and 19 [125], as well as the X chromosome [353].

Clinical Features

Presenting Signs and Symptoms

Adolescents with scoliosis usually seek medical evaluation because someone has noticed an asymmetrical appearance to their back - often this is first appreciated during school screening programs for scoliosis or “well child checks” by the primary care provider. Though uncommon, back pain is present in individuals with idiopathic scoliosis more often than was previously thought. Nearly one-third of adolescents with idiopathic scoliosis complain of back discomfort at some [632,

791, 795, 871]. Interestingly, this is similar to the incidence of back pain reported in population based studies of adolescents [343, 388].

When an adolescent with presumed idiopathic scoliosis has back pain, a careful history should be obtained, a thorough physical examination performed, and plain radiographs ordered. If the pain does not limit activities and the neurologic exam is normal, a diagnosis of idiopathic scoliosis can be made, the scoliosis can be treated appropriately, and nonsurgical treatment of the back pain can be initiated.

Physical Examination

Physical examination of an adolescent with idiopathic scoliosis should be performed with the patient dressed in undergarments and an examination gown open at the back. The patient's entire back, including the shoulders and iliac crests, and lower extremities including feet must be visible. The skin is inspected closely for abnormalities such as midline discolorations, hair tufts, and dimpling in the lumbosacral region as these findings may be associated with the presence of an underlying spinal cord abnormality.

With the patient standing, the examiner determines whether the iliac crests are level. If they are not, a lower limb length discrepancy is likely, which can be quantified by placing measured blocks under the short extremity until the iliac crests are level. Leg length discrepancy can be responsible for the appearance of scoliosis. The back is then examined for asymmetry of the shoulders and flank creases, unequal scapular prominence, prominent iliac crest. Although these findings are consistent with scoliosis, the best clinical test for evaluating spinal curvature is the Adams forward-bending test [10, 100, 152]. As described above, the Adams forward bending test will reveal the degree and direction of vertebral rotation (see Figs. 28.3 and 28.4) [97]. It is prudent to remember that obesity will mask the vertebral rotation; thus, as the patient's weight increases the threshold to assess radiographically should decrease [487].

Frequently, if the patient is inspected from the front, asymmetry of the pectoral regions, breasts, or rib cage may be evident. Although these asymmetries are probably related to the spinal curvature, they may also occur in individuals without scoliosis. Occasionally, breast asymmetry is the primary concern of the patient and parents [637]. Families should be informed that correcting the scoliosis may have little, if any, influence on this asymmetry [485].

Spinal balance should be assessed in the coronal (frontal) and sagittal planes. Although patients with AIS may have a coronal shift in the trunk (rib cage) – most commonly to the right in a single thoracic curve - the head will almost always be centered over the pelvis (Fig. 28.3). Similarly, although AIS in the thoracic spine is hypo-kyphotic; patients will be compensated in global sagittal balance. If a patient has significant coronal or sagittal imbalance there should be a high degree of suspicion for a non-idiopathic etiology of the scoliosis.

A thorough neurologic assessment is mandatory in every patient with scoliosis. Range of motion and strength should be assessed in all four extremities. The hands and feet should be examined for abnormal posture and for evidence of abnormal



Fig. 28.5 Abdominal reflexes are assessed by the abdomen

sensation (excessive callus formation or nail bed irregularities). The patellar and Achilles tendon reflexes should also be tested, with the expectation that they will be symmetric [331, 888]. Finally, examination of the superficial abdominal reflexes should be performed. The abdominal reflex examination is performed with the patient supine on an examination table and the arms relaxed along the side of the body. An area approximately 10 cm above and below the umbilicus and to each anterior axillary line is exposed. With the patient relaxed, the bluntly pointed handle of a reflex hammer is used to lightly stroke the skin in each quadrant over a distance of 10 cm (Fig. 28.5).

The stroke starts lateral to the umbilicus near the anterior axillary line and is directed diagonally toward the umbilicus in each quadrant. The umbilicus is observed for deviation toward the side on which the test is performed. If these reflexes are consistently present on one side and absent on the other side, further evaluation is warranted because this finding may be the only clinical evidence of underlying pathology of the neural axis, such as syringomyelia.

Patient Maturity

Maturity can be assessed during the physical examination according to the Tanner system [782], which assesses breast and pubic hair development in girls and genital and pubic hair development in boys. Although the Tanner system may provide an indication of the patient's physical maturity, more commonly, the clinical emphasis is placed on the patient's menarchal status, increase in height over time (Peak Height Velocity or PHV) and on assessment of skeletal indicators of maturity (e.g., open or closed tri-radiate cartilage, Risser and Sanders sign and Proximal Humeral Ossification System or PHOS).

Radiographic Findings

Plain Radiography

With today's use of computed radiography and picture archive and communication systems (PACS), the initial examination of the spine should include full-length

posteroanterior (PA) and lateral radiographs on imaging plates inside 36 × 14-inch cassettes. With this, nearly all the important radiographic features can be assessed on a single image. On the PA projection, such features include the curve pattern in its entirety, the presence of any congenitally abnormal vertebrae, the overall balance of the spine and trunk, skeletal maturity (as determined by the tri-radiate cartilage, Risser sign or PHOS), and the presence of a lower limb length discrepancy (pelvic tilt). The lateral image is useful initially to evaluate the global sagittal contour of the thoracic and lumbar spine, determine the presence and severity of thoracic hypokyphosis, and screen for spondylolysis and spondylolisthesis.

A newer imaging technique, the EOS 2D/3D imaging system (EOS Imaging, Paris), has been used with increasing frequency over the past 10 years. The advantages of this system are the significant reduction in radiation dose (50–80% less than conventional x-rays), the ability to obtain simultaneous anteroposterior and lateral 2D images of the whole body, and the ability to perform 3D reconstructions. Although the "microdose" images have slightly less clarity qualitatively, the reliability of curve measurements are comparable to standard radiologic techniques [261, 323, 327, 502, 514, 570].

Measurement of Curve Magnitude

The Cobb method is considered the standard for measuring curve size [38] (Fig. 28.1). Although the Cobb method has good overall reliability [399], some variation among different observers' measurements is always present. Such variability averages 7.2° if the end vertebrae are not preselected but improves to 6.3° when they are preselected [546]. Another aspect of the accuracy of the Cobb method is that to achieve 95% statistical confidence that a true change in curve size has occurred, a measurement difference of 10° between radiographs taken at different times would be needed [118]. The intra- and inter-rater variability in Cobb angle measurements is of substantial clinical significance as the decision to initiate brace treatment is often made on documented progression in skeletally immature patients. Additionally, most of the literature uses a criteria of a 5–6° increase in curve size to determine the success or failure of brace treatment.

Measurement of Vertebral Rotation

Today, the EOS 2D/3D imaging system can reliably measure apical vertebral rotation preoperatively and postoperatively [885]. This requires reconstruction of the spine into a 3D image. Historically, the Perdriolle method and the Nash-Moe method were the two most common means of assessing vertebral rotation on plain frontal radiographic films. Neither of these two methods are commonly used today as most PACs systems do not include software that allows these measurements [370, 559, 591, 612, 644].

Diagnosis of Exclusion

Patients with a syringomyelia or syrinx frequently present without symptoms and with scoliosis as the only physical finding (Fig. 28.6).



Fig. 28.6 Sagittal MRI showing holocord cervical syrinx

Thus, it is important to have a high degree of suspicion to identify these patients. Most commonly, the convexity of thoracic curves in AIS is directed to the right. Left thoracic curves are more common in those with an underlying syrinx [129, 178, 216, 579, 595, 632, 693, 799]. Sagittal plane deformity in an individual with apparent idiopathic scoliosis may also be an indicator of syringomyelia [595, 628, 646]. If hypokyphosis is *absent*, a diagnosis of idiopathic scoliosis should be made only after a syrinx has been ruled out. As previously noted, asymmetrical abdominal reflexes are also associate with syringomyelia.

Magnetic Resonance Imaging

MRI is a valuable tool in the assessment of scoliosis as it can identify soft tissue abnormalities within the neural axis. Syringomyelia, Chiari malformations, abnormalities in the brainstem, spinal cord tumors, spinal cord tethering, diastematomyelia, and intervertebral disc herniation have all been identified in individuals previously thought to have idiopathic scoliosis [178, 257, 321, 331, 420, 436, 598, 646, 693, 710, 860]. MRI is usually reserved for patients with an atypical manifestation of scoliosis [185, 542]. Although atypical manifestations have never been specifically defined, they generally include patients with abnormal neurologic findings such as weakness, hyper-reflexia, progressive foot deformities or asymmetric abdominal reflexes; patients with left thoracic curves or excessive thoracic kyphosis

or unusually rapid curve progression. Routine preoperative MRI is not indicated for typical AIS if findings on the neurologic examination are normal [178, 582, 710, 860].

Treatment

There are three widely scientifically accepted treatments for AIS: observation, bracing and surgery. Scoliosis specific exercises are another treatment undergoing intensive investigation. The magnitude and location of the curve and the remaining growth of the patient are the primary considerations in determining treatment (see Table 28.2). Immature patients with small curves that may not progress [19, 457] and mature patients with curves below the threshold for progression (45–50° depending on curve location) are observed. Bracing has been shown to be effective in limiting curve progression (but does not make the curve smaller) in patients who have not achieved skeletal maturity and have curves between 25° and 45° [659]. In most cases, growing adolescents with curves exceeding 45–50° require operative stabilization because non-operative forms of treatment are ineffective in controlling or correcting the scoliosis. Skeletally mature individuals with curves exceeding 50–55° are also at risk for continued curve progression and should be considered for surgical treatment [847]. Possible exceptions include patients with well-balanced double curves less than 60 who are not bothered by their clinical appearance.

Observation

In general, regardless of the patient's maturity, no treatment is needed for curves less than 20°. The frequency of follow-up examination is dependent on the patient's remaining growth and the magnitude of the curve. During adolescent growth, if curves progress, they do so at an average of a degree a month; given this fact and the 5–7° measurement variability in Cobb angles, observation during growth is most commonly done at six-month intervals. Patients with 15–25° curves who are just entering peak growth velocity (TRC open, Sanders 1–2, PHO 1–2) may be seen at 4 month intervals to facilitate timely brace initiation should their curves progress. Patients who are near or at the end of growth (Risser 4–5, Sanders 6–8, PHO 4–5) may be seen yearly. After skeletal maturity patients need to be seen very infrequently (2–10 years).

Orthotic (Brace) Treatment

In 1946 the Milwaukee brace was developed to replace postoperative plaster cast immobilization that was used during that era. Later, use of the brace was expanded as a method of nonoperative scoliosis treatment with the thought that the passive, active, and distraction forces exerted by this brace might be beneficial in preventing curve progression. In the 1960s, thermoplastics were introduced into orthosis manufacturing, which led to the thoracolumbosacral orthoses (TLSOs) used today. In recent years, computer-assisted design and computer-assisted manufacturing (CAD/CAM) have been used to fashion spinal orthoses, and the superiority of this technique compared to previous plaster-cast designs has been shown [144, 159, 205, 638].

Evidence Supporting Bracing Effectiveness

Historical studies were inconclusive regarding brace effectiveness, largely because of the inability to measure brace wear compliance and the inclusion of patients who were relatively skeletally mature and at low risk for progression [41, 75, 363, 364, 528, 563, 607, 846]. With the introduction of temperature sensors, that measure brace wear compliance, a number of studies have shown braces effective in limiting curve progression in growing patients. The most prominent of these studies by Weinstein [846], Katz [365], and Karol [363] objectively quantified compliance (hours in brace) through the use of a heat sensor in the orthosis. In Weinstein's BrAIST study of a randomized cohort and a preference cohort, bracing significantly decreased the progression of high-risk curves to the threshold for surgery and the benefit increased with longer hours of brace wear. In Katz' study, the total number of hours of brace wear inversely correlated with curve progression. This effect was most significant in patients who were at Risser stage 0 or 1 at the beginning of treatment. Curves did not progress in 82% of patients who wore the brace more than 12 hours/day, as opposed to only 31% of those who wore the brace less than 7 hours/day. However, in Karol's study, patients at Risser stage 0 are at risk for surgery despite brace wear. In these patients, 12.9 hours of daily wear (the number of hours linked with a successful outcome in Weinstein's BRAIST study) did not prevent surgery. Patients with open triradiate cartilage were at highest risk, especially those with curves of ≥30°.

Indications for Brace Treatment

Brace treatment is indicated in children to prevent curve progression during further skeletal growth. In general, bracing is indicated in growing adolescents (Risser 0–2, Sanders 1–5, PHO 1–4) who on initial evaluation have curves in the range of 25–45°. The concept for early bracing (curves 15–25° with concurrent clinical deformity and documented progression) in the immature patient (TRC open, Sanders 1–2, PHO 1–2) is gaining increased acceptance as evidence is emerging that predictable progression toward surgery can occur in immature patients with curves ≥30° at the time of brace initiation [363]. Patients should consider their existing deformities cosmetically acceptable and must be willing to wear the brace the prescribed amount of time.

Brace Treatment Protocols

Current braces are designed to be worn full time (16–20 hours/day; these include Boston, Custom TLSO or Chenau braces) or night time (8 hours/day: Providence or Charleston brace). The dynamic flexible Spine-Cor brace has shown failure rates significantly higher than that of a rigid spinal orthosis that have limited its widespread use [279, 873]. The Charleston "night time bending brace" was designed to achieve more correction of the scoliosis in hopes that this would allow it to be as effective as a full time brace – when only worn 8 hours at night. Multiple studies have shown that it is effective in treating thoracolumbar or lumbar curves <35° [75, 159, 219, 341, 366, 623, 624]. Recently, the Chenau brace has been introduced in an attempt to brace the scoliosis in three dimensions rather than classic Boston/TLSO/

night time bending braces which have only addressed the coronal plane [23, 166, 215, 394, 648].

There is a growing body of evidence that the amount of prescribed brace wear should correlate with the patient's growth potential. Karol has recommended that Risser stage-0 patients should be prescribed a minimum of 18 hours of brace wear per day [296, 363]. Her work also demonstrated that providing patients undergoing bracing for AIS with feedback about their compliance with brace wear improves that compliance [364]. Table 28.3 summarizes the current consensus guidelines at our institution regarding observation or brace and type of brace.

After a brace has been prescribed and delivered the patient returns after a month of brace wear for an in-brace radiograph. In brace correction for full time TLSO braces should be 30–50% and 80–100% for night-time bending braces. Patients then return at six-month intervals with coronal radiographs out of the brace to monitor potential curve progression/brace effectiveness.

With female patients, when a brace has been successful in controlling curve progression, the braced is discontinued when no further increase in height has occurred; this usually corresponds to 18–24 months post-menses; Risser 4, Sanders 7 and PHO 5. In male patients, the spine continues to grow to Risser 4–5 [362]. Therefore, in boys, bracing may need to be continued until Risser 5, Sanders 7 and PHO 5 is achieved. Frequently, this does not occur until the later teenage years, which makes compliance with brace wear a challenge.

Physical Therapy and Biofeedback

Consistent asymmetry in torso rotation strength has been documented in patients with AIS [540]. Although muscle conditioning is beneficial to a patient's overall well-being, only modest evidence supports the concept that physical therapy programs are helpful in controlling or improving scoliosis. In Europe, the use of physical therapy exercises and biofeedback to enhance the effectiveness of bracing (Schroth technique and others) is common [65, 561, 562, 728, 892], and has been reported beneficial in several North American centers [688, 895].

Electrical Stimulation

Electrical stimulation was used as an alternative to bracing in the early 1980s. Surface muscle stimulators were placed over the muscles on the convex side of the scoliotic curve and were activated for approximately 8–10 hours each night. In Canada, electrode stimulators were actually implanted in the paraspinal muscles. Although some preliminary success was reported with transcutaneous stimulation [26], most studies found that this form of treatment did nothing to favorably alter the natural history of scoliosis [63, 199, 556, 585]. Today, electrical stimulation is no longer considered a useful method in the management of idiopathic scoliosis.

Surgical Treatment

The primary surgical treatment for AIS is spinal fusion. Today this is done most commonly through a posterior approach using pedicle screw instrumentation and allograft bone [381, 424]. In 2019 the FDA approved a flexible tethering device that

can be used to surgically treat scoliosis without fusion. It is placed into the vertebral bodies anteriorly – often via a thoracoscopic approach. Historically, the goal of spinal fusion was to halt progression of the curve; however, modern surgical techniques provide powerful means to correct the deformity, thus patients today can expect a spine that is well balanced in both the coronal and sagittal plane and significant radiographic and clinical improvement in their deformity.

Indications for Surgery

The primary indication for surgical treatment is curve magnitude. Thoracic curves and double major curves that exceed 50° and thoracolumbar/lumbar curves that exceed 45° at skeletal maturity have a significant probability of worsening over time and warrant operative intervention [847]. Because of the variability in Cobb angle measurements, there is some "gray area" in curves between 40° and 60°. Particularly in these patients, the clinical appearance (as perceived by the patient, the family, and the surgeon) plays a role in surgical decision making.

Pain is usually not an indication for surgery in AIS. As previously discussed, approximately 30% of patients with AIS have non-activity limiting back pain. Interestingly, recent studies suggest that many patients with pain may improvement with surgical treatment [184, 262].

Preoperative Planning

Preoperative planning involves careful clinical and radiographic assessment to determine which curves and levels to include. The clinical exam should assess for shoulder height differences, waistline asymmetry and trunk balance with the patient standing. The Adam's forward bend test demonstrates axial plane deformity in the thoracic and lumbar spine (Figs. 28.3 and 28.4).

Curves in males appear to be more rigid than those in females with AIS [303, 760]. When planning surgery in males, less curve correction and greater blood loss should be expected. Although short- and long-term functional outcomes are similar to those in females [408], complication rates are higher [168].

Curve Flexibility

Determination of preoperative curve flexibility is an integral part of the pre-operative assessment and necessary to use the Lenke classification – the most widely accepted system for classifying operative AIS. Curve flexibility can be assessed using including supine best-effort side-bending radiographs; the fulcrum bend test; a supine resting radiograph or traction radiographs. A recent literature review suggests that the supine best-effort bend radiograph can be used for both thoracic and lumbar curves while the traction view is best for larger curves and the fulcrum bend is best for thoracic curves (and) and not lumbar curves [362]. We routinely use supine best-bend radiographs.

Lenke Classification System

Lenke and co-workers developed a new classification system for AIS in 1997 designed to encourage three-dimensional analysis of scoliosis, help determine

which curves should be fused and to achieve greater intraobserver and interobserver reliability [427]. The Lenke classification system is dependent on curve measurements in both the frontal and sagittal planes [427, 428]. The three main variables requiring evaluation are curve type (Table 28.4), lumbar spine modifier, and thoracic sagittal modifier.

Although this system has good inter and intraobserver reliability [427, 428], there different surgeons still have significant variations in fusion levels [118, 546].

Table 28.4 Lenke classification system

Curve type	Description	Characteristic curve patterns			Structural region
		Proximal thoracic	Main thoracic	Thoracolumbar or lumbar	
1	Main thoracic	Nonstructural	Structural (major) Cobb angle: ≥25° on side-bending radiographs Kyphosis: +20° between T10 and L2	Nonstructural	Main thoracic
2	Double thoracic	Structural Cobb angle: ≥25° on side-bending radiographs Kyphosis: +20° between T2 and T5	Structural (major) Cobb angle: ≥25° on side-bending radiographs Kyphosis: +20° between T10 and L2	Nonstructural	Proximal thoracic, main thoracic
3	Double major	Nonstructural	Structural (major) Cobb angle: ≥25° on side-bending radiographs Kyphosis: +20° between T10 and L2	Structural Cobb angle: ≥25° on side-bending radiographs Kyphosis: +20° between T10 and L2	Main thoracic, thoracolumbar, or lumbar
4	Triple major	Structural Cobb angle: ≥25° on side-bending radiographs Kyphosis: +20° between T2 and T5	Structural (major) Cobb angle: ≥25° on side-bending radiographs Kyphosis: +20° between T10 and L2	Structural (major) Cobb angle: ≥25° on side-bending radiographs Kyphosis: +20° between T10 and L2	Proximal thoracic, main thoracic, thoracolumbar, or lumbar

(continued)

Table 28.4 (continued)

Curve type	Description	Characteristic curve patterns			Structural region
		Proximal thoracic	Main thoracic	Thoracolumbar or lumbar	
5	Thoracolumbar or lumbar	Nonstructural	Nonstructural	Structural (major) Cobb angle: ≥25° on side-bending radiographs Kyphosis: +20° between T10 and L2	Thoracolumbar or lumbar
6	Thoracolumbar or lumbar, main thoracic	Nonstructural	Structural Cobb angle: ≥25° on side-bending radiographs Kyphosis: +20° between T10 and L2	Structural (major) Cobb angle: ≥25° on side-bending radiographs Kyphosis: +20° between T10 and L2	Thoracolumbar or lumbar, main thoracic

From Tachdijain's Pediatric Orthopaedics 6th Edition

Despite this limitation, the Lenke classification system offers a more comprehensive preoperative radiographic classification of patients with AIS than was available with previous systems and appears to correlate with surgical treatment of structural regions of the spine [59, 426, 431].

The first parameter to identify in the Lenke classification system is curve type which is determined by identifying the largest curve. The other curves are then deemed structural by magnitudes that are greater than 25° on the supine best-bend radiograph or if junctional kyphosis (measured between T2 and T5 for the proximal and main thoracic curves and between T10 and L2 for the main thoracic and thoracolumbar/lumbar curves) is greater than 20°. The second variable, the lumbar spine modifier, is determined by drawing the center sacral vertical line (CSVL-vertical line constructed upward from the center of the sacrum) and assessing the concave pedicle of the apical lumbar vertebra relative to the CSVL. The final variable, the thoracic sagittal modifier (T5-12), is then assessed to define the sagittal plane deformity. The sagittal modifier is hypokyphotic (<10°), normal (10–40°), or hyperkyphotic (>40°).

Fusion Levels

Although the Lenke classification provides a framework to discuss and compare different AIS curves, choosing fusion levels for AIS remains a nuanced skill. In addition to the radiographic parameters identified in the Lenke system, the determination of fusion levels must also take into consideration the clinical appearance, skeletal maturity the patient and the expectations of the family [46, 517].

Lenke type 1A and 1B curves are single thoracic structural curves with nonstructural lumbar curves that do not cross the midline (CSVL). Clinically, trunk imbalance is more pronounced to the right in these patients than in those with double-curve patterns. For posterior constructs the upper instrumented vertebra (UIV) is generally the upper end vertebrae (UEV) or one proximal to this when the UEV translated off the midline. The selection of the lower instrumented vertebrae (LIV) is more varied, but most commonly is the last vertebra substantially touched by the CSVL as one travels from distal to proximal (LSTV).

Further subdivisions of the 1A pattern based on the tilt of L4 provides some guidance as to extend the fusion more distal. In the 1A "R", where L4 is tilted to the right and the stable vertebra is more distal (usually L3 or L4) fusion is often necessary to L2 while the 1A "L" curve have L4 tilted to the left, with a stable vertebra at the thoracolumbar junction, and the LIV is usually T12 [532]. This 1A-R curve essentially describes the "King 4" curve which is a thoracic curve with the L4 vertebra tilted into the curve and fusion more distal is appropriate [385].

Patients with Lenke type 1C curves have single thoracic structural curves with nonstructural lumbar curves with the apical lumbar vertebra completely crossing the midline. This curve pattern has generated controversy whether to perform fusion of only the thoracic curve (selective thoracic fusion, STF) or to include the lumbar curve in the fusion. Proponents of STF note the thoracic curve is the only structurall curve and saving motion segments provides greater lumbar flexibility which benefits the long-term health of the spine. Supporting this argument, Enercan et al. compared STF patients to an *age-matched normal group* and demonstrated overall excellent results in the STF group with only mild degeneration of the disc levels [207]. The primary concerns with performing a selective fusion has been whether the patient remains balanced over time and avoiding decompensation [429, 643]. These concerns are manifested in the differences with which surgeons approach these curves; only 49% of surgeons from one multicenter study chose a STF. The factors which correlated with performing the STF were smaller lumbar curves with less clinical deformity, larger ratio of thoracic to lumbar Cobb angles and patients who were less concerned about their clinical appearance. Twenty year follow up of STF patients demonstrates a stable lumbar curve magnitude and maintenance of the L4 with similar SRS outcome scores compared to long fusion into the lumbar spine for similar curves [412]. In general, we prefer to perform a selective thoracic fusion wherever possible as we think preserved motion segments in a well-balanced spine is superior in the long-term to longer fusion with fewer lumbar motion segments (Fig. 28.7).

Patients with Lenke type 2 curves have double thoracic structural curves with nonstructural lumbar curves. The first thoracic vertebra is tilted into the upper curve, with junctional kyphosis between the proximal and main thoracic curves. The patient's shoulder on the side of the convexity of the upper curve (most commonly the left) is nearly always elevated unless the main thoracic curve is so large thus compensating for the shoulder elevation produced by the structural proximal thoracic (PT) curve. Inclusion of the PT curve with fusion to T2 should be strongly

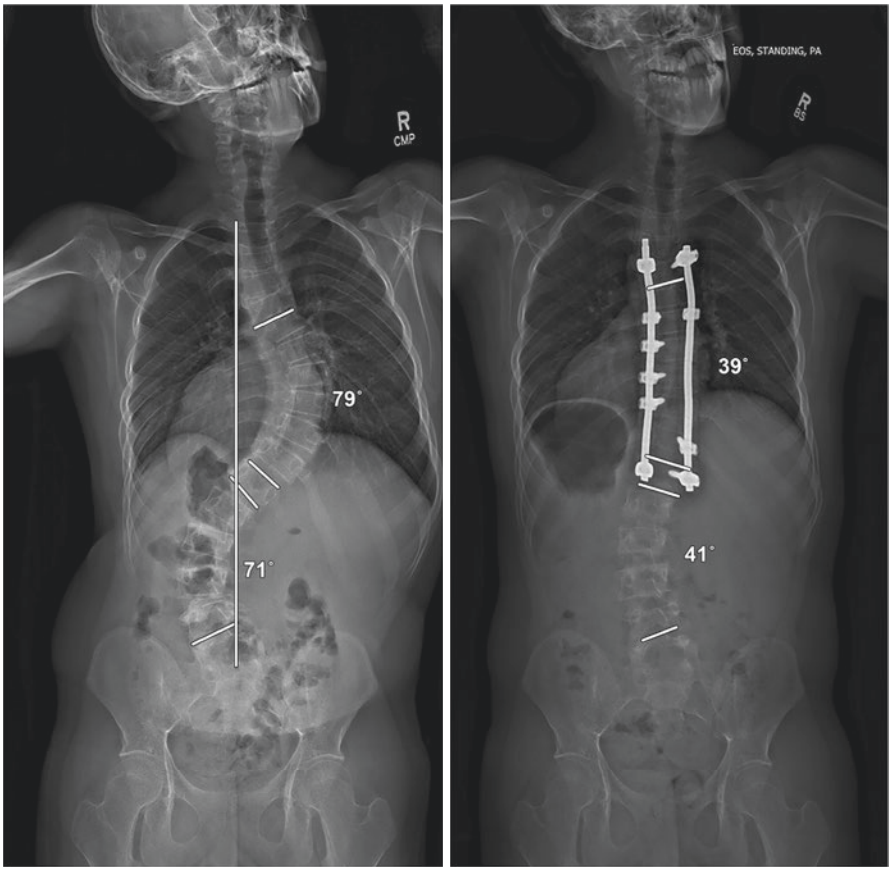


Fig. 28.7 Pre- and post-operative AP radiographs of a patient treated with an instrumented selective thoracic posterior fusion

considered when the T1 is tilted into the upper curve and the shoulder is elevated on the convex side of the upper curve [398, 419, 430, 856, 857]. Although left shoulder elevation on the clinical examination is an indication for inclusion of the PT curve, level shoulders or even a slightly elevated right shoulder may require fusion of the PT curve when the main thoracic (MT) curve is >75° and the intended correction of this curve is significant. The correction of this MT curve will push the left shoulder up unless the instrumentation includes fusion to T2.

Lenke type 3 curves represent double major structural curve patterns in which both the thoracic and lumbar components require instrumentation. The choice of LIV first depends on the distal end vertebra of the lumbar curve with the most common controversy being whether to stop at L3 or L4. This decision should be made with careful consideration of the distal end vertebra, the magnitude of the lumbar cure and the location of the apixal vertebra. Curve characteristics that allow stopping short of the end vertebra include thoracolumbar or lumbar curves smaller than

55°, flexible curves, and apex of the thoracolumbar or lumbar curves that is two or more levels proximal to the intended LIV. Selective fusion of 3C curves is possible in very few situations and risks significant coronal imbalance greater than 2 cm (56% vs 10%) as demonstrated recently by Singla et al. who reviewed 74 patients with Lenke 3C curves comparing those who had a STF and those who did not [719]. Useful guidelines have been developed to help differentiate these curve patterns using relative ratios between the thoracic and lumbar curves with regard to their size, rotation, and deviation from midline can be assessed preoperatively on a standing radiograph [429]. If thoracic curve Cobb:lumbar curve Cobb ratio is less than 1.0, both curves will require fusion. If the ratio is greater than 1.2 selective thoracic fusion can be performed safely. When determining whether to perform a STF or include the lumbar curve in the fusion, a careful assessment of the clinical appearance of the patient is necessary to identify structural lumbar rotational deformities and to understand the patient's perception of their deformity especially as it relates to the lumbar curve and whether this is acceptable as this deformity will remain following a STF (Fig. 28.7).

Lenke Type 4 curves are triple major curve patterns in which both thoracic curves, in addition to the lumbar curve, are structural. All three curves require posterior instrumentation.

In Lenke type 5 curves, only the thoracolumbar or lumbar curve is structural. In the past, the anterior approach was the most common method to treat the curves with excellent three-dimensional correction [255, 370, 834, 836]. Today, the posterior approach with pedicle screw fixation is the most common method to treat these patients. Several studies many have compared the two methods with equivocal results [2, 190, 194, 256, 530, 584]. The choice between the two approaches often comes down to the site of the surgical incision as well as the risk profile of each approach. Operative time, blood loss and length or stay are less with a posterior approach. The anterior approach risks a higher pseudoarthrosis rate while having no /little risk for postoperative infection. The posterior approach has little risk of pseudoarthrosis but risks infection- both acute and delayed [312, 344, 358, 359, 812].

Lenke type 6 curves represent double curve patterns, with the primary thoracolumbar or lumbar curve accompanied by a smaller but structural main thoracic curve. Generally, fusion of both curves is appropriate since both curves are structural. A selective lumbar fusion can be considered if the thoracic deformity is small. Sanders et al. found satisfactory results with selective fusion if the lumbar Cobb: thoracic Cobb ratio was 1.25 or greater [680, 690].

Transfusion Requirements

Significant research has focused on methods to limit allogeneic blood transfusion in patients undergoing surgery for AIS. Current strategies to limit blood loss/transfusion include controlled hypotensive anesthesia (during exposure, with return to normal during correction of deformity to insure adequate spinal cord perfusion), tranexamic acid (TXA) and intraoperative salvage of lost blood [25, 147, 149, 220, 229, 230, 253, 324, 351, 396, 470, 541, 553, 590, 615, 715, 717, 766, 814, 880].

Bone Grafting

Spinal fusion is enhanced by meticulous "stripping of the spine", appropriate facetectomies, careful decortication and adequate bone grafting. Although autogenous iliac crest bone grafting (ICBG) is the "historical standard" [141, 824] the postoperative pain and second incision/scar associated with these are not insignificant [274, 725]. Numerous studies of successful fusions using allograft bone as a substitute for autogenous bone have been reported without an increase in pseudarthrosis rates [42, 72, 187, 211, 274, 409, 754, 790]. A recent multi-institution study of 461 AIS patients undergoing posterior spinal fusion and instrumentation had no differences in complication rates including pseudoarthrosis whether autologous iliac crease bone graft, allograft or bone graft substitutes were used [409]. A meta-analysis which compiled 2389 patients from 12 studies compared autologous ICBG with allograft following AIS surgery. The fusion rates were the same between groups but the ICBG patients had greater operative time and blood loss with more postoperative pain [387]. These data suggest little need for autologous bone graft for AIS surgery. To minimize the risk of transmitting human immunodeficiency virus, hepatitis virus, and any other potential viral pathogens, the donor blood and tissue are tested at the site of recovery, and testing is usually continued throughout the harvesting process. Freeze-dried cancellous bone is usually exposed to low-dose gamma radiation to sterilize all nonsystemic bacterial and fungal contaminants.

Spinal Cord Monitoring

Spinal cord monitoring using both spinal somatosensory evoked potentials (SSEPs) and motor evoked potentials (MEPs) is the standard of care during scoliosis surgery and is critical to the safety of any spine deformity surgery. SSEPs record the sensory function of the spinal cord and provide continuous monitoring throughout the procedure [29, 130, 131, 133, 305, 468, 547, 581, 602]. This test may, however, be adversely affected by changes in anesthetic level and critical changes tend to lag behind MEPs. With impending neurologic deficit, MEPs are used to monitor the anterior spinal cord motor tracts and are ideally performed by applying a stimulus to the motor cortex of the brain (transcranial MEPs [tcMEPs]) and detecting a distal response in the muscles of the arms and legs [61, 263, 599, 692, 751, 779]. Total intravenous anesthesia (TIVA) with propofol is necessary to obtain good tcMEP data.

Critical neuromonitoring changes are a decline of 50% of SSEP data or a 60–80% decline in TcMEP data. The intra-op response to critical neuromonitoring changes should be planned in advance and a checklist is important to ensure all members of the surgical team are working together to ensure the patient awakens without permanent neurologic deficit. A checklist was developed using a Delphi method with expert surgeon, neurologist and neurophysiologist participation [829] (Fig. 28.8).

The critical initial steps include ensuring there are no technical problems with the monitoring, raising the mean arterial pressure, ensuring normal body temperature, checking a hemoglobin/hematocrit [882]. Following this, the steps of the procedure should be reversed in the order each step was performed to include reducing the amount of surgical correction with removal of rod(s) and checking screw position. Ultimately, the correct management may include waking the patient fully

Checklist for the response to intraoperative neuromonitoring changes in patients with a stable spine

Gain control of room	Anesthetic/systemic	Technical/ neurophysiologic	Surgical
☐ Intraoperative pause: stop case and announce to the room	☐ Optimize mean arterial pressure (MAP)	☐ Discuss status of anesthetic agents	☐ Discuss events and actions just prior to signal loss and consider reversing actions: ☐ Remove traction (if applicable) ☐ Decrease/remove distraction or other corrective forces ☐ Remove rods ☐ Remove screws and probe for breach
☐ Eliminate extraneous stimuli (e.g., music, conversations)	☐ Optimize hematocrit	☐ Check extent of neuromuscular blockade and degree of paralysis	☐ Evaluate for spinal cord compression, examine osteotomy and laminotomy sites
☐ Summon ATTENDING anesthesiologist, SENIOR neurologist or neurophysiologist, and EXPERIENCED nurse	☐ Optimize blood pH and pCO_2 ☐ Seek normothermia	☐ Check electrodes and connections ☐ Determine pattern and timing of signal changes	☐ Intraoperative and/or perioperative imaging (e.g., O-arm, fluoroscopy, x-ray) to evaluate implant placement
☐ Anticipate need for intraoperative and/or perioperative imaging if not readily available	☐ Discuss POTENTIAL need for wake-up test with ATTENDING anesthesiologist	☐ Check neck and limb positioning; check limb position on table especially if unilateral loss	

Ongoing considerations

- ☐ REVISIT anesthetic/systemic considerations and confirm that they are optimized
- ☐ Wake-up test
- ☐ Consultation with a colleague
- ☐ Continue surgical procedure versus staging procedure
- ☐ IV steroid protocol: Methylprednisolone 30 mg/kg in first hour, then 5.4 mg/kg/h for next 23 hours

Fig. 28.8 Checklist for best practices in intraoperative neuromonitoring in spine. (From Tachdijain's Pediatric Orthopaedics 6th Edition)

following closure of the wound to perform a complete and comprehensive neurologic examination and returning to the operating room another day.

When MEPs are used in conjunction with SSEPs, the chance of unrecognized injury to the spinal cord is minimized A large multicenter study of 1121 patients demonstrated that 38 (3.4%) had a critical change on monitoring when tcMEPs and SSEPs were used. The tcMEP/SSEP combination did not miss any patient with a transient motor or sensory deficit [691]. We have recently completed a 20 year review of 1524 AIS patients who underwent multimodal spinal cord monitoring and demonstrated a critical alert in 2.1% of patients. There were two transient and no permanent neurologic deficit [763]. Similar studies have demonstrated excellent results with combined multimodal intraoperative neuromonitoring during AIS surgery [221, 608, 798, 891].

The wake-up test, a gross evaluation of motor function, was first developed and used routinely prior to the development of spinal cord monitoring. The wake-up test can still be used today when critical changes in SSEPs or MEPs are noted during correction of the spine because spinal cord injury may exist even when monitored variables return to baseline [577]. For this test the anesthesiologist allows the patient to regain partial consciousness and motor function during the surgical procedure [285].

Technique for Posterior Spinal Fusion with Instrumentation

Regardless of the diagnosis, instrumented posterior spinal fusion can be broken into the following steps: exposure, facetectomies, anchor (screws, hooks, bands or wires) placement, spine mobilization (when indicated), deformity correction, decortication/bone grafting and closure. Meticulous detail to each step will facilitate efficient completion of the next step and, with selection of appropriate fusion levels and deformity correction, should result in a solid arthrodesis and a well-balanced spine in both planes.

The use of pedicle screws has dramatically changed the operative treatment of spine deformity providing and maintaining greater three dimensional correction [49, 288, 770]. Suk first reported the routine use of pedicle screws for scoliosis correction in the thoracic spine in 1995. They demonstrated improved coronal plane correction in the screw group (72%) compared to hooks (55%) and hybrid constructs (66%) [771]. The improved three dimensional correction achieved with pedicle screws has led to near universal adoption with improved radiographic correction compared with more traditional hook constructs [115, 381, 382, 398, 443, 620, 735, 753, 772, 821].

Placement of pedicle screws can be performed with a variety of methods including the free-hand technique [395, 583, 601, 603, 604, 816, 817, 900, 901] (use of anatomic landmarks together with fluoroscopy when needed), fluoroscopically-guided screw placement (use of fluoroscopy to visualize the exact trajectory of the screw) [297, 401, 769] or navigation systems [9, 124, 241, 280, 356, 403, 404, 480, 600, 625, 636, 815, 890] (use of a preop or intraop computed tomography which is registered with a computer system to identify and watch the trajectory of the screws as they are placed).

Regardless of how they are placed, it is important to understand the anatomy of pedicles in the scoliotic spine. In general, the width of the thoracic pedicle is smaller in the proximal part of the thoracic spine, on the concavity of the upper and main thoracic curves, and with greater curve magnitude. The spinal cord is positioned adjacent to the concave pedicles with less than 1 mm of epidural space, compared to 3–5 mm on the convex side [442]. At the apex of a right thoracic scoliosis, the aorta is positioned more lateral and posterior to the vertebral body than in a normal, straight spine [442, 757]. The most challenging pedicles are the concave screws, in general, and especially those of the proximal thoracic curve [164]. Despite the challenges, pedicle screws are safe: “breech rates” have been reported between 1.5% and 15% with documented improvement with increasing surgeon experience and few neurologic complications [6, 57, 177, 398, 677, 769].

There is great variability between surgeons in “implant density” – the number of anchors/level fused. On the extreme end, some surgeons routinely place screws on each side of every level fused (implant density 2.0) – regardless of curve size/stiffness. Other surgeons are more strategic in their use of screws/anchors. The appropriate implant density is not known. Some studies have shown improved coronal correction with high density constructs [143, 414] while others have failed to demonstrate a difference between high and low density constructs [68, 171, 254, 268,

415, 663]. Higher density constructs are more costly, one study reported a potential savings of 11–20 million dollars a year with low density constructs [413].

Spine Mobilization

Traditionally, a facetectomy referred to the removal of the inferior facet and is the standard technique to achieve fusion. The use of pedicle screws provides opportunity for greater three-dimensional correction and has led to use of more aggressive spine mobilization procedures [27]. In increasing order of "spine mobilization" (and subsequently, increasing neurologic risk) these techniques include: complete facetectomies (including superior facet) and ligamentum release (Ponte or Smith-Petersen osteotomies), concave and/or convex rib resections, vertebral body decancellation with wedge resection of the vertebra, pedicle subtraction osteotomy, and finally, a vertebral column resection (VCR).

A Ponte-style osteotomy generally refers to complete removal of both the superior and inferior facet and ligamentum. It was originally described for the treatment of Scheuermann's kyphosis, allowing shortening of the posterior column [618]. The Smith-Petersen osteotomy, typically refers to performance of these complete facetectomies in the setting of a fully or partially arthrodesed anterior column. The effectiveness of the Ponte osteotomy to allow posterior column shortening when treating hyper-kyphosis is unquestionable. However, there is significant controversy regarding their routine use in spinal fusion for AIS. In general, we do not routinely use Ponte osteotomies for a typical 45–65° thoracic AIS, rather we selectively perform these in patients with larger curves (>70°) especially when there is significant hypokyphosis and rotational deformity. We recently compared 34 AIS patients with curves averaging 70° who had Ponte osteotomies to a matched group without Ponte osteotomies. The osteotomy group had 10% (7°) more coronal plane correction but no difference in sagittal correction or SRS outcomes scores. The incidence of IONM changes was significantly higher in the Ponte group – a finding identified by others including Shah et al. who had an 8% incidence of IONM changes in AIS patients undergoing Ponte osteotomies [700].

The next level of posterior release is rib resections on the concave side of the spine to allow lateral and posterior translation of the spine. We do not generally perform these rib resections to improve curve flexibility because experience has shown limited appreciable correction and the risk for postoperative pulmonary issues is high.

The VCR procedure is the most aggressive technique for achieving correction of the spine because it removes one or more vertebral segments at the apex of the deformity, however it is rarely necessary in AIS treatment. It was initially described for severe spine deformity/trunk shift and was performed using an a combined anterior/posterior approach. Bradford and Tribus reported 50% curve correction and 80% translation correction in 24 patients [86]. Suk and co-workers were the first to describe the posterior-only approach with 62% correction in the coronal plane and 45° of correction in the sagittal plane in 70 patients with severe deformity from kyphoscoliosis, post-infectious kyphosis, and adult scoliosis. Similar to previous

reports, the complication rate was relatively high, with 24 patients having complications, including 2 with postoperative spinal cord deficits who had neurologic deficits preoperatively [767]. Riley, Lenke et al. recently reported a large series of both adult and pediatric patients who had minimum 5 year follow-up after a VCR [649]. The pediatric patient population consisted of 31 patients who achieved a 61.6% coronal correction, with 16.1% of patients having a proximal junctional kyphosis, and 9% incidence of neurologic deficit. However, patients reported excellent improvement in self-image and satisfaction. We perform VCR in individuals with very severe deformity—angular scoliosis or kyphosis—especially when previous fusion has been performed. This procedure should be performed only by skilled and experienced surgeons who have worked their way up the learning curve by performing less arduous spine mobilization procedures. A multicenter study of 147 pediatric VCR procedures performed by senior surgeons reported complications in 59% of patients, including 27% who had an intraoperative neurologic event without paraplegia [432].

Techniques for Deformity Correction

Historically, Harrington achieved coronal plane correction with distraction. This had the unfortunate consequence of creating abnormalities in the sagittal plane. Cotrel and Dubousset introduced the concept of a 90° concave rod rotation maneuver in an attempt to improve the three-dimensional deformity. The rod was shaped to the coronal plane deformity, and for the typical right thoracic curve, the left rod was rotated counterclockwise so that the scoliotic bend in the rod became the kyphotic bend in the rod to maintain or restore kyphosis. Today, with modern implants (including stiff rods), a better understanding of deformity and improved ability to monitor the spinal cord, better correction in all three planes is safely achieved – even in large AIS deformities.

The most common method of deformity correction today is to use the anchors (typically screws, occasionally wires or bands) to translate the apex of the spine posteriorly and laterally to an over-contoured stiff rod. A stiff cobalt-chrome 6.0-mm diameter rod is preferred in general by the authors, especially for severely hypo-kyphotic spines. The left rod is seated and fixed in the most distal screw the proximal set screw is left loose in more rigid curves to allow for some lengthening of the spine. The reducing devices on the concave side are then used to bring the left apex up to the rod to improve the coronal, sagittal and axial planes of the deformity. The stiff rod should allow nice correction and may need to be partially derotated due to the forces acting on the spine during the translation correction. During this translational correction the bone-screw interface should be visualized to ensure significant screw pullout does not occur and it should be recognized that the rod will flatten out to a certain extent depending on the stiffness of the deformity and the desired amount of correction.

Following initial placement of the left rod, in situ coronal bending can be performed as well as distraction to improve the main thoracic curve and compression to pull the left shoulder distal for proximal thoracic curves. Further direct vertebral rotation at the apex can be performed by attaching instruments to de-rotate either

segmentally, with each screw head being manipulated individually to correct the axial plane or en-block after linking the screws together [18, 421, 534, 767]. The convex (usually right) rod is then placed. It is often less stiff and under-contoured. to push down on the apex of the spine [533].

Postoperative Pain Management

The post-operative pain management of patients undergoing PSF for AIS has recently changed significantly. Historically, epidural analgesia and patient-controlled analgesia (PCA)were frequently used for the first 24–48 hours post-operatively [31, 204, 374, 466, 484, 709, 758]. More recently, a number of other non-narcotic medications have been utilized; including: continuous intravenous dose of Precedex® (dexmedetomidine), Toradol (ketorolac trimethromene), an injectable nonsteroidal anti-inflammatory drug, is effective for the short-term management of moderate to severe postoperative pain and does not affect spinal arthrodesis [762, 826], gabapentin, clonidine, intravenous acetaminophen and long acting local anesthetics such as bupivacaine liposome [138, 495]. A number of recent studies have shown that with aggressive use of non-narcotic pain meds and standardized pathways length of stay can be reduced to 2–3 days [224, 225, 679].

Complications of Posterior Instrumentation and Fusion in AIS

The primary complications associated with posterior surgery and instrumentation include infection, implant-related problems such as discomfort or prominence, and implant failure associated with pseudarthrosis; Although most feared, neurologic deficit is also the least common. The overall incidence of reoperation within 10 years after posterior instrumentation and fusion between 5% and 10% [37, 51, 119, 231, 467, 519, 634].

Infection may be acute (<90 days) or delayed (usually greater than 12 months). Acute infection rates are generally less than 2%, with the primary risk factor being obesity (BMI >95%) [489]. Delayed infections have been reported between 1% and 10%. Although it has been hypothesized that delayed infection is related to micromotion at the hook or screw and rod interface, it is more likely that delayed infections result from low-virulence organisms that are seeded at the time of surgery and remain quiescent over an extended period [142, 158, 197, 642, 645, 823, 851]. Delayed infection seems to have decreased with the decreased use of ¼ inch stainless steel implants [519].

The incidence of pseudarthrosis is very low reported recently as 1.4% in a meta-analysis when modern double-rod systems, segmental instrumentation, and the use of allograft bone were used [316].

The most feared complication in spine deformity surgery is neurologic deficit, whose incidence has remained steady through the years and is still below 1% for AIS surgery [103, 145, 472, 639]. Intraoperative spinal cord monitoring (IONM) with the use of somatosensory evoked potentials (SSEP) and transcranial motor evoked potentials (TcMEP) provides the optimum opportunity for safe surgery. The incidence of IONM critical changes is between 2% and 5% with the TcMEP changes occurring more frequently and earlier than SSEP changes [95, 675, 792]. Reames

and coauthors reported the most recent analysis of the SRS database, which found a 0.8% incidence of neurologic deficit following surgery for AIS [639]. The likelihood of complete or partial recovery of neurologic deficits is high in all series and is dependent on an organized response to critical IONM changes to include the use of a checklist to ensure the entire operative team is working in unison [829]. The immediate responses include raising the mean arterial blood pressure above 80 mmHg [882], ensuring normal body temperature, good blood counts with the measurement of the hemoglobin, and reversing any surgical maneuvers performed. The incidence of neurologic deficit is regarded to be higher with combined anterior/posterior surgery and when osteotomies are performed and is generally thought to be of vascular origin [90, 639].

Other complications include "adding on" (progressive coronal plane imbalance - often due to continued spine growth or inappropriate fusion levels) and sagittal plane issues (often due to failure to restore appropriate thoracic kyphosis (under or over correction) or inappropriate fusion levels) [137, 163, 169, 326, 452].

Antibiotic Prophylaxis for Dental Procedures

Recent evidence suggests that the cost-effectiveness of utilizing antibiotics prior to dental procedures for those patients who have implants is questionable [510, 723, 727]. The American Academy of Orthopaedic Surgeons Appropriate Use Criteria suggests that it is "rarely appropriate" to use prophylactic antibiotics for dental procedures for a healthy patient [629, 630]. If antibiotic prophylaxis is given, the following regimen is recommended: patients who are not allergic to penicillin can be treated with cephalexin, cephradine, or amoxicillin, 2 g orally 1 hour before the dental procedure; patients who are allergic to penicillin should receive clindamycin, 600 mg orally 1 hour before the dental procedure.

Anterior Spinal Surgery

With removal of the intervertebral disc and endplate, anterior spinal surgery has the theoretical advantage of allowing greater mobilization of the spine to facilitate deformity correction, shortening the anterior column to increase kyphosis, providing an abundant area of cancellous bone for fusion and eliminating growth potential. However, with the wide adoption of pedicle screws, stiff rods and aggressive posterior mobilization techniques, there are fewer indications for anterior surgery [222, 711]. Nevertheless, it remains an important technique in the spine surgeon's armamentarium. It is most commonly used today in patients who are at risk for crankshaft and in non-idiopathic patients who have a risk for pseudoarthrosis.

Instrumented Anterior Fusion for AIS

Instrumented anterior spinal fusion for thoracic deformity was introduced by Dwyer in the 1960s. However, the correction was insufficient with the cable instrumentation system [200]. Harms began re-popularizing the idea hypothesizing that anterior correction without a posterior derotation maneuver would prevent the lumbar curve from decompensating, as had been described following selective posterior instrumentation [66, 425, 441]. However, instrumented anterior thoracic fusion, has fallen

out of favor due to a high pseudarthrosis rate' the need for a chest tube (and potentially longer length of stay) and longer surgery despite the advantage of similar correction and fewer fusion levels when compared to the posterior approach [441, 565, 571, 572, 761, 872].

Zielke introduced instrumented anterior fusion with a rod for thoraco-lumbar and lumbar curves. While this technique provided excellent coronal plane correction, there were high rates of pseudarthrosis and sagittal imbalance [537]. TSRH and Isola instrumentation extended the concepts of Zielke by using a stiff, smooth, solid rod as the longitudinal connection between vertebral screws [344, 346, 812]. The resulting stiffer, fatigue-resistant construct enhanced the maintenance of correction and the likelihood of arthrodesis without postoperative external immobilization. However, the early results of anterior single-rod treatment of thoracolumbar and lumbar curves included a high incidence of pseudarthrosis, which improved with rib strut grafts [594, 812]. The addition of anterior interbody structural support in the form of a titanium cage or femoral ring allograft significantly increased the flexion–extension stiffness of the construct when using single-rod implants [239, 465, 586]. Kaneda took the concept of instrumented anterior fusion further, introducing dual-rod constructs as a method of increasing the stiffness of the construct to maintain coronal and sagittal plane correction and preventing pseudarthrosis [132, 256, 301, 312, 313, 359, 423, 494, 696, 764, 773, 833, 836, 842]. Recent studies comparing posterior and anterior instrumented fusions for thoraco-lumbar and lumber AIS have demonstrated similar levels of fusion and correction of deformity. However, because the posterior approach is associated with a shorter operative time and hospital stay without the need for a chest tube or a general surgeon to perform the surgical approach the anterior approach is seldom used [2, 102, 255, 530, 584].

Anterior Spinal Fusion for Crankshaft

Dubousset coined the term "crankshaft" to describe progressive deformity in young children after successful posterior fusion. He observed the anterior column of the spine continued to grow and twist around the axis of the fusion mass (in a manner similar to an automobile crankshaft) [196]. Radiographic changes of more than 10° in curve size, apical vertebral rotation, and the rib–vertebral angle difference are all thought to reflect progression of the deformity secondary to the crankshaft phenomenon [287, 410, 682]. Methods to limit this phenomenon include the use of anterior fusion and greater use of, and correction with pedicle screw fixation. For patients undergoing PSF for AIS who have not reached their peak height velocity, are under 10 years of age and whose triradiate cartilage remains open, strong consideration should be given to combining anterior and posterior fusion to prevent the crankshaft phenomenon [189, 410, 684, 713]. For anterior spinal fusion, a conventional open thoracotomy approach has been compared with the newer, less invasive video-assisted thoracoscopic surgery (VATS) [309, 511, 573, 759, 831]. Some reports suggest that stiff posterior constructs, particularly when screws are used at nearly every level in the segment fused, may be strong enough to prevent the crankshaft phenomenon in immature patients, thus avoiding the need for anterior fusion [101, 424, 784].

Anterior Release and Fusion to Increase Deformity Correction

Anterior release including discectomy, and possibly rib head resection to increase flexibility and deformity correction can be performed through an open thoracotomy or through a thoracoscopic approach [357, 662]. The thoracoscopic approach has some advantage over the open thoracotomy because smaller incisions are used with less chest wall disruption and less detrimental impact on pulmonary function, especially when it is performed in the prone position with double lung ventilation using a regular single-lumen endotracheal tube [759]. The combined thoracoscopic release in the prone position and posterior instrumented fusion has resulted in overall excellent radiographic and clinical results.

The choice of spine mobilization procedure is dependent on several factors related to the spine deformity, including its severity, flexibility, amount of previous fusion, and experience of the surgeon. In general, for curves between 50° and 65°, we utilize stiff 6.0 CoCr rods on the concavity without the use of osteotomies; above 65° especially when significant hypokyphosis is present, the use of Ponte osteotomies is appropriate; when curves get above 90°, especially when the patient is skeletally immature, then anterior release/fusion is appropriate, and when previously multiply-operated patients with very severe (>100°) curves present to us then a VCR is a consideration but only performed following an exhaustive list of other options is discussed with the family.

Fusionless Surgery: Tethering

Surgical correction of scoliosis without fusion has the theoretical advantage of treating the scoliosis without creating stiffness associated with fusion of multiple motion segments. Conceptually, tethering vertebral body growth anteriorly on the concave side of the scoliosis could allow both sagittal and coronal plane correction. This use of the Hueter-Volkmann principle to produce "guided growth" has been used to address angular deformity in the legs for decades. Newton demonstrated the potential for an anterior tether to create scoliosis and kyphosis in a bovine model and studied the effect of the tether on the involved intervertebral discs [567, 568]. Braun also created deformity in a goat model using an anterior stable between vertebral bodies [88].

Crawford and Lenke reported a single case of scoliosis correction using anterior vertebral body screws and a flexible tether in 2010 [157] and a series of 32 patients with 1 year follow up after was published in 2015 [673]. In 2018 Newton reported 2–4 year follow up on 17 patients– 41% of whom required a revision. In 2019 the FDA approved Zimmer-Biomet's product: The Tether™ – Vertebral Body Tethering System that consists of an anterior vertebral body screw and flexible cable.

Currently there is a great deal of enthusiasm – from surgeons, patients and parents – regarding "motion preserving" or "fusion-less" scoliosis surgery; with some advocating this treatment in patients who would be indicated for brace treatment. It is important to remember that this is an evolving technology and is an invasive surgical procedure on the spine that carries many of the inherent risks associated with spinal fusion. Patients should be carefully selected and appropriately counseled. Newton currently considers growth modulation with tethering in patients with primary thoracic curves of 45–65° and Risser 0 or 1, Sanders 3–4. He wisely notes:

"Patients often over-estimate the loss of mobility and function associated with a thoracic spinal fusion and under-estimate the challenges associated with "minimally invasive" spinal surgery" [566]. Further investigations will determine if this is indeed an improvement over the well-defined, predictable and quite good results achieved with modern posterior spinal fusion.

Juvenile Idiopathic Scoliosis

Children between the ages of 3 and 9 with scoliosis without an underlying cause are diagnosed with juvenile idiopathic scoliosis. Juvenile idiopathic scoliosis (JIS) represents 10–15% of scoliosis in children [339, 656]. Approximately 20–25% of children less than 10 years of age with a 20° scoliosis will be found to have neural axis abnormality (and subsequently not be deemed idiopathic) [210, 277, 436, 693, 739, 899]. As a result, some authors recommend MRI during the initial evaluation of patients presumed to have JIS. If scoliosis surgery is planned, it is imperative that preoperative MRI evaluation be undertaken. Neurologic deficits following spinal surgery have been reported in patients with neural axis abnormalities that were not recognized preoperatively [579].

Treatment options for children with JIS are the same as those for AIS. Because they have more growth, non-operative treatments are less likely to be successful in patients with juvenile idiopathic scoliosis. Some patients with JIS may be treated with casting as outlined in the section "Early-Onset Scoliosis."

Infantile Idiopathic Scoliosis

Infantile Idiopathic Scoliosis (IIS) is defined as scoliosis without a known etiology in the first 3 years of life. It represents only 1% of idiopathic scoliosis and unlike AIS and JIS, boys are slightly more effected than girls, left thoracic curves are as prevalent as right curves and curves will spontaneously resolve up to 80% of the time.

Mehta described radiographic parameters (measurement of the rib-vertebral angle difference (RVAD) and the phase 1 or 2 rib head at the apical vertebra) associated with progressive IIS. 80% of patients with a phase 1 rib (not covering the apical vertebral edge), and a RVAD $\geq 20°$ will have progression of their curve. If the rib head has reached phase 2, overlapping the vertebral edge, the curve will progress regardless of the RVAD (Fig. 28.9) [512].

These patients are at risk to develop thoracic insufficiency syndrome, their treatment can be exceptionally challenging and is discussed in the section "Early-Onset Scoliosis".

Congenital Spinal Deformities

Congenital deformities of the spine are caused by anomalies of the vertebrae. These anomalies may be subtle and found incidentally on radiographs obtained for some other reason, or they may be complex and lead to severe spinal deformity with accompanying neurologic deficits. Congenital scoliosis, congenital kyphosis, and a combination of the two are the deformities encountered. They are much less common than idiopathic scoliosis.

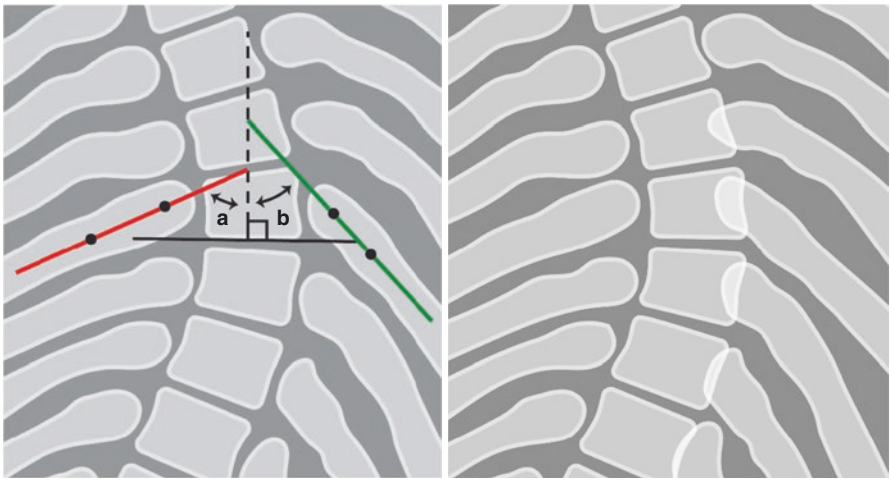


Fig. 28.9 With a Phase 1 rib, the rib vertebral angle difference (RVAD) should be calculated. If the RVAD is greater than 25°, the curve is likely to progress. In a phase 2 rib the rib on the convex side is medial to the edge of the vertebral body. These curves are progressive

Etiology

The cause of congenital vertebral anomalies remains unknown. During embryologic development, these abnormalities develop in the spine between the fifth and eighth weeks of gestation. Investigation of genetic causes has provided modest insight. A positive family history can be found in approximately 1% of patients with congenital spinal deformities [854]. Idiopathic scoliosis has been reported in 17% of families of children with congenital scoliosis [626]. An isolated anomaly, such as a hemivertebra, usually occurs as a sporadic event and carries no risk for a similar abnormality in other offspring [877]. Studies of identical twins, only one of whom was affected, showed no genetic pattern [295, 613, 619], but other reports of twins with similar congenital deformities suggested the possibility of genetic causes [227, 539, 755]. Scientists have identified the human gene *HuP48*, a member of the *Pax* family of developmental control genes, as having a role in establishing the segmented pattern of the vertebral column [730]. As yet, no mutations in this gene have been found in those with vertebral segmentation defects. A chromosomal aberration, deletion of 17p11.2, has been reported in congenital scoliosis but needs further verification [329]. Analysis of the candidate gene *DLL3* has raised the possibility of its involvement in congenital scoliosis [208, 482]. However, no definitive cause of anomalous vertebral development has yet been established.

Associated Abnormalities

A nonspecific insult during embryonic may result in congenital malformation of any organ undergoing concurrent epigenesis [266]. The most common associated

finding is intraspinal anomaly, a general category that includes s tethered cord, diastematomyelia, syringomyelia and lipoma [54, 58, 85, 503, 506, 621, 622, 765]. The reported incidence of associated neural axis abnormalities ranges from 21% to 37%. In addition to neural axis abnormalities, approximately 60% of patients have associated abnormalities affecting other systems [56]. Approximately 20–30% of patients have an anomaly of the genitourinary system [54, 154, 201, 275, 473], and cardiac anomalies are seen in approximately 12–26% of patients [54, 56, 70]. The VACTERL association includes vertebral defects, anal atresia, cardiac defects, tracheoesophageal fistula, renal anomalies, and limb abnormalities. Other abnormalities reported with congenital vertebral abnormalities include cranial nerve palsy, clubfoot, dislocated hip and Sprengel deformity.

Congenital Scoliosis

Even though the vertebral anomalies are present at birth, congenital scoliosis can be diagnosed at any age. The vertebral anomalies are often noted as incidental findings on chest x-rays or KUBs ordered for evaluation of common medical illnesses. The variety of vertebral anomalies that can exist leads to an unpredictable natural history. The deformity may remain mild, or it may progress dramatically over time and ultimately result in severe spinal deformity and pulmonary compromise [160]. Understanding which vertebral anomalies place the spine at greatest risk for progressive deformity allows the treating physician to intervene at the appropriate time.

Classification

Congenital vertebral anomalies are either defects of vertebral formation and defects of vertebral segmentation. Hemivertebrae and wedged vertebrae are examples of defects of formation. Defects of segmentation include block vertebrae and unilateral bars. Many patients have a combination of deformities in which one type predominates. In 2009 a three-dimensional CT classification system for congenital spinal deformities was introduced [368, 557]. Four types of congenital vertebral abnormalities were introduced: type 1, solitary simple; type 2, multiple simple; type 3, complex; and type 4, segmentation failure.

Defects of Formation

Defects of formation may be partial or complete. Partial unilateral failure of formation produces a wedged or trapezoid-shaped vertebra that contains two pedicles, although one of them may be hypoplastic. The associated scoliosis worsens slowly and may not require treatment.

True hemivertebrae are caused by complete failure of formation on one side and result in laterally based wedges consisting of half the vertebral body, a single pedicle, and a hemilamina. Occasionally, the lamina associated with the hemivertebra may be incorporated into that of the adjacent normal-appearing vertebra. When this occurs, differentiating between the anterior vertebral abnormality and the corresponding posterior abnormality becomes difficult. Hemivertebrae in the thoracic

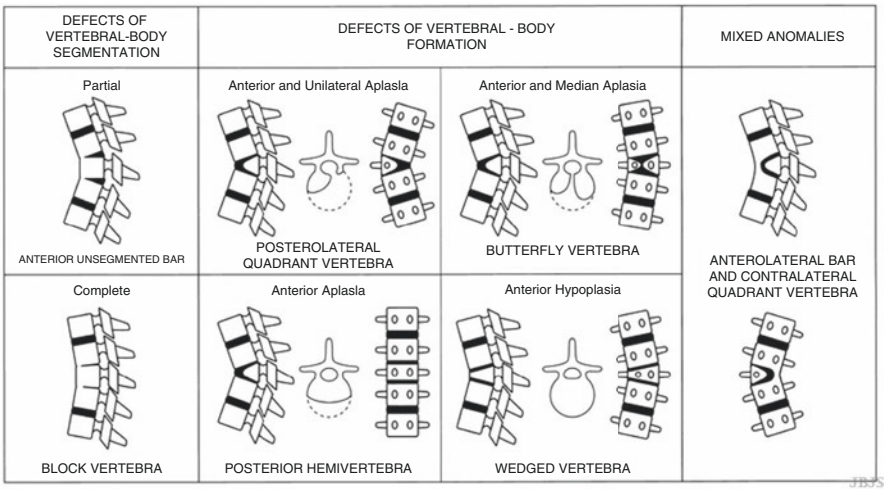


Fig. 28.10 Cong scoliosis classification. (From: McMaster and Singh [508])

spine are usually accompanied by an extra rib. Hemivertebrae may be fully segmented (most common), semisegmented, nonsegmented, or incarcerated (least common) (Fig. 28.10).

Fully segmented hemivertebra has the highest likelihood of progressive deformity because it is separated from the adjacent vertebrae by intact end-plates and intervertebral disks. The hemivertebra is nearly always located at the apex of the scoliosis. Lower thoracic and thoracolumbar curves tend to worsen more rapidly than do curves at other levels. When two or more hemivertebrae are present on the same side of the spine, the deformity progresses at a faster rate. Conversely, the spinal deformity may be balanced and nonprogressive if two hemivertebrae are situated opposite each other. A fully segmented hemivertebra at the lumbosacral junction creates significant obliquity between the spine and pelvis and is usually accompanied by a long compensatory scoliosis in the lumbar or thoracolumbar region. This readily apparent deformity is best treated surgically (usually by hemivertebrectomy) at an early age, before the compensatory curve becomes fixed.

A semisegmented hemivertebra is separated from one adjacent vertebra (superior or inferior) by a normal vertebral growth plate and disk but is fused to the other adjacent vertebra. Although growth of the spine should remain balanced, the hemivertebra can induce a slowly progressive scoliosis. Treatment is necessary only if the deformity is progressive.

A nonsegmented hemivertebra is fused to both adjacent vertebrae (above and below) and therefore has no vertebral end-plates or adjacent disks. In the absence of any asymmetric growth, a nonsegmented hemivertebra does not cause progressive spinal deformity. An incarcerated hemivertebra is more ovoid and smaller than a fully segmented (nonincarcerated) hemivertebra. The vertebrae above and below compensate for this hemivertebra, and as a result, minimal if any scoliosis is present.

Defects of Segmentation

Defects of segmentation result in a bony bar or bridge between two or more vertebrae, either unilaterally or involving the entire segment. Circumferential, symmetric failure of segmentation leads to a block vertebra. This does not cause any angular or rotational spinal deformity but does lead to some loss of longitudinal growth.

Unilateral failure of segmentation of two or more vertebrae (unilateral bar) occurs when a bar of bone fuses the disk spaces, pedicles, and facet joints on one side of the spine, thus precluding growth and creating the concavity of a curve. If/when growth proceeds opposite the bar (on the convexity) the curve progresses. Rib fusions or other rib abnormalities on the concavity of the scoliosis are often seen (22% frequency) adjacent to the bony bar bridging the vertebrae [507].

Some patients with unilateral failure of segmentation have one or more hemivertebrae located on the opposite (convex) side of the curve. This combination carries the worst prognosis because it produces the most severe and rapidly progressive deformity. Without treatment, patients with thoracolumbar, midthoracic, or lumbar curves may develop significant deformity at an early age because of a combination of shoulder imbalance, severe distortion of the rib cage, decompensation of the trunk, and pelvic obliquity that produces an apparent leg length discrepancy.

In addition to deformities involving the thoracic and lumbar spine, congenital scoliosis affecting the cervical and cervicothoracic spine can lead to significant deformities of the neck and an abnormal head position [733]. The neck deformities can result in persistent tilt of the head (apparent torticollis) because the relatively few normal vertebrae above the area of the segmentation defects cannot provide sufficient compensation for balance. Nearly 50% of those with congenital cervical or cervicothoracic scoliosis have associated Klippel-Feil abnormalities [733, 789, 797].

Natural History

The rate of curve progression and the final severity of congenital scoliosis are related to two factors: the type of vertebral anomalies present and the patient's remaining growth at the time of diagnosis. The two periods of accelerated growth during which congenital scoliosis worsens most rapidly are the first 2 years of life and the adolescent growth spurt.

Curve progression is certain in patients with a unilateral unsegmented bar and a contralateral hemivertebra (one or more) [80, 505, 506, 852, 855]. Thoracolumbar curves of this type have the worst prognosis and may progress up to 7° per year before the age of 10 years; this increases to 14° per year during the adolescent growth spurt. Curve progression cis also seen with (in decreasing order) isolated unilateral unsegmented bars, multiple fully segmented hemivertebrae, a single fully segmented hemivertebra.

The progression of scoliosis caused by mixed abnormalities is extremely difficult to predict, numerous visits may be required before the nature of the curve becomes evident. The most worrisome potential outcome is the development of thoracic insufficiency syndrome, in which growth of the thorax is so retarded that

normal lung growth and respiration cannot be supported [116]. This concept is explained in more detail in the section "Early-Onset Scoliosis".

Compensatory curves develop more commonly in patients with congenital scoliosis with an apex in the mid thoracic spine. As the congenital curve progresses, this secondary curve may also increase, become inflexible, and require treatment [505]. Patients with large congenital lumbar or thoracolumbar curves may be unable to develop compensatory curves large enough to maintain a balanced trunk. In this instance, pelvic obliquity and apparent lower limb length inequality are compensatory mechanisms used to keep the trunk vertical.

Radiographic Findings

Radiographic details of the vertebral abnormalities are best seen on films obtained before the development of significant deformity—often during infancy—on a radiograph taken while the child is supine. As the child grows and the congenital scoliosis progressively worsens, the bony detail becomes less clear. At the initial evaluation, coned-down radiographs of the affected area provide the most information about the vertebral anomalies. Associated abnormalities that may also be noted on plain radiographs include diastematomyelia (midline bone spur), spina bifida occulta, and congenital rib fusions on the concavity of the curve [258].

Although early supine radiographs reveal bony detail, they cannot be used to assess curve progression. The initial upright radiograph must serve as the baseline study against which further curve progression is measured. The variability in measuring angles in congenital scoliosis is reportedly larger than that in idiopathic scoliosis because of skeletal immaturity, incomplete ossification, and anomalous development of the end vertebrae [212, 451]. Concerted effort should be made to measure the curves with similar end points to detect subtle yet steady progression of the curvature and assess secondary or compensatory curves. The current radiograph should be carefully compared to the *earliest* upright radiograph to ascertain whether progression has occurred. (It is not uncommon for radiographs taken 4–6 months previously to reveal only slight progression when compared with current radiographs. If comparisons are made with radiographs obtained several years earlier, the changes become more evident.)

In severe congenital scoliosis, plain radiographs may not provide sufficient detail of the vertebral abnormalities. Should surgical intervention be necessary, CT with three-dimensional reconstruction is helpful for preoperative planning, particularly in visualizing posterior vertebral anomalies associated with hemivertebrae [104, 298, 368, 515, 557, 569, 667, 786].

MRI of the spine should be performed in all patients with congenital scoliosis who are undergoing surgical intervention and should also be strongly considered during the initial evaluation because 37–55% of patients may have an intraspinal abnormality [54, 506, 802]. In the first year of life ultrasound of the spine may reveal tethered cord, lipomas and other intradural abnormalities.

Nonoperative Treatment

Use of a brace to control curve progression secondary to congenital scoliosis is universally unsuccessful and should not be attempted. However, serial casting and bracing can be considered as a means of temporarily controlling a compensatory curve below (or less commonly above) the congenital component [347].

Operative Treatment

In skeletally immature patients, once a congenital curve has been documented to progress, surgical management is indicated. If patients are less than 10 years of age, consideration must be given to pulmonary development and the potential for thoracic insufficiency syndrome. This is discussed in the section "Early-Onset Scoliosis" at the end of this chapter.

For older children with congenital scoliosis, even with relatively small curves (<40°) once progression has been confirmed, surgical intervention should be undertaken if a significant amount of growth remains. Preoperative MRI of the neural canal is essential to rule out diastematomyelia, tethered cord, syrinx, and other abnormalities. If any of these lesions are identified, they should be addressed prior to spinal fusion. Children and adolescents who have undergone spinal fusion should be monitored to maturity because a progressive deformity can develop above or below the fused sites. Additional surgery may be required in these individuals.

Various operative approaches can be used; the choice depends on the maturity of the patient, the location of the deformity, and the type of congenital deformity. Historically, many congenital curves were treated with in-situ fusion without instrumentation. Today, with smaller implants, almost all patients are treated instrumented fusion.

Anterior and Posterior Fusion

This approach is used in immature individuals in whom continued anterior growth on the convexity would lead to development of the crankshaft phenomenon [196, 302, 788]. Children most in need of this approach are those who have unilateral unsegmented bars with (or sometimes without) contralateral hemivertebrae. In a young child, fusion should extend to one level above and one level below the anomalous vertebrae; this may prevent "adding on" of the curve in subsequent years.

Posterior Spinal Fusion

Posterior spinal fusion is indicated in older children with progressive congenital scoliosis with less growth potential and therefor who are less likely to develop the crankshaft phenomenon or in younger children who do not have normal anterior growth potential. Crankshaft progression does not develop in many young children with congenital scoliosis because the anterior growth plates are abnormal [372, 460, 858, 863, 865]. However, these patients are not always easily identified preoperatively, so the decision whether to include anterior fusion is difficult. The use of reduced-size spinal instrumentation in young patients is safe and efficacious [299]. Any deformity correction is usually obtained through the flexible, normal vertebral segments.

Hemivertebra Excision

This procedure is most commonly performed in patients with a single hemivertebrae in the lumbar or lower thoracic spine. Hemivertebra excision carries a risk for temporary and occasionally permanent neurologic injury [311]. Excision distal to L2 is safer because nerve roots are more tolerant of manipulation than the spinal cord. The major advantage of resection of the hemivertebra is that it allows maximal correction of the deformity and realignment of the spine [55, 83, 111, 127, 173, 311, 314, 338, 386, 558, 660, 661, 712].

Excision of a hemivertebra can be accomplished from posterior only or combined anterior and posterior approach [77, 300, 391, 879]. Historically, in the combined technique, the anterior approach allows removal of the body of the hemivertebra and its adjacent disks back to the spinal canal, along with removal of the anterior half of the pedicle. The patient is next repositioned and the posterior elements are excised through a secondary midline approach. Correction is then achieved internally with posterior compression instrumentation on the convexity. More recently, a number of studies have reported successful hemivertebra excision via a posterior approach alone [338, 558, 660, 661, 712, 886]. This method, combined with transpedicular instrumentation, is safe and provides excellent correction in both the frontal and sagittal planes.

Vertebral Column Resection

We perform VCR in individuals with very severe deformity —angular scoliosis or kyphosis— usually neglected or previously treated. This procedure should be performed only by skilled and experienced surgeons who have worked their way up the learning curve by performing less arduous spine mobilization procedures [40, 433, 434, 768]. A multicenter study of 147 pediatric VCR procedures performed by senior surgeons reported complications in 59% of patients, including 27% who had an intraoperative neurologic event without paraplegia [432].

Congenital Kyphosis

Congenital kyphosis represents an abrupt posterior angulation of the spine because of a localized congenital malformation of one or more vertebrae [195, 276, 852, 859]. Although this condition is less common than congenital scoliosis, paraplegia is a far greater risk in those with congenital kyphosis.

Classification

Congenital kyphosis is caused by defects of vertebral body formation (type I), defects of vertebral body segmentation (type II), or a combination of the two (type III) (Fig. 28.10).

In contrast to congenital scoliosis, failure of formation is the most common type of congenital kyphosis, and it tends to produce more severe deformities than those seen with kyphosis resulting from failure of segmentation. These vertebral abnormalities may also lead to frontal plane deformity and result in kyphoscoliosis.

Defects of Formation (Type I)

In kyphosis caused by defects in vertebral body formation, part or all of the vertebral body is deficient. Several contiguous levels may be affected, which produces greater deformity. In general, the posterior elements (spinous processes, pedicles, transverse processes) are present. Growth continues normally in the posterior portion of the spine, but not anteriorly. As a result, relentless progression of the deformity usually occurs.

Defects of formation place the patient at a much greater risk for the development of paraplegia than do defects of segmentation. The kyphotic junction may be unstable, particularly when the apex is between T4 and T9. Paraplegia can occur at any age but is most common during the adolescent growth spurt.

Defects of Segmentation (Type II)

In kyphosis caused by failure of segmentation, the anterior portions of two or more adjacent vertebral bodies are fused. This deformity tends to be less progressive, produces less deformity, and is associated with a much lower risk for paraplegia than is kyphosis caused by defects in formation [496]. The area most commonly affected is the lower thoracic or thoracolumbar spine.

Natural History

The apical area of the kyphosis can occur at any level but is most commonly located between the tenth thoracic and first lumbar levels [508]. There appears to be no relationship between the severity of the kyphosis and its location in the spine. Progression of these deformities is most rapid during the adolescent growth spurt.

Congenital kyphosis from either failure of formation (type I) or mixed anomalies (type III) is much more likely to be progressive [488, 508, 614, 864]. Deformities caused by two adjacent type I vertebral anomalies progress more rapidly and with more severity than do deformities caused by a similar single anomaly. Kyphosis from failure of segmentation (type II) is much less progressive, produces less severe deformity, and has a very low likelihood of resulting in paraplegia.

Clinical Features

Although congenital kyphosis has been diagnosed prenatally, it may not be clinically evident in a newborn or infant [737]. Suspicion may first be raised after a chest radiograph is obtained for evaluation of an unrelated event, such as a respiratory infection. As the child begins standing and walking, a localized prominence may become noticeable or palpable. The child is usually asymptomatic and has no spinal tenderness. In adolescents, the predominant clinical complaint tends to be lower back discomfort caused by secondary lumbar hyperlordosis. Mild scoliosis may accompany the kyphosis.

On occasion, myelopathy or paraplegia secondary to spinal cord compression may develop in a child with congenital kyphosis [375]. Reports of mild trauma producing a sudden onset of paraplegia in children who have unrecognized, acute type I kyphosis highlight the delicate underlying neurologic status in this condition. When congenital kyphosis caused by a defect in vertebral formation is diagnosed, a

meticulous neurologic examination should be performed to identify any subtle abnormalities.

Radiographic Findings

Congenital kyphosis is best visualized on a lateral radiograph of the spine. It may not be evident on the frontal view. Once identified, a coned-down lateral view of the specific area provides greater bony detail. Three-dimensional CT imaging of the spine with reconstructed images is very useful in the evaluation of vertebral anomalies, especially in older children [569]. MRI provides the clearest picture of the spinal cord and vertebral bodies in very young children. It should be ordered immediately for those whose kyphosis is due to failure of formation. Spinal cord compression may be evident on MRI before any clinical neurologic deficits become apparent.

Treatment

Nonoperative treatment has no beneficial effect on congenital kyphosis, and use of an orthosis is inappropriate. Once type I or type III kyphosis is recognized, plans for surgical intervention should be considered. For adolescents with mild type II kyphosis, close monitoring for progression is reasonable.

Type I Kyphosis

Historically, if the kyphosis is recognized in a child younger than 5 years and is less than 45–50°, simple posterior fusion without instrumentation may be considered. A hyperextension cast is used postoperatively for 4–6 months, followed by a TLSO for another 6 months. Successful outcomes with posterior fusion have been reported [508, 509, 861]. This approach allows some growth to occur anteriorly in the abnormal region of the spine, which may result in progressive improvement in the localized kyphosis over time. Reexploration and augmentation of the graft at 6 months have been advocated. An alternative approach for young children is to combine anterior fusion using a rib strut with posterior fusion during the same surgical intervention. This approach produces some immediate improvement in sagittal plane alignment and increases the likelihood of a solid fusion, but it eliminates any further correction that might occur as a result of anterior growth.

In an older child, two approaches can be used. The first and older approach consists of the combination of anterior and posterior arthrodesis [866]. The anterior arthrodesis is performed first. Following excision of the gristlelike soft tissue anteriorly, some distraction is attempted. Any distraction that is achieved can then be maintained with rib strut grafts. Vascularized rib struts heal more rapidly and should always be used in those who have had previously unsuccessful attempts at anterior fusion; they may also be considered for the initial fusion procedure [82, 84, 699].

The second and more common current approach, involves posterior surgery only. Through VCR, the anterior deformity can be resected, decompression achieved, and an interbody spacer placed [40, 432–434, 597, 756, 768]. Posterior pedicle screw fixation is required to maintain spinal stability during resection of the anterior elements. This procedure should be performed only by skilled and experienced surgeons who have worked their way up the learning curve.

If a neurologic deficit is present at the time that the congenital kyphosis is recognized, treatment should be undertaken immediately. If the deficit is minimal (increased reflexes, Babinski sign, or both, but no loss of motor, bowel, or bladder function), formal anterior decompression of the spinal cord is not necessary. Following a solid anterior and posterior arthrodesis, or following a VCR, these subtle neurologic deficits may resolve. On occasion, patients have mild paraparesis of recent onset. In these individuals the apical flexibility of the kyphotic deformity should be assessed with a hyperextension radiograph. If the apex is flexible, some improvement in the paraparesis may be achieved by resting the recently compromised spinal cord with a halo vest, cast, or minimal halo traction [195]. *Halo traction should not be considered in those with a rigid, inflexible kyphotic apex because of the risk for progressive neurologic deterioration.* Very close monitoring is needed. If recovery occurs, spinal fusion can be performed without the need for decompression [383]. If the deficits do not resolve, arthrodesis must be combined with anterior decompression of the spinal cord. Unless the child is very small, these procedures can be accomplished during the same operative episode. The decompression must be performed anterior to the compressed cord by removing the posterior aspect of the vertebral body. Posterior laminectomy does not relieve the spinal cord compression. VCR through a costotransversectomy is an effective approach for these complex kyphotic deformities of the thoracic spine. It should be undertaken only by those experienced with this technique.

Type II Kyphosis

Defects of segmentation are best treated at a young age, before significant deformity has developed. Historically, posterior spinal fusion followed by cast immobilization was performed across the unsegmented levels plus one level farther both cephalad and caudad. Correction of the kyphosis should not be expected, although mild improvement from the cephalic and caudal extensions is possible [380]. Today, posterior osteotomies and instrumentation are commonly performed to achieve some correction and avoid immobilization .

In an older child with severe kyphosis, some correction of the deformity may be achieved through osteotomy of the unsegmented anterior region [731]. When combined with posterior compression instrumentation, this approach may result in some improvement in the sagittal plane.

Type III Kyphosis

Mixed anomalies are least common but usually produce a kyphoscoliotic deformity. Because of their association with failure of segmentation, type III anomalies generally require posterior arthrodesis only.

Segmental Spinal Dysgenesis, Congenital Vertebral Displacement, and Congenital Dislocation of the Spine

Segmental spinal dysgenesis, congenital vertebral displacement, and congenital dislocation of the spine may be variations of the same deformity, although this is not universally accepted [179]. They all create severe localized kyphosis of the spine

and lead to a neurologic deficit in 50–60% of patients [213, 232, 322, 706, 897]. These are the most severe (and rare) congenital deformities and may require surgical treatment in the first 2 years of life.

Segmental spinal dysgenesis is characterized by a focal spinal deformity, usually located at the thoracolumbar junction or in the upper lumbar spine [213, 232, 235, 322, 801, 894]. The deformity frequently includes severe kyphosis; anterior, posterior, or lateral subluxation of the spine; scoliosis in association with a severely stenotic spinal canal; and absent nerve roots. All these patients have localized stenosis of the spinal canal at the level of involvement, and the osseous canal has an hourglass shape. No pedicles, spinous processes, or transverse processes are seen at the level of involvement. Commonly, an offset in the sagittal plane is present between the cephalic and caudal segments of the spine at the level of dysgenesis. Decompression of the stenotic canal results in some improvement in neurologic function in 20% of patients. Early anterior and posterior arthrodesis in patients with segmental spinal dysgenesis is indicated because progressive kyphosis inevitably develops and often results in neurologic deficits.

Congenital vertebral displacement occurs when the spinal column is displaced at a single vertebral level and results in abrupt displacement of the neural canal [706, 807, 822, 896]. The displacement can occur in the presence of a posteriorly located hemivertebra in which the pedicles, transverse processes, and spinous processes may be present. As with segmental spinal dysgenesis, the potential for severe neurologic deficits is high. Combined anterior and posterior arthrodesis of the spine is needed in an effort to prevent the development of such deficits. For those with neurologic deficits of recent onset or progressive neurologic deficits, decompression of the spinal cord is indicated.

The congenitally dislocated spine was first described in 1973 by Dubousset [897]. It, too, is associated with spinal kyphosis and a high likelihood of neurologic deterioration. The posterior elements are abnormal in all patients with congenital dislocation of the spine. The various stages of posterior dysraphism range from agenesis of the laminae with pathologic changes in the articular facets to total absence of the posterior elements and the spinal cord under otherwise normal skin. Anterior and posterior spinal fusion is indicated because posterior fusion alone is insufficient to achieve solid fusion with this type of congenital instability. Function must be favored over cosmetic appearance. Neurosurgical decompression should be used only for a proven recent and progressive neurologic deficit.

For these three entities, all of which involve a severe form of localized kyphosis, early recognition is imperative, and the appropriate operative intervention should be undertaken. Prenatal diagnosis is possible and can be useful for parental counseling and obstetric management [238, 670].

Scoliosis in Cerebral Palsy

Scoliosis affects between 25% and 68% of patients with cerebral palsy (CP) [481, 499, 678]. The incidence is highest in patients who are nonambulatory and have

total body involvement (i.e., GMFCS level 4 and 5). Series that include patients who are ambulatory and have milder neurologic involvement yield a lower incidence, whereas those that study institutionalized patients show a higher frequency. Up to 64% of institutionalized adults with CP have scoliosis [481, 793].

The typical curve pattern in scoliosis secondary to CP is a long sweeping curve that extends to the pelvis, with the apex of the curve at the thoracolumbar junction. Pelvic obliquity and rotation accompanies the coronal-plane curvature (Fig. 28.11) [32, 777].

Scoliosis can create difficulty with seating and pelvic obliquity, which can produce uneven pressure on the ischial tuberosities and eventually pressure sores [734]. Seating imbalance forces the child to lean on the upper extremities and become a hands-dependent or a propped sitter.

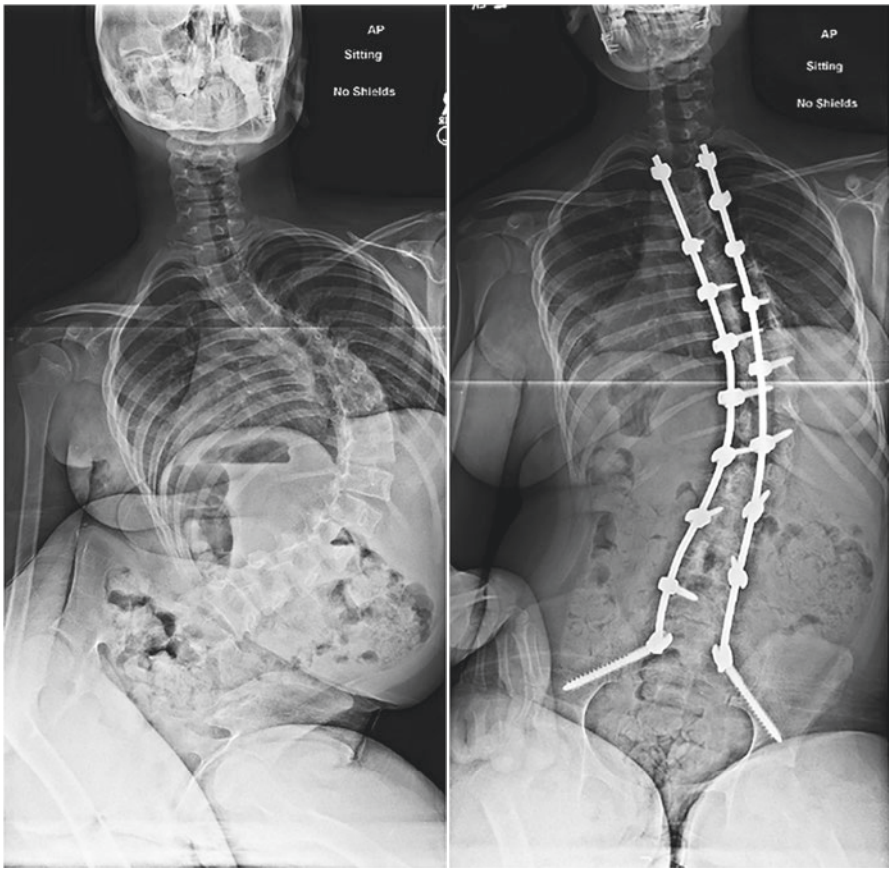


Fig. 28.11 Pre- and post-operative AP radiographs of a non-ambulatory patient with cerebral palsy treated with an instrumented posterior fusion to the pelvis

Nonoperative Treatment

Curve progression is not controlled by bracing, a fact that has been proved in many studies. Bracing had no impact on scoliosis curve, shape, or rate of progression in patients with spastic quadriplegia who were observed by Miller and colleagues until fusion [520]. Letts and colleagues found that seating was made somewhat easier when a soft orthosis was prescribed. The brace was used only to allow comfortable seating, not to treat the curve [435]. Wheelchair adaptations can usually be made to position a child in a functional position and avoid pressure sores, but do not effect progression of the curvature.

Surgical Treatment

Surgical management of scoliosis associated with CP carries sufficient risks (5 year complication rates as high as 45% and mortality as high as 5%) [531] and unclear benefits. Thus it is wise to approach each patient with a "shared decision making" model where the risks and benefits of both surgical treatment and observation are thoroughly reviewed with the family. It has been shown that the rate of progression of scoliosis is related to the patient's GMFCS level, with the most involved nonambulatory patients worsening at a faster pace than an ambulatory group [249]. Majd and colleagues monitored all adult patients with CP in a nursing home and documented whether they had scoliosis and whether their curves progressed [483]. They found that 18% of patients had significant progression of their curves. The larger curves tended to progress in adulthood at a rate of 4.4° per year. Three patients had decubitus ulcers, and their average curves were greater than 100°, with more than 45° of pelvic obliquity [483]. Thometz and Simon found similar results, but the rate of progression of curves greater than 50° at skeletal maturity averaged only 1.4° per year. They also found that thoracolumbar and lumbar curves tended to progress more than thoracic curves did [793]. If a curve is greater than 40° by 15 years of age, it is likely to progress [666].

These studies looked at whether curves progressed after skeletal maturity, but they did not address whether patients with larger curves were less healthy or more difficult to nurse. Kalen and associates compared 14 residents of a nursing home who had scoliosis of between 51° and 105° with 42 residents who had either no scoliosis or small curves. They found that patients with larger curves had more orthopaedic deformities such as hip dislocations and that they needed modified wheelchairs. No difference was found in the incidence of decubitus ulcers, functional level or loss of function, or oxygen saturation, however [354]. They concluded that problems with functional loss and decubitus ulcers were seen in equal proportions in both groups, so the surgical indications for spinal fusion in those with CP were not clear.

Cassidy and associates analyzed the health and nursing care of a group of institutionalized patients with CP who had undergone spinal fusion surgery and a similar group of patients with scoliosis of greater than 50° who had not [122]. They found no significant difference in pain, pulmonary status, decubitus ulcers, function, or time required for daily care. The nurses caring for these patients, however, believed that those who had undergone spinal fusion were more comfortable. Based on this

study and the study by Kalen and colleagues [354], the indications for surgery in patients who are institutionalized and severely mentally retarded remain clouded.

Preoperative Evaluation

Once the decision to operate has been made, a thorough medical evaluation is necessary [867]. Malnutrition is frequently a problem in these patients and, when present, predisposes to infection and delayed wound healing. Laboratory studies, including measurement of serum protein and albumin and a total lymphocyte count, are useful in assessing the nutritional status of the child. A serum albumin level of 35 g/L and a total lymphocyte count of 1500 cells/mm^3 have been established as levels below which complications occur more frequently [342]. Gastrostomy tube feedings may be necessary preoperatively to lessen the risk for complications. Aspiration has been documented in 69% of patients with total body involvement of CP [192]. In patients with gastroesophageal reflux, pancreatitis and feeding difficulties are also more likely to develop postoperatively [79]. Preoperative swallowing studies should be performed in patients suspected of aspirating.

Certain seizure medications that are frequently used in patients with CP can increase blood loss by interfering with coagulation. Patients treated with divalproex (Depakote) or valproate (Depakene) will have normal routine coagulation profiles—that is, prothrombin time and partial thromboplastin time—but prolonged bleeding times. Platelet counts may also be decreased by these medications [867]. Use of antifibrinolytic medications such as tranexamic acid at the start of surgery has been shown to decrease blood loss but not decrease transfusion requirements [174]. ***Preparations for large intraoperative blood loss must be made*** [335].

The use of indwelling intrathecal baclofen pumps has become more prevalent in patients with CP and scoliosis. It should be expected that the catheter will be in the way of the surgery, so a repair kit should be made available in these cases. We find it easiest to clamp and transect the tubing during exposure of the spine and reanastomose it at completion of the procedure. In patients in whom the catheter requires reinsertion, spinal headache can develop postoperatively [694].

Intra-operative Neuromonitoring

Intraoperative spinal cord monitoring should be performed. Transcranial motor evoked potentials have been used safely in the cerebral palsy population although monitoring may be more difficult to obtain and interpret than in idiopathic patients [181, 289, 609]. Concerns regarding worsening of seizure disorders in children with CP who have transcranial motor evoked potential monitoring appear to be unfounded [669].

Surgical Technique

Most commonly, instrumented fusion from T2 to the pelvis is performed (Fig. 28.11). Historically, this was done with segmental Luque wire fixation of the thoracic and lumbar spine and fixation to the pelvis with one of several techniques. Today fusion is done with segmental pedicle screws and pelvic fixation is most commonly achieved with SAI screws, although iliac screws may be necessary in patients with

severe pelvic obliquity/deformity [3, 5, 243, 310, 337, 531, 806, 887]. Intraoperative traction is helpful in this patient population. If pedicle screws cannot be placed, sub-laminar polyester bands are now available. Theoretical advantage of these bands over wires include decrease likelihood of penetration into the spinal canal and better distribution of force on the lamina in osteopenic patients [20, 117]. Patients with extreme hyperlordosis can be the most difficult to treat as exposure is associated with increased time and blood loss and instrumentation is difficult.

Although, improved posterior fixation and spine mobilization techniques have made anterior release and fusion less common it is still occasionally performed. The indications are not clearly defined and are very surgeon dependent [293, 334]. Anterior release decreases the rate of pseudarthrosis and crankshaft and increases the deformity correction. The addition of anterior fusion reduced the pseudarthrosis rate from 22% to 5.4% in a series published in 1983 by Lonstein and Akbarnia [455]. In younger patients with open triradiate cartilage, the crankshaft phenomenon may develop with isolated posterior fusion in the presence of postoperative anterior vertebral growth. These patients may benefit from anterior fusion combined with posterior instrumentation and fusion. Severe, stiff curves may require anterior release and fusion to improve the surgical correction of the deformity [180]. Boachie-Adjei and colleagues recommended preliminary anterior release and fusion for curves greater than 90° and for curves in which a stretch supine radiograph showed lack of correction of pelvic obliquity [73]. The literature is inconclusive as to the benefits and risk of staged or same day anterior and posterior fusion [334, 804].

Complications

The complication rates of surgical treatment of scoliosis associated with CP are reported to be as high as 45–62% with mortality of 5% [458, 531, 830]. Pulmonary complications are frequent in the early post-operative period and are associated with increased intraoperative blood loss and lead to an increase in ICU stays [674]. Pancreatitis has also been described in the cerebral palsy population, particularly in those children who have gastrostomy tubes [4]. A multicenter study from 2016 subclassified patients who were GMFCS5 by whether they had a tracheostomy, gastrostomy, a seizure disorder, and were nonverbal. 49 of 100 patients who had 3 or more of these risk factors suffered complications including death in five children [336].

Wound infection can occur in up to 20% of patients with CP having scoliosis surgery [455, 538, 635, 744, 778]. Wound infection is most likely in malnourished patients [193, 389], those that require gastrostomy tubes [575] and in those with greater cognitive impairment [742]. Infection with gram-negative organisms and polymicrobial infections are seen more frequently in the CP population because of contamination from the diaper in patients with bowel and bladder incontinence [635]. When infection does occur, it generally responds to antibiotics combined with multiple surgical irrigations and débridements, hardware removal is rarely necessary, some curve correction may be lost, and time to recovery will be prolonged [342, 635, 778]. The addition of gentamicin to the allograft or vancomycin in the wound has been shown to decrease infection rates in these patients [78, 492, 538,

635, 744]. Careful attention by the surgical team to the appropriate dose of intravenous antibiotic prophylaxis and timing of redosing is merited [635].

Decubitus ulcers can occur after spinal fusion. Surgical management changes the pelvic obliquity and can produce new weight bearing areas on the ischium. These can lead to skin breakdown; thus we insure every patient has pressure mapping of their seating system prior to discharge from the hospital [252].

Results

Several reports of parent and caregiver perceptions about the outcome of spinal fusion in children with CP have been published. Parents voice high satisfaction with the results of surgery and state that appearance, ease of care, and quality of life are improved after spinal fusion [76, 805]. Studies matching operative with nonoperated children with scoliosis due to cerebral palsy show improved cpCHILD outcome scores as well as activity scores following surgical correction of spinal deformity [697, 698]. Although, improvements in caregiver outcome scores measured at 1 year have been shown to return to preoperative levels at 2 years in one study [182], a more recent multi-center study showed improvements in HRQoL at 5 years that did not correlate with the high (45%) complication rate [531].

Spinal Deformity in Myelomeningocele

Spinal deformity in patients with myelomeningocele occurs frequently, can be complex, and often requires treatment. Deformities can be congenital or acquired, specific to myelomeningocele or similar to deformities seen in other conditions. Congenital spinal anomalies include scoliosis secondary to vertebral malformations, congenital kyphosis related to posterior dysplasia, and intrathecal anomalies such as diastematomyelia. Acquired deformities include idiopathic-like or neuromuscular curves. Problems created by spinal deformity include pressure sores or interference with sitting balance in non-ambulatory patients, unstable skin over the deformity in the case of kyphosis and pulmonary compromise. Although generalizations can be made, treatment must be individualized, based on the cause, severity, and risk of progression of the deformity, the patient's age and ambulatory status, and the impact of the deformity on the patient's well-being.

Radiographic evaluation of the entire spinal column should be carried out in infants with myelomeningocele, looking specifically for the presence, location, and severity of kyphosis, the last level of posterior element closure, and any evidence of congenital spinal deformity. Routine physical examination and periodic radiographic screening for evidence of scoliosis should be performed in all patients with spina bifida because the prevalence of this deformity is so high.

Scoliosis

Congenital scoliosis is managed as if any other patient; if the deformity is progressive in a skeletally immature patient, appropriate spinal fusion should be performed.

Progressive neuromuscular (noncongenital) or idiopathic like curves are treated according to their severity, evidence of progression, and the patient's skeletal

maturity. First, the overall health of the patient's neurologic system should be evaluated, particularly in those with newly evident or rapidly progressive deformities. Shunt function should be assessed, and the spinal cord evaluated for evidence of tethering. Curves between 25° and 45° in skeletally immature patients may be considered for total-contact orthoses. Bracing in ambulatory patients dependent on extensive lower extremity braces, particularly HKAFOs, can be challenging, but because spinal orthoses can at least delay the rate of progression of deformity [62, 527], they should be considered; bracing should probably be recommended for young patients in whom deferral of spinal fusion is warranted. As for patients with idiopathic scoliosis, spinal fusion should be considered for curves greater than 55° unless the patient is a community ambulator. In this case, because a long stiff spine may adversely affect ambulation, it may be prudent to delay surgical management until the patient becomes largely wheelchair-reliant or the curve becomes significantly worse.

Patients with myelomeningocele who undergo spinal surgery are particularly likely to experience peri- and postoperative complications, making attentive treatment by a multi-disciplinary team essential. Even with such care, pressure sores, urinary tract infections, wound breakdown, deep infections, pseudarthrosis, and progression of the deformity are more frequent than in all other patient populations with spinal deformities.

Preoperatively, the treating surgeon must ensure that the patient's shunt function is stable, there is no ongoing urinary tract infection, the nutritional status is optimized, the weight-bearing skin of the pelvis and upper thighs is free of pressure sores, and the skin over the portion of the spine to be operated on is healthy. Because the posterior skin can be significantly scarred, consideration should be given to consultation with a plastic surgeon for wound management. We have found tissue expansion helpful in some of these patients [328, 838] (Fig. 28.12a–c).

Preoperative assessment should include careful assessment and neurosurgical consultation to assess the potential need for prior or concurrent detethering of the spinal cord [676]. To minimize the possibility of a potential urinary tract infection leading to a bacteremia-induced spinal wound infection, we treat patients the evening before surgery with parenteral gentamicin.

In most patients, the posterior element deficiency mandates combined anterior and posterior spinal fusion [47, 444, 504, 592, 593, 606, 745, 749, 750, 839, 887]. Most commonly we perform a single-stage combined anterior spinal release and fusion, followed by posterior spinal fusion with instrumentation to the pelvis. We use pedicle screws when possible, pedicle wires or screws in the area of posterior element insufficiency, and SAI screws or iliac screws if there is significant deformity for fixation to the pelvis [47].

Postoperatively, the patient's urinary management routine should return to the preoperative technique, usually clean intermittent catheterization, as soon as possible, with postoperative urine cultures and prompt aggressive treatment of any early urinary tract infection. Increasingly, we use an impermeable, negative-pressure (vacuum-assisted closure [VAC]) dressing to cover the wound for several weeks following the surgery. Fusion to the pelvis may alter the weight-bearing areas of the

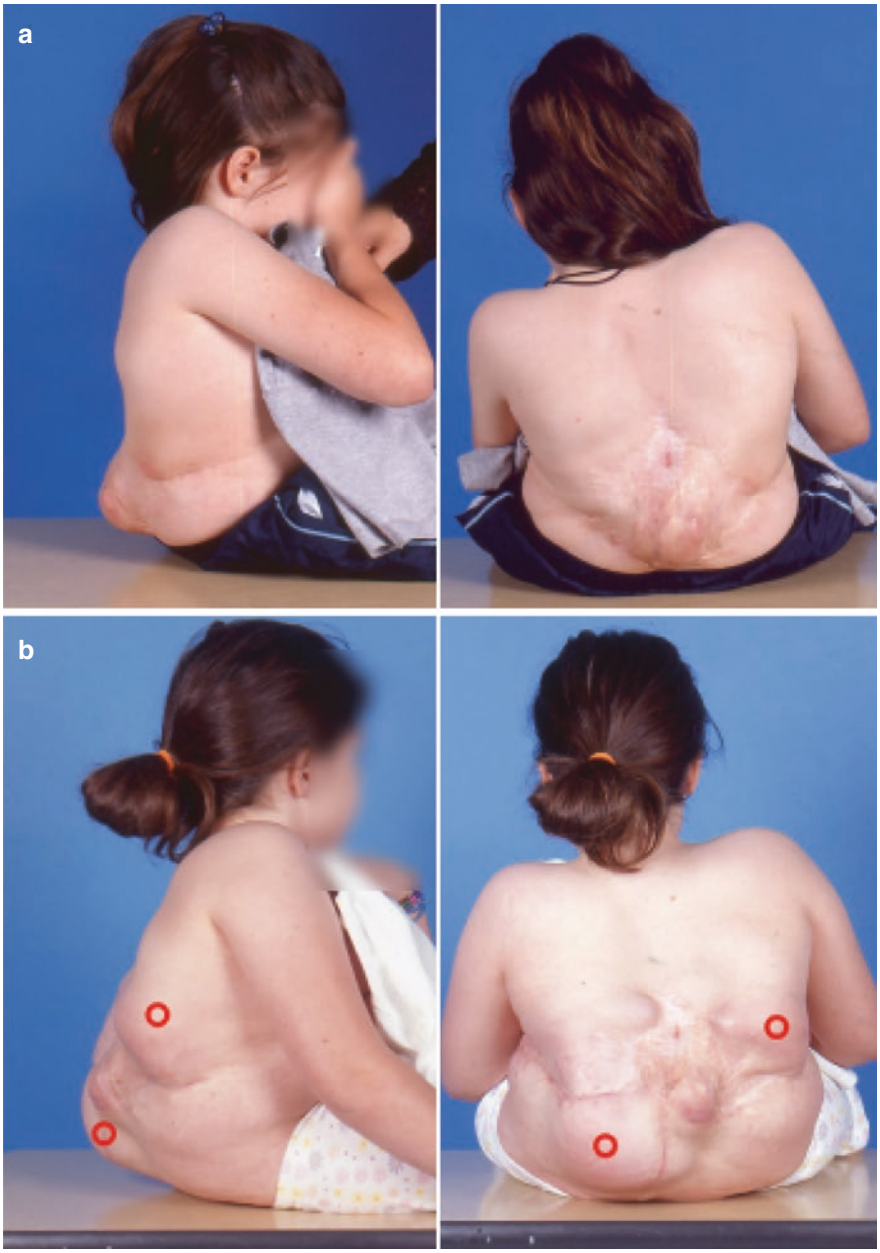


Fig. 28.12 (**a**) Preoperative clinical photographs of a patient with myelomeningocele and severe kyphosis producing recurrent skin breakdown. (**b**) Tissue expanders (note the red dots) have been placed to facilitate wound closure after kyphectomy. (**c**) Final clinical result following kyphectomy and removal of the tissue expanders



Fig. 28.12 (continued)

ischium, thus the seating system should be pressure mapped and modified as necessary prior to discharge.

Although surgical management of scoliosis can have a positive effect on pulmonary function in patients with myelomeningocele [48, 121], it is wise to educate patients and families that the long stiff spine produced by fusion can have a negative impact on the child's overall mobility and ADLs (including self-dressing and self-catheterization) [74, 497, 552, 554, 687].

Postoperative complications, including deep wound infection, pseudarthrosis, worsening of neurologic deficits, or pressure sores have been reported in more than 50% of these patients, regardless of the surgical technique [749, 839]. A 2014 study compared long term outcomes between patients treated operatively and those managed conservatively. In the surgical group there was a 32.4% infection rate, 17.6% pseudarthrosis rate, and 20% required removal of implants. There was no difference in walking capacity, neurological motor level, sitting balance, or health related quality of life between the two groups at follow-up [377].

Kyphosis

Kyphosis of the lumbar spine is a common deformity in myelomeningocele patients (20–46%). Kyphosis is more commonly seen in patients with thoracic and upper lumbar levels of paralysis. Progressive kyphosis is usually associated with a compensatory thoracic lordosis (Fig. 28.13) [120, 188, 529].



Fig. 28.13 Clinical photograph of severe kyphosis associated with myelomeningocele. Note the compensatory thoracic lordosis proximal to the gibbus

The treatment of myelomeningocele-related kyphosis is always challenging. Lumbar kyphosis can be problematic from birth, causing difficulty closing the skin and the meningeal defect. Later difficulties include skin breakdown with sitting, sitting balance problems, and even pulmonary compromise caused by pressure on the thoracic cavity from the collapsing abdomen and diaphragm. Chronic skin breakdown can leave the neural elements and the spinal column exposed and at risk for infection.

Patients with skin breakdown over a stable kyphosis that does not need treatment should have their wheelchair supports and activities carefully evaluated and any irritants causing the breakdown removed. If these efforts are unsuccessful, rotational or free flaps can be used to cover the kyphotic area with thicker, more stable skin.

There appears to be little, if any, role for bracing in an attempt to control or correct kyphosis associated with myelomeningocele. Definitive management of kyphosis consists of kyphectomy (which typically includes cordectomy and vertebral body resection) and posterior spinal fusion with instrumentation [24, 140, 148, 202, 203, 244, 251, 286, 307, 320, 392, 444–446, 462, 491, 498, 574, 576, 587, 672, 686, 708, 747, 796, 800, 840].

As in patients with scoliosis, careful preoperative assessment is necessary. The function of the shunt must be determined. The skin over the kyphosis must be as

stable as possible and, if it is of poor quality, a plastic surgeon should be consulted to assess the use of tissue expanders or rotational flaps. Patients should have careful nutritional assessment and their nutritional status maximized prior to surgery. The patient should be treated for any urinary tract infection preoperatively, and renal function should be evaluated. The aorta typically bridges the area of kyphosis and thus is not at great risk during vertebrectomy; however, the kidneys are often nestled within the kyphotic area and may be inadvertently injured during surgery [240, 450]. Cordectomy may result in improved bladder function, as evidenced by increased bladder compliance and capacity [405].

The surgical technique involves identification and transection of the spinal cord/placode, after resection of the cord, the lumbar spine is dissected extraperiosteally from the posterior approach to the anterior aspect of the vertebral bodies. Two or more vertebral bodies are resected so that the kyphosis can be reduced. Fixation to the pelvis is then carried out. Historically, a number of instrumentation techniques have been described, including fixation with Harrington compression instrumentation, Luque-Galveston instrumentation to the iliac crests, the Dunn-McCarthy modification of Luque instrumentation to the sacral alae, the Fackler technique: Luque rods contoured to fit through the first sacral foramen (a modification of the Dunn-McCarthy technique) [840], vertebral body plates, and figure-eight wire loops around the pedicular remnants, with immobilization in a cast or brace. Currently we most commonly use pedicle screws (placed in the pedicle remnant in the dysplastic vertebrae) with iliac screw fixation or Fackler fixation in the pelvis. In younger patients, we have also used sublaminar wires without fusion in the upper thoracic spine to allow for growth. There have been recent reports of using the vertical expandable prosthetic titanium rib (VEPTR) to treat young patients with severe kyphotic deformity [233, 732]. The high rate of complications in patients undergoing one-stage, definitive spine surgery has resulted in bias against the use of so-called growing techniques, which require multiple planned operations in patients with myelomeningocele; we prefer to use nonoperative delaying tactics (i.e., braces) in very young patients and perform single-stage definitive surgery after the age of 6 years.

Results of kyphectomy demonstrate the complexity of the procedure. Warner and Fackler found less recurrence (0/12 compared to 8/21) of kyphosis when the modified Dunn-McCarthy pelvic fixation was utilized [840]. Improved deformity correction using the Dunn modification of segmental fixation also has been reported by others [307, 498]. McCall [498] reported that preoperative deformity averaged 110°, postoperative deformity averaged only 15°, and loss of correction averaged only 5° on follow-up. Of 16 patients, 8 had complications, and blood loss averaged 1100 mL [498].

Garg and colleagues achieved improved seating balance and skin conditions in 17 of 18 patients undergoing kyphectomy [251]. Seven patients required reoperation and three developed deep infection. One patient who had removal of implants following deep infection developed recurrent deformity. As a result of this experience, anterior fusion prior to implant removal is now recommended for patients who develop deep infection.

Odent and associates [587] reported nine patients who underwent a two-stage procedure consisting of a posterior kyphectomy using lumbar pedicle screws and long, S-shaped rods buttressing the anterior sacrum and a thoracoabdominal approach to the spine, with an inlay strut graft from T10 to S1 as a second operation several weeks later. Kyphosis was corrected from a mean of 110° before surgery to 15° afterward, with no instrumentation failure, loss of correction, or pseudarthrosis. The authors believe that this technique improves biomechanical and biologic fusion mass anteriorly and should prevent late instrumentation failure and loss of correction. Caution is advised, however. It is generally agreed that kyphectomy with instrumentation is a major surgical procedure, intraoperative blood loss is usually well in excess of 1000 mL, perioperative deaths have occurred, and postoperative complications, including skin breakdown, infection, loss of fixation, and recurrence of deformity, occur more frequently than after most other orthopaedic procedures [24, 148, 251, 286, 307, 392, 446, 462, 491, 498, 672, 800, 840].

Scoliosis in Duchene Muscular Dystrophy

Scoliosis develops in nearly all children with Duchenne muscular dystrophy who are not treated with steroids, and becomes increasingly pronounced after the boy is nonambulatory [360, 704]. The use of corticosteroids in boys with Duchenne muscular dystrophy is reducing the incidence and delaying the development of scoliosis. In a study comparing steroid treated patients with those not receiving steroids, 20% of the treated boys required scoliosis surgery compared to 92% of those not treated. At 15 years' follow-up, 78% of steroid treated patients avoided surgery compared to only 8% of the untreated cohort [416]. The spinal curves are classically neuromuscular: long and sweeping with pelvic obliquity. Thoracolumbar kyphosis is commonly present, but lumbar hyperlordosis may be seen in some boys. If left untreated, many curves progress beyond 90°. Such curves make it difficult for the child to sit comfortably and lead to skin breakdown because the muscle weakness interferes with the patient's ability to relieve pressure during sitting [729].

Bracing has been tried but is not recommended in this patient population for several reasons. First, the goal of bracing is to prevent progression of the curvature, yet progression occurs in these patients despite bracing [113, 146, 198]. Second, bracing can impede full respiratory effort. Pulmonary function is already precarious in these children, with forced vital capacity (FVC) decreasing by approximately 4% each year and by another 4% for each 10° of thoracic scoliosis [402]. Because curve progression is the rule rather than the exception and because pulmonary function deteriorates rapidly when the patient is no longer able to walk, it is preferable to perform surgery earlier, when the child's respiratory status is functionally better. Delaying surgery because of brace treatment may make any subsequent operation less safe as a result of the presence of pulmonary disease [647, 775, 776].

The indications for spinal fusion to correct scoliosis in patients with Duchenne muscular dystrophy are different from those for idiopathic scoliosis. Surgery is performed once a curve reaches 30° and the patient is nonambulatory because curve progression is very likely and pulmonary function will deteriorate as the curve worsens [318, 729, 774, 775]. Mubarak and associates recommended surgery for

curves greater than 20° in children whose FVC is greater than 40% of normal [549]. Surgery is best tolerated before the patient's FVC is less than 35% of age-matched normal values [525]. Although surgery has been performed successfully in children with more advanced pulmonary disease [490, 780], the risk for prolonged mechanical ventilation (possibly requiring tracheostomy) and postoperative pneumonia increases. Use of noninvasive mask ventilation such as bilevel positive airway pressure (BIPAP) has improved postoperative outcomes in patients with poor preoperative pulmonary function (i.e., FVC <30%) [291]. Preoperative planning must include cardiac evaluation [702] and pulmonary function tests. Malignant hyperthermia has been associated with muscular dystrophies, particularly the Duchenne and Becker types. Use of succinylcholine and inhalational agents should be avoided during surgery [369, 411]. Intraoperative cardiac arrest [333], intraoperative anaphylaxis as a result of latex allergy [191], and complete airway obstruction because of tracheobronchial compression after intubation [652] have also been described in children with Duchenne muscular dystrophy. Hypotensive anesthetic techniques to minimize blood loss have been used in selected patients with Duchenne muscular dystrophy and mild scoliosis [105, 237].

Posterior spinal fusion with segmental pedicle screw fixation is currently favored by most surgeons because of improved curve correction and reduced blood loss in comparison to sublaminar wires [33, 284]. Historically, there was debate as to the need to extend the fusion to the pelvis. Mubarak and associates reported that for mild curves without preexisting pelvic obliquity, fusion to L5 was sufficient [549]. More recently, Sengupta and co-workers found that with smaller curves and no preoperative pelvic obliquity, fixation to L5 with lumbar pedicle screws and thoracic sublaminar wires prevented pelvic obliquity at a 3.5-year follow-up [695]. Most recently, all pedicle screw constructs to L5 have been shown to maintain correction of severe scoliosis with pelvic obliquity as long as L5 tilt measured less than 15° preoperatively [780]. Alman and Kim are proponents of fusion to the pelvis in boys with Duchenne muscular dystrophy [22]. In a review of 38 patients with the pelvis fused to L5, worsening pelvic obliquity occurred in 10 patients, 2 of whom required further spinal surgery.

However, in clinical practice, most patients have preexisting pelvic obliquity at the time of treatment of the spinal curvature. Because one of the primary goals of the operation is to ensure a level pelvis for comfortable seating, most surgeons continue to fuse to the pelvis.

The effect of spinal fusion and correction of scoliosis on pulmonary function has been studied by a number of investigators. Most authors have found no difference in the rate of pulmonary deterioration or long-term survival between patients who underwent spinal fusion and those who did not, although all agree that surgery improves sitting [371, 521, 522, 702]. Conversely, Velasco and colleagues found that the rate of decline in pulmonary function was half the annual preoperative rate of decline in a group of 56 patients following posterior spinal fusion [818]. Additionally, an average perioperative decrease in pulmonary function of 1% has been reported, which should be considered in the preoperative assessment of the

patient [653]. Galasko and associates, on the other hand, found that children whose scoliosis was stabilized maintained better pulmonary function and lived longer [246, 247].

An often unanticipated complication of spinal fusion is the loss of ability to self-feed. Prior to fusion the patient is able to lean forward and downward against a table such that the table flexes his elbows and brings his hands to his mouth. After surgery the stiffness of the spine makes this impossible [325]. Devices such as mobile arm supports are useful to restore self-feeding functions [884].

The complication rate of spinal surgery in patients with Duchenne muscular dystrophy is a concern. Major complications occurred in 27% in one study [633]. During spinal fusion, loss of blood can be substantial [701]. Although the results of laboratory analysis of platelet function are normal, bleeding times may be increased, and blood vessel reactivity is impaired [578, 813]. Platelet adhesion has also been found to be deficient in boys with Duchenne muscular dystrophy [236]. Therefore, one should be prepared for the transfusion of several units of blood [843]. Intravenous administration of an antifibrinolytic medication such as tranexamic acid has been shown to decrease blood loss in this patient group [705]. Postoperative infection is not uncommon, and instrumentation failure can occur. Medical complications, such as pneumonia, also occur more frequently in this patient population. Miller and Hoffman noted pulmonary complications in 17% of 183 patients who underwent surgery [526]. Cardiac complications have been reported during anesthesia [333] and in the postoperative period [123]. Sudden death can occur on rare occasion in these children during the perioperative period [633].

Studies have shown that the families of children with Duchenne muscular dystrophy believe that the patients' quality of life is enhanced by spinal fusion surgery [271, 633]. Without surgery, scoliosis interferes with comfortable sitting in a wheelchair, thereby deterring children from getting out into the community and forcing them into their beds during the terminal phase of the disease. Postoperative malnutrition has been documented in some of these children [325]. Families should be counseled about the serious risks associated with this surgery and the consequences if the surgery is not performed.

Scoliosis in Spinal Muscular Atrophy

Spinal muscular atrophy (SMA) is a hereditary disease characterized by degeneration of the anterior horn cells of the spinal cord and occasionally of the motor neurons of the cranial nerves (cranial nerves V through XII). The classic infantile form of SMA was first described by Werdnig in 1891 [848]; the less severe form was described by Kugelberg and Welander much later, in 1956 [397]. SMA manifests with progressive hypotonia and weakness involving the lower extremities to a greater degree than the upper extremities and the proximal muscles more than the distal. Sensation is normal, as is intelligence. The incidence of SMA is approximately 1 in 15, 000–20, 000 live births [93], and the prevalence of the carrier state is 1 in 80.

Spinal muscular atrophy is caused by deletions or mutations in the Survival Motor Neuron 1 (SMA) gene. This results in lack of production of sufficient

survival motor neuron protein which is essential for spinal cord motor neuron survival and function [223]. The disease severity depends on the amounts of normal SMN protein translated by the backup SMN2 gene, which usually excludes exon 7 and therefore produces too little functional SMN protein. A therapeutic drug, nusinersen, is now available to treat patients with SMA. It is an antisense oligonucleotide delivered intrathecally which binds repressive sites within SMN2 exon 7 resulting in increased inclusion of exon 7 with SMN2. This results in increased production of functional SMN protein [223].

Spinal muscular atrophy is usually inherited in an autosomal recessive pattern. It is the second most common disease inherited in this manner to affect children, after cystic fibrosis [93]. The genetic locus for SMA has been identified on chromosome 5q [93]. At this 5q locus, two genes have been identified: the gene for neuronal apoptosis inhibitory protein (*NAIP*), which is present in 67% of patients, and the survival motor neuron (*SMN*) gene, which was found to have deletions in more than 98% of SMA patients [93, 422]. A less common form of the disease is inherited in an autosomal dominant pattern, and its genetic locus is on 5q13 [81, 848]. With molecular genetic technology, first-trimester prenatal diagnosis is now possible [449, 868].

Spinal muscular atrophy is an extremely heterogeneous condition, with a wide variety of clinical manifestations. Classification systems have been developed primarily for prognostic value. The Byers and Banker classification is the one used most often [106, 107]. It defines three different types of SMA—type I, acute infantile SMA or Werdnig-Hoffmann disease; type II, chronic infantile SMA; and type III, a milder form, also known as Kugelberg-Welander disease. These types are genetically similar but differ in age of presentation and clinical course. As a rule, the younger the age at disease onset, the worse the prognosis.

Scoliosis is universal in nonambulatory patients with SMA and is prevalent in children with the Kugelberg-Welander form of the disease as well. Granata and associates found that all but one of their patients with the infantile form of SMA developed scoliosis; the mean age at which a curve was discovered was 4 years 4 months [272]. All patients with mild SMA who lost the ability to ambulate also developed scoliosis, although at an older age, 9 years 10 months. Of 19 ambulatory patients, 12 developed scoliosis while able to walk, but the curves tended to progress more slowly than in sitting patients. Others have had similar findings [209].

Long sweeping curves are most common in SMA, with a predominance of thoracolumbar curves. Kyphosis is present in association with scoliosis in approximately 30% of patients [272]. Despite the often large magnitude of the curves, they are more flexible than those seen in typical idiopathic scoliosis; however, they progress more rapidly.

Nonoperative treatment of scoliosis in SMA is difficult. Orthoses make sitting easier, but they are ineffective in preventing curve progression or altering the need for surgery [209, 272, 516]. Curves in nonambulatory patients were found to increase at a rate of 8°/year, despite brace use [516]. Respiratory function may be significantly depressed in patients with severe SMA, and rigid orthoses can further

tax their compromised respiratory status. Seating systems that support flexible curves are usually better tolerated than rigid orthoses [30]. A soft, custom-made thoracolumbosacral orthosis may be tolerated in young children with flexible curves between 20° and 40° to allow growth before definitive instrumentation and fusion [92].

The indications for surgical treatment are progressive spinal deformity despite orthotic management, with a curve magnitude greater than 50–65°. A frank discussion among the family, surgeon, and neurologist should occur before the decision is made to proceed with surgery. If a patient is being treated with an intrathecal drug, planning for intrathecal access should be included in the surgical preparation. Preoperative traction is an excellent way to increase the flexibility of the spine while also improving pulmonary function [30, 616].

Posterior spinal fusion with segmental spinal instrumentation is the treatment of choice for scoliosis in patients who can tolerate surgery. Fusion should include the entire thoracic and lumbar spine and should extend to the pelvis. The goal of surgery is to obtain a balanced trunk over a level pelvis to facilitate comfortable seating. In young patients with tenuous pulmonary status, the risks of anterior surgery should be discussed thoroughly with the neurologist and pulmonologist and carefully weighed against the risks of crankshaft [1]. Recently there have been reports of the successful use of growing rod techniques to manage young patients with severe spine deformity [126, 501]. Although these reports are encouraging, it is important to weigh the burden of multiple operations with a high likelihood of complications associated with growing rods against the good results reported with single-stage, definitive surgery.

Although curve correction and stabilization can be achieved through spinal arthrodesis, the tradeoff is a possible decline in the ability to carry out ADLs. Specifically, the rigid and upright spine creates difficulty in self-feeding, drinking, and self-hygiene because the patient cannot bring his or her hands up to the face because of upper extremity proximal muscle weakness [92, 209]. Most studies, however, report patient and family satisfaction and a willingness to choose the surgery again if faced with that decision [30, 89]. Patients most at risk for losing function are those who are weakest preoperatively [245]. In general, an improved outcome was seen in sitting balance, cosmesis with respect to trunk position, and overall quality of life; intermediate to good outcomes were seen with regard to pulmonary status, pain, and self-image. Counseling patients and their families before surgery is essential. Occupational therapists should evaluate the children for the appropriateness of adaptive equipment following surgery.

There are conflicting reports in the literature regarding the effect of scoliosis surgery on pulmonary function. There is agreement that a decline in respiratory status correlates with an increasingly severe scoliosis [657]. Some authors have found that respiratory parameters improve after spinal arthrodesis [657], whereas others have found a continuous decline in pulmonary function [30, 616]. Nonetheless, aggressive pulmonary care must be provided perioperatively to avoid respiratory complications such as pneumonia and the need for prolonged mechanical ventilation.

Scoliosis in an ambulatory patient poses a treatment dilemma. Up to 50% of walking patients develop scoliosis. Often, lumbar lordosis and pelvic motion compensate for the proximal muscle weakness of the lower extremities and are essential to the gait in these patients. Because the ability to walk may be lost in some patients after spinal arthrodesis [209, 245], surgical treatment of scoliosis should be delayed when reasonable in ambulatory patients [703].

Scoliosis in Neurofibromatosis

Scoliosis is the most common skeletal manifestation of neurofibromatosis [155, 156, 379, 809]. It can be either nondystrophic or dystrophic, depending on accompanying abnormalities specific to this disorder [112, 156, 242, 379, 406, 588, 721, 849]. Differentiation between the two types is important because the prognosis and management differ significantly. Dystrophic scoliosis is more common, has a greater tendency to progress, and includes a subgroup of patients (those with severe kyphoscoliosis) at risk for neurologic deficits [721]. Nondystrophic scoliosis more closely resembles idiopathic scoliosis in both curve patterns and behavior. It is now recognized that nondystrophic curves in younger children can modulate into the more worrisome dystrophic type over the course of several years [156]. The overall modulation rate is 65% but varies depending on the age at which scoliosis clinically presents. Modulation occurs in 81% of NF-1 patients with scoliosis before age 7 years, and 25% of those after the age of 7 years.

Nondystrophic Scoliosis

The nondystrophic scoliosis is similar to idiopathic scoliosis in clinical appearance, radiographic findings, and behavior of the curve. However, nondystrophic deformities usually become apparent at an earlier age than idiopathic curves and have a slightly higher likelihood of progressive deformity and over time, some nondystrophic curves evolve or "modulate" into dystrophic curves. Management of nondystrophic curves is similar to that for idiopathic scoliosis. Curves less than 25° can be observed closely without active intervention. Brace treatment appears to be effective for skeletally immature individuals with curves between 25° and 40° [379]. However, once the nondystrophic curves of neurofibromatosis exceed 40°, posterior spinal fusion with instrumentation is recommended.

Dystrophic Scoliosis

In dystrophic scoliosis, short (4–6 spinal levels), sharply angled curves develop at an early age, often as young as 3 years. Radiographic features that help differentiate dystrophic from nondystrophic curves include vertebral scalloping, spindled transverse processes, severe apical vertebral wedging and rotation, foraminal enlargement, deficient pedicles, penciling (narrowing of the proximal portion) of the ribs, the presence of paravertebral soft tissue lesions, and rarely, subluxation between vertebral bodies. Some of these findings may result from direct erosion of the bone by intraspinal neurofibromas, paraspinal neurofibromas, or dural ectasia. Dural ectasia is an expansion in the width of the thecal sac thought to be due to an increase in hydrostatic pressure.

Fortunately, most dystrophic curves are not accompanied by an excessive amount of kyphosis [721]. Individuals with this combination have significant potential for the development of neurologic deficits. Kyphosis may occur in one of two ways. An abrupt angular kyphosis may be present in the very early stages of the deformity, or a more gradual kyphosing scoliosis might result from progression and rotation of the scoliosis (Fig. 28.14) [242].

Once kyphosis is established, prompt combined anterior and posterior spinal fusions are required.

Nonoperative management of dystrophic scoliosis is almost always unsuccessful. These curve patterns need early and aggressive surgical intervention, even in a young child. Delay leads only to progressive deformity, which may be as rapid as 8° per year in the frontal plane and 11° per year in the sagittal plane [112]. Most patients exhibit marked progression before 10 years of age, and severe deformity can be seen before the adolescent growth spurt. Characteristics of dystrophic scoliosis that correlate with excessive risk for progression include early age at onset, a high Cobb angle at the time of initial evaluation, and the presence of vertebral scalloping, penciling of multiple ribs, and apical vertebral rotation exceeding 11° (Perdriolle measurements [612]) [242].

Before surgery, a thorough neurologic examination is essential to identify any subtle abnormalities. MRI and CT should always be performed [809]. MRI may demonstrate neurofibromatosis lesions in the neck, thorax, paravertebral region, neural foramina, or spinal canal. Chiari I malformations, dural ectasia, pseudomeningoceles, and spinal cord compression (secondary to localized kyphosis, rib impingement, or a mass effect from neurofibromas) [34, 167, 250, 376, 378, 471, 631, 808, 810]. CT demonstrates scalloping of the vertebral bodies anteriorly, erosion of the posterior portion of the vertebral body or lamina from dural ectasia, and the presence of ribs within the spinal canal [1, 110, 811, 837, 881]. Three-dimensional CT reconstruction is invaluable in clarifying the anatomy of severe deformities and is helpful in preoperative planning.

Posterior spinal fusion with instrumentation alone can be used for certain patients with dystrophic curves between 20° and 50° (perhaps even greater) and kyphosis of less than 50° (no sharp angulation). Because the risk for pseudarthrosis is higher than that in the idiopathic scoliosis population, consideration should be given to performing imaging studies (tomography) 6 months after surgery. If the fusion mass appears inadequate, repeated bone graft augmentation may be necessary.

Anterior fusion in addition to posterior fusion is needed for most patients with dystrophic curves [290, 393, 605, 718, 785]. The combination of anterior and posterior fusion increases the likelihood of successful fusion. Longer fusions are generally indicated, even in young patients. Curve progression can occur in patients with neurofibromatosis, even in those with a solid arthrodesis [849].

Severe kyphoscoliosis absolutely requires anterior fusion in addition to posterior fusion. Thorough anterior diskectomy, bone grafting, and rib (or tibia) strut graft placement are needed. In some patients with exaggerated kyphosis, the apical rotation may be so severe that the vertebral body faces posterolaterally. With this deformity, placement of the strut graft can be extremely difficult, and the anterior

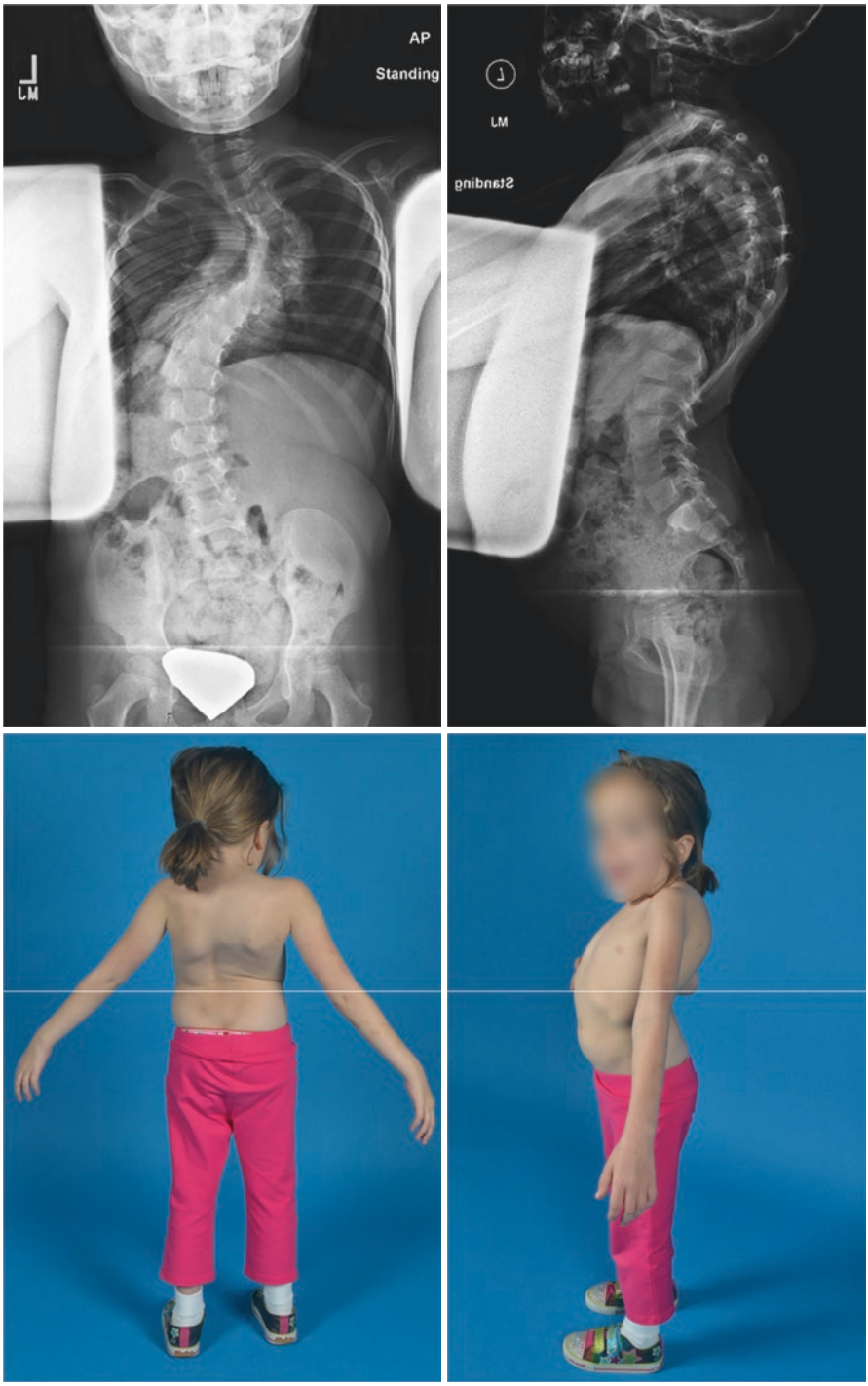


Fig. 28.14 Clinical and radiographic representation of scoliosis associated with neurofibromatosis. Radiographically the curve is short, sharp and kyphotic. Clinically note the café au lait spots

approach to the spine may need to be undertaken from the concave side. Vertebral body erosion secondary to intrathoracic neurofibroma or dural ectasia can also significantly interfere with anterior exposure and fusion. Dysplastic posterior elements limit the ability to achieve strong posterior internal fixation. Despite meticulous attempts at anterior and posterior fusion, pseudarthrosis is a significant concern [156, 721, 862].

Excessive kyphosis is the most frequent cause of neurologic deficits in patients with neurofibromatosis and spinal deformities. This can occur in either the thoracic or cervical spine [304, 367]. Should a neurologic deficit be present, VCR in the thoracic region may be needed to decompress the spinal cord [752]. Laminectomy for spinal cord decompression or prophylactic laminectomy for kyphoscoliosis should be avoided because it destabilizes the spine, increases the kyphosis, removes bone stock needed for successful posterior fusion, and most important, does not relieve the anterior compression on the spinal cord. Neurologic deficits can also result from cord impingement by neurofibroma lesions within the spinal canal [28, 617]. Differentiation of neurofibroma impingement from kyphotic impingement is required to correctly address the problem surgically. MRI can help clarify the situation.

Scoliosis in Marfan Syndrome

Marfan syndrome, one of the more common connective tissue disorders, has a 0.01% prevalence in the general population [741]. Scoliosis is the most common spinal deformity in this condition, with a prevalence approaching 63% [654, 655, 741, 781]. In addition, 6% of patients with Marfan syndrome have spondylolisthesis. Although Marfan syndrome is an autosomal dominant disorder, no familial pattern of scoliosis has been identified.

The curve patterns seen in Marfan syndrome are similar to those seen in idiopathic scoliosis, although Marfan syndrome has a slightly higher rate of triple curves and thoracolumbar curves [71]. Scoliosis is equally distributed between males and females, in contrast to the female preponderance in idiopathic scoliosis.

Back pain is more frequent in patients with Marfan syndrome than in the general population. However, no significant difference in back pain between patients with scoliosis and those without has been noted. Back pain is associated with the presence of dural ectasia, a finding that is more common in those with Marfan syndrome than in the normal population [13, 14, 217, 281, 518, 740].

No well-defined natural history studies of scoliosis in patients with Marfan syndrome exist, although certain trends are evident. Curves identified in infancy progress dramatically [743]. These curves do not resemble the curves of infantile idiopathic scoliosis in that they are not expected to resolve spontaneously and are largely right thoracic in configuration. In older but still skeletally immature patients, all curves greater than 30° will probably progress at least 10° and will reach at least 40° by maturity.

Unfortunately, brace treatment is not effective in controlling scoliosis in Marfan syndrome [69, 172, 400, 741, 743, 853], with a reported success rate of just 17% [740]. Most skeletally immature patients (Risser grade 2 or less) with curves

exceeding 25° will reach the stage at which surgery is necessary, even with brace treatment. In infants, curves almost always progress to the point of needing operative intervention. Nevertheless, bracing curves less than 40° in infants may be a useful technique for postponing surgery. Bracing in older children may also be temporarily beneficial in that it may allow sufficient maturity to be gained that only posterior surgery is needed.

Spinal stabilization for scoliotic deformities is indicated when the magnitude of the curve exceeds 45° in adolescents or 50° in adults. Because of an increased risk for atlantoaxial rotatory instability, special attention to intubation and positioning, both intraoperatively and postoperatively, is necessary [306]. The spinal procedure of choice is posterior spinal fusion with segmental instrumentation [176, 352, 400, 439, 627, 853]. When compared with idiopathic scoliosis, patients with Marfan syndrome will have atypical curve patterns; require more levels of surgical correction, more distal fusion, greater correction of sagittal balance, and more reoperations; and have more cerebrospinal fluid leaks and instrumentation-related complications [172, 259, 260, 349]. Patients with Marfan syndrome tend to have a higher incidence of pseudarthrosis, although its true incidence is unknown [349, 400]. A higher incidence of perioperative complications, including increased blood loss, infection, and curve decompensation, has been reported [349].

Relative contraindications to performing corrective surgery for spinal deformity in patients with Marfan syndrome include cardiac insufficiency and a dissecting aortic aneurysm. These conditions should be treated before orthopaedic intervention is undertaken. Splenic rupture has been reported following posterior spinal instrumentation [139].

Early-Onset Scoliosis

Early-Onset Scoliosis is defined as scoliosis of *any etiology* diagnosed prior to age 10 years [724]. Children presenting with significant scoliosis prior to the age 6 are at high-risk for *thoracic insufficiency syndrome* [116], defined as an inability of the thorax to support normal respiration and lung growth. In managing these patients, the emphasis is on controlling the spine deformity while maintaining growth of the spine and thorax to promote increased lung volume throughout the critical first decade of life. Because of the multitude of etiologies with varying co-morbidities and natural histories, the **C-EOS** classification system (Fig. 28.15) has been developed and validated [850] to help generate prognostic details and guide care for this challenging and potentially lethal condition.

Scoliosis Effect on Respiratory Function

Respiratory failure from *untreated* scoliosis presenting before 5–8 years of age has been documented for at least 3 decades [87, 273, 716]. Untreated Swedish patients with infantile (0–3 years) and juvenile (4–9 years) onset were found to have a significant increase in observed mortality in comparison to the general population; in contrast, patients with adolescent (older than 10 years) onset had the same mortality rate as the general population [610]. Not surprisingly, the magnitude of the scoliosis also plays a role in the demise of untreated patients: untreated curves greater than

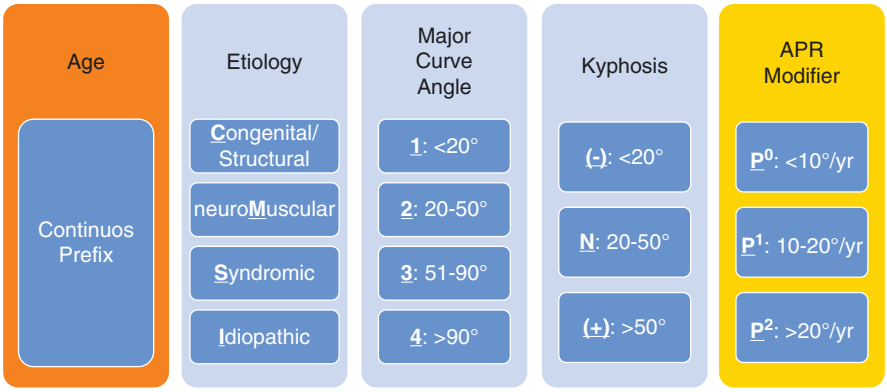


Fig. 28.15 EOS classification. (From Williams et al. [850])

70° result in a higher mortality [610]. In a Scottish study of children with infantile idiopathic or congenital curves, a decrease in vital capacity was directly correlated with increasing deformity, whereas in adolescent-onset deformity, vital capacity was not effected by an increasing Cobb angle [551]. The combination of onset before 6 years and a curve over 100° can produce respiratory failure as early as the third decade [716]. The source of the respiratory failure is twofold: diminished lung volume and impaired chest wall function.

Lung Volume

Alveolar hyper*plasia* has traditionally been considered the main source of lung growth until age 8, at which point hyper*trophy* takes over to increase volume until maturity [640]. The volume necessary for this hyperplasia and hypertrophy to occur comes from the growth of the thoracic cage. Thoracic volume depends on the length of the T1-12 segment, as well as thoracic coronal width and sagittal depth provided by the rib cage. The length of the thoracic spine increases by 50% (from 12 to 18 cm) from birth to 5 years of age [183], achieving 60% of the adult spine length in the first 5 years. The circumference (coronal width and sagittal depth) of the thorax *doubles* in size *after* age 10 [183].

The traditional method of halting curve progression is spinal fusion. However, in a young child fusion can impair the growth of the entire thorax (depending on the age at which the fusion is performed and the number and location of segments fused) and has the potential to produce thoracic insufficiency [114].

Impaired Chest Wall Function

Scoliosis produces rib and chest wall deformities that decrease compliance and disturb respiratory function [60, 114]. In non-congenital deformities, the rib deformity secondary to the scoliosis produces inefficient respiration. The intercostal spaces on the concave hemithorax are narrowed and unable to expand – a restrictive condition. Meanwhile, the convex hemithorax has widened intercostal spaces that cannot generate normal expiratory function [350]. In congenital scoliosis, patients have been

found to have decreased vital capacity relative to idiopathic curves of the same magnitude, presumably as a result of concomitant rib anomalies producing additional chest wall dysfunction [596]. When fusion "in situ" is performed at an early age, *without* sufficient correction of the spine and/or chest deformity, the pulmonary function is poor [267, 361, 827].

Thus, it is important to understand the goals in treating young children with large or progressive spinal deformities. Volume *and* compliance are important. A shorter spine *without deformity* may be the best outcome that can be expected [348]. Although it is important to avoid fusion of the thoracic spine, especially the proximal segments, before 5 years of age, it is also important to control and correct the deformity. It is the combination of progressive deformity without growth which is potentially lethal. The goal must be to control the spinal deformity without impeding thoracic growth, attempting to prevent the development of thoracic insufficiency.

Treatment

Non-operative/"Delaying Tactics"

Attempting to delay definitive spinal fusion until 8–10 years of age by non-operative means is the first approach in a patient at risk for thoracic insufficiency. If fusion can be delayed until 10 years of age, the need for anterior fusion may be avoided [345]. If it is not possible to delay until a definitive fusion can be performed, we work aggressively to delay initiation of "growth friendly" surgical techniques as long as possible, ideally not before 4 or 5 years of age.

Bracing

Bracing is often the first treatment for non-congenital deformities if the patient has no coexisting conditions where a brace would adversely affect respiratory function by circumferential chest or abdominal compression. Although bracing efficacy is difficult to define or prove by the standard criteria of success, orthotic management can be considered successful if progression is prevented for several years, thereby delaying any surgical management until the child is older.

When using a brace in a child younger than 5 years, it is important to monitor for brace-induced deformities on rib cage or obliteration of normal sagittal contours (lumbar hypolordosis and thoracic hypokyphosis). Iatrogenic rib and sagittal spine deformities should not add to the existing deformity. A well-fitted orthosis is generally accepted by children younger than 6; poor acceptance often indicates poor brace fit, which may be due to progression and/or increased rigidity of the deformity.

Casting

Serial casting is frequently used in the non-operative management of EOS with the goal of curing young patients with small curves and delaying operative treatment in older patients with larger curves. The cast must be applied under general anesthesia on a special casting table, with neck halter and pelvic strap to apply traction (Fig. 28.16).



Fig. 28.16 Intraoperative photographs of a patient at the conclusion of the application of a Mehta cast. Note the cervical and pelvic traction and the window was cut into the cast

Casting complications include pressure sores, especially in patients who are not cognitively normal. For this reason, casting may be relatively contraindicated in such patients [513, 671].

Mehta reviewed a 20-year experience of treating infantile-onset, non-congenital scoliosis with serial casts [513]. and reported that 69% (94/136) of patients obtained "full" correction (curve <10° at maturity) when treated aggressively (mean age 19 months with an average curve of 32° pre-casting) Patients who started treatment later (mean age 30 months) with larger curves (52°) did not gain the same correction, but their deformities did not progress (46°) at follow-up. Her protocol required cast changes under anesthesia every 2–3 months in children younger than 2 years, with a minimum of 5 casts. The goal was to achieve a straight spine, at which time the patient was switched to a brace. Children older than 2 years required cast changes every 3–4 months. Older children demonstrating "recurrence" were placed back in a cast for 4 months to "re-correct" the deformity before continuing with bracing. Mehta's important contribution was to demonstrate that serial casting in young children (even infants), if pursued appropriately, can "cure" the deformity over a long follow-up period. Similar experience with casting has also been reported by others – some with less remarkable results [294, 332, 681].

In our practice, casting is also a valuable method to delay surgical intervention in curves near surgical magnitude. As a curve increases in severity and stiffness, bracing may no longer be tolerated. A series of casts applied under anesthesia can provide significant curve correction, improve the flexibility of the spine, and permit resumption of brace wear. Fletcher reported the TSRH experience treating thoracic

curves averaging 69° in 4 year-old patients, with correction to 39° during casting and delaying surgery 39 ± 25 months [226]. A multicenter study reported similar results – 27 patients with average curves of 65° had surgery delayed 1.7 years by casting [347].

Traction

We have found halo–gravity traction (HGT) to be a valuable tool in managing patients with EOS as it achieves deformity correction in both coronal and particularly the sagittal plane and, indirectly, improves respiratory mechanics [407, 720]. The technique was originally described by Stagnara [748] and later demonstrated to TSRH staff by Zielke after a visit to the latter's clinic in Germany in 1984. A halo is first applied with six to eight pins and the child under general anesthesia [548, 720]. Experience has shown that using more pins actually decreases the chance of pin infection/loosening for any single pin. Pins are tightened to a torque of one-foot pound per year of age of the child (for example, a 4-year-old patient's pins are tightened to 4 foot-pounds). Traction is achieved via a bale on the halo which is connected to a spring based dynamic traction device attached to a wheelchair or walker (Fig. 28.17).



Fig. 28.17 Clinical photograph of a patient in halo gravity traction. Note the multiple pins in the halo and the spring containing device that allows the patient to increase or decrease the traction

Traction is initiated at 5–10 lb and increased in weight and duration (with careful neurologic monitoring) over 10–14 days to a goal of 50–80% of body weight (up to 50 pounds) and 10 hours a day sitting and 4 hours walking. The traction is increased to the point the patient's buttocks are lifted slightly off the wheelchair seat while sitting; the patient should be up just on tiptoes in the walker. In response to pain or neurologic symptoms, the patient can automatically relieve the traction by pushing up on the arms of the wheelchair or walker. All patients need careful neurologic assessment twice daily while traction is being increased. It is important to assess all cranial nerves, including the 11th nerve for trapezius strength (shoulder shrug). Lower extremity strength and reflexes are also monitored. In our most recent review of 107 cases, there were four traction-related complications: one patient with a pin site infection necessitating pin exchange; another patient with *hyperpyrexia* that resolved with cessation of traction; a third developed incontinence and tremors felt to be associated with HGT as it resolved with traction cessation; and a fourth patient fell and dislodged pins on their halo, necessitating pin revision [407]. We have also encountered an 11th nerve palsy in a patient with myopathy, where the palsy was only discovered after traction was discontinued with insertion of growing rod instrumentation. The ability to mobilize patients with weakness, osteopenia, and respiratory compromise during the traction period is invaluable in preparing them for surgery. Nutritional support, often via g-tube, is an important part of the preoperative preparation that should also be addressed while the patient is in HGT. We have used HGT to mobilize the spine prior to casting or bracing, prior to growth friendly or definitive surgery and, in a few isolated cases, as definitive treatment for patients with severe curves and medical fragility (up to 9 years).

Operative Treatment

Instrumentation Without Fusion: Growing Rod Instrumentation (GRI)

In 1978 Moe introduced the technique of *subcutaneous* single Harrington "growing rod instrumentation" for the management of early-onset scoliosis [536]. With this technique the spine is exposed subperiosteally only at the end vertebrae, hoping for continued growth of unexposed intercalary segments after distraction between end vertebral anchors (hooks in this initial report) and the patient returns to the operating room at six moth intervals for repeated lengthening/distraction of the overlapping rods. Moe reported that in the early cases, the apex of the curve remained unfused until the definitive fusion whereas the end vertebrae uniformly fused spontaneously [536]. Subsequent reports showed that the intercalary segments ankylosed even though they were never actually exposed [8], and decreasing effectiveness of repeated distractions resulted in little additional correction of the stiffened spine [390, 787]. The amount of lengthening achieved was modest (1.2–3.1 cm) with complication rates ranging from 11% to 30%.

In an effort to improve the outcome with "growing" instrumentation, dual-rod subcutaneous instrumentation was introduced. The second rod provided better stability (eliminating the need for long-term bracing) and decreased the amount of crankshaft [16, 355]. Early results in non-congenital cases confirmed these

advantages along with an increase in T1-S1 length of 5 cm at the initial procedure and an additional 4.6 cm in serial lengthening. No improvement in Cobb angle following the original distraction occurred, but correction of around 40% was maintained [794]. However, patients with preoperative hyperkyphosis (>40°) continue to be poor candidates for growing rod instrumentation because of the increased likelihood of failure of the proximal anchors, rod fractures and the biomechanical inefficiency of correcting kyphosis by distraction [689]. In these patients preliminary treatment with halo traction may reduce the kyphosis and facilitate successful GRI.

Two decades of experience with of GRI has resulted in improved understanding of the importance of proximal anchor density. Five or more proximal anchors, (pedicle screws, sublaminar wires or tapes or and hooks on spine or ribs) or a construct length/proximal anchor ratio <3.5 significantly protects against upper anchor migration and failure [315, 828]. Despite these advances, complications associated with GRI, including anchor and rod failure, wound problems [64, 841, 883], spontaneous ankyloses [109, 234] and the "law of diminishing returns" [685] remain prevalent.

The only study of pulmonary and functional outcomes of GRI patients who have completed treatment was published in 2017 from TSRH [348]. Twelve patients were evaluated some 3 years following final fusion or at least 2 years since the most recent lengthening if simply being observed. Radiographic outcomes were quite satisfactory – nearly 47% correction of Cobb angle, an average of 9 cm increase in T1-12 length and over 1 liter of absolute lung volume gained via a mean of 10 procedures (average 7 lengthenings and 2 revisions). However, in terms of spine height and pulmonary function, they were <5th percentile around 50% predicted, similar to the values at the beginning of treatment. Even with these modest results (patient parameters were not improved but simply maintained), patients were as active in daily movement as controls (measured by a step-activity monitor) and during treadmill exercise, subjects walked and reached age-specific heart rate targets the same as controls. It appears that a *realistic* long-term goal of early-onset scoliosis treatment is to maintain spine elongation and pulmonary function *no worse* than at the initiation of treatment. Equally important in this series were patient – reported outcomes (SRS-30, EOSQ-24), which documented impairment in general health and physical function scores. Interestingly, in comparison to reports administered to patients *still undergoing treatment*, these patients who were 2–3 years removed from treatment were clearly better, suggesting that the graduates were reporting improved quality of life *after* the 2–3 year hiatus from their most recent surgery [151, 186] (Fig. 28.18a–h).

Magnetic Controlled Growing Rod (MCGR)

The development of a magnetically controlled growing rod (MCGR) by Ellipse technologies (Irvine, CA) is the most recent advance in the treatment of EOS as it allows rod lengthening without a surgical procedure. Available outside the US since 2009, the device was approved by FDA in 2014. The device is a titanium rod with an actuator section containing a magnet-driven distraction-retraction mechanism. When the magnet in the actuator is rotated by a hand-held controller placed over the patient's back; the rod housed within the actuator can be advanced (or retracted).

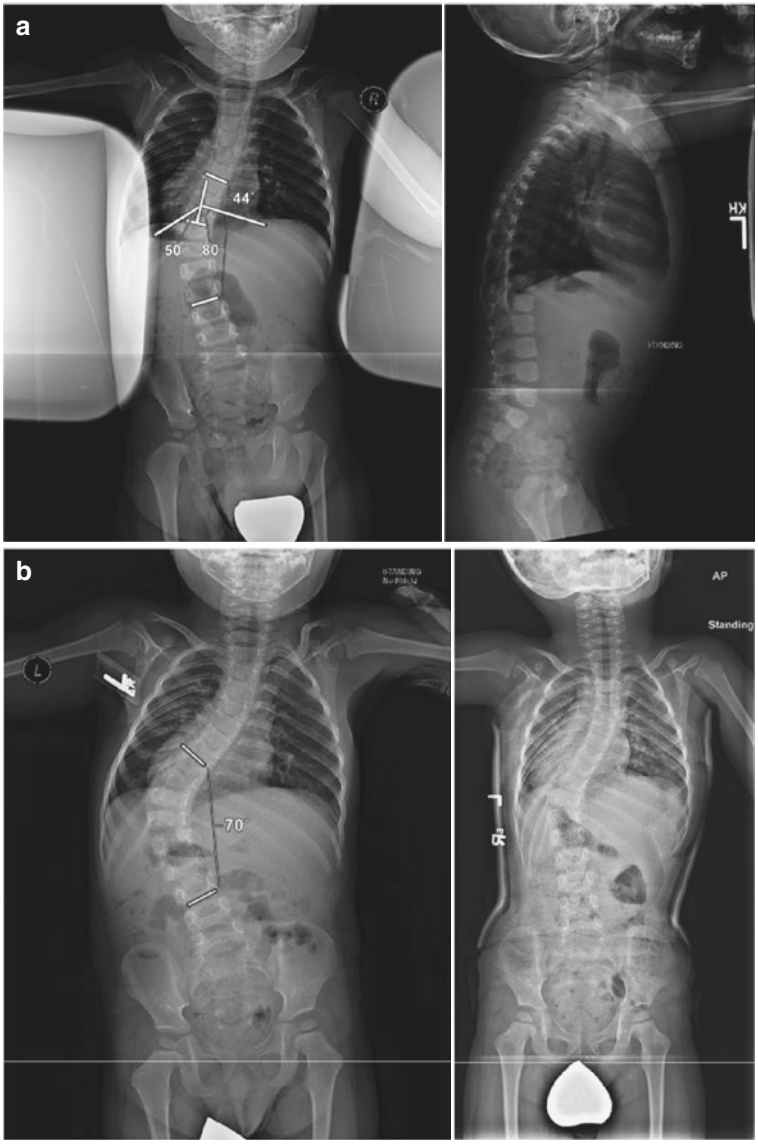


Fig. 28.18 (**a**) PA and lateral radiographs of an 18-month-old with idiopathic scoliosis. Despite a rib vertebral angle difference of 30° the family elected observation rather than Mehta casting. (**b**) The curve progressed and Mehta casting was initiated. (**c**) Casting and bracing were utilized as "delaying tactics" for 2.5 years. At 5 years of age halo gravity traction was initiated. (**d**) PA and lateral standing radiographs following placement traditional growing rods after 2 months of traction. (**e**) After four lengthenings the proximal pedicle screw anchors have migrated posteriorly. (**f**) The proximal anchors were revised in the traditional growing rods exchanged for magnetically controlled rods. The patient is 8 years of age. (**g**) Between the ages of 8 and 14 the patient underwent 14 Magic lengthenings. Note the extension of the rods and the posterior migration of the proximal anchors and the development of a proximal junctional kyphosis. (**h**) The magnetically controlled rods were removed and the patient underwent definitive fusion at 14 years of age. Note the correction of the sagittal plane deformities with correction of the proximal junctional kyphosis, restoration of thoracic kyphosis and lumbar lordosis

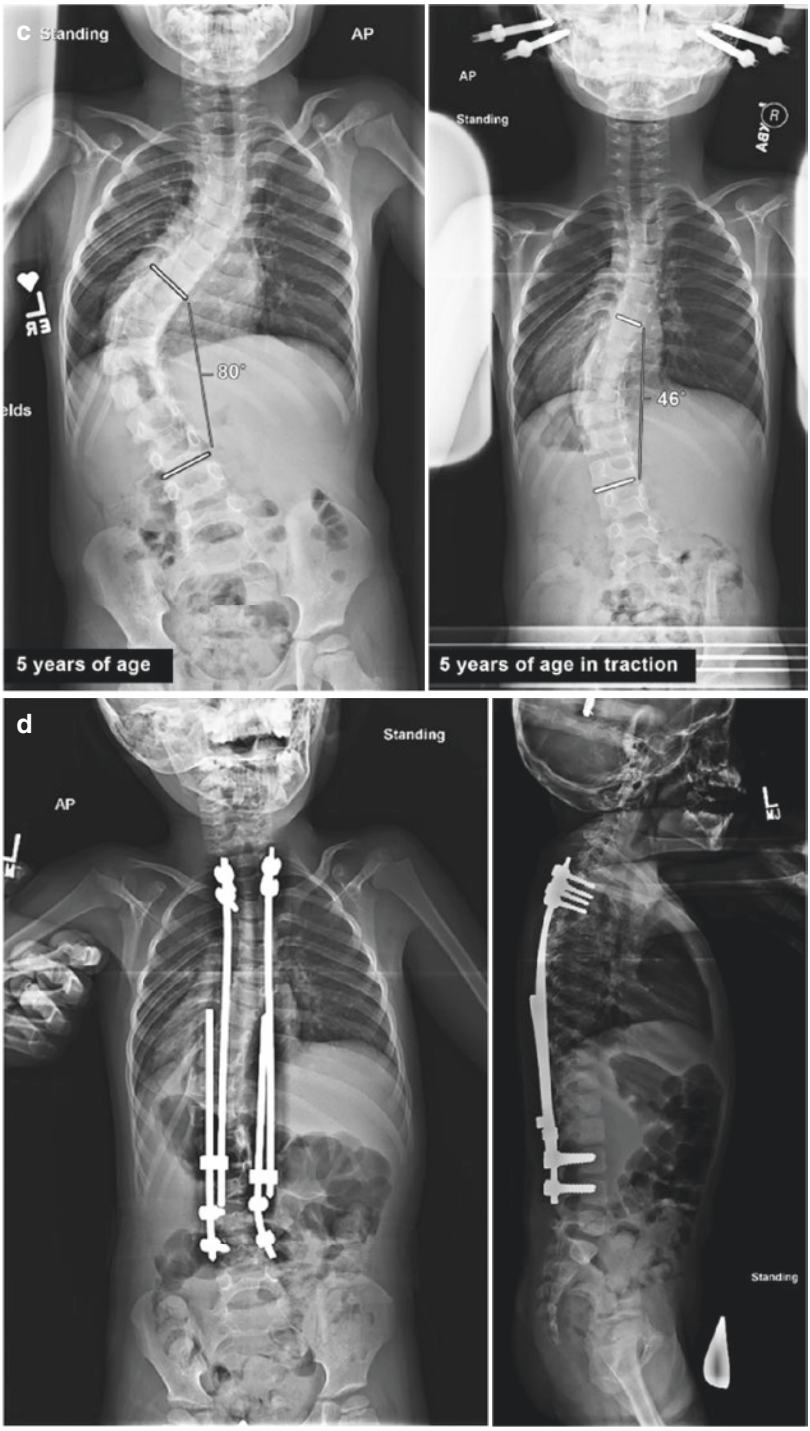


Fig. 28.18 (continued)

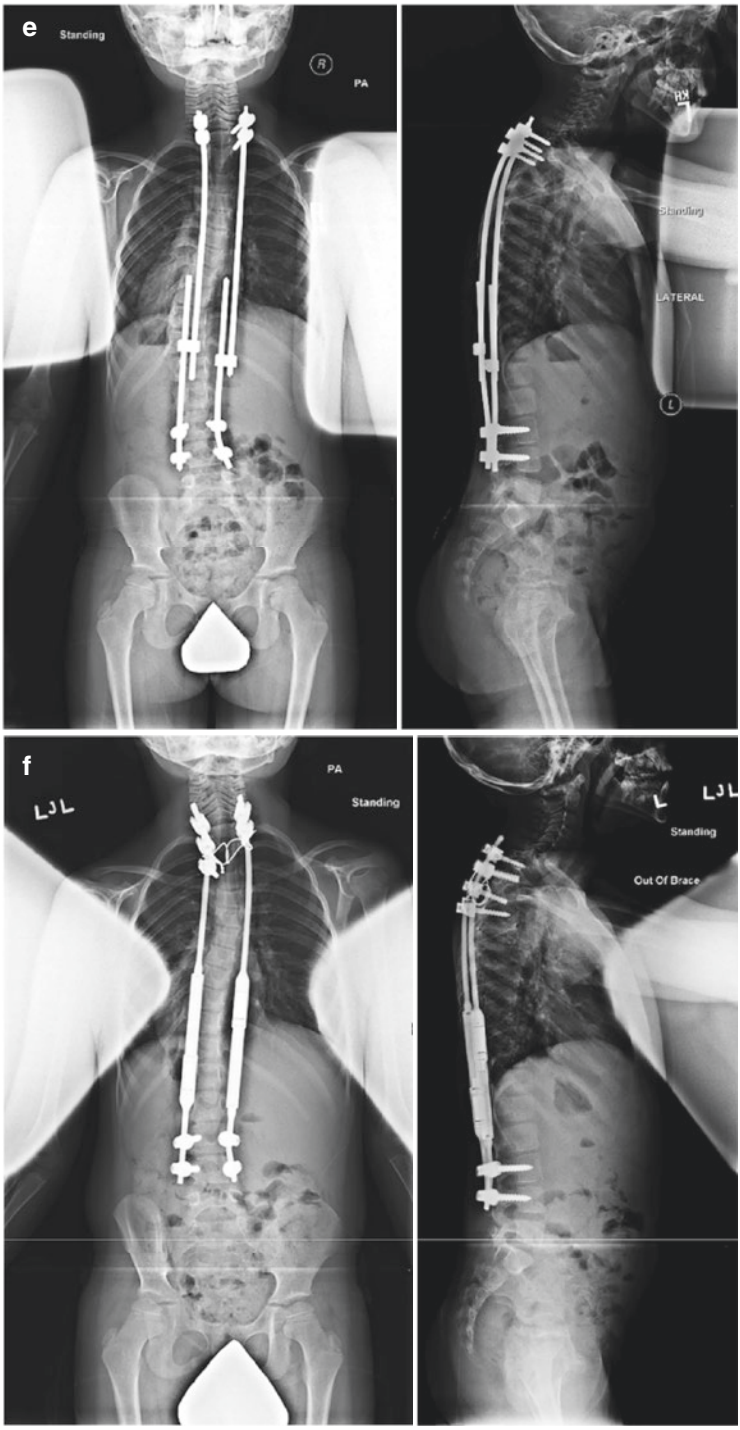


Fig. 28.18 (continued)

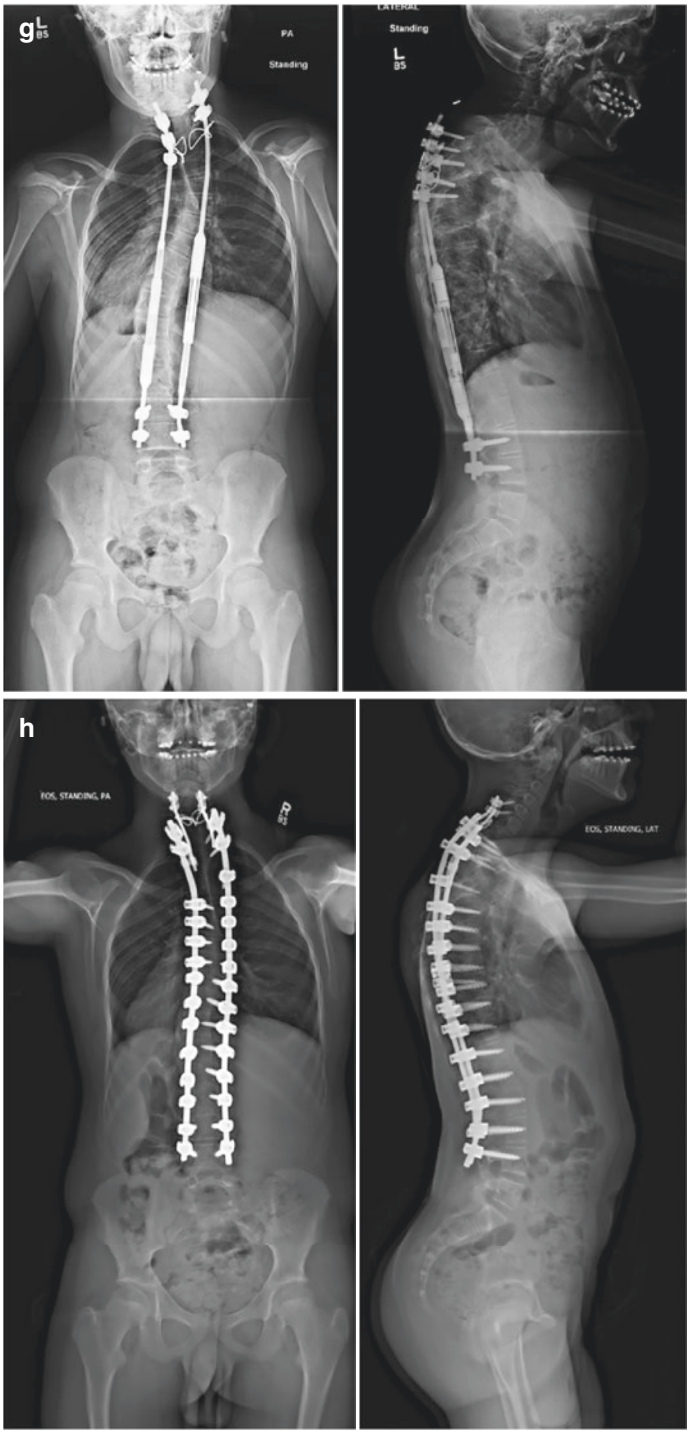


Fig. 28.18 (continued)

The critical feature of this device is that the actuator portion (70 or 90 mm) cannot be contoured, and so all necessary contouring for implantation – usually in the sagittal plane - must occur *outside* the actuator zone. Patient selection for MCGR becomes critical because there are cases of severe convex rib hump deformity, or hyperkyphosis, where a perfectly straight, non-contourable segment 7 or 9 cm long is difficult if not impossible to implant.

Contraindications to MCGR), besides kyphotic deformity which cannot be accommodated, include patients with implanted pacemakers or cardioverters and those requiring serial MRI. The latter is a relative contraindication in that if the area of interest is remote from the spine, then there will be little image degradation by the magnetic devices. However, for patients needing high resolution MRI for spinal cord surveillance for intradural or spinal canal pathology, the image degradation by the magnetic rod is too extended over a long segment, and MCGR should be avoided [96].

Because experience with the MCGR is limited by its abbreviated time of availability, reported results of treatment are equally limited. Cheung in Hong Kong published the initial report in 2012 in five patients who underwent monthly outpatient lengthenings, gaining 57% curve correction and 30 mm of T1-12 length gain in 2 year follow-up [135]. Akbarnia reported the "next generation" of growth-sparing technique by assembling a European and Middle Eastern series and determined that dual MCGR's were more effective than single rods [15]. A comparison between traditional growing rod instrumentation (TGR = GRI) and MCGR's in 2014 showed that curve corrections were similar, that TGR patients gained more length, but required 73 surgeries (including revisions) to do so while MCGR) patients had only 16 surgeries (including revisions) and 137 outpatient lengthenings. Although complication rates were similar, the most striking finding of this early study was that the spine length obtained by MCGR constructs was only one-third of that gained by TGR over the T1-S1 segment, and was only one-seventh of that gained by TGR between T1 and T12 [12, 17, 417]. Unquestionably the ideal patient for MCGR implantation is a hypokyphotic curve or a neuromuscular patient for whom recurring anesthesias would be undesirable.

Expansion Thoracoplasty with VEPTR

In 1987, faced with what appeared to be untreatable scoliosis in a 6-month-old infant with absent ribs and flail chest who was respirator dependent and could not be weaned, Campbell and associates [114] implanted a "chest wall prosthesis" made of vertically oriented Steinmann pins wired to the vestigial ribs. Not only was the child subsequently weaned from the respirator, but the scoliosis was significantly improved by the rib distraction produced and maintained by the pins. To deal with the new problem of how to continue treatment as growth inevitably occurred, an expandable rib prosthesis was required. Thus, the concept of expansion thoracoplasty by rib distraction was born. This led to the development of a unique prosthetic rib implant—the vertically expandable prosthetic titanium rib (VEPTR). The current device (VEPTR II) is essentially two curvilinear sleeves sliding one within the other, the device can be sequentially expanded to produce an opening wedge

correction of the scoliosis from the concavity while providing chest wall stability for flail segments and volume expansion of the hypoplastic thorax in a patient with, for example, fused ribs. The device includes anchors on ribs via "cradles" capturing one or two consecutive ribs and has spine anchors consisting of hooks, screws, or an "S" rod for use on the iliac crest. Concomitant growth of the spine has been documented, even in patients with congenital unsegmented bars in whom previously it was assumed that growth of the concavity was impossible [114].

The expansion thoracoplasty technique has revolutionized the treatment of young children with congenital scoliosis and chest wall abnormalities, such as fused ribs. Formerly treatment of these children emphasized stopping the progressive deformity by early spinal fusion, but as discussed earlier, the pulmonary cost of an early fusion has been well documented, with follow-up studies identifying thoracic insufficiency syndrome because of lack of growth of the thorax [114, 165, 267, 361].

The ability to lengthen the thoracic spine and simultaneously correct the scoliosis without performing surgery on the spine itself is proposed as the major advantage of the expansion thoracoplasty technique and eventually became the justification for expanding the indications for VEPTR) use to *non-congenital* deformities.

Although parameters of thoracic cavity volume, such as coronal thoracic width and sagittal depth of the thorax [114] have been increased by the technique [35], actual improved pulmonary outcomes have been difficult to document, in part because of an inability to test young patients preoperatively. Since correlation between increases in anatomic volume and improvement in concomitant physiologic function tests (e.g., forced vital capacity, forced expiratory volume in 1 second) has been elusive, the decreased chest wall compliance inherently created by a rib anchoring device has raised significant questions about the overall value of the technique- especially in patients *without* a congenital chest wall deformity.

A comparison of VEPTR) to traditional spine-based growing rod (GRI) for treatment of *idiopathic* deformities showed inferior correction and more complications in the patients treated with VEPTR [43]. GRI patients achieved 50% curve correction compared to 27% for the VEPTR group. The GRI group gained 24% in thoracic height compared to 12% for the VEPTRs. GRI patients also had smaller curves at follow-up – 43 vs 60° for VEPTR – and much l kyphosis, 47 vs 64°. Finally, compared to the GRI group, VEPTR patients had a higher incidence of infection (41 vs 14%, p = .001) and double the rate of complications. Given the concerns over decreased chest wall compliance [170, 206] and the documented inferior deformity correction the use of chest wall expansion in patients who do not have primary chest wall anomalies is generally avoided.

Growth Guidance Constructs

The concept of applying segmental, non-rigid fixation to the growing spine, in order to "guide" growth by encouraging vertebrae to slide along rods, was first devised by Eduardo Luque in the 1980's [469]. Known as the Luque "trolley", he inserted smooth rods on each side of the spine, attaching them via sublaminar wires at each

vertebral segment without fusion, with excess rod left either at each end vertebra or in some cases having two U-shaped rods with the open ends of the "U" facing each other from cephalad and caudad, with apical overlap to allow the vertebrae to grow away from the apex along the guiding rods, thereby controlling the scoliosis. Due to the subperiosteal dissection required for the sublaminar wire passage, the eventual lengthening of the construct often was often unsatisfactory due to spontaneous fusion limiting the possible growth [486].

The concept was refined by Richard McCarthy, who after a visit to Seoul, South Korea in the early 2000s, developed a method using aggressive correction of just the apical segments of a scoliosis by segmental pedicle fixation and fusion (sometimes preceded by an anterior release/vertebrectomy to enhance correction), and then connecting the apical segments to "sliding" screws attached to upper and lower end vertebrae, which are instrumented percutaneously to minimize potential fusion at non-apical segments. Multiaxial flanged screws (called "Shilla" after a Korean icon) non-rigidly captured the end vertebrae to permit growth directed along the rods spanning the entire deformity [500]. McCarthy's 5 year results in 33 patients demonstrated a 74% reduction in the number of procedures compared to traditional growing rods. However, he also reported a 73% incidence of total complications including a 30% incidence of infection and a 70% incidence of implant-related complications requiring unplanned revision procedures. The eventual 5-year spine growth was about 8 cm (T1-S1), and deformity correction was 44%, with 19% loss of correction from the initial postoperative correction. Since its inception the Shilla operation has been performed at only a small number of centers. Recently a 5 year mean follow-up report of 21 patients from centers other than the originator's confirmed the high revision rate (30 surgeries for 15 patients) and a smaller length gained (6.8 cm) for T1-S1, with a 21% loss of correction in the post-implantation period [560]. Thus the Shilla concept remains in evolution as a reliable method to control deformity while appropriately addressing the continued growth (lengthening) of the spine and thorax.

Psychological Effects

It is important to recognize the psychological effects of treatment in EOS patients undergoing serial surgical procedures. The psychosocial effects of repetitive surgeries with respect to emotional and conduct problems, hyperactivity and attention deficit, and peer relations have been described [39, 493]. Abnormal psychosocial assessments are correlated with younger age at first growth-friendly surgery as well as the number of repetitive surgeries, while depression and generalized anxiety disorder have been found in 24–43% of early-onset surgical patients. While the development of the magnetic controlled growing rod (MCGR) is a significant response to this concern, early comparative reports on psychosocial function and other patient-reported domains have not found major improvement in the MCGR patient cohorts [186].

Summary

Several treatment methods are available and used with some efficacy for the myriad of types and complexities of early onset scoliosis. Most are still under investigation, due to the lack of long-term outcomes in "graduates. Serial casting and bracing are appropriate approaches for children without major chest wall deformities in an effort to either definitively treat or delay corrective surgery, with fusion ideally being delayed until around age 10. Larger and stiffer curves often require halo-gravity traction, followed by either a return to bracing, growth-friendly instrumentation if the child is immature, or definitive fusion to "lock-in" the correction achieved in traction. Growing rod constructs – traditional (GRI) or magnetically-controlled (MCGR) – are recommended for patients who have failed non-operative or delaying tactics. Expansion thoracoplasty is reserved for those with spinal deformities complicated by chest wall abnormalities that assume the primary pathology with potential of thoracic insufficiency syndrome. The primary goal in EOS treatment is to avoid early growth arrest by definitive fusion and facilitate spine/thoracic length increase while *correcting /controlling* the deformity.

References

1. Abdulian MH, Liu RW, Son-Hing JP, et al. Double rib penetration of the spinal canal in a patient with neurofibromatosis. J Pediatr Orthop. 2011;31(1):6–10.
2. Abel MF, Singla A, Feger MA, et al. Surgical treatment of Lenke 5 adolescent idiopathic scoliosis: comparison of anterior vs posterior approach. World J Orthop. 2016;7(9):553–60.
3. Abousamra O, Nishnianidze T, Rogers KJ, et al. Correction of pelvic obliquity after spinopelvic fixation in children with cerebral palsy: a comparison study with minimum two-year follow-up. Spine Deform. 2016;4(3):217–24.
4. Abousamra O, Nishnianidze T, Rogers KJ, et al. Risk factors for pancreatitis after posterior spinal fusion in children with cerebral palsy. J Pediatr Orthop B. 2018;27(2):163–7.
5. Abousamra O, Sullivan BT, Samdani AF, et al. Three methods of pelvic fixation for scoliosis in children with cerebral palsy: differences at 5-year follow-up. Spine (Phila Pa 1976). 2019;44(1):E19–25.
6. Abul-Kasim K, Ohlin A. The rate of screw misplacement in segmental pedicle screw fixation in adolescent idiopathic scoliosis. Acta Orthop. 2011;82(1):50–5.
7. Acaroglu E, Akel I, Alanay A, et al. Comparison of the melatonin and calmodulin in paravertebral muscle and platelets of patients with or without adolescent idiopathic scoliosis. Spine (Phila Pa 1976). 2009;34(18):E659–63.
8. Acaroglu E, Yazici M, Alanay A, et al. Three-dimensional evolution of scoliotic curve during instrumentation without fusion in young children. J Pediatr Orthop. 2002;22(4):492–6.
9. Acosta FL Jr, Thompson TL, Campbell S, et al. Use of intraoperative isocentric C-arm 3D fluoroscopy for sextant percutaneous pedicle screw placement: case report and review of the literature. Spine J. 2005;5(3):339–43.
10. Adams W. Lectures on the pathology and treatment of lateral and other forms of curvature of the spine. London: Churchill; 1865.
11. Ahl T, Albertsson-Wikland K, Kalen R. Twenty-four-hour growth hormone profiles in pubertal girls with idiopathic scoliosis. Spine. 1988;13(2):139–42.
12. Ahmad A, Subramanian T, Panteliadis P, et al. Quantifying the 'law of diminishing returns' in magnetically controlled growing rods. Bone Joint J. 2017;99-B(12):1658–64.

13. Ahn NU, Nallamshetty L, Ahn UM, et al. Dural ectasia and conventional radiography in the Marfan lumbosacral spine. Skelet Radiol. 2001;30(6):338–45.
14. Ahn NU, Sponseller PD, Ahn UM, et al. Dural ectasia is associated with back pain in Marfan syndrome. Spine. 2000;25(12):1562–8.
15. Akbarnia BA, Cheung K, Noordeen H, et al. Next generation of growth-sparing techniques: preliminary clinical results of a magnetically controlled growing rod in 14 patients with early-onset scoliosis. Spine (Phila Pa 1976). 2013;38(8):665–70.
16. Akbarnia BA, Marks DS, Boachie-Adjei O, et al. Dual growing rod technique for the treatment of progressive early-onset scoliosis: a multicenter study. Spine. 2005;30(17 Suppl):S46–57.
17. Akbarnia BA, Pawelek JB, Cheung KM, et al. Traditional growing rods versus magnetically controlled growing rods for the surgical treatment of early-onset scoliosis: a case-matched 2-year study. Spine Deform. 2014;2(6):493–7.
18. Akcali O, Alici E, Kosay C. Apical instrumentation alters the rotational correction in adolescent idiopathic scoliosis. Eur Spine J. 2003;12(2):124–9.
19. Albanese S. Idiopathic scoliosis: etiology and evaluation; natural history and nonsurgical management. In: Richards BS, editor. Orthopaedic knowledge update--pediatrics. Rosemont: American Academy of Orthopaedic Surgeons; 1996. p. 97.
20. Albert MC, LaFleur BC. Hybrid fixation with sublaminar polyester bands in the treatment of neuromuscular scoliosis: a comparative analysis. J Pediatr Orthop. 2015;35(2):172–7.
21. Alexander MA, Season EH. Idiopathic scoliosis: an electromyographic study. Arch Phys Med Rehabil. 1978;59(7):314–5.
22. Alman BA, Kim HK. Pelvic obliquity after fusion of the spine in Duchenne muscular dystrophy. J Bone Joint Surg Br. 1999;81(5):821–4.
23. Almansour H, Pepke W, Bruckner T, et al. Three-dimensional analysis of initial brace correction in the setting of adolescent idiopathic scoliosis. J Clin Med. 2019;8(11):1804.
24. Altiok H, Finlayson C, Hassani S, et al. Kyphectomy in children with myelomeningocele. Clin Orthop Relat Res. 2011;469(5):1272–8.
25. Anand N, Idio FG Jr, Remer S, et al. The effects of perioperative blood salvage and autologous blood donation on transfusion requirements in scoliosis surgery. J Spinal Disord. 1998;11(6):532–4.
26. Anciaux M, Lenaert A, Van Beneden ML, et al. Transcutaneous electrical stimulation (TCES) for the treatment of adolescent idiopathic scoliosis: preliminary results. Acta Orthop Belg. 1991;57(4):399–405.
27. Anderson AL, McIff TE, Asher MA, et al. The effect of posterior thoracic spine anatomical structures on motion segment flexion stiffness. Spine (Phila Pa 1976). 2009;34(5):441–6.
28. Ando K, Imagama S, Ito Z, et al. Cervical myelopathy caused by bilateral C1-2 dumbbell ganglioneuromas and C2-3 and C3-4 neurofibromas associated with neurofibromatosis type 1. J Orthop Sci. 2014;19(4):676–81.
29. Apel DM, Marrero G, King J, et al. Avoiding paraplegia during anterior spinal surgery. The role of somatosensory evoked potential monitoring with temporary occlusion of segmental spinal arteries. Spine. 1991;16(8 Suppl):S365–70.
30. Aprin H, Bowen JR, MacEwen GD, et al. Spine fusion in patients with spinal muscular atrophy. J Bone Joint Surg Am. 1982;64(8):1179–87.
31. Arms DM, Smith JT, Osteyee J, et al. Postoperative epidural analgesia for pediatric spine surgery. Orthopedics. 1998;21(5):539–44.
32. Aronsson DD, Stokes IA, Ronchetti PJ, et al. Comparison of curve shape between children with cerebral palsy, Friedreich's ataxia, and adolescent idiopathic scoliosis. Dev Med Child Neurol. 1994;36(5):412–8.
33. Arun R, Srinivas S, Mehdian SM. Scoliosis in Duchenne's muscular dystrophy: a changing trend in surgical management: a historical surgical outcome study comparing sublaminar, hybrid and pedicle screw instrumentation systems. Eur Spine J. 2010;19(3):376–83.
34. Asazuma T, Hashimoto T, Masuoka K, et al. Acute thoracic myelopathy after a traumatic episode in a patient with neurofibromatosis associated with sharply angular scoliosis: a case report. J Orthop Sci. 2003;8(5):721–4.

35. Asher M, Green P, Orrick J. A six-year report: spinal deformity screening in Kansas school children. J Kans Med Soc. 1980;81(12):568–71.
36. Asher MA, Burton DC. Adolescent idiopathic scoliosis: natural history and long term treatment effects. Scoliosis. 2006;1(1):2.
37. Asher MA, Lai SM, Burton DC. Analysis of instrumentation/fusion survivorship without reoperation after primary posterior multiple anchor instrumentation and arthrodesis for idiopathic scoliosis. Spine J. 2010;10(1):5–15.
38. Asher MA, Strippgen WE, Heinig CF, et al. Isola spinal implant system. Semin Spine Surg. 1992;(4):175.
39. Aslan C, Olgun ZD, Ertas ES, et al. Psychological profile of children who require repetitive surgical procedures for early onset scoliosis: is a poorer quality of life the cost of a straighter spine? Spine Deform. 2017;5(5):334–41.
40. Auerbach JD, Lenke LG, Bridwell KH, et al. Major complications and comparison between 3-column osteotomy techniques in 105 consecutive spinal deformity procedures. Spine (Phila Pa 1976). 2012;37(14):1198–210.
41. Aulisa AG, Guzzanti V, Falciglia F, et al. Lyon bracing in adolescent females with thoracic idiopathic scoliosis: a prospective study based on SRS and SOSORT criteria. BMC Musculoskelet Disord. 2015;16:316.
42. Aurori BF, Weierman RJ, Lowell HA, et al. Pseudarthrosis after spinal fusion for scoliosis. A comparison of autogeneic and allogeneic bone grafts. Clin Orthop Relat Res. 1985;(199):153–8.
43. Bachabi M, McClung A, Pawelek JB, et al. Idiopathic early-onset scoliosis: growing rods versus vertically expandable prosthetic titanium ribs at 5-year follow-up. J Pediatr Orthop. 2020;40(3):142–8.
44. Bagnall K, Raso VJ, Moreau M, et al. The effects of melatonin therapy on the development of scoliosis after pinealectomy in the chicken. J Bone Joint Surg Am. 1999;81(2):191–9.
45. Bagnall KM, Beuerlein M, Johnson P, et al. Pineal transplantation after pinealectomy in young chickens has no effect on the development of scoliosis. Spine. 2001;26(9):1022–7.
46. Bai J, Chen K, Wei Q, et al. Selecting the LSTV as the lower instrumented vertebra in the treatment of Lenke types 1A and 2A adolescent idiopathic scoliosis-a minimal 3-year follow-up. Spine (Phila Pa 1976). 2017;43(7):E390–8.
47. Banta JV. Combined anterior and posterior fusion for spinal deformity in myelomeningocele. Spine. 1990;15(9):946–52.
48. Banta JV, Park SM. Improvement in pulmonary function in patients having combined anterior and posterior spine fusion for myelomeningocele scoliosis. Spine. 1983;8(7):765–70.
49. Barr SJ, Schuette AM, Emans JB. Lumbar pedicle screws versus hooks. Results in double major curves in adolescent idiopathic scoliosis. Spine. 1997;22(12):1369–79.
50. Barrack RL, Wyatt MP, Whitecloud TS 3rd, et al. Vibratory hypersensitivity in idiopathic scoliosis. J Pediatr Orthop. 1988;8(4):389–95.
51. Bartley CE, Yaszay B, Bastrom TP, et al. Perioperative and delayed Major complications following surgical treatment of adolescent idiopathic scoliosis. J Bone Joint Surg Am. 2017;99(14):1206–12.
52. Bashiardes S, Veile R, Allen M, et al. SNTG1, the gene encoding gamma1-syntrophin: a candidate gene for idiopathic scoliosis. Hum Genet. 2004;115(1):81–9.
53. Bashiardes S, Veile R, Wise CA, et al. Positional cloning strategies for idiopathic scoliosis. Stud Health Technol Inform. 2002;91:86–9.
54. Basu PS, Elsebaie H, Noordeen MH. Congenital spinal deformity: a comprehensive assessment at presentation. Spine. 2002;27(20):2255–9.
55. Basu S, Tikoo A. Posterior hemivertebrectomy and short segment fixation-long term results. Childs Nerv Syst. 2017;33(2):321–8.
56. Beals RK, Robbins JR, Rolfe B. Anomalies associated with vertebral malformations. Spine. 1993;18(10):1329–32.
57. Belmont PJ Jr, Klemme WR, Robinson M, et al. Accuracy of thoracic pedicle screws in patients with and without coronal plane spinal deformities. Spine. 2002;27(14):1558–66.

58. Belmont PJ Jr, Kuklo TR, Taylor KF, et al. Intraspinal anomalies associated with isolated congenital hemivertebra: the role of routine magnetic resonance imaging. J Bone Joint Surg Am. 2004;86-A(8):1704–10.
59. Berg AO. Screening for adolescent idiopathic scoliosis: a report from the United States preventive services task force. J Am Board Fam Pract. 1993;6(5):497–501.
60. Bergofsky EH, Turino GM, Fishman AP. Cardio-respiratory failure in kypho-scoliosis. Medicine. 1959;36:456.
61. Bernard JM, Pereon Y, Fayet G, et al. Effects of isoflurane and desflurane on neurogenic motor- and somatosensory-evoked potential monitoring for scoliosis surgery. Anesthesiology. 1996;85(5):1013–9.
62. Berned Muller E, Nordwall A, von Wendt L. The influence of scoliosis brace treatment on function in children with myelomeningocele. Acta Paediatr. 1992;81:925.
63. Bertrand SL, Drvaric DM, Lange N, et al. Electrical stimulation for idiopathic scoliosis. Clin Orthop Relat Res. 1992;(276):176–81.
64. Bess S, Akbarnia BA, Thompson GH, et al. Complications of growing-rod treatment for early-onset scoliosis: analysis of one hundred and forty patients. J Bone Joint Surg Am. 2010;92(15):2533–43.
65. Bettany-Saltikov J, Parent E, Romano M, et al. Physiotherapeutic scoliosis-specific exercises for adolescents with idiopathic scoliosis. Eur J Phys Rehabil Med. 2014;50(1):111–21.
66. Betz RR, Clements DH, et al. Anterior instrumentation for thoracic idiopathic scoliosis. Semin Spine Surg. 1997;9:141.
67. Beuerlein M, Wilson J, Moreau M, et al. The critical stage of pinealectomy surgery after which scoliosis is produced in young chickens. Spine. 2001;26(3):237–40.
68. Bharucha NJ, Lonner BS, Auerbach JD, et al. Low-density versus high-density thoracic pedicle screw constructs in adolescent idiopathic scoliosis: do more screws lead to a better outcome? Spine J. 2013;13(4):375–81.
69. Birch JG, Herring JA. Spinal deformity in Marfan syndrome. J Pediatr Orthop. 1987;7(5):546–52.
70. Bitan F, Rigault P, Houfani B, et al. Scoliosis and congenital heart diseases in children. Apropos of 44 cases. Rev Chir Orthop Reparatrice Appar Mot. 1991;77(3):179–88.
71. Bitterman AD, Sponseller PD. Marfan syndrome: a clinical update. J Am Acad Orthop Surg. 2017;25(9):603–9.
72. Blanco JS, Sears CJ. Allograft bone use during instrumentation and fusion in the treatment of adolescent idiopathic scoliosis. Spine. 1997;22(12):1338–42.
73. Boachie-Adjei O, Lonstein JE, Winter RB, et al. Management of neuromuscular spinal deformities with Luque segmental instrumentation. J Bone Joint Surg Am. 1989;71(4):548–62.
74. Boemers TM, Soorani-Lunsing IJ, de Jong TP, et al. Urological problems after surgical treatment of scoliosis in children with myelomeningocele. J Urol. 1996;155(3):1066–9.
75. Bohl DD, Telles CJ, Golinvaux NS, et al. Effectiveness of Providence nighttime bracing in patients with adolescent idiopathic scoliosis. Orthopedics. 2014;37(12):e1085–90.
76. Bohtz C, Meyer-Heim A, Min K. Changes in health-related quality of life after spinal fusion and scoliosis correction in patients with cerebral palsy. J Pediatr Orthop. 2011;31(6):668–73.
77. Bollini G, Docquier PL, Viehweger E, et al. Lumbar hemivertebra resection. J Bone Joint Surg Am. 2006;88(5):1043–52.
78. Borkhuu B, Borowski A, Shah SA, et al. Antibiotic-loaded allograft decreases the rate of acute deep wound infection after spinal fusion in cerebral palsy. Spine (Phila Pa 1976). 2008;33(21):2300–4.
79. Borkhuu B, Nagaraju D, Miller F, et al. Prevalence and risk factors in postoperative pancreatitis after spine fusion in patients with cerebral palsy. J Pediatr Orthop. 2009;29(3):256–62.
80. Bosch B, Heimkes B, Stotz S. Course and prognosis of congenital scoliosis. Z Orthop Ihre Grenzgeb. 1994;132(5):363–70.
81. Boylan KB, Cornblath DR, Glass JD, et al. Autosomal dominant distal spinal muscular atrophy in four generations. Neurology. 1995;45(4):699–704.

82. Bradford DS. Anterior vascular pedicle bone grafting for the treatment of kyphosis. Spine. 1980;5(4):318–23.
83. Bradford DS, Boachie-Adjei O. One-stage anterior and posterior hemivertebral resection and arthrodesis for congenital scoliosis. J Bone Joint Surg Am. 1990;72(4):536–40.
84. Bradford DS, Daher YH. Vascularised rib grafts for stabilisation of kyphosis. J Bone Joint Surg Br. 1986;68(3):357–61.
85. Bradford DS, Heithoff KB, Cohen M. Intraspinal abnormalities and congenital spine deformities: a radiographic and MRI study. J Pediatr Orthop. 1991;11(1):36–41.
86. Bradford DS, Tribus CB. Vertebral column resection for the treatment of rigid coronal decompensation. Spine (Phila Pa 1976). 1997;22(14):1590–9.
87. Branthwaite MA. Cardiorespiratory consequences of unfused idiopathic scoliosis. Br J Dis Chest. 1986;80(4):360–9.
88. Braun JT, Ogilvie JW, Akyuz E, et al. Fusionless scoliosis correction using a shape memory alloy staple in the anterior thoracic spine of the immature goat. Spine (Phila Pa 1976). 2004;29(18):1980–9.
89. Bridwell KH, Baldus C, Iffrig TM, et al. Process measures and patient/parent evaluation of surgical management of spinal deformities in patients with progressive flaccid neuromuscular scoliosis (Duchenne's muscular dystrophy and spinal muscular atrophy). Spine. 1999;24(13):1300–9.
90. Bridwell KH, McAllister JW, Betz RR, et al. Coronal decompensation produced by Cotrel-Dubousset “derotation” maneuver for idiopathic right thoracic scoliosis. Spine. 1991;16(7):769–77.
91. Brodner W, Krepler P, Nicolakis M, et al. Melatonin and adolescent idiopathic scoliosis. J Bone Joint Surg Br. 2000;82(3):399–403.
92. Brown JC, Zeller JL, Swank SM, et al. Surgical and functional results of spine fusion in spinal muscular atrophy. Spine. 1989;14(7):763–70.
93. Brzustowicz LM, Lehner T, Castilla LH, et al. Genetic mapping of chronic childhood-onset spinal muscular atrophy to chromosome 5q11.2-13.3. Nature. 1990;344(6266):540–1.
94. Buckner JMH. A longitudinal study of adolescent growth. London: Springer; 1990.
95. Buckwalter JA, Yaszay B, Ilgenfritz RM, et al. Analysis of intraoperative neuromonitoring events during spinal corrective surgery for idiopathic scoliosis. Spine Deform. 2013;1(6):434–8.
96. Budd HR, Stokes OM, Meakin J, et al. Safety and compatibility of magnetic-controlled growing rods and magnetic resonance imaging. Eur Spine J. 2016;25(2):578–82.
97. Bunnell WP. An objective criterion for scoliosis screening. J Bone Joint Surg Am. 1984;66(9):1381–7.
98. Bunnell WP. The natural history of idiopathic scoliosis before skeletal maturity. Spine. 1986;11(8):773–6.
99. Bunnell WP. Outcome of spinal screening. Spine. 1993;18(12):1572–80.
100. Bunnell WP, MacEwen GD, Jayakumar S. The use of plastic jackets in the non-operative treatment of idiopathic scoliosis. Preliminary report. J Bone Joint Surg Am. 1980;62(1):31–8.
101. Burton DC, Asher MA, Lai SM. Scoliosis correction maintenance in skeletally immature patients with idiopathic scoliosis. Is anterior fusion really necessary? Spine. 2000;25(1):61–8.
102. Burton DC, Asher MA, Lai SM. Patient-based outcomes analysis of patients with single torsion thoracolumbar-lumbar scoliosis treated with anterior or posterior instrumentation: an average 5- to 9-year follow-up study. Spine. 2002;27(21):2363–7.
103. Burton DC, Carlson BB, Place HM, et al. Results of the Scoliosis Research Society morbidity and mortality database 2009-2012: a report from the morbidity and mortality committee. Spine Deform. 2016;4(5):338–43.
104. Bush CH, Kalen V. Three-dimensional computed tomography in the assessment of congenital scoliosis. Skelet Radiol. 1999;28(11):632–7.
105. Bushby K, Finkel R, Birnkrant DJ, et al. Diagnosis and management of Duchenne muscular dystrophy, part 2: implementation of multidisciplinary care. Lancet Neurol. 2010;9(2):177–89.

106. Byers RK, Banker BQ. Infantile muscular atrophy: an eleven year experience. Trans Am Neurol Assoc. 1960;85:10–4.
107. Byers RK, Banker BQ. Infantile muscular atrophy. Arch Neurol. 1961;5:140–64.
108. Byrd JA 3rd. Current theories on the etiology of idiopathic scoliosis. Clin Orthop Relat Res. 1988;(229):114–9.
109. Cahill PJ, Marvil S, Cuddihy L, et al. Autofusion in the immature spine treated with growing rods. Spine (Phila Pa 1976). 2010;35(22):E1199–203.
110. Cai S, Zhang J, Shen J, et al. Posterior correction without rib-head resection for patients with Neurofibromatosis type 1, dystrophic scoliosis, and rib-head protrusion into the Spinal Canal. Clin Spine Surg. 2017;30(1):32–7.
111. Callahan BC, Georgopoulos G, Eilert RE. Hemivertebral excision for congenital scoliosis. J Pediatr Orthop. 1997;17(1):96–9.
112. Calvert PT, Edgar MA, Webb PJ. Scoliosis in neurofibromatosis. The natural history with and without operation. J Bone Joint Surg Br. 1989;71(2):246–51.
113. Cambridge W, Drennan JC. Scoliosis associated with Duchenne muscular dystrophy. J Pediatr Orthop. 1987;7(4):436–40.
114. Campbell RM Jr, Hell-Vocke AK. Growth of the thoracic spine in congenital scoliosis after expansion thoracoplasty. J Bone Joint Surg Am. 2003;85-A(3):409–20.
115. Campbell RM Jr, Smith MD, Hell-Vocke AK. Expansion thoracoplasty: the surgical technique of opening-wedge thoracostomy. Surgical technique. J Bone Joint Surg Am. 2004;86-A(Suppl 1):51–64.
116. Campbell RM Jr, Smith MD, Mayes TC, et al. The characteristics of thoracic insufficiency syndrome associated with fused ribs and congenital scoliosis. J Bone Joint Surg Am. 2003;85-A(3):399–408.
117. Canavese F, Charles YP, Samba A, et al. Safety and efficacy of sublaminar bands and Ponte osteotomies in rigid deformity: preliminary results in a prospective series of 20 neuromuscular scoliosis patients. J Pediatr Orthop B. 2017;26(3):233–9.
118. Carman DL, Browne RH, Birch JG. Measurement of scoliosis and kyphosis radiographs. Intraobserver and interobserver variation. J Bone Joint Surg Am. 1990;72(3):328–33.
119. Carreon LY, Puno RM, Lenke LG, et al. Non-neurologic complications following surgery for adolescent idiopathic scoliosis. J Bone Joint Surg Am. 2007;89(11):2427–32.
120. Carstens C, Koch H, Brocai DR, et al. Development of pathological lumbar kyphosis in myelomeningocele. J Bone Joint Surg Br. 1996;78(6):945–50.
121. Carstens C, Paul K, Niethard FU, et al. Effect of scoliosis surgery on pulmonary function in patients with myelomeningocele. J Pediatr Orthop. 1991;11(4):459–64.
122. Cassidy C, Craig CL, Perry A, et al. A reassessment of spinal stabilization in severe cerebral palsy. J Pediatr Orthop. 1994;14(6):731–9.
123. Chalkiadis GA, Branch KG. Cardiac arrest after isoflurane anaesthesia in a patient with Duchenne's muscular dystrophy. Anaesthesia. 1990;45(1):22–5.
124. Chan A, Parent E, Narvacan K, et al. Intraoperative image guidance compared with free-hand methods in adolescent idiopathic scoliosis posterior spinal surgery: a systematic review on screw-related complications and breach rates. Spine J. 2017;17(9):1215–29.
125. Chan V, Fong GC, Luk KD, et al. A genetic locus for adolescent idiopathic scoliosis linked to chromosome 19p13.3. Am J Hum Genet. 2002;71(2):401–6.
126. Chandran S, McCarthy J, Noonan K, et al. Early treatment of scoliosis with growing rods in children with severe spinal muscular atrophy: a preliminary report. J Pediatr Orthop. 2011;31(4):450–4.
127. Chang DG, Kim JH, Ha KY, et al. Posterior hemivertebra resection and short segment fusion with pedicle screw fixation for congenital scoliosis in children younger than 10 years: greater than 7-year follow-up. Spine (Phila Pa 1976). 2015;40(8):E484–91.
128. Charles YP, Canavese F, Dimeglio A. Curve progression risk in a mixed series of braced and nonbraced patients with idiopathic scoliosis related to skeletal maturity assessment on the olecranon. J Pediatr Orthop B. 2017;26(3):240–4.

129. Charry O, Koop S, Winter R, et al. Syringomyelia and scoliosis: a review of twenty-five pediatric patients. J Pediatr Orthop. 1994;14(3):309–17.
130. Cheliout-Heraut F, Daunois O, Pouliquen JC, et al. Value of somatosensory evoked potentials in spinal surgery monitoring in children and adolescents: 110 cases. Neurophysiol Clin. 1993;23(2–3):163–78.
131. Cheliout-Heraut F, Mariambourg G, Fahed M, et al. Contribution of somatosensory evoked potentials in the surveillance of the spinal cord during spinal surgery. Rev Chir Orthop Reparatrice Appar Mot. 1991;77(5):344–52.
132. Chen J, Yang C, Ran B, et al. Correction of Lenke 5 adolescent idiopathic scoliosis using pedicle screw instrumentation: does implant density influence the correction? Spine (Phila Pa 1976). 2013;38(15):E946–51.
133. Chen ZY, Wong HK, Chan YH. Variability of somatosensory evoked potential monitoring during scoliosis surgery. J Spinal Disord Tech. 2004;17(6):470–6.
134. Cheng JC, Guo X, Sher AH. Persistent osteopenia in adolescent idiopathic scoliosis. A longitudinal follow up study. Spine. 1999;24(12):1218–22.
135. Cheung KM, Cheung JP, Samartzis D, et al. Magnetically controlled growing rods for severe spinal curvature in young children: a prospective case series. Lancet. 2012;379(9830):1967–74.
136. Cheung KM, Lu DS, Poon AM, et al. Effect of melatonin suppression on scoliosis development in chickens by either constant light or surgical pinealectomy. Spine. 2003;28(17):1941–4.
137. Cho SK, Kim YJ, Lenke LG. Proximal junctional kyphosis following spinal deformity surgery in the Pediatric patient. J Am Acad Orthop Surg. 2015;23(7):408–14.
138. Choudhry DK, Brenn BR, Sacks K, et al. Evaluation of gabapentin and clonidine use in children following spinal fusion surgery for idiopathic scoliosis: a retrospective review. J Pediatr Orthop. 2019;39(9):e687–93.
139. Christodoulou AG, Ploumis A, Terzidis IP, et al. Spleen rupture after surgery in Marfan syndrome scoliosis. J Pediatr Orthop. 2004;24(5):537–40.
140. Christofersen MR, Brooks AL. Excision and wire fixation of rigid myelomeningocele kyphosis. J Pediatr Orthop. 1985;5(6):691–6.
141. Chugh S, Marks DS, Mangham DC, et al. Autologous bone grafting in staged scoliosis surgery. The patient as bone bank. Spine. 1998;23(16):1793–5.
142. Clark CE, Shufflebarger HL. Late-developing infection in instrumented idiopathic scoliosis. Spine. 1999;24(18):1909–12.
143. Clements DH, Betz RR, Newton PO, et al. Correlation of scoliosis curve correction with the number and type of fixation anchors. Spine (Phila Pa 1976). 2009;34(20):2147–50.
144. Cobetto N, Aubin CE, Clin J, et al. Braces optimized with computer-assisted design and simulations are lighter, more comfortable, and more efficient than plaster-cast braces for the treatment of adolescent idiopathic scoliosis. Spine Deform. 2014;2(4):276–84.
145. Coe JD, Arlet V, Donaldson W, et al. Complications in spinal fusion for adolescent idiopathic scoliosis in the new millennium. A report of the Scoliosis Research Society morbidity and mortality committee. Spine (Phila Pa 1976). 2006;31(3):345–9.
146. Colbert AP, Craig C. Scoliosis management in Duchenne muscular dystrophy: prospective study of modified Jewett hyperextension brace. Arch Phys Med Rehabil. 1987;68(5 Pt 1):302–4.
147. Cole JW, Murray DJ, Snider RJ, et al. Aprotinin reduces blood loss during spinal surgery in children. Spine. 2003;28(21):2482–5.
148. Comstock SA, Cook PC, Leahey JL, et al. Posterior kyphectomy for myelomeningocele with anterior placement of fixation: a retrospective review. Clin Orthop Relat Res. 2011;469(5):1265–71.
149. Copley LA, Richards BS, Safavi FZ, et al. Hemodilution as a method to reduce transfusion requirements in adolescent spine fusion surgery. Spine. 1999;24(3):219–22; discussion 223–214.
150. Cordover AM, Betz RR, Clements DH, et al. Natural history of adolescent thoracolumbar and lumbar idiopathic scoliosis into adulthood. J Spinal Disord. 1997;10(3):193–6.

151. Corona J, Matsumoto H, Roye DP, et al. Measuring quality of life in children with early onset scoliosis: development and initial validation of the early onset scoliosis questionnaire. J Pediatr Orthop. 2011;31(2):180–5.
152. Cote P, Cassidy JD. Re: a study of the diagnostic accuracy and reliability of the scoliometer and Adam's forward bend test (spine. 1999;23:796–802). Spine. 1999;24(22):2411–2.
153. Cowell HR, Hall JN, MacEwen GD. Genetic aspects of idiopathic scoliosis. A Nicholas Andry award essay, 1970. Clin Orthop Relat Res. 1972;86:121–31.
154. Cowell HR, MacEwen GD, Hubben C. Incidence of abnormalities of the kidney and ureter in congenital scoliosis. Birth Defects Orig Artic Ser. 1974;10(4):142–5.
155. Crawford AH. Pitfalls of spinal deformities associated with neurofibromatosis in children. Clin Orthop Relat Res. 1989;(245):29–42.
156. Crawford AH. Neurofibromatosis. In: Weinstein SL, editor. The pediatric spine: principles and practice. New York: Raven Press; 1994. p. 619.
157. Crawford CH 3rd, Lenke LG. Growth modulation by means of anterior tethering resulting in progressive correction of juvenile idiopathic scoliosis: a case report. J Bone Joint Surg Am. 2010;92(1):202–9.
158. Cunningham BW, Orbegoso CM, Dmitriev AE, et al. The effect of titanium particulate on development and maintenance of a posterolateral spinal arthrodesis: an in vivo rabbit model. Spine. 2002;27(18):1971–81.
159. D'Amato CR, Griggs S, McCoy B. Nighttime bracing with the Providence brace in adolescent girls with idiopathic scoliosis. Spine. 2001;26(18):2006–12.
160. Dalal A, Upasani VV, Bastrom TP, et al. Apical vertebral rotation in adolescent idiopathic scoliosis: comparison of uniplanar and polyaxial pedicle screws. J Spinal Disord Tech. 2011;24(4):251–7.
161. Danielsson AJ. Natural history of adolescent idiopathic scoliosis: a tool for guidance in decision of surgery of curves above 50 degrees. J Child Orthop. 2013;7(1):37–41.
162. Daruwalla JS, Balasubramaniam P, Chay SO, et al. Idiopathic scoliosis. Prevalence and ethnic distribution in Singapore schoolchildren. J Bone Joint Surg Br. 1985;67(2):182–4.
163. Daubs MD. Sagittal alignment changes and proximal junctional kyphosis in adolescent idiopathic scoliosis. Spine J. 2016;16(6):784–5.
164. Davis CM, Grant CA, Pearcy MJ, et al. Is there asymmetry between the concave and convex pedicles in adolescent idiopathic scoliosis? A CT investigation. Clin Orthop Relat Res. 2017;475(3):884–93.
165. Day GA, Upadhyay SS, Ho EK, et al. Pulmonary functions in congenital scoliosis. Spine. 1994;19(9):1027–31.
166. De Giorgi S, Piazzolla A, Tafuri S, et al. Cheneau brace for adolescent idiopathic scoliosis: long-term results. Can it prevent surgery? Eur Spine J. 2013;22(Suppl 6):S815–22.
167. de Kleuver M, van Jonbergen JP, Langeloo DD. Asymptomatic massive dural ectasia associated with neurofibromatosis type 1 threatening spinal column support: treatment by anterior vascularized fibula graft. J Spinal Disord Tech. 2004;17(6):539–42.
168. De la Garza Ramos R, Goodwin CR, Abu-Bonsrah N, et al. Patient and operative factors associated with complications following adolescent idiopathic scoliosis surgery: an analysis of 36, 335 patients from the Nationwide inpatient sample. J Neurosurg Pediatr. 2016;25(6):730–6.
169. de Mendonca RG, Sawyer JR, Kelly DM. Complications after surgical treatment of adolescent idiopathic scoliosis. Orthop Clin North Am. 2016;47(2):395–403.
170. Dede O, Motoyama EK, Yang CI, et al. Pulmonary and radiographic outcomes of VEPTR (vertical expandable prosthetic titanium rib) treatment in early-onset scoliosis. J Bone Joint Surg Am. 2014;96(15):1295–302.
171. Delikaris A, Wang X, Boyer L, et al. Implant density at the apex is more important than overall implant density for 3D correction in thoracic adolescent idiopathic scoliosis using rod Derotation and En bloc vertebral Derotation technique. Spine (Phila Pa 1976). 2018;43(11):E639–47.
172. Demetracopoulos CA, Sponseller PD. Spinal deformities in Marfan syndrome. Orthop Clin North Am. 2007;38(4):563–72, vii.

173. Deviren V, Berven S, Smith JA, et al. Excision of hemivertebrae in the management of congenital scoliosis involving the thoracic and thoracolumbar spine. J Bone Joint Surg Br. 2001;83(4):496–500.
174. Dhawale AA, Shah SA, Sponseller PD, et al. Are antifibrinolytics helpful in decreasing blood loss and transfusions during spinal fusion surgery in children with cerebral palsy scoliosis? Spine (Phila Pa 1976). 2012;37(9):E549–55.
175. Di Pauli von Treuheim T, Li DT, Mikhail C, et al. Reliable skeletal maturity assessment for an AIS patient cohort: external validation of the proximal humerus ossification system (PHOS) and relevant learning methodology. Spine Deform. 2020;8(4):613–20.
176. Di Silvestre M, Greggi T, Giacomini S, et al. Surgical treatment for scoliosis in Marfan syndrome. Spine. 2005;30(20):E597–604.
177. Di Silvestre M, Parisini P, Lolli F, et al. Complications of thoracic pedicle screws in scoliosis treatment. Spine (Phila Pa 1976). 2007;32(15):1655–61.
178. Diab M, Landman Z, Lubicky J, et al. Use and outcome of MRI in the surgical treatment of adolescent idiopathic scoliosis. Spine (Phila Pa 1976). 2011;36(8):667–71.
179. Dias MS, Li V, Landi M, et al. The embryogenesis of congenital vertebral dislocation: early embryonic buckling? Pediatr Neurosurg. 1998;29(6):281–9.
180. Dias RC, Miller F, Dabney K, et al. Surgical correction of spinal deformity using a unit rod in children with cerebral palsy. J Pediatr Orthop. 1996;16(6):734–40.
181. DiCindio S, Theroux M, Shah S, et al. Multimodality monitoring of transcranial electric motor and somatosensory-evoked potentials during surgical correction of spinal deformity in patients with cerebral palsy and other neuromuscular disorders. Spine (Phila Pa 1976). 2003;28(16):1851–5; discussion 1855–1856.
182. DiFazio RL, Miller PE, Vessey JA, et al. Health-related quality of life and care giver burden following spinal fusion in children with cerebral palsy. Spine (Phila Pa 1976). 2017;42(12):E733–9.
183. Dimeglio A, Bonnel F. Le rachis en croissance. Paris: Springer; 1990.
184. Djurasovic M, Glassman SD, Sucato DJ, et al. Improvement in SRS22R pain scores after surgery for adolescent idiopathic scoliosis. Spine (Phila Pa 1976). 2016;16(10):S343–4.
185. Do T, Fras C, Burke S, et al. Clinical value of routine preoperative magnetic resonance imaging in adolescent idiopathic scoliosis. A prospective study of three hundred and twenty-seven patients. J Bone Joint Surg Am. 2001;83-A(4):577–9.
186. Doany ME, Olgun ZD, Kinikli GI, et al. Health-related quality of life in early-onset scoliosis patients treated surgically: EOSQ scores in traditional growing rod versus magnetically controlled growing rods. Spine (Phila Pa 1976). 2018;43(2):148–53.
187. Dodd CA, Fergusson CM, Freedman L, et al. Allograft versus autograft bone in scoliosis surgery. J Bone Joint Surg Br. 1988;70(3):431–4.
188. Doers T, Walker JL, van den Brink K, et al. The progression of untreated lumbar kyphosis and the compensatory thoracic lordosis in myelomeningocele. Dev Med Child Neurol. 1997;39(5):326–30.
189. Dohin B, Dubousset JF. Prevention of the crankshaft phenomenon with anterior spinal epiphysiodesis in surgical treatment of severe scoliosis of the younger patient. Eur Spine J. 1994;3(3):165–8.
190. Dong Y, Weng X, Zhao H, et al. Lenke 5C curves in adolescent idiopathic scoliosis: anterior vs posterior selective fusion. Neurosurgery. 2016;78(3):324–31.
191. Dormans JP, Templeton JJ, Edmonds C, et al. Intraoperative anaphylaxis due to exposure to latex (natural rubber) in children. J Bone Joint Surg Am. 1994;76(11):1688–91.
192. Drvaric DM, Roberts JM, Burke SW, et al. Gastroesophageal evaluation in totally involved cerebral palsy patients. J Pediatr Orthop. 1987;7(2):187–90.
193. Drvaric DM, Schmitt EW, Nakano JM. The Grice extra-articular subtalar arthrodesis in the treatment of spastic hindfoot valgus deformity. Dev Med Child Neurol. 1989;31(5):665–9.
194. Dubory A, Miladi L, Ilharreborde B, et al. Cobb-1 versus cobb-to-cobb anterior fusion for adolescent idiopathic scoliosis Lenke 5C curves: a radiological comparative study. Eur Spine J. 2017;26(6):1711–20.

195. Dubousset J. Congenital kyphosis and lordosis. In: Weinstein SL, editor. The pediatric spine: principles and practice. New York: Raven Press; 1994. p. 245.
196. Dubousset J, Herring JA, Shufflebarger H. The crankshaft phenomenon. J Pediatr Orthop. 1989;9(5):541–50.
197. Dubousset J, Shufflebarger H, Wenger DR. Late infection with CD instrumentation. Orthop Trans. 1994;(18):121.
198. Duport G, Gayet E, Pries P, et al. Spinal deformities and wheelchair seating in Duchenne muscular dystrophy: twenty years of research and clinical experience. Semin Neurol. 1995;15(1):29–37.
199. Durham JW, Moskowitz A, Whitney J. Surface electrical stimulation versus brace in treatment of idiopathic scoliosis. Spine. 1990;15(9):888–92.
200. Dwyer AF, Newton NC, Sherwood AA. An anterior approach to scoliosis. A preliminary report. Clin Orthop Relat Res. 1969;62:192–202.
201. Eckford SD, Westgate J. Solitary crossed renal ectopia associated with unicornuate uterus, imperforate anus and congenital scoliosis. J Urol. 1996;156(1):221.
202. Eckstein HB, Vora RM. Spinal osteotomy for severe kyphosis in children with myelomeningocele. J Bone Joint Surg Br. 1972;54(2):328–33.
203. Edvardsen P. Physeo-epiphyseal injuries of lower extremities in myelomeningocele. Acta Orthop Scand. 1972;43(6):550–7.
204. Ekatodramis G, Min K, Cathrein P, et al. Use of a double epidural catheter provides effective postoperative analgesia after spine deformity surgery. Can J Anaesth. 2002;49(2):173–7.
205. Eldeeb H, Boubekri N, Asfour S, et al. Design of thoracolumbosacral orthosis (TLSO) braces using CT/MR. J Comput Assist Tomogr. 2001;25(6):963–70.
206. Emans JB, Caubet JF, Ordonez CL, et al. The treatment of spine and chest wall deformities with fused ribs by expansion thoracostomy and insertion of vertical expandable prosthetic titanium rib: growth of thoracic spine and improvement of lung volumes. Spine. 2005;30(17 Suppl):S58–68.
207. Enercan M, Kahraman S, Cobanoglu M, et al. Selective thoracic fusion provides similar health-related quality of life but can cause more lumbar disc and facet joint degeneration: a comparison of adolescent idiopathic scoliosis patients with Normal population 10 years after surgery. Spine Deform. 2015;3(5):469–75.
208. Erol B, Tracy MR, Dormans JP, et al. Congenital scoliosis and vertebral malformations: characterization of segmental defects for genetic analysis. J Pediatr Orthop. 2004;24(6):674–82.
209. Evans GA, Drennan JC, Russman BS. Functional classification and orthopaedic management of spinal muscular atrophy. J Bone Joint Surg Br. 1981;63B(4):516–22.
210. Evans SC, Edgar MA, Hall-Craggs MA, et al. MRI of 'idiopathic' juvenile scoliosis. A prospective study. J Bone Joint Surg Br. 1996;78(2):314–7.
211. Fabry G. Allograft versus autograft bone in idiopathic scoliosis surgery: a multivariate statistical analysis. J Pediatr Orthop. 1991;11(4):465–8.
212. Facanha-Filho FA, Winter RB, Lonstein JE, et al. Measurement accuracy in congenital scoliosis. J Bone Joint Surg Am. 2001;83-A(1):42–5.
213. Faciszewski T, Winter RB, Lonstein JE, et al. Segmental spinal dysgenesis. A disorder different from spinal agenesis. J Bone Joint Surg Am. 1995;77(4):530–7.
214. Fagan AB, Kennaway DJ, Sutherland AD. Total 24-hour melatonin secretion in adolescent idiopathic scoliosis. A case-control study. Spine. 1998;23(1):41–6.
215. Fang MQ, Wang C, Xiang GH, et al. Long-term effects of the Cheneau brace on coronal and sagittal alignment in adolescent idiopathic scoliosis. J Neurosurg Spine. 2015;23(4):505–9.
216. Farley FA, Song KM, Birch JG, et al. Syringomyelia and scoliosis in children. J Pediatr Orthop. 1995;15(2):187–92.
217. Fattori R, Nienaber CA, Descovich B, et al. Importance of dural ectasia in phenotypic assessment of Marfan's syndrome. Lancet. 1999;354(9182):910–3.
218. Faust MS. Somatic development of adolescent girls. Monogr Soc Res Child Dev. 1977;42(1):1–90.

219. Federico DJ, Renshaw TS. Results of treatment of idiopathic scoliosis with the Charleston bending orthosis. Spine. 1990;15(9):886–7.
220. Feldman JM, Roth JV, Bjoraker DG. Maximum blood savings by acute normovolemic hemodilution. Anesth Analg. 1995;80(1):108–13.
221. Feng B, Qiu G, Shen J, et al. Impact of multimodal intraoperative monitoring during surgery for spine deformity and potential risk factors for neurological monitoring changes. J Spinal Disord Tech. 2012;25(4):E108–14.
222. Ferrero E, Pesenti S, Blondel B, et al. Role of thoracoscopy for the sagittal correction of hypokyphotic adolescent idiopathic scoliosis patients. Eur Spine J. 2014;23(12):2635–42.
223. Finkel RS, Chiriboga CA, Vajsar J, et al. Treatment of infantile-onset spinal muscular atrophy with nusinersen: a phase 2, open-label, dose-escalation study. Lancet. 2016;388(10063):3017–26.
224. Fletcher ND, Andras LM, Lazarus DE, et al. Use of a novel pathway for early discharge was associated with a 48% shorter length of stay after posterior spinal fusion for adolescent idiopathic scoliosis. J Pediatr Orthop. 2017;37(2):92–7.
225. Fletcher ND, Glotzbecker MP, Marks M, et al. Development of consensus-based best practice guidelines for postoperative care following posterior spinal fusion for adolescent idiopathic scoliosis. Spine (Phila Pa 1976). 2017;42(9):E547–54.
226. Fletcher ND, McClung A, Rathjen KE, et al. Serial casting as a delay tactic in the treatment of moderate-to-severe early-onset scoliosis. J Pediatr Orthop. 2012;32(7):664–71.
227. Floman Y. "Mirror image" congenital scoliosis in siblings. J Spinal Disord. 1991;4(3):366–8.
228. Floman Y, Liebergall M, Robin GC, et al. Abnormalities of aggregation, thromboxane A2 synthesis, and 14C serotonin release in platelets of patients with idiopathic scoliosis. Spine. 1983;8(3):236–41.
229. Florentino-Pineda I, Blakemore LC, Thompson GH, et al. The effect of epsilon-aminocaproic acid on perioperative blood loss in patients with idiopathic scoliosis undergoing posterior spinal fusion: a preliminary prospective study. Spine. 2001;26(10):1147–51.
230. Florentino-Pineda I, Thompson GH, Poe-Kochert C, et al. The effect of amicar on perioperative blood loss in idiopathic scoliosis: the results of a prospective, randomized double-blind study. Spine. 2004;29(3):233–8.
231. Flynn JM, Betz RR, O'Brien MF, et al. Radiographic classification of complications of instrumentation in adolescent idiopathic scoliosis. Clin Orthop Relat Res. 2010;468(3):665–9.
232. Flynn JM, Otsuka NY, Emans JB, et al. Segmental spinal dysgenesis: early neurologic deterioration and treatment. J Pediatr Orthop. 1997;17(1):100–4.
233. Flynn JM, Ramirez N, Emans JB, et al. Is the vertebral expandable prosthetic titanium rib a surgical alternative in patients with spina bifida? Clin Orthop Relat Res. 2011;469(5):1291–6.
234. Flynn JM, Tomlinson LA, Pawelek J, et al. Growing-rod graduates: lessons learned from ninety-nine patients who completed lengthening. J Bone Joint Surg Am. 2013;95(19):1745–50.
235. Ford EG, Jaufmann BA, Kaste SC, et al. Successful staged surgical correction of congenital segmental spinal dysgenesis and complete rotary subluxation of the thoracolumbar spine in an infant. J Pediatr Surg. 1996;31(7):960–4.
236. Forst J, Forst R, Leithe H, et al. Platelet function deficiency in Duchenne muscular dystrophy. Neuromuscul Disord. 1998;8(1):46–9.
237. Fox HJ, Thomas CH, Thompson AG. Spinal instrumentation for Duchenne's muscular dystrophy: experience of hypotensive anaesthesia to minimise blood loss. J Pediatr Orthop. 1997;17(6):750–3.
238. Fratelli N, Rich P, Jeffrey I, et al. Prenatal diagnosis of segmental spinal dysgenesis. Prenat Diagn. 2007;27(10):979–81.
239. Fricka KB, Mahar AT, Newton PO. Biomechanical analysis of anterior scoliosis instrumentation: differences between single and dual rod systems with and without interbody structural support. Spine. 2002;27(7):702–6.
240. Fromm B, Carstens C, Niethard FU, et al. Aortography in children with myelomeningocele and lumbar kyphosis. J Bone Joint Surg Br. 1992;74(5):691–4.

241. Fu TS, Chen LH, Wong CB, et al. Computer-assisted fluoroscopic navigation of pedicle screw insertion: an in vivo feasibility study. Acta Orthop Scand. 2004;75(6):730–5.
242. Funasaki H, Winter RB, Lonstein JB, et al. Pathophysiology of spinal deformities in neurofibromatosis. An analysis of seventy-one patients who had curves associated with dystrophic changes. J Bone Joint Surg Am. 1994;76(5):692–700.
243. Funk S, Lovejoy S, Mencio G, et al. Rigid instrumentation for neuromuscular scoliosis improves deformity correction without increasing complications. Spine (Phila Pa 1976). 2016;41(1):46–52.
244. Furderer S, Eysel P, Hopf C, et al. Sagittal static imbalance in myelomeningocele patients: improvement in sitting ability by partial and total gibbus resection. Eur Spine J. 1999;8(6):451–7.
245. Furumasu J, Swank SM, Brown JC, et al. Functional activities in spinal muscular atrophy patients after spinal fusion. Spine. 1989;14(7):771–5.
246. Galasko CS, Delaney C, Morris P. Spinal stabilisation in Duchenne muscular dystrophy. J Bone Joint Surg Br. 1992;74(2):210–4.
247. Galasko CS, Williamson JB, Delaney CM. Lung function in Duchenne muscular dystrophy. Eur Spine J. 1995;4(5):263–7.
248. Gao X, Gordon D, Zhang D, et al. CHD7 gene polymorphisms are associated with susceptibility to idiopathic scoliosis. Am J Hum Genet. 2007;80(5):957–65.
249. Garg S, Engelman G, Yoshihara H, et al. The relationship of gross motor functional classification scale level and hip dysplasia on the pattern and progression of scoliosis in children with cerebral palsy. Spine Deform. 2013;1(4):266–71.
250. Garg S, Hosalkar H, Dormans JP. Quadriplegia in a 10 year-old boy due to multiple cervical neurofibromas. Spine. 2003;28(17):E339–43.
251. Garg S, Oetgen M, Rathjen K, et al. Kyphectomy improves sitting and skin problems in patients with myelomeningocele. Clin Orthop Relat Res. 2011;469(5):1279–85.
252. Gau YL, Lonstein JE, Winter RB, et al. Luque-Galveston procedure for correction and stabilization of neuromuscular scoliosis and pelvic obliquity: a review of 68 patients. J Spinal Disord. 1991;4(4):399–410.
253. Gaudiche O, Loose JP, Egu JF, et al. Autotransfusion using cell saver III. Experience with 90 cases of surgery of the spine in childhood and adolescence. Cah Anesthesiol. 1988;36(6):451–7.
254. Gebhart S, Alton TB, Bompadre V, et al. Do anchor density or pedicle screw density correlate with short-term outcome measures in adolescent idiopathic scoliosis surgery? Spine (Phila Pa 1976). 2014;39(2):E104–10.
255. Geck MJ, Rinella A, Hawthorne D, et al. Comparison of surgical treatment in Lenke 5C adolescent idiopathic scoliosis: anterior dual rod versus posterior pedicle fixation surgery: a comparison of two practices. Spine (Phila Pa 1976). 2009;34(18):1942–51.
256. Geck MJ, Rinella A, Hawthorne D, et al. Anterior dual rod versus posterior pedicle fixation surgery for the surgical treatment in Lenke 5C adolescent idiopathic scoliosis: a Multicenter, matched case analysis of 42 patients. Spine Deform. 2013;1(3):217–22.
257. Geissele AE, Kransdorf MJ, Geyer CA, et al. Magnetic resonance imaging of the brain stem in adolescent idiopathic scoliosis. Spine. 1991;16(7):761–3.
258. Ghandhari H, Tari HV, Ameri E, et al. Vertebral, rib, and intraspinal anomalies in congenital scoliosis: a study on 202 Caucasians. Eur Spine J. 2015;24(7):1510–21.
259. Gjolaj JP, Sponseller PD, Shah SA, et al. Spinal deformity correction in Marfan syndrome versus adolescent idiopathic scoliosis: learning from the differences. Spine (Phila Pa 1976). 2012;37(18):1558–65.
260. Glard Y, Launay F, Edgard-Rosa G, et al. Scoliotic curve patterns in patients with Marfan syndrome. J Child Orthop. 2008;2(3):211–6.
261. Glaser DA, Doan J, Newton PO. Comparison of 3-dimensional spinal reconstruction accuracy: biplanar radiographs with EOS versus computed tomography. Spine (Phila Pa 1976). 2012;37(16):1391–7.

262. Glassman SD, Sucato DJ, Carreon LY, et al. Does thoracic hypokyphosis matter in Lenke type 1 adolescent idiopathic scoliosis? Spine Deform. 2013;1(1):40–5.
263. Glassman SD, Zhang YP, Shields CB, et al. Transcranial magnetic motor-evoked potentials in scoliosis surgery. Orthopedics. 1995;18(10):1017–23.
264. Goldberg CJ, Dowling FE, Fogarty EE, et al. Adolescent idiopathic scoliosis and cerebral asymmetry. An examination of a nonspinal perceptual system. Spine. 1995;20(15):1685–91.
265. Goldberg CJ, Dowling FE, Hall JE, et al. A statistical comparison between natural history of idiopathic scoliosis and brace treatment in skeletally immature adolescent girls. Spine. 1993;18(7):902–8.
266. Goldberg CJ, Fogarty EE, Moore DP, et al. Fluctuating asymmetry and vertebral malformation. A study of palmar dermatoglyphics in congenital spinal deformities. Spine. 1997;22(7):775–9.
267. Goldberg CJ, Gillic I, Connaughton O, et al. Respiratory function and cosmesis at maturity in infantile-onset scoliosis. Spine (Phila Pa 1976). 2003;28(20):2397–406.
268. Gotfryd AO, Avanzi O. Randomized clinical study on surgical techniques with different pedicle screw densities in the treatment of adolescent idiopathic scoliosis types Lenke 1A and 1B. Spine Deform. 2013;1(4):272–9.
269. Goultidis TT, Papavasiliou KA, Petropoulos AS, et al. Higher levels of melatonin in early stages of adolescent idiopathic scoliosis: toward a new scenario. J Pediatr Orthop. 2014;34(8):768–73.
270. Gozdzialska A, Jaskiewicz J, Knapik-Czajka M, et al. Association of Calcium and Phosphate Balance, vitamin D, PTH, and calcitonin in patients with adolescent idiopathic scoliosis. Spine (Phila Pa 1976). 2016;41(8):693–7.
271. Granata C, Merlini L, Cervellati S, et al. Long-term results of spine surgery in Duchenne muscular dystrophy. Neuromuscul Disord. 1996;6(1):61–8.
272. Granata C, Merlini L, Magni E, et al. Spinal muscular atrophy: natural history and orthopaedic treatment of scoliosis. Spine. 1989;14(7):760–2.
273. Grassi V, Tantucci C. Respiratory prognosis in chest wall diseases. Monaldi Arch Chest Dis. 1993;48(2):183–7.
274. Grogan DP, Kalen V, Ross TI, et al. Use of allograft bone for posterior spinal fusion in idiopathic scoliosis. Clin Orthop Relat Res. 1999;369:273–8.
275. Guerrero G, Saieh C, Dockendorf I, et al. Genitourinary anomalies in children with congenital scoliosis. Rev Chil Pediatr. 1989;60(5):281–3.
276. Guille JT, Forlin E, Bowen JR. Congenital kyphosis. Orthop Rev. 1993;22(2):235–9.
277. Gupta P, Lenke LG, Bridwell KH. Incidence of neural axis abnormalities in infantile and juvenile patients with spinal deformity. Is a magnetic resonance image screening necessary? Spine. 1998;23(2):206–10.
278. Gurnett CA, Alaee F, Bowcock A, et al. Genetic linkage localizes an adolescent idiopathic scoliosis and pectus excavatum gene to chromosome 18 q. Spine (Phila Pa 1976). 2009;34(2):E94–100.
279. Gutman G, Benoit M, Joncas J, et al. The effectiveness of the SpineCor brace for the conservative treatment of adolescent idiopathic scoliosis. Comparison with the Boston brace. Spine J. 2016;16(5):626–31.
280. Haberland N, Ebmeier K, Grunewald JP, et al. Incorporation of intraoperative computerized tomography in a newly developed spinal navigation technique. Comput Aided Surg. 2000;5(1):18–27.
281. Habermann CR, Weiss F, Schoder V, et al. MR evaluation of dural ectasia in Marfan syndrome: reassessment of the established criteria in children, adolescents, and young adults. Radiology. 2005;234(2):535–41.
282. Hadley-Miller N, Mims B, Milewicz DM. The potential role of the elastic fiber system in adolescent idiopathic scoliosis. J Bone Joint Surg Am. 1994;76(8):1193–206.
283. Hagglund G, Karlberg J, Willner S. Growth in girls with adolescent idiopathic scoliosis. Spine. 1992;17(1):108–11.

284. Hahn F, Hauser D, Espinosa N, et al. Scoliosis correction with pedicle screws in Duchenne muscular dystrophy. Eur Spine J. 2008;17(2):255–61.
285. Hall JE, Levine CR, Sudhir KG. Intraoperative awakening to monitor spinal cord function during Harrington instrumentation and spine fusion. Description of procedure and report of three cases. J Bone Joint Surg Am. 1978;60(4):533–6.
286. Hall JE, Poitras B. The management of kyphosis in patients with myelomeningocele. Clin Orthop Relat Res. 1977;(128):33–40.
287. Hamill CL, Bridwell KH, Lenke LG, et al. Posterior arthrodesis in the skeletally immature patient. Assessing the risk for crankshaft: is an open triradiate cartilage the answer? Spine. 1997;22(12):1343–51.
288. Hamill CL, Lenke LG, Bridwell KH, et al. The use of pedicle screw fixation to improve correction in the lumbar spine of patients with idiopathic scoliosis. Is it warranted? Spine. 1996;21(10):1241–9.
289. Hammett TC, Boreham B, Quraishi NA, et al. Intraoperative spinal cord monitoring during the surgical correction of scoliosis due to cerebral palsy and other neuromuscular disorders. Eur Spine J. 2013;22(Suppl 1):S38–41.
290. Hanna BG, Pill SG, Drummond DS. Irreducible thoracic spondyloptosis in a child with neurofibromatosis: a rationale for treatment. Spine. 2002;27(14):E342–7.
291. Harper CM, Ambler G, Edge G. The prognostic value of pre-operative predicted forced vital capacity in corrective spinal surgery for Duchenne's muscular dystrophy. Anaesthesia. 2004;59(12):1160–2.
292. Harrington PR. The etiology of idiopathic scoliosis. Clin Orthop Relat Res. 1977;126:17–25.
293. Hasler CC. Operative treatment for spinal deformities in cerebral palsy. J Child Orthop. 2013;7(5):419–23.
294. Hassanzadeh H, Nandyala SV, Puvanesarajah V, et al. Serial Mehta cast utilization in infantile idiopathic scoliosis: evaluation of radiographic predictors. J Pediatr Orthop. 2017;37(6):387–91.
295. Hattaway GL. Congenital scoliosis in one of monozygotic twins: a case report. J Bone Joint Surg Am. 1977;59(6):837–8.
296. Hawary RE, Zaaroor-Regev D, Floman Y, et al. Brace treatment in adolescent idiopathic scoliosis: risk factors for failure-a literature review. Spine J. 2019;19(12):1917–25.
297. Heary RF, Bono CM, Black M. Thoracic pedicle screws: postoperative computerized tomography scanning assessment. J Neurosurg. 2004;100(4 Suppl Spine):325–31.
298. Hedequist DJ, Emans JB. The correlation of preoperative three-dimensional computed tomography reconstructions with operative findings in congenital scoliosis. Spine. 2003;28(22):2531–4; discussion 2531.
299. Hedequist DJ, Hall JE, Emans JB. The safety and efficacy of spinal instrumentation in children with congenital spine deformities. Spine. 2004;29(18):2081–6; discussion 2087.
300. Hedequist DJ, Hall JE, Emans JB. Hemivertebra excision in children via simultaneous anterior and posterior exposures. J Pediatr Orthop. 2005;25(1):60–3.
301. Hee HT, Yu ZR, Wong HK. Comparison of segmental pedicle screw instrumentation versus anterior instrumentation in adolescent idiopathic thoracolumbar and lumbar scoliosis. Spine (Phila Pa 1976). 2007;32(14):1533–42.
302. Hefti FL, McMaster MJ. The effect of the adolescent growth spurt on early posterior spinal fusion in infantile and juvenile idiopathic scoliosis. J Bone Joint Surg Br. 1983;65(3):247–54.
303. Helenius I, Remes V, Yrjonen T, et al. Does gender affect outcome of surgery in adolescent idiopathic scoliosis? Spine. 2005;30(4):462–7.
304. Helenius IJ, Sponseller PD, Mackenzie W, et al. Outcomes of spinal fusion for cervical kyphosis in children with Neurofibromatosis. J Bone Joint Surg Am. 2016;98(21):e95.
305. Helmers SL, Hall JE. Intraoperative somatosensory evoked potential monitoring in pediatrics. J Pediatr Orthop. 1994;14(5):592–8.
306. Herzka A, Sponseller PD, Pyeritz RE. Atlantoaxial rotatory subluxation in patients with Marfan syndrome. A report of three cases. Spine. 2000;25(4):524–6.

307. Heydemann JS, Gillespie R. Management of myelomeningocele kyphosis in the older child by kyphectomy and segmental spinal instrumentation. Spine. 1987;12(1):37–41.
308. Hilibrand AS, Blakemore LC, Loder RT, et al. The role of melatonin in the pathogenesis of adolescent idiopathic scoliosis. Spine. 1996;21(10):1140–6.
309. Holcomb GW 3rd, Mencio GA, Green NE. Video-assisted thoracoscopic diskectomy and fusion. J Pediatr Surg. 1997;32(7):1120–2.
310. Hollenbeck SM, Yaszay B, Sponseller PD, et al. The pros and cons of operating early versus late in the progression of cerebral palsy scoliosis. Spine Deform. 2019;7(3):489–93.
311. Holte DC, Winter RB, Lonstein JE, et al. Excision of hemivertebrae and wedge resection in the treatment of congenital scoliosis. J Bone Joint Surg Am. 1995;77(2):159–71.
312. Hopf A, Eysel P, Dubousset J. CDH: preliminary report on a new anterior spinal instrumentation. Eur Spine J. 1995;4(3):194–9.
313. Hopf CG, Eysel P, Dubousset J. Operative treatment of scoliosis with Cotrel-Dubousset-Hopf instrumentation. New anterior spinal device. Spine. 1997;22(6):618–27; discussion 627–618.
314. Hosalkar HS, Luedtke LM, Drummond DS. New technique in congenital scoliosis involving fixation to the pelvis after hemivertebra excision. Spine. 2004;29(22):2581–7.
315. Hosseini P, Akbarnia BA, Nguyen S, et al. Construct levels to anchored levels ratio and rod diameter are associated with implant-related complications in traditional growing rods. Spine Deform. 2018;6(3):320–6.
316. How NE, Street JT, Dvorak MF, et al. Pseudarthrosis in adult and pediatric spinal deformity surgery: a systematic review of the literature and meta-analysis of incidence, characteristics, and risk factors. Neurosurg Rev. 2019;42(2):319–36.
317. Hresko MT, Talwalkar V, Schwend R, et al. Early detection of idiopathic scoliosis in adolescents. J Bone Joint Surg Am. 2016;98(16):e67.
318. Hsu JD. The natural history of spine curvature progression in the nonambulatory Duchenne muscular dystrophy patient. Spine (Phila Pa 1976). 1983;8(7):771–5.
319. Hsu JD, Slager UT, Swank SM, et al. Idiopathic scoliosis: a clinical, morphometric, and histopathological correlation. J Pediatr Orthop. 1988;8(2):147–52.
320. Huang TJ, Lubicky JP. Kyphectomy and segmental spinal instrumentation in young children with myelomeningocele kyphosis. J Formos Med Assoc. 1994;93(6):503–8.
321. Huber M, Gilbert G, Roy J, et al. Sensitivity of MRI parameters within intervertebral discs to the severity of adolescent idiopathic scoliosis. J Magn Reson Imaging. 2016;44(5):1123–31.
322. Hughes LO, McCarthy RE, Glasier CM. Segmental spinal dysgenesis: a report of three cases. J Pediatr Orthop. 1998;18(2):227–32.
323. Hui SC, Pialasse JP, Wong JY, et al. Radiation dose of digital radiography (DR) versus microdose x-ray (EOS) on patients with adolescent idiopathic scoliosis: 2016 SOSORT- IRSSD "John Sevastic award" winner in imaging research. Scoliosis Spinal Disord. 2016;11:46.
324. Hur SR, Huizenga BA, Major M. Acute normovolemic hemodilution combined with hypotensive anesthesia and other techniques to avoid homologous transfusion in spinal fusion surgery. Spine. 1992;17(8):867–73.
325. Iannaccone ST, Owens H, Scott J, et al. Postoperative malnutrition in Duchenne muscular dystrophy. J Child Neurol. 2003;18(1):17–20.
326. Ilharreborde B. Sagittal balance and idiopathic scoliosis: does final sagittal alignment influence outcomes, degeneration rate or failure rate? Eur Spine J. 2018;27(Suppl 1):48–58.
327. Ilharreborde B, Ferrero E, Alison M, et al. EOS microdose protocol for the radiological follow-up of adolescent idiopathic scoliosis. Eur Spine J. 2016;25(2):526–31.
328. Imahiyerobo T, Minkara AA, Matsumoto H, et al. Plastic multilayered closure in Pediatric nonidiopathic scoliosis is associated with a lower than expected incidence of wound complications and surgical site infections. Spine Deform. 2018;6(4):454–9.
329. Imaizumi K, Masuno M, Ishii T, et al. Congenital scoliosis (hemivertebra) associated with de novo balanced reciprocal translocation, 46, XX, t(13;17)(q34;p11.2). Am J Med Genet. 1997;73(3):244–6.
330. Inoh H, Kawakami N, Matsuyama Y, et al. Correlation between the age of pinealectomy and the development of scoliosis in chickens. Spine. 2001;26(9):1014–21.

331. Inoue M, Minami S, Nakata Y, et al. Preoperative MRI analysis of patients with idiopathic scoliosis: a prospective study. Spine. 2005;30(1):108–14.
332. Iorio J, Orlando G, Diefenbach C, et al. Serial casting for infantile idiopathic scoliosis: radiographic outcomes and factors associated with response to treatment. J Pediatr Orthop. 2017;37(5):311–6.
333. Irwin MG, Henderson M. Cardiac arrest during major spinal scoliosis surgery in a patient with Duchenne's muscular dystrophy undergoing intravenous anaesthesia. Anaesth Intensive Care. 1995;23(5):626–9.
334. Jackson T, Yaszay B, Sponseller PD, et al. Factors associated with surgical approach and outcomes in cerebral palsy scoliosis. Eur Spine J. 2019;28(3):567–80.
335. Jain A, Njoku DB, Sponseller PD. Does patient diagnosis predict blood loss during posterior spinal fusion in children? Spine (Phila Pa 1976). 2012;37(19):1683–7.
336. Jain A, Sponseller PD, Shah SA, et al. Subclassification of GMFCS Level-5 cerebral palsy as a predictor of complications and health-related quality of life after spinal arthrodesis. J Bone Joint Surg Am. 2016;98(21):1821–8.
337. Jain A, Thompson JM, Brooks JT, et al. Implant-related fractures in children with proximal femoral osteotomy: blade plate versus screw-side plate constructs. J Pediatr Orthop. 2016;36(1):e1–5.
338. Jalanko T, Rintala R, Puisto V, et al. Hemivertebra resection for congenital scoliosis in young children: comparison of clinical, radiographic, and health-related quality of life outcomes between the anteroposterior and posterolateral approaches. Spine (Phila Pa 1976). 2011;36(1):41–9.
339. James JI. Idiopathic scoliosis; the prognosis, diagnosis, and operative indications related to curve patterns and the age at onset. J Bone Joint Surg Br. 1954;36-B(1):36–49.
340. James JI. The etiology of scoliosis. J Bone Joint Surg Br. 1970;52(3):410–9.
341. Janicki JA, Poe-Kochert C, Armstrong DG, et al. A comparison of the thoracolumbosacral orthoses and providence orthosis in the treatment of adolescent idiopathic scoliosis: results using the new SRS inclusion and assessment criteria for bracing studies. J Pediatr Orthop. 2007;27(4):369–74.
342. Jevsevar DS, Karlin LI. The relationship between preoperative nutritional status and complications after an operation for scoliosis in patients who have cerebral palsy. J Bone Joint Surg Am. 1993;75(6):880–4.
343. Johansson MS, Jensen Stochkendahl M, Hartvigsen J, et al. Incidence and prognosis of midback pain in the general population: a systematic review. Eur J Pain. 2017;21(1):20–8.
344. Johnston CE. Anterior correction of thoracolumbar and lumbar idiopathic scoliosis. Semin Spine Surg. 1997;9:150.
345. Johnston CE 2nd, Hakala MW, Rosenberger R. Paralytic spinal deformity: orthotic treatment in spinal discontinuity syndromes. J Pediatr Orthop. 1982;2(3):233–41.
346. Johnston CE, Herring JA, Ashman RB. Texas Scottish Rite Hospital (TSRH) universal spinal instrumentation system. In: An HS, Cotler JM, editors. Spinal instrumentation. Baltimore: Williams & Wilkins; 1992. p. 127.
347. Johnston CE, McClung AM, Thompson GH, et al. Comparison of growing rod instrumentation versus serial cast treatment for early-onset scoliosis. Spine Deform. 2013;1(5):339–42.
348. Johnston CE, Tran DP, McClung A. Functional and radiographic outcomes following growth-sparing Management of Early-Onset Scoliosis. J Bone Joint Surg Am. 2017;99(12):1036–42.
349. Jones KB, Erkula G, Sponseller PD, et al. Spine deformity correction in Marfan syndrome. Spine. 2002;27(18):2003–12.
350. Jones RS, Kennedy JD, Hasham F, et al. Mechanical inefficiency of the thoracic cage in scoliosis. Thorax. 1981;36(6):456–61.
351. Jonker D, Castelein R. Blood saving in spine surgery. Curr Orthop. 2000;14:365.
352. Joseph KN, Kane HA, Milner RS, et al. Orthopedic aspects of the Marfan phenotype. Clin Orthop Relat Res. 1992;(277):251–61.
353. Justice CM, Miller NH, Marosy B, et al. Familial idiopathic scoliosis: evidence of an X-linked susceptibility locus. Spine. 2003;28(6):589–94.

354. Kalen V, Conklin MM, Sherman FC. Untreated scoliosis in severe cerebral palsy. J Pediatr Orthop. 1992;12(3):337–40.
355. Kamaci S, Demirkiran G, Ismayilov V, et al. The effect of dual growing rod instrumentation on the apical vertebral rotation in early-onset idiopathic scoliosis. J Pediatr Orthop. 2014;34(6):607–12.
356. Kamimura M, Ebara S, Itoh H, et al. Accurate pedicle screw insertion under the control of a computer-assisted image guiding system: laboratory test and clinical study. J Orthop Sci. 1999;4(3):197–206.
357. Kandwal P, Goswami A, Vijayaraghavan G, et al. Staged anterior release and posterior instrumentation in correction of severe rigid scoliosis (cobb angle >100 degrees). Spine Deform. 2016;4(4):296–303.
358. Kaneda K, Satoh S, Fujiya N. Analysis of results with Zielke instrumentation for thoracolumbar and lumbar curvature. Nippon Seikeigeka Gakkai Zasshi. 1985;59(8):841–51.
359. Kaneda K, Shono Y, Satoh S, et al. New anterior instrumentation for the management of thoracolumbar and lumbar scoliosis. Application of the Kaneda two-rod system. Spine. 1996;21(10):1250–61; discussion 1261–1252.
360. Karol LA. Scoliosis in patients with Duchenne muscular dystrophy. J Bone Joint Surg Am. 2007;89(Suppl 1):155–62.
361. Karol LA, Johnston C, Mladenov K, et al. Pulmonary function following early thoracic fusion in non-neuromuscular scoliosis. J Bone Joint Surg Am. 2008;90(6):1272–81.
362. Karol LA, Johnston CE 2nd, Browne RH, et al. Progression of the curve in boys who have idiopathic scoliosis. J Bone Joint Surg Am. 1993;75(12):1804–10.
363. Karol LA, Virostek D, Felton K, et al. The effect of the Risser stage on bracing outcome in adolescent idiopathic scoliosis. J Bone Joint Surg Am. 2016;98(15):1253–9.
364. Karol LA, Virostek D, Felton K, et al. Effect of compliance Counseling on brace use and success in patients with adolescent idiopathic scoliosis. J Bone Joint Surg Am. 2016;98(1):9–14.
365. Katz DE, Herring JA, Browne RH, et al. Brace wear control of curve progression in adolescent idiopathic scoliosis. J Bone Joint Surg Am. 2010;92(6):1343–52.
366. Katz DE, Richards BS, Browne RH, et al. A comparison between the Boston brace and the Charleston bending brace in adolescent idiopathic scoliosis. Spine. 1997;22(12):1302–12.
367. Kawabata S, Watanabe K, Hosogane N, et al. Surgical correction of severe cervical kyphosis in patients with neurofibromatosis type 1. J Neurosurg Spine. 2013;18(3):274–9.
368. Kawakami N, Tsuji T, Imagama S, et al. Classification of congenital scoliosis and kyphosis: a new approach to the three-dimensional classification for progressive vertebral anomalies requiring operative treatment. Spine (Phila Pa 1976). 2009;34(17):1756–65.
369. Kelfer HM, Singer WD, Reynolds RN. Malignant hyperthermia in a child with Duchenne muscular dystrophy. Pediatrics. 1983;71(1):118–9.
370. Kelly DM, McCarthy RE, McCullough FL, et al. Long-term outcomes of anterior spinal fusion with instrumentation for thoracolumbar and lumbar curves in adolescent idiopathic scoliosis. Spine (Phila Pa 1976). 2010;35(2):194–8.
371. Kennedy JD, Staples AJ, Brook PD, et al. Effect of spinal surgery on lung function in Duchenne muscular dystrophy. Thorax. 1995;50(11):1173–8.
372. Kesling KL, Lonstein JE, Denis F, et al. The crankshaft phenomenon after posterior spinal arthrodesis for congenital scoliosis: a review of 54 patients. Spine. 2003;28(3):267–71.
373. Kesling KL, Reinker KA. Scoliosis in twins. A meta-analysis of the literature and report of six cases. Spine. 1997;22(17):2009–14; discussion 2015.
374. Kester K. Epidural pain management for the pediatric spinal fusion patient. Orthop Nurs. 1997;16(6):55–60; quiz 61–52.
375. Khanna N, Molinari R, Lenke L. Exertional myelopathy in type 2 congenital kyphosis. Spine. 2002;27(22):E488–92.
376. Khong PL, Goh WH, Wong VC, et al. MR imaging of spinal tumors in children with neurofibromatosis 1. AJR Am J Roentgenol. 2003;180(2):413–7.

377. Khoshbin A, Vivas L, Law PW, et al. The long-term outcome of patients treated operatively and non-operatively for scoliosis deformity secondary to spina bifida. Bone Joint J. 2014;96-B(9):1244–51.
378. Khoshhal KI, Ellis RD. Paraparesis after posterior spinal fusion in neurofibromatosis secondary to rib displacement: case report and literature review. J Pediatr Orthop. 2000;20(6):799–801.
379. Kim HW, Weinstein SL. Spine update. The management of scoliosis in neurofibromatosis. Spine. 1997;22(23):2770–6.
380. Kim HW, Weinstein SL. Atypical congenital kyphosis. Report of two cases with long-term follow-up. J Bone Joint Surg Br. 1998;80(1):25–9.
381. Kim YJ, Lenke LG, Cho SK, et al. Comparative analysis of pedicle screw versus hook instrumentation in posterior spinal fusion of adolescent idiopathic scoliosis. Spine. 2004;29(18):2040–8.
382. Kim YJ, Lenke LG, Kim J, et al. Comparative analysis of pedicle screw versus hybrid instrumentation in posterior spinal fusion of adolescent idiopathic scoliosis. Spine. 2006;31(3):291–8.
383. Kim YJ, Otsuka NY, Flynn JM, et al. Surgical treatment of congenital kyphosis. Spine. 2001;26(20):2251–7.
384. Kindsfater K, Lowe T, Lawellin D, et al. Levels of platelet calmodulin for the prediction of progression and severity of adolescent idiopathic scoliosis. J Bone Joint Surg Am. 1994;76(8):1186–92.
385. King HA, Moe JH, Bradford DS, et al. The selection of fusion levels in thoracic idiopathic scoliosis. J Bone Joint Surg Am. 1983;65(9):1302–13.
386. King JD, Lowery GL. Results of lumbar hemivertebral excision for congenital scoliosis. Spine. 1991;16(7):778–82.
387. Kirzner N, Hilliard L, Martin C, et al. Bone graft in posterior spine fusion for adolescent idiopathic scoliosis: a meta-analysis. ANZ J Surg. 2018;88(12):1247–52.
388. Kjaer P, Wedderkopp N, Korsholm L, et al. Prevalence and tracking of back pain from childhood to adolescence. BMC Musculoskelet Disord. 2011;12:98.
389. Klein JD, Garfin SR. Nutritional status in the patient with spinal infection. Orthop Clin North Am. 1996;27(1):33–6.
390. Klemme WR, Denis F, Winter RB, et al. Spinal instrumentation without fusion for progressive scoliosis in young children. J Pediatr Orthop. 1997;17(6):734–42.
391. Klemme WR, Polly DW, Orchowski JR. Hemivertebral excision for congenital scoliosis in very young children. J Pediatr Orthop. 2001;21(6):761–4.
392. Ko AL, Song K, Ellenbogen RG, et al. Retrospective review of multilevel spinal fusion combined with spinal cord transection for treatment of kyphoscoliosis in pediatric myelomeningocele patients. Spine (Phila Pa 1976). 2007;32(22):2493–501.
393. Koptan W, ElMiligui Y. Surgical correction of severe dystrophic neurofibromatosis scoliosis: an experience of 32 cases. Eur Spine J. 2010;19(9):1569–75.
394. Korovessis P, Syrimpeis V, Tsekouras V, et al. Effect of the Cheneau brace in the natural history of moderate adolescent idiopathic scoliosis in girls: cohort analysis of a selected homogenous population of 100 consecutive skeletally immature patients. Spine Deform. 2018;6(5):514–22.
395. Kotani T, Minami S, Takahashi K, et al. Three dimensional analysis of chest wall motion during breathing in healthy individuals and patients with scoliosis using an ultrasonography-based system. Stud Health Technol Inform. 2002;91:135–9.
396. Kruger LM, Colbert JM. Intraoperative autologous transfusion in children undergoing spinal surgery. J Pediatr Orthop. 1985;5(3):330–2.
397. Kugelberg E, Welander L. Heredofamilial juvenile muscular atrophy simulating muscular dystrophy. AMA Arch Neurol Psychiatry. 1956;75(5):500–9.
398. Kuklo TR, Lenke LG, O'Brien MF, et al. Accuracy and efficacy of thoracic pedicle screws in curves more than 90 degrees. Spine. 2005;30(2):222–6.

399. Kuklo TR, Potter BK, Polly DW Jr, et al. Reliability analysis for manual adolescent idiopathic scoliosis measurements. Spine. 2005;30(4):444–54.
400. Kumar SJ, Guille JT. Marfan syndrome. In: Weinstein SL, editor. The pediatric spine: principles and practice. New York: Raven Press; 1994. p. 665.
401. Kuntz CT, Maher PC, Levine NB, et al. Prospective evaluation of thoracic pedicle screw placement using fluoroscopic imaging. J Spinal Disord Tech. 2004;17(3):206–14.
402. Kurz LT, Mubarak SJ, Schultz P, et al. Correlation of scoliosis and pulmonary function in Duchenne muscular dystrophy. J Pediatr Orthop. 1983;3(3):347–53.
403. Kwan MK, Chiu CK, Gani SM, et al. Accuracy and safety of pedicle screw placement in adolescent idiopathic scoliosis patients: a review of 2020 screws using computed tomography assessment. Spine (Phila Pa 1976). 2017;42(5):326–35.
404. Laine T, Lund T, Ylikoski M, et al. Accuracy of pedicle screw insertion with and without computer assistance: a randomised controlled clinical study in 100 consecutive patients. Eur Spine J. 2000;9(3):235–40.
405. Lalonde F, Jarvis J. Congenital kyphosis in myelomeningocele. The effect of cordotomy on bladder function. J Bone Joint Surg Br. 1999;81(2):245–9.
406. Lam KS, Mehdian H, White B. The effects of simple trauma on patients with cervical spine neurofibromatosis: two case reports. Eur Spine J. 1997;6(1):77–80.
407. LaMont LE, Jo C, Molinari S, et al. Radiographic, pulmonary, and clinical outcomes with halo gravity traction. Spine Deform. 2019;7(1):40–6.
408. Landman Z, Oswald T, Sanders J, et al. Prevalence and predictors of pain in surgical treatment of adolescent idiopathic scoliosis. Spine (Phila Pa 1976). 2011;36(10):825–9.
409. Lansford TJ, Burton DC, Asher MA, et al. Radiographic and patient-based outcome analysis of different bone-grafting techniques in the surgical treatment of idiopathic scoliosis with a minimum 4-year follow-up: allograft versus autograft/allograft combination. Spine J. 2013;13(5):523–9.
410. Lapinksy AS, Richards BS. Preventing the crankshaft phenomenon by combining anterior fusion with posterior instrumentation. Does it work? Spine. 1995;20(12):1392–8.
411. Larsen UT, Juhl B, Hein-Sorensen O, et al. Complications during anaesthesia in patients with Duchenne's muscular dystrophy (a retrospective study). Can J Anaesth. 1989;36(4):418–22.
412. Larson AN, Fletcher ND, Daniel C, et al. Lumbar curve is stable after selective thoracic fusion for adolescent idiopathic scoliosis: a 20-year follow-up. Spine (Phila Pa 1976). 2012;37(10):833–9.
413. Larson AN, Polly DW Jr, Ackerman SJ, et al. What would be the annual cost savings if fewer screws were used in adolescent idiopathic scoliosis treatment in the US? J Neurosurg Spine. 2016;24(1):116–23.
414. Larson AN, Polly DW Jr, Diamond B, et al. Does higher anchor density result in increased curve correction and improved clinical outcomes in adolescent idiopathic scoliosis? Spine (Phila Pa 1976). 2014;39(7):571–8.
415. Le Naveaux F, Aubin CE, Larson AN, et al. Implant distribution in surgically instrumented Lenke 1 adolescent idiopathic scoliosis: does it affect curve correction? Spine (Phila Pa 1976). 2015;40(7):462–8.
416. Lebel DE, Corston JA, McAdam LC, et al. Glucocorticoid treatment for the prevention of scoliosis in children with Duchenne muscular dystrophy: long-term follow-up. J Bone Joint Surg Am. 2013;95(12):1057–61.
417. Lebon J, Batailler C, Wargny M, et al. Magnetically controlled growing rod in early onset scoliosis: a 30-case multicenter study. Eur Spine J. 2017;26(6):1567–76.
418. Ledonio CG, Rosenstein BE, Johnston CE, et al. Pulmonary function tests correlated with thoracic volumes in adolescent idiopathic scoliosis. J Orthop Res. 2017;35(1):175–82.
419. Lee CK, Denis F, Winter RB, et al. Analysis of the upper thoracic curve in surgically treated idiopathic scoliosis. A new concept of the double thoracic curve pattern. Spine. 1993;18(12):1599–608.
420. Lee CS, Hwang CJ, Kim NH, et al. Preoperative magnetic resonance imaging evaluation in patients with adolescent idiopathic scoliosis. Asian Spine J. 2017;11(1):37–43.

421. Lee S-M, Suk S-I, Chung E-R. Direct vertebral rotation: a new technique of three-dimensional deformity correction with segmental pedicle screw fixation in adolescent idiopathic scoliosis. Spine. 2004;29(3):343–9.
422. Lefebvre S, Burglen L, Reboullet S, et al. Identification and characterization of a spinal muscular atrophy-determining gene. Cell. 1995;80(1):155–65.
423. Lehman RA Jr, Lenke LG, Keeler KA, et al. Operative treatment of adolescent idiopathic scoliosis with posterior pedicle screw-only constructs: minimum three-year follow-up of one hundred fourteen cases. Spine (Phila Pa 1976). 2008;33(14):1598–604.
424. Lenke LG. Debate: resolved, a 55 degrees right thoracic adolescent idiopathic scoliotic curve should be treated by posterior spinal fusion and segmental instrumentation using thoracic pedicle screws: pro: thoracic pedicle screws should be used to treat a 55 degrees right thoracic adolescent idiopathic scoliosis. J Pediatr Orthop. 2004;24(3):329–34; discussion 338–321.
425. Lenke LG, Betz RR, Bridwell KH, et al. Spontaneous lumbar curve coronal correction after selective anterior or posterior thoracic fusion in adolescent idiopathic scoliosis. Spine. 1999;24(16):1663–71; discussion 1672.
426. Lenke LG, Betz RR, Clements D, et al. Curve prevalence of a new classification of operative adolescent idiopathic scoliosis: does classification correlate with treatment? Spine. 2002;27(6):604–11.
427. Lenke LG, Betz RR, Haher TR, et al. Multisurgeon assessment of surgical decision-making in adolescent idiopathic scoliosis: curve classification, operative approach, and fusion levels. Spine. 2001;26(21):2347–53.
428. Lenke LG, Betz RR, Harms J, et al. Adolescent idiopathic scoliosis: a new classification to determine extent of spinal arthrodesis. J Bone Joint Surg Am. 2001;83-A(8):1169–81.
429. Lenke LG, Bridwell KH, Baldus C, et al. Preventing decompensation in King type II curves treated with Cotrel-Dubousset instrumentation. Strict guidelines for selective thoracic fusion. Spine. 1992;17(8 Suppl):S274–81.
430. Lenke LG, Bridwell KH, O'Brien MF, et al. Recognition and treatment of the proximal thoracic curve in adolescent idiopathic scoliosis treated with Cotrel-Dubousset instrumentation. Spine. 1994;19(14):1589–97.
431. Lenke LG, Edwards CC 2nd, Bridwell KH. The Lenke classification of adolescent idiopathic scoliosis: how it organizes curve patterns as a template to perform selective fusions of the spine. Spine. 2003;28(20):S199–207.
432. Lenke LG, Newton PO, Sucato DJ, et al. Complications after 147 consecutive vertebral column resections for severe Pediatric spinal deformity: a Multicenter analysis. Spine (Phila Pa 1976). 2013;38(2):119–32.
433. Lenke LG, O'Leary PT, Bridwell KH, et al. Posterior vertebral column resection for severe pediatric deformity: minimum two-year follow-up of thirty-five consecutive patients. Spine (Phila Pa 1976). 2009;34(20):2213–21.
434. Lenke LG, Sides BA, Koester LA, et al. Vertebral column resection for the treatment of severe spinal deformity. Clin Orthop Relat Res. 2010;468(3):687–99.
435. Letts M, Rathbone D, Yamashita T, et al. Soft Boston orthosis in management of neuromuscular scoliosis: a preliminary report. J Pediatr Orthop. 1992;12(4):470–4.
436. Lewonowski K, King JD, Nelson MD. Routine use of magnetic resonance imaging in idiopathic scoliosis patients less than eleven years of age. Spine. 1992;17(6 Suppl):S109–16.
437. Li DT, Cui JJ, DeVries S, et al. Humeral head ossification predicts peak height velocity timing and percentage of growth remaining in children. J Pediatr Orthop. 2018;38(9):e546–50.
438. Li DT, Linderman GC, Cui JJ, et al. The proximal humeral ossification system improves assessment of maturity in patients with scoliosis. J Bone Joint Surg Am. 2019;101(20):1868–74.
439. Li ZC, Liu ZD, Dai LY. Surgical treatment of scoliosis associated with Marfan syndrome by using posterior-only instrumentation. J Pediatr Orthop B. 2011;20(2):63–6.
440. Liebergall M, Floman Y, Eldor A. Functional, biochemical, and structural anomalies in platelets of patients with idiopathic scoliosis. J Spinal Disord. 1989;2(2):126–30.

441. Liljenqvist U, Halm H, Bullmann V. Spontaneous lumbar curve correction in selective anterior instrumentation and fusion of idiopathic thoracic scoliosis of Lenke type C. Eur Spine J. 2013;22(Suppl 2):S138–48.
442. Liljenqvist UR, Allkemper T, Hackenberg L, et al. Analysis of vertebral morphology in idiopathic scoliosis with use of magnetic resonance imaging and multiplanar reconstruction. J Bone Joint Surg Am. 2002;84-A(3):359–68.
443. Liljenqvist UR, Halm HF, Link TM. Pedicle screw instrumentation of the thoracic spine in idiopathic scoliosis. Spine. 1997;22(19):2239–45.
444. Lindseth RE. Myelomeningocele spine. In: Weinstein SL, editor. The pediatric spine: principles and practice. New York: Raven Press; 1994. p. 1043.
445. Lindseth RE, Stelzer L Jr. Vertebral excision for kyphosis in children with myelomeningocele. J Bone Joint Surg Am. 1979;61(5):699–704.
446. Lintner SA, Lindseth RE. Kyphotic deformity in patients who have a myelomeningocele. Operative treatment and long-term follow-up. J Bone Joint Surg Am. 1994;76(9):1301–7.
447. Little DG, Song KM, Katz D, et al. Relationship of peak height velocity to other maturity indicators in idiopathic scoliosis in girls. J Bone Joint Surg Am. 2000;82(5):685–93.
448. Liu T, Chu WC, Young G, et al. MR analysis of regional brain volume in adolescent idiopathic scoliosis: neurological manifestation of a systemic disease. J Magn Reson Imaging. 2008;27(4):732–6.
449. Lo Cicero S, Capon F, Melchionda S, et al. First-trimester prenatal diagnosis of spinal muscular atrophy using microsatellite markers. Prenat Diagn. 1994;14(6):459–62.
450. Loder RT, Shapiro P, Towbin R, et al. Aortic anatomy in children with myelomeningocele and congenital lumbar kyphosis. J Pediatr Orthop. 1991;11(1):31–5.
451. Loder RT, Urquhart A, Steen H, et al. Variability in cobb angle measurements in children with congenital scoliosis. J Bone Joint Surg Br. 1995;77(5):768–70.
452. Lonner BS, Ren Y, Newton PO, et al. Risk factors of proximal junctional kyphosis in adolescent idiopathic scoliosis-the pelvis and other considerations. Spine Deform. 2017;5(3):181–8.
453. Lonstein JE. Natural history and school screening for scoliosis. Orthop Clin North Am. 1988;19(2):227–37.
454. Lonstein JE. Adolescent idiopathic scoliosis: screening and diagnosis. Instr Course Lect. 1989;38:105–13.
455. Lonstein JE, Akbarnia A. Operative treatment of spinal deformities in patients with cerebral palsy or mental retardation. An analysis of one hundred and seven cases. J Bone Joint Surg Am. 1983;65(1):43–55.
456. Lonstein JE, Bjorklund S, Wanninger MH, et al. Voluntary school screening for scoliosis in Minnesota. J Bone Joint Surg Am. 1982;64(4):481–8.
457. Lonstein JE, Carlson JM. The prediction of curve progression in untreated idiopathic scoliosis during growth. J Bone Joint Surg Am. 1984;66(7):1061–71.
458. Lonstein JE, Koop SE, Novachek TF, et al. Results and complications after spinal fusion for neuromuscular scoliosis in cerebral palsy and static encephalopathy using Luque Galveston instrumentation: experience in 93 patients. Spine (Phila Pa 1976). 2012;37(7):583–91.
459. Lonstein JE, Winter RB. The Milwaukee brace for the treatment of adolescent idiopathic scoliosis. A review of one thousand and twenty patients. J Bone Joint Surg Am. 1994;76(8):1207–21.
460. Lopez-Sosa F, Guille JT, Bowen JR. Rotation of the spine in congenital scoliosis. J Pediatr Orthop. 1995;15(4):528–34.
461. Low WD, Chew EC, Kung LS, et al. Ultrastructures of nerve fibers and muscle spindles in adolescent idiopathic scoliosis. Clin Orthop Relat Res. 1983;(174):217–21.
462. Lowe GP, Menelaus MB. The surgical management of kyphosis in older children with myelomeningocele. J Bone Joint Surg Br. 1978;60(1):40–5.
463. Lowe T, Lawellin D, Smith D, et al. Platelet calmodulin levels in adolescent idiopathic scoliosis: do the levels correlate with curve progression and severity? Spine. 2002;27(7):768–75.

464. Lowe TG, Burwell RG, Dangerfield PH. Platelet calmodulin levels in adolescent idiopathic scoliosis (AIS): can they predict curve progression and severity? Summary of an electronic focus group debate of the IBSE. Eur Spine J. 2004;13(3):257–65.
465. Lowe TG, Enguidanos ST, Smith DA, et al. Single-rod versus dual-rod anterior instrumentation for idiopathic scoliosis: a biomechanical study. Spine. 2005;30(3):311–7.
466. Lowry KJ, Tobias J, Kittle D, et al. Postoperative pain control using epidural catheters after anterior spinal fusion for adolescent scoliosis. Spine. 2001;26(11):1290–3.
467. Luhmann SJ, Lenke LG, Bridwell KH, et al. Revision surgery after primary spine fusion for idiopathic scoliosis. Spine (Phila Pa 1976). 2009;34(20):2191–7.
468. Luk KD, Hu Y, Wong YW, et al. Evaluation of various evoked potential techniques for spinal cord monitoring during scoliosis surgery. Spine. 2001;26(16):1772–7.
469. Luque ER. Paralytic scoliosis in growing children. Clin Orthop Relat Res. 1982;163:202–9.
470. Lykissas MG, Crawford AH, Chan G, et al. The effect of tranexamic acid in blood loss and transfusion volume in adolescent idiopathic scoliosis surgery: a single-surgeon experience. J Child Orthop. 2013;7(3):245–9.
471. Lykissas MG, Schorry EK, Crawford AH, et al. Does the presence of dystrophic features in patients with type 1 neurofibromatosis and spinal deformities increase the risk of surgery? Spine (Phila Pa 1976). 2013;38(18):1595–601.
472. MacEwen GD, Bunnell WP, Sriram K. Acute neurological complications in the treatment of scoliosis. A report of the Scoliosis Research Society. J Bone Joint Surg Am. 1975;57(3):404–8.
473. MacEwen GD, Winter RB, Hardy JH. Evaluation of kidney anomalies in congenital scoliosis. J Bone Joint Surg Am. 1972;54(7):1451–4.
474. Machida M, Dubousset J, Imamura Y, et al. An experimental study in chickens for the pathogenesis of idiopathic scoliosis. Spine. 1993;18(12):1609–15.
475. Machida M, Dubousset J, Imamura Y, et al. Role of melatonin deficiency in the development of scoliosis in pinealectomised chickens. J Bone Joint Surg Br. 1995;77(1):134–8.
476. Machida M, Dubousset J, Imamura Y, et al. Pathogenesis of idiopathic scoliosis: SEPs in chicken with experimentally induced scoliosis and in patients with idiopathic scoliosis. J Pediatr Orthop. 1994;14(3):329–35.
477. Machida M, Dubousset J, Imamura Y, et al. Melatonin. A possible role in pathogenesis of adolescent idiopathic scoliosis. Spine. 1996;21(10):1147–52.
478. Machida M, Dubousset J, Satoh T, et al. Pathologic mechanism of experimental scoliosis in pinealectomized chickens. Spine. 2001;26(17):E385–91.
479. Machida M, Miyashita Y, Murai I, et al. Role of serotonin for scoliotic deformity in pinealectomized chicken. Spine. 1997;22(12):1297–301.
480. Macke JJ, Woo R, Varich L. Accuracy of robot-assisted pedicle screw placement for adolescent idiopathic scoliosis in the pediatric population. J Robot Surg. 2016;10(2):145–50.
481. Madigan RR, Wallace SL. Scoliosis in the institutionalized cerebral palsy population. Spine (Phila Pa 1976). 1981;6(6):583–90.
482. Maisenbacher MK, Han JS, O'Brien ML, et al. Molecular analysis of congenital scoliosis: a candidate gene approach. Hum Genet. 2005;116(5):416–9.
483. Majd ME, Muldowny DS, Holt RT. Natural history of scoliosis in the institutionalized adult cerebral palsy population. Spine (Phila Pa 1976). 1997;22(13):1461–6.
484. Malviya S, Merkel S. Acute pain management. In: Richards BS, editor. Orthopaedic knowledge update--pediatrics. Rosemont: American Academy of Orthopaedic Surgeons; 1996. p. 47.
485. Mao SH, Shi B, Sun X, et al. Morphometric analysis of iatrogenic breast asymmetry secondary to operative breast shape changes in thoracic adolescent idiopathic scoliosis. Eur Spine J. 2016;25(10):3075–81.
486. Mardjetko SM, Hammerberg KW, Lubicky JP, et al. The Luque trolley revisited. Review of nine cases requiring revision. Spine (Phila Pa 1976). 1992;17(5):582–9.
487. Margalit A, McKean G, Constantine A, et al. Body mass hides the curve: thoracic Scoliometer readings vary by body mass index value. J Pediatr Orthop. 2017;37(4):e255–60.

488. Marks DS, Qaimkhani SA. The natural history of congenital scoliosis and kyphosis. Spine (Phila Pa 1976). 2009;34(17):1751–5.
489. Marks MC, Newton PO, Bastrom TP, et al. Surgical site infection in adolescent idiopathic scoliosis surgery. Spine Deform. 2013;1(5):352–8.
490. Marsh A, Edge G, Lehovsky J. Spinal fusion in patients with Duchenne's muscular dystrophy and a low forced vital capacity. Eur Spine J. 2003;12(5):507–12.
491. Martin J Jr, Kumar SJ, Guille JT, et al. Congenital kyphosis in myelomeningocele: results following operative and nonoperative treatment. J Pediatr Orthop. 1994;14(3):323–8.
492. Master DL, Poe-Kochert C, Son-Hing J, et al. Wound infections after surgery for neuromuscular scoliosis: risk factors and treatment outcomes. Spine (Phila Pa 1976). 2011;36(3):E179–85.
493. Matsumoto H, Williams BA, Corona J, et al. Psychosocial effects of repetitive surgeries in children with early-onset scoliosis: are we putting them at risk? J Pediatr Orthop. 2014;34(2):172–8.
494. Maurice B. Anterior instrumentation (dual screws single rod system) for the surgical treatment of idiopathic scoliosis in the lumbar area: a prospective study on 33 adolescents and young adults, based on a new system of classification. Eur Spine J. 2013;22(Suppl 2):S149–63.
495. Mayell A, Srinivasan I, Campbell F, et al. Analgesic effects of gabapentin after scoliosis surgery in children: a randomized controlled trial. Paediatr Anaesth. 2014;24(12):1239–44.
496. Mayfield JK, Winter RB, Bradford DS, et al. Congenital kyphosis due to defects of anterior segmentation. J Bone Joint Surg Am. 1980;62(8):1291–301.
497. Mazur J, Menelaus MB, Dickens DR, et al. Efficacy of surgical management for scoliosis in myelomeningocele: correction of deformity and alteration of functional status. J Pediatr Orthop. 1986;6(5):568–75.
498. McCall RE. Modified Luque instrumentation after myelomeningocele kyphectomy. Spine. 1998;23(12):1406–11.
499. McCarthy JJ, D'Andrea LP, Betz RR, et al. Scoliosis in the child with cerebral palsy. J Am Acad Orthop Surg. 2006;14(6):367–75.
500. McCarthy RE, Luhmann S, Lenke L, et al. The Shilla growth guidance technique for early-onset spinal deformities at 2-year follow-up: a preliminary report. J Pediatr Orthop. 2014;34(1):1–7.
501. McElroy MJ, Shaner AC, Crawford TO, et al. Growing rods for scoliosis in spinal muscular atrophy: structural effects, complications, and hospital stays. Spine (Phila Pa 1976). 2011;36(16):1305–11.
502. McKenna C, Wade R, Faria R, et al. EOS 2D/3D X-ray imaging system: a systematic review and economic evaluation. Health Technol Assess. 2012;16(14):1–188.
503. McMaster MJ. Occult intraspinal anomalies and congenital scoliosis. J Bone Joint Surg Am. 1984;66(4):588–601.
504. McMaster MJ. Anterior and posterior instrumentation and fusion of thoracolumbar scoliosis due to myelomeningocele. J Bone Joint Surg Br. 1987;69(1):20–5.
505. McMaster MJ. Congenital scoliosis. In: Weinstein SL, editor. The pediatric spine: principles and practice. New York: Raven Press; 1994. p. 227.
506. McMaster MJ. Congenital scoliosis caused by a unilateral failure of vertebral segmentation with contralateral hemivertebrae. Spine. 1998;23(9):998–1005.
507. McMaster MJ, McMaster ME. Prognosis for congenital scoliosis due to a unilateral failure of vertebral segmentation. J Bone Joint Surg Am. 2013;95(11):972–9.
508. McMaster MJ, Singh H. Natural history of congenital kyphosis and kyphoscoliosis. A study of one hundred and twelve patients. J Bone Joint Surg Am. 1999;81(10):1367–83. https://doi.org/10.2106/00004623-199910000-00002. PMID: 10535587.
509. McMaster MJ, Singh H. The surgical management of congenital kyphosis and kyphoscoliosis. Spine. 2001;26(19):2146.
510. McNally CM, Visvanathan R, Liberali S, et al. Antibiotic prophylaxis for dental treatment after prosthetic joint replacement: exploring the orthopaedic surgeon's opinion. Arthroplast Today. 2016;2(3):123–6.

511. Mehlman CT, Crawford AH, Wolf RK. Video-assisted thoracoscopic surgery (VATS). Endoscopic thoracoplasty technique. Spine. 1997;22(18):2178–82.
512. Mehta MH. The rib-vertebra angle in the early diagnosis between resolving and progressive infantile scoliosis. J Bone Joint Surg Br. 1972;54(2):230–43.
513. Mehta MH. Growth as a corrective force in the early treatment of progressive infantile scoliosis. J Bone Joint Surg Br. 2005;87(9):1237–47.
514. Melhem E, Assi A, El Rachkidi R, et al. EOS((R)) biplanar X-ray imaging: concept, developments, benefits, and limitations. J Child Orthop. 2016;10(1):1–14.
515. Menten R, Mousny M, Saint-Martin C, et al. Planispheric multiplanar reformatted CT: a new method for evaluation of paediatric congenital spine abnormalities. Pediatr Radiol. 2005;35(6):627–9.
516. Merlini L, Granata C, Bonfiglioli S, et al. Scoliosis in spinal muscular atrophy: natural history and management. Dev Med Child Neurol. 1989;31(4):501–8.
517. Merriman M, Hu C, Noyes K, et al. Selection of the lowest level for fusion in adolescent idiopathic scoliosis-a systematic review and meta-analysis. Spine Deform. 2015;3(2):128–35.
518. Mesfin A, Ahn NU, Carrino JA, et al. Ten-year clinical and imaging follow-up of dural ectasia in adults with Marfan syndrome. Spine J. 2013;13(1):62–7.
519. Mignemi M, Tran D, Ramo B, et al. Repeat surgical interventions following "definitive" instrumentation and fusion for idiopathic scoliosis: 25-year update. Spine Deform. 2018;6(4):409–16.
520. Miller A, Temple T, Miller F. Impact of orthoses on the rate of scoliosis progression in children with cerebral palsy. J Pediatr Orthop. 1996;16(3):332–5.
521. Miller F, Moseley CF, Koreska J. Spinal fusion in Duchenne muscular dystrophy. Dev Med Child Neurol. 1992;34(9):775–86.
522. Miller F, Moseley CF, Koreska J, et al. Pulmonary function and scoliosis in Duchenne dystrophy. J Pediatr Orthop. 1988;8(2):133–7.
523. Miller NH, Justice CM, Marosy B, et al. Identification of candidate regions for familial idiopathic scoliosis. Spine. 2005;30(10):1181–7.
524. Miller NH, Mims B, Child A, et al. Genetic analysis of structural elastic fiber and collagen genes in familial adolescent idiopathic scoliosis. J Orthop Res. 1996;14(6):994–9.
525. Miller RG, Chalmers AC, Dao H, et al. The effect of spine fusion on respiratory function in Duchenne muscular dystrophy. Neurology. 1991;41(1):38–40.
526. Miller RG, Hoffman EP. Molecular diagnosis and modern management of Duchenne muscular dystrophy. Neurol Clin. 1994;12(4):699–725.
527. Milunsky A, Jick H, Jick SS, et al. Multivitamin/folic acid supplementation in early pregnancy reduces the prevalence of neural tube defects. JAMA. 1989;262(20):2847–52.
528. Minsk MK, Venuti KD, Daumit GL, et al. Effectiveness of the Rigo Cheneau versus Boston-style orthoses for adolescent idiopathic scoliosis: a retrospective study. Scoliosis Spinal Disord. 2017;12:7.
529. Mintz LJ, Sarwark JF, Dias LS, et al. The natural history of congenital kyphosis in myelomeningocele. A review of 51 children. Spine. 1991;16(8 Suppl):S348–50.
530. Miyanji F, Nasto LA, Bastrom T, et al. A detailed comparative analysis of anterior versus posterior approach to Lenke 5C curves. Spine (Phila Pa 1976). 2018;43(5):E285–91.
531. Miyanji F, Nasto LA, Sponseller PD, et al. Assessing the risk-benefit ratio of scoliosis surgery in cerebral palsy: surgery is worth it. J Bone Joint Surg Am. 2018;100(7):556–63.
532. Miyanji F, Pawelek JB, Van Valin SE, et al. Is the lumbar modifier useful in surgical decision making?: defining two distinct Lenke 1A curve patterns. Spine (Phila Pa 1976). 2008;33(23):2545–51.
533. Mladenov KV, Vaeterlein C, Stuecker R. Selective posterior thoracic fusion by means of direct vertebral derotation in adolescent idiopathic scoliosis: effects on the sagittal alignment. Eur Spine J. 2011;20(7):1114–7.
534. Modi HN, Suh SW, Srinivasalu S, et al. Comparison of apical axial derotation between adolescent idiopathic and neuromuscular scoliosis with pedicle screw instrumentation. Asian Spine J. 2008;2(2):74–80.

535. Moe JH. Historical aspects of scoliosis. In: Moe JH, editor. Moe's textbook of scoliosis and other spinal deformities. 1. Philadelphia: W.B. Saunders; 1987.
536. Moe JH, Kharrat K, Winter RB, et al. Harrington instrumentation without fusion plus external orthotic support for the treatment of difficult curvature problems in young children. Clin Orthop Relat Res. 1984;(185):35–45.
537. Moe JH, Purcell GA, Bradford DS. Zielke instrumentation (VDS) for the correction of spinal curvature. Analysis of results in 66 patients. Clin Orthop Relat Res. 1983;(180):133–53.
538. Mohamed Ali MH, Koutharawu DN, Miller F, et al. Operative and clinical markers of deep wound infection after spine fusion in children with cerebral palsy. J Pediatr Orthop. 2010;30(8):851–7.
539. Mooney JF 3rd. Identical type I congenital kyphosis in male twins: a brief report. J Surg Orthop Adv. 2005;14(2):99–101.
540. Mooney V, Gulick J, Pozos R. A preliminary report on the effect of measured strength training in adolescent idiopathic scoliosis. J Spinal Disord. 2000;13(2):102–7.
541. Moran MM, Kroon D, Tredwell SJ, et al. The role of autologous blood transfusion in adolescents undergoing spinal surgery. Spine. 1995;20(5):532–6.
542. Morcuende JA, Dolan LA, Vazquez JD, et al. A prognostic model for the presence of neurogenic lesions in atypical idiopathic scoliosis. Spine. 2004;29(1):51–8.
543. Morcuende JA, Minhas R, Dolan L, et al. Allelic variants of human melatonin 1A receptor in patients with familial adolescent idiopathic scoliosis. Spine. 2003;28(17):2025–8; discussion 2029.
544. Moreau A, da Wang S, Forget S, et al. Melatonin signaling dysfunction in adolescent idiopathic scoliosis. Spine. 2004;29(16):1772–81.
545. Morrissy RT. School screening for scoliosis. Spine. 1999;24(24):2584–91.
546. Morrissy RT, Goldsmith GS, Hall EC, et al. Measurement of the Cobb angle on radiographs of patients who have scoliosis. Evaluation of intrinsic error. J Bone Joint Surg Am. 1990;72(3):320–7.
547. Mostegl A, Bauer R, Eichenauer M. Intraoperative somatosensory potential monitoring. A clinical analysis of 127 surgical procedures. Spine. 1988;13(4):396–400.
548. Mubarak SJ, Camp JF, Vuletich W, et al. Halo application in the infant. J Pediatr Orthop. 1989;9(5):612–4.
549. Mubarak SJ, Morin WD, Leach J. Spinal fusion in Duchenne muscular dystrophy--fixation and fusion to the sacropelvis? J Pediatr Orthop. 1993;13(6):752–7.
550. Muhlrad A, Yarom R. Contractile protein studies on platelets from patients with idiopathic scoliosis. Haemostasis. 1982;11(3):154–60.
551. Muirhead A, Conner AN. The assessment of lung function in children with scoliosis. J Bone Joint Surg Br. 1985;67(5):699–702.
552. Muller EB, Nordwall A, von Wendt L. Influence of surgical treatment of scoliosis in children with spina bifida on ambulation and motoric skills. Acta Paediatr. 1992;81(2):173–6.
553. Murray DJ, Forbes RB, Titone MB, et al. Transfusion management in pediatric and adolescent scoliosis surgery. Efficacy of autologous blood. Spine. 1997;22(23):2735–40.
554. Mustard WT. A follow-up study of iliopsoas transfer for hip instability. J Bone Joint Surg Br. 1959;41-B(2):289–98.
555. Nachemson A. A long term follow-up study of non-treated scoliosis. Acta Orthop Scand. 1968;39(4):466–76.
556. Nachemson AL, Peterson LE. Effectiveness of treatment with a brace in girls who have adolescent idiopathic scoliosis. A prospective, controlled study based on data from the Brace Study of the Scoliosis Research Society. J Bone Joint Surg Am. 1995;77(6):815–22.
557. Nakajima A, Kawakami N, Imagama S, et al. Three-dimensional analysis of formation failure in congenital scoliosis. Spine (Phila Pa 1976). 2007;32(5):562–7.
558. Nakamura H, Matsuda H, Konishi S, et al. Single-stage excision of hemivertebrae via the posterior approach alone for congenital spine deformity: follow-up period longer than ten years. Spine. 2002;27(1):110–5.
559. Nash CL Jr, Moe JH. A study of vertebral rotation. J Bone Joint Surg Am. 1969;51(2):223–9.

560. Nazareth A, Skaggs DL, Parent S. Growth guideance constructs with pedicle screw (Shilla) results in 1/5 of normal T1-S1 Growth. San Diego, CA: International Congress on Early Onset Scoliosis; 2017.
561. Negrini S, Atanasio S, Fusco C, et al. Effectiveness of complete conservative treatment for adolescent idiopathic scoliosis (bracing and exercises) based on SOSORT management criteria: results according to the SRS criteria for bracing studies – SOSORT Award 2009 Winner. Scoliosis. 2009;4:19.
562. Negrini S, Fusco C, Minozzi S, et al. Exercises reduce the progression rate of adolescent idiopathic scoliosis: results of a comprehensive systematic review of the literature. Disabil Rehabil. 2008;30(10):772–85.
563. Negrini S, Minozzi S, Bettany-Saltikov J, et al. Braces for idiopathic scoliosis in adolescents. Spine (Phila Pa 1976). 2016;41(23):1813–25.
564. Nelson LM, Ward K, Ogilvie JW. Genetic variants in melatonin synthesis and signaling pathway are not associated with adolescent idiopathic scoliosis. Spine (Phila Pa 1976). 2011;36(1):37–40.
565. Newton PO. Thoracoscopic treatment of pediatric spinal deformity. Semin Spine Surg. 2003;15(3):244.
566. Newton PO. Spinal growth tethering: indications and limits. Ann Transl Med. 2020;8(2):27.
567. Newton PO, Farnsworth CL, Faro FD, et al. Spinal growth modulation with an anterolateral flexible tether in an immature bovine model: disc health and motion preservation. Spine (Phila Pa 1976). 2008;33(7):724–33.
568. Newton PO, Fricka KB, Lee SS, et al. Asymmetrical flexible tethering of spine growth in an immature bovine model. Spine (Phila Pa 1976). 2002;27(7):689–93.
569. Newton PO, Hahn GW, Fricka KB, et al. Utility of three-dimensional and multiplanar reformatted computed tomography for evaluation of pediatric congenital spine abnormalities. Spine. 2002;27(8):844–50.
570. Newton PO, Khandwala Y, Bartley CE, et al. New EOS imaging protocol allows a substantial reduction in radiation exposure for scoliosis patients. Spine Deform. 2016;4(2):138–44.
571. Newton PO, Marks MC, Bastrom TP, et al. Surgical treatment of Lenke 1 main thoracic idiopathic scoliosis: results of a prospective, multicenter study. Spine (Phila Pa 1976). 2013;38(4):328–38.
572. Newton PO, Shea KG, Granlund KF. Defining the pediatric spinal thoracoscopy learning curve: sixty-five consecutive cases. Spine. 2000;25(8):1028–35.
573. Newton PO, Wenger DR, Mubarak SJ, et al. Anterior release and fusion in pediatric spinal deformity. A comparison of early outcome and cost of thoracoscopic and open thoracotomy approaches. Spine. 1997;22(12):1398–406.
574. Niall DM, Dowling FE, Fogarty EE, et al. Kyphectomy in children with myelomeningocele: a long-term outcome study. J Pediatr Orthop. 2004;24(1):37–44.
575. Nishnianidze T, Bayhan IA, Abousamra O, et al. Factors predicting postoperative complications following spinal fusions in children with cerebral palsy scoliosis. Eur Spine J. 2016;25(2):627–34.
576. Nolden MT, Sarwark JF, Vora A, et al. A kyphectomy technique with reduced perioperative morbidity for myelomeningocele kyphosis. Spine. 2002;27(16):1807–13.
577. Noonan KJ, Walker T, Feinberg JR, et al. Factors related to false- versus true-positive neuromonitoring changes in adolescent idiopathic scoliosis surgery. Spine. 2002;27(8):825–30.
578. Noordeen MH, Haddad FS, Muntoni F, et al. Blood loss in Duchenne muscular dystrophy: vascular smooth muscle dysfunction? J Pediatr Orthop B. 1999;8(3):212–5.
579. Noordeen MH, Taylor BA, Edgar MA. Syringomyelia. A potential risk factor in scoliosis surgery. Spine. 1994;19(12):1406–9.
580. Nowak R, Szota J, Mazurek U. Vitamin D receptor gene (VDR) transcripts in bone, cartilage, muscles and blood and microarray analysis of vitamin D responsive genes expression in paravertebral muscles of juvenile and adolescent idiopathic scoliosis patients. BMC Musculoskelet Disord. 2012;13:259.

581. Nuwer MR, Dawson EG, Carlson LG, et al. Somatosensory evoked potential spinal cord monitoring reduces neurologic deficits after scoliosis surgery: results of a large multicenter survey. Electroencephalogr Clin Neurophysiol. 1995;96(1):6–11.
582. O'Brien MF, Lenke LG, Bridwell KH, et al. Preoperative spinal canal investigation in adolescent idiopathic scoliosis curves > or = 70 degrees. Spine. 1994;19(14):1606–10.
583. O'Brien MF, Lenke LG, Mardjetko S, et al. Pedicle morphology in thoracic adolescent idiopathic scoliosis: is pedicle fixation an anatomically viable technique? Spine. 2000;25(18):2285–93.
584. O'Donnell C, Michael N, Pan X, et al. Anterior spinal fusion and posterior spinal fusion both effectively treat Lenke type 5 curves in adolescent idiopathic scoliosis: a Multicenter study. Spine Deform. 2018;6(3):231–40.
585. O'Donnell CS, Bunnell WP, Betz RR, et al. Electrical stimulation in the treatment of idiopathic scoliosis. Clin Orthop Relat Res. 1988;229:107–13.
586. Oda I, Cunningham BW, Lee GA, et al. Biomechanical properties of anterior thoracolumbar multisegmental fixation: an analysis of construct stiffness and screw-rod strain. Spine. 2000;25(18):2303–11.
587. Odent T, Arlet V, Ouellet J, et al. Kyphectomy in myelomeningocele with a modified Dunn-McCarthy technique followed by an anterior inlayed strut graft. Eur Spine J. 2004;13(3):206–12.
588. Ogilvie J. Neurofibromatosis. In: Lonstein J, Winter R, Ogilvie J, editors. Moe's textbook of scoliosis and other deformities. Philadelphia: W.B. Saunders; 1994. p. 33.
589. Olafsson Y, Odergren T, Persson HE, et al. Somatosensory testing in idiopathic scoliosis. Dev Med Child Neurol. 2002;44(2):130–2.
590. Olsfanger D, Jedeikin R, Metser U, et al. Acute normovolaemic haemodilution and idiopathic scoliosis surgery: effects on homologous blood requirements. Anaesth Intensive Care. 1993;21(4):429–31.
591. Omeroglu H, Ozekin O, Bicimoglu A. Measurement of vertebral rotation in idiopathic scoliosis using the Perdriolle torsionmeter: a clinical study on intraobserver and interobserver error. Eur Spine J. 1996;5(3):167–71.
592. Osebold WR. Stability of myelomeningocele spines treated with the Mayfield two-stage anterior and posterior fusion technique. Spine. 2000;25(11):1344–51.
593. Osebold WR, Mayfield JK, Winter RB, et al. Surgical treatment of paralytic scoliosis associated with myelomeningocele. J Bone Joint Surg Am. 1982;64(6):841–56.
594. Ouellet JA, Johnston CE 2nd. Effect of grafting technique on the maintenance of coronal and sagittal correction in anterior treatment of scoliosis. Spine. 2002;27(19):2129–35; discussion 2135–2126.
595. Ouellet JA, LaPlaza J, Erickson MA, et al. Sagittal plane deformity in the thoracic spine: a clue to the presence of syringomyelia as a cause of scoliosis. Spine. 2003;28(18):2147–51.
596. Owange-Iraka JW, Harrison A, Warner JO. Lung function in congenital and idiopathic scoliosis. Eur J Pediatr. 1984;142(3):198–200.
597. Ozturk C, Alanay A, Ganiyusufoglu K, et al. Short-term X-ray results of posterior vertebral column resection in severe congenital kyphosis, scoliosis, and kyphoscoliosis. Spine (Phila Pa 1976). 2012;37(12):1054–7.
598. Ozturk C, Karadereler S, Ornek I, et al. The role of routine magnetic resonance imaging in the preoperative evaluation of adolescent idiopathic scoliosis. Int Orthop. 2010;34(4):543–6.
599. Padberg AM, Wilson-Holden TJ, Lenke LG, et al. Somatosensory- and motor-evoked potential monitoring without a wake-up test during idiopathic scoliosis surgery. An accepted standard of care. Spine. 1998;23(12):1392–400.
600. Pan Y, Lu GH, Kuang L, et al. Accuracy of thoracic pedicle screw placement in adolescent patients with severe spinal deformities: a retrospective study comparing drill guide template with free-hand technique. Eur Spine J. 2018;27(2):319–26.
601. Panjabi MM, Takata K, Goel V, et al. Thoracic human vertebrae. Quantitative three-dimensional anatomy. Spine. 1991;16(8):888–901.

602. Papastefanou SL, Henderson LM, Smith NJ, et al. Surface electrode somatosensory-evoked potentials in spinal surgery: implications for indications and practice. Spine. 2000;25(19):2467–72.
603. Parent S, Labelle H, Skalli W, et al. Thoracic pedicle morphometry in vertebrae from scoliotic spines. Spine. 2004;29(3):239–48.
604. Parent S, Labelle H, Skalli W, et al. Morphometric analysis of anatomic scoliotic specimens. Spine. 2002;27(21):2305–11.
605. Parisini P, Di Silvestre M, Greggi T, et al. Surgical correction of dystrophic spinal curves in neurofibromatosis. A review of 56 patients. Spine. 1999;24(21):2247–53.
606. Parsch D, Geiger F, Brocai DR, et al. Surgical management of paralytic scoliosis in myelomeningocele. J Pediatr Orthop B. 2001;10(1):10–7.
607. Pasquini G, Cecchi F, Bini C, et al. The outcome of a modified version of the Cheneau brace in adolescent idiopathic scoliosis (AIS) based on SRS and SOSORT criteria: a retrospective study. Eur J Phys Rehabil Med. 2016;52(5):618–29.
608. Pastorelli F, Di Silvestre M, Plasmati R, et al. The prevention of neural complications in the surgical treatment of scoliosis: the role of the neurophysiological intraoperative monitoring. Eur Spine J. 2011;20(Suppl 1):S105–14.
609. Pastorelli F, Di Silvestre M, Vommaro F, et al. Intraoperative monitoring of somatosensory (SSEPs) and transcranial electric motor-evoked potentials (tce-MEPs) during surgical correction of neuromuscular scoliosis in patients with central or peripheral nervous system diseases. Eur Spine J. 2015;24(Suppl 7):931–6.
610. Pehrsson K, Larsson S, Oden A, et al. Long-term follow-up of patients with untreated scoliosis. A study of mortality, causes of death, and symptoms. Spine. 1992;17(9):1091–6.
611. Peleg I, Eldor A, Kahane I, et al. Altered structural and functional properties of myosins, from platelets of idiopathic scoliosis patients. J Orthop Res. 1989;7(2):260–5.
612. Perdriolle R. The torsion meter: a critical review. J Pediatr Orthop. 1991;11(6):789.
613. Peterson HA, Peterson LF. Hemivertebrae in identical twins with dissimilar spinal columns. J Bone Joint Surg Am. 1967;49(5):938–42.
614. Philips MF, Dormans J, Drummond D, et al. Progressive congenital kyphosis: report of five cases and review of the literature. Pediatr Neurosurg. 1997;26(3):130–43.
615. Phillips WA, Hensinger RN. Control of blood loss during scoliosis surgery. Clin Orthop Relat Res. 1988;(229):88–93.
616. Piasecki JO, Mahinpour S, Levine DB. Long-term follow-up of spinal fusion in spinal muscular atrophy. Clin Orthop Relat Res. 1986;(207):44–54.
617. Pollack IF, Colak A, Fitz C, et al. Surgical management of spinal cord compression from plexiform neurofibromas in patients with neurofibromatosis 1. Neurosurgery. 1998;43(2):248–55; discussion 255–246.
618. Ponte A, Orlando G, Siccardi GL. The true ponte osteotomy: by the one who developed it. Spine Deform. 2018;6(1):2–11.
619. Pool RD. Congenital scoliosis in monozygotic twins. Genetically determined or acquired in utero? J Bone Joint Surg Br. 1986;68(2):194–6.
620. Potter BK, Kuklo TR, Lenke LG. Radiographic outcomes of anterior spinal fusion versus posterior spinal fusion with thoracic pedicle screws for treatment of Lenke Type I adolescent idiopathic scoliosis curves. Spine. 2005;30(16):1859–66.
621. Prahinski JR, Polly DW, McHale KA, et al. Occult intraspinal anomalies in congenital scoliosis. J Pediatr Orthop. 2000;20(1):59–63.
622. Pravda J, Ghelman B, Levine DB. Syringomyelia associated with congenital scoliosis. A case report. Spine. 1992;17(3):372–4.
623. Price CT, Scott DS, Reed FE Jr, et al. Nighttime bracing for adolescent idiopathic scoliosis with the Charleston bending brace. Preliminary report. Spine. 1990;15(12):1294–9.
624. Price CT, Scott DS, Reed FR Jr, et al. Nighttime bracing for adolescent idiopathic scoliosis with the Charleston Bending Brace: long-term follow-up. J Pediatr Orthop. 1997;17(6):703–7.
625. Privitera DM, Matsumoto H, Gomez JA, et al. Are breech rates for pedicle screws higher in the upper thoracic spine? Spine Deform. 2013;1(3):189–95.

626. Purkiss SB, Driscoll B, Cole WG, et al. Idiopathic scoliosis in families of children with congenital scoliosis. Clin Orthop Relat Res. 2002;(401):27–31.
627. Qiao J, Xu L, Liu Z, et al. Surgical treatment of scoliosis in Marfan syndrome: outcomes and complications. Eur Spine J. 2016;25(10):3288–93.
628. Qiao J, Zhu Z, Zhu F, et al. Indication for preoperative MRI of neural axis abnormalities in patients with presumed thoracolumbar/lumbar idiopathic scoliosis. Eur Spine J. 2013;22(2):360–6.
629. Quinn RH, Murray JN, Pezold R, et al. Management of patients with orthopaedic implants undergoing dental procedures. J Am Acad Orthop Surg. 2017;25(7):e138–41.
630. Quinn RH, Murray JN, Pezold R, et al. The American Academy of Orthopaedic Surgeons appropriate use criteria for the management of patients with orthopaedic implants undergoing dental procedures. J Bone Joint Surg Am. 2017;99(2):161–3.
631. Ramachandran M, Tsirikos AI, Lee J, et al. Whole-spine magnetic resonance imaging in patients with neurofibromatosis type 1 and spinal deformity. J Spinal Disord Tech. 2004;17(6):483–91.
632. Ramirez N, Johnston CE, Browne RH. The prevalence of back pain in children who have idiopathic scoliosis. J Bone Joint Surg Am. 1997;79(3):364–8.
633. Ramirez N, Richards BS, Warren PD, et al. Complications after posterior spinal fusion in Duchenne's muscular dystrophy. J Pediatr Orthop. 1997;17(1):109–14.
634. Ramo BA, Richards BS. Repeat surgical interventions following "definitive" instrumentation and fusion for idiopathic scoliosis: five-year update on a previously published cohort. Spine (Phila Pa 1976). 2012;37(14):1211–7.
635. Ramo BA, Roberts DW, Tuason D, et al. Surgical site infections after posterior spinal fusion for neuromuscular scoliosis: a thirty-year experience at a single institution. J Bone Joint Surg Am. 2014;96(24):2038–48.
636. Rampersaud YR, Pik JH, Salonen D, et al. Clinical accuracy of fluoroscopic computer-assisted pedicle screw fixation: a CT analysis. Spine. 2005;30(7):E183–90.
637. Ramsay J, Joncas J, Gilbert G, et al. Is breast asymmetry present in girls with adolescent idiopathic scoliosis? Spine Deform. 2014;2(5):374–9.
638. Raux S, Kohler R, Garin C, et al. Tridimensional trunk surface acquisition for brace manufacturing in idiopathic scoliosis. Eur Spine J. 2014;23(Suppl 4):S419–23.
639. Reames DL, Smith JS, Fu KM, et al. Complications in the surgical treatment of 19, 360 cases of pediatric scoliosis: a review of the Scoliosis Research Society Morbidity and Mortality database. Spine (Phila Pa 1976). 2011;36(18):1484–91.
640. Reid L. Pathologic changes in the lungs in scoliosis. In: Zorab PA, editor. Scoliosis. Springfield: Charles C. Thomas; 1969. p. 67.
641. Reuber M, Schultz A, McNeill T, et al. Trunk muscle myoelectric activities in idiopathic scoliosis. Spine. 1983;8(5):447–56.
642. Richards BR, Emara KM. Delayed infections after posterior TSRH spinal instrumentation for idiopathic scoliosis: revisited. Spine. 2001;26(18):1990–6.
643. Richards BS. Lumbar curve response in type II idiopathic scoliosis after posterior instrumentation of the thoracic curve. Spine. 1992;17(8 Suppl):S282–6.
644. Richards BS. Measurement error in assessment of vertebral rotation using the Perdriolle torsionmeter. Spine. 1992;17(5):513–7.
645. Richards BS. Delayed infections following posterior spinal instrumentation for the treatment of idiopathic scoliosis. J Bone Joint Surg Am. 1995;77(4):524–9.
646. Richards BS, Sucato DJ, Johnston CE, et al. Right thoracic curves in presumed adolescent idiopathic scoliosis: which clinical and radiographic findings correlate with a preoperative abnormal magnetic resonance image? Spine (Phila Pa 1976). 2010;35(20):1855–60.
647. Rideau Y, Glorion B, Delaubier A, et al. The treatment of scoliosis in Duchenne muscular dystrophy. Muscle Nerve. 1984;7(4):281–6.
648. Rigo M, Jelacic M. Brace technology thematic series: the 3D Rigo Cheneau-type brace. Scoliosis Spinal Disord. 2017;12:10.

649. Riley MS, Lenke LG, Chapman TM Jr, et al. Clinical and radiographic outcomes after posterior vertebral column resection for severe spinal deformity with five-year follow-up. J Bone Joint Surg Am. 2018;100(5):396–405.
650. Riseborough EJ, Wynne-Davies R. A genetic survey of idiopathic scoliosis in Boston, Massachusetts. J Bone Joint Surg Am. 1973;55(5):974–82.
651. Risser JC. The Iliac apophysis; an invaluable sign in the management of scoliosis. Clin Orthop. 1958;11:111–9.
652. Rittoo DB, Morris P. Tracheal occlusion in the prone position in an intubated patient with Duchenne muscular dystrophy. Anaesthesia. 1995;50(8):719–21.
653. Roberto R, Fritz A, Hagar Y, et al. The natural history of cardiac and pulmonary function decline in patients with duchenne muscular dystrophy. Spine (Phila Pa 1976). 2011;36(15):E1009–17.
654. Robin H, Damsin JP, Filipe G, et al. Spinal deformities in Marfan disease. Rev Chir Orthop Reparatrice Appar Mot. 1992;78(7):464–9.
655. Robins PR, Moe JH, Winter RB. Scoliosis in Marfan's syndrome. Its characteristics and results of treatment in thirty-five patients. J Bone Joint Surg Am. 1975;57(3):358–68.
656. Robinson CM, McMaster MJ. Juvenile idiopathic scoliosis. Curve patterns and prognosis in one hundred and nine patients. J Bone Joint Surg Am. 1996;78(8):1140–8.
657. Robinson D, Galasko CS, Delaney C, et al. Scoliosis and lung function in spinal muscular atrophy. Eur Spine J. 1995;4(5):268–73.
658. Rogala EJ, Drummond DS, Gurr J. Scoliosis: incidence and natural history. A prospective epidemiological study. J Bone Joint Surg Am. 1978;60(2):173–6.
659. Rowe DE, Bernstein SM, Riddick MF, et al. A meta-analysis of the efficacy of non-operative treatments for idiopathic scoliosis. J Bone Joint Surg Am. 1997;79(5):664–74.
660. Ruf M, Harms J. Hemivertebra resection by a posterior approach: innovative operative technique and first results. Spine. 2002;27(10):1116–23.
661. Ruf M, Harms J. Posterior hemivertebra resection with transpedicular instrumentation: early correction in children aged 1 to 6 years. Spine. 2003;28(18):2132–8.
662. Ruf M, Letko L, Matis N, et al. Effect of anterior mobilization and shortening in the correction of rigid idiopathic thoracic scoliosis. Spine (Phila Pa 1976). 2013;38(26):E1662–8.
663. Rushton PR, Elmalky M, Tikoo A, et al. The effect of metal density in thoracic adolescent idiopathic scoliosis. Eur Spine J. 2016;25(10):3324–30.
664. Sabato S, Rotman A, Robin GC, et al. Platelet aggregation abnormalities in idiopathic scoliosis. J Pediatr Orthop. 1985;5(5):558–63.
665. Sahgal V, Shah A, Flanagan N, et al. Morphologic and morphometric studies of muscle in idiopathic scoliosis. Acta Orthop Scand. 1983;54(2):242–51.
666. Saito N, Ebara S, Ohotsuka K, et al. Natural history of scoliosis in spastic cerebral palsy. Lancet. 1998;351(9117):1687–92.
667. Saito T, Kawakami N, Tsuji T, et al. Three-dimensional analysis of hemimetameric segmental shift in congenital scoliosis. J Pediatr Orthop. 2018;38(4):217–22.
668. Salehi LB, Mangino M, De Serio S, et al. Assignment of a locus for autosomal dominant idiopathic scoliosis (IS) to human chromosome 17p11. Hum Genet. 2002;111(4–5):401–4.
669. Salem KM, Goodger L, Bowyer K, et al. Does transcranial stimulation for motor evoked potentials (TcMEP) worsen seizures in epileptic patients following spinal deformity surgery? Eur Spine J. 2016;25(10):3044–8.
670. Sales de Gauzy J, Accadbled F, Sarramon MF, et al. Prenatal sonographic diagnosis of the congenital dislocated spine: a case report. Spine. 2003;28(2):E41–4.
671. Salsano V, Montanaro A, Turturro F. Casting. In: Weinstein SL, editor. The pediatric spine: principles and practice. New York: Raven Press; 1994. p. 1257.
672. Samagh SP, Cheng I, Elzik M, et al. Kyphectomy in the treatment of patients with myelomeningocele. Spine J. 2011;11(3):e5–11.
673. Samdani AF, Ames RJ, Kimball JS, et al. Anterior vertebral body tethering for immature adolescent idiopathic scoliosis: one-year results on the first 32 patients. Eur Spine J. 2015;24(7):1533–9.

674. Samdani AF, Belin EJ, Bennett JT, et al. Major perioperative complications after spine surgery in patients with cerebral palsy: assessment of risk factors. Eur Spine J. 2016;25(3):795–800.
675. Samdani AF, Bennett JT, Ames RJ, et al. Reversible intraoperative neurophysiologic monitoring alerts in patients undergoing arthrodesis for adolescent idiopathic scoliosis: what are the outcomes of surgery? J Bone Joint Surg Am. 2016;98(17):1478–83.
676. Samdani AF, Fine AL, Sagoo SS, et al. A patient with myelomeningocele: is untethering necessary prior to scoliosis correction? Neurosurg Focus. 2010;29(1):E8.
677. Samdani AF, Ranade A, Sciubba DM, et al. Accuracy of free-hand placement of thoracic pedicle screws in adolescent idiopathic scoliosis: how much of a difference does surgeon experience make? Eur Spine J. 2010;19(1):91–5.
678. Samilson RL, Bechard R. Scoliosis in cerebral palsy: incidence, distribution of curve patterns, natural history, and thoughts on etiology. Curr Pract Orthop Surg. 1973;5:183.
679. Sanders AE, Andras LM, Sousa T, et al. Accelerated discharge protocol for posterior spinal fusion patients with adolescent idiopathic scoliosis decreases hospital postoperative charges 22. Spine (Phila Pa 1976). 2017;42(2):92–7.
680. Sanders AE, Baumann R, Brown H, et al. Selective anterior fusion of thoracolumbar/lumbar curves in adolescents: when can the associated thoracic curve be left unfused? Spine. 2003;28(7):706–13; discussion 714.
681. Sanders JO, D'Astous J, Fitzgerald M, et al. Derotational casting for progressive infantile scoliosis. J Pediatr Orthop. 2009;29(6):581–7.
682. Sanders JO, Herring JA, Browne RH. Posterior arthrodesis and instrumentation in the immature (Risser-grade-0) spine in idiopathic scoliosis. J Bone Joint Surg Am. 1995;77(1):39–45.
683. Sanders JO, Khoury JG, Kishan S, et al. Predicting scoliosis progression from skeletal maturity: a simplified classification during adolescence. J Bone Joint Surg Am. 2008;90(3):540–53.
684. Sanders JO, Little DG, Richards BS. Prediction of the crankshaft phenomenon by peak height velocity. Spine. 1997;22(12):1352–6; discussion 1356–1357.
685. Sankar WN, Skaggs DL, Yazici M, et al. Lengthening of dual growing rods and the law of diminishing returns. Spine (Phila Pa 1976). 2011;36(10):806–9.
686. Sarwark JF. Kyphosis deformity in myelomeningocele. Orthop Clin North Am. 1999;30(3):451–5, viii–ix.
687. Schoenmakers MA, Gulmans VA, Gooskens RH, et al. Spinal fusion in children with spina bifida: influence on ambulation level and functional abilities. Eur Spine J. 2005;14(4):415–22.
688. Schreiber S, Parent EC, Moez EK, et al. The effect of Schroth exercises added to the standard of care on the quality of life and muscle endurance in adolescents with idiopathic scoliosis-an assessor and statistician blinded randomized controlled trial: "SOSORT 2015 Award Winner". Scoliosis. 2015;10:24.
689. Schroerlucke SR, Akbarnia BA, Pawelek JB, et al. How does thoracic kyphosis affect patient outcomes in growing rod surgery? Spine (Phila Pa 1976). 2012;37(15):1303–9.
690. Schulte TL, Liljenqvist U, Hierholzer E, et al. Spontaneous correction and derotation of secondary curves after selective anterior fusion of idiopathic scoliosis. Spine. 2006;31(3):315–21.
691. Schwartz DM, Auerbach JD, Dormans JP, et al. Neurophysiological detection of impending spinal cord injury during scoliosis surgery. J Bone Joint Surg Am. 2007;89(11):2440–9.
692. Schwartz DM, Drummond DS, Ecker ML. Influence of rigid spinal instrumentation on the neurogenic motor evoked potential. J Spinal Disord. 1996;9(5):439–45.
693. Schwend RM, Hennrikus W, Hall JE, et al. Childhood scoliosis: clinical indications for magnetic resonance imaging. J Bone Joint Surg Am. 1995;77(1):46–53.
694. Segal LS, Wallach DM, Kanev PM. Potential complications of posterior spine fusion and instrumentation in patients with cerebral palsy treated with intrathecal baclofen infusion. Spine (Phila Pa 1976). 2005;30(8):E219–24.
695. Sengupta DK, Mehdian SH, McConnell JR, et al. Pelvic or lumbar fixation for the surgical management of scoliosis in duchenne muscular dystrophy. Spine (Phila Pa 1976). 2002;27(18):2072–9.

696. Senkoylu A, Luk KD, Wong YW, et al. Prognosis of spontaneous thoracic curve correction after the selective anterior fusion of thoracolumbar/lumbar (Lenke 5C) curves in idiopathic scoliosis. Spine J. 2014;14(7):1117–24.
697. Sewell MD, Malagelada F, Wallace C, et al. A preliminary study to assess whether spinal fusion for scoliosis improves carer-assessed quality of life for children with GMFCS level IV or V cerebral palsy. J Pediatr Orthop. 2016;36(3):299–304.
698. Sewell MD, Wallace C, Malagelada F, et al. Does spinal fusion and scoliosis correction improve activity and participation for children with GMFCS level 4 and 5 cerebral palsy? Medicine (Baltimore). 2015;94(49):e1907.
699. Shaffer JW, Davy DT, Field GA, et al. The superiority of vascularized compared to non-vascularized rib grafts in spine surgery shown by biological and physical methods. Spine. 1988;(13):1150.
700. Shah SA, Dhawale AA, Oda JE, et al. Ponte osteotomies with pedicle screw instrumentation in the treatment of adolescent idiopathic scoliosis. Spine Deform. 2013;1(3):196–204.
701. Shapiro F, Sethna N. Blood loss in pediatric spine surgery. Eur Spine J. 2004;13(Suppl 1):S6–17.
702. Shapiro F, Sethna N, Colan S, et al. Spinal fusion in Duchenne muscular dystrophy: a multi-disciplinary approach. Muscle Nerve. 1992;15(5):604–14.
703. Shapiro F, Specht L. The diagnosis and orthopaedic treatment of childhood spinal muscular atrophy, peripheral neuropathy, Friedreich ataxia, and arthrogryposis. J Bone Joint Surg Am. 1993;75(11):1699–714.
704. Shapiro F, Zurakowski D, Bui T, et al. Progression of spinal deformity in wheelchair-dependent patients with Duchenne muscular dystrophy who are not treated with steroids: coronal plane (scoliosis) and sagittal plane (kyphosis, lordosis) deformity. Bone Joint J. 2014;96-B(1):100–5.
705. Shapiro F, Zurakowski D, Sethna NF. Tranexamic acid diminishes intraoperative blood loss and transfusion in spinal fusions for duchenne muscular dystrophy scoliosis. Spine (Phila Pa 1976). 2007;32(20):2278–83.
706. Shapiro J, Herring J. Congenital vertebral displacement. J Bone Joint Surg Am. 1993;75(5):656–62.
707. Sharma S, Gao X, Londono D, et al. Genome-wide association studies of adolescent idiopathic scoliosis suggest candidate susceptibility genes. Hum Mol Genet. 2011;20(7):1456–66.
708. Sharrard WJ. Posterior iliopsoas transplantation in the treatment of paralytic dislocation of the hip. J Bone Joint Surg Br. 1964;46:426–44.
709. Shaw BA, Watson TC, Merzel DI, et al. The safety of continuous epidural infusion for post-operative analgesia in pediatric spine surgery. J Pediatr Orthop. 1996;16(3):374–7.
710. Shen WJ, McDowell GS, Burke SW, et al. Routine preoperative MRI and SEP studies in adolescent idiopathic scoliosis. J Pediatr Orthop. 1996;16(3):350–3.
711. Shi Z, Chen J, Wang C, et al. Comparison of thoracoscopic anterior release combined with posterior spinal fusion versus posterior-only approach with an all-pedicle screw construct in the treatment of rigid thoracic adolescent idiopathic scoliosis. J Spinal Disord Tech. 2015;28(8):E454–9.
712. Shono Y, Abumi K, Kaneda K. One-stage posterior hemivertebra resection and correction using segmental posterior instrumentation. Spine. 2001;26(7):752–7.
713. Shufflebarger HL, Clark CE. Prevention of the crankshaft phenomenon. Spine. 1991;16(8 Suppl):S409–11.
714. Shyy W, Wang K, Gurnett CA, et al. Evaluation of GPR50, hMel-1B, and ROR-alpha melatonin-related receptors and the etiology of adolescent idiopathic scoliosis. J Pediatr Orthop. 2010;30(6):539–43.
715. Siller TA, Dickson JH, Erwin WD. Efficacy and cost considerations of intraoperative autologous transfusion in spinal fusion for idiopathic scoliosis with predeposited blood. Spine. 1996;21(7):848–52.
716. Simonds AK, Carroll N, Branthwaite MA. Kyphoscoliosis as a cause of cardio-respiratory failure--pitfalls of diagnosis. Respir Med. 1989;83(2):149–50.

717. Simpson MB, Georgopoulos G, Eilert RE. Intraoperative blood salvage in children and young adults undergoing spinal surgery with predeposited autologous blood: efficacy and cost effectiveness. J Pediatr Orthop. 1993;13(6):777–80.
718. Singh K, Samartzis D, An HS. Neurofibromatosis type I with severe dystrophic kyphoscoliosis and its operative management via a simultaneous anterior-posterior approach: a case report and review of the literature. Spine J. 2005;5(4):461–6.
719. Singla A, Bennett JT, Sponseller PD, et al. Results of selective thoracic versus nonselective fusion in Lenke type 3 curves. Spine (Phila Pa 1976). 2014;39(24):2034–41.
720. Sink EL, Karol LA, Sanders J, et al. Efficacy of perioperative halo-gravity traction in the treatment of severe scoliosis in children. J Pediatr Orthop. 2001;21(4):519–24.
721. Sirois JL 3rd, Drennan JC. Dystrophic spinal deformity in neurofibromatosis. J Pediatr Orthop. 1990;10(4):522–6.
722. Sitoula P, Verma K, Holmes L Jr, et al. Prediction of curve progression in idiopathic scoliosis: validation of the Sanders skeletal maturity staging system. Spine (Phila Pa 1976). 2015;40(13):1006–13.
723. Skaar DD, Park T, Swiontkowski MF, et al. Cost-effectiveness of antibiotic prophylaxis for dental patients with prosthetic joints: comparisons of antibiotic regimens for patients with total hip arthroplasty. J Am Dent Assoc. 2015;146(11):830–9.
724. Skaggs DL, Guillaume T, El-Hawary R, et al. Early onset scoliosis consensus statement, SRS Growing Spine Committee, 2015. Spine Deform. 2015;3(2):107.
725. Skaggs DL, Samuelson MA, Hale JM, et al. Complications of posterior iliac crest bone grafting in spine surgery in children. Spine. 2000;25(18):2400–2.
726. Skogland LB, Miller JA. Growth related hormones in idiopathic scoliosis. An endocrine basis for accelerated growth. Acta Orthop Scand. 1980;51(5):779–80.
727. Slover JD, Phillips MS, Iorio R, et al. Is routine antibiotic prophylaxis cost effective for total joint replacement patients? J Arthroplast. 2015;30(4):543–6.
728. Smania N, Picelli A, Romano M, et al. Neurophysiological basis of rehabilitation of adolescent idiopathic scoliosis. Disabil Rehabil. 2008;30(10):763–71.
729. Smith AD, Koreska J, Moseley CF. Progression of scoliosis in Duchenne muscular dystrophy. J Bone Joint Surg Am. 1989;71(7):1066–74.
730. Smith CA, Tuan RS. Human PAX gene expression and development of the vertebral column. Clin Orthop Relat Res. 1994;(302):241–50.
731. Smith JT, Gollogly S, Dunn HK. Simultaneous anterior-posterior approach through a costotransversectomy for the treatment of congenital kyphosis and acquired kyphoscoliotic deformities. J Bone Joint Surg Am. 2005;87(10):2281–9.
732. Smith JT, Novais E. Treatment of Gibbus deformity associated with myelomeningocele in the young child with use of the vertical expandable prosthetic titanium rib (VEPTR): a case report. J Bone Joint Surg Am. 2010;92(12):2211–5.
733. Smith MD. Congenital scoliosis of the cervical or cervicothoracic spine. Orthop Clin North Am. 1994;25(2):301–10.
734. Smith RM, Emans JB. Sitting balance in spinal deformity. Spine (Phila Pa 1976). 1992;17(9):1103–9.
735. Smorgick Y, Millgram MA, Anekstein Y, et al. Accuracy and safety of thoracic pedicle screw placement in spinal deformities. J Spinal Disord Tech. 2005;18(6):522–6.
736. Song KM, Little DG. Peak height velocity as a maturity indicator for males with idiopathic scoliosis. J Pediatr Orthop. 2000;20(3):286–8.
737. Song TB, Kim YH, Oh ST, et al. Prenatal ultrasonographic diagnosis of congenital kyphosis due to anterior segmentation failure. Asia Oceania J Obstet Gynaecol. 1994;20(1):31–3.
738. Spencer GS, Zorab PA. Spinal muscle in scoliosis. Part 1. Histology and histochemistry. J Neurol Sci. 1976;30(1):137–42.
739. Sponseller PD. Syringomyelia and Chiari I malformation presenting with juvenile scoliosis as sole manifestation. J Spinal Disord. 1992;5(2):237–9; discussion 239–244.
740. Sponseller PD, Ahn NU, Ahn UM, et al. Osseous anatomy of the lumbosacral spine in Marfan syndrome. Spine. 2000;25(21):2797–802.

741. Sponseller PD, Hobbs W, Riley LH 3rd, et al. The thoracolumbar spine in Marfan syndrome. J Bone Joint Surg Am. 1995;77(6):867–76.
742. Sponseller PD, LaPorte DM, Hungerford MW, et al. Deep wound infections after neuromuscular scoliosis surgery: a multicenter study of risk factors and treatment outcomes. Spine (Phila Pa 1976). 2000;25(19):2461–6.
743. Sponseller PD, Sethi N, Cameron DE, et al. Infantile scoliosis in Marfan syndrome. Spine. 1997;22(5):509–16.
744. Sponseller PD, Shah SA, Abel MF, et al. Infection rate after spine surgery in cerebral palsy is high and impairs results: multicenter analysis of risk factors and treatment. Clin Orthop Relat Res. 2010;468(3):711–6.
745. Sponseller PD, Young AT, Sarwark JF, et al. Anterior only fusion for scoliosis in patients with myelomeningocele. Clin Orthop Relat Res. 1999;(364):117–24.
746. Spoonamore MJ, Dolan LA, Weinstein SL. Use of the Rosenberger brace in the treatment of progressive adolescent idiopathic scoliosis. Spine. 2004;29(13):1458–64.
747. Sriram K, Bobechko WP, Hall JE. Surgical management of spinal deformities in spina bifida. J Bone Joint Surg Br. 1972;54(4):666–76.
748. Stagnara P. Cranial traction using the "Halo" of Rancho Los Amigos. Rev Chir Orthop Reparatrice Appar Mot. 1971;57(4):287–300.
749. Stark A, Saraste H. Anterior fusion insufficient for scoliosis in myelomeningocele. 8 children 2-6 years after the Zielke operation. Acta Orthop Scand. 1993;64(1):22–4.
750. Stella G, Ascani E, Cervellati S, et al. Surgical treatment of scoliosis associated with myelomeningocele. Eur J Pediatr Surg. 1998;8(Suppl 1):22–5.
751. Stephen JP, Sullivan MR, Hicks RG, et al. Cotrel-dubousset instrumentation in children using simultaneous motor and somatosensory evoked potential monitoring. Spine. 1996;21(21):2450–7.
752. Stoker GE, Lenke LG, Dorward IG. Posterior vertebral column resection for the treatment of dystrophic kyphosis associated with type-1 neurofibromatosis: a case report and review of the literature. Spine (Phila Pa 1976). 2012;37(26):E1659–64.
753. Storer SK, Vitale MG, Hyman JE, et al. Correction of adolescent idiopathic scoliosis using thoracic pedicle screw fixation versus hook constructs. J Pediatr Orthop. 2005;25(4):415–9.
754. Stricker SJ, Sher JS. Freeze-dried cortical allograft in posterior spinal arthrodesis: use with segmental instrumentation for idiopathic adolescent scoliosis. Orthopedics. 1997;20(11):1039–43.
755. Sturm PF, Chung R, Bomze SR. Hemivertebra in monozygotic twins. Spine. 2001;26(12):1389–91.
756. Sucato DJ. Management of severe spinal deformity: scoliosis and kyphosis. Spine (Phila Pa 1976). 2010;35(25):2186–92.
757. Sucato DJ, Duchene C. The position of the aorta relative to the spine: a comparison of patients with and without idiopathic scoliosis. J Bone Joint Surg Am. 2003;85-A(8):1461–9.
758. Sucato DJ, Duey-Holtz A, Elerson E, et al. Postoperative analgesia following surgical correction for adolescent idiopathic scoliosis: a comparison of continuous epidural analgesia and patient-controlled analgesia. Spine. 2005;30(2):211–7.
759. Sucato DJ, Erken YH, Davis S, et al. Prone thoracoscopic release does not adversely affect pulmonary function when added to a posterior spinal fusion for severe spine deformity. Spine (Phila Pa 1976). 2009;34(8):771–8.
760. Sucato DJ, Hedequist D, Karol LA. Operative correction of adolescent idiopathic scoliosis in male patients. A radiographic and functional outcome comparison with female patients. J Bone Joint Surg Am. 2004;86-A(9):2005–14.
761. Sucato DJ, Kassab F, Dempsey M. Analysis of screw placement relative to the aorta and spinal canal following anterior instrumentation for thoracic idiopathic scoliosis. Spine. 2004;29(5):554–9; discussion 559.
762. Sucato DJ, Lovejoy JF, Agrawal S, et al. Postoperative ketorolac does not predispose to pseudoarthrosis following posterior spinal fusion and instrumentation for adolescent idiopathic scoliosis. Spine (Phila Pa 1976). 2008;33(10):1119–24.

763. Sucato DJ, Poppino K, AS T, et al. Neuromonitoring for AIS: a 20-year analysis of AIS patient incidence of critical changes and predictive factors to define patients at risk at a single center. International Meeting of Advanced Spine Techniques, Capetown, South Africa; 2017.
764. Sudo H, Ito M, Kaneda K, et al. Long-term outcomes of anterior dual-rod instrumentation for thoracolumbar and lumbar curves in adolescent idiopathic scoliosis: a twelve to twenty-three-year follow-up study. J Bone Joint Surg Am. 2013;95(8):e49.
765. Suh SW, Sarwark JF, Vora A, et al. Evaluating congenital spine deformities for intraspinal anomalies with magnetic resonance imaging. J Pediatr Orthop. 2001;21(4):525–31.
766. Sui WY, Ye F, Yang JL. Efficacy of tranexamic acid in reducing allogeneic blood products in adolescent idiopathic scoliosis surgery. BMC Musculoskelet Disord. 2016;17:187.
767. Suk SI. Pedicle screw instrumentation for adolescent idiopathic scoliosis: the insertion technique, the fusion levels and direct vertebral rotation. Clin Orthop Surg. 2011;3(2):89–100.
768. Suk SI, Chung ER, Kim JH, et al. Posterior vertebral column resection for severe rigid scoliosis. Spine (Phila Pa 1976). 2005;30(14):1682–7.
769. Suk SI, Kim WJ, Lee SM, et al. Thoracic pedicle screw fixation in spinal deformities: are they really safe? Spine. 2001;26(18):2049–57.
770. Suk SI, Lee CK, Chung SS. Comparison of Zielke ventral derotation system and Cotrel-Dubousset instrumentation in the treatment of idiopathic lumbar and thoracolumbar scoliosis. Spine. 1994;19(4):419–29.
771. Suk SI, Lee CK, Kim WJ, et al. Segmental pedicle screw fixation in the treatment of thoracic idiopathic scoliosis. Spine. 1995;20(12):1399–405.
772. Suk SI, Lee SM, Chung ER, et al. Selective thoracic fusion with segmental pedicle screw fixation in the treatment of thoracic idiopathic scoliosis: more than 5-year follow-up. Spine. 2005;30(14):1602–9.
773. Sun Z, Qiu G, Zhao Y, et al. Lowest instrumented vertebrae selection for selective posterior fusion of moderate thoracolumbar/lumbar idiopathic scoliosis: lower-end vertebra or lower-end vertebra+1? Eur Spine J. 2014;23(6):1251–7.
774. Sussman M. Duchenne muscular dystrophy. J Am Acad Orthop Surg. 2002;10(2):138–51.
775. Sussman MD. Advantage of early spinal stabilization and fusion in patients with Duchenne muscular dystrophy. J Pediatr Orthop. 1984;4(5):532–7.
776. Sussman MD. Treatment of scoliosis in Duchenne muscular dystrophy. Dev Med Child Neurol. 1985;27(4):522–4.
777. Suzuki S, Kasahara Y, Yamamoto S, et al. Three-dimensional spinal deformity in scoliosis associated with cerebral palsy and with progressive muscular dystrophy. Spine (Phila Pa 1976). 1993;18(15):2290–4.
778. Szoke G, Lipton G, Miller F, et al. Wound infection after spinal fusion in children with cerebral palsy. J Pediatr Orthop. 1998;18(6):727–33.
779. Tabaraud F, Boulesteix JM, Moulies D, et al. Monitoring of the motor pathway during spinal surgery. Spine. 1993;18(5):546–50.
780. Takaso M, Nakazawa T, Imura T, et al. Surgical management of severe scoliosis with high risk pulmonary dysfunction in Duchenne muscular dystrophy: patient function, quality of life and satisfaction. Int Orthop. 2010;34(5):695–702.
781. Tallroth K, Malmivaara A, Laitinen ML, et al. Lumbar spine in Marfan syndrome. Skelet Radiol. 1995;24(5):337–40.
782. Tanner J. Growth and endocrinology of the adolescent. In: Gardner L, editor. Endocrine and Genetic diseases of childhood. Phildelphia: W.B. Saunders; 1975. p. 14.
783. Tanner J, Whitehouse RH, Takaishi M. Standards from birth to maturity for height, weight, height velocity, and weight velocity: British children. Parts I and II. Arch Dis Child. 1966;(41):454.
784. Tao F, Zhao Y, Wu Y, et al. The effect of differing spinal fusion instrumentation on the occurrence of postoperative crankshaft phenomenon in adolescent idiopathic scoliosis. J Spinal Disord Tech. 2010;23(8):e75–80.

785. Tauchi R, Kawakami N, Castro MA, et al. Long-term surgical outcomes after early definitive spinal fusion for early-onset scoliosis with neurofibromatosis type 1 at mean follow-up of 14 years. J Pediatr Orthop. 2020;40(1):42–7.
786. Tauchi R, Tsuji T, Cahill PJ, et al. Reliability analysis of cobb angle measurements of congenital scoliosis using X-ray and 3D-CT images. Eur J Orthop Surg Traumatol. 2016;26(1):53–7.
787. Tello C, Bersusky E, Francheri-Wilson A, et al. Severe infantile socliosis treated by repetitive distractions followed by definitive arthrodesis. Eur Spine J. 2005, in press.
788. Terek RM, Wehner J, Lubicky JP. Crankshaft phenomenon in congenital scoliosis: a preliminary report. J Pediatr Orthop. 1991;11(4):527–32.
789. Theiss SM, Smith MD, Winter RB. The long-term follow-up of patients with Klippel-Feil syndrome and congenital scoliosis. Spine. 1997;22(11):1219–22.
790. Theologis AA, Tabaraee E, Lin T, et al. Type of bone graft or substitute does not affect outcome of spine fusion with instrumentation for adolescent idiopathic scoliosis. Spine (Phila Pa 1976). 2015;40(17):1345–51.
791. Theroux J, Stomski N, Hodgetts CJ, et al. Prevalence of low back pain in adolescents with idiopathic scoliosis: a systematic review. Chiropr Man Ther. 2017;25:10.
792. Thirumala PD, Crammond DJ, Loke YK, et al. Diagnostic accuracy of motor evoked potentials to detect neurological deficit during idiopathic scoliosis correction: a systematic review. J Neurosurg Spine. 2017;26(3):374–83.
793. Thometz JG, Simon SR. Progression of scoliosis after skeletal maturity in institutionalized adults who have cerebral palsy. J Bone Joint Surg Am. 1988;70(9):1290–6.
794. Thompson GH, Akbarnia BA, Kostial P, et al. Comparison of single and dual growing rod techniques followed through definitive surgery: a preliminary study. Spine. 2005;30(18):2039–44.
795. Thompson RM, Hubbard EW, Jo CH, et al. Brace success is related to curve type in patients with adolescent idiopathic scoliosis. J Bone Joint Surg Am. 2017;99(11):923–8.
796. Thomsen M, Lang RD, Carstens C. Results of kyphectomy with the technique of Warner and Fackler in children with myelodysplasia. J Pediatr Orthop B. 2000;9(3):143–7.
797. Thomsen MN, Schneider U, Weber M, et al. Scoliosis and congenital anomalies associated with Klippel-Feil syndrome types I-III. Spine. 1997;22(4):396–401.
798. Thuet ED, Winscher JC, Padberg AM, et al. Validity and reliability of intraoperative monitoring in pediatric spinal deformity surgery: a 23-year experience of 3436 surgical cases. Spine (Phila Pa 1976). 2010;35(20):1880–6.
799. Tomlinson RJ Jr, Wolfe MW, Nadall JM, et al. Syringomyelia and developmental scoliosis. J Pediatr Orthop. 1994;14(5):580–5.
800. Torode I, Godette G. Surgical correction of congenital kyphosis in myelomeningocele. J Pediatr Orthop. 1995;15(2):202–5.
801. Tortori-Donati P, Fondelli MP, Rossi A, et al. Segmental spinal dysgenesis: neuroradiologic findings with clinical and embryologic correlation. AJNR Am J Neuroradiol. 1999;20(3):445–56.
802. Trenga AP, Singla A, Feger MA, et al. Patterns of congenital bony spinal deformity and associated neural anomalies on X-ray and magnetic resonance imaging. J Child Orthop. 2016;10(4):343–52.
803. Trivedi JM, Thomson JD. Results of Charleston bracing in skeletally immature patients with idiopathic scoliosis. J Pediatr Orthop. 2001;21(3):277–80.
804. Tsirikos AI, Chang WN, Dabney KW, et al. Comparison of one-stage versus two-stage anteroposterior spinal fusion in pediatric patients with cerebral palsy and neuromuscular scoliosis. Spine (Phila Pa 1976). 2003;28(12):1300–5.
805. Tsirikos AI, Chang WN, Dabney KW, et al. Comparison of parents' and caregivers' satisfaction after spinal fusion in children with cerebral palsy. J Pediatr Orthop. 2004;24(1):54–8.
806. Tsirikos AI, Mains E. Surgical correction of spinal deformity in patients with cerebral palsy using pedicle screw instrumentation. J Spinal Disord Tech. 2012;25(7):401–8.
807. Tsirikos AI, McMaster MJ. Infantile developmental thoracolumbar kyphosis with segmental subluxation of the spine. J Bone Joint Surg Br. 2010;92(3):430–5.

808. Tsirikos AI, Ramachandran M, Lee J, et al. Assessment of vertebral scalloping in neurofibromatosis type 1 with plain radiography and MRI. Clin Radiol. 2004;59(11):1009–17.
809. Tsirikos AI, Saifuddin A, Noordeen MH. Spinal deformity in neurofibromatosis type-1: diagnosis and treatment. Eur Spine J. 2005;14(5):427–39.
810. Tubbs RS, Rutledge SL, Kosentka A, et al. Chiari I malformation and neurofibromatosis type 1. Pediatr Neurol. 2004;30(4):278–80.
811. Turgut M, Yenisey C, Uysal A, et al. The effects of pineal gland transplantation on the production of spinal deformity and serum melatonin level following pinealectomy in the chicken. Eur Spine J. 2003;12(5):487–94.
812. Turi M, Johnston CE 2nd, Richards BS. Anterior correction of idiopathic scoliosis using TSRH instrumentation. Spine. 1993;18(4):417–22.
813. Turturro F, Rocca B, Gumina S, et al. Impaired primary hemostasis with normal platelet function in Duchenne muscular dystrophy during highly-invasive spinal surgery. Neuromuscul Disord. 2005;15(8):532–40.
814. Urban MK, Beckman J, Gordon M, et al. The efficacy of antifibrinolytics in the reduction of blood loss during complex adult reconstructive spine surgery. Spine. 2001;26(10):1152–6.
815. Urbanski W, Jurasz W, Wolanczyk M, et al. Increased radiation but no benefits in pedicle screw accuracy with navigation versus a freehand technique in scoliosis surgery. Clin Orthop Relat Res. 2018;476(5):1020–7.
816. Vaccaro AR, Rizzolo SJ, Allardyce TJ, et al. Placement of pedicle screws in the thoracic spine. Part I: morphometric analysis of the thoracic vertebrae. J Bone Joint Surg Am. 1995;77(8):1193–9.
817. Vaccaro AR, Rizzolo SJ, Balderston RA, et al. Placement of pedicle screws in the thoracic spine. Part II: an anatomical and radiographic assessment. J Bone Joint Surg Am. 1995;77(8):1200–6.
818. Velasco MV, Colin AA, Zurakowski D, et al. Posterior spinal fusion for scoliosis in duchenne muscular dystrophy diminishes the rate of respiratory decline. Spine (Phila Pa 1976). 2007;32(4):459–65.
819. Venn G, Mehta MH, Mason RM. Solubility of spinal ligament collagen in idiopathic and secondary scoliosis. Clin Orthop Relat Res. 1983;(177):294–301.
820. Verma K, Sitoula P, Gabos P, et al. Simplified skeletal maturity scoring system: learning curve and methods to improve reliability. Spine (Phila Pa 1976). 2014;39(26):E1592–8.
821. Viau M, Tarbox BB, Wonglertsiri S, et al. Thoracic pedicle screw instrumentation using the "Funnel Technique": part 2. Clinical experience. J Spinal Disord Tech. 2002;15(6):450–3.
822. Viehweger E, Giacomelli MC, Glard Y, et al. Congenital dislocated spine: implications for orthopaedic management. J Pediatr Orthop. 2009;29(4):362–8.
823. Viola RW, King HA, Adler SM, et al. Delayed infection after elective spinal instrumentation and fusion. A retrospective analysis of eight cases. Spine. 1997;22(20):2444–50; discussion 2450–2441.,.
824. Violas P, Chapuis M, Bracq H. Local autograft bone in the surgical management of adolescent idiopathic scoliosis. Spine. 2004;29(2):189–92.
825. Vira S, Husain Q, Jalai C, et al. The interobserver and intraobserver reliability of the Sanders classification versus the Risser stage. J Pediatr Orthop. 2017;37(4):e246–9.
826. Vitale MG, Choe JC, Hwang MW, et al. Use of ketorolac tromethamine in children undergoing scoliosis surgery. An analysis of complications. Spine J. 2003;3(1):55–62.
827. Vitale MG, Matsumoto H, Bye MR, et al. A retrospective cohort study of pulmonary function, radiographic measures, and quality of life in children with congenital scoliosis: an evaluation of patient outcomes after early spinal fusion. Spine (Phila Pa 1976). 2008;33(11):1242–9.
828. Vitale MG, Matsumoto H, Feinberg N, et al. Paper#35 proximal rib vs proximal spine anchors in growing rods: a multicenter prospective cohort study. Spine Deform. 2015;6(3):626–7.
829. Vitale MG, Skaggs DL, Pace GI, et al. Best practices in intraoperative neuromonitoring in spine deformity surgery: development of an intraoperative checklist to optimize response. Spine Deform. 2014;2(5):333–9.

830. Vivas AC, Pahys JM, Jain A, et al. Early and late hospital readmissions after spine deformity surgery in children with cerebral palsy. Spine Deform. 2020;8(3):507–16.
831. Waisman M, Saute M. Thoracoscopic spine release before posterior instrumentation in scoliosis. Clin Orthop Relat Res. 1997;(336):130–6.
832. Wajchenberg M, Lazar M, Cavacana N, et al. Genetic aspects of adolescent idiopathic scoliosis in a family with multiple affected members: a research article. Scoliosis. 2010;5:7.
833. Wang F, Xu XM, Wei XZ, et al. Spontaneous thoracic curve correction after selective posterior fusion of thoracolumbar/lumbar curves in Lenke 5C adolescent idiopathic scoliosis. Medicine (Baltimore). 2015;94(29):e1155.
834. Wang T, Zeng B, Xu J, et al. Radiographic evaluation of selective anterior thoracolumbar or lumbar fusion for adolescent idiopathic scoliosis. Eur Spine J. 2008;17(8):1012–8.
835. Wang WW, Man GC, Wong JH, et al. Abnormal response of the proliferation and differentiation of growth plate chondrocytes to melatonin in adolescent idiopathic scoliosis. Int J Mol Sci. 2014;15(9):17100–14.
836. Wang Y, Fei Q, Qiu G, et al. Anterior spinal fusion versus posterior spinal fusion for moderate lumbar/thoracolumbar adolescent idiopathic scoliosis: a prospective study. Spine (Phila Pa 1976). 2008;33(20):2166–72.
837. Wang Z, Fu C, Leng J, et al. Treatment of dystrophic scoliosis in neurofibromatosis Type 1 with one-stage posterior pedicle screw technique. Spine J. 2015;15(4):587–95.
838. Ward JP, Feldman DS, Paul J, et al. Wound closure in nonidiopathic scoliosis: does closure matter? J Pediatr Orthop. 2017;37(3):166–70.
839. Ward WT, Wenger DR, Roach JW. Surgical correction of myelomeningocele scoliosis: a critical appraisal of various spinal instrumentation systems. J Pediatr Orthop. 1989;9(3):262–8.
840. Warner WC Jr, Fackler CD. Comparison of two instrumentation techniques in treatment of lumbar kyphosis in myelodysplasia. J Pediatr Orthop. 1993;13(6):704–8.
841. Watanabe K, Uno K, Suzuki T, et al. Risk factors for complications associated with growing-rod surgery for early-onset scoliosis. Spine (Phila Pa 1976). 2013;38(8):E464–8.
842. Watkins RGT, Hussain N, Freeman BJ, et al. Anterior instrumentation for thoracolumbar adolescent idiopathic scoliosis: do structural interbody grafts preserve sagittal alignment better than morselized rib autografts? Spine (Phila Pa 1976). 2006;31(20):2337–42.
843. Weimann RL, Gibson DA, Moseley CF, et al. Surgical stabilization of the spine in Duchenne muscular dystrophy. Spine (Phila Pa 1976). 1983;8(7):776–80.
844. Weinstein S. Natural history of idiopathic scoliosis: Back pain at 50-year follow-up. New York: Scoliosis Research Society; 1998.
845. Weinstein S. Untreated adolescent idiopathic scoliosis: psychosocial characteristics at 50-year follow-up. New York: Scoliosis Research Society; 1998.
846. Weinstein SL, Dolan LA, Wright JG, et al. Effects of bracing in adolescents with idiopathic scoliosis. N Engl J Med. 2013;369(16):1512–21.
847. Weinstein SL, Ponseti IV. Curve progression in idiopathic scoliosis. J Bone Joint Surg Am. 1983;65(4):447–55.
848. Werdnig G. Zwei fruhim famile hereditare Fall von progressiver Muskelatrophie unter dem Bild der Dystrophie, aber auf neurotischer Grundlage. Arch Psychiatr. 1891;22:237.
849. Wilde PH, Upadhyay SS, Leong JC. Deterioration of operative correction in dystrophic spinal neurofibromatosis. Spine. 1994;19(11):1264–70.
850. Williams BA, Matsumoto H, McCalla DJ, et al. Development and initial validation of the Classification of Early-Onset Scoliosis (C-EOS). J Bone Joint Surg Am. 2014;96(16):1359–67. https://doi.org/10.2106/JBJS.M.00253.
851. Wimmer C, Gluch H. Management of postoperative wound infection in posterior spinal fusion with instrumentation. J Spinal Disord. 1996;9(6):505–8.
852. Winter R. Congenital spinal deformity. In: Lonstein J, Winter R, Ogilvie J, editors. Moe's text book of scoliosis and other spinal deformities. Phildelphia: W.B. Saunders; 1994. p. 257.
853. Winter R. Marfan's syndrome. In: Lonstein J, Winter R, Ogilvie J, editors. Moe's text book of scoliosis and other spinal deformities. Phildelphia: W.B. Saunders; 1994. p. 547.

854. Winter R, Moe JH, Lonstein J. A review of family histories in patients with congenital spinal deformities. Orthop Trans. 1983;(7):32.
855. Winter RB. Congenital scoliosis. Clin Orthop Relat Res. 1973;(93):75–94.
856. Winter RB. The idiopathic double thoracic curve pattern. Its recognition and surgical management. Spine. 1989;14(12):1287–92.
857. Winter RB, Denis F. The King V curve pattern. Its analysis and surgical treatment. Orthop Clin North Am. 1994;25(2):353–62.
858. Winter RB, Lonstein JE. Congenital scoliosis with posterior spinal arthrodesis T2-L3 at age 3 years with 41-year follow-up. A case report. Spine. 1999;24(2):194–7.
859. Winter RB, Lonstein JE, Boachie-Adjei O. Congenital spinal deformity. Instr Course Lect. 1996;45:117–27.
860. Winter RB, Lonstein JE, Heithoff KB, et al. Magnetic resonance imaging evaluation of the adolescent patient with idiopathic scoliosis before spinal instrumentation and fusion. A prospective, double-blinded study of 140 patients. Spine. 1997;22(8):855–8.
861. Winter RB, Moe JH. The results of spinal arthrodesis for congenital spinal deformity in patients younger than five years old. J Bone Joint Surg Am. 1982;64(3):419–32.
862. Winter RB, Moe JH, Bradford DS, et al. Spine deformity in neurofibromatosis. A review of one hundred and two patients. J Bone Joint Surg Am. 1979;61(5):677–94.
863. Winter RB, Moe JH, Lonstein JE. Posterior spinal arthrodesis for congenital scoliosis. An analysis of the cases of two hundred and ninety patients, five to nineteen years old. J Bone Joint Surg Am. 1984;66(8):1188–97.
864. Winter RB, Moe JH, Wang JF. Congenital kyphosis. Its natural history and treatment as observed in a study of one hundred and thirty patients. J Bone Joint Surg Am. 1973;55(2):223–56.
865. Winter RB, Smith MD, Lonstein JE. Congenital scoliosis due to unilateral unsegmented bar: posterior spine fusion at age 12 months with 44-year follow-up. Spine. 2004;29(3):E52–5.
866. Winter RB, Turek-Shay LA. Twenty-eight-year follow-up of anterior and posterior fusion for congenital kyphosis. A case report. Spine. 1997;22(18):2183–7.
867. Winter S. Preoperative assessment of the child with neuromuscular scoliosis. Orthop Clin North Am. 1994;25(2):239–45.
868. Wirth B, Rudnik-Schoneborn S, Hahnen E, et al. Prenatal prediction in families with autosomal recessive proximal spinal muscular atrophy (5q11.2-q13.3): molecular genetics and clinical experience in 109 cases. Prenat Diagn. 1995;15(5):407–17.
869. Wise CA, Barnes R, Gillum J, et al. Localization of susceptibility to familial idiopathic scoliosis. Spine. 2000;25(18):2372–80.
870. Wise CA, Gao X, Shoemaker S, et al. Understanding genetic factors in idiopathic scoliosis, a complex disease of childhood. Curr Genomics. 2008;9(1):51–9.
871. Wong AYL, Samartzis D, Cheung PWH, et al. How common is back pain and what biopsychosocial factors are associated with back pain in patients with adolescent idiopathic scoliosis? Clin Orthop Relat Res. 2019;477(4):676–86.
872. Wong HK, Hee HT, Yu Z, et al. Results of thoracoscopic instrumented fusion versus conventional posterior instrumented fusion in adolescent idiopathic scoliosis undergoing selective thoracic fusion. Spine. 2004;29(18):2031–8; discussion 2039.
873. Wong MS, Cheng JC, Lam TP, et al. The effect of rigid versus flexible spinal orthosis on the clinical efficacy and acceptance of the patients with adolescent idiopathic scoliosis. Spine (Phila Pa 1976). 2008;33(12):1360–5.
874. Worthington V, Shambaugh P. Nutrition as an environmental factor in the etiology of idiopathic scoliosis. J Manip Physiol Ther. 1993;16(3):169–73.
875. Wyatt MP, Barrack RL, Mubarak SJ, et al. Vibratory response in idiopathic scoliosis. J Bone Joint Surg Br. 1986;68(5):714–8.
876. Wynne-Davies R. Familial (idiopathic) scoliosis. A family survey. J Bone Joint Surg Br. 1968;50(1):24–30.
877. Wynne-Davies R. Congenital vertebral anomalies: aetiology and relationship to spina bifida cystica. J Med Genet. 1975;12(3):280–8.

878. Xu H, Qiu G, Wu Z, et al. Expression of transforming growth factor and basic fibroblast growth factor and core protein of proteoglycan in human vertebral cartilaginous endplate of adolescent idiopathic scoliosis. Spine. 2005;30(17):1973–8.
879. Xu W, Yang S, Wu X, et al. Hemivertebra excision with short-segment spinal fusion through combined anterior and posterior approaches for congenital spinal deformities in children. J Pediatr Orthop B. 2010;19(6):545–50.
880. Yagi M, Hasegawa J, Nagoshi N, et al. Does the intraoperative tranexamic acid decrease operative blood loss during posterior spinal fusion for treatment of adolescent idiopathic scoliosis? Spine (Phila Pa 1976). 2012;37(21):E1336–42.
881. Yalcin N, Bar-on E, Yazici M. Impingement of spinal cord by dislocated rib in dystrophic scoliosis secondary to neurofibromatosis type 1: radiological signs and management strategies. Spine (Phila Pa 1976). 2008;33(23):E881–6.
882. Yang J, Skaggs DL, Chan P, et al. Raising mean arterial pressure alone restores 20% of intraoperative neuromonitoring losses. Spine (Phila Pa 1976). 2018;43(13):890–4.
883. Yang J, Sponseller P, Thompson GH, et al. Risk factors for rod fracture in growing rod surgery. J Child Orthop. 2009;3(2):147.
884. Yasuda YL, Bowman K, Hsu JD. Mobile arm supports: criteria for successful use in muscle disease patients. Arch Phys Med Rehabil. 1986;67(4):253–6.
885. Yaszay B, Bastrom TP, Bartley CE, et al. The effects of the three-dimensional deformity of adolescent idiopathic scoliosis on pulmonary function. Eur Spine J. 2017;26(6):1658–64.
886. Yaszay B, O'Brien M, Shufflebarger HL, et al. Efficacy of hemivertebra resection for congenital scoliosis: a multicenter retrospective comparison of three surgical techniques. Spine (Phila Pa 1976). 2011;36(24):2052–60.
887. Yazici M, Asher MA, Hardacker JW. The safety and efficacy of Isola-Galveston instrumentation and arthrodesis in the treatment of neuromuscular spinal deformities. J Bone Joint Surg Am. 2000;82(4):524–43.
888. Yngve D. Abdominal reflexes. J Pediatr Orthop. 1997;17(1):105–8.
889. Yoshihara H, Kawakami N, Matsuyama Y, et al. A histomorphologic study of scoliosis in pinealectomized chickens. Spine. 2005;30(20):2244–51.
890. Youkilis AS, Quint DJ, McGillicuddy JE, et al. Stereotactic navigation for placement of pedicle screws in the thoracic spine. Neurosurgery. 2001;48(4):771–8; discussion 778–779.
891. Zaarour C, Engelhardt T, Strantzas S, et al. Effect of low-dose ketamine on voltage requirement for transcranial electrical motor evoked potentials in children. Spine (Phila Pa 1976). 2007;32(22):E627–30.
892. Zaina F, Negrini S, Atanasio S, et al. Specific exercises performed in the period of brace weaning can avoid loss of correction in Adolescent Idiopathic Scoliosis (AIS) patients: Winner of SOSORT's 2008 Award for Best Clinical Paper. Scoliosis. 2009;4:8.
893. Zamecnik J, Krskova L, Hacek J, et al. Etiopathogenesis of adolescent idiopathic scoliosis: expression of melatonin receptors 1A/1B, calmodulin and estrogen receptor 2 in deep paravertebral muscles revisited. Mol Med Rep. 2016;14(6):5719–24.
894. Zana E, Chalard F, Mazda K, et al. An atypical case of segmental spinal dysgenesis. Pediatr Radiol. 2005;35(9):914–7.
895. Zapata K, Parent EC, Sucato D. Immediate effects of scoliosis-specific corrective exercises on the cobb angle after one week and after one year of practice. Scoliosis Spinal Disord. 2016;11(Suppl 2):36.
896. Zarzycki D, Rymarczyk A, Bakalarek B, et al. Surgical treatment of congenital vertebral displacement Type A in the sagittal plane only: a retrospective study involving eleven cases. Spine. 2002;27(1):72–7.
897. Zeller RD, Ghanem I, Dubousset J. The congenital dislocated spine. Spine. 1996;21(10):1235–40.
898. Zetterberg C, Aniansson A, Grimby G. Morphology of the paravertebral muscles in adolescent idiopathic scoliosis. Spine. 1983;8(5):457–62.

899. Zhang W, Sha S, Xu L, et al. The prevalence of intraspinal anomalies in infantile and juvenile patients with “presumed idiopathic” scoliosis: a MRI-based analysis of 504 patients. BMC Musculoskelet Disord. 2016;17:189.
900. Zindrick MR, Knight GW, Sartori MJ, et al. Pedicle morphology of the immature thoracolumbar spine. Spine. 2000;25(21):2726–35.
901. Zindrick MR, Wiltse LL, Doornik A, et al. Analysis of the morphometric characteristics of the thoracic and lumbar pedicles. Spine. 1987;12(2):160–6.
902. Beauchamp EC, Anderson RCE, Vitale MG. Modern Surgical management of early onset and adolescent idiopathic scoliosis. Neurosurgery. 2019;84(2):291–304. https://doi.org/10.1093/neuros/nyy267. PMID: 30016462.

Anesthesia for Pediatric Spinal Deformity

29

Christopher Bryan McLeod

Natural History

The historical origin of "scoliosis" is derived from the Greek word "skolios" (crooked or curved) [1]. Scoliosis can be described as a curvature in the coronal plane of 10 degrees or greater. Due to curvature of the spine, there is a rotational component of the vertebrae as well [2]. Rotation of the spinous process occurs towards the concave side of the curvature and the vertebral body will rotate towards the convex side. In turn, this rotation forces the ribs posteriorly on the convex side and can produce a rib hump deformity [3]. On the concave side, the ribs are pushed laterally and anteriorly, thus causing restriction in chest wall compliance (Fig. 29.1). The lungs on the concave side are able to achieve a normal end-expiratory position but unable to achieve normal end-inspiratory position. The hemi-thorax on the convex side can achieve normal end-inspiratory position, but unable to achieve normal end-expiratory position [4]. Furthermore, dynamic breathing MRI has shown the main factor in respiratory dysfunction in scoliosis is limited chest wall motion. Diaphragm function remains normal. [5]

Classification of the severity of the curvature relies upon the Cobb angle [6]. A line is drawn parallel to the upper end of the proximal vertebrae of the curvature and a line drawn parallel to the lower end of the distal vertebrae of the curvature. Two more lines are drawn perpendicular to each of these, so they intersect. The angle of this intersection is then measured in degrees (Fig. 29.2). If the scoliosis progresses, characteristic cardiopulmonary changes occur such as restrictive lung disease, dyspnea on exertion and eventual right heart strain when curvature reaches 100 degrees or greater [7].

C. B. McLeod (✉)
Scottish Rite for Children, Dallas, TX, USA
e-mail: Christopher.McLeod@tsrh.org

© The Author(s), under exclusive license to Springer Nature Switzerland AG 2022

C. E. Noe (ed.), *Multidisciplinary Spine Care*,
https://doi.org/10.1007/978-3-031-04990-3_29

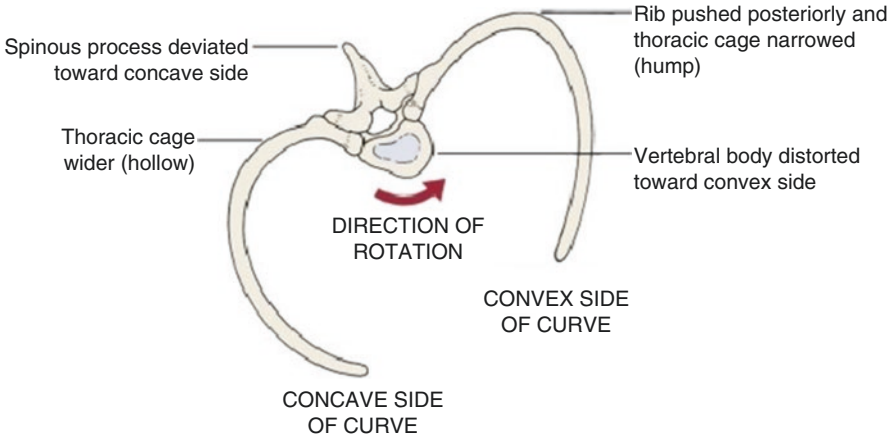


Fig. 29.1 Pathological changes in the ribs and vertebra with idiopathic scoliosis in the thoracic spine. (From Magee DJ. Orthopedic physical assessment: Elsevier Health Sciences; 2013)

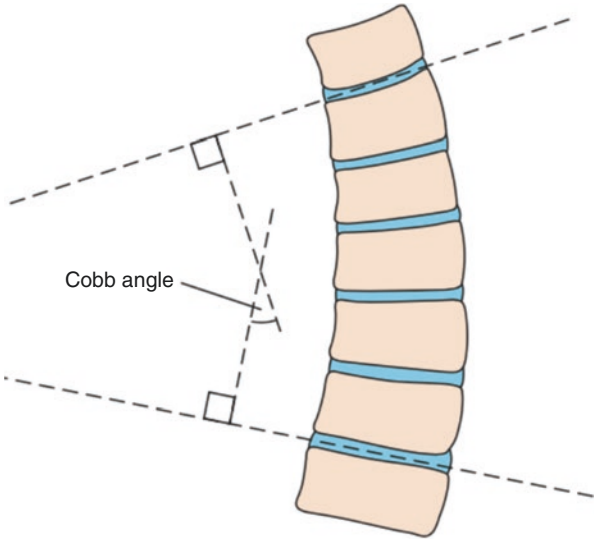


Fig. 29.2 Calculation of Cobb angle. A line is drawn parallel to the upper end of the proximal vertebrae of the curvature and a line drawn parallel to the lower end of the distal vertebrae of the curvature. Two more lines are drawn perpendicular to each of these, so they intersect. The angle of this intersection is then measured in degrees. (From Simonds AK. Scoliosis and Kyphoscoliosis. Elsevier; 2012. p. 756–62)

Classification

Idiopathic Scoliosis

Classification of curvature can vary depending on the age of onset, underlying etiology, and the location of the curvature. A diagnosis of idiopathic scoliosis is made when a non-idiopathic cause has been ruled out. Non-idiopathic causes of scoliosis include neuromuscular disease, neurofibromatosis, mesenchymal disease, trauma, and tumors [8] (Table 29.1).

Table 29.1 Classification of scoliosis

Idiopathic
Infantile (0–3 years)
Juvenile (4–9 years)
Adolescent (>10 years)
Congenital
Abnormal vertebral development
Hemivertebrae
Abnormal spinal cord development
Myelodysplasia
Neuromuscular
Neuropathic - Upper or Lower Motor Neuron
Cerebral Palsy
Spinal Muscular Atrophy
Myopathic
Muscular dystrophies
Neurofibromatosis
Mesenchymal Disorders
Congenital
Marfan's
Arthrogryposis
Acquired
Rheumatoid arthritis
Trauma
Fracture
Radiation
Surgery (thoracotomy)
Neoplasm
Spinal cord tumor
Osteoid osteoma

Modified from Goldstein and Waugh [8]

Additionally, further definition of idiopathic scoliosis is made with the Lenke classification. This system, developed in 2001, is a standardized way to describe idiopathic curvature and guides research and treatment [9, 10]. Curves are numbered 1 through 6 based upon location of the major curvature. A major curve is defined as having a Cobb angle of ≥25 degrees [11]. Type 1 is a single, main thoracic curve. A type 2 is a double thoracic curve, proximal and main thoracic structural curves. Type 3 is a double major curve, main thoracic and thoracolumbar/lumbar structural curve. Type 4, a triple major, is proximal thoracic, main thoracic and thoracolumbar/lumbar structural curves. Type 5 is a thoracolumbar/lumbar structural curve. Type 6 thoracolumbar/lumbar structural curve and main thoracic, with the thoracolumbar/lumbar being the main structural curve.

The lumbar spine has modifiers (A,B,C) to further define the relation of a line from the center of the sacrum through the lumbar spine on a coronal radiograph, the central sacral vertebral line (Fig. 29.3) The sagittal thoracic spine modifiers are (−, N, or +) to describe lordosis, neutral, or kyphosis in the thoracic spine from T5-T12. A normal thoracic spine has some degree of kyphosis, hence the N, or normal kyphosis of 30°, with a range of 10° to 40°. A (−) sign refers to a lack of kyphosis, or hypokyphosis, and a kyphosis of less than 10°. A (+) sign refers to hyperkyphosis, or a curvature of greater than 40°.

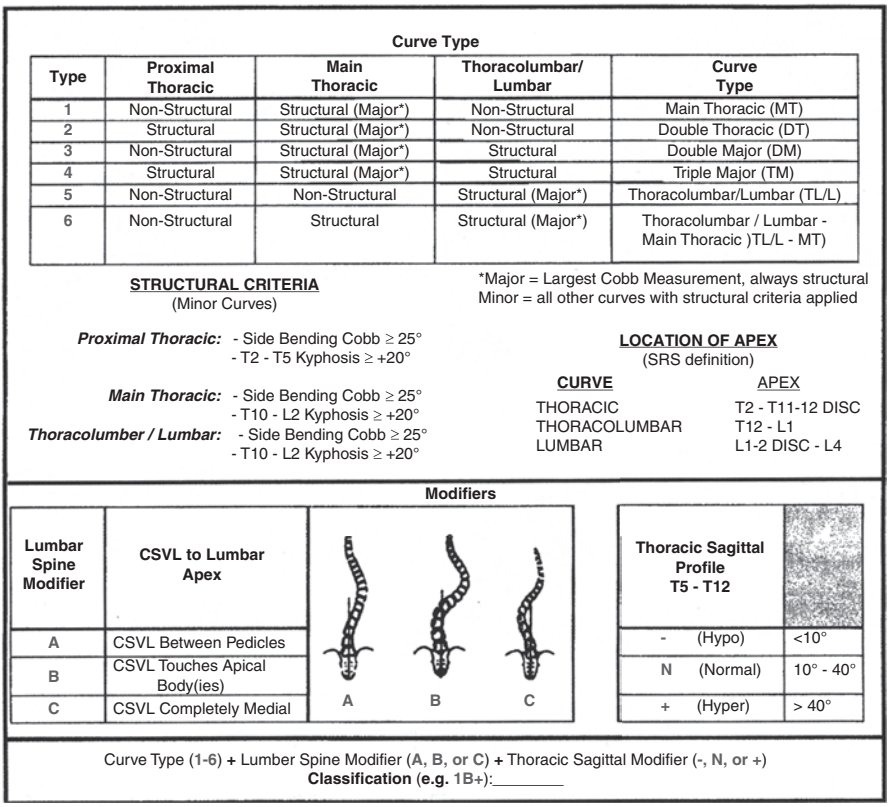


Fig. 29.3 Lenke Classification for Adolescent Idiopathic Scoliosis. SRS Scoliosis Research Society, CSVL central sacral vertebral line. A naming system commonly used to describe characteristics of idiopathic scoliosis curvature. For example, a Lenke 1AN describes a patient with a Major curve in the thoracic region, a CSVL between the pedicles of the lumbar apex, and a neutral curvature in the thoracic sagittal plane. (From Lenke et al. [9])

Idiopathic scoliosis is traditionally classified by the age at the time of diagnosis, infantile (ages 0–3), juvenile (ages 3–10), and adolescent (between age 10 and skeletal maturity) [12]. Infantile idiopathic scoliosis (IIS) accounts for less than 1 percent of all cases of idiopathic scoliosis [13]. It is typically seen in males, with a left convex curvature in the thoracic or thoracolumbar region. Development of curvature commonly occurs in the first six months of life [14]. Early identification and treatment of progressive IIS is essential to aid in lung development. Post-natal development of alveoli is greatest through the first two years of life and continues until age 8 [15]. Thus, early identification and treatment of IIS is imperative for quality of life. The differential for idiopathic scoliosis includes congenital scoliosis, neuromuscular scoliosis, and scoliosis due to intraspinal pathology [12]. Non-operative treatment for progressive curvature is serial body casting, which will be described later in this chapter.

Juvenile idiopathic is scoliosis that is diagnosed between ages 4–10. It represents 12–21% of all cases of idiopathic scoliosis [16]. Two-thirds of juvenile idiopathic scoliosis (JIS) curvature is a right thoracic convexity. Right convexity predominance is similar to adolescent idiopathic scoliosis (AIS) [17]. As with other idiopathic scoliosis diagnoses, any potential underlying causes of curvature should be investigated. Consideration for surgery in both infantile and juvenile cases depends on the age of the child at presentation and the amount of growth remaining in the spine [12]. Left untreated, IIS and JIS, but not AIS, can lead to increased mortality by the age of 40 due to death from respiratory failure [18].

Adolescent idiopathic scoliosis remains the most prevalent form with rates reported from 0.47–5.2% in the population. It is responsible for 90% of idiopathic cases, but fortunately formal treatment is only necessary in <10% of patients [1, 10]. Smaller curvature shows an equal male to female predominance, but as the magnitude increases, the ratio of female to male is 4 to 1 [19]. A decision to treat depends upon the magnitude of the major curve and risk for progression. Risk of progression depends upon location of the curvature, in particular thoracic, and signs of skeletal maturity. If a patient is skeletally immature, curvature less than 25 degrees can be observed, and bracing can be used for curvature 25–50 degrees. Patients who are skeletally mature with a curvature of 45 degrees are considered for surgery. Skeletally immature patients who have failed or cannot tolerate bracing and have progressive curvature of 40 degrees or more are also considered for surgical treatment [20].

However, if left untreated, pulmonary hypertension and right heart failure are rare in AIS [21]. The Iowa natural history studies followed patients retrospectively and then prospectively beginning in 1976. Upon follow up in the early 1990s, patients had an average age of 66, and had initially presented between 1932–1948. They were compared to age and sex-matched volunteers. There was no evidence to associate untreated AIS with increased mortality or cardiopulmonary issues related to the curvature. The untreated AIS group did have more back pain, but it was rated as “little, or moderate”. Patients with a curvature greater than 80 degrees also reported more shortness of breath with everyday activities and a higher degree of body dissatisfaction compared to the volunteer cohorts [22]. Additionally, a group of 45 patients with untreated idiopathic scoliosis were followed over a 20 year period. Patients with a vital capacity less than 45% and a curvature greater than 110 degrees at enrollment were more likely to develop respiratory failure [23].

A multicenter, prospective study from 1995–2003 looked at 631 AIS patients to determine the effect of curve magnitude on deterioration of pulmonary function. The authors noted that even patients with curvature less than 50 degrees could show moderate and severe pulmonary impairment (moderate described as forced expiratory volume in one second of 50 to 65% of predicted value and severe less than 50%) [24].

Early Onset Scoliosis

Early onset scoliosis (EOS) is a more recent all-encompassing term for scoliosis onset prior to age 10 years old, not necessarily the age at diagnosis [25]. In addition

to IIS and JIS, other diagnoses included in EOS include congenital, neuromuscular, and syndromic scoliosis. Congenital scoliosis is due to one or more vertebral defects during the sixth week of fetal development, with or without associated rib fusions [25, 26]. Associated cardiorenal abnormalities are common, as is association with VATER/VACTERL syndromes [26].

Treatment goals of EOS are to facilitate lung development and prevent the degradation of lung function to the point of thoracic insufficiency syndrome (TIS). Thoracic insufficiency syndrome is the inability of the thorax to support normal respiration or lung growth [27]. As the thoracic deformity progresses, respiratory effort becomes highly dependent upon the diaphragm due to lack of chest wall mobility. Naturally, as compensatory mechanisms fail, these patients can progress to respiratory failure and failure to thrive [27]. For patients with severe EOS that has progressed to the point of TIS, quality of life is worse than those compared to patients with epilepsy, cancer, or even heart disease [28].

When posterior spinal fusion is performed at a young age in complex multi-level EOS, the posterior spinal elements become fused and no longer grow. The anterior portion of the spine will continue to grow and create further deformity, known as crankshaft phenomenon [29]. More modern techniques are classified as growth friendly techniques, which allow for continued spine and thorax growth and control of curvature [30]. Understanding of the methods for spine correction with growth-friendly techniques is important to the anesthesiologist. These children will require frequent anesthetics over the course of their treatment span and at times, a definitive fusion surgery when skeletal maturity is reached. However, these techniques are not without complications: infection, rod breakage, anchor displacement, kyphosis, and unintended fusion occur [25].

Distraction-based implants are common implants used in the treatment of EOS. The general principle is that a distractive force is applied between proximal and distal "anchors" that are joined by an expandable rod. As the spine grows, the rods can be lengthened to lessen curve progression. With the traditional growing rod (TGR), limited fusion is performed at the distal and proximal anchor sites to allow continued spine growth. Lengthening is performed at approximately six- month intervals, thus necessitating repeat general anesthetics [26]. The vertical expandable prosthetic titanium rib (VEPTR) device is another distraction-based implant that uses the ribs as anchors to provide primarily thoracic expansion. This device also requires frequent anesthetics for lengthening procedures. A hybrid system uses the concepts of TGR and VEPTR combined. A distal anchor site is created in the lower spine, but the proximal anchor site is a rib. A rod connects the two sites and requires repeated lengthening. This perhaps makes the spine more flexible as there is no spinal fusion site in the proximal portion. The magnetically controlled growing rod (MCGR) is also similar to the TGR, but the rods can be lengthened externally. This is possible due to internal magnets in the rod construct. With external lengthening, generally no anesthetic is required [26]. Rarely, light sedation will be required in cases of behavioral issues (Fig. 29.4).

Additional surgical techniques that the anesthetist should be aware of include guided growth implants and compression-based implants. The Shilla technique is a



Fig. 29.4 Patient with surgically implanted magnetically controlled growing rods (MCGRs). Internal magnets are located inside the rod construct and which allows for generally pain free external lengthening with a handheld device at approximately six-month intervals. Repeated anesthetics are limited as opposed to the traditional growing rod. (From Scottish Rite for Children)

method of guided growth implants. Minimal dissection is used to place pedicle screws in hopes of avoiding boney fusion. The curvature is then corrected with rod placement and the spine is allowed to grow along the contour of the rods. These patients require less anesthetics than the distraction-based implants (TGR, VEPTR, MCGR). However, correction of scoliosis may not be as effective as MCGR [31]. A compression-based technique involves arrest of the growth on the convex side of the curvature. Via a trans-thoracic approach, staples or tethers are applied to the anterior portion of vertebrae on the convexity of the curve, which will arrest growth plate. This in turns allows the concave side of the curve to grow and help to correct the curvature. A potential concern for the anesthesiologist is the need for repeat trans-thoracic surgery [26].

Neuromuscular Scoliosis

Neuromuscular causes of scoliosis provide the greatest challenges in perioperative care for spine surgery. The diagnosis of neuromuscular scoliosis (NMS) is a broad term that includes many underlying conditions. So while the orthopedic presentation can be similar, the complexity of the medical conditions associated with it must be respected and optimized [32]. The classic neuromuscular deformity is a long thoracolumbar curve to the pelvis, which in turn induces pelvic imbalance [33]. Deformity of the spine can be induced due to trunk muscle weakness, imbalanced

spasticity, or dyskinesia [33, 34]. Deformities can become quite severe and make wheelchair positioning challenging and painful. The goal of surgical treatment is restoring upright posture and preventing curve progression.

Understanding the underlying etiology of the scoliosis and associated medical co-morbidities allows for proper planning to minimize the severity of complications [32]. A retrospective review of the Healthcare Cost and Utilization Project Kids Inpatient Database showed patients with NMS had longer hospitalizations, higher costs, more total procedures, more respiratory complications and higher rates of surgical site infections when compared to patients with idiopathic scoliosis [35].

Cerebral Palsy

Cerebral palsy (CP) is the most common neuromuscular diagnosis associated with scoliosis [32]. Anywhere from 21–64% of patients with cerebral palsy have scoliosis, and unlike idiopathic scoliosis, the deformity in neuromuscular scoliosis often progresses past the age of skeletal maturity [36–38].

Despite the high risk of peri-operative complications in cerebral palsy patients, a prospective questionnaire administered to caregivers of severe CP patients who underwent surgical repair of scoliosis indicated that at one year post-operatively, patient pain and fatigue, happiness and parental satisfaction improved significantly compared to pre-operative surveys [39]. A large systematic review also showed significant quality of life improvements in CP and muscular dystrophy patients after spinal fusion [40].

For patients with more flaccid forms of CP or other non-spastic NMS, with an apex of the curvature at L2 or higher and minimal pelvic obliquity, fusion may stop at L5 [41, 42]. However, the majority of NMS patients will have severe pelvic obliquity and require a fusion that extends to the sacral level. Extension of the fusion to pelvis is of concern to the anesthesiologist as it is associated with more blood loss perioperatively, increased infection rates, failed fusion, and limited patient mobility [32, 43].

Patients with CP often have co-existing seizure disorders. A careful review of medications and seizure history is necessary. Anti-epileptics can upregulate hepatic enzymes, making response to anesthetic and analgesic agents often unpredictable [44]. In addition, the anti-convulsant sodium valproate is known to cause bleeding concerns due to many mechanisms including impaired platelet function, decreased platelet count, and decreased clotting factor function [45].

Other mechanisms for increased bleeding in cerebral palsy patients included poor nutritional status leading to clotting factor insufficiency and also impaired primary hemostasis due to altered connective tissue structure in blood vessels. A prospective comparison between NMS and AIS patients compared normal clotting parameters and thromboelastogram (TEG) measurements at baseline and then after 15% loss of estimated blood volume. After a 15% loss of estimated blood volume, a transient hypercoagulable state would be expected, however, CP patients showed a decreased maximum amplitude on the TEG analysis, suggesting altered platelet function or impaired platelet-fibrinogen interaction. Prothromin time and partial thrombplastin time were also outside of normal range compared to normal values in

the AIS group [46]. Finally, CP and other NMS patients typically require more extensive fusions which require longer operative times and instrumentation of more vertebral levels, both of which will naturally increase blood loss [47].

In addition to a high prevalence of gastro-esophageal reflux disease, children with CP can also have swallowing incoordination and significant pooling of oral secretions, which puts them at a high risk of aspiration. Ideally, a swallow study will be performed prior to major spinal fusion to identify patients at highest risk of aspiration [48]. Evaluation of feeding problems and nutritional deficiency should be a high priority [49]. Patients with albumin levels >3.5 g/dL have lower rates of infection, shorter duration of post-operative intubation, and shorter hospitalization [50]. Optimization of nutritional status may require several weeks or months of nocturnal feeding via nasogastric tube or placement of a gastrostomy tube in anticipation of spinal fusion [33].

CP patients are also at increased risk for sleep disruption due to a variety of factors: upper airway obstruction, severe visual impairment leading to altered sleep-wake cycles, brainstem dysfunction leading to altered cardiopulmonary function, micro-aspiration, and somnolence secondary to epilepsy medications. The gold standard for evaluation is a polysomnogram (PSG). If there is a high suspicion for sleep disordered breathing and a PSG is not available, clinical judgement in combination with a lateral airway radiograph (to evaluated enlarged adenoidal tissue), a measure of serum bicarbonate, and sleep oximetry can be utilized. Continuous positive airway pressure can be beneficial in overcoming upper pharyngeal obstruction due to poor muscle tone. In patients with altered respiratory drive, more complex support with bi-level positive airway pressure devices (BiPAP) may be necessary that includes a backup rate [51].

Myelodysplasia

In patients with myelodysplasia, the incidence of scoliosis is over 70% if the deformity is at T12 or higher [52]. Prevalence of scoliosis in this population can be as high 89%, with deformity at L3 or higher increasing the risk of severe scoliosis. The type of deformity is dependent upon the level of the defect [43].

Complicating the condition is a high rate of renal anomalies, incontinence, latex allergy, insensate skin, urinary tract colonization, and lower extremity contractures [43]. Patients with myelodysplasia commonly have hydrocephalus requiring a ventricular shunt. Peri-operative complication rates are high, reported from 48–53% [53, 54]. Recent shunt evaluation by a neurosurgeon prior to surgery is critical to ensure patency and function. Shunt malfunction can occur post-operatively and can lead to acute hydrocephalus and death [53].

Because of a neurogenic bladder, myelomeningocele patients frequently have asymptomatic urinary tract infections which can be associated with post-operative wound infections [55]. Evaluation and treatment in the immediate pre-operative period can help to decrease the risk of peri-operative wound infection [56].

The risk of latex allergy is also a concern specific to myelomeningocele patients, with an estimated risk of an intraoperative anaphylactic reaction to latex that is 500 times greater than the general population [57]. Latex allergy refers to immediate

hypersensitivity symptoms caused by contact (urticaria, erythema, angioedema, etc.) with a positive IgE test. Latex sensitivity refers to patients with IgE antibody results specific to latex, but do not show symptoms on contact with latex [58]. Given that 18–40% of patients may have an allergy, latex testing is prudent prior to an operation [59].

Duchenne's Muscular Dystrophy

Duchenne's Muscular Dystrophy (DMD) is an x-linked recessive disorder caused by a frameshift mutation in the dystrophin gene, which results in breakdown of muscle cells with replacement by fibrofatty tissue [60, 61]. The progressive disease can result in eventual loss of ambulation, scoliosis, and cardiopulmonary deterioration [62]. Ninety percent of untreated males will develop scoliosis [63]. Fortunately, initiation of steroids, such as Deflazacort, can prevent the progression of scoliosis and substantially decrease the need for surgical treatment [64–66].

For patients presenting for surgery, particular attention to cardiopulmonary status is warranted. The loss of dystrophin affects cardiac myocytes, with cardiac functioning beginning to decline between ages 12–14 [62, 67, 68]. Use of beta-blockers and angiotensin converting enzyme inhibitors or angiotensin II receptor blockers is common in disease management [62]. Peri-operative dosing continuation should be in consultation with the patient's cardiologist.

DMD patients are not able to be reliably evaluated with the New York Heart Association (NYHA) classification of heart failure. Subtle signs and symptoms of heart failure include weight loss, vomiting, abdominal pain, sleep disturbance, decreased urinary output, and fatigue. Chest pain attributed to musculoskeletal etiology could mask cardiac concerns. Sinus tachycardia is also common, and can occur in the absence of ventricular dysfunction [69]. Potential etiologies include dysregulation of the autonomic nervous system [70].

Baseline evaluation such as electrocardiogram and echocardiogram should be reviewed. Ventricular ectopy is common in the older patient and can be evaluated by a Holter monitor [69]. It is important, however, to recognize that a resting echocardiogram will not provide information as to how the heart will respond to surgery and hemodynamic shifts. In addition, ultrasound acoustic windows are difficult to obtain in older patients with adiposity and scoliosis [71]. Consideration should be given to a dobutamine stress echocardiogram or a cardiac magnetic resonance imaging study (CMR) should more information be needed [69]. CMR can provide benefit over standard echocardiography when there is difficulty in image acquisition. It allows for more precise distinction between blood and the endocardium which allows for accurate chamber volume assessment. Subjective and objective wall motion characteristics are also better visualized with CMR [72].

Pre-operative workup should include pulmonary function testing. Forced vital capacity (FVC) less than 35% has been associated with an increased rate of pulmonary complications [73]. However, more recent studies have shown that patients with FVC <30% can safely undergo surgery with a similar post-operative outcome as patients with FVC >30%. The routine use of post-operative non-invasive ventilation to facilitate early extubation is critical [74].

Not to be under-appreciated is the potential for difficult intubation in DMD patients. In a retrospective review of 292 cases, 4% of DMD patients were labeled as difficult laryngoscopies. They were typically older children with obesity, large tongues, small mouth openings, and restricted cervical motion [75]. Progressive fibrosis of musculature accounts for difficulty in mouth opening and cervical motion [69].

Use of muscle relaxants should occur with caution. Due to upregulated fetal nicotinic acetylcholine receptors, life threatening hyperkalemia can result with depolarizing neuromuscular blocking agents such as succinylcholine [75–77]. Onset of action and duration of action for non-depolarizing agents can be prolonged in DMD patients [78, 79]. With newer reversal agents such as sugammadex, reversal of profound rocuronium induced neuromuscular block is possible [80].

Total intravenous anesthesia is preferred in DMD due to concern for anesthesia induced rhabdomyolysis as well as unexplained fever when patients are exposed to volatile anesthetics. There is no increased risk of malignant hyperthermia in this patient population. Intraoperative rhabdomyolysis and secondary hyperkalemia are thought to be due to the action of inhaled anesthetics on already vulnerable muscle membranes [75, 76].

The anesthesiologist should also be prepared for larger than expected blood loss. The lack of dystrophin in vascular smooth muscle leads to a poor vasoconstrictive response to bleeding [81]. In addition, platelet responsiveness is impaired in DMD [82].

Syndromic Scoliosis

Similar to neuromuscular scoliosis, syndromic scoliosis is an umbrella term used to describe scoliosis occurring as part of a systemic disease. Co-existing conditions include Marfan syndrome, Down syndrome, neurofibromatosis, Rett syndrome, achondroplasia, Ehlers- Danlos syndrome, Prader-Willi syndrome, Friedrich's ataxia, and Osteogenesis Imperfecta [83]. The more common syndromes with their anesthetic implications will be reviewed here.

Marfan Syndrome

Patients with Marfan syndrome, an autosomal dominant condition due to defect in genetic coding for fibrillin-1, have scoliosis in 60% of cases [84]. Although curvatures can be similar to idiopathic scoliosis, surgical correction typically requires a greater number of levels to be fused. Complications such as extensive blood loss related to abnormal fibrillin and cerebrospinal fluid leaks related to dural ectasia are more common [83, 84]. The anesthesiologist must be aware of any potential airway difficulties due to a high arched palate or impaired oral opening due to temporomandibular joint dysfunction [85, 86]. Additionally, respiratory concerns include an increased incidence of spontaneous pneumothorax, up to 4–15%. A heightened awareness should remain throughout the anesthetic and hospital stay for this potential complication [85, 87]. Cardiovascular concerns should also be addressed preoperatively. Progressive aortic root dilation and mitral valve prolapse are the most significant abnormalities in patients with Marfan syndrome. Aortic root dilation can

lead to aortic valve regurgitation as well as eventual aortic dissection [88]. Continued deterioration of the mitral valve prolapse can lead to mitral valve regurgitation. Extensive pre-operative cardiac workup is warranted in these patients as is strict hemodynamic control intraoperatively.

Additionally, due to risk of perioperative visual loss (POVL) in spine surgery, the patient's ocular history should be reviewed [89]. Ectopis lentis or lens subluxation, corneal abnormalities, glaucoma, and retinal detachments are possible due to abnormal fibrillin. Information on recent ophthalmic exams should be obtained pre-operatively [90].

Down Syndrome

A patient with Down syndrome, a trisomy of chromosome 21, provides unique anesthetic challenges due to systemic clinical manifestations [91]. Scoliosis can present in 7% of the patient population, with rates in institutionalized patients as high as 50% [92, 93]. Complications after scoliosis surgery are high with this patient population, most notably spinal hardware failure and infection [91, 94].

When a patient with trisomy 21 presents for scoliosis spine surgery, several systemic considerations should be accounted for. Cervical spine stability should be evaluated pre-operatively. Atlantoaxial and atlanto-occipital instability can occur concurrently in a patient with Down syndrome [95]. Screening for instability can be challenging in infants and toddlers due to lack of ossification of the spine. Flexion and extension cervical spine radiographs to evaluate the atlantodens interval (ADI) can be obtained. Normal radiographs do not preclude possibility of an injury occurring. A baseline assessment of strength and movement should be obtained pre-operatively and a repeat assessment should occur post-operatively. A decision for cervical spine radiographs should be made between the surgeon and the anesthesiologist [96].

A complete atrioventricular septal defect is the most common cardiac lesion, with ventricular septal defect and atrial septal defect being next most common [97]. A recent echocardiogram should be obtained to assess for undiagnosed or residual heart disease in addition to possible signs of pulmonary arterial hypertension [96]. Should inhalation induction be required, the anesthesiologist should be prepared for bradycardia in response to sevoflurane. This phenomenon is likely due to impaired autonomic cardiac regulation [98].

Upper airway obstruction is also common due to multiple possible sites of restriction: macroglossia, flattened nasal bridge, pharyngeal muscle hypotonia, tonsillar and adenoidal hypertrophy, congenital subglottic stenosis, and tracheomalacia. Because of upper airway anomalies, obstructive sleep apnea is common and previous diagnostic studies should be reviewed [99].

Additional Spinal Pathologies

Kyphosis

Kyphosis is from the Greek work 'kyphos', which means bowed forward, in the sagittal plane [43]. Kyphosis of 20–40° is considered normal and increases as a

person ages [100, 101]. Curvature is measured on lateral radiographs via the Cobb angle method as described previously [102]. Congenital kyphosis is due to abnormal vertebral segmentation of formation and can progress quickly to the point of spinal cord compression and paraplegia [102]. Prevention of progression with bracing is ineffective and surgical correction is often required before age 5 and before the curvature progresses to 50° [102].

Scheuermann Kyhposis is a structure deformity that presents clinically in late childhood, ages 8–12, with wedging of three adjacent thoracic vertebral bodies [103]. A more severe, fixed form, appears at ages 12–16 [104]. The incidence ranges from 1% to 8%. Kyphosis that is greater than 80° in the thoracic spine or 65° in the thoracolumbar spine typically requires surgical intervention [102]. Pain and neurologic deficits are possible if progressive curvature is left untreated. As curvature approaches 100°, restrictive lung disease can be seen [103].

Spondylolysis and Spondylolisthesis

Spondylolysis can also affect pediatric patients and can at times require surgical management. From the Greek roots, "spondylos" meaning vertebra and "lysis" meaning defect, it is pathology of the pars interarticularis. When the defect occurs bilaterally, there can be forward displacement of the upper vertebrae over the lower vertebrae, or spondylolisthesis (derived from the Greek word 'olisthesis,' meaning movement or slippage). Only rarely does spondylolysis require surgical fixation when all conservative measures have failed to treat lumbar pain [105].

Spondylolisthesis severity depends upon the amount of anterior slippage of the superior vertebrae when compared to the vertebrae below. As with spondylolysis, clinical manifestations can be highly variable. When symptoms do appear in young patients, it coincides during the time of rapid growth during puberty, ages 10–15. Severity can be defined by the amount of slippage of the superior vertebrae. Grade I is displacement of 0–25%, Grade II is displacement of 26–50%, and in grade III the displacement may be up to 75%. Displacement of 75–100% is classed as grade IV. Cauda equina syndrome can occur in cases of grade IV spondylolisthesis [105, 106].

Preoperative Evaluation for Spinal Deformity Surgery

For children with multi-organ system pathology who present for spinal deformity surgery, a thorough preoperative assessment should include evaluation by a multi-disciplinary team (MDT). This could be comprised of a scoliosis surgeon, general pediatrician, pulmonologist, anesthesiologist and cardiologist [107]. Early involvement of a complex care pediatrician is prudent, as this lessens the burden on the anesthesia care team for last-minute referrals and recommendations.

Respiratory Evaluation

Pre-operative impairment of respiratory function can be anticipated in most patients who have scoliosis of surgical magnitude. Severe scoliosis will show a reduced total lung capacity and forced vital capacity (FVC). A reduction in FVC will mirror severity of curvature [7]. When patients present with neuromuscular disease as well as an FVC <40%, they are more likely to require prolonged mechanical ventilation [108].

Maximum inspiratory pressure (MIP) is also decreased in scoliosis due to effects of chest wall deformity on the ability to generate appropriate inspiratory forces. The MIP is important, as a value of less than 30 cm H_20 can be predictive of need for post-operative ventilation [7].

However, for patients that were historically not candidates for early post-operative extubation, use of non-invasive ventilation and cough assist devices increases chances for success. Pre-operative training with non-invasive positive pressure ventilation (NPPV) and a mechanical insufflator–exsufflator (MI–E) device in a small series of non-idiopathic scoliosis patients undergoing corrective surgery showed great success in minimizing respiratory complications. 13 patients with neuromuscular scoliosis were intubated for a mean of 19.9 hours post-operatively, had an average intensive care unit stay of 2.5 days and had no respiratory complications. MI-E was provided by a Cough Assist™ device (JH Emerson Company, Cambridge, MA, USA) to help facilitate clearance of airway secretions [109, 110].

Cardiac Evaluation

Patients with scoliosis secondary to neuromuscular or syndromic conditions will benefit from evaluation by a cardiologist to include electrocardiogram and echocardiography. Not all otherwise healthy idiopathic scoliosis patients will undergo cardiac evaluation beyond a basic clinical assessment. In a retrospective review of 212 otherwise healthy patients with idiopathic scoliosis who underwent screening electrocardiogram and echocardiogram, 85 percent had normal findings. However, 4.2 percent of patients demonstrated significant abnormalities, including two with atrial septal defects that required repair prior to surgery [111]. The prevalence of echocardiogram abnormalities in a population of 357 randomly selected junior high school students, only 7 patients (1.9 percent) had significant abnormalities [112]. No formal recommendations exist for cardiac evaluation prior to pediatric spinal fusion surgery.

The anesthesiologist should also be keenly aware of right heart changes and pulmonary pressure increases that can be expected with severe curvature. With increased curvature and lung compression, the number of vascular units per unit volume of lung is less and therefore increases pulmonary vascular resistance. In addition, lung compression by the chest wall deformity limits alveoli expansion and

causes shunting of blood flow through the higher resistance extra-alveolar blood vessels [113]. Changes in right sided heart pressures can be seen when curvature approaches 100 degrees or higher [7].

Non-Surgical Treatment

Serial Casting

In young children who would not tolerate a thoracolumbar brace, serial application of a corrective plaster jacket can be performed under general anesthesia. The cast is worn for 8 to 16 weeks to allow the spine time to grow into an improved direction [114]. In some cases, such as idiopathic early onset scoliosis, a cure can be obtained. However, often times, the casting is to delay severe progression as a bridge to operative approaches when the child ages [26].

The technique, initially developed by Cotrel and Morel, has regained popularity after Mehta's 2005 report [115]. The casting procedure is on a table which allows access to the body but supports the head, arms, and legs [116]. To facilitate correction of the curve, traction is applied at the head and the hips. A soft bite block should be placed to prevent compression of the airway after the chin strap is applied. Endotracheal intubation is recommended, as during cast application, one can expect a rise in airway pressure as the cast material hardens. Following placement of the cast, a large chest window as well as a window over the concavity of the curvature is created to allow lung expansion [114]. Children typically go home the same day, however, observation can be required when there is respiratory difficulty post-procedure. Cast removal is sometimes necessary (Fig. 29.5).

Of growing concern, however, is recurrent exposure to anesthetics in young children could potentially lead to poor neurodevelopmental outcomes [117, 118]. Awake casting can be an option in older children but treatment outcomes depend on



Fig. 29.5 Patient undergoing placement of a corrective plaster jacket on a specialized frame. Traction is applied at the head and the hips to facilitate scoliosis curvature reduction. Casting material is molded around the patient's torso. (From Scottish Rite for Children)

patient selection, with idiopathic scoliosis patients achieving the best outcomes [119]. As more is understood about the risk of early and repetitive exposure to anesthesia, perhaps more aggressive awake or sedated options will be pursued.

Operative Approaches

Surgical techniques vary given the patient age, anticipated growth, and characteristics of the curvature. The earliest generation of spinal instrumentation was developed by Harrington in the 1960s. The technique involved placement of hooks on the posterior elements of the spine at the distal and proximal portion of the curvature which allowed for rod placement and correction of the spine in the coronal plane. Patients would remain in body casts post-operatively to allow time for osseous fusion [120, 121].

Posterior Fusion

Modern posterior spinal fusion relies upon transpedicular screw placement, which allows for a strong point of fixation for de-rotation, compression, or distraction of the spinal deformity around a stiff metal rod (Fig. 29.6) [120]. The anesthesiologist should be prepared for potential complications related to screw misplacement. Potential issues include direct spinal cord injury, dural tear, epidural hematoma, intrathoracic screw placement with pneumothorax or hemothorax, and potential aortic abutment [122].

Anterior Procedures

Surgical treatment of spinal deformity can require a combined anterior and posterior procedure [123]. The anterior spine release is utilized to facilitate greater spine

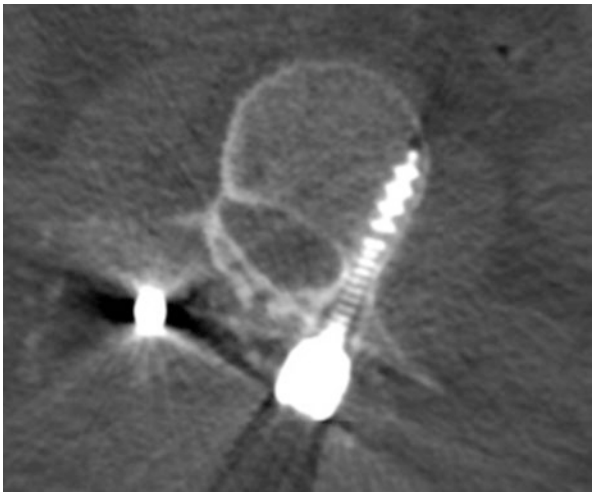


Fig. 29.6 Pedicle screw placement demonstrating the proximity of the screw to spinal canal. Pedicle screws provide strong fixation points for anchoring of metal rods. (From Scottish Rite for Children)

flexibility by removing intervertebral discs at the apex of the curvature [124]. This is then followed by a posterior instrumentation and fusion. The anterior spine can be approached with an open posterolateral thoracotomy or via a thoracoscopic technique [123, 124]. A large retrospective review showed no difference in pulmonary function recovery between patients undergoing an open or thoracoscopic approach [125]. In either procedure, one lung ventilation may be necessary depending on surgeon preference, anticipated technical difficulty and patient habitus. Generally, patients can tolerate a same day combined anterior and posterior procedure [126].

Anesthetic Intraoperative Management

Intraoperative care for spinal deformity is complex and requires careful consideration of medical comorbidities in the face of prolonged surgery with large fluid shifts. Effort should be made to minimize necessity of allogenic blood transfusion, maintain stable anesthetic conditions for proper intraoperative neuromonitoring and provide proper analgesia for a highly painful surgery (Table 29.2).

Monitoring

Patient monitoring should be individualized given patient co-morbidities. At the minimum, ASA standard monitoring should be utilized. The majority of scoliosis surgery will require beat to beat blood pressure monitoring with a peripheral arterial line. Central venous access should be considered on a case-by-case basis. One must be aware that central venous pressures could be misleading. In a study of 12 pediatric patients undergoing scoliosis surgery, central venous pressure (CVP) data was correlated with transesophageal echocardiography. When patients were placed prone, CVP increased from 8.7 mmHg to 17.7 mmHg with decreased left ventricular end diastolic diameter from 37.1 mm to 33.2 mm. Fractional shortening, left ventricular end systolic diameter and heart rate did not change when going from supine to prone. This suggests that CVP does not accurately reflect preload [127].

Positioning

When positioned prone for posterior spinal fusion, it is necessary to avoid pressure on several key areas including the anterior superior iliac spine to protect the lateral femoral cutaneous nerve, the knees, male genitalia, and the nipples. Care must be given to avoid abduction of arms more than 90 degrees to protect the ulnar nerve from injury. Patient positioning is wrought with challenges, particularly when patients have pronounced contractures [3, 128]. At times it is not possible to straighten the arms significantly, so positioning with the aid of disposable polyurethane foam positioners can accommodate contractures.

Table 29.2 Sample anesthetic plan for an AIS patient undergoing PSF

Premedication	Diazepam 0.1 mg/kg PO 2 hours pre-procedure Midazolam 2 mg IV immediately pre-procedure Aprepitant 40 mg PO if personal history of post-operative nausea or vomiting (PONV)
Monitoring and Lines	Standard ASA monitors, arterial line, 2 peripheral intravenous catheters SSEP, MEP, Single channel electroencephalogram
Positioning	Arms less than 90 degrees of abduction Prone pillow to protect eyes and face Abdomen freely hanging Well-padded extremities
Induction Agents	Propofol 2–3 mg/kg Sufentanil 0.1 mcg/kg Lidocaine 1 mg/kg Ketamine 0.5 mg/kg Rocuronium 0.3 mg/kg
Maintenance	Propofol 250 mcg/kg/min titrated to 80–100 mcg/kg/min by wound closure Sufentanil 0.3 mcg/kg/hr titrated to 0.1 mcg/kg/min Dexmedetomidine 0.5 mcg/kg/hr titrated to 0.1 mcg/kg/hr Ketamine 0.25 mg/kg bolus every hour
Emergence	Cessation of IV Infusions following last neuro-monitoring exam Nitrous Oxide or low dose volatile agent after last neuro-monitoring exam and until extubation
Intraoperative Multi-Modal Pain Control	Acetaminophen 15 mg/kg IV up to 1000 mg Ketorolac 0.5 mg/kg IV up to 30 mg Local infiltration analgesia into muscle, fascia and subcutaneous tissue and skin with Liposomal Bupivacaine, Bupivacaine HCl and Normal Saline mixture prior to wound closure
PONV prophylaxis	Dexamethasone 0.1 mg/kg up to 4 mg at induction Ondansetron 0.1 mg/kg up to 4 mg at skin closure
Blood Management	Tranexamic acid 30 mg/kg up to 2 grams then 10 mg/kg/hr until skin closure Maintenance of normothermia with preoperative forced air warming, intraoperative forced air warming, and fluid warming Intraoperative cell salvage Controlled normotension during spine exposure with Nicardipine 0.1–0.5 mcg/kg/min IV. Discontinued during screw placement and in advance of corrective spine maneuvers (i.e. rod placement)
Disposition	Extubation in operating room, recovery in post anesthesia care unit and transfer to an Intermediate Care Unit or Intensive Care Unit Neurological checks every 2 hours Multi-modal pain control with Oral or Intravenous opioid, Dexmedetomidine, Ibuprofen, Acetaminophen, Diazepam

The abdomen should be hanging freely to avoid increases in intra-abdominal pressure, which has several consequences. It can impair chest compliance, engorge epidural veins and therefore increase bleeding, in addition to impairing venous return due to inferior vena cava compression. Use of a Jackson frame (Mizuho OSI; Hollywood, CA) table allows for the abdomen to hang free and will avoid many of these consequences [128].

Attention must be given to the eyes and face. Use of a specialized prone pillow allows for the eyes to remain free of pressure and the weight of the head can be distributed on the face and the forehead. Risk of perioperative visual loss (POVL) is a relevant concern, even in children. Rates of POVL range from 0.03–0.2% in adults undergoing prone spine surgery [129]. In children, rates have been reported at 0.16% in a large retrospective national database review [130]. Ischemic optic neuropathy (ION) is the most common cause of visual loss following spinal surgery due to decreased perfusion of the optic nerve. Other causes include external ocular injury, cortical blindness, and central retinal artery occlusion [130]. Central retinal artery occlusion can occur from direct pressure to the eye and is usually a unilateral occurrence [131]. Cortical blindness is possible due to watershed blood supply to the visual cortex and susceptibility to hypoperfusion insults [130].

In a large case control study from 17 institutions, risk factors identified for development of ION in adults undergoing spine surgery in the prone position included obesity, male sex, Wilson frame use, longer anesthetic duration, greater estimated blood loss, and decreased colloid administration relative to crystalloid [132]. In children, however, younger age, male gender, pre-existing iron deficiency anemia, having Medicaid insurance, and fusion of 8 or more spinal levels were identified as risk factors for POVL in patients who underwent elective idiopathic scoliosis repair [130]. A most recent 2019 practice advisory update from the American Society of Anesthesiologists Task Force on Perioperative Visual Loss has several recommendations to mitigate risk (Table 29.3).

In the event of an emergency, the prone position is not ideal to provide resuscitation. Chest compressions have been described in several case reports with patients in the prone position [133, 134]. A hard surface is required under the patient's chest to provide sternal counter pressure to the thoracic compression [134]. During surgery, the patient's stretcher should remain close to the operating room in the event that patient needs to be turned from prone to supine. Turning for prone to supine is

Table 29.3 Summary of recommendations from Practice Advisory for Perioperative Visual Loss Associated with Spine Surgery

Blood Pressure Management	Use deliberate hypotension in high-risk patients only when the anesthesiologist and surgeon agree that its use is essential Treat prolonged significant decreases in blood pressure
Management of Blood Loss and Administration of Fluid	Use of transfusion of blood as deemed appropriate Crystalloids or colloids alone or in combination
Use of Vasopressors	Adrenergic agents when necessary to correct hypotension on a case-by-case basis
Patient and Head Positioning Devices	Avoid direct pressure on the eye to prevent retinal artery occlusion Check the position of the eyes periodically during surgery Position the high-risk patient so that the head is level with or higher than the rest of the body when possible
Staging of Surgical Procedures	On a case-by-case basis for high-risk patients

Modified from Practice Advisory for Perioperative Visual Loss Associated with Spine Surgery 2019. Anesthesiology. 2019;130(1):12–30.

a complex effort and rehearsed simulation with the peri-operative team can prepare for this event.

Blood Loss

Given the large incision and prolonged exposure of raw bone surfaces, blood loss in scoliosis surgery can be substantial. As suggested throughout the chapter, numerous factors contribute to the degree of blood loss. From pooled studies, mean estimated blood loss for idiopathic scoliosis on the posterior spine is 600 to 1000 mL, with 65–100 mL per level fused. For anterior surgery, blood loss averages from 350–650 mL due to a smaller number of levels fused. In neuromuscular conditions, such as cerebral palsy, amounts range from 1300–2200 mL, and an amount of 100–190 mL per level fused. Values for patients with DMD are similarly elevated, with amounts as high as 3000–4000 mL reported [135].

Strategies to Limit Blood Loss and Allogenic Blood Transfusion

Induced Hypotension

Pharmacologically induced hypotension can be achieved with numerous agents to lower mean arterial pressure (MAP) and subsequently decrease blood loss. No one agent has been proven superior to another. An international consensus of pediatric spine surgeons agreed that a target MAP of 60–70 mmHg was ideal for optimal care during spine exposure [136]. This is much higher than historical values, in which systolic blood pressures of 80 mm Hg were recommended for a hypotensive anesthetic technique [137].

However, there remains great debate as to what a normal pediatric blood pressure is under anesthesia, with some suggesting a MAP < 60 as abnormal in idiopathic scoliosis patients [138]. There is value in the use of hypotensive anesthesia as it can reduce transfusion requirements by 55% and improve visualization of the surgical field [139]. It should be noted that most likely patients with pre-existing end organ damage or patients undergoing prolonged surgery are not good candidates for hypotensive anesthesia. With regards to spinal cord perfusion, when significant neuromonitoring changes suggestive of ischemia occurred in a large series of patients, MAPs were 55 mmHg. In addition to spinal cord perfusion concerns, the anesthesiologist should be aware of potential renal, cerebral, ophthalmologic and cardiac complications due to excessive and prolonged induced hypotension.

Primary agents used for induced hypotension are calcium channel blockers and beta blockers [140]. Clevidipine (Cleviprex, The Medicines Company, Parsippany, NJ) is a newer short-acting calcium channel blocker with a half-life of 1 to 3 minutes due to metabolism by non-specific blood and tissue esterases. In a series of 30 patients with neuromuscular scoliosis undergoing a PSF, target mean arterial pressures were reached in an average of 8.9 minutes, and more than half reached the

target MAP of 55–65 mmHg within 5 minutes. Of equal importance, MAPs returned to 65 mmHg or higher within 10 minutes upon discontinuation of the infusion [141].

Antifibrinolytic Agents

Another essential component of patient blood management is the use of antifibrinolytic agents. Tranexamic acid (TXA) and epsilon-aminocaproic acid (EACA) are synthetic lysine analogs that competitively inhibit activation of plasminogen to plasmin. This in turn reduces plasmin breakdown of fibrin [142]. A large Cochrane review in 2016 included 9 studies and 455 participants undergoing pediatric scoliosis surgery showed that antifibrinolytics decreased blood loss by 427 mL, which is a 20% reduction compared to placebo. The volume of blood transfused was also decreased by 327 mL. The only adverse event reported was 3 cases of deep vein thrombosis (DVT) in a placebo group [143].

Dosing of antifibrinolytics in scoliosis surgery has not been fully elucidated. A retrospective review of 116 patients who underwent PSF for idiopathic scoliosis showed difference in blood loss between a high dose technique (50 mg/kg loading dose followed by 5 mg/kg/h) and a low dose technique (10 mg/kg loading dose followed by 1 mg/kg/h). Higher dose TXA was associated with decreased intraoperative and whole hospitalization transfusion requirements [144]. Dosing for EACA is also variable as well. A retrospective review compared EACA bolus dose of 100 mg/kg followed by infusion rates of 33 mg/kg/hr compared to 10 mg/kg/hr. The 33 mg/kg/hr group actually showed higher blood loss compared to the lower dose group (25.3 ml/kg vs. 17.4 ml/kg) [145]. Potential explanation for this unanticipated difference is that EACA can interfere with platelet-vessel wall interaction [146].

Some potential safety concerns with antifibrinolytics include seizures, thromboembolism and renal dysfunction [147]. A large national database review of pediatric cardiac surgery patients showed that patients who received TXA compared to a cohort that did not, seizures were significantly higher (1.6% compared to 0.2%). There were no other differences in length of hospital stay or mortality [148].

Intraoperative Cell Salvage

Intraoperative cell salvage systems collect blood shed from the surgical field and filter out debris such as platelets, white blood cells, clotting factors, irrigation solution and metal debris [149, 150]. Concerns do exist with intraoperative autologous transfusion (IAT) including altered hemostasis and coagulation due to residual heparin [151]. However, even in children, heparin levels in transfused salvaged blood are zero to insignificant [149]. When IAT was instituted at a high volume center performing PSF for idiopathic scoliosis, intraoperative allogenic transfusion rates decreased from 55% to 6% and salvaged blood was used in 85% of cases [149].

Preoperative Autologous Blood Donation

Preoperative autologous blood donation (PABD) can increase the likelihood that a patient undergoing scoliosis surgery will not need an allogenic blood transfusion [152]. Potential benefits include reduced risk of viral transmission and antibody formation [153]. However, patients who participated in a PABD program had a

lower starting hematocrit (37.8 vs. 40.2) and were also transfused at a higher hematocrit (28 vs. 23.7) compared to patients who did not participate. Additionally, with a PABD program, there is higher cost of preparation, wasted units of autologous blood, and burden on the family for repeat visits to the hospital [154, 155].

In instances where a large amount of blood is to be collected, patients may benefit from preoperative erythropoietin (EPO) to stimulate blood production. Adding erythropoietin to PABD strategies results in higher hematocrit values preoperatively, more PABD units obtained, and subsequently lower allogenic transfusion rates [156].

Acute Normovolemic Hemodilution

Acute normovolemic hemodilution (ANH) can be an additional strategy to minimize allogenic blood transfusion by way of removing blood through a large bore cannula and replacing with crystalloid or colloid prior to the beginning of the operation. The blood is then reinfused at the end of the procedure [157]. Cardiac index and oxygen extraction will increase with this technique and systemic vascular resistance will decrease [158]. When ANH was used in a series of patients with AIS undergoing PSF, transfusion rates were 79% in a non-hemodilution group compared to 37% in the hemodilution group. A reduction of hematocrit to 30% was used with the ANH technique [159]. More substantial levels of hemodilution have been obtained in a series of patients with AIS undergoing PSF. Hemoglobin decreased from 10.0 to an average of 3.0 g/dL, while mixed venous oxygen saturations decreased from 90.8% to 72.3%. No patients had adverse outcomes from the technique, albeit patients were otherwise young and healthy [158].

Normothermia

In an effort to reduce blood loss, maintaining normothermia should be a high priority for the anesthesiologist and perioperative team. Intraoperative hypothermia refers to a core body temperature less than 36 degrees Celsius and can consequently affect platelet function and enzymatic function in the coagulation cascade [160, 161]. Patients undergoing spine surgery have significant portions of their body exposed to ambient air and large open wounds making them at high risk for hypothermia. Institution of a pre-operative forced air warming protocol has shown ability to increase the first measured patient temperature in the operating room by 0.5 °C and reduce the duration of the case spent hypothermic by 111.1 minutes [162]. Similar retrospective review demonstrated an association with less allogenic blood transfusion in patients who were pre-warmed [163]. Adult literature suggests that hypothermia is associated with transfusion of allogenic blood products in high blood loss cases such as total knee or total hip arthroplasty [164]. More conclusive studies are needed to demonstrate a causal relationship between hypothermia and allogenic blood transfusion in pediatric spinal deformity surgery.

Additional concerns about infection risk occur with hypothermia. Natural killer cell function as well as cell-mediated antibody production are diminished during conditions of mild hypothermia [165]. A 1 °C decrease in core temperature during surgery can diminish lymphocyte function for 48 hours after surgery [166]. In a case

control study of 326 VEPTR surgeries, a temperature of 35.0 °C or less was identified as a risk factor for surgical site infection (SSI) [167]. The World Health Organization recommends the use of warming devices with the purpose of reducing SSI. This is a conditional recommendation with moderate quality of evidence [168].

Transfusion Threshold

Restrictive transfusion thresholds can be utilized as an additional approach to limit allogenic blood transfusion. The AABB (formerly known as the American Association of Blood Banks) recommends a transfusion threshold of a hemoglobin of 8 g/dL for patients undergoing orthopedic surgery. This is a strong recommendation with moderate quality evidence and is applicable to adults and children [169]. When considering patients with idiopathic scoliosis and a cobb angle of 40 to 90 degrees, international consensus is that a transfusion threshold of 7.0 g/dL intraoperatively is appropriate [136]. Organ specific anemia tolerance varies based on oxygen demands. As demonstrated in animal models, different organs tolerate different degrees of anemia. Tissue hypoxia in anesthetized pigs developed more quickly in the kidneys and skeletal muscle as opposed to the brain and heart [170]. Even in high risk adult populations, large randomized controlled trials show non-inferiority and reduction in blood transfusion with a hemoglobin transfusion threshold of 7.0 g/dL [171].

Iron Therapy and Anemia

In a large study of AIS patients requiring allogenic blood transfusion, preoperative anemia was identified as a significant risk factor. Of 210 patients,16 (8%) had a preoperative hemoglobin level of 12 g/dL or less, which increased odds of transfusion by 9 times [172]. Importantly, significant association between pre-operative anemia and in-patient mortality has also been observed on review of the American College of Surgeons National Surgical Quality Improvement Program (ACS NSQIP) database from 2012–2014 [173]. Preoperative screening and treatment of mild anemia in adult patients undergoing hip and knee arthroplasty has demonstrated reduced transfusion rates, length of stay, and critical care admission [174]. Iron deficiency anemia affects children with a prevalence of 0.9% to 4.4% [173]. Given success in adult populations in screening and treating for iron deficiency anemia, it remains to be seen if similar programs can have success in children undergoing posterior spinal fusion [175].

Spinal Cord Monitoring

Spinal cord injury during scoliosis surgery ranges from small sensory disturbances to paraplegia [176]. Reports from 2004–2016 looking at 84,320 patients undergoing surgery for AIS, the rate of new neurological deficit was 0.35%, or 235 patients. The deficit was detected intraoperatively in 134 patients, immediately postoperative in 103, and delayed in 54 patients. Recovery was reported in 203 patients, but unfortunately 15 did not recover [177]. Imperative to recovery from injury is recognition of the insult as soon as possible [176].

Intraoperatively, neurologic insult can be from direct nerve trauma from an instrument or implant, spinal cord ischemia related to hypotension, and stretching of the spinal cord during corrective maneuvers. The use of somatosensory evoked potentials (SSEPs) and transcranial evoked motor potentials (TcMEPs) provides timely information at the time of a neurologic insult [178]. Historically, prior to use of intraoperative neurophysiologic monitoring (IONM), the Stagnara wake-up test was the only method to detect an intraoperative neurologic insult [179]. As the name implies, patients are awoken from their anesthetic and asked to move their hands to evaluate consciousness. Then, the patient is instructed to move their feet or wiggle their toes to evaluate motor function. The test is a high-risk endeavor, with risk of accidental intubation, unpleasant recall, increased surgical time, and importantly, a delay in the actual diagnosis of an abnormality [180]. The combination of SSEPs and TcMEPs has a sensitivity of 100% and specificity of 88% for detecting a true neurologic event and has largely supplanted the wake-up test [181].

However, monitoring can be challenging in certain populations. In a study of 39 patients with cerebral palsy undergoing corrective surgery, TcMEPs were monitorable in only 63% of patients with mild-moderate CP and 39% of those with severe disease. SSEPs were monitorable in 82% of all CP patients [182]. Similarly, patients with neural axis abnormalities such as syringomyelia, tethered cord, and diastematomyelia underwent successful SSEP monitoring (85.4%) and MEP monitoring (82.6%) in a series of 41 patients [183].

Somatosensory Evoked Potentials

SSEPs were the first opportunity for real time monitoring of neurologic function intraoperatively [184, 185]. The integrity of the dorsal column-medial lemniscus pathway is monitored and consists of a primary afferent signal in the periphery that travels up the dorsal column and is monitored with cortical and subcortical responses [180]. There is no monitoring of motor function with SSEPs, and despite normal SSEPs during surgery, patients can wake up with deficits [186].

Stimulating electrodes are placed over the posterior tibial nerves and occasionally, if responses are unobtainable, more proximally at the common peroneal nerve in the popliteal fossa. The median nerves are monitored in the upper extremities [185]. The nerves are stimulated at fixed intervals throughout the surgery and the amplitude (height) and latency (time of occurrence) are compared to a baseline recording [3, 185]. Amplitude is measured as the peak to peak voltage difference (microvolts) and latency is the time from stimulus to the peak (milliseconds). Concerning changes are an amplitude decreased by 50% and/or latency that is increased by 10% [186].

Selecting Anesthetic Agents

SSEPs are sensitive to volatile inhaled anesthetics, however, satisfactory monitoring can be obtained with 0.5–1 MAC [187]. The addition of nitrous oxide to an inhaled anesthetic can decrease amplitude of SSEPs by 75% [188]. Latency remains

Table 29.4 Anesthetic agent effects on somatosensory evoked potentials

Medication	Latency	Amplitude
Volatile agents	Increased	Decreased
Nitrous oxide	Minimal change	Decreased
Opioids	Minimal change	Minimal change
Dexmedetomidine	Minimal change	Minimal change
Midazolam	Minimal change	Decreased
Ketamine	Minimal change	Increased
Etomidate	Minimal change	Increased
Propofol	Minimal change	Minimal change

unchanged with nitrous oxide [187]. Midazolam also decreases amplitude with no changes to latency. Ketamine and etomidate both increase amplitude with little change in latency, which can be useful during times where monitoring changes occur [187, 189]. Opioids generally have no change on amplitude or latency. However, changes can occur with large bolus doses but less likely with continuous infusions [187, 190]. Propofol, when used in combination with an opioid such as sufentanil, produces a decrease in amplitude and increase in latency. However, 30 minutes after induction, these changes stabilize and allow for adequate SSEP monitoring during surgery [187, 191]. Dexmedetomidine produces minimal changes in SSEPs or MEPs [192]. See Table 29.4 for common anesthetic agents and their effects on SSEPs.

Motor Evoked Potentials

Descending motor pathways are stimulated via scalp electrodes and recording of signals is via epidural electrodes or in peripheral muscles via compound muscle action potentials (CMAPs) [185]. The importance of TcMEPs was highlighted in a review of 1121 pediatric patients who underwent corrective spine surgery and had monitoring with both TcMEPs and SSEPs. Overall, 38 patients (3.4%) had a neuromonitoring signal change. Of 7 patients with a confirmed motor deficit, SSEP failed to detect this change in 4 of the patients. Furthermore, when neuromonitoring changes occurred, SSEP changes lagged behind TcMEP by an average of 5 minutes [176].

Placement of epidural leads in the surgical field allows for recording of Direct (D) waves [180, 193]. D-waves are the result of direct stimulation of motor neurons via one transcranial electrical stimulus [194, 195]. They are not routinely monitored as they require epidural lead placement directly in the operative field or percutaneously into muscle [185]. They are, however, less sensitive to the effects of halogenated agents compared to CMAPs [193]. Additionally, with epidural recording of D-waves, neuromuscular blocking agents can be used because activity of peripheral muscles is not being monitored [196].

CMAPs are monitored via electrodes placed in peripheral muscles. Commonly used muscles are the abductor pollicis brevis, adductor hallucis brevis, and tibialis anterior [185]. The generation of a CMAP is dependent upon interaction of cortical

axon synapses and spinal cord anterior horn cells, both of which are sensitive to the effects of anesthetics [194, 197]. The stimulus can be delivered in a single impulse or multiple pulses, typically 5–7, as part of a train of pulses [198]. Multiple pulses can facilitate a process known as temporal summation. Upon stimulation, sodium channels remain open for 1–2 mS and then excitatory post-synaptic potential decays over the next 10–15 mS. A repeated stimulus delivered during this time will result in increased responses due to temporal summation [199].

All volatile anesthetics and nitrous oxide produce dose dependent decreases in MEP amplitudes [197]. Opioids have little effect on MEPs, as does Ketamine [200]. Use of total intravenous anesthetic (TIVA) limits the need for volatile agents or nitrous oxide. A combination of dexmedetomidine, propofol, and remifentanil infusion titrated to maintain a stable depth of anesthesia is one example of an anesthetic that is compatible with neuromonitoring [192]. Similarly, when ketamine is added to an anesthetic infusion of propofol and remifentanil, the voltage required to achieve maximum amplitude MEPs did not change [201]. Maintenance of a stable level of anesthetic and avoidance of bolus dosing will lead to more interpretable and reliable MEP recordings [197].

The term "anesthetic fade" is of potential importance during lengthy corrective surgery. Despite a consistent anesthetic regimen, it is common to see that the minimum voltage threshold required to produce an adequate MEP response increases as anesthetic duration is prolonged. This can lead to an increased incidence of "false positive" alerts. In a review of 703 adult spine surgery patients, the incidence of "false positive" alerts were 14%. An alert was noted if the TcMEP was ≤30% of the baseline. The authors noted an independent association between total propofol dose (>1550 mg), large variations in body temperature (1 °C), and greater blood loss (500 mL) [202]. One potential explanation for propofol's effect on TcMEP is that as blood loss continues during a long surgery, drug concentration will rise despite continuous infusion [203, 204].

Electromyography

Monitoring of spinal cord function with SSEP and MEP does not allow for identification of specific nerve root injury. Use of electrical and mechanical electromyography (EMG) allows the surgeon to identify excessive nerve root traction or mechanical injury. Mechanically elicited EMG, also known as spontaneous or free-run EMG, are passively continuous EMGs [180, 205]. A "burst" on EMG can simply be from mechanical contact with a spinal nerve root. A "train" of activity can imply traction on a nerve, mechanical irritation, or perhaps effect of cold irrigation solution [205]. Electrically elicited EMG also known as stimulus-evoked EMG or triggered EMG can be used to identify a cortical breach with a pedicle screw [206]. If the pedicle screw has breached the boney cortex, electrical stimulation will cause the nerve root at the given spinal level to depolarize at a much lower current than what is typical. This will in turn cause the corresponding muscle of that nerve root to contract, which can be recorded as a CMAP. As with TcMEPs, neuromuscular blocker usage should be limited when EMG is utilized [205].

Incidence, Cause, and Response to IONM Changes

In a retrospective review of 1121 consecutive patients with AIS undergoing corrective surgery, 38 (3.4%) of patients met criteria for a signal change. Nine of these patients experienced decreased TcMEP, but unchanged SSEPs, deemed to be the result of hypotension. The average mean arterial pressure (MAP) was 59 mm Hg, which was below the suggested MAP of 65 mm Hg in the study. The amplitude of the TcMEPs returned to baseline within 5 minutes of increasing the MAP to 90 mm Hg. The other 27 patients experienced IONM changes related to surgical technique, with the application of corrective forces being most common cause [176].

Additionally, in a series of 1155 patients undergoing pediatric spinal deformity correction, 8 transient false positive events were related to low mean arterial pressure (<60 mm Hg). They were considered as false positive due to improvement following augmented blood pressure. A true positive event (11 total) was more likely related to surgical instrument placement, spinal osteotomies, or corrective maneuvers [207].

Response to Intraoperative Neuromonitoring Changes

Use of surgical checklists can result in decreased complication and mortality rates [208]. A checklist, such as that published in 2014 by a consensus group of expert spine surgeons, identifies several key areas to address when faced with a crisis such as significant IONM changes [209]. The anesthesiologist has a direct and critical role in response to IONM changes (Fig. 29.7). Several factors need to be optimized by the anesthesiologist including choice of anesthetic agent and maintenance of normal physiologic variables (temperature, oxygenation and ventilation, blood pressure, and hemoglobin). Although transfusion recommendations are typically at a hemoglobin of 8 g/dL or less, this value should be reconsidered in the face of persistent neuromonitoring changes that do not respond to simple corrective measures [210]. Based on study of responses to neuromonitoring events, a MAP of 80 mmHg or higher is an appropriate target in the face of an IONM change [176, 207, 211–213].

Should neuromonitoring signals not improve, the anesthesiologist should be prepared for a potential wake up test and for the administration of high dose corticosteroids [209]. Although its use remains controversial, methylprednisolone 30 mg/kg followed by 5.4 mg/kg/hr for 24 hours is an option in the face of persistent neurologic deficits [214–216]. IONM changes can present stressful times in the operating room and coordinated efforts of all team members is imperative to generate positive outcomes.

Multi-Modal Pain Management

Corrective surgery for spinal deformity is a highly painful procedure for children and adolescents and use of multi-modal analgesia is critical for recovery and for the prevention of chronic post-surgical pain. Pain is a high concern collectively

Checklist for the Response to Intraoperative Neuromonitoring Changes in Patients with a Stable Spine

GAIN CONTROL OF ROOM	ANESTHETIC/SYSTEMIC	TECHNICAL/ NEUROPHYSIOLOGIC	SURGICAL
☐ Intraoperative pause: stop case and announce to the room	☐Optimize mean arterial pressure (MAP)	☐ Discuss status of anesthetic agents	☐ Discuss events and actions just prior to signal loss and consider reversing actions:
☐ Eliminate extraneous stimuli (e.g., music, conversations, etc.)	☐Optimize hematocrit	☐ Check extent of neuromuscular blockade and degree of paralysis	☐ Remove traction (if applicable)
☐ Summon ATTENDING anesthesiologist, SENIOR neurologist or neurophysiologist, and EXPERIENCED nurse	☐Optimize blood pH and pCO_2	☐ Check electrodes and connections	☐ Decrease/remove distraction or other corrective forces
☐ Anticipate need for intraoperative and/or perioperative imaging if not readily available	☐Seek normothermia	☐ Determine pattern and timing of signal changes	☐ Remove rods
	☐Discuss POTENTIAL need for wake-up test with ATTENDING anesthesiologist	☐ Check neck and limb positioning; check limb position on table especially if unilateral loss	☐ Remove screws and probe for breach
			☐ Evaluate for spinal cord compression, examine osteotomy and laminotomy sites
			☐ Intraoperative and/or perioperative imaging (e.g., O-arm, fluoroscopy, x-ray) to evaluate implant placement

ONGOING CONSIDERATIONS

- ☐ REVISIT anesthetic/systemic considerations and confirm that they are optimized
- ☐ Wake-up test
- ☐ Consultation with a colleague
- ☐ Continue surgical procedure versus staging procedure
- ☐ IV steroid protocol: Methylprednisolone 30 mg/kg in first hour, then 5.4 mg/kg/h for next 23 hrs

Fig. 29.7 Multi-disciplinary response to IONM changes as suggested by a consensus group of expert spine surgeons. (From Vitale et al. [209])

for patients and parents of children undergoing surgery for AIS [217]. By using multiple agents that work via different pharmacologic mechanisms, multi-modal analgesia allows for combination of drugs to be used at lower doses in order to limit untoward side effects of a single drug and maximize analgesic properties [218–220].

Pre-emptive analgesia refers to the concept of administering medication prior to surgery in order to diminish the establishment of pain hypersensitivity intraoperatively [221]. In AIS patients, highest pain scores are expected 12 hours after surgery and typically subside to more tolerable levels by post-operative day 4 [222]. Several methods to control post-operative pain exist, which begins in the pre-operative period.

Gabapentin, when begun on the day of surgery, has shown efficacy in reduction postoperative pain via reduction of afferent neuronal hyperexcitability [223]. Although use is off label for pediatric pain control, several dosing strategies have been developed [224]. A single dose of 600 mg in AIS patients did not show benefit in reducing pain or opioid consumption in a double-blind randomized control trial [225]. When dosed for several days in a double-blind randomized control trial (15 mg/kg on day of surgery followed by 5 mg/kg three times a day for five days), the gabapentin group showed less opioid use on through post-operative day 2 and lower pain scores in the recovery room and morning after surgery. No differences in

morphine related side effects (oxygen consumption, foley catheter use, bowel movement) were noted between the placebo and treatment group [226].

Diazepam, a long-acting benzodiazepine, can be administered prior to surgery to facilitate anxiolysis as well as muscle relaxation. When administered at doses of 0.3 mg/kg orally, similar sedation scores can be seen as with oral midazolam at doses of 0.5 mg/kg [227]. In AIS, a reasonable dose to administer is 0.1 mg/kg orally up to a maximum of 10 mg two hours prior to the procedure. This can be followed by 2 mg of intravenous midazolam immediately pre-procedure. Diazepam can be continued in the post-operative period during the first few days of recovery [228].

Systemic opioids have historically been the "gold" standard for pain control post-operatively [220]. Use of patient controlled analgesia (PCA) with morphine is a common practice in pain control for adolescents undergoing spinal fusion [225, 229, 230]. In addition, children as young as 6 can safely receive a PCA [231–233]. Severely delayed or medically complex spinal fusion patients may not be appropriate candidates for a PCA device. Intraoperatively, intrathecal morphine can be administered prior to incision to facilitate analgesia for several hours postoperatively and delay the need for systemic opioids [234–236]. Caution is advised with high doses (9–19 μg/kg) as this can increase likelihood of respiratory depression and pediatric intensive care unit (PICU) admission [237]. A lower dose range, 5–10 μg/kg, provides effective analgesia and less likelihood of a PICU admission. However, side effects such as nausea, vomiting, and pruritus are to be expected [236].

Epidural catheters placed in the surgical field can be utilized for post-operative pain control. There is a large variation in drug mixtures, method of administration, and the position or number of catheters [220]. Pain control was compared in a prospective trial of PCA, single epidural catheter, or dual epidural catheter techniques. Patients who received dual epidural catheters had lower pain intensity and did not experience any episodes of respiratory depression or neurologic changes [238]. Patients with single epidural catheters compared to PCA also show improved pain control. However, a large retrospective study comparing 413 epidural patients to 200 PCA patients showed premature discontinuation of catheters in 54 patients due to poor analgesia (61.1%) or neurologic changes (14.8%). No patients had permanent deficits [239].

Clonidine and dexmedetomidine are α2-adrenergic receptor agonists utilized to augment pain control and anxiolysis in pediatric spine surgery [230, 240]. Analgesia occurs via agonism at the α2-adrenergic receptor at the dorsal horn of the spinal cord and anxiolysis occurs via decreased central sympathetic output at the locus ceruleus [241]. When transdermal clonidine was added to a post-operative care pathway consisting of gabapentin and morphine PCA, the number PCA attempts decreased significantly and time to ambulation was decreased. In a prospective trial of morphine infusion compared to dexmedetomidine infusion for patients undergoing PSF for AIS, patients who received a dexmedetomidine infusion for the first 24 hours post-operatively had similar pain scores but statically significant less-opioid requirements throughout their hospitalization. Additionally, 15.6% of patients in the morphine group developed an ileus post-operatively compared to 3.2% in the dexmedetomidine group [230].

Beyond use as a component of balanced anesthesia, low dose perioperative ketamine can be utilized to decrease post-operative opioid requirements and improve analgesia [220]. A meta-analysis of 14 randomized controlled trials showed adjunctive ketamine in adult spine surgery patients lowers pain scores for the first 24 hours and decreased opioid requirements for the first 24 hours with no increased in adverse side effects from ketamine such as unpleasant dreams, hallucinations, or dysphoria [242]. However, when perioperative ketamine use in AIS was studied in a double-blind randomized placebo-controlled study, results did not demonstrate significant benefit in post-operative pain control. Patients received 0.5 mg/kg at induction of surgery followed by 2 μg/kg/min until 72 hours post-surgery. There was no difference in morphine usage between the two groups [243]. A similar trial evaluated morphine usage, but took into account patient weight to report in terms of milligrams per kg. Morphine usage (mg/kg), pain scores, nausea and vomiting were all decreased compared to placebo when ketamine was administered 0.5 mg/kg at induction followed by 0.2 mg/kg/h for 48 hours after surgery [244].

Acetaminophen, whether oral or intravenous, should be included in standardized care pathways. Data suggesting value of intravenous over oral acetaminophen is lacking in the scoliosis population. A randomized controlled trial with 30 mg/kg of intravenous acetaminophen at the conclusion of scoliosis surgery followed by 2 additional doses at 8-hour intervals showed a reduction in pain scores but no difference in total opioid administered. The authors used a total 24-hour dose of 90 mg/kg and drug levels were well below toxic thresholds. However, the sample size was small (n = 18) [245]. In adult joint arthroplasty patients, a single dose of intravenous acetaminophen as pre-emptive analgesia showed benefit in pain control in the first four hours after surgery. However, when intravenous was continued for an additional three doses post-operatively, it showed no benefit over the much less costly oral formulation [246].

In addition to acetaminophen, nonsteroidal anti-inflammatory drugs (NSAIDs) are key components of multi-modal pain control in pediatric spine deformity surgery [220]. In a small retrospective review of 60 patients, use of ketorolac did not increase complications such as transfusion, bleeding, or re-operation in patients undergoing scoliosis surgery [247]. Given the concern for poor bone healing related to prostaglandin inhibition, pseudoarthrosis remains a concern in patients receiving NSAIDs. A larger retrospective review of 158 patients receiving ketorolac post-operatively showed no differences in rates of failed boney fusion when compared to a cohort of 161 patients that did not receive ketorolac [248]. Short term exposure of less than 14 days to normal doses of NSAIDs likely does not affect bone healing in adult spine surgery patients [249]. Use of NSAIDs is surgeon and institution dependent.

Emerging methods to control pain include local infiltration anesthesia with a long acting local anesthetic, liposomal bupivacaine (Exparel, Pacira Pharmaceutical,Parsippany, NJ) (Fig. 29.8). When injected into the surgical wound, it can provide up to 72 hours of analgesia via slow release of bupivacaine from multi-vesicular liposomes [250]. Use has been well established in adult orthopedic procedures, including singe level spine decompression [251, 252]. Results

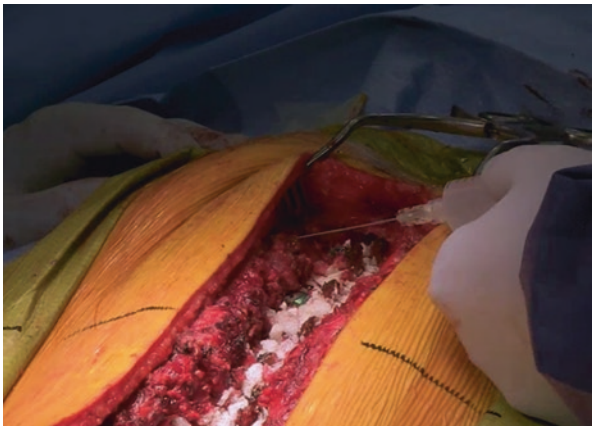


Fig. 29.8 Local infiltration into paraspinal musculature, fascia, and subcutaneous tissue during PSF from T5-L1 for AIS. The local anesthetic mixture consisted of liposomal bupivacaine (Exparel), bupivacaine HCl, and normal saline. The patient began oral opioids on the night of surgery and was discharged home on postoperative day 2. (From Scottish Rite for Children)

regarding pain scores, opioid usage, and time to discharge are mixed and more rigorous study is needed. Safety in pediatric spine surgery patients has been demonstrated without report of local anesthetic systemic toxicity [253, 254].

Postoperative Recovery

Multi-modal analgesia is a key component of rapid recovery pathways for scoliosis surgery. Such pathways are designed to facilitate patient recovery by reducing opioid consumption, encourage early oral intake and mobilization and ultimately an earlier discharge [228]. For example, when compared to traditional care at a high volume children's spine hospital, use of an accelerated discharge pathway resulted in a length of stay of 2.2 days compared to 4.2 days [229]. Key components of the accelerated pathway included resumption of a diet on the first post-operative day regardless of return of bowel function and early transition from PCA to oral opioids with use of multi-modal analgesia. Mobilization was begun on the morning postoperatively and continued two to three times a day. Absence of a bowel movement did not preclude discharge. Readmission rates and complications were similar between the two groups of AIS patients [229]. Highly important for success of the program was the education of patients and families to expect discharge on postoperative day 2 [229, 255–257].

Recovery in Intensive Care Unit Versus General Floor

In high volume spine centers taking care of otherwise healthy AIS patients, admission to the intensive care unit is generally unnecessary baring any intraoperative complications [258, 259]. In a retrospective study at a high volume institution, patients that recovered on the general floor had lower requirements for analgesic and antianxiety medicine, less blood draws, and shorter hospital stays [258]. Critical

to limiting adverse events in the study was nurse-to-patient ratios of 1:1 the first postoperative night, tenured nursing staff taking care of general floor patients, and comprehensive neurological assessment every two hours [258].

Delayed post-operative neurologic deficits (DPNDs) are a feared complication and highlight the importance of close hemodynamic and neurologic monitoring post-operatively. Estimated rates for DPNDs in pediatric spine deformity surgery are 1 in 9910 cases (0.01%), with 64% occurring in the first 24 hours post-operatively and 90% within 48 hours. Potential causes include delayed spinal cord ischemia due to spinal cord stretch or post-operative hypotension and anemia. Compression related issues from an epidural hematoma or instrumentation are other causes [260]. Continued vigilance in the immediate post-operative period is paramount to allow for corrective action to occur as soon as possible [259, 260].

Neuromuscular patients with co-morbidities that undergo lengthy and high blood loss procedures in the prone position may not be candidates for extubation in the immediate post-operative period and require ICU admission. However, at experienced centers, many patients can be managed in an intermediate level of care [261]. In a series of 197 patients with neuromuscular scoliosis undergoing PSF, only 15% were admitted to the ICU. The majority of these admissions were due to pulmonary complications or concerns. The majority of patients, 142, had severe cerebral palsy as the primary diagnosis. In institutions with adequate nursing and ancillary staff resources, ICU admission can be a case by case decision [261].

Summary

A key understanding of the patient's disease process contributing to spinal deformity is the foundation for a safe peri-operative course. Thorough pre-operative evaluation is essential as is preparation for care in the post-operative period. Hospital recovery can range from a two day stay on a general surgical floor to a prolonged ICU admission. Tailoring the anesthetic regimen to the patient's condition and proposed surgery is not unique to pediatric spine deformity but has heightened importance in the case of a complex operation. Meticulous intraoperative care leads to the possibility for early extubation and can limit use of intensive care units. Growing in popularity are standardized care pathways that provide uniformity and decreased variation amongst care teams in order to provide safe, cost-effective, and high-quality recovery from spine surgery.

References

1. Konieczny MR, Senyurt H, Krauspe R. Epidemiology of adolescent idiopathic scoliosis. J Child Orthop. 2013;7(1):3–9.
2. Asher MA, Burton DC. Adolescent idiopathic scoliosis: natural history and long term treatment effects. Scoliosis. 2006;1(1):2.
3. Davis PJ, Cladis FP, Zuckerberg AL, Yaster M. Smith's anesthesia for infants and children. Elsevier; 2017. p. 865–91.

4. Jones R, Kennedy J, Hasham F, Owen R, Taylor J. Mechanical inefficiency of the thoracic cage in scoliosis. Thorax. 1981;36(6):456–61.
5. Kotani T, Minami S, Takahashi K, Isobe K, Nakata Y, Takaso M, et al. An analysis of chest wall and diaphragm motions in patients with idiopathic scoliosis using dynamic breathing MRI. Spine. 2004;29(3):298–302.
6. JR C. Outline for the study of scoliosis. AAOS, Instructional Course Lecures. 1948;5:261–75.
7. Tsiligiannis T, Grivas T. Pulmonary function in children with idiopathic scoliosis. Scoliosis. 2012;7(1):7.
8. Goldstein L, Waugh T. Classification and terminology of scoliosis. Clin Orthop Relat Res (1976-2007). 1973;93:10–22.
9. Lenke LG, Betz RR, Harms J, Bridwell KH, Clements DH, Lowe TG, et al. Adolescent idiopathic scoliosis: a new classification to determine extent of spinal arthrodesis. JBJS. 2001;83(8):1169–81.
10. Slattery C, Verma K. Classifications in brief: The Lenke Classification for adolescent idiopathic scoliosis. Clin Orthop Relat Res. 2018;476(11):2271.
11. James JIP. Idiopathic scoliosis: the prognosis, diagnosis, and operative indications related to curve patterns and the age at onset. J Bone Joint Surg. 1954;36(1):36–49.
12. Dobbs MB, Weinstein SL. Infantile and juvenile scoliosis. Orthop Clin North Am. 1999;30(3):331–41.
13. James J, Lloyd-Roberts G, Pilcher M. Patterns in idiopathic structural scoliosis. J Bone Joint Surg Br. 1959;41:719.
14. Morgan T, Scott J. Treatment of infantile idiopathic scoliosis. J Bone Joint Surg. 1956;38(2):450–7.
15. Thurlbeck WM. Postnatal human lung growth. Thorax. 1982;37(8):564–71.
16. Ponseti IV, Friedman B. Prognosis in idiopathic scoliosis. J Bone Joint Surg Am. 1950;32(2):381–95.
17. Figueiredo U, James J. Juvenile idiopathic scoliosis. J Bone Joint Surg. 1981;63(1):61–6.
18. Pehrsson K, Larsson S, Oden A, Nachemson A. Long-term follow-up of patients with untreated scoliosis. A study of mortality, causes of death, and symptoms. Spine. 1992;17(9):1091–6.
19. Miller NH. Cause and natural history of adolescent idiopathic scoliosis. Orthop Clin. 1999;30(3):343–52.
20. Rose PS, Lenke LG. Classification of operative adolescent idiopathic scoliosis: treatment guidelines. Orthop Clin North Am. 2007;38(4):521–9.
21. Barton CB, Weinstein SL. Adolescent idiopathic scoliosis: natural history. Pathogenesis of Idiopathic Scoliosis. Springer; 2018. p. 27–50.
22. Weinstein SL, Dolan LA, Spratt KF, Peterson KK, Spoonamore MJ, Ponseti IV. Health and function of patients with untreated idiopathic scoliosis: a 50-year natural history study. JAMA. 2003;289(5):559–67.
23. Pehrsson K, Bake B, Larsson S, Nachemson A. Lung function in adult idiopathic scoliosis: a 20 year follow up. Thorax. 1991;46(7):474–8.
24. Newton PO, Faro FD, Gollogly S, Betz RR, Lenke LG, Lowe TG. Results of preoperative pulmonary function testing of adolescents with idiopathic scoliosis. J Bone Joint Surg. 2005;87(9):1937–46.
25. Karol LA. The natural history of early-onset scoliosis. J Pediatr Orthop. 2019;39(6, Supplement 1):S38-S43.
26. Yang S, Andras LM, Redding GJ, Skaggs DL. Early-onset scoliosis: a review of history, current treatment, and future directions. Pediatrics. 2016;137(1):e20150709.
27. Campbell RM Jr, Smith MD, Mayes TC, Mangos JA, Willey-Courand DB, Kose N, et al. The characteristics of thoracic insufficiency syndrome associated with fused ribs and congenital scoliosis. JBJS. 2003;85(3):399–408.
28. Vitale MG, Matsumoto H, Roye DP Jr, Gomez JA, Betz RR, Emans JB, et al. Health-related quality of life in children with thoracic insufficiency syndrome. J Pediatr Orthop. 2008;28(2):239–43.

29. Skaggs DL, Guillaume T, El-Hawary R, Emans J, Mendelow M, Smith J. Early onset scoliosis consensus statement, SRS Growing Spine Committee, 2015. Spine Deformity. 2015;3(2):107.
30. Skaggs D, Akbarnia B, Flynn J, Myung K, Sponseller P, Vitale M, Chest Wall and Spine Deformity Study Group; Growing Spine Study Group; Pediatric Orthopaedic Society of North America; Scoliosis Research Society Growing Spine Study Committee. A classification of growth friendly spine implants. J Pediatr Orthop. 2014;34(3):260–74.
31. Andras LM, Joiner ER, McCarthy RE, McCullough L, Luhmann SJ, Sponseller PD, et al. Growing rods versus Shilla growth guidance: better cobb angle correction and T1–S1 length increase but more surgeries. Spine Deformity. 2015;3(3):246–52.
32. Murphy RF, Mooney JF. Current concepts in neuromuscular scoliosis. Curr Rev Musculoskelet Med. 2019;12(2):220–7.
33. Vialle R, Thévenin-Lemoine C, Mary P. Neuromuscular scoliosis. Orthop Traumatol Surg Res. 2013;99(1):S124–S39.
34. Halawi MJ, Lark RK, Fitch RD. Neuromuscular scoliosis: current concepts. Orthopedics. 2015;38(6):e452–e6.
35. Murphy NA, Firth S, Jorgensen T, Young PC. Spinal surgery in children with idiopathic and neuromuscular scoliosis. what's the difference? J Pediatr Orthop. 2006;26(2):216–20.
36. Balmer G, MacEwen GD. The incidence and treatment of scoliosis in cerebral palsy. J Bone Joint Surg. 1970;52(1):134–7.
37. Madigan RR, Wallace SL. Scoliosis in the institutionalized cerebral palsy population. Spine. 1981;6(6):583–90.
38. Persson-Bunke M, Hägglund G, Lauge-Pedersen H, Wagner P, Westbom L. Scoliosis in a total population of children with cerebral palsy. Spine. 2012;37(12):E708–E13.
39. Jones KB, Sponseller PD, Shindle MK, McCarthy ML. Longitudinal parental perceptions of spinal fusion for neuromuscular spine deformity in patients with totally involved cerebral palsy. J Pediatr Orthop. 2003;23(2):143–9.
40. Mercado E, Alman B, Wright JG. Does spinal fusion influence quality of life in Neuromuscular Scoliosis? Spine. 2007;32(Supplement):S120–S5.
41. McCall RE, Hayes B. Long-term outcome in neuromuscular scoliosis fused only to lumbar 5. Spine. 2005;30(18):2056–60.
42. Takaso M, Nakazawa T, Imura T, Fukuda M, Takahashi K, Ohtori S. Segmental pedicle screw instrumentation and fusion only to L5 in the surgical treatment of flaccid neuromuscular scoliosis. Spine. 2018;43(5):331–8.
43. Berven S, Bradford DS, editors. Neuromuscular scoliosis: causes of deformity and principles for evaluation and management. Seminars in neurology; 2002: Copyright© 2002 by Thieme Medical Publishers, Inc., 333 Seventh Avenue, New ….
44. Perks A, Cheema S, Mohanraj R. Anaesthesia and epilepsy. Br J Anaesth. 2012;108(4):562–71.
45. Koenig S, Gerstner T, Keller A, Teich M, Longin E, Dempfle C-E. High incidence of vaproate-induced coagulation disorders in children receiving valproic acid: a prospective study. Blood Coagul Fibrinolysis. 2008;19(5):375–82.
46. Brenn BR, Theroux MC, Dabney KW, Miller F. Clotting parameters and thromboelastography in children with neuromuscular and idiopathic scoliosis undergoing posterior spinal fusion. Spine. 2004;29(15):E310–E4.
47. Bendon AA, George KA, Patel D. Perioperative complications and outcomes in children with cerebral palsy undergoing scoliosis surgery. Pediatr Anesth. 2016;26(10):970–5.
48. McCarthy JJ, D'Andrea LP, Betz RR, Clements DH. Scoliosis in the child with cerebral palsy. J Am Acad Orthop Surg. 2006;14(6):367–75.
49. Sullivan P, Juszczak E, Lambert B, Rose M, Ford-Adams M, Johnson A. Impact of feeding problems on nutritional intake and growth: Oxford Feeding Study II. Dev Med Child Neurol. 2002;44(7):461–7.
50. Jevsevar DS, Karlin LI. The relationship between preoperative nutritional status and complications after an operation for scoliosis in patients who have cerebral palsy. J Bone Joint Surg Am. 1993;75(6):880–4.

51. Fitzgerald DA, Follett J, Asperen V. Assessing and managing lung disease and sleep disordered breathing in children with cerebral palsy. Paediatr Respir Rev. 2009;10(1):18–24.
52. Piggott H. The natural history of scoliosis in myelodysplasia. J Bone Joint Surg. 1980;62(1):54–8.
53. Geiger F, Parsch D, Carstens C. Complications of scoliosis surgery in children with myelomeningocele. Eur Spine J. 1999;8(1):22–6.
54. Banit DM, Iwinski HJ Jr, Talwalkar V, Johnson M. Posterior spinal fusion in paralytic scoliosis and myelomeningocele. J Pediatr Orthop. 2001;21(1):117–25.
55. Elliott SP, Villar R, Duncan B. Bacteriuria management and urological evaluation of patients with spina bifida and neurogenic bladder: a multicenter survey. J Urol. 2005;173(1):217–20.
56. Hatlen T, Song K, Shurtleff D, Duguay S. Contributory factors to postoperative spinal fusion complications for children with Myelomeningocele. Spine. 2010;35:1294.
57. Kelly KJ, Pearson ML, Kurup VP, Havens PL, Byrd RS, Setlock MA, et al. A cluster of anaphylactic reactions in children with spina bifida during general anesthesia: epidemiologic features, risk factors, and latex hypersensitivity. J Allergy Clin Immunol. 1994;94(1):53–61.
58. Parisi CA, Petriz NA, Busaniche JN, Cortines MC, Frangi FA, Portillo SA, et al. Prevalence of latex allergy in a population of patients diagnosed with myelomeningocele. Arch Argent Pediatr. 2016;114(1):30–5.
59. Banta JV, Bonanni C, Prebluda J. Latex anaphylaxis during spinal surgery in children with myelomeningocele. Dev Med Child Neurol. 1993;35(6):543–8.
60. Kunkel LM. Analysis of deletions in DNA from patients with Becker and Duchenne muscular dystrophy. Nature. 1986;322(6074):73–7.
61. Hoffman EP, Fischbeck KH, Brown RH, Johnson M, Medori R, Loire JD, et al. Characterization of dystrophin in muscle-biopsy specimens from patients with Duchenne's or Becker's muscular dystrophy. N Engl J Med. 1988;318(21):1363–8.
62. Archer JE, Gardner AC, Roper HP, Chikermane AA, Tatman AJ. Duchenne muscular dystrophy: the management of scoliosis. J Spine Surg. 2016;2(3):185–94.
63. Bushby K, Finkel R, Birnkrant DJ, Case LE, Clemens PR, Cripe L, et al. Diagnosis and management of Duchenne muscular dystrophy, part 1: diagnosis, and pharmacological and psychosocial management. Lancet Neurol. 2010;9(1):77–93.
64. Houde S, Filiatrault M, Fournier A, Dubé J, D'Arcy S, Bérubé D, et al. Deflazacort use in Duchenne muscular dystrophy: an 8-year follow-up. Pediatr Neurol. 2008;38(3):200–6.
65. Lebel DE, Corston JA, McAdam LC, Biggar WD, Alman BA. Glucocorticoid treatment for the prevention of scoliosis in children with Duchenne muscular dystrophy: long-term follow-up. J Bone Joint Surg Am Volume. 2013;95(12):1057–61.
66. King W, Ruttencutter R, Nagaraja HN, Matkovic V, Landoll J, Hoyle C, et al. Orthopedic outcomes of long-term daily corticosteroid treatment in Duchenne muscular dystrophy. Neurology. 2007;68(19):1607–13.
67. Kirchmann C, Kececioglu D, Korinthenberg R, Dittrich S. Echocardiographic and electrocardiographic findings of cardiomyopathy in Duchenne and Becker–Kiener muscular dystrophies. Pediatr Cardiol. 2005;26(1):66–72.
68. Strehle E-M, Straub V. Recent advances in the management of Duchenne muscular dystrophy. Arch Dis Child. 2015;100(12):1173–7.
69. Cripe LH, Tobias JD. Cardiac considerations in the operative management of the patient with D uchenne or B ecker muscular dystrophy. Pediatr Anesth. 2013;23(9):777–84.
70. Thrush PT, Allen HD, Viollet L, Mendell JR. Re-examination of the electrocardiogram in boys with Duchenne muscular dystrophy and correlation with its dilated cardiomyopathy. Am J Cardiol. 2009;103(2):262–5.
71. Romfh A, McNally EM. Cardiac assessment in Duchenne and Becker muscular dystrophies. Curr Heart Fail Rep. 2010;7(4):212–8.
72. Soslow JH, Xu M, Slaughter JC, Stanley M, Crum K, Markham LW, et al. Evaluation of echocardiographic measures of left ventricular function in patients with Duchenne muscular dystrophy: assessment of reproducibility and comparison to cardiac magnetic resonance imaging. J Am Soc Echocardiogr. 2016;29(10):983–91.

73. Miller F, Moseley CF, Koreska J. Spinal fusion in Duchenne muscular dystrophy. Dev Med Child Neurol. 1992;34(9):775–86.
74. Harper CM, Ambler G, Edge G. The prognostic value of pre-operative predicted forced vital capacity in corrective spinal surgery for Duchenne's muscular dystrophy. Anaesthesia. 2004;59(12):1160–2.
75. Muenster T, Mueller C, Forst J, Huber H, Schmitt HJ. Anaesthetic management in patients with Duchenne muscular dystrophy undergoing orthopaedic surgery. Eur J Anaesthesiol. 2012;29(10):489–94.
76. Gurnaney H, Brown A, Litman RS. Malignant hyperthermia and muscular dystrophies. Anesth Anal. 2009;109(4):1043–8.
77. Henderson W. Succinylcholine-induced cardiac arrest in unsuspected Duchenne muscular dystrophy. Can Anaesth Soc J. 1984;31(4):444–6.
78. Wick S, Muenster T, Schmidt J, Forst J, Schmitt HJ. Onset and duration of Rocuronium-induced meeting abstracts in patients with Duchenne muscular dystrophy. Anesthesiology J Am Soc Anesthesiol. 2005;102(5):915–9.
79. Ihmsen H, Schmidt J, Schwilden H, Schmitt HJ, Muenster T. Influence of disease progression on the neuromuscular blocking effect of Mivacurium in children and adolescents with Duchenne muscular dystrophy. Anesthesiology. 2009;110(5):1016–9.
80. de Boer HD, van Esmond J, Booij LH, Driessen JJ. Reversal of rocuronium-induced profound neuromuscular block by sugammadex in Duchenne muscular dystrophy. Pediatric Anesthesia. 2009;19(12):1226–8.
81. Noordeen M, Haddad F, Muntoni F, Gobbi P, Hollyer J, Bentley G. Blood loss in Duchenne muscular dystrophy: vascular smooth muscle dysfunction? J Pediatr Orthop B. 1999;8(3):212–5.
82. Forst J, Forst R, Leithe H, Maurin N. Platelet function deficiency in Duchenne muscular dystrophy. Neuromuscul Disord. 1998;8(1):46–9.
83. Levy BJ, Schulz JF, Fornari ED, Wollowick AL. Complications associated with surgical repair of syndromic scoliosis. Scoliosis. 2015;10(1):14.
84. Gjolaj JP, Sponseller PD, Shah SA, Newton PO, Flynn JM, Neubauer PR, et al. Spinal deformity correction in Marfan syndrome versus adolescent idiopathic scoliosis. Spine. 2012;37(18):1558–65.
85. Yamaguchi Y, Samora W, Klamar JE, Tumin D, Tobias JD. Anesthetic management of an adolescent with Marfan syndrome during anterior spinal fusion. J Med Cases. 2018;9(11):366–70.
86. Bauss O, Sadat-Khonsari R, Fenske C, Engelke W, Schwestka-Polly R. Temporomandibular joint dysfunction in Marfan syndrome. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2004;97(5):592–8.
87. Karpman C, Aughenbaugh GL, Ryu JH. Pneumothorax and bullae in Marfan syndrome. Respiration. 2011;82(3):219–24.
88. Van Karnebeek CDM. Natural history of cardiovascular manifestations in Marfan syndrome. Arch Dis Child. 2001;84(2):129–37.
89. Kitaba A, Martin DP, Gopalakrishnan S, Tobias JD. Perioperative visual loss after nonocular surgery. J Anesth. 2013;27(6):919–26.
90. Nemet AY, Assia EI, Apple DJ, Barequet IS. Current concepts of ocular manifestations in Marfan syndrome. Surv Ophthalmol. 2006;51(6):561–75.
91. Milbrandt TA, Johnston CE. Down syndrome and scoliosis: a review of a 50-year experience at one institution. Spine. 2005;30(18):2051–5.
92. Diamond L, Lynne D, Sigman B. Orthopedic disorders in patients with Down's syndrome. Orthop Clin North Am. 1981;12(1):57.
93. Merrick J, Ezra E, Josef B, Hendel D, Steinberg DM, Wientroub S. Musculoskeletal problems in down syndrome European paediatric orthopaedic society survey: the Israeli sample. J Pediatr Orthop B. 2000;9(3):185–92.
94. Lerman JA, Emans JB, Hall JE, Karlin LI. Spinal arthrodesis for scoliosis in Down syndrome. J Pediatr Orthop. 2003;23(2):159–61.

95. Caird MS, Wills BP, Dormans JP. Down syndrome in children: the role of the orthopaedic surgeon. J Am Acad Orthop Surg. 2006;14(11):610–9.
96. Lewanda AF, Matisoff A, Revenis M, Harahsheh A, Futterman C, Nino G, et al. Preoperative evaluation and comprehensive risk assessment for children with D own syndrome. Pediatr Anesth. 2016;26(4):356–62.
97. Irving CA, Chaudhari MP. Cardiovascular abnormalities in Down's syndrome: spectrum, management and survival over 22 years. Arch Dis Child. 2012;97(4):326–30.
98. Bai W, Voepel-Lewis T, Malviya S. Hemodynamic changes in children with Down syndrome during and following inhalation induction of anesthesia with sevoflurane. J Clin Anesth. 2010;22(8):592–7.
99. Banjar HH, Jamil M, Kattan H, Jancy J, Al-Zabin S, Al-Harbi H, et al. Sleep study abnormalities in patients with Down syndrome. Curr Pediatr Res. 2013;17:49–54.
100. Campos MA, Weinstein SL. Pediatric scoliosis and kyphosis. Neurosurg Clin N Am. 2007;18(3):515–29.
101. Fon GT, Pitt MJ, Thies AC Jr. Thoracic kyphosis: range in normal subjects. Am J Roentgenol. 1980;134(5):979–83.
102. McMaster MJ, Singh H. The surgical management of congenital kyphosis and kyphoscoliosis. Spine. 2001;26(19):2146–54.
103. Murray PM, Weinstein SL, Spratt KF. The natural history and long-term follow-up of Scheuermann kyphosis. J Bone Joint Surg Am. 1993;75(2):236–48.
104. Wenger DR, Frick SL. Scheuermann kyphosis. Spine. 1999;24(24):2630.
105. Logroscino G, Mazza O, Aulisa A, Pitta L, Pola E, Aulisa L. Spondylolysis and spondylolisthesis in the pediatric and adolescent population. Childs Nerv Syst. 2001;17(11):644–55.
106. Lonstein JE. Spondylolisthesis in children: cause, natural history, and management. Spine. 1999;24(24):2640.
107. Cloake T, Gardner A. The management of scoliosis in children with cerebral palsy: a review. J Spine Surg. 2016;2(4):299–309.
108. Yuan N, Skaggs DL, Dorey F, Keens TG. Preoperative predictors of prolonged postoperative mechanical ventilation in children following scoliosis repair. Pediatr Pulmonol. 2005;40(5):414–9.
109. Khirani S, Bersanini C, Aubertin G, Bachy M, Vialle R, Fauroux B. Non-invasive positive pressure ventilation to facilitate the post-operative respiratory outcome of spine surgery in neuromuscular children. Eur Spine J. 2014;23(S4):406–11.
110. Florentino-Pineda I. Anesthesia and postoperative management of spinal deformity surgery in growing children. Berlin Heidelberg: Springer; 2011. p. 499–513.
111. Ipp L, Flynn P, Blanco J, Green D, Boachie-Adjei O, Kozich J, et al. The findings of preoperative cardiac screening studies in adolescent idiopathic scoliosis. J Pediatr Orthop. 2011;31(7):764–6.
112. Steinberger J, Moller JH, Berry JM, Sinaiko AR. Echocardiographic diagnosis of heart disease in apparently healthy adolescents. Pediatrics. 2000;105(4):815–8.
113. Kafer ER. Respiratory and cardiovascular functions in scoliosis and the principles of anesthetic management. Anesthesiology J Am Soc Anesthesiol. 1980;52(4):339–51.
114. Mehta M. Growth as a corrective force in the early treatment of progressive infantile scoliosis. J Bone Joint Surg. 2005;87(9):1237–47.
115. Fedorak GT, D'Astous JL, Nielson AN, Macwilliams BA, Heflin JA. Minimum 5-year follow-up of Mehta casting to treat idiopathic early-onset scoliosis. J Bone Joint Surg. 2019;101(17):1530–8.
116. D'Astous JL, Sanders JO. Casting and traction treatment methods for scoliosis. Orthop Clin North Am. 2007;38(4):477–84.
117. Baky FJ, Milbrandt TA, Flick R, Larson AN. Cumulative anesthesia exposure in patients treated for early-onset scoliosis. Spine deformity. 2018;6(6):781–6.
118. Food U. Drug administration. FDA Drug Safety Communication: FDA review results in new warnings about using general anesthetics and sedation drugs in young children and pregnant women; 2017.

119. LaValva SM, MacAlpine EM, Kawakami N, Gandhi JS, Morishita K, Sturm PF, et al. Awake serial body casting for the management of infantile idiopathic scoliosis: is general anesthesia necessary? Spine Deform. 2020;8:1109.
120. Mohan AL, Das K. History of surgery for the correction of spinal deformity. Neurosurg Focus. 2003;14(1):1–5.
121. Harrington PR. The history and development of Harrington Instrumentation. Clin Orthop Relat Res. (1976-2007) 1988. 227:3–5.
122. Hicks JM, Singla A, Shen FH, Arlet V. Complications of Pedicle Screw Fixation in Scoliosis Surgery. Spine. 2010;35(11):E465–E70.
123. Kokoska ER, Gabriel KR, Silen ML. Minimally invasive anterior spinal exposure and release in children with scoliosis. J Soc Laparoendoscopic Surg. 1998;2(3):255.
124. Suk S-I, Kim J-H, Cho K-J, Kim S-S, Lee J-J, Han Y-T. Is anterior release necessary in severe scoliosis treated by posterior segmental pedicle screw fixation? Eur Spine J. 2007;16(9):1359–65.
125. Verma K, Lonner BS, Kean KE, Dean LE, Valdevit A. Maximal pulmonary recovery after spinal fusion for adolescent idiopathic scoliosis. Spine. 2011;36(14):1086–95.
126. Tsirikos AI, Chang W-N, Dabney KW, Miller F. Comparison of One-Stage Versus Two-Stage Anteroposterior Spinal Fusion in Pediatric Patients With Cerebral Palsy and Neuromuscular Scoliosis. Spine. 2003;28(12):1300–5.
127. Soliman D, Maslow A, Bokesch P, Strafford M, Karlin L, Rhodes J, et al. Transoesophageal echocardiography during scoliosis repair: comparison with CVP monitoring. Can J Anaesth. 1998;45(10):925–32.
128. Coté CJ, Lerman J, Anderson BJ, Wilson NC. Coté and Lerman's a practice of anesthesia for infants and children. Philadelphia: Elsevier; 2019.
129. Patil CG, Lad EM, Lad SP, Ho C, Boakye M. Visual loss after spine. Surgery. 2008;33(13):1491–6.
130. De la Garza-Ramos R, Samdani AF, Sponseller PD, Ain MC, Miller NR, Shaffrey CI, et al. Visual loss after corrective surgery for pediatric scoliosis: incidence and risk factors from a nationwide database. Spine J. 2016;16(4):516–22.
131. Lee LA, Roth S, Posner KL, Cheney FW, Caplan RA, Newman NJ, et al. The American Society of Anesthesiologists postoperative visual loss registryanalysis of 93 spine surgery cases with postoperative visual loss. Anesthesiology J Am Soc Anesthesiol. 2006;105(4):652–9.
132. Group PVLS. Risk factors associated with ischemic optic neuropathy after spinal fusion surgery. Anesthesiology. 2012;116(1):15–24.
133. Brown J, Rogers J, Soar J. Cardiac arrest during surgery and ventilation in the prone position: a case report and systematic review. Resuscitation. 2001;50(2):233–8.
134. Bhatnagar V, Jinjil K, Dwivedi D, Verma R, Tandon U. Cardiopulmonary resuscitation: Unusual techniques for unusual situations. J Emerg Trauma Shock. 2018;11(1):31.
135. Shapiro F, Sethna N. Blood loss in pediatric spine surgery. Haemostasis in Spine Surgery. Springer; 2005. p. 6–17.
136. De Kleuver M, Lewis SJ, Germscheid NM, Kamper SJ, Alanay A, Berven SH, et al. Optimal surgical care for adolescent idiopathic scoliosis: an international consensus. Eur Spine J. 2014;23(12):2603–18.
137. Hampton LJ. Complications associated with the use of "controlled hypotension" in anesthesia. Arch Surg. 1953;67(4):549.
138. Santana L, Kiebzak GM, Toomey N, Maul TM. Blood pressure measurements during intraoperative pediatric scoliosis surgery. Saudi J Anaesth. 2020;14(2):152.
139. Malcolm-Smith NA, McMaster MJ. The use of induced hypotension to control bleeding during posterior fusion for scoliosis. J Bone Joint Surg. 1983;65(3):255–8.
140. Tobias JD. Anesthesia for Spinal Surgery in Children. Wiley-Blackwell; 2011. p. 654–77.
141. Kako H, Gable A, Martin D, Beebe A, Thung A, Samora W, et al. A prospective, open-label trial of clevidipine for controlled hypotension during posterior spinal fusion. J Pediatr Pharmacol Therap. 2015;20(1):54–60.

142. Henry D, Carless P, Moxey A, O'Connell D, Stokes B, Fergusson D. Anti-fibrinolytic drugs for reducing blood loss and the need for red blood cell transfusion during and after surgery. Cochrane Database Syst Rev. 2011;19(1):1.
143. McNicol ED, Tzortzopoulou A, Schumann R, Carr DB, Kalra A. Antifibrinolytic agents for reducing blood loss in scoliosis surgery in children. Cochrane Database Syst Rev. 2016;3:CD006883.
144. Johnson DJ, Johnson CC, Goobie SM, Nami N, Wetzler JA, Sponseller PD, et al. High-dose versus low-dose tranexamic acid to reduce transfusion requirements in pediatric scoliosis surgery. J Pediatr Orthop. 2017;37(8):e552–e7.
145. Lam H, Austin T, Nguyen T, Martus J, Schoenecker J. Unexpected higher blood loss associated with higher dose ε-aminocaproic acid in pediatric scoliosis surgery. J Pediatr Orthop B. 2019;28(1):40–4.
146. Green D, Ts' ao C-H, Cerullo L, Cohen I, Ruo TI, Atkinson AJ. Clinical and laboratory investigation of the effects of ε-aminocaproic acid on hemostasis. J Lab Clin Med. 1985;105(3):321–7.
147. Verma K, Errico TJ, Vaz KM, Lonner BS. A prospective, randomized, double-blinded single-site control study comparing blood loss prevention of tranexamic acid (TXA) to epsilon aminocaproic acid (EACA) for corrective spinal surgery. BMC Surg. 2010;10(1):13.
148. Maeda T, Sasabuchi Y, Matsui H, Ohnishi Y, Miyata S, Yasunaga H. Safety of tranexamic acid in pediatric cardiac surgery: a nationwide database study. J Cardiothorac Vasc Anesth. 2017;31(2):549–53.
149. Bowen RE, Gardner S, Scaduto AA, Eagan M, Beckstead J. Efficacy of intraoperative cell salvage systems in pediatric idiopathic scoliosis patients undergoing posterior spinal fusion with segmental spinal instrumentation. Spice. 2010;35(2):246–51.
150. Keeling MM, Gray LA Jr, Brink MA, Hillerich VK, Bland KI, Intraoperative autotransfusion. Experience in 725 consecutive cases. Ann Surg. 1983;197(5):536.
151. Murphy GJ, Rogers CS, Lansdowne WB, Channon I, Alwair H, Cohen A, et al. Safety, efficacy, and cost of intraoperative cell salvage and autotransfusion after off-pump coronary artery bypass surgery: a randomized trial. J Thorac Cardiovasc Surg. 2005;130(1):20–8.
152. Anand N, Idio FG Jr, Remer S, Hoppenfeld S. The effects of perioperative blood salvage and autologous blood donation on transfusion requirements in scoliosis surgery. J Spinal Disord. 1998;11(6):532–4.
153. Program NBRE. National Blood Resource Education Program's (transfusion Therapy Guidelines for Nurses: US Department of Health and Human Services, Public Health Service, National …; 1990.
154. Bess RS, Lenke LG, Bridwell KH, Steger-May K, Hensley M. Wasting of preoperatively donated autologous blood in the surgical treatment of adolescent idiopathic scoliosis. Spine. 2006;31(20):2375–80.
155. Vitale MG, Privitera DM, Matsumoto H, Gomez JA, Waters LM, Hyman JE, et al. Efficacy of preoperative erythropoietin administration in pediatric neuromuscular scoliosis patients. Spine. 2007;32(24):2662–7.
156. Colomina MJ, Godet C, Bagó J, Pellisé F, Villanueva C. Preoperative erythropoietin in spine surgery. Haemostasis in spine surgery. Springer; 2005. p. 40–9.
157. Du Toit G, Relton J, Gillespie R. Acute haemodilutional autotransfusion in the surgical management of scoliosis. J Bone Joint Surg. 1978;60(2):178–80.
158. Fontana JL, Welborn L, Mongan PD, Sturm P, Martin G, Bunger R. Oxygen consumption and cardiovascular function in children during profound intraoperative normovolemic hemodilution. Anesth Analg. 1995;80(2):219–25.
159. Copley LA, Richards BS, Safavi FZ, Newton PO. Hemodilution as a method to reduce transfusion requirements in adolescent spine fusion surgery. Spine. 1999;24(3):219–22.
160. Rajagopalan S, Mascha E, Na J, Sessler DI. The effects of mild perioperative hypothermia on blood loss and transfusion requirement. Anesthesiology. 2008;108(1):71–7.
161. Young V, Watson M. Prevention of perioperative hypothermia in plastic surgery. Aesthet Surg J. 2006;26(5):551–71.

162. Görges M, Ansermino JM, Whyte SD. A retrospective audit to examine the effectiveness of preoperative warming on hypothermia in spine deformity surgery patients. Pediatr Anesth. 2013;23(11):1054–61.
163. Görges M, West NC, Cheung W, Zhou G, Miyanji F, Whyte SD. Preoperative warming and undesired surgical and anesthesia outcomes in pediatric spinal surgery—a retrospective cohort study. Pediatr Anesth. 2016;26(9):866–75.
164. Pan P, Song K, Yao Y, Jiang T, Jiang Q. The impact of intraoperative hypothermia on blood loss and allogenic blood transfusion in total knee and hip arthroplasty: a retrospective study. Biomed Res Int. 2020;2020:1096743.
165. Doufas AG. Consequences of inadvertent perioperative hypothermia. Best Pract Res Clin Anaesthesiol. 2003;17(4):535–49.
166. Beilin B, Shavit Y, Razumovsky J, Wolloch Y, Zeidel A, Bessler H. Effects of mild perioperative hypothermia on cellular immune responses. Anesthesiology J Am Soc Anesthesiol. 1998;89(5):1133–40.
167. Crews JD, Mina M, Johnson E, Guillen J, Simmons J, Joshi A. Risk factors for surgical site infections following vertical expandable prosthetic titanium rib (VEPTR) surgery in children. Spine deformity. 2018;6(6):791–6.
168. Allegranzi B, Zayed B, Bischoff P, Kubilay NZ, de Jonge S, de Vries F, et al. New WHO recommendations on intraoperative and postoperative measures for surgical site infection prevention: an evidence-based global perspective. Lancet Infect Dis. 2016;16(12):e288–303.
169. Carson JL, Guyatt G, Heddle NM, Grossman BJ, Cohn CS, Fung MK, et al. Clinical practice guidelines from the AABB. JAMA. 2016;316(19):2025.
170. Lauscher P, Kertscho H, Schmidt O, Zimmermann R, Rosenberger P, Zacharowski K, et al. Determination of organ-specific anemia tolerance*. Crit Care Med. 2013;41(4):1037–45.
171. Spahn DR, Spahn GH, Stein P. Evidence base for restrictive transfusion triggers in high-risk patients. Transfus Med Hemother. 2015;42(2):110–4.
172. Ohrt-Nissen S, Bukhari N, Dragsted C, Gehrchen M, Johansson PI, Dirks J, et al. Blood transfusion in the surgical treatment of adolescent idiopathic scoliosis-a single-center experience of patient blood management in 210 cases. Transfusion. 2017;57(7):1808–17.
173. Faraoni D, DiNardo JA, Goobie SM. Relationship between preoperative anemia and in-hospital mortality in children undergoing noncardiac surgery. Anesth Anal. 2016;123(6):1582–7.
174. Pujol-Nicolas A, Morrison R, Casson C, Khan S, Marriott A, Tiplady C, et al. Preoperative screening and intervention for mild anemia with low iron stores in elective hip and knee arthroplasty. Transfusion. 2017;57(12):3049–57.
175. Fontanals M, O'Leary JD, Zaarour C, Skelton T, Faraoni D. Preoperative anemia increases the risk of red blood cell transfusion and prolonged hospital length of stay in children undergoing spine arthrodesis surgery. Transfusion. 2019;59(2):492–9.
176. Schwartz DM, Auerbach JD, Dormans JP, Flynn J, Drummond DS, Bowe JA, et al. Neurophysiological detection of impending spinal cord injury during scoliosis surgery. J Bone Joint Surg. 2007;89(11):2440–9.
177. Kwan KY, Koh HY, Blanke KM, Cheung KM. Complications following surgery for adolescent idiopathic scoliosis over a 13-year period: a review of 84,320 patients from the Scoliosis Research Society Morbidity and Mortality Database. Bone Joint J. 2020;102(4):519–23.
178. Murphy RF, Mooney JF. Complications following spine fusion for adolescent idiopathic scoliosis. Curr Rev Musculoskelet Med. 2016;9(4):462–9.
179. Vauzelle C, Stagnara P, Jouvinroux P. Functional monitoring of spinal cord activity during spinal surgery. Clin Orthop Relat Res. (1976-2007) 1973. 93:173–8.
180. Glover CD, Carling NP. Neuromonitoring for scoliosis surgery. Anesthesiol Clin. 2014;32(1):101–14.
181. Vitale MG, Moore DW, Matsumoto H, Emerson RG, Booker WA, Gomez JA, et al. Risk factors for spinal cord injury during surgery for spinal deformity. J Bone Joint Surg-Am Vol. 2010;92(1):64–71.
182. Dicindio S, Theroux M, Shah S, Miller F, Dabney K, Brislin RP, et al. Multimodality monitoring of transcranial electric motor and somatosensory-evoked potentials during surgical

correction of spinal deformity in patients with cerebral palsy and other neuromuscular disorders. Spine. 2003;28(16):1851–5.
183. El-Hawary R, Sucato DJ, Sparagana S, McClung A, Van Allen E, Rampy P. Spinal cord monitoring in patients with spinal deformity and neural axis abnormalities. Spine. 2006;31(19):E698–706.
184. Nash CL Jr, Lorig RA, Schatzinger LA, Brown RH. Spinal cord monitoring during operative treatment of the spine. Clin Orthop Relat Res. 1977;126:100–5.
185. Mendiratta A, Emerson RG. Neurophysiologic intraoperative monitoring of scoliosis surgery. Anethesiol Clin. 2009;26(2):62–9.
186. Nuwer MR, Dawson EG, Carlson LG, Kanim LEA, Sherman JE. Somatosensory evoked potential spinal cord monitoring reduces neurologic deficits after scoliosis surgery: results of a large multicenter survey. Electroencephalograph Clin Neurophysiol/Evoked Potent Sect. 1995;96(1):6–11.
187. Banoub M, Tetzlaff JE, Schubert A. Pharmacologic and physiologic influences affecting sensory evoked potentialsimplications for perioperative monitoring. Anesthesiology J Am Soc Anesthesiol. 2003;99(3):716–37.
188. Wolfe DE, Drummond JC. Differential effects of isoflurane/nitrous oxide on posterior tibial somatosensory evoked responses of cortical and subcortical origin. Anesth Anal. 1988;67(9):852–9.
189. Schubert A, Licina MG, Lineberry PJ. The effect of ketamine on human somatosensory evoked potentials and its modification by nitrous oxide. Anesthesiology J Am Soc Anesthesiol. 1990;72(1):33–9.
190. Pathak KS, Brown R, Cascorbi HF, Nash JC. Effects of fentanyl and morphine on intraoperative somatosensory cortical-evoked potentials. Anesth Analg. 1984;63(9):833–7.
191. Borrissov B, Langeron O, Lille F, Gomola A, Saillant G, Riou B, et al. Effets de l'association propofol-sufentanil sur les potentiels évoqués somesthésiques au cours de la chirurgie du rachis. 1995;14(4):326–30.
192. Tobias JD, Goble TJ, Bates G, Anderson JT, Hoernschemeyer DG. Effects of dexmedetomidine on intraoperative motor and somatosensory evoked potential monitoring during spinal surgery in adolescents. Pediatr Anesth. 2008;18(11):1082–8.
193. Burke D, Hicks R, Stephen J, Woodforth I, Crawford M. Assessment of corticospinal and somatosensoty conduction simultaneously during scoliosis surgery. Electroencelophalogr Clin Neurophisiol. 1992;85(6):388–96.
194. Sloan TB, Heyer EJ. Anesthesia for intraoperative neurophysiologic monitoring of the spinal cord. J Clin Neurophysiol. 2002;19(5):430–43.
195. Park J-H, Hyun S-J. Intraoperative neurophysiological monitoring in spinal surgery. World J Clin Cases. 2015;3(9):765.
196. Burke D, Hicks RG. Surgical monitoring of motor pathways. J Clin Neurophysiol. 1998;15(3):194–205.
197. Wang AC, Than KD, Etame AB, La Marca F, Park P. Impact of anesthesia on transcranial electric motor evoked potential monitoring during spine surgery: a review of the literature. Neurosurg Focus. 2009;27(4):E7.
198. Rohde V. Measurement of motor evoked potentials following repetitive magnetic motor cortex stimulation during isoflurane or propofol anaesthesia. Br J Anesth. 2003;91(4):487–92.
199. Kalkman CJ, Ubags LH, Been HD, Swaan A, Drummond JC. Improved amplitude of myogenic motor evoked responses after paired transcranial electrical stimulation during sufentanil/nitrous oxide anesthesia. Anesthesialogy. 1995;83(2):270–6.
200. Ubags LH, Kalkman CJ, Been HD, Porsius M, Drummond JC. The use of ketamine or etomidate to supplement sufentanil/N2O anesthesia does not disrupt monitoring of myogenic transcranial motor evoked responses. J Neurosurg Anesthesiol. 1997;9(3):228–33.
201. Zaarour C, Engelhardt T, Strantzas S, Pehora C, Lewis S, Crawford MW. Effect of low-dose ketamine on voltage requirement for transcranial electrical motor evoked potentials in children. Spine. 2007;32(22):E627–E30.

202. Ushirozako H, Yoshida G, Kobayashi S, Hasegawa T, Yamato Y, Yasuda T, et al. Impact of total propofol dose during spinal surgery: anesthetic fade on transcranial motor evoked potentials. J Neurosurg Spine. 2019;30(5):705–13.
203. Ushirozako H, Yoshida G, Hasegawa T, Yamato Y, Yasuda T, Banno T, et al. Characteristics of false-positive alerts on transcranial motor evoked potential monitoring during pediatric scoliosis and adult spinal deformity surgery: an "anesthetic fade" phenomenon. J Neurosurg Spine. 2020;32(3):423–31.
204. Johnson KB, Egan TD, Kern SE, White JL, McJames SW, Syroid N, et al. The influence of hemorrhagic shock on propofol. Anesthesiology. 2003;99(2):409–20.
205. Devlin VJ, Schwartz DM. Intraoperative neurophysiologic monitoring during spinal surgery. J Am Acad Orthop Surg. 2007;15(9):549–60.
206. Min W-K, Lee H-J, Jeong W-J, Oh C-W, Bae J-S, Cho H-S, et al. Reliability of triggered EMG for prediction of safety during pedicle screw placement in adolescent idiopathic scoliosis surgery. Asian Spine J. 2011;5(1):51.
207. Tsirikos AI, Duckworth AD, Henderson LE, Michaelson C. Multimodal intraoperative spinal cord monitoring during spinal deformity surgery: efficacy, diagnostic characteristics, and algorithm development. Med Princ Pract. 2020;29(1):6–17.
208. Mayer EK, Sevdalis N, Rout S, Caris J, Russ S, Mansell J, et al. Surgical checklist implementation project. Ann Surg. 2016;263(1):58–63.
209. Vitale MG, Skaggs DL, Pace GI, Wright ML, Matsumoto H, Anderson RC, et al. Best practices in intraoperative neuromonitoring in spine deformity surgery: development of an intraoperative checklist to optimize response. Spine deformity. 2014;2(5):333–9.
210. Lewis SJ, Wong IHY, Strantzas S, Holmes LM, Vreugdenhil I, Bensky H, et al. Responding to intraoperative neuromonitoring changes during pediatric coronal spinal deformity surgery. Global Spine J. 2019;9(1_suppl):15S–21S.
211. Buckwalter JA, Yaszay B, Ilgenfritz RM, Bastrom TP, Newton PO, Group HS. Analysis of intraoperative neuromonitoring events during spinal corrective surgery for idiopathic scoliosis. Spine Deformity. 2013;1(6):434–8.
212. Samdani AF, Bennett JT, Ames RJ, Asghar JK, Orlando G, Pahys JM, et al. Reversible intraoperative neurophysiologic monitoring alerts in patients undergoing arthrodesis for adolescent idiopathic scoliosis. J Bone Joint Surg. 2016;98(17):1478–83.
213. Yang J, Skaggs DL, Chan P, Shah SA, Vitale MG, Neiss G, et al. Raising mean arterial pressure alone restores 20% of intraoperative neuromonitoring losses. Spine. 2018;43(13):890–4.
214. Bracken MB, Shepard MJ, Holford TR, Leo-Summers L, Aldrich EF, Fazl M, et al. Administration of methylprednisolone for 24 or 48 hours or tirilazad mesylate for 48 hours in the treatment of acute spinal cord injury: results of the third national acute spinal cord injury randomized controlled trial. JAMA. 1997;277(20):1597–604.
215. Pahys JM, Guille JT, D'Andrea LP, Samdani AF, Beck J, Betz RR. Neurologic injury in the surgical treatment of idiopathic scoliosis: guidelines for assessment and management. J Am Acad Orthop Surg. 2009;17(7):426–34.
216. Bowers CA, Kundu B, Hawryluk GW. Methylprednisolone for acute spinal cord injury: an increasingly philosophical debate. Neural Regen Res. 2016;11(6):882.
217. Chan P, Skaggs DL, Sanders AE, Villamor GA, Choi PD, Tolo VT, et al. Pain is the greatest preoperative concern for patients and parents before posterior spinal fusion for adolescent idiopathic scoliosis. Spine. 2017;42(21):E1245–E50.
218. Connelly M, Fulmer RD, Prohaska J, Anson L, Dryer L, Thomas V, et al. Predictors of postoperative pain trajectories in adolescent idiopathic scoliosis. Spine. 2014;39(3):E174–E81.
219. Kehlet H, Dahl JB. The value of "multimodal" or "balanced analgesia" in postoperative pain treatment. Anesth Anal. 1993;77(5):1048–56.
220. Seki H, Ideno S, Ishihara T, Watanabe K, Matsumoto M, Morisaki H. Postoperative pain management in patients undergoing posterior spinal fusion for adolescent idiopathic scoliosis: a narrative review. Scoliosis Spinal Disorders. 2018;13(1):17.
221. Woolf CJ, Chong M-S. Preemptive analgesia—treating postoperative pain by preventing the establishment of central sensitization. Anesth Anal. 1993;77(2):362–79.

222. Kwan MK, Chiu CK, Chan TS, Chong KI, Mohamad SM, Hasan MS, et al. Trajectory of postoperative wound pain within the first 2 weeks following posterior spinal fusion surgery in adolescent idiopathic scoliosis patients. Spine. 2017;42(11):838–43.
223. Kukkar A, Bali A, Singh N, Jaggi AS. Implications and mechanism of action of gabapentin in neuropathic pain. Arch Pharm Res. 2013;36(3):237–51.
224. Trzcinski S, Rosenberg RE, Vasquez Montes D, Sure A, Zhou P, Tishelman J, et al. Use of Gabapentin in posterior spinal fusion is associated with decreased postoperative pain and opioid use in children and adolescents. Clin Spine Surg. 2019;32(5):210–4.
225. Mayell A, Srinivasan I, Campbell F, Peliowski A. Analgesic effects of gabapentin after scoliosis surgery in children: a randomized controlled trial. Pediatr Anesth. 2014;24(12):1239–44.
226. Rusy LM, Hainsworth KR, Nelson TJ, Czarnecki ML, Tassone JC, Thometz JG, et al. Gabapentin use in pediatric spinal fusion patients: a randomized, double-blind, controlled trial. Anesth Anal. 2010;110(5):1393–8.
227. Martinez JL, Sutters KA, Waite S, Davis J, Medina E, Montano N, et al. A comparison of oral diazepam versus midazolam, administered with intravenous meperidine, as premedication to sedation for pediatric endoscopy. J Pediatr Gastroenterol Nutr. 2002;35(1):51–8.
228. Gornitzky AL, Flynn JM, Muhly WT, Sankar WN. A rapid recovery pathway for adolescent idiopathic scoliosis that improves pain control and reduces time to inpatient recovery after posterior spinal fusion. Spine Deformity. 2016;4(4):288–95.
229. Fletcher ND, Andras LM, Lazarus DE, Owen RJ, Geddes BJ, Cao J, et al. Use of a novel pathway for early discharge was associated with a 48% shorter length of stay after posterior spinal fusion for adolescent idiopathic scoliosis. J Pediatr Orthop. 2017;37(2):92–7.
230. Naduvanahalli Vivekanandaswamy A, Prasad Shetty A, Mugesh Kanna R, Shanmuganathan R. An analysis of the safety and efficacy of dexmedetomidine in posterior spinal fusion surgery for adolescent idiopathic scoliosis: a prospective randomized study. Eur Spine J. 2020;30:698.
231. Doyle E, Robinson D, Morton N. Comparison of patient-controlled analgesia with and without a background infusion after lower abdominal surgery in children. Br J Anaesth. 1993;71(5):670–3.
232. Gaukroger PB, Tomkins D, Van Der Walt J. Patient-controlled analgesia in children. Anaesth Intensive Care. 1989;17(3):264–8.
233. Berde CB, Lehn BM, Yee JD, Sethna NF, Russo D. Patient-controlled analgesia in children and adolescents: a randomized, prospective comparison with intramuscular administration of morphine for postoperative analgesia. J Pediatr. 1991;118(3):460–6.
234. Gall O, Aubineau J-V, Bernière J, Desjeux L, Murat I. Analgesic effect of low-dose intrathecal morphine after spinal fusion in children. Anesthesiology. 2001;94(3):447–52.
235. Ibach BW, Loeber C, Shukry M, Hagemann TM, Harrison D, Johnson PN. Duration of intrathecal morphine effect in children with idiopathic scoliosis undergoing posterior spinal fusion. J Opioid Manag. 2015;11(4):295–303.
236. Li Y, Hong RA, Robbins CB, Gibbons KM, Holman AE, Caird MS, et al. Intrathecal morphine and oral analgesics provide safe and effective pain control after posterior spinal fusion for adolescent idiopathic scoliosis. Spine. 2017;1
237. Tripi PA, Poe-Kochert C, Potzman J, Son-Hing JP, Thompson GH. Intrathecal morphine for postoperative analgesia in patients with idiopathic scoliosis undergoing posterior spinal fusion. Spine. 2008;33(20):2248–51.
238. Klatt JWB, Mickelson J, Hung M, Durcan S, Miller C, Smith JT. A randomized prospective evaluation of 3 techniques of postoperative pain management after posterior spinal instrumentation and fusion. Spine. 2013;38(19):1626–31.
239. Sucato DJ, Duey-Holtz A, Elerson E, Safavi F. Postoperative analgesia following surgical correction for adolescent idiopathic scoliosis: a comparison of continuous epidural analgesia and patient-controlled analgesia. 2005;30(2):211–7.
240. Choudhry DK, Brenn BR, Sacks K, Shah S. Evaluation of Gabapentin and Clonidine use in children following spinal fusion surgery for idiopathic scoliosis. J Pediatr Orthop. 2019;39(9):e687–e93.

241. Tobias J. Dexmedetomidine: applications in pediatric critical care and pediatric anesthesiology. Pediatr Crit Care Med. 2007;8(2):115–31.
242. Pendi A, Field R, Farhan S-D, Eichler M, Bederman SS. Perioperative ketamine for analgesia in spine surgery. Spine. 2018;43(5):E299–307.
243. Perelló M, Artés D, Pascuets C, Esteban E, Ey Batlle AM. Prolonged perioperative low-dose ketamine does not improve short and long-term outcomes after pediatric idiopathic scoliosis surgery. Spine. 2017;42(5):E304–E12.
244. Ricciardelli RM, Walters NM, Pomerantz M, Metcalfe B, Afroze F, Ehlers M, et al. The efficacy of ketamine for postoperative pain control in adolescent patients undergoing spinal fusion surgery for idiopathic scoliosis. Spine Deformity. 2020;8:1–8.
245. Hiller A, Helenius I, Nurmi E, Neuvonen PJ, Kaukonen M, Hartikainen T, et al. Acetaminophen improves analgesia but does not reduce opioid requirement after major spine surgery in children and adolescents. Spine. 2012;37(20):E1225–E31.
246. Politi JR, Davis RL, Matrka AK. Randomized prospective trial comparing the use of intravenous versus oral acetaminophen in total joint arthroplasty. J Arthroplasty. 2017;32(4):1125–7.
247. Vitale MG, Choe JC, Hwang MW, Bauer RM, Hyman JE, Lee FY, et al. Use of ketorolac tromethamine in children undergoing scoliosis surgery. Spine J. 2003;3(1):55–62.
248. Sucato DJ, Lovejoy JF, Agrawal S, Elerson E, Nelson T, McClung A. Postoperative Ketorolac does not predispose to pseudoarthrosis following posterior spinal fusion and instrumentation for adolescent idiopathic scoliosis. Spine. 2008;33(10):1119–24.
249. Li Q, Zhang Z, Cai Z. High-dose Ketorolac affects adult spinal fusion. Spine. 2011;36(7):E461–E8.
250. Hu D, Onel E, Singla N, Kramer WG, Hadzic A. Pharmacokinetic profile of liposome bupivacaine injection following a single administration at the surgical site. Clin Drug Investig. 2013;33(2):109–15.
251. Tomov M, Tou K, Winkel R, Puffer R, Bydon M, Nassr A, et al. Does subcutaneous infiltration of liposomal Bupivacaine following single-level transforaminal lumbar interbody fusion surgery improve immediate postoperative pain control? Asian Spine J. 2018;12(1):85–93.
252. Puffer RC, Tou K, Winkel RE, Bydon M, Currier B, Freedman BA. Liposomal bupivacaine incisional injection in single-level lumbar spine surgery. Spine J. 2016;16(11):1305–8.
253. Cloyd C, Moffett BS, Bernhardt MB, Monico EM, Patel N, Hanson D. Efficacy of liposomal bupivacaine in pediatric patients undergoing spine surgery. Pediatr Anesth. 2018;28(11):982–6.
254. Chughtai M, Sultan AA, Hudson B, Goodwin RC, Seif J, Khlopas A, et al. Liposomal Bupivacaine is both safe and effective in controlling postoperative pain after spinal surgery in children. Clin Spine Surg Spine Publication. 2020;33:E533. Publish Ahead of:1
255. Rao RR, Hayes M, Lewis C, Hensinger RN, Farley FA, Li Y, et al. Mapping the road to recovery. 2016;1
256. Jones S, Alnaib M, Kokkinakis M, Wilkinson M, St Clair Gibson A, Kader D. Pre-operative patient education reduces length of stay after knee joint arthroplasty. Ann Royal Coll Surg Engl. 2011;93(1):71–5.
257. Julien-Marsollier F, Michelet D, Assaker R, Doval A, Louisy S, Madre C, et al. Enhanced recovery after surgical correction of adolescent idiopathic scoliosis. Pediatr Anesth. 2020;31:376.
258. Shan L-Q, Skaggs DL, Lee C, Kissinger C, Myung KS. Intensive care unit versus hospital floor. J Bone Joint Surg. 2013;95(7):e40.
259. Haber LL, Womack ED, Sathyamoorthy M, Moss JA, Shrader MW. Who needs a pediatric intensive care unit after posterior spinal fusion for adolescent idiopathic scoliosis? Spine deformity. 2018;6(2):137–40.
260. Auerbach JD, Kean K, Milby AH, Paonessa KJ, Dormans JP, Newton PO, et al. Delayed postoperative neurologic deficits in spinal deformity surgery. Spine. 2016;41(3):E131–E8.
261. Fletcher ND, Bellaire LL, Dilbone ES, Ward LA, Bruce RW. Variability in length of stay following neuromuscular spinal fusion. Spine Deformity. 2020;8:1–8.

Regenerative Therapies

30

Nenna Nwazota and Amr Hegazi

Introduction

Historically, the term 'regenerative medicine' was first used in a 1992 article on hospital administration when the author listed the technologies which would impact the future of hospitals [1]. After many years of basic research, this approach is beginning to represent a valuable treatment option for acute injuries, chronic diseases and congenital malformations.

Future applications of stem cells include Parkinson's disease, coronary artery disease, cardiomyopathy, congestive heart failure, bone marrow transplants, leukemia, and cell replacement therapy in neurological disease to name a few [2]. Furthermore, the advancements of medical science presume using stem cells to treat cancer, muscle damage, autoimmune disease, and spinal cord injuries among several other impairments and diseases. In general, stem cell research has created hope for potential therapeutic application.

Regulatory Landscape

The U.S. Food and Drug Administration (FDA)'s role has expanded to include new therapies, such as biologics, as long as they are used to treat serious illnesses. The FDA's Center for Biologics Evaluation and Research's (CBER) Office of Cellular,

N. Nwazota
Department of Anesthesiology and Pain Management, UT Southwestern Medical Center, Dallas, TX, USA
e-mail: Nenna.Nwazota@utsouthwestern.edu

A. Hegazi (✉)
Department of Physical Medicine and Rehabilitation, UT Southwestern Medical Center, Dallas, TX, USA
e-mail: Hegazi@utsouthwestern.edu

© The Author(s), under exclusive license to Springer Nature Switzerland AG 2022

C. E. Noe (ed.), *Multidisciplinary Spine Care*,
https://doi.org/10.1007/978-3-031-04990-3_30

Tissue, and Gene Therapies (OCTGT) has been tasked with overseeing use of human cell and tissue-based products (HCT/P). In 2016 the FDA passed the twenty-first Century Cures Act which allows for expedited processing of regenerative medicine advanced therapies. The Act requires expedited review of regenerative therapies intended to "treat, modify, reverse, or cure" life-threatening conditions if there is an unmet need. There is less federal regulation around regenerative products that are (1) "minimally manipulated", (2) intended for homologous use, (3) not combined with any other product type and (4) have limited systemic effect or intended for autologous use. This includes most blood-derived regenerative therapies such as platelet rich plasma and autologous conditioned serum. Sections 351 and 361 of the FDA's *Public Health Services Act* regulates HCT/Ps that do not meet these criteria.

Hematopoietic stem cells are the only stem cells which are FDA approved, all others are considered investigational. Research or research sponsors must apply for an Investigational New Drug designation prior to conducting clinical trials using HCT/Ps. This oversight is important – some people feel the field of regenerative or stem cell therapy is far less advanced than the public has been led to believe. To that end, in 2017 the FDA announced increased enforcement of regulation and oversight of stem cell clinics.

The Federal State Medical Board created a working group in 2018 to address stem cells and regenerative therapies at the behest of the U.S. Congress. They have created guidelines surrounding best practices for the regulation, promotion, communication and treatments offered at stem cell clinics in the United States. Their recommendations include the FDA requirement for minimal manipulation, guidance around marketing of benefits of these investigational therapies, disclosure requirements for providers and facilities among others. The International Society for Cell & Gene Therapy has convened a global regulatory task force with similar objectives.

While these regulations have clear benefits, there are some drawbacks [3]. Canada and Europe have their own regulatory agencies, Health Canada and European Medicines Agency, respectively. This means requirements vary by country and the complex regulatory landscape makes multi-center regenerative trials challenging. The costly nature of HCT/P development limits trials to academic centers and centers with comparable funding. Despite recent legislation to expedite innovative therapies, there are still several barriers that regenerative researchers need to overcome. This creates difficulty in generating sufficient data in human studies to assess the efficacy of HCT/Ps. Further, there is a lack of formal mechanisms for reporting outcomes of HCT/P therapies and there is no mechanism to report adverse outcomes of these therapies.

Blood-Derived Products

Platelet-Rich Plasma

PRP is also known as platelet-rich growth factor (GFs), platelet-rich fibrin (PRF) matrix, and platelet concentrations (PC). PRP is the most commonly used

biologic in the lumbar spine. Hematologists first created the term PRP in the 1970s to describe the autologous preparations and enrichment of platelets from plasma-concentrate (i.e. with platelet count above that of peripheral blood). It was initially used as a transfusion product to treat patient with thrombocytopenia.

In the late twentieth century PRP found use in surgical applications for aiding wound closure, reducing inflammation, and increasing new cell growth. At that time PRP was mostly used in periodontal surgery, maxillofacial surgery, cosmetic surgery and skin grafting. Over the past 2 decades, PRP therapy has expanded to several other clinical areas including neurosurgery, head and neck surgery, urology, orthopedic/spine surgery, cardiothoracic and general surgery [4]. Despite this broad clinical use, PRP is only FDA approved for use with ligament grafting and approximation of bony matrices during reconstruction.

The main function of platelets is to participate in primary hemostasis through four steps: adhesion, activation, secretion, and aggregation. Platelet granules contain different bioactive chemical mediators, many of which have a fundamental role in hemostasis and tissue healing. Platelets are highly responsive and can alter the environment through the release of growth factors, chemokines, coagulant factors, RNA species, and extracellular vesicles.

PRP is a biological product defined as a portion of the plasma fraction of autologous blood with platelets more than what is typically found in blood [5]. As such, PRP contains not only a high level of platelets but also the full complement of clotting factors. It is enriched with a range of GFs, chemokines and other plasma proteins. The concentration of platelets, and thereby, the concentration of GFs can be 5–10 times greater than usual.

PRP products are divided into four families, based on leukocytes and fibrin content:

1. *Pure platelet-rich plasma (P-PRP)* which is also known as plasma rich in growth factors (PRGF)
2. *Leukocyte-and platelet-rich plasma (L-PRP)*. They are usually in the form of gel or liquid and are characterized by a low-density fibrin network, with or without leukocytes
3. *Platelet-rich fibrin matrix (PRFM)* or *pure platelet-rich fibrin*
4. *Leukocyte-and platelet-rich fibrin (L-PRF)* [6].

P-PRP and P-RFM are the most widely used since they allow for injection and are not prone to clotting.

Growth factors derived from PRP can contribute to tissue regeneration by assisting cell migration, proliferation, differentiation and extra-cellular matrix synthesis [7]. Table 30.1 summarizes numerous growth factors.

The varying GF concentration may have different biologic effects, resulting in the fact that individual differences in GF levels should be considered for reliable interpretation of the biologic functions and standardized application of PRP. There are several factors affecting GF concentration in PRP – donor-related (e.g., age, gender, comorbidities, medications, nutritional status), processing-related (e.g.,

Table 30.1 Growth factors with roles in tissue regeneration

Growth Factor	Function
Platelet-derived (PDGF)	Enhances collagen synthesis, proliferative activity, macrophage activation.
Transforming growth factor beta (TGF-B)	Enhances synthesis of type one collagen, promotes angiogenesis, stimulates, chemotaxis of immune cells, inhibits osteoclasts formation and bone resorption.
Vascular endothelial growth factor (VEGF)	Stimulates angiogenesis, migration and mitosis of endothelial cells, increases permeability of the vessels, stimulates chemotaxis of macrophages, and neutrophils.
Epidermal growth factor (EGF)	Stimulates cellular proliferation, differentiation of epithelial cells, promotes cytokine secretion by mesenchymal and epithelial cells.
Insulin-like growth factor (IGF)	Promotes cell growth, differentiation, recruitment in bone, blood vessel, skin and other tissue, stimulates collagen synthesis together with PDGF.
Hepatocyte growth factor (HGF)	Promotes angiogenesis, promotes cellular proliferation and resists to apoptosis.
Fibroblast growth factor (FGF)	Promotes proliferation of mesenchymal cell, chondrocytes osteoblasts and stimulates the growth and differentiation of chondrocytes and osteoblasts.

collection & storage conditions, spin protocol, activation protocol, storage), delivery-related (e.g., form of delivery, timing in relation to isolation, timing in relation to activation), host factors, and chronicity of injury to name a few.

There are many commercial systems available to create PRP from autologous whole blood. In most of the available systems, whole blood is centrifuged, which separates the samples cellular products based on different specific gravity. PRP can be prepared by the:

1. PRP method: it is performed by two consecutive centrifugations. During the first one (10 minutes), the blood separates into red corpuscular base, buffy coat and the platelet poor plasma. The last two components are re-centrifuged for 10 more minutes after which PRP will be collected in the bottom of the tube and suspended in a minimal quantity of plasma (2–4 ml) by gently shaking the tube [8].
2. Buffy-coat method: Before centrifugation whole blood is stored at 20–24 C. The blood is centrifuged at a high speed resulting in three layers: a bottom layer consists of red blood cells; a middle layer consists of platelets and white blood cells (Buffy coat) and a top layer contains platelet-poor plasma (PPP). The top layer is discarded from the tube. The buffy-coat layer is transferred to another sterile tube, then centrifuged at low speed to separate white blood cells.

In order to optimize PRP extraction, it is necessary to follow several important parameters. First, the centrifugation process should be sterile and accurately performed to produce optimal platelet separation and sequestration with minimal damage or lysis, which could result in premature release of growth factors. Second, platelet concentration should be at least 2.5 times higher than the platelet

concentration in plasma [9]. Some cite a concentration of at least 300–400% greater in order for PRP to have therapeutic effect [10].

There is no consensus on whether platelets must be activated before their application. Some authors activate platelets with thrombin or calcium while others apply platelets without activating them claiming better results [11]. The choice of exogenous pre-activation of platelets and the number of applications is important in choosing a suitable protocol. Anti-thrombotic drugs are likely to effect PRP efficacy and they should be held prior to PRP application. Similarly, NSAIDs may alter the efficacy of PRP due to their inhibitory platelet effects and bone healing.

Studies have shown that both PRP and stem cells (SC) can complement each other and might have advantage when used in combination. For example, PRP may offer a suitable microenvironment for MSCs by promoting proliferation and differentiation and accelerating wound healing capabilities. PRP can be a powerful tool to attract cell populations, such as MSCs, a combination of which provides a promising approach for treatment [12]. It has also been mentioned that PRP can contribute to the proliferation of bone marrow mesenchymal stem cells and their differentiation into osteoblasts [13]. These authors also felt PRP may be the ideal origin of GFs for many applications.

Autologous Conditioned Serum

Autologous conditioned serum (ACS) was first described in the 1990s as a method of delivering IL-1Ra to musculoskeletal tissues. IL-1Ra is the first naturally occurring receptor antagonist of any cytokine or hormone to be described in scientific literature. IL-1Ra is a natural anti-inflammatory protein in arthritis, colitis, and some pulmonary diseases. Its use has been approved by the FDA for treatment of rheumatoid arthritis. Published studies on use of IL-1Ra, commercially known as anakinra, in sepsis and rheumatoid arthritis have shown weak results. However, there have been more robust responses in treatment of juvenile arthritis, gout, pseudogout and other rare autoinflammatory disorders [14].

ACS is produced by drawing peripheral blood into a syringe containing glass beads which allow adherent cells to attach. Then the blood is incubated during which time platelets degranulate and mononuclear cells synthesize and secrete IL-1Ra among other cytokines. Thus, ACS contains many cytokines and growth factors that may play a role in its regenerative capacity. Most research on ACS to date has studied intra- and peri-articular administration, but there are some studies on use of ACS in the cervical and lumbar spine.

Cellular Products

Cellular therapies typically fall into two broad categories: embryonic stem cells and tissue-derived (somatic) stem cells. Somatic stem cells can be further subdivided based on the tissue of origin. Fetal stem cells are isolated from fetal tissues (e.g.,

placenta, decidual tissue, umbilical cord blood, amniotic fluid, etc.). Adult stem cells are similarly isolated from various tissues (e.g., adipose (ASCs), bone marrow (BMSCs), dental pulp, skeletal muscle, etc.). Two newer somatic stem cells have recently been described – induced pluripotent stem cells (iPSCs) and induced tissue-specific stem cells (iTS).

Stem cells (SCs) were first reported in 1909 with the discovery of hematopoietic stem cells isolated from bone marrow. These cells were first identified as multipotent in the 1970s. The term "mesenchymal stem cells" (MSCs) became popularized as these cells were seen to be multipotent, self-renewing with immunomodulatory properties. The abbreviation MSC may refer to "marrow stromal cell", "multipotent stromal cells", mesodermal stem cells" or "mesenchymal stromal cells" because it is now known that these cells represent a heterogenous population of nonclonal cells. MSCs are currently the most studied experimental cellular therapy likely due to their potential for broad therapeutic application [15]. Research to date has demonstrated their ease of isolation and cellular expansion; multipotency; immunomodulatory, antimicrobial and regenerative effects; homing/migratory capacity to sites of injury; and safety profile. MSCs are capable of restoring damaged tissues through their angiogenic and paracrine anti-inflammatory properties.

There are several factors affecting the translational application of MSCs. Namely, we have yet to identify the optimal mode of administration, culture and expansion technique, or viability and preservation method. Though current research has provided some insight.

The origin of MSCs is important since the source typically dictates differentiation potential. For example, MSCs derived from adipose tissue default to adipogenic potential and those from bone marrow to osteogenic potential [9]. However, there are other factors affecting differentiation such as culture medium. Also, mechanical loading has shown to increase MSC chondrogenesis in several studies [16]. Similarly, cellular expansion techniques can affect MSC growth, survival and differentiation *in vitro* [17, 18].

MSCs do not express histocompatibility complexes and do not require immunosuppression after administration. They are most effective in degenerative diseases where there is minimal inflammation [9]. They are capable of homing to sites of injury through a network of chemoattractants [9].

After intravenous infusion MSCs are mostly sequestered in the lung due to a pulmonary first pass effect [19]. MSC survival is short lived after injection into ischemic tissue. Culture media with low oxygen tension and low glucose concentrations may precondition MSCs for survival in ischemic and avascular tissues [17].

MSC preservation is a similar challenge for researchers and clinicians. The thawing process can induce apoptosis, though this cell death may be overcome by acquisition of MSCs and injection in the same setting. One downside to this approach is that it will not achieve the benefits of culture media. Capacity of MSCs is not affected by needle gauge used at time of extraction when comparing needle gauges [9]. It must be noted, though, that larger bore needles are associated with less

apoptosis. It has also been postulated that the recipient's immunologic profile can influence the effect of MSCs such that patients with higher levels of systemic inflammation may exhibit a decreased therapeutic effect [17].

True adult stem cells are scarce and do not differentiate well so facilities commonly expand and enrich MSCs for their intended use. There is no standardized process for MSC manufacturing. One 2019 study [18] examined 15 such facilities and found the intended use of MSCs was broad ranging from wound healing to pulmonary disease. Several facilities cited three or more intended uses. 93.3% of facilities isolated MSCs from bone marrow and only 10 facilities cited a single tissue source. Most facilities validated their product based on cell surface marker expression. Purity was defined by negative marker expression ranging from less than 10% to less than 2% and for positive markers the range was >10% to >95%. This broad range reflects the heterogeneity that exists in the MSC manufacturer market.

Embryonic Stem Cells

Embryonic stem cells (ESCs) are derived from the pre-implantation blastocyst and possess the ability to differentiate into any mature cell type. ESCs have the hallmark ability for self-renewal, pluripotency and genomic stability. Use of ESCs in clinical medicine is limited, however, due to the real risk of teratoma formation, difficult acquisition process and ethical controversy.

Non-Embryonic Stem Cells

Adult stem cells, hereafter referred to as MSCs, arise from the perivascular space and can be derived from nearly any vascularized tissue. The International Society for Cellular Therapy has published minimum criteria for MSCs. They must be plastic adherent and express certain, defined cell surface markers such as CD34 which is most closely associated with "stemness" [20]. In order to be classified as MSCs they must also possess the capacity for *in vitro* differentiation to three tissue types – osteoblasts, adipocytes and chondroblasts. They typically exist in a quiescent state until they are activated by a specific event such as local trauma.

Fetal SCs are multipotent with less ethical restrictions than embryonic stem cells. They have less differential potential than their embryonic counterparts but possess increased differentiation potential compared to other adult SCs. Notably, some researchers do not distinguish between adult and fetal SCs as certain sources contain both maternal and fetal cellular products. Umbilical cord derived MSCs are thought to be superior to BM-MSCs and ASCs in terms of culture length, proliferation capacity, expansion capacity and storage length [15]. A major downside to umbilical cord derived MSCs is its procoagulant effects with reports of thrombotic and thromboembolic events in human studies.

Bone marrow (BM-MSC) and adipose-derived stem cells (ASC) are the most studied to date. ASCs and BM-MSCs have angiogenic properties and are good for use in hypovascular and avascular regions.

BM-MSC contains mostly hematopoietic stem cells and only a small quantity of true pluripotent cells. Thus, use of BM-MSCs requires a large volume of bone marrow. Marrow is typically harvested from the sternum or the posterior iliac crest. There are reports of fatal embolic events related to aspiration process.

Adipose-derived stem cells are easier to acquire and have relatively fewer complications associated with their retrieval. The concept of ASCs was first described in 1964 when stromal vascular progenitor cells were isolated from rat adipose tissue [21]. Much later in 2001 MSCs were isolated from human adipose tissue [22]. Zuks et al. showed that adipose tissue contains a cellular fraction capable of differentiation to adipogenic, chondrogenic, osteogenic and myogenic lineages. Their research also showed that ASCs are easily expanded with less cellular senescence than BM-MSCs. This finding was very useful and, increasingly, ASCs have become the MSC of choice due to their relative ease of procurement, faster proliferation, excellent differentiation ability, increased viability, and minimal adverse effects or ethical concerns. There is still the need for a standardized protocol, but ASCs have demonstrated immense potential in the field of regenerative medicine.

In order to retrieve ASCs, the products of liposuction (commonly known as the lipoaspirate) undergo enzymatic digestion by collagenase to form an aqueous byproduct. This byproduct is known as the stromal vascular fraction (SVF) and it is composed of a heterogenous cell mixture that includes ASCs, endothelial precursors, endothelial cells, macrophages, lymphocytes, and pericytes among others. Based on the processing method, ASCs may comprise up to 10% of the SVF [15]. Studies have shown that SVF has therapeutic potential and, in certain applications, may perform better than ASCs [20]. SVF is also much easier to obtain as it does not require culture or expansion. This lack of exposure to reagents may also make it safer for human use. However, due to its heterogeneity, SVF is more likely to elicit an immune response and autologous use is preferable to allotransplantation. SVF is subject to less stringent regulatory criteria than ASCs, especially when the newer, mechanical methods of SVF extraction are implemented rather than enzymatic digestion. There are several point-of-care SVF isolation devices in development.

Induced pluripotent stem cells are yet another type of cellular regenerative therapy. iPSCs are reprogrammed somatic cells which resemble embryonic stem cells [23]. iPSCs have the capacity for self-renewal and pluripotency. Their creation involves co-culture with primary cells, derivation using growth factors or small molecules, differentiation through progenitors (e.g., MSCs, osteoblasts, myoblasts) and/or differentiation through embryoid body formation [24]. They can be derived from easily accessible cells unlike most other adult MSC. Also, MSCs have several downsides including variation among donors, effects of donor age and health status, heterogenicity, cell scarcity and invasive techniques required to obtain MSCs. iPSCs obviate many of these downsides and national repositories of iPSCs are being created to model nearly every human disease. Despite this, the scarcity of standardized differentiation protocols may limit use of this technology.

Other

Another biologic of interest are exosomes. Exosomes are tiny microvesicles released by cells during various physiologic events. Exosomes are 30–100 nanometers big and microvesicles are larger than 100 nanometers. They are secreted from a range of cells from T-cells to dendritic cells and are thought to play a role in cellular regulation. They secrete proteins, micro RNA and messenger RNA and home to cites of injury similar to MSCs. There are several advantages of exosomes over MSCs including no clumping, no pulmonary first pass effect when administered intravenously and ability to cross the blood-brain barrier [9].

Gene therapy is another alternative regenerative therapy that has shown potential in treating a variety of conditions. It involves transfection of degenerated cells with genes promoting repair. A variety of vectors have been studied, though traditionally viral vectors have been used. Gene therapy can be conducted *ex vivo* or, more recently, *in vivo*. Most clinical trials are aimed at treating cancer, monogenic diseases, cardiovascular disease and infectious disease [25]. There is some evidence that gene therapy can be used to treat degenerative joint conditions. However, due to the limited innate regenerative capacity within degenerated IVDs, some researchers advocate a combination of gene therapy with stem cell therapy in treatment of DDD [26].

Alternative regenerative therapies include prolotherapy and tenotomy. Prolotherapy involves injection of a small volume of irritant in order to initiate a local inflammatory response. It is believed that this inflammatory response can cause hypertrophy and strengthen lax collagenous structures such as ligaments and tendon insertions. Prolotherapy has been used to treat musculoskeletal conditions including joint pain, headache and low back pain for nearly a century [27]. Percutaneous tenotomy involves passage of a needle into injured tendons. The thought behind this is the fenestration of damaged tissue can convert a chronic, degenerative process into an acute, inflammatory process which will ultimately lead to healing. Bleeding associated with the tenotomy can also cause local release of growth factors that can aid the healing process.

Regenerative Therapy in Spinal Disease

There is emerging literature on the use of biologics in chronic neck and low back pain. There are studies showing improved analgesia and decreased disability with epidural administration of ACS to treat lumbar [28] and cervical radiculopathy [29]. The majority of the data surrounds use of regenerative therapies in degenerative disc disease. Degenerated intervertebral discs exhibit an altered homeostatic balance between anabolic and catabolic processes favoring proliferation of proteinases and pro-inflammatory cytokines. It is postulated that even degenerated intervertebral discs contain progenitor cells that can be stimulated via cellular or gene therapy to proliferate and differentiate [30]. As mentioned previously co-culture may represent an option for treating DDD. Marrow-derived MSCs from rodents that were cultured

with intact intervertebral disc tissue differentiated into intradiscal-like cells [16]. Culture medium can confer benefit and pre-condition MSCs for use in the intradiscal space since the use of MSCs in degenerated discs requires survival in a depleted environment (e.g., low oxygen tension, acidic pH, poor nutrient, high mechanical load, etc.).

The ideal biologic would not only provide analgesia but also slow or reverse the degeneration and restore normal tissue. Several articles have shown prolonged analgesia and decreased disability following injection of MSCs into degenerated intervertebral discs in human subjects [31, 32]. Few human studies have shown restoration of tissue following MSC injection. The American Society for Interventional Pain Physicians published a position paper citing level 3 evidence for use of MSC and PRP for treatment of lumbar discogenic pain and level 4 evidence for use of PRP for treatment of lumbar facet joint disorders, use of PRP in the epidural space to treat lumbar radiculopathy and use of PRP to treat sacroiliac joint pain [9].

There are several ongoing clinical trials evaluating the efficacy of regenerative therapies in lumbar spinal conditions. More information is needed before this therapy can become widespread. We need to confirm whether this therapy is safe and identify the ideal candidate likely to experience meaningful benefits. Also, to date there are no published studies on the use of cell-based therapies in the cervical spine and much of the supportive literature consists of animal studies. Human studies are limited in power and scope. There is little data on safety of cellular therapies and there is no literature on cell-based therapies for treatment of degenerative disc disease in the cervical spine. We need to study allogeneic MSCs more in humans to determine actual risk of rejection. More research is needed to determine appropriate dose and viability of injected cells and to answer question around cell leakage, culture, culture and expansion protocol, preservation, thawing, cost efficiency, etc.

References

1. Kaiser LR. The future of multi- hospital systems. Top Health Care Finance. 1992;18(4):82–45.
2. Kumar R, Sharma A, Varadwaj K. Stem cells: an overview with respect to cardiovascular and renal disease. J Nat BioMed. 2010;1(1):45–52.
3. Kleiderman E, Boily A, Hasilo C, Knoppers BM. Overcoming barriers to facilitate the regulation of multi-centre regnerative medicine clinical trials. Stem Cell Res Ther. 2018;9(1):307–15.
4. Lacci KM, Dardik A. Platelet- rich plasma: support for its use in wound healing. Yale J Biol Med. 2010;83(1):1–9.
5. Alves R, Grimalt R. A randomized placebo-controlled, double- blinded, half-head study to assess the efficacy of platelet-rich plasma on the treatment of androgenetic alopecia. Dermatol Surg. 2016;42:491–7.
6. Dohan Ehrenfest DM, Andia I, Zumstein MA, Zhang CA, Pinto NR, Bieleeki T. Classification of platelet concentrates (Platelet-Rich Plasma, PRP; Platelet-Rich Fibrin, PRF) for topical and infiltrative use in orthopedic and sports medicine: current consensus, clinical implications and perspective. Ligaments Tendons J. 2014;4(1):3–9.
7. Isaac C, Gharaibeh B, Witt M, Wright VJ, Huard J. Biologic approaches to enhance rotator cuff healing after injury. J Shoulder Elb Surg. 2012;21:181–90.

8. Sweeney J, Grossman BJ. Blood collection, storage and component preparation methods. In: Breeher M, editor. Technical manual. 14th ed. Bethesda MD: Am. Assoc. Blood Banks (AABB); 2002. p. 955–8.
9. Navani A, Manchikanti L, Albers SL, Latchaw RE, Sanapati J, Kaye AD, Atluri S, Jordan S, Gupta A, Cedeno D, Vallejo A, Fellows B, Knezevic NN, Pappolla M, Diwan S, Trescot AM, Soin A, Kaye AM, Aydin SM, Calodney AK, Candido KD, Bakshi S, Benyamin RM, Vallejo R, Watanabe A, Beall D, Stitik TP, Foye PM, Helander EM, Hirsch JA. Responsible, safe, and effective use of biologics in the management of low back pain: American Society of Interventional Pain Physicians (ASIPP) guidelines. Pain Physician. 2019;22:S1–S74.
10. Marx RE. Platelet-rich plasma: evidence to support its use. J Oral Maxillofacial Surg. 2004;62:489–96.
11. Scherer SS, Tobalem M, Vigato E, Heit Y, Modorressi A, Heinz B. Non -activated versus thrombin-activated platelets in wound healing and fibroblast -to- myofibroblast differentiation in Vivo and in Vitro. Plast Reconstruction Surg. 2012;129:46e–54e.
12. Rubio D, Garcia-Castro J, Martin MC, de la Fuentes R, Cigudosa JC, Lloyd AC. Spontaneous human adult stem cell transformation. Cancer Res. 2005;65:3033–9.
13. Montgomery SR, Petrigliano FA, Gamradt SC. Biologic augmentation of rotator cuff repair. Curr Rev Musculoskeletal Med. 2011;4:221.
14. Evans CH, Chevalier X, Wehling P. Autologous conditioned serum. Phys Med Rehabil Clin N Am. 2016, nOV;27(4):893–908.
15. Ntege EH, Sunami H, Shimizu Y. Advances in regenerative therapy: a review of the literature and future directions. Regen Ther. 2020;14:136–53.
16. Huang Y, Leung VYL, Lu WW, Luk KDK. The effects of micro-environment in mesenchymal stem cell based regeneration of intervertebral disk. Spine J. 2013;13:352–62.
17. Nolta JA, Galipeau J, Phinney DG. Improving mesenchymal stem/stromal cell potency and survival. Proceedings from the International Society of Cell Therapy (ISCT) MSC preconference held in May 2018, Palais de Congres de Montréal, Organized by the ISCT MSC Scientific Committee. Cytotherapy. 2020;22:123–6.
18. Phinney DG, Galipeau J. Manufacturing mesenchymal stromal cells for clinical applications: a survey of Good Manufacturing Practices at U.S. academic centers. Cytotherapy. 2019;21:782–92.
19. Buchheit T, Huh Y, Maixner W, Cheng J, Ji R-R. Neuroimmune modulation of pain and regenerative pain medicine. J Clin Investig. 2020;130(5):2164–76.
20. Bora P, Majumdar AS. Adipose tissue-derived stromal vascular fraction in regenerative medicine: a brief review on biology and translation. Stem Cell Res Ther. 2017;8:145–55.
21. Rodbell M. Localization of lipoprotein lipase in fat cells of rat adipose tissue. J Biol Chem. 1964;289(3):753–5.
22. Zuk PA, Zhu M, Mizuno H, Huang J, Futrell JW, Katz AJ, et al. Multilineage cells from human adipose tissue: implications for cell-based therapies. Tissue Eng. 2001;7(2):211–28.
23. Xia K, Zhu J, Hua J, Gong Z, Yu C, Zhou X, et al. Intradiscal Injection of Induced Pluripotent Stem Cell-Derived nucleus pulposus-like cell-seeeded polymeric microspheres promotes rat disc regeneration. Stem Cells Int. 2019;2019:6806540.
24. Sanjurjo-Rodriguez C, Castro-Vinuelas R, Pineiro-Ramil M, Rodriguez-Fernandez S, Fuentes-Boquete I, Blanco FJ, Diaz-Prado S. Versatility of induced pluripotent stem cells (iPSCs) for improving the knowledge on musculoskeletal diseases. Int J Mol Sci. 2020;21:6124–51.
25. Wang F, Qin Z, Lu H, He S, Luo J, Jin C, Song X. Clinical translation of gene medicine. J Gene Med. 2019;21(7):e3108–16.
26. Mobasheri A, Richardson SM. Cell and gene therapy for spine regeneration: mammalian protein production platforms for overproduction of therapeutic proteins and growth factors. Neurosurg Clin N Am. 2020;31(1):131–9.
27. Rabago D, Best TM, Beamsley M, Patterson J. A systematic review of prolotherapy for chronic musculoskeletal pain. Clin J Sports Med. 2005;15(5):E376–86.
28. Becker C, Heidersdorf S, Drewlo S, Zirke de Rodriguez S, Kramer J, Willburger RE. Efficacy of epidural perineural injections with autologous conditioned serum for lumbar radicular

compression: an investigator-initiated, prospective, double-blind, reference-controlled study. Spine. 2007;32(17):1803–8.
29. Goni VG, Jhala SS, Gopinathan NR, Behera P, Batra YK, Arjun RHH, et al. Efficacy of epidural perineural injection of autologous conditioned serum in unilateral cervical radiculopathy. Spine. 2015;40(16):E915–21.
30. Sivakamasundari V, Lufkin T. Stemming the degeneration: IVD stem cells and stem cell regenerative therapy for degenerative disk disease. Adv Stem Cells. 2013;2013:724547.
31. Sanapati J, Manchikanti L, Atluri S, Jordan S, Albers SL, Pappolla MA, Kaye AD, Candido KD, Pampati V, Hirsch JA. Do regenerative medicine therapies provide long-term relief in chronic low back pain: a Systematic review and meta-analysis. Pain Physician. 2018;21:515–40.
32. Pettine KA, Suzuki RK, Sand TT, Murphy MB. Autologous bone marrow concentrate intradiscal injection for the treatment of degenerative disc disease with three-year follow-up. Int Orthop. 2017;41:2097–103.

Epidural Catheter Infusion for Post-operative Analgesia for Major Spine Surgery

31

Anthony Machi and Enas Kandil

Why Use an Epidural Catheter

Major spine surgery causes significant pain in the immediate post-operative period. Soft tissue and bone trauma stimulate nociception, inflammation, neuropathic pain and precipitate muscle spasm. Neuropathic pain has been implicated in the occurrence of failed back surgery syndrome (FBSS) where pain persists despite pathology correction [1]. These factors contribute to neurohumoral physiologic consequences including peripheral and central sensitization, transcriptional and post-transcriptional dysregulation, augmented facilitation and disinhibition [2]. In simple terms: surgical spine patients experience significant amount of pain, specifically in the days immediately following surgery, and the degree of pain is an important factor that affects speed of recovery, hospital length of stay and long term surgical outcome [3]. Multimodal analgesia with a combination of pharmaceutical therapies is a cornerstone of perioperative pain management for major spine surgery, while comprehensive multidisciplinary Enhanced Recovery Protocols are increasingly utilized to deploy this intensive pain management, improve outcomes and hasten functional recovery [4–8]. However, for some subsets of patients these interventions and approaches alone are insufficient. One modality which may augment these strategies and maybe useful in select patients and surgical populations is continuous epidural infusion of local anesthetic with or without low-dose opioids or other adjuvants via epidural catheters placed perioperatively [9, 10].

A. Machi (✉) · E. Kandil
Department of Anesthesiology and Pain Management, University of Texas Southwestern Medical Center, Dallas, TX, USA
e-mail: anthony.machi@utsouthwestern.edu; enas.kandil@utsouthwestern.edu

© The Author(s), under exclusive license to Springer Nature Switzerland AG 2022

C. E. Noe (ed.), *Multidisciplinary Spine Care*,
https://doi.org/10.1007/978-3-031-04990-3_31

Benefits

Perioperative epidural analgesia can provide powerful analgesia and can have a multitude of beneficial effects when used for a variety of major surgeries [11, 12]. Due to blockade of nociception and inhibition of sympathetic stimulation these include reduced risk of perioperative mortality, beneficial effects on major cardiovascular, pulmonary and gastrointestinal complications such as decreased risk of venous thromboembolism, myocardial infarction, pneumonia, respiratory depression and ileus. When examined specifically for major spine surgery, the evidence of beneficial effects is more limited because studies of perioperative epidural analgesia for spine surgery have been small (<100 patients) and serious perioperative complications such as myocardial infarction are rare following major spine surgery, occurring in less than 1–2% of patients [13, 14]. Perioperative epidural analgesia is effective in relieving pain following major spine surgery as well as decreasing the incidence of postoperative nausea and vomiting, promoting earlier return of bowel function, and improving patient satisfaction [15–22]. Examination of the effect of post-operative epidural analgesia on the surgical stress response following major spine surgery when compared with systemic opioid analgesia revealed attenuation of pro-inflammatory cytokines IL-1ß, IL-6 and IL-10, decreased hyperglycemia and decreased cortisol which translated into better pain control, earlier mobility, less blood loss, less nausea and vomiting and better patient satisfaction [23]. Similar benefits are found when an epidural catheter is placed directly into the epidural space during surgery (as in many of the cited articles) as when placed preoperative percutaneously by an anesthesiologist [24].

Perioperative epidural analgesia may also provide effective analgesia in special subpopulations of major spine surgery patients at risk for perioperative complication. For example, children with neuromuscular disorders that can cause both pulmonary dysfunction and scoliosis, such as Duchenne muscular dystrophy and spinal muscular atrophy, are a particularly challenging group to provide analgesia without risking further respiratory compromise. In a small retrospective pilot study Saito and colleagues reported safe, excellent and better analgesia in a group that received multimodal analgesia in combination with epidural analgesia compared to a group that received multimodal analgesia alone [25].

Risks

Perioperative epidural analgesia via an epidural catheter is safe with low risk of serious complication. The most common limitations to good analgesia from an epidural infusion are incorrect placement of the epidural catheter and inadequate spread of analgesia. Both of these are resolved in the major spine surgery patient population because the surgeon most commonly places the epidural catheter directly at the correct level at the conclusion of surgery and prior to closure of the surgical wound [26, 27]. In general, rare but serious complications can occur including infection, such as epidural abscess, bleeding, such as epidural hematoma, nerve injury and local anesthetic systemic toxicity [28, 29]. The exact incidence of these serious complications

are unknown but are estimated to be 1/1000–1/150,000 depending on patient and surgical risk factors [28, 30]. We could not find any reports in the literature of infection, hematoma, catheter breakage, catheter retention, nerve injury or local anesthetic systemic toxicity related to continuous perioperative epidural analgesia for spine surgery. This may be a reflection of the improved safety of placement when placed directly into the epidural space during surgery, and that we estimate that fewer than 2000 patients have been reported in the literature utilizing continuous perioperative epidural analgesia for spine surgery. The authors are also unaware of any of these complications occurring in their institution where approximately 150 patients per year receive an epidural catheter infusion for analgesia for major spine surgery.

Evidence of functional limitations of continuous perioperative epidural analgesia for major spine surgery are similarly lacking in the literature but are appreciated in the course of clinical care. The two most common limitations are sympathectomy and obscuring postoperative neurologic assessment. Because the nerves of the sympathetic nervous system originate at the thoracolumbar vertebral levels, blockade with local anesthetic causes inhibition of this system. Such blockade has positive effects on the gastrointestinal system during the perioperative period but can have negative effects on the cardiovascular system in the perioperative period by causing vasodilation of the peripheral and central vasculature that lead to hypotension. Due to operative blood loss, patients undergoing major spine surgery are often hypovolemic and more sensitive to epidural blockade mediated vasodilation. This can be an impediment to delivering sufficient local anesthetic to provide additional adequate post-operative analgesia (epidural infusion of local anesthesia may have to be decreased to the point that pain increases to permit adequate mean arterial pressure for goal end-organ perfusion). Another limitation is the potential need for intraoperative or post-operative neurologic assessment. Intraoperative neuromonitoring of somatosensory evoked potentials and motor evoked potentials may preclude intraoperative use of local anesthetic via an epidural catheter. In addition, if there is concern for operative neurologic compromise in the post-operative period, a local anesthetic infusion through an epidural catheter could temporarily obscure neurologic assessment. Both the sympathectomy and blockade of motor nerves are issues which result from local anesthetic infusion through an epidural catheter. One solution to this would be the temporary usage of opioid or other adjuvant only through the epidural catheter; however, this may reduce the effectiveness of the analgesia that results from the epidural infusion. [29, 31] Additional limitations that can occur relative to the type of medication (local anesthesia vs opioid) that is utilized in the epidural infusion include nausea, vomiting, pruritus, urinary retention, respiratory depression, sedation, dysesthesia, and paresthesia (see Table 31.1). In general,

Table 31.1 Limitations to utilization of epidural medications

Local Anesthesia	Opioid
Sympathectomy: hypotension	Nausea and vomiting
Motor blockade: weakness	Constipation
Affect neurologic assessment	Pruritus
Paresthesia	Urinary retention
Dysesthesia	Respiratory depression
	Delirium
	Sedation

opioids administered via an epidural catheter led to fewer and less significant side effects than when administered systemically at equipotent analgesic amounts with the exception of pruritus and possibly nausea and vomiting [32, 33].

Surgical Procedures and Patient Populations

Given the benefits, risks and limitations, continuous perioperative epidural analgesia is most useful for major open spine surgeries involving multi-day hospitalizations associated with severe pain. These include adult and pediatric scoliosis correction surgery of the thoracic and lumbar spine, such T10-pelvis fusion and T4-L5 fusion, single or multilevel open posterior lumbar decompression and interbody fusion, and open transforaminal lumbar interbody fusion [15, 17, 20, 25]. It would not be recommended for minimally invasive approaches or approaches limited to decompression or disc herniation due to less tissue trauma involvement and generally more rapid recovery. Consequently, it would be most useful for a relatively small portion of the major spine surgery population [5, 34, 35].

Similarly, continuous perioperative epidural analgesia may be most useful in specific patient populations. Patients who have chronic pain and opioid tolerance and adults or children with preoperative pulmonary dysfunction are two such populations. Those with chronic pain and opioid tolerance often exhibit hyperalgesia and allodynia for which blockade with local anesthesia can be very helpful and may not have their analgesia needs otherwise sufficiently addressed by systemic multimodal analgesic agents. Because epidural analgesia does not adversely impact pulmonary function, it can be particularly useful for patients with baseline pulmonary compromise as a powerful analgesic in place of others that directly or indirectly cause respiratory depression, such as opioids, muscle relaxants, and gabapentinoids.

Infusion Solutions and Techniques

Multiple techniques and solutions have been utilized for perioperative analgesia with a continuous epidural infusion for major spine surgery. Examples of regimens of local anesthetics range from bupivacaine 0.0625% at 4 ml/hr. to bupivacaine 0.125% at 10 ml/hr. or ropivacaine 0.1% at 12 ml/hr. to ropivacaine 0.3% 10 ml/hr. [15] It is clear from studies of perioperative epidural analgesia for other types of surgery that a combination of local anesthetic and opioid can provide synergistic analgesia. Few studies have compared different epidural solutions in the setting of major spine surgery, and an optimal regimen is unknown. More likely, there is not a single best regimen nor a single best medication, rather a combination of medications within a range of doses that will likely yield the best analgesic results with the fewest epidural infusion related side effects [31]. The most important characteristic of local anesthesia in an epidural infusion is a longer duration of action that can provide a stable plateau of blockade and analgesia. Examples of local anesthetics

Table 31.2 Common medications used for epidural infusions and administration recommendations

Medication	Concentration	Dose range/hour	Rate	Max dose/hr
Bupivacaine	0.0625–0.125%	8–12 mg/hr	6–14 ml/hr	20 mg
Ropivacaine	0.1–0.3%	10–15 mg/hr	5–15 ml/hr	25 mg
Fentanyl	2–5 mcg/ml	20–30 mcg/hr	6–14 ml/hr	40 mcg
Hydromorphone	5–20 mcg/ml	30–120 mcg/hr	6–12 ml/hr	120 mcg

that provide this are bupivacaine, levobupivacaine and ropivacaine. Lower concentration of local anesthetics decreases the degree of motor blockade that results from an epidural infusion. Similarly, the most important characteristics of opioids in an epidural infusion are their hydrophilicity vs lipophilicity and their duration of action. These characteristics help determine the most analgesic value while mitigating epidural opioid related side effects. Examples of useful epidural opioids include fentanyl, hydromorphone and sufentanil while morphine would be too hydrophilic (leading to rostral spread and increasing the risk of respiratory depression) and others like alfentanil would be too short acting. Potential dose ranges are fentanyl 2–5 mcg/ml and hydromorphone 5–20 mcg/ml with an example beneficial dose per hour range being fentanyl 20–30 mcg/hr. (see Table 31.2) [29, 31]. Epidural infusions may be administered continuously, and may benefit from a patient controlled bolus as well as intermittent automated or patient controlled bolus [24, 36, 37]. At our institution, we favor initiation of an epidural infusion of bupivacaine 0.0625% with fentanyl 2 mcg/ml running at 6–8 ml/hr. and with a 2 ml bolus and 20 minute lockout for surgeries such as T10-pelvis fusion in adults for scoliosis correction. Subsequent titration occurs based on patient needs. This relatively dilute solution is favored to mitigate primarily against the local anesthesia associated sympathectomy and motor blockade and is used in conjunction with multimodal systemic analgesia. Last, other medications such as alpha-2 adrenergic agonists or epinephrine, may be used in epidural infusions but the risk-benefit balance generally weights against their usage, and we do not recommend them [31, 38].

Like the heterogeneity in epidural infusion solutions that can be utilized, a variety of methods and equipment may be utilized as well. An epidural catheter may be placed pre-surgery percutaneously when an anterior surgical approach is being utilized or it may be placed directly by the surgeon prior to surgical closure and following surgical instrumentation or scoliosis correction [23, 24]. A multi-orifice 19 or 20 gauge flexible spring wound epidural catheter is typically utilized and then hooked up to a programmable electronic pump. For extensive multilevel instrumentation and fusion, such as occurs in major scoliosis correction of 10 or more levels, where a single epidural catheter optimally placed may not provide sufficient spread, a double epidural catheter technique may be utilized. An example approach is placing the upper catheter at the cranial end of the wound and directing it 4–5 cm cephalad to T1-4 while placing the lower catheter at the caudal end of the wound and directing it 4–5 cm cephalad to L1-4 or more simply the upper catheter at the cranial end of the instrumentation and the lower catheter at the caudal end of the instrumentation and directing each 3 cm cephalad [18, 21].

Integration of Perioperative Epidural Analgesia into Enhanced Recovery Protocols

The goal of an Enhanced Recovery Protocol (ERP) is to hasten high quality functional recovery after surgery through a coordinated multidisciplinary approach. This leads to improved early functional ability that results in shorter hospital stays, reduced hospital costs and improved quality of life. The concept has its origins in the work of Danish surgeon Henrik Kehlet over 20 years ago but has only recently been applied to spine surgery [5, 7, 8, 39, 40]. Common key features of ERPs include intensive pain management, attenuation of surgical stress, and early mobilization. Perioperative epidural analgesia directly addresses the first two components and facilitates the third, and it has been successfully employed in many published protocols to help achieve these goals [5, 7, 8, 39, 41].

Nonetheless, perioperative epidural analgesia is a resource heavy, labor intensive addition to post-operative analgesia for major spine surgery. It requires regular monitoring by nurses and an inpatient consult acute pain team, typically led by an anesthesiologist or pain management specialist, to manage it. Frequent adjustments may occur in the first 24 hours and then as needed thereafter. To help it be a successful component of an ERP a number of factors should be addressed: a protocol for placement and initiation of the epidural infusion, good communication among the surgeon, acute pain team, nurses and therapists to address any issues that may arise, frequent assessment when initiating the infusion to provide as much analgesia as possible without causing significant side effects, low concentration and dose of local anesthetic in the immediate post-operative period to permit neurologic assessment, mitigate sympathectomy related hypotension and facilitate early mobilization, and coordination of appropriate prophylactic anticoagulation for the situation. List 31.1 summarizes surgical procedures using continuous epidural analgesia for post- operative pain management.

List 31.1 Continuous post-operative epidural analgesia is indicated for the following surgeries:

- Adult and pediatric scoliosis correction surgery of the thoracic and lumbar spine
- Single or multilevel open posterior lumbar decompression and interbody fusion
- Open transforaminal lumbar interbody fusion

References

1. Cho JH, Lee JH, Song KS, Hong JY. Neuropathic pain after spinal surgery. Asian Spine J. 2017;11(4):642–52.
2. Woolf CJ. Pain: moving from symptom control toward mechanism-specific pharmacologic management. Ann Intern Med. 2004;140(6):441–51.
3. Gerbershagen HJ, Aduckathil S, van Wijck AJ, Peelen LM, Kalkman CJ, Meissner W. Pain intensity on the first day after surgery: a prospective cohort study comparing 179 surgical procedures. Anesthesiology. 2013;118(4):934–44.

4. Machi AT. Perioperative pain management. In: Noe CE, editor. Pain management for clinicians. Springer; 2020. p. 31–72.
5. Dietz N, Sharma M, Adams S, Alhourani A, Ugiliweneza B, Wang D, et al. Enhanced recovery after surgery (ERAS) for spine surgery: a systematic review. World Neurosurg. 2019;130:415–26.
6. Kim SI, Ha KY, Oh IS. Preemptive multimodal analgesia for postoperative pain management after lumbar fusion surgery: a randomized controlled trial. Eur Spine J. 2016;25(5):1614–9.
7. Mathiesen O, Dahl B, Thomsen BA, Kitter B, Sonne N, Dahl JB, et al. A comprehensive multimodal pain treatment reduces opioid consumption after multilevel spine surgery. Eur Spine J. 2013;22(9):2089–96.
8. Devin CJ, McGirt MJ. Best evidence in multimodal pain management in spine surgery and means of assessing postoperative pain and functional outcomes. J Clin Neurosci. 2015;22(6):930–8.
9. Borgeat A, Blumenthal S. Postoperative pain management following scoliosis surgery. Curr Opin Anaesthesiol. 2008;21(3):313–6.
10. Tobias JD. A review of intrathecal and epidural analgesia after spinal surgery in children. Anesth Analg. 2004;98(4):956–65, table of contents.
11. Popping DM, Elia N, Van Aken HK, Marret E, Schug SA, Kranke P, et al. Impact of epidural analgesia on mortality and morbidity after surgery: systematic review and meta-analysis of randomized controlled trials. Ann Surg. 2014;259(6):1056–67.
12. Block BM, Liu SS, Rowlingson AJ, Cowan AR, Cowan JA Jr, Wu CL. Efficacy of postoperative epidural analgesia: a meta-analysis. JAMA. 2003;290(18):2455–63.
13. Shaw R, Skovrlj B, Cho SK. Association between age and complications in adult scoliosis surgery: an analysis of the scoliosis research society morbidity and mortality database. Spine. 2016;41(6):508–14.
14. Arshi A, Park HY, Blumstein GW, Wang C, Buser Z, Wang JC, et al. Outpatient posterior lumbar fusion: a population-based analysis of trends and complication rates. Spine. 2018;43(22):1559–65.
15. Sharma S, Balireddy RK, Vorenkamp KE, Durieux ME. Beyond opioid patient-controlled analgesia: a systematic review of analgesia after major spine surgery. Reg Anesth Pain Med. 2012;37(1):79–98.
16. Schenk MR, Putzier M, Kügler B, Tohtz S, Voigt K, Schink T, et al. Postoperative analgesia after major spine surgery: patient-controlled epidural analgesia versus patient-controlled intravenous analgesia. Anesth Analg. 2006;103(5):1311–7.
17. Wenk M, Liljenqvist U, Kaulingfrecks T, Gurlit S, Ermert T, Pöpping DM, et al. Intra- versus postoperative initiation of pain control via a thoracic epidural catheter for lumbar spinal fusion surgery. Minerva Anestesiol. 2018;84(7):796–802.
18. Blumenthal S, Borgeat A, Nadig M, Min K. Postoperative analgesia after anterior correction of thoracic scoliosis: a prospective randomized study comparing continuous double epidural catheter technique with intravenous morphine. Spine. 2006;31(15):1646–51.
19. Gottschalk A, Freitag M, Tank S, Burmeister MA, Kreil S, Kothe R, et al. Quality of postoperative pain using an intraoperatively placed epidural catheter after major lumbar spinal surgery. Anesthesiology. 2004;101(1):175–80.
20. Park SY, An HS, Lee SH, Suh SW, Kim JL, Yoon SJ. A prospective randomized comparative study of postoperative pain control using an epidural catheter in patients undergoing posterior lumbar interbody fusion. Eur Spine J. 2016;25(5):1601–7.
21. Blumenthal S, Min K, Nadig M, Borgeat A. Double epidural catheter with ropivacaine versus intravenous morphine: a comparison for postoperative analgesia after scoliosis correction surgery. Anesthesiology. 2005;102(1):175–80.
22. Toktaş ZO, Konakçı M, Yılmaz B, Ekşi M, Aksoy T, Yener Y, et al. Pain control following posterior spine fusion: patient-controlled continuous epidural catheter infusion method yields better post-operative analgesia control compared to intravenous patient controlled analgesia method. A retrospective case series. Eur Spine J. 2016;25(5):1608–13.

23. Ezhevskaya AA, Mlyavykh SG, Anderson DG. Effects of continuous epidural anesthesia and postoperative epidural analgesia on pain management and stress response in patients undergoing major spinal surgery. Spine. 2013;38(15):1324–30.
24. Wenk M, Ertmer C, Weber TP, Liljenqvist UR, Pöpping DM, Möllmann M, et al. Feasibility and efficacy of preoperative epidural catheter placement for anterior scoliosis surgery. Anesthesiology. 2010;113(2):353–9.
25. Saito W, Inoue G, Imura T, Takenami T, Ueno M, Nakazawa T, et al. Safety and efficacy of continuous epidural anesthesia following scoliosis surgery in respiratory-impaired neuromuscular children: a pilot study. Spine Deform. 2015;3(3):272–6.
26. Tran DQ, Van Zundert TC, Aliste J, Engsusophon P, Finlayson RJ. Primary failure of thoracic epidural analgesia in training centers: the invisible elephant? Reg Anesth Pain Med. 2016;41(3):309–13.
27. Hermanides J, Hollmann MW, Stevens MF, Lirk P. Failed epidural: causes and management. Br J Anaesth. 2012;109(2):144–54.
28. von Hösslin T, Imboden P, Lüthi A, Rozanski MJ, Schnider TW, Filipovic M. Adverse events of postoperative thoracic epidural analgesia: a retrospective analysis of 7273 cases in a tertiary care teaching hospital. Eur J Anaesthesiol. 2016;33(10):708–14.
29. Manion SC, Brennan TJ. Thoracic epidural analgesia and acute pain management. Anesthesiology. 2011;115(1):181–8.
30. Rosero EB, Joshi GP. Nationwide incidence of serious complications of epidural analgesia in the United States. Acta Anaesthesiol Scand. 2016;60(6):810–20.
31. Curatolo M, Schnider TW, Petersen-Felix S, Weiss S, Signer C, Scaramozzino P, et al. A direct search procedure to optimize combinations of epidural bupivacaine, fentanyl, and clonidine for postoperative analgesia. Anesthesiology. 2000;92(2):325–37.
32. Waxler B, Dadabhoy Zerin P, Stojiljkovic L, Rabito Sara F, Warltier DC. Primer of postoperative pruritus for anesthesiologists. Anesthesiology. 2005;103(1):168–78.
33. Gehling M, Tryba M. Risks and side-effects of intrathecal morphine combined with spinal anaesthesia: a meta-analysis. Anaesthesia. 2009;64(6):643–51.
34. Martin BI, Mirza SK, Spina N, Spiker WR, Lawrence B, Brodke DS. Trends in lumbar fusion procedure rates and associated hospital costs for degenerative spinal diseases in the United States, 2004 to 2015. Spine. 2019;44(5):369–76.
35. Theologis AA, Sing DC, Chekeni F, Diab M. National trends in the surgical management of adolescent idiopathic scoliosis: analysis of a national estimate of 60,108 children from the national inpatient sample over a 13-year time period in the United States. Spine Deform. 2017;5(1):56–65.
36. Erdogan MA, Ozgul U, Ucar M, Korkmaz MF, Aydogan MS, Ozkan AS, et al. Patient-controlled intermittent epidural bolus versus epidural infusion for posterior spinal fusion after adolescent idiopathic scoliosis: prospective, randomized, double-blinded study. Spine. 2017;42(12):882–6.
37. Wong CA, McCarthy RJ, Hewlett B. The effect of manipulation of the programmed intermittent bolus time interval and injection volume on total drug use for labor epidural analgesia: a randomized controlled trial. Anesth Analg. 2011;112(4):904–11.
38. Tschopp C, Tramèr MR, Schneider A, Zaarour M, Elia N. Benefit and harm of adding epinephrine to a local anesthetic for neuraxial and locoregional anesthesia: a meta-analysis of randomized controlled trials with trial sequential analyses. Anesth Analg. 2018;127(1):228–39.
39. Wainwright TW, Immins T, Middleton RG. Enhanced recovery after surgery (ERAS) and its applicability for major spine surgery. Best Pract Res Clin Anaesthesiol. 2016;30(1):91–102.
40. Kehlet H. Multimodal approach to control postoperative pathophysiology and rehabilitation. Br J Anaesth. 1997;78(5):606–17.
41. Chakravarthy VB, Yokoi H, Coughlin DJ, Manlapaz MR, Krishnaney AA. Development and implementation of a comprehensive spine surgery enhanced recovery after surgery protocol: the Cleveland Clinic experience. Neurosurg Focus. 2019;46(4):E11.

Telemedicine Physical Examination for Spine

32

Daltry Dott, Jason Zafereo, Stephanie Jones, and Carl E. Noe

Introduction

The advent of telemedicine dates back to the 1950s or 1960s. Since that time, the utilization of telemedicine has increased and the capabilities are rapidly expanding. Now, the novel coronavirus (COVID-19) pandemic has catapulted telemedicine into mainstream medicine. Many aspects of a clinic visit, including chief complaint, past medical history, and review of systems can often be obtained directly from the patient. Electronic medical records contain previously completed medical testing including imaging and laboratory and pathology records. Electronic prescription records and monitoring programs can be used to identify most of the high risk prescription drugs patients may be taking. However, performance of a physical examination is potentially limited with a telemedicine visit.

To provide the highest quality of care, documenting a physical examination is important in order to meet the standard of care [1]. While the time-based evaluation and management coding is encouraged for telemedicine, several pitfalls exist for this practice. Using time-based coding may result in a lack of performance of important examinations that can be at least attempted using telemedicine. If attempts at examination are unsatisfactory, this can be documented just as it is for an in-person examination. In many states, documentation of a problem-focused exam specific to the presenting chief complaint of the patient is a minimum requirement

D. Dott (✉) · S. Jones · C. E. Noe
Department of Anesthesiology and Pain Management, University of Texas Southwestern Medical Center, Dallas, TX, USA
e-mail: Daltry.Dott@UTSouthwestern.edu; Stephanie.Jones@UTSouthwestern.edu; Carl.Noe@UTSouthwestern.edu

J. Zafereo
Department of Physical Therapy, University of Texas Southwestern Medical Center, Dallas, TX, USA
e-mail: Jason.Zafereo@UTSouthwestern.edu

© The Author(s), under exclusive license to Springer Nature Switzerland AG 2022
C. E. Noe (ed.), *Multidisciplinary Spine Care*,
https://doi.org/10.1007/978-3-031-04990-3_32

for prescribing controlled substance for the treatment or chronic pain. Also, The Federation of State Medical Boards Guidelines for the Chronic Use of Opioid Analgesics requires documentation of a relevant physical examination [2]. In addition, the Drug Enforcement Agency (DEA) requires a real time, two-way audiovisual telemedicine visit for initiating opioid treatment to allow for completion and documentation of a physical exam.

To effectively perform a physical exam via telemedicine, providers and patients may need to employ some ingenuity. Patients may need to be instructed on camera adjustments or may need someone to assist in camera positioning so that the area being examined can be seen via two-way video systems. Care should be taken in patients who may be at risk for falls, and these patients should be chaperoned and should have assistance when performing any maneuvers that may put them at risk of falling. Even with the limitations that telemedicine may pose for the completion of a physical exam, it can often be completed efficiently. This chapter will summarize evidence- based physical examination techniques that can be performed using telemedicine to support diagnosis and treatment of commonly presenting problems in the field of pain management.

Vital Signs

Respiratory rate may be measured using telemedicine and is especially relevant for patients who are taking opioids or other respiratory depressants. Pulse may be counted by the patient and timed by the physician. Patients may also have health monitoring accessories that have heart rate monitoring capabilities which can be reviewed by the physician. Blood pressure, temperature, height and weight can be measured if the patient has the necessary equipment at home or if patients have access to a local pharmacy that can perform blood pressure readings at a nearby location.

A randomized trial has shown that hypertension can be controlled just as well using remote blood pressure monitoring [3].

General Appearance

Visualization of the patient via a two-way audio visual system allows for evaluation of the patient's general appearance and grooming. Distress can be assessed directly by watching the patient and any facial expressions that may change with movement or different parts of the exam. Patient's mental status, level of alertness, and orientation can be assessed through visualization of the patient through the video component as well as by asking specific orientation questions. Speech and any unusual facial movements or speech patterns, including slurred speech, word-finding difficulties, and mood can be assessed. Tremors and other abnormal movements can be generally observed. Dementia and mild cognitive impairment can be screened for with the Rapid Cognitive Screen [4].

Table 32.1 Psychiatric status screen

Examination	Tests
Perception and cognition	Level of consciousness
	Orientation to person, place and time
	Recent and remote memory
	Attention span and concentration
	Language, naming objects, repeating phrases
Thought content and process	Fund of Knowledge—current events, past history, vocabulary
	Associations-loose, tangential, circumstantial
	Speech-rate, volume, articulation, coherence, spontaneity, perseveration, paucity of language
	Thought-rate of thoughts, contest of thoughts, logical vs illogical, tangential, abstract reasoning, computation
	Judgement concerning everyday situations
Affect and insight	Mood and affect—suicidal ideation, anxiety, depression, sleep pattern
	Hallucinations, delusions, preoccupation, obsessions
	Insight into psychological component of pain
Drug-seeking behavior	Congruous patient report of prescription or illicit drug use compared to electronic prescription records and laboratory findings
	Behavior consistent with self-escalation of drug doses, doctor shopping, substance use disorder, overmedication or diversion
	Adherence to laws, rules and medication agreements regarding the prescribing of scheduled drugs

Source: https://www.cms.gov/Outreach-and-Education/Medicare-Learning-Network-MLN/MLNEdWebGuide/Downloads/95Docguidelines.pdf

Table 32.2 Opioid withdrawal screen

Examination	Sign
Vital signs	Tachycardia
Skin inspection	Diaphoresis
	Piloerection
Facial inspection	Midriasis
	Rhinorrhea
	Yawning
Neuropsychological	Tremor
	Anxiety or irritability
	Restlessness

Source: Wesson and Ling [5]

Additional examinations to assess psychiatric status are listed in Table 32.1. Opioid withdrawal signs that can be evaluated via telemedicine are listed in Table 32.2 [5].

Inspection of Painful Area

Patients can expose their painful area for inspection, which can help to identify any rash, discoloration, swelling, deformity, atrophy, abnormal muscle activity or motion, or wounds. Patients can also directly identify their painful area by pointing. This is important to identify the specific area being discussed as patents often use terms to describe and localize pain that are not consistent. For example, a complaint

of “hip pain” is frequently localized to the upper buttock. “Neck pain” may localize to the trapezius area. “Low back pain” may localize to the thoracolumbar, lumbar, or sacral area. “Shoulder pain” may be anterior, lateral, or posterior and each area has a potentially different differential diagnosis. Inspection of the arms and legs for needle track marks or skin popping is possible using telemedicine to detect signs of intravenous drug abuse.

However, telemedicine has limitations and patients who are determined to need an in person evaluation should be scheduled for a timely in person follow up visit or advised to go to the emergency room based on their complaints [6].

Range of Motion

If pain is lateralized, range of motion (ROM) of the symptomatic side can be compared to the asymptomatic side by asking the patient to perform specific maneuvers, such as abduction, adduction, internal and external rotation, and other movements. ROM may be limited by pain, weakness, or tightness. If weakness or muscular pain limits movement, active ROM may be significantly more limited than passive ROM. Active and passive ROM will be limited similarly if tightness limits movement or if joint pain limits movement [7].

Cranial Nerve (CN) Exam

Although a full evaluation of the cranial nerves is limited using telemedicine, several examinations are possible and can be documented:

CNI: typically, is not examined during a physical exam, in person or telemedicine

CNII: visual acuity and visual field testing can be performed through telemedicine using an online chart or web-based system

CNIII-IV, VI: Eye movements ban be assessed by tasking the patient to move their eyes into each of the four quadrants typically tested in person. Pupillary size and accommodation can be examined by asking the patient to move close to the camera to visualize the pupils. The patient is asked to close their eyes so the pupils will dilate. Then the patient is asked to open their eyes and the pupils will normally accommodate to room light.

CNV: Sensation in the distributions of the three branches of the trigeminal nerve can be evaluated aby asking the patient to stroke their skin with a tissue in each dermatome.

CNVII: Facial neuromuscular exam can be performed by asking patients to elevate their eyebrows, smile and frown.

CNVIII: Hearing can be evaluated by asking the patient to rub their fingers gently near their ears.

CNIX: Patients can be asked to open their mouth wide and elevate their palate by saying “ahh”

CNX: Patients can test their own gag reflex

CNXI: Patients can be asked to shrug their shoulders
CNXII: Patients can be asked to protrude their tongue to examine for any atrophy or abnormal movements.

Sensorimotor Exam

Sensation, including allodynia, hyperalgesia and normal sensation can be assessed by having the patient stroke the skin with a tissue at the painful area. This can be repeated on the contralateral side for comparison.

Upper extremity strength can be evaluated by asking patients to hold weighted objects in the hand such as a bottle of water or a gallon jug. Shoulder elevation (deltoid) strength can be assessed by asking patients to repeatedly lift a gallon jug and then comparing scores to age-referenced normative values [8].

Asymmetry during strength testing may indicate weakness. Triceps strength can be evaluated by asking patients to hold the objects with the shoulders abducted and internally rotated. Then the patient is instructed to flex the elbow to 90 degrees to move the object toward the floor and then extend the elbow to straighten the arm. Biceps strength can be assessed by flexing the elbow with the arm resting at the side. Normative values exist for men (8lbs) and women (5lbs) on the biceps curl test according to age ranges [9]. See Table 32.3 for a summary of the age-based normative values for upper extremity strength testing.

Table 32.3 Upper extremity strength testing

Gallon jug lift test			
Age in years	Number of repetitions in 30 seconds		
	Below average	Average	Above average
Male			
19–35	≤24	25–30	≥31
36–65	≤22	23–27	≥28
65+	≤13	14–21	≥22
Female			
19–35	≤19	20–24	≥25
36–65	≤15	16–18	≥19
65+	≤7	8–14	≥15
Arm curl test			
Age in years	Number of repetitions in 30 seconds		
	Poor	Average	Excellent
Male (8lbs)			
60–64	≤15	18–20	≥23
65–69	≤13	18–19	≥23
70–74	≤13	17–18	≥22
75–79	≤12	16–17	≥21
80–84	≤12	15–17	≥20
Female (5lbs)			
60–64	≤12	15–17	≥20
65–69	≤11	15–16	≥19
70–74	≤10	14–15	≥19
75–79	≤10	13–15	≥18
80–84	≤9	12–14	≥17

Table 32.4 Lower extremity strength testing

5-time Sit-to-stand test			
Age in years	Time in seconds to complete 5 repetitions		
	Poor	Average	Excellent
19–29	≤7.2	6.3–5.7	≤4.8
30–39	≤7.3	6.4–5.8	≤4.9
40–49	≤9.2	8.0–7.2	≤6
50–59	≤9.9	7.1–8.3	≤5.5
60–69	≤9.9	7.2–8.4	≤5.7
70–79	≤11.1	8.8–9.8	≤7.5
80+	≤13.0	10.2–11.4	≤8.6
Single leg heel raise test			
Age in years	Maximum number of full repetitions completed		
	Poor	Average	Excellent
Male			
19–29	≤33	37–38	≥42
30–39	≤29	32–34	≥37
40–49	≤24	28–29	≥33
50–59	≤20	23–25	≥28
60–69	≤16	19–20	≥23
70–79	≤16	19–20	≥23
80+	≤7	10–11	≥14
Female			
19–29	≤26	30–31	≥35
30–39	≤24	27–29	≥32
40–49	≤21	25–26	≥29
50–59	≤19	22–23	≥27
60–69	≤16	19–20	≥24
70–79	≤16	19–20	≥24
80+	≤11	14–15	≥18

Heel and toe walking can evaluate L4 and S1 myotome strength, respectively. Normative values are reported by age for the heel raise test [10].

A 5-time Sit-to-Stand test can be performed to provide an age-referenced assessment of lower extremity strength and function [11]. See Table 32.4 for a summary of the age-based normative values for lower extremity strength testing.

Coordination

Coordination can be evaluated using rapid alternating movements, heel shin, and finger-nose testing. For finger-nose testing, patients may alternate touching different points of their computer or tablet or points in space instead of touching an examiner's finger. A tap test may also be performed on a computer keyboard using the index finger and compared to age-referenced normative values [12–14]. See Table 32.5 for a summary of the age-based normative values for upper extremity coordination testing.

Table 32.5 Upper extremity coordination testing

Computer tap test: dominant side			
Age in years	Max number of spacebar taps in 10 seconds		
	Poor	Average	Excellent
Male			
25–39	≤55	59–61	≥66
40–54	≤49	55–58	≥65
55–64	≤41	46–48	≥53
65–74	≤39	44–46	≥52
Female			
25–39	≤50	56–59	≥64
40–54	≤49	54–55	≥59
55–64	≤35	39–41	≥46
65–74	≤31	37–39	≥45
Computer tap test: non-dominant side			
Age in years	Max number of spacebar taps in 10 seconds		
	Poor	Average	Excellent
Male			
25–39	≤49	53–55	≥59
40–54	≤46	49–50	≥54
55–64	≤38	42–44	≥48
65–74	≤36	41–43	≥48
Female			
25–39	≤46	49–51	≥54
40–54	≤45	48–49	≥53
55–64	≤34	38–39	≥43
65–74	≤30	35–37	≥41

Gait and Station

The typical office assessment can be performed via telemedicine by observing multiple points during gait and station examination. Posture during ambulation may reveal kyphosis, lordosis, scoliosis or other postural changes. Facial examination may reveal anxiety, grimacing due to pain, Parkinsonian facies, or other important information.

Different types of gaits may indicate differing underlying pathologies. A Trendelenburg gait is often due to hip abductor weakness. An antalgic gait has a shortened support (stance) phase due to pain with weight bearing. A shuffling or festinating gait may be associated with Parkinson's disease. A steppage or equine gate is often associated with foot drop. A circumduction gait is common with hemiparesis. A waddling gait can be seen with myopathy. A wide stance may indicate a cerebellar gait. A diplegic cage can be seen in cerebral palsy. Tandem gate can be used to identify ataxia. A slow wide-based gait can be seen in spondylotic myelopathy. Such a gait disturbance may prompt further evaluation of cervical and/or thoracic pathology [15].

For those patients with stairs in their home, a test of ascending and descending a flight of 11 steps can be performed and compared to age-referenced normative values [11]. Additionally, the Timed Up and Go (TUG) test, which has established

Table 32.6 Functional gait testing

Timed stair test			
Age in years	Time in seconds to ascend/descent 11 stairs		
	Poor	Average	Excellent
Male			
20–29	≥6.5	5.3–5.7	≤4.5
30–39	≥7.2	5.8–6.4	≤5.0
40–49	≥7.0	5.6–6.2	≤4.8
50–59	≥8.5	6.3–7.3	≤5.1
60–69	≥10.8	8.3–9.3	≤6.8
70–79	≥11.8	9.0–10.2	≤7.4
Female			
20–29	≥7.8	6.7–7.1	≤6
30–39	≥8.4	6.6–7.4	≤5.6
40–49	≥8.6	7.0–7.6	≤6
50–59	≥10.0	8.0–8.8	≤6.8
60–69	≥12.2	9.5–10.7	≤8
70–79	≥14.5	11.0–12.6	≤9.1
Timed up and go test			
Age in years	Time in seconds to complete 1 trial		
	Below average	Average	Above average
20–39	≥7.3	5.9–7.4	≤5.8
40–59	≥7.9	6.3–7.8	≤6.2
60–69	≥9.1	7.1–9.0	≤7.0
70–79	≥10.3	8.2–10.2	≤8.1
80–99	≥12.8	10.0–12.7	≤9.9

predictive and normative values, can easily performed in the home with a chair and a 3-meter walkway [16, 17]. See Table 32.6 for a summary of the age-based normative values for functional gait testing.

Finally, in patients with suspected balance deficits, a test of single limb balance can be performed and compared to normative values [18, 19]. Patients under age 50 should be able to stand on one limb for at least 60 seconds, whereas those in their 50s can typically balance for 50 seconds. Balance capacity drops about 10 seconds for every additional 5 years of life beginning at age 60 (e.g., patients 60–65 can balance for 40 seconds, 66–70 for 30 seconds, etc.). Patients should be tested near a wall or furniture to prevent a fall during testing [20].

Tests for Specific Conditions

Low Cerebrospinal Fluid (CSF) Pressure Headache

The Valsalva maneuver is performed by having the patient bear down against a closed glottis to increase the intraabdominal pressure. This maneuver may improve a low cerebrospinal fluid pressure headache. Muller Maneuver is performed by having a patient attempt to inhale against a closed glottis. This may produce the opposite response of a Valsalva maneuver and may worsen a low CSF headache.

Temporomandibular Joint Dysfunction (TMD)

By asking a patient to open their mouth as wide as possible, oral opening can be assessed. The normal opening between the incisal edges of the maxillary and mandibular incisors is 35-55 mm, or the width of 3 fingers. Reduced opening is criterion for TMD.

Cervical Myelopathy

Lhermitte's sign may be performed by asking the patient to flex the cervical spine and reporting any pain down the spine or in the extremities [21].

Wartenberg's sign is positive when the fifth finger abducts spontaneously when the fingers are extended [22].

Discoordination with a lower extremity foot tapping test may also suggest the presence of cervical myelopathy if scores fall significantly outside the age-reference normative values [23].

Cervical Instability

Rust sign or lifting the head manually is a sign of possible cervical instability.

Cervical Pain

The cervical compression test if performed by asking the patient to move their chin to the shoulder and then extend the neck. Pain on the concave side suggests nerve root or facet pain. Pain on the convex side suggests musculoskeletal pain, particularly if the pain is relieved with contralateral scapular elevation.

Brachial Plexopathy

The test for Bikele's sign is performed by asking a seated patient to abduct the arm at the shoulder to 90 degrees and flex the elbow. Then the patient extends the elbow. Reproduction of pain is a positive sign for brachial plexus lesion, nerve root tension or meningitis.

Cervical Radiculitis

The shoulder abduction test (Bakody's Sign) is performed by asking the patient to place the hand of their painful arm on top of their head. Relief is a sign of cervical radiculitis [24, 25].

Thoracic Outlet Syndrome

The Roos test or elevated arm stress test (EAST) is a test for thoracic outlet syndrome. The patient is seated and holds both arms with the shoulders in 90 degrees abduction and external rotation with the shoulders and elbows in the frontal plane. The fists are opened and closed for 3 minutes. Reproduction of symptom suggests thoracic outlet syndrome [26].

Shoulder Pain

The Apley inferior and superior scratch test if performed by asking the patient to scratch their shoulder blade with the contralateral hand from superiorly and inferiorly. This range of motion is limited in patients with rotator cuff problems, labral tears and adhesive capsulitis [27].

Rotator Cuff

The drop arm test is used to evaluate patients for rotator cuff tears. The patient abducts the shoulder and slowly lowers the arm to the waist. Patients with rotator cuff tears or supraspinatus weakness will be unable to slowly drop the arm and the arm will fall [28].

The empty can test can also be used to identify rotator cuff pathology. The patient is asked to hold the affected shoulder at 90 degrees abduction with the hand extended to the front and the patient is asked to internally rotate the hand like they would if they were trying to empty a can. Reproduction of pain can indicate rotator cuff pathology.

Subscapularis Muscle Tear

The Lift off test is an evaluation for subscapularis tears. The hand is placed behind the back and the dorsum of the hand is lifted off the back. Weakness of this motion indicated subscapularis weakness [29].

The belly press test (Napoleon test) is performed by the patient pressing their palm into the epigastric area while maintain the elbow and wrist in the horizontal plane. This isolates the subscapularis muscle function to internally rotate the shoulder [30].

Subacromial Impingement

The painful ARC test is performed by the patient abducting the shoulder to 180 degrees. Pain between 60 and 120 degrees is a positive test for sub-acromial impingement pain [31].

Acromioclavicular Joint Pathology

The cross arm test is performed by the patient lifting the arm to 90 degrees and adducting the arm across the body. Pain in the acromioclavicular joint is a positive sign [27].

Cubital Tunnel Syndrome

The elbow flexion test if performed by the patient flexing the elbow as much as possible for 3 minutes. Reproduction of pain and numbness in an ulnar distribution is a positive test [32].

Lateral Epicondylitis

The wrist drop test if performed by the patient positioning the hands in the praying position with the hands together and the wrists extended. The patient separates the hands and holds the wrist and hands in the same positions for 1 minute. A positive test is an inability to hold the wrist in extension due to epicondylitis or radial nerve dysfunction [33].

Carpal Tunnel Syndrome

Tinel's sign can be tested by asking the patient to tap over the median nerve at the wrist to evaluate carpal tunnel syndrome.

Thenar atrophy is a sign of late or severe carpal tunnel syndrome. The recurrent branch of the ulnar nerve innervates the opponens pollicus, abductor brevis, flexor pollicis brevis [34].

The hand evaluation test is performed by the patient lifting the hands for 2 minutes. If symptom reproduction occurs within 2 minutes, the test is positive [35].

The closed fist test (Berger test) is performed by the patient making a fist and holding for 30–60 seconds. Reproduction of symptoms is a positive test [36].

The Phalen's test is performed by the patient flexing the wrists and holding the dorsal surfaces of both hands together for 30–60 seconds. Reproduction of symptoms indicates a positive test.

The reverse Phalen's test is performed by the patient holding their hand in the prayer position with the wrists and fingers fully extended. Reproduction of symptoms indicates a positive test.

The wringing test if performed by the patient wringing a towel with both hands. Paresthesias in the hand indicate a positive test.

Neuropathy

The shrivel test (O'rianin sign or Leukens' test) is performed by having the patient place their fingers in warm water for 30 minutes. Denervated fingertip skin will not shrivel.

Thoracic Spine Pathology

Thoracic range of motion may be evaluated by having the patient bend from side to side. Forestier's bowstring sign is when there is asymmetrical motion with lateral bending which may indicate paraspinous muscle spasm, ankylosing spondylitis or other painful spinal condition.

First Thoracic Nerve Root Pain

The first thoracic nerve root test is performed by the patient abducting the shoulder to 90 degrees, flexing the elbow to 90 degrees, and pronating the forearm to 90 degrees from neutral. Then, the hand is placed behind the neck. The occurrence of scapular pain indicates a positive sign of T1-T2 nerve root compression.

Lumbar Spine Instability

The spinal instability catch sign test is performed by the patient bending forward as much as possible and then attempting to return to the upright position. An inability to return to the upright position is a positive test [37].

The stork test is performed by the patient standing on one foot with hands on the hips and positioning the other foot against the knee of the standing leg. The average time of balancing in this posture is 25–39 seconds. The stork test is positive for spinal instability if pain is aggravated from this position. The stork test is also used to evaluate balance and sacroiliac pain. The test for balance terminates if the hands are removed from the hips, the standing leg moves, or if the foot is removed from the knee. The flamingo test is similar but the patient stands on a board [38].

General trunk weakness would be suspected in patients with underlying spinal instability. The dynamic or static 1/4 Sit-Up test, provides age-referenced normative values for abdominal strength [39].

Similarly, the dynamic or static Chest Raise test provides age-referenced normative values for back extensor strength [39].

Lumbar Radiculitis

Radicular pain may be worsened with the Valsalva maneuver, coughing, sneezing or straining.

Straight leg raising (Lasegue test) is performed by the examiner raising the patient's leg; however, one study has reported a high correlation between active straight leg raising and passive straight leg raising [40].

Bragad's test can be used to increase the straight raise leg test sensitivity. To do this the leg is lowered below the level that induces the radicular pain and the foot is dorsiflexed. Increase in radicular pain with foot dorsiflexion is considered a positive test. Another way to increase sensitivity of the straight leg raise is to have the patient flex the knee, which will typically improve pain. The seated straight leg raise is less sensitive compared to the supine straight leg raise test [41].

The cross straight leg raising is performed by lifting the asymptomatic leg. Reproduction of pain is a positive sign.

The reverse leg raising is performed by lifting the leg with the patient in the prone position. This is a test to detect upper lumbar nerve root pathology.

The Slump test is performed with the patient seated with the hands behind the back. The patient slumps forward with the thoracolumbar back. If this is not painful, the patient flexes the neck and extends one knee. If the knee extension causes pain, the neck is extended to the neutral position. If pain persists, a positive sign is considered. If knee extension does not cause pain, the ankle is dorsiflexed. If pain is reproduced, the test is positive.

Neri's sign (Neri's bowstring sign) test is performed standing. The patient bends forward to touch the toes. If the patient flexes the knees the test if positive.

Lumbar Spondylosis

The extension quadrant test (facet loading) is performed by the patient extending the lumbar spine and rotating to one side. Pain is associated with degenerative lumbar spine disease such a spondylosis [42].

Lumbar Stenosis

Patients with lumbar stenosis will often report pain associated with standing or walking and relief with sitting. Patients can be asked to stand and walk. Patients with spinal stenosis will often have symptomatic relief with leaning forward and walking. If a patient has pain with standing and leaning forward onto a chair or cart improves their pain, it may indicate lumbar spinal stenosis [43].

Lumbar extension may aggravate pain in patients with lumbar stenosis.

Compression Fracture

The supine test is performed by the patient reclining in the supine position with one pillow. Severe spine pain indicates a positive test [44].

Sacroiliac Pain

The Fortin finger test is performed by having the patient use one finger to localize their pain. A positive test is when the patient twice identifies the painful region as within 1 cm of inferomedial to the posterior superior iliac spine [45].

Hip Pain

Patients with hip pain may stand with the hip and knee slightly flexed on the affected side [46].

While sitting, patients may lean away for the affected side with less flexion in the affected hip.

The C sign is positive when the patient grabs the hip with the hand in a C shape and the thumb is posterior to the hip and the fingers are in the groin area.

Flexion, abduction, external rotation (FABER) or flexion, adduction, internal rotation (FADIR) testing may be useful for identifying signs of osteoarthritis or hip impingement, respectively [47].

These tests require an examiner but the positions can be reproduced by the patient with instruction to give some screening information about the hip.

The Thomas test if performed by the patient lying supine and flexing the asymptomatic hip with the knee to the chest. A positive test is indicated by limited in extension in the affected hip. Hip flexion contracture or psoas syndrome are possible diagnoses.

Gluteus Medius Tear

Trendelenburg's sign is a reliable exam for the gluteus medial muscle tears. The Trendelenburg test is performed by the patient standing on one leg. The pelvis dropping is a sign of weak hip abductor weakness and is a positive sign. During the gait of exam, the truck flexes toward the affected side [48].

Greater Trochanter Pain Syndrome

The single leg stance test is performed by the patient standing on one leg for 30 seconds. Pain in the hip of the standing leg is a positive test [49].

Piriformis Syndrome

The Beatty maneuver is positive when pain is reproduced in the buttock, not the lumbar spine, when the patient actively abducts the leg in a side lying position [50, 51].

Knee Meniscus Tear

The Ege test is performed by the patient standing with the feet 30–40 cm apart. The feet are internally rotated to test for lateral meniscus tears. The patient squats and stands. Pain or crepitus is considered a positive test. For testing the medical meniscus, the feet are externally rotated, and the same procedure is followed [52].

The Childress (Duck waddle, squat waddle) test is performed by the patient squatting and walking like a duck. Inability to perform the test, pain or crepitus is considered a positive test [53].

Patellofemoral Syndrome

The eccentric step test is performed by the patient stepping down from a raised platform with the hands on the hips. Reproduction of pain indicates a positive test [54].

Discussion

Telemedicine has been available for decades but its use has been limited due to concerns about improper technology or equipment, safety, reimbursement, HIPPA compliance, and other issues. Many of the barriers to the use of telemedicine have been terminated during the COVID-19 pandemic, and the use of telemedicine has increased substantially. With the substantial rise in the use of telemedicine and limited ability to see in-person clinic visits, it is vital that providers understand the limitations that may accompany telemedicine visits and adapt to work within these constraints. A common misconception is that the physical exam is unable to be performed via telemedicine visit. However, telemedicine has been used in multiple settings, including outpatient and inpatient, and has been shown to have similar safety and quality compared to in-person clinic visits in both new and follow up surgical and nonsurgical patients [55, 56]. General neurological telemedicine examinations have good interrater reliability in a study of emergency room patients [57].

We have demonstrated that telemedicine may be used to perform physical examinations in the field of pain management to evaluate and treat patients with a wide variety of painful conditions caused by spinal pathology or conditions that may mimic spine pathology. Patients who are inadequately evaluated using telemedicine may be scheduled for timely in-person evaluations or directed to the emergency department for further evaluation.

References

1. Centers for Medicare and Medicaid Services. 1995 Documentation guidelines for evaluation aEnd management services. https://www.cms.gov/Outreach-and-Education/Medicare-Learning-Network-MLN/MLNEdWebGuide/Downloads/95Docguidelines.pdf.

2. Federation of State Medical boards. Guidelines for the chronic use of opioid analgesics. 2017. https://www.fsmb.org/siteassets/advocacy/policies/opioid_guidelines_as_adopted_april-2017_final.pdf.
3. Madsen LB, Kirkegaard P, Pedersen EB. Blood pressure control during telemonitoring of home blood pressure. A randomized controlled trial during 6 months. Blood Press. 2008;17:78–86.
4. Malmstrom TK, Voss VB, Cruz-Oliver DM, Cummings-Vaughn LA, Tumosa N, Grossberg GT, Morley JE. The rapid cognitive screen (RCS): a point-of-cre screening for dementia and mild cognitive impairment. J Nutr Health Aging. 2015;19(7):741–4. https://doi.org/10.1007/s12603-015-0564-2.
5. Wesson DR, Ling W. The clinical opiate withdrawal scale (COWS). J Psychoactive Drugs. 2003;35(2):253–9. https://doi.org/10.1080/02791072.2003.10400007.
6. Weinstein RS, Krupinski EA, Doarn CR. Clinical examination component of telemedicine, telehealth, mHealth, and connected health medical practices. Med Clin North Am. 2018;102(3):533–44. https://doi.org/10.1016/j.mcna.2018.01.002.
7. Fritz JM, Delitto A, Erhard RE, Roman M. An examination of the selective tissue tension scheme, with evidence for the concept of a capsular pattern of the knee. Phys Ther. 1998;78(10):1046–56.
8. Shah KM, et al. Early development and reliability of the timed functional arm and shoulder test. J Orthop Sports Phys Ther. 2017;47(6):420–31. Epub 3 Mar 2017. https://doi.org/10.2519/jospt.2017.7136.
9. Rikli RE, Jones CJ. Functional fitness normative scores for community residing older adults, ages 60–94. J Aging Phys Act. 1999;7:162–81.
10. Hébert-Losier K, et al. Updated reliability and normative values for the standing heel-rise test in healthy adults. Physiotherapy. 2017;103(4):446–52.
11. Bohannon RW, et al. Sit-to-stand test: performance and determinants across the age-span. Isokinet Exerc Sci. 2010;18(4):235–40.
12. Christianson MK, Leathem JM. Development and standardisation of the computerised finger tapping test: comparison with other finger tapping instruments. N Z J Psychol. 2004;33(2):44–9.
13. Ashendorf L, Vanderslice-Barr JL, McCaffrey RJ. Motor tests and cognition in healthy older adults. Appl Neuropsychol. 2009;16(3):171–6.
14. Austin, et al. Measuring motor speed through typing: a surrogate for the finger tapping test. Behav Res Methods. 2011;43(4):903–9. https://doi.org/10.3758/s13428-011-0100-1.
15. Haddas R, Ju KL, Belanger T, et al. The use of gait analysis in the assessment of patients afflicted with spinal disorders. Eur Spine J. 2018;27:1712–23.
16. Brustio PR, Magistro D, Zecca M, Rabaglietti E, Liubicich ME. Age-related decrements in dual-task performance: Comparison of different mobility and cognitive tasks. A cross sectional study. PLoS ONE. 2017;12(7):e0181698.
17. Bohannon RW. Reference values for the timed up and go test: a descriptive meta-analysis. J Geriatr Phys Ther. 2006;29(2):64–8.
18. Springer B, Marin R, Cyhan T, et al. Normative values for the unipedal stance test with eyes open and closed. J Geriatr Phys Ther. 2007;30(1):8–15.
19. Kjær, et al. Normative values for musculoskeletal and neuromotor fitness in apparently healthy Norwegian adults and the association with obesity: a cross-sectional study. BMC Sports Sci Med Rehabil. 2016;8:37.
20. Van Gerpen JA. Romberg testing evaluates proprioception and balance. Semin Neurol. 2011;31(1):078–04.
21. Uchihara T, Furukawa T, Tsukagoshi H. Compression of brachial plexus as a diagnostic test of cervical cord lesion. Spine. 1994;19:2170–3.
22. Kiely P, Baker JF, O'hEireamhoin S, et al. The evaluation of the inverted supinator reflex in asymptomatic patients. Spine. 2010;35(9):955–7. https://doi.org/10.1097/BRS.0b013e3181bccf0f.
23. Numasawa, et al. Simple foot tapping test as a quantitative objective assessment of crvical myelopathy. Spine. 2012, 37;(2):108–13.
24. Rubinstein SM, Pool JJ, van Tulder MW, Riphagen II, de Vet HC. A systematic review of the diagnostic accuracy of provocative tests of the neck for diagnosing cervical radiculopathy. Eur Spine J. 2007;16(3):307–19. https://doi.org/10.1007/s00586-006-0225-6.

25. Viikari-Juntura E, Porras M, Laasonen EM. Validity of clinical tests in the diagnosis of root compression in cervical disc disease. Spine. 1989;14(3):253–7. https://doi.org/10.1097/00007632-198903000-00003.
26. Gillard J, Pérez-Cousin M, Hachulla É, Remy J, Hurtevent JF, Vinckier L, Thévenon A, Duquesnoy B. Diagnosing thoracic outlet syndrome: contribution of provocative tests, ultrasonography, electrophysiology, and helical computed tomography in 48 patients. Joint Bone Spine. 2001;68(5):416–24.
27. Woodward TW, Best TM. The painful shoulder: part I. Clinical evaluation. Am Fam Physician. 2000;61(10):3079–88.
28. Sgroi M, Loitsch T, Reichel H, Kappe T. Diagnostic value of clinical tests for supraspinatus tendon tears. Arthroscopy. 2018;34(8):2326–33.
29. Gerber C, Krushell RJ. Isolated rupture of the tendon of the subscapularis muscle. Clinical features in 16 cases. J Bone Joint Surg Br. 1991;73(3):389–94.
30. Barth JR, Burkhart SS, De Beer JF. The bear-hug test: a new and sensitive test for diagnosing a subscapularis tear. Arthroscopy. 2006;22(10):1076–84.
31. Park HB, Yokota A, Gill HS, El Rassi G, McFarland EG. Diagnostic accuracy of clinical tests for the different degrees of subacromial impingement syndrome. J Bone Joint Surg Am. 2005;87(7):1446–55.
32. Rosati M, Martignoni R, Spagnolli G, Nesti C, Lisanti M. Clinical validity of the elbow flexion test for the diagnosis of ulnar nerve compression at the cubital tunnel. Acta Orthop Belg. 1998;64(4):366–70.
33. Han BR, Cho YJ, Yang JS, et al. Clinical features of wrist drop caused by compressive radial neuropathy and its anatomical considerations. J Korean Neurosurg Soc. 2014;55(3):148–51.
34. D'Arcy C, McGee S. Does this patient have carpal tunnel syndrome? JAMA. 2000;283:3110–7.
35. Amirfeyz R, Gozzard C, Leslie IJ. Hand elevation test for assessment of carpal tunnel syndrome. J Hand Surg Br. 2005;30(4):3614.
36. Yii NW, Elliot D. A study of the dynamic relationship of the lumbrical muscles and the carpal tunnel. J Hand Surg Br. 1994;19(4):439–43.
37. Kasai Y, Morishita K, Kawakita E, Kondo T, Uchida A. A new evaluation method for lumbar spinal instability: passive lumbar extension test. Phys Ther. 2006;86(12):1661–7.
38. Panta K, Arulsingh W, Raj JO, Sinha M, Rahman M. A study to associate the Flamingo Test and the Stork Test in measuring static balance on healthy adults. Foot Ankle Online J. 2011;8(3):4.
39. McIntosh G, Wilson L, Affleck M, Hall H. Trunk and lower extremity muscle endurance: normative data. J Rehabil Outcomes Measur. 1998;2(4):20–39.
40. McIlroy S, et al. Correlation between the active and the passive straight leg raise test in adults with lumbar neural impingement. Results of a prospective clinical evaluation. Spine J. 17(3):S27.
41. Rabin A, Gerszten PC, Karausky P, Bunker CH, Potter DM, Welch WC. The sensitivity of the seated straight-leg raise test compared with the supine straight-leg raise test in patients presenting with magnetic resonance imaging evidence of lumbar nerve root compression. Arch Phys Med Rehabil. 2007;88(7):840–3.
42. Lyle MA, Manes S, McGuinness M, Ziaei S, Iversen MD. Relationship of physical examination findings and self-reported symptom severity and physical function in patients with degenerative lumbar conditions. Phys Ther. 2005;85(2):120–33.
43. Fritz JM, Erhard RE, Delitto A, et al. Preliminary results of the use of a two-stage treadmill test as a clinical diagnostic tool in the differential diagnosis of lumbar spinal stenosis. J Spinal Disord. 1997;10:410–6.
44. Langdon J, Way A, Heaton S, Bernard J, Molloy S. Vertebral compression fractures–new clinical signs to aid diagnosis. Ann R Coll Surg Engl. 2010;92(2):163–6.
45. Fortin JD, Falco FJ. The Fortin finger test: an indicator of sacroiliac pain. Am J Orthop. 1997;26(7):477–80.
46. Byrd JW. Evaluation of the hip: history and physical examination. N Am J Sports Phys Ther. 2007;2(4):231–40.
47. Kuhlman GS, Domb BG. Hip impingement: identifying and treating a common cause of hip pain. Am Fam Physician. 2009;80(12):1429–34.

48. Bird PA, Oakley SP, Shnier R, Kirkham BW. Prospective evaluation of magnetic resonance imaging and physical examination findings in patients with greater trochanteric pan syndrome. Arthritis Rheum. 2001;44(9):2138–45PA, Oakley.
49. Lequesne M, Mathieu P, Vuillemin-Bodaghi V, Bard H, Djian P. Gluteal tendinopathy in refractory greater trochanter pain syndrome: diagnostic value of two clinical tests. Arthritis Rheum. 2008;59(2):241–6.
50. Beatty RA. The piriformis muscle syndrome: a simple diagnostic maneuver. Neurosurgery. 1994;34(3):512–4.
51. Kirschner JS, Foye PM, Cole JL. Piriformis syndrome, diagnosis and treatment. Muscle Nerve. 2009;40:10–8. https://doi.org/10.1002/mus.21318.
52. Akseki D, Ozcan O, Boya H, Pinar H. A new weight-bearing meniscal test and a comparison with McMurray's test and joint line tenderness. Arthroscopy. 2004;20(9):951–8.
53. Pookarnianamorakot C, Korsantirat T, Woratanarat P. Meniscal lesions in the anterior crutiate sinsufficient knee: the accuracy of clinical evaluation. J Med Assoc Thail. 2004;87(6):618–23.
54. Nijs J, Van Geel C, Van der Auwera C, Van de Velde B. Diagnostic value of five clinical tests in patellofemoral pain syndrome. Manual Ther. 2006;11(1):69–77.
55. Daruwalla ZJ, Wong KL, Thambiah J. The application of telemedicine in orthopedic surgery in Singapore: a pilot study on a secure, mobile telehealth application and messaging platform. JMIR Mhealth Uhealth. 2014;2(2):e28.
56. Buvik A, Bugge E, Knutsen G, Småbrekke A, Wilsgaard T. Quality of care for remote orthopaedic consultations using telemedicine: a randomized controlled trial. BMC Health Serv Res. 2016;16(1):483.
57. Awadallah M, Janssen F, Körber B, Breuer L, Scibor M, Handschu R. Telemedicine in general neurology: interrater reliability of clinical neurological examination via audio-visual telemedicine. Eur Neurol. 2018;80(5–6):289–94. https://doi.org/10.1159/000497157.

The Future of Spine Care

33

Kathleen R. Bell, Hunt Batjer, and Charles Whitten

The conundrum of spine disorders remains unanswered at present. While surgical techniques and certain technologies have advanced, the overall approach to spine care remains fragmented to a large degree. However, there are a number of evolving approaches that offer great promise. The future of spine care will include new technological advancements as well as widespread use of evidence- based, cost effective interventions that have not yet been fully adopted by patients and physicians. The information from large electronic medical record databases about the natural history of back pain, other spine disorders and treatments will change the approach to management. Last but perhaps most importantly, the formation of interdisciplinary teams to evaluate and treat pain with the patient as an essential team member will permit the most judicious management plan. In considering the future of spine care, we will discuss new technology and techniques, the advent of big data, team medicine, and the continued struggles with pain management and legal challenges.

K. R. Bell
Department of Physical Medicine and Rehabilitation, University of Texas Southwestern Medical Center, Dallas, TX, USA
e-mail: Charles.Whitten@UTSouthwestern.edu

H. Batjer
University of Texas Southwestern Medical Center, Dallas, TX, USA
e-mail: Kathleen.Bell@UTSouthwestern.edu

C. Whitten (✉)
Department of Anesthesiology and Pain Management, University of Texas Southwestern Medical Center, Dallas, TX, USA

© The Author(s), under exclusive license to Springer Nature Switzerland AG 2022

C. E. Noe (ed.), *Multidisciplinary Spine Care*,
https://doi.org/10.1007/978-3-031-04990-3_33

Technology and Technique

Injections

Injections for spine pain are criticized for being over-utilized and trends toward decreasing unnecessary injections will likely occur [1]. The practice of performing multiple types of injections for chronic pain symptoms will likely decline. Instead, using injections in conjunction with surgical planning is a likely trend. The complications from injections have increased over the past decades along with the number of injections being performed.

A number of recommendations have been made to reduce the risk associated with injections for spine pain [2]. These recommendations will be incorporated into widespread practice in the future. For cervical interlaminar injections, imaging is recommended for needle placement. The recommended level for cervical interlaminar injection is C7-T1. Contrast injection is also recommended. For transforaminal injections, contrast injection using extension tubing and continuous fluoroscopy or digital subtractions is recommended. Particulate steroid is not recommended. Moderate to heavy sedation is not recommended. These recommendations will likely be implemented into practice. Reimbursement for these procedures may become tied to documentation of performance of these steps. Additional safety recommendations have been proposed by the Benelux work group [3]. These recommendations are likely to influence future practice as well.

In summary, a test dose of local anesthetic should be used for transforaminal injections before steroid injection. Limiting the doses of steroid to 40 mg of methylprednisolone, 20 mg for triamcinolone and 10 mg dexamethasone, is recommended. Dexamethasone (as opposed to particulate steroid) is recommended for transforaminal injections above the L3 level. Imaging should be reviewed prior to injections to make sure the epidural space is large enough at the level of injection to accommodate the volume of injected fluid. The volume of injected fluid should be limited to 4 ml. The needle tip should be placed in the posterior foramen. Air bubbles should be removed from the tubing.

Cervical transforaminal injections are falling out of favor due to complications from intra-arterial injections and injuries. Furthermore, a randomized trial reported no difference between cervical transforaminal steroid injections and controls [4]. In another study of cervical transforaminal epidural steroid injections versus facet injections, no difference was shown [5].

In the future, complications associated with injection procedures will be reduced with improved techniques and also by limiting procedures to evidence-based procedures and cost-effectiveness strategies.

Biological treatments will be studied and in the future some therapies will be abandoned while others may be prove to be effective. A phase 1 trial of intradiscal adipose derived mesenchymal stem cell injections demonstrated safety [6].

Patients reported reduced pain and disability but studies with a control group are needed in this area of research.

Multimodal Postoperative Analgesia

Multimodal analgesia with gabapentinioids, magnesium, corticosteroid and ketamine has become popular [7]. However, none of these drugs have FDA labeled indications for perioperative pain. A multimodal regimen of acetaminophen, gabapentin, ketamine and iv lidocaine was not shown to be superior to placebos of each drug in patients undergoing multilevel spine surgery [8].

In the future, multimodal analgesia may be simplified to use fewer drugs and focus on using acetaminophen, NSAID and local anesthetic. Opioids will be used as rescue analgesics. Also, non-drug interventions such as relaxation techniques (e.g. diaphragmatic breathing) and physical modalities (e.g., cold) will be used routinely.

Surgical Innovation

Several factors will improve surgical treatments of spine conditions in the future. New techniques to preserve motion will be developed as an alternative to fusion. Preoperative planning will be more accurate using advanced imaging. 3 dimensional imaging for hardware placement will be used. Robotics will be used for minimally invasive techniques. Implanted devices will be customized on site on the day of surgery. New materials for devices will increase the options for many procedures.

Neuroaugmentation

The use of spinal cord stimulation for back pain may increase as new high frequency stimulation patterns are proving to be effective [9, 10].

Big Data

Electronic Medical Records and Registries

Electronic medical records will become easier to use and more efficient. Electronic medical records may help identify patients who are over utilizing resources early. For example, one study found, through electronic medical record information, that male gender, workers' compensation and smoking were associated with higher treatment costs [11].

Also, a clinical decision support tool has been embedded in the electronic medical record to help guide return to work prescriptions successfully [12].

Big data research using data from electronic medical records will lead to the identification of risk factors and risk mitigation factors that will change practice.

Machine learning will empower physicians to make individualized and specific treatment recommendations to each patient. Spine care patients will be a major

beneficiary of this new technology if we create the accurate data input gained from new technologies. Studies are currently underway to use artificial intelligence, genomic analysis, genome guided therapy, microfluidics, genome editing, wearable monitors, digital health and biomaterials to customize treatment for individual patients in a variety of clinical settings [13].

Machine learning and artificial intelligence will be able to predict non-surgical versus surgical outcomes more accurately than regression models [14]. This will allow patients to participate in shared decision making in a more meaningful way.

Guidelines

When clinicians were asked if the medical community would ever reach a consensus about how to treat nonspecific low back pain, 83% said no. [15]

Patients were asked what helped them the most. 63% said a combination of treatments followed by medication (14%), physical therapy and exercise (12%), psychotherapy (4%), relaxation therapy (3%), heat (2%), chiropractic manipulation (2%), yoga or tai chi (0%) and acupuncture (0%). In the future, Clinical Practice Guidelines will be used on a more widespread basis. Using non-pharmacological treatments and self-care will be promoted with education delivered electronically [16, 17].

Different groups have promulgated different guidelines based on their own analysis of evidence and opinion [16–23]. The possibility of bias exists towards the services offered by the group formulating the guidelines. Also, bias may exist based on other factors including financial relationships with interested parties. In the future, guidelines will be improved to be more universally accepted by surgical and non-surgical specialties. Cost effectiveness and patient preferences may be included as factors.

Outcome Documentation

In the future, physicians will be increasingly required to document their outcomes; however, there is no established standard outcome reporting system for spine care. The North American Spine Society has a long track record of work developing instruments for cervical and lumbar spine conditions [24–26].

Several instruments have been validated to evaluate cervical spine patients including the neck disability index, the NASS cervical questionnaire and the core outcome measures index (COMI) for cervical spine problems [27–29]. The Oswestry low back disability questionnaire, Quebec back pain disability scale, the North American Spine Society Lumbar Spine Outcome Assessment Instrument and the Roland Morris low back pain measure have been validated for lumbar spine problems [29–32].

For pain research, several critical outcome domains have been identified including (1) pain, (2) physical functioning, (3) emotional functioning, (4) participant ratings of improvement and satisfaction with treatment, (5) symptoms and adverse events, (6) participant disposition (e.g. adherence to the treatment regimen and reasons for premature withdrawal from the trial) [33].

Also, outcome instruments have been developed to measure comprehensive medical outcomes including the SF-36 and PROMIS system [34, 35]. The PROMIS system has been used to develop a measure for pain interference and pain behavior [36–38].

In the early 1990's the commission for the accreditation (CARF) promoted an outcome system with 10 domains:

1. Medical findings
2. Pain severity
3. Medications
4. Physcial fucntion
5. Social function
6. Psychological function
7. Productivity
8. Healthcare utilization
9. Patient costs
10. System costs

While these outcome domains are too comprehensive for most practicing clinicians to implement, they include important items. Medications and doses are important to track due to the opioid epidemic. Productivity is a major outcome that most self-report instruments fail to capture. Healthcare utilization and costs are obviously important but they are difficult to quantify.

The Canadian Occupational Performance Measure (COPM) is an instrument that allows patients to set their own goals and individualize their outcome data [39]. It is a truly patient centric outcome instrument and is validated.

A major limitation of self-report outcome instruments is patient participation. Many outcome measures are repetitive and lengthy. Patients do not complete online questionnaires or paper forms reliably. The amount of missing data is problematic. In the future, collecting data may include using smart phones and remote monitoring devices to monitor patients' activity and outcomes continuously in real time instead of periodically with snapshot lists of self-report questions.

Electronic medical records may become a meaningful source of outcome data if discreet data fields can be standardized for spine care [40].

Until a standardized outcome system for spine has been developed, patient satisfaction and treatment costs will likely continue to be outcomes that payors use to discriminate between providers.

Team Medicine

Interdisciplinary Spine Rehabilitation Programs

The future will include interdisciplinary pain management being used more frequently and earlier in the course of spine pain. A systematic review of multidisciplinary biopsychosocial rehabilitation for chronic low back pain concluded that the

multidisciplinary model is more effective than physical treatments alone [41]. Interdisciplinary pain management programs can not only improve chronic pain but can prevent chronic low back pain and the associated bad outcomes (e.g. Opioid use, overutilization) from progressing [42, 43]. Several studies have compared interdisciplinary pain management to lumbar spine fusion surgery [44–48]. The results suggest that interdisciplinary pain management programs may be a good alternative for many patients. In the future, interdisciplinary pain management may be required before or in conjunction with lumbar spine fusion surgery. Also, interdisciplinary pain management programs are an effective treatment for opioid reduction [49]. Interdisciplinary pain programs will be an important alternative to chronic opioid therapy and may be a useful adjunctive therapy for patients with co-existing substance use disorder and chronic pain. However, interdisciplinary pain programs have become less abundant. One estimate concluded that we have only 1 program for every 670,000 Americans [50]. In the future, an increase in the number of these programs is necessary in order to provide the best care for many patients with chronic pain.

Prevention and Self Management

The future will include improved utilization of measures to prevent back pain. We already have evidence that exercise and education prevent back pain [51]. However, only 23% of Americans between the ages of 18 and 64 meet guidelines recommendations for exercise [52].

Educational material about proper body mechanics for lifting and other activity is available but not widely used [53, 54]. Avoiding heavy lifting and lifting and twisting simultaneously may prevent back pain. Avoiding bed rest as a treatment for non-specific low back pain is essential. Also, smoking cessation may reduce the risk of developing back pain. Maintaining a healthy weight with proper nutrition may also be beneficial. Self-care for acute episodes of back pain using heat and over the counter analgesics will help patients manage most episodes of uncomplicated, acute back pain. Providing education in a format that is effective for the patient is critical [55].

Improved methods for motivating patients to prevent back pain will be developed in the future. Motivational interviewing has been successful in patients with other conditions [56, 57]. Preliminary evidence suggests that motivational interviewing may be successful in managing subacute low back pain [58].

ERAS Programs

Enhanced recovery after Surgery (ERAS) programs have been used for a wide variety of cases including spine surgery. In the future, these programs will be studied and refined to retain effective interventions and abandon interventions that are not cost effective. For spine surgery, a comprehensive preoperative plan including a prehabilitation period has been used [59].

Minimally invasive surgical techniques allow for rapid postoperative mobilization, same day or short stay hospitalization and rapid return to work. In the future, new surgical techniques will minimize tissue trauma and the associated recovery time.

Healthcare System Changes

Incremental changes in the healthcare system are more likely than a sudden change to a single-payer system. However, the costs associated with back pain evaluation and treatment will get more scrutiny in the future. Direct costs for back pain healthcare are $34,167,000,000 (2010 dollars) in the United States [59–61].

A recent study found that 55.7% of patients with newly diagnosed back pain received no medical treatment and only 1.2% had surgery. However, the cost of surgical treatment was 29.3% of the total costs over 12 months. The non-surgical patient's evaluation and treatment cost 1.8 billion dollars. 32.3% had imaging within 30 days of diagnosis and 35.3% had imaging without first having a trial of physical therapy [62].

Indirect costs have been estimated to be twice that of the indirect costs [63]. An early intervention interdisciplinary pain rehabilitation program is an effective way to reduce indirect costs associated with back pain [64].

In this study, adding a work transition program to the early intervention program did not add benefits. In the future, early intervention approaches will emerge to reduce not only the direct costs of treatment but the indirect costs.

New Models of Care

Managed care will enforce guidelines for evaluation and treatment, particularly for imaging and procedures. Telemedicine can provide more frequent contacts at lower costs per contact. This may facilitate moving patients along a care path. Direct access to spine clinics will streamline care and provide most evaluation and treatment services in one location.

Risk stratification methods will specifically determine the spinal level involved for an episode of care. This will focus care on a specific injury or diagnosis rather than open up reimbursement for treatment of other problems. Risk stratification methods will also determine the severity of spine pathology and will guide treatment options rather than allow a non-specific series of conservative treatment trials. Risk factors for disability and long term overutilization will be identified during the acute and subacute phases.

The STarT instrument has been used to evaluate patients with back pain of any duration and to classify patients as low, moderate or high risk [65, 66]. A treatment plan based on the StarT risk level has been associated with better outcomes [67].

Interventions to mitigate that risk will be implemented in a timelier fashion rather than delaying risk management until patients have chronic symptoms or are

at catastrophic risk levels. Care paths for specific populations and diagnoses will focus resources to maximize cost effectiveness and reduce complications.

Medical Training

The number of spine specialists has increased significantly over the past decades. If the number of spine procedures being performed is too high, perhaps the number of training positions for interventional specialists should decrease and the number of specialists with non-invasive training should increase. This would produce a better match between the supply and demand for evidence based treatment. In the future, compensation models will shift to incentivize medical students to enter training programs in specialties in short supply.

The Opioid Epidemic

The future of spine care will continue to include the use of opioids for pain control, but the average duration of treatment and dose will decline. Multiple factors are moving spine care away from opioid therapy, especially for chronic back pain. The number of deaths in the United States from drug overdoses during the opioid epidemic has surpassed 700,000 and most of these involve prescription opioids, obtained either legally or illicitly [68].

In 2017, 18 million Americans (5.5%) took opioids daily for pain. 4.2% of the US population aged 12 or older misused opioids (including heroin). 92% of the people who misused were taking prescription opioids, whether acquired legally or illegally [69, 70]. From this, it seems clear that there is too much prescription opioid being dispensed to the public to maintain a reasonable level of safety.

Treating osteoarthritis with tramadol as a first line drug is associated with higher 1 year mortality compared to initial treatment with NSAID [71]. One study found that over 50% of non-fatal overdoses occur within 90 days of opioid treatment and that 30% occur on doses below 50 mg per day of morphine equivalents [72]. This contradicts the notion that only patients on high dose, long term opioid therapy are at high risk for accidental overdoses.

In the future, opioids will continue to be used to treat postoperative pain but the trend will be that opioids will be tapered off after an appropriate duration of time and non-opioid analgesics will be used as alternatives for long term use.

Lack of Efficacy of Opioids for Long Term Use

In addition to a lack of safety, there is a lack of long- term evidence from randomized controlled trials demonstrating more than a small analgesic with opioids in patients with chronic pain [73]. The SPACE trial showed that in patients with chronic back, hip and knee pain, non-opioid analgesics were actually more effective

analgesics than opioids [74]. No difference between the groups was found for functional status. Another study found that only 12% of patients on long-term opioid therapy experience both significant analgesia (>30%) and high levels of functioning [75].

So treating patients with opioids long term is of questionable efficacy, as well as safety. In the future, the percentage of the spine patient population taking opioids long-term will reduce by 50% or more. Also, the average dose will decrease by 50% or more. Intrathecal opioid pump use for back pain is likely to decline as well.

Opioids and Surgical Outcomes

Preoperative opioid use is associated with worse postoperative outcomes [76]. Reducing opioids before surgery may mitigate against the adverse effects of chronic opioid use [77]. Also, prescribing less opioid than in the past for post-operative pain doesn't seem to worsen outcomes and has the positive effect of reducing the amount of opioid available for diversion or overdose, accidental or intentional [78].

In one study of patients having spine surgery, 12.8% of opioid naive patients continued opioids chronically after surgery [79]. 77% of long-term preoperative opioid users continued opioids long term after surgery. Preoperative opioid doses and the number of postoperative prescribers were the strongest associated factors. However, in another study, opioid naive patients, only 0.2% of patients took opioids longer than 6 months after surgery [80]. This suggests that patients on opioids preoperatively may be a bigger problem than opioid naive patients who can be easily tapered off opioids after surgery.

Checking electronic prescription records prior to prescribing opioids and other scheduled drugs will become required in most, if not all, states in the US. Also checking the electronic prescription record prior to scheduling surgery may be used in the future to evaluate perioperative risk. The opioid dose, number of prescriptions, prescribers, pharmacies, and distance traveled for prescriptions and out of pocket purchases have been identified as risk factors for overdose or misuse [81–83].

Regulators at the state and federal level will become more active with disciplinary actions against physicians about opioid prescribing during the perioperative period, as well as the non-surgical spine episodes of care. Surgeons will prescribe opioids for shorter durations for postoperative pain and will need to see patients on a more frequent basis to manage opioids in the postoperative period.

Regulatory and Medicolegal Environment

The future will bring more malpractice and medical board action due to complications and dissatisfaction with results. More physicians have been trained to treat spine pain and they will face increased scrutiny [84–86]. Increases in liability associated with cervical injections and medication management have occurred over the past decades [87]. This will likely result in less aggressive use of cervical injections

and opioid prescribing. A study of spine surgery litigation in the United States reported that the average settlement amount was $2,384,775 and the average jury award was $3,945,456 [88].

The future will include additional attempts at tort reform and more peer review of complications and patient complaints.

Patient Satisfaction

Patient satisfaction is higher among outpatients with chronic musculoskeletal pain who are treated with opioids [89]. In another study, higher patient satisfaction scores were associated with fewer emergency room visits but with more hospitalizations, higher health care costs including prescription drug costs [90].

Paradoxically, higher satisfaction was associated with higher mortality. However, in a surgical population, opioid prescribing was decreased by 50% without adverse effects on satisfaction scores [91]. In this study, the surgeons set clear expectations about pain and opioids preoperatively and prescribed effective non-opioid analgesics as an opioid sparring intervention.

In the future, patient satisfaction surveys will use computer adaptive testing to gain more information about the reasons for dissatisfaction. In this way, dissatisfaction related to factors beyond the physicians control will not be held against them.

COVID-19

At the time of this writing, the world is being overwhelmed with the new Covid 19 disease (Severe acute respiratory syndrome coronavirus-2). The impact of Covid 19 on spine care is uncertain but it is likely that vaccines will be successful along with moderately effective treatments. The economic impact of this disease may have a long term effect on investment in new technology for spine care and may result in a realignment of healthcare priorities.

The expanded use of telemedicine during the pandemic will have a lasting effect on the way we manage our outpatient practice for Spine Disease.

Conclusions

The future of spine care will be characterized by significant technological advances and simultaneous healthcare market forces to increase cost effectiveness. The physicians and health systems that can deliver results on both will prosper.

References

1. Kennedy DJ, Baker RM, Rathmell JP. Use of spinal injections for low back pain. JAMA. 2013;310(16):1736. https://doi.org/10.1001/jama.2013.277957. https://www.painnewsnetwork.org/stories/2015/8/19/experts-say-epidural-injections-overused.

2. Rathmell JP, Benzon HT, Dreyfuss P, Huntoon M, Wallace M, Baker R, Riew KD, Rosenquist RW, Aprill C, Rost NS, Buvanendran A, Kreiner DS, Bogduk N, Fourney DR, Fraifeld E, Horn S, Stone J, Vorenkamp K, Lawler G, Summers J, Kloth D, O'Brien D, Tutton S. Safeguards to prevent neurologic complications after epidural steroid injections: consensus opinions from a Multidisciplinary Working Group and National Organizations. Anesthesiology. 2015;122(5):974–84. https://doi.org/10.1097/ALN.0000000000000614.
3. Van Boxem K, Rijsdijk M, Hans G, de Jong J, Kallewaard JW, Vissers K, van Kleef M, Rathmell JP, Van Zundert J. Safe use of epidural corticosteroid injections: recommendations of the WIP Benelux Work Group. Pain Pract. 2019;19:61–92. https://doi.org/10.1111/papr.12709.
4. Anderberg L, Annertz M, Persson L, Brandt L, Säveland H. Transforaminal steroid injections for the treatment of cervical radiculopathy: a prospective and randomised study. Eur Spine J. 2007;16(3):321–8. https://doi.org/10.1007/s00586-006-0142-8.
5. Bureau NJ, Dagher JH, Shedid D, Li M, Brassard P, Leduc BE. Transforaminal versus intraarticular facet corticosteroid injections for the treatment of cervical radiculopathy: a randomized, double-blind, controlled study. Am J Neuroradiol. 2014;35(8):1467–74.
6. Kumar H, Ha DH, Lee EJ, Park JH, Shim JH, Ahn TK, Kim KT, Ropper AE, Sohn S, Kim CH, Thakor DK, Lee SH, Han IB. Safety and tolerability of intradiscal implantation of combined autologous adipose-derived mesenchymal stem cells and hyaluronic acid in patients with chronic discogenic low back pain: 1-year follow-up of a phase I study. Stem Cell Res Ther. 2017;8(1):262.
7. Bruhn J, Scheffer G, van Geffen G-J. Clinical application of perioperative multimodal analgesia. Curr Opin Support Palliat Care. 2017;11(2):106–11. https://doi.org/10.1097/SPC.0000000000000267. PMID: 28333865. ISSN Print: 1751–4258. Publication Date: June 2017.
8. Maheshwari K, Avitsian R, Sessler DI, Makarova N, Tanios M, Raza S, Traul D, Rajan S, Manlapaz M, Machado S, Krishnaney A, Machado A, Rosenquist R, Kurz A. Multimodal analgesic regimen for spine surgery: a randomized placebo-controlled trial. Anesthesiology. 2020;132(5):992–1002. https://doi.org/10.1097/ALN.0000000000003143.
9. Kapural L, et al. Comparison of 10-kHz high-frequency and traditional low-frequency spinal cord stimulation for the treatment of chronic back and leg pain: 24-month results from a multicenter, randomized, controlled pivotal trial. Neurosurgery. 2016;79:667–77. https://doi.org/10.1227/neu.0000000000001418.
10. Amirdelfan K, et al. Long-term quality of life improvement for chronic intractable back and leg pain patients using spinal cord stimulation: 12-month results from the SENZA-RCT. Qual Life Res. 2018;27:2035–44. https://doi.org/10.1007/s11136-018-1890-8.
11. Maeng DD, Stewart WF, Yan X, et al. Use of electronic health records for early detection of high-cost, low back pain patients. Pain Res Manag. 2015;20(5):234–40. https://doi.org/10.1155/2015/862702.
12. McLellan RK, Haas NS, Kownacki RP, Pransky GS, Talmage JB, Dreger M. Using electronic health records and clinical decision support to provide return-to-work guidance for primary care practitioners for patients with low back pain. J Occup Environ Med. 2017;59(11):e240–4. https://doi.org/10.1097/JOM.0000000000001180.
13. Ho D, Quake SR, McCabe ERB, et al. Enabling technologies for personalized and precision medicine. Trends Biotechnol. 2020;38(5):497–518. https://doi.org/10.1016/j.tibtech.2019.12.021.
14. Khan O, Badhiwala JH, Grasso G, Fehlings MG. Use of machine learning and artificial intelligence to drive personalized medicine approaches for spine care. World Neurosurg. 2020;140:512–8. https://doi.org/10.1016/j.wneu.2020.04.022.
15. Nierengarten MB. Chronic low back pain: can we find a treatment consensus? Pract Pain Manag. 20(2):29–31.
16. Chou R, Deyo R, Friedly J, et al. Nonpharmacologic therapies for low back pain: a systematic review for an American College of Physicians Clinical Practice Guideline. Ann Intern Med. 2017;166:493–505. [Epub ahead of print 14 February 2017]. https://doi.org/10.7326/M16-2459.

17. Qaseem A, Wilt TJ, McLean RM, et al; for the Clinical Guidelines Committee of the American College of Physicians. Noninvasive treatments for acute, subacute, and chronic low back pain: a clinical practice guideline from the American College of Physicians. Ann Intern Med. 2017;166:514–30. [Epub ahead of print 14 February 2017]. https://doi.org/10.7326/M16-2367.
18. VA/DoD clinical practice guideline for diagnosis and treatment of low back pain. Version 2.0–2017. https://www.healthquality.va.gov/guidelines/Pain/lbp/VADoDLBPCPG092917.pdf.
19. Chou R, Deyo R, Friedly J, et al. Systemic pharmacologic therapies for low back pain: a systematic review for an American College of Physicians Clinical Practice Guideline. Ann Intern Med. 2017;166:480–92. [Epub ahead of print 14 February 2017]. https://doi.org/10.7326/M16-2458.
20. Oliveira CB, Maher CG, Pinto RZ, et al. Clinical practice guidelines for the management of non-specific low back pain in primary care: an updated overview. Eur Spine J. 2018;27:2791–803. https://doi.org/10.1007/s00586-018-5673-2.
21. Evidence-based clinical guidelines for multidisciplinary spine care: diagnosis & treatment of low back pain. North American Spine Society 2020. https://www.spine.org/Portals/0/assets/downloads/ResearchClinicalCare/Guidelines/LowBackPain.pdf.
22. Manchikanti L, Abdi S, Atluri S, et al. An update of comprehensive evidence-based guidelines for interventional techniques in chronic spinal pain. Part II: Guidance and recommendations. Pain Physician. 2013;16:S49–S283.
23. Bussières AE, Stewart G, Al-Zoubi F, Decina P, Descarreaux M, Haskett D, Hincapié C, Pagé I, Passmore S, Srbely J, Stupar M, Weisberg J, Ornelas J. Spinal manipulative therapy and other conservative treatments for low back pain: a guideline from the Canadian Chiropractic Guideline Initiative. J Manipulative Physiol Ther. 2018;41(4):265–93, . ISSN 0161-4754. https://doi.org/10.1016/j.jmpt.2017.12.004.
24. Gatchel RJ. Compendium of outcome instruments for assessment and research of spinal disorders. LaGrange, IL: North American Spine Society; 2001.
25. Gatchel RJ. Compendium of outcome instruments for assessment and research of spinal disorders. 2nd ed. LaGrange, IL: North American Spine Society; 2006.
26. Katz JN. Measures of adult back and neck function: the North American Spine Society (NASS) Lumbar Spine Outcome Assessment Instrument, Neck Disability Index, Oswestry Low Back Pain Disability Index, Quebec Back Pain Disability Scale, and Roland-Morris Low Back Pain Measure. Arthritis Rheum. 2003;49:S43–9. https://doi.org/10.1002/art.11399.
27. Vernon H, Mior S. The neck pain disability index: a study of reliability and validity. J Manip Physiol Ther. 1991;14:409–15.
28. Stoll T, Huber E, Bachmann S, Baumeler H, Mariacher S, Rutz M, Schneider W, Spring H, Aeschlimann A, Stucki G, Steiner W. Validity and sensitivity to change of the NASS questionnaire for patients with cervical spine disorders. Spine (Phila Pa 1976). 2004;29:2851–5. https://doi.org/10.1097/01.brs.0000147802.57484.77.
29. Fankhauser CD, Mutter U, Aghayev E, Mannion AF. Validity and responsiveness of the Core Outcome Measures Index (COMI) for the neck [published correction appears in Eur Spine J. 2012;21(3):561]. Eur Spine J. 2012;21(1):101–14. https://doi.org/10.1007/s00586-011-1921-4.
30. Kopec JA, Esdaile JM, Abrahamowicz M, Abenhaim L, Wood-Dauphinee S, Lamping DL, et al. The Quebec Back Pain Disability. Scale: measurement properties. Spine. 1995;20:341–52.
31. Daltroy LH, Cats-Baril WL, Katz JN, Fossel AH, Liang MH. The North American Spine Society lumbar spine outcome assessment instrument. Spine. 1996;21:741–9.
32. Roland M, Morris R. A study of the natural history of back pain: Part I. Development of a reliable and sensitive measure of disability in low-back pain. Spine. 1983;8:141–4.
33. Dworkin RH, Turk DC, Farrar JT, Haythornthwaite JA, Jensen MP, Katz NP, et al. Core outcome measures for chronic pain clinical trials: IMMPACT recommendations. Pain. 2005;113(1):9–19.
34. Ware JE Jr, Sherbourne CD, The MOS. 36-item short-form health survey (SF-36). I. Conceptual framework and item selection. Med Care. 1992;30(6):473–83.

35. Cella D, Riley W, Stone AA, Rothrock N, Reeve BB, Yount S, Amtmann DDB, Choi S, Cook KF, et al. The Patient Reported Outcomes Measurement Information System (PROMIS) developed and tested its first wave of adult selfreported health outcome item banks: 2005–2008. J Clin Epidemiol. 2010;63:1179–94.
36. Amtmann DA, Cook KF, Jensen MP, Chen W-H, Choi SW, Revicki D, Cella D, Rothrock N, Keefe F, Callahan L, Lai J-S. Development of a PROMIS item bank to measure pain interference. Pain. 2010;150(1):173–82.
37. Revicki DA, Chen W-H, Harnam N, Cook K, Amtmann D, Callahan LF, Jensen MP, Keefe FJ. Development and psychometric analysis of the PROMIS pain behavior item bank. Pain. 2009;146(1–2):158–69.
38. Cook KF, Keefe F, Jensen MP, Roddey TS, Callahan LF, et al. Development and validation of a new self-report measure of pain behaviors. Pain. 2013;154(12):2867–76.
39. Law M, Baptiste S, McColl M, Opzoomer A, Polatajko H, Pollock N. The Canadian occupational performance measure: an outcome measure for occupational therapy. Can J Occup Ther. 1990;57(2):82–7. https://doi.org/10.1177/000841749005700207.
40. Lee JH. Future of the smartphone for patients and healthcare providers. Healthc Inform Res. 2016;22(1):1–2. https://doi.org/10.4258/hir.2016.22.1.1.
41. Kamper SJ, Apeldoorn AT, Chiarotto A, Smeets RJ, Ostelo RWJG, Guzman J, van Tulder MW. Multisciplinary biopsychosocial rehabilitation for chronic low back pain. Cochrane Database Syst Rev. 2014;9:CD000963. https://doi.org/10.1002/14651858.CD000963.pub3.
42. Gatchel RJ, Polatin PB, Noe C, et al. Treatment and cost-effectiveness of early intervention for acute low back pain patients: a one-year prospective study. J Occup Rehabil. 2003;13:1–9.
43. Whitfill T, Haggard R, Bierner SM, et al. Early intervention options for acute low back pain patients: a randomized clinical trial with one-year follow-up outcomes. J Occup rehabil. 2010;20:256–63. https://doi.org/10.1007/s10926-010-9238-4.
44. Fairbank J, Frost H, Wilson-MacDonald J, et al. Randomised controlled trial to compare surgical stabilisation of the lumbar spine with an intensive rehabilitation programme for patients with chronic low back pain: the MRC spine stabilisation trial. BMJ. 2005;330:1233–9.
45. Fritzell P, Hagg O, Wessberg P, et al. Volvo Award Winner in Clinical Studies: Lumbar fusion versus nonsurgical treatment for chronic low back pain: a multicenter randomized controlled trial from the Swedish Lumbar Spine Study Group. Spine (Phila Pa 1976). 2001;26:2521–32; discussion 32–34.
46. Ohtori S, Koshi T, Yamashita M, et al. Surgical versus nonsurgical treatment of selected patients with discogenic low back pain: a small sized randomized trial. Spine (Phila Pa 1976). 2011;36:347–54.
47. Brox JI, Sorensen R, Friis A, et al. Randomized clinical trial of lumbar instrumented fusion and cognitive intervention and exercises in patients with chronic low back pain and disc degeneration. Spine (Phila Pa 1976). 2003;28:1913–21.
48. Brox JI, Reikeras O, Nygaard O, et al. Lumbar instrumented fusion compared with cognitive intervention and exercises in patients with chronic back pain after previous surgery for disc herniation: a prospective randomized controlled study. Pain. 2006;122:145–55.
49. Frank JW, Lovejoy TI, Becker WC, et al. Patient outcomes in dose reduction or discontinuation of long-term opioid therapy: a systematic review. Ann Intern Med. 2017;167:181–91. [Epub ahead of print 11 July 2017]. https://doi.org/10.7326/M17-0598.
50. Schatman ME. Interdisciplinary chronic pain management: international perspectives. Pain. 2012;20(7)
51. Foster NE, Anema JR, Cherkin D, Chou R, Cohen SP, Gross DP, Ferreira PH, Fritz JM, Koes BW, Peul W, Turner JA, Maher CG, on behalf of the Lancet Low Back Pain Series Working Group. Prevention and treatment of low back pain: evidence, challenges, and promising directions. Lancet. 2018;391:2368–83.
52. Blackwell DL, Clarke TC. State variation in meeting the 2008 federal guidelines for both aerobic and muscle-strengthening activities through leisure-time physical activity among adults aged 18–64: United States 2010–2015. National Health Statistics Reports; no 112.

Hyattsville, MD: National Center for Health Statistics. 2018. https://www.cdc.gov/nchs/data/nhsr/nhsr112.pdf.
53. Royal College of General Practitioners and NHS Executive. Get back active–the back book video. London: Stationery Office; 2002. http://www.biomechanicsofbackpain.com/video.php
54. Burton K, et al. The back book – UK edition. 2nd ed. TSO (The Stationery Office).
55. Marcus C. Strategies for improving the quality of verbal patient and family education: a review of the literature and creation of the EDUCATE model. Health Psychol Behav Med. 2014;2(1):482–95. https://doi.org/10.1080/21642850.2014.900450.
56. Poudel N, Kavookjian J, Scalese MJ. Motivational interviewing as a strategy to impact outcomes in heart failure patients: a systematic review. Patient. 2020;13:43–55. https://doi.org/10.1007/s40271-019-00387-6.
57. Dellasega C, Gabbay R, Durdock K, Martinez-King N. Motivational interviewing (MI) to change type 2DM self care behaviors: a nursing intervention. J Diabetes Nurs. 2010;14(3):112–8.
58. O'Halloran PD, Holden J, Breckon J, et al. Embedded Motivational Interviewing combined with a smartphone app to increase physical activity in people with sub-acute low back pain: study protocol of a cluster randomised control trial. Contemp Clin Trials Commun. 2019;17:100511. Published 2019 Dec 24. https://doi.org/10.1016/j.conctc.2019.100511.
59. Nielsen PR, Jørgensen LD, Dahl B, Pedersen T, Tønnesen H. Prehabilitation and early rehabilitation after spinal surgery: randomized clinical trial. Clin Rehabil. 2010;24(2):137–48. https://doi.org/10.1177/0269215509347432.
60. Nielsen PR, Andreasen J, Asmussen M, Tønnesen H. Costs and quality of life for prehabilitation and early rehabilitation after surgery of the lumbar spine. BMC Health Serv Res. 2008;8:209. https://doi.org/10.1186/1472-6963-8-209.
61. Institute of Medicine (US) Committee on Advancing Pain Research, Care, and Education. Relieving pain in America: a blueprint for transforming prevention, care, education, and research. Washington, DC, National Academies Press (US); 2011.
62. Gaskin DJ, Richard P. The economic costs of pain in the United States. In: Institute of Medicine (US) Committee on Advancing Pain Research, Care, and Education, editor. Relieving pain in America: a blueprint for transforming prevention, care, education, and research. Washington, DC: National Academies Press (US); 2011. Appendix C. Available from: https://www.ncbi.nlm.nih.gov/books/NBK92521/.
63. Kim LH, Vail D, Azad TD, et al. Expenditures and health care utilization among adults with newly diagnosed low back and lower extremity pain. JAMA Netw Open. 2019;2(5):e193676. https://doi.org/10.1001/jamanetworkopen.2019.3676. Katz JN. Lumbar disc disorders and low-back pain: socioeconomic factors and consequences. J Bone Joint Surg Am. 2006;88 Suppl 2:21–4.
64. Rogerson MD, Gatchel RJ, Bierner SM. A cost utility analysis of interdisciplinary early intervention versus treatment as usual for high-risk acute low back pain patients. Pain Pract. 2010;10(5):382–95. https://doi.org/10.1111/j.1533-2500.2009.00344.x.
65. Keele University. STarT back screening tool website. http://www.keele.ac.uk/sbst/. Accessed 23 Jul 2013.
66. Hill JC, Dunn KM, Lewis M, Mullis R, Main CJ, Foster NE, Hay EM. A primary care back pain screening tool: identifying patient subgroups for initial treatment. Arthritis Care Res. 2008;59:632–41. http://onlinelibrary.wiley.com/doi/10.1002/art.23563/full. Accessed 29 Jul 2013.
67. Hill JC, Whitehurst DG, Lewis M, Bryan S, Dunn KM, Foster NE, Konstantinou K, Main CJ, Mason E, Somerville S, Sowden G, Vohora K, Hay EM. Comparison of stratified primary care management for low back pain with current best practice (STarT Back): a randomised controlled trial. Lancet. 2011;378:1560–71. http://www.ncbi.nlm.nih.gov/pubmed/21963002. Accessed 29 Jul 2013.
68. Wide-ranging online data for epidemiologic research (WONDER). Atlanta, GA: CDC, National Center for Health Statistics; 2020. Available at http://wonder.cdc.gov. https://www.cdc.gov/drugoverdose/data/index.html.

69. Bose J, Hedden SL, Lipari RN, Park-Lee E; Substance Abuse and Mental Health Services Administration. Key substance use and mental health indicators in the United States: results from the 2017 national survey on drug use and health. https://www.samhsa.gov/data/report/2017-nsduh-annual-national-report.
70. Ashburn MA, Fleisher LA. Increasing evidence for the limited role of opioids to treat chronic non-cancer pain. JAMA. 2018;320(23):2427–8.
71. Zeng C, Dubreuil M, LaRochelle MR, et al. Association of tramadol with all-cause mortality among patients with osteoarthritis. JAMA. 2019;321(10):969–82. https://doi.org/10.1001/jama.2019.1347.
72. Fulton-Kehoe D, Garg RK, Turner JA, Bauer AM, Sullivan MD, Wickizer TM, Franklin GM. Opioid poisonings and opioid adverse effects in workers in Washington State. Am J Ind Med. 2013;56(12):1452–62.
73. Busse JW, Wang L, Kamaleldin M, et al. Opioids for chronic noncancer Pain: a systematic review and meta-analysis. JAMA. 2018;320(23):2448–60. https://doi.org/10.1001/jama.2018.18472.
74. Krebs EE, Gravely A, Nugent S, et al. Effect of opioid vs nonopioid medications on pain-related function in patients with chronic back pain or hip or knee osteoarthritis pain: the SPACE Randomized Clinical Trial. JAMA. 2018;319(9):872–82. https://doi.org/10.1001/jama.2018.0899.
75. Goesling J, Moser SE, Lin LA, Hassett AL, Wasserman RA, Brummett CM. Discrepancies between perceived benefit of opioids and self-reported patient outcomes. Pain Med. 2018;19(2):297–306. https://doi.org/10.1093/pm/pnw263.
76. Villavicencio AT, Nelson EL, Kantha V, Burneikiene S. Prediction based on preoperative opioid use of clinical outcomes after transforaminal lumbar interbody fusions. J Neurosurg Spine. 2017;26(2):144–9. https://doi.org/10.3171/2016.7.SPINE16284. Epub 2016 Sep 30.
77. Nguyen L-CL, Sing DC, Bozic KJ. Preoperative reduction of opioid use before total joint arthroplasty. J Arthroplasty. 2016;31(9, Supplement):282–7. , ISSN 0883-5403,. https://doi.org/10.1016/j.arth.2016.01.068.
78. Hartford LB, Van Koughnett JAM, Murphy PB, et al. Standardization of outpatient procedure (STOP) narcotics: a prospective non-inferiority study to reduce opioid use in outpatient general surgical procedures. J Am Coll Surg. 2019; https://doi.org/10.1016/j.jamcollsurg.2018.09.008.
79. Deyo RA, Hallvik SE, Hildebran C, Marino M, O'Kane N, Carson J, Van Otterloo J, Wright DA, Millet LM, Wakeland W. Use of prescription opioids before and after an operation for chronic pain (lumbar fusion surgery). Pain. 2018;159(6):1147–54. https://doi.org/10.1097/j.pain.0000000000001202.
80. Schoenfeld AJ, et al. Surgical intensity and risk factors for prolonged opioid use following spine surgery. Spine J. 17(10):S63.
81. Paulozzi LJ, Kilbourne EM, Shah NG, Nolte KB, Desai HA, Landen MG, Harvey W, Loring LD. A history of being prescribed controlled substances and risk of drug overdose death. Pain Med. 2012;13(1):87–95. https://doi.org/10.1111/j.1526-4637.2011.01260.x.
82. Cepeda MS, Fife D, Chow W, Mastrogiovanni G, Henderson SC. Opioid shopping behavior: how often, how soon, which drugs, and what payment method. J Clin Pharmacol. 2013;53:112–7. https://doi.org/10.1177/0091270012436561.
83. Soledad Cepeda M, Fife D, Yuan Y, Mastrogiovanni G. Distance traveled and frequency of interstate opioid dispensing in opioid shoppers and nonshoppers. J Pain. 2013;14(10):1158–61, ISSN 1526-5900,. https://doi.org/10.1016/j.jpain.2013.04.014.
84. Abrecht CR, Greenberg P, Song E, et al. A contemporary medicolegal analysis of implanted devices for chronic pain management. Anesth Analg. 2017;124(4):1304–10.
85. https://www.practicalpainmanagement.com/treatments/pharmacological/opioids/dea-enforcement-versus-pain-practice.
86. Abrecht CR, Brovman EY, Greenberg P, et al. A contemporary medicolegal analysis of outpatient medication management in chronic pain. Anesth Analg. 125, 5:1761, 2017–8.

87. Pollak KA, Stephens LS, Posner KL, Rathmell JP, Fitzgibbon DR, Dutton RP, Michna E, Domino KB. Trends in pain medicine liability. Anesthesiology. 2015;123(5):1133–41. https://doi.org/10.1097/ALN.0000000000000855.
88. Makhni MC, Park PJ, Jimenez J, Saifi C, Caldwell J-M, Ha A, Figueroa-Santana B, Lehman RA, Weidenbaum M. The medicolegal landscape of spine surgery: how do surgeons fare? Spine J. 2018;18(2):209–15, ISSN 1529-9430,. https://doi.org/10.1016/j.spinee.2017.06.038.
89. Sites BD, Harrison J, Herrick MD, Masaracchia MM, Beach ML, Davis MA. Prescription opioid use and satisfaction with care among adults with musculoskeletal conditions. Ann Fam Med. 2018;16:6–13. https://doi.org/10.1370/afm.2148.
90. Fenton JJ, Jerant AF, Bertakis KD, Franks P. The cost of satisfaction: a national study of patient satisfaction, health care utilization, expenditures, and mortality. Arch Intern Med. 2012;172(5):405–11. https://doi.org/10.1001/archinternmed.2011.1662.
91. Louie CE, Kelly JL, Barth RJ. Association of decreased postsurgical opioid prescribing with patients' satisfaction with surgeons. JAMA Surg. 2019;154(11):1049–54. https://doi.org/10.1001/jamasurg.2019.2875.

Index

A
Acetaminophen, 171–172
Achilles tendinopathy, 122
Acupuncture, 157, 346
Acute/chronic mechanical back pain, 115
Acute kidney injury (AKI), 388
Acute lung injury (ALI), 387
Acute normovolemic hemodilution (ANH), 688
Acute respiratory distress syndrome (ARDS), 387
Acute spine pain, 138
Addison's syndrome, 403
Adjacent-level degeneration, 525
Adjacent segment degeneration (ASD), 530
Adolescent idiopathic scoliosis (AIS), 551–553, 671
  anterior spinal surgery, 578
  antibiotic prophylaxis, 578
  brace effectiveness, 564
  brace treatment protocols, 564, 565
  clinical features, 558
  connective tissue abnormalities, 558
  curve flexibility, 566
  curve magnitude, 554, 555, 561
  deformity correction, 576, 577
  diagnosis of exclusion, 562
  fusion levels, 568–571
  genetic factors, 558
  Lenke classification system, 566, 568
  magnetic resonance imaging, 562
  neurologic factors, 557
  orthotic (brace) treatment, 563
  patient maturity, 560
  physical examination, 559
  physical therapy, 565
  plain radiography, 561
  preoperative planning, 566
  prevalence, 552
  scoliosis screening, 555
  spine mobilization, 575
  surgical treatment, 565
Advanced catheter fastening technique, 262
Aerobic exercise, 11, 14, 142
Albumin, 335
Alcohol cessation, 334
American College of Cardiology (ACC), 356
American Heart Association (AHA) guidelines, 356
American Spinal Injury Association (ASIA), 382
Amyotrophic lateral sclerosis (ALS), 477
Anemia of chronic disease, 360
Anesthesia dolorosa (AD), 430
Anesthetic intraoperative management, 683
Aneurysmal bone cysts (ABCs), 509, 510
Angiotensin converting enzyme (ACE) inhibitors, 362
Anorexia, 335
Anterior cervical discectomy and fusion (ACDF), 459
Anterior cingulate cortex (ACC), 202
Anterior cutaneous nerve entrapment syndrome (ACNES), 131
Anterolateral spinothalamic tract (STT), 414
Anterolisthesis, 76
Anticonvulsants, 176–179
Antifibrinolytic agents, 687
Aquatic exercises, 341
Arachnoiditis, 309
Arteriovenous fistulae (AVFs), 510, 511
Arteriovenous malformations (AVMs), 511
Associated abnormalities, 583
Autologous conditioned serum (ACS), 712, 715
Axial neck pain, 469, 471
Axial spondyloarthritis (axSpA), 109

© The Editor(s) (if applicable) and The Author(s), under exclusive license to Springer Nature Switzerland AG 2022

C. E. Noe (ed.), *Multidisciplinary Spine Care*,
https://doi.org/10.1007/978-3-031-04990-3

B
Babinski reflex, 41
Back pain, 749, 752
Back schools, 149
Bakody's sign, 44
Bilateral buttocks pain, 123
Biofeedback, 346
Blood-derived products
  autologous conditioned serum, 715
  platelet-rich plasma, 713–715
Blood loss, 686
Bone healing, 334
Bone mineral density (BMD), 227
Bone scintigraphy, 232
Bony spine, 35
Brace treatment, 564
Bracing, 156, 406, 407, 614
Brown-Sequard syndrome, 304
Burst fracture, 519

C
Calcitonin, 236
Capsaicin, 187–188
Cardiac arrest, 384
Cardiac arrhythmias, 384
Carpal tunnel syndrome, 741
Carpometacarpal (CMC) joint, 25
Casting, 614, 615
Catheter placement, 275, 276, 293
Catheter tape down technique, 268–270
Cauda equina syndrome (CES), 502
Cellular products, 715, 716
  embryonic stem cells, 717
  non-embryonic stem cells, 717, 718
Center sacral vertical line (CSVL), 569
Cerebral palsy (CP), 674
Cerebrospinal fluid (CSF), 500
Cervical disc replacement (CDR), 476
Cervical disk disease, 313
Cervical dystonia, 127
Cervical manipulation, 472
Cervical neural flossing, 298–300
Cervical radiculopathy, 473
Cervical spine, 469–471, 474, 475, 479, 517, 518, 544, 545
Cervical spine injury classification severity score (CSISS), 518
Cervical Spine Outcomes Questionnaire (CSOQ), 545
Cervical spondylotic myelopathy (CSM), 477, 478
Chemical shift imaging (CSI), 100
Chronic low back pain (CLBP), 18, 200
Cobb method, 561
Cognitive behavior therapy (CBT), 204, 206, 345, 346
Combined physical therapy, 206
Complex regional pain syndrome (CRPS), 424
Complex repetitive discharges (CRDs), 60
Compound muscle action potential (CMAP), 58
Computed tomography (CT), 85–88
Congenital kyphosis, 588
  classification, 588, 589
  clinical features, 589
  radiographic findings, 590
  treatment, 590, 591
Congenital scoliosis, 597, 598, 600
  classification, 583–585
  natural history, 585, 586
  nonoperative treatment, 587
  operative treatment, 587, 588
  radiographic findings, 586
Congenital spinal deformities, 582
Congestive heart failure (CHF), 345
Contrast-enhanced MRI studies, 231
Contrast injection, 264
Coordination, 736
Coronary artery bypass grafting (CABG), 345
Coronary artery disease (CAD), 357
Corticosteroids, 182–183
COX-inhibitors, 172–174
Cryotherapy, 153
Cutaway diagram, 295
Cyclobenzaprine, 180

D
Deep brain stimulation (DBS), 414
Deep venous thrombosis (DVT) prophylaxis protocols, 406
Degenerative spondylolisthesis, 4
Delirium, 383
De Quervain disease, 122
Dexmedetomidine, 373
Diffuse idiopathic skeletal hyperostosis (DISH), 115, 479
Diffusion non-Tensor Imaging (q-space), 101
Diffusion tensor imaging (DTI), 97, 101, 102
Diffusion-weighted imaging (DWI), 97
Digital video fluoroscopy (DVF), 79, 82, 83, 85
Disability-adjusted life years (DALY), 75
Disc degeneration, 470
  epidemiology, 490
  imaging findings, 491
  initial management, 491

pathophysiology, 490
physical exam findings, 490
surgical management, 491
Disc dessication, 88
Disc herniation, 469, 474
Discography, 85
Discoligamentous complex (DLC), 519
Disease modifying antirheumatic drugs (DMARDs), 117, 480
Disease-specific measures
cervical spine, 544, 545
thoracolumbar spine, 546
Distraction injury, 519
Dorsal approaches, 487
Dorsal Root Entry Zone (DREZ) lesioning, 416, 417
Dorsal root ganglionectomy (DRGectomy), 419
Dorsolateral approaches, 488
Down syndrome, 678
Dual-energy x-ray absorptiometry (DEXA), 535
Duchenne's Muscular Dystrophy (DMD), 676
Duke Activity Status Index (DASI), 356
Dupuytren contracture, 122
Dural laceration/puncture, 292
Dural tug maneuver, 251
Dynamic fluoroscopy, 81
Dysphagia, 389
Dystrophic scoliosis, 608, 609, 611

E

Early onset scoliosis (EOS), 612, 671
Electrical muscle stimulation (EMS), 154, 565
Electroencephalogram (EEG), 19
Electrolyte disorders, 388
Electromyography (EMG), 474, 692
clinical outcomes, 69
limitations of, 64–67
lumbar spinal stenosis, 68
MUAP, 61
radiculopathy, 62–64
timing of findings, 67–68
Electronic medical records, 751
Elevated arm stress test (EAST), 740
Embryonic stem cells (ESCs), 717
Endoscopic lumbar spine, 440
Endoscopic posterior cervical foraminotomy, 448, 449
Endoscopic spine surgery, 450
endoscopic lumbar spine, 440
endoscopic posterior cervical foraminotomy, 448, 449
endoscopic thoracic discectomy, 449–450
history, 439–440
interlaminar approach, 443–445
TLIF, 446, 447
transforaminal approach
lumbar perineural cyst, 442
outcomes, 441
PTED *vs*. microdiscectomy, 441–442
re-herniation, 441
technique, 440–441
Endoscopic thoracic discectomy, 449–450
Enhanced perioperative care (EPOC) pathway, 372
Enhanced Recovery After Surgery (ERAS) approach, 336, 542, 754
Enhanced Recovery Protocol (ERP), 728
Epidermal growth factor (EGF), 714
Epidural catheter, 723
Epidural infusion, 724, 725
Epidural lysis of adhesions
advanced catheter fastening technique, 259–263, 265
applied anatomy, 253
catheter injections, 283–286
catheter tape down technique, 267–270, 296
caudal approach, 255–259
cervical injections, 296–297
cervical interlaminar epidural catheter placement, 286–287
complications, 305–307
dural flossing exercises, 303
flexion rotation and chin to shoulder maneuver, 287–296
hypertonic saline, 252
indications, 254–255
informed consent, 305
lumbar transforaminal catheter placement, 270–277
medicolegal concerns, 303–304
neural flossing exercises, 298–303
prevention of complications, 307–312
recent modifications, 251
sacral foraminal catheter placement, 277–283
site-specific catheter placement, 252
techniques, 255
trainees, 307
Epidural steroid injections, 69
Epidurography, 255
Epimed's Stingray™ Connector, 276
Erythropoiesis-stimulating agent (ESA), 360
European Quality of Life five-dimension (EuroQoL-5D), 543

Exercise tolerance, 333
Extra-articular manifestations, 111

F
Facet arthropathy, 49–50, 476
Facet loading, 45
Failed back surgery syndrome (FBSS), 424
Femoral stretch, 303
Fibroblast growth factor (FGF), 714
Fibromyalgia syndrome (FMS), 125
First thoracic nerve root pain, 742
Flatback syndrome, 528
Flexion Abduction External Rotation (FABER) test, 46
Flexion-extension radiographs, 81
Fluoroscopy, 81
Fluoxetine, 175
Forced vital capacity (FVC), 676
F wave, 58

G
Gabapentinoids, 369
Gadolinium based contrast agents (GBCA), 74
Gaenslen's test, 45
Gastrointestinal system, 389–390
General adaptation syndrome, 22
Gluteal bursitis, 129
Gluteus maximus, 121
Glycosaminoglycans (GAGs), 470
Goniometry, 40
Greater trochanteric pain syndrome, 128, 129
Growing rod instrumenatinon (GRI), 617, 618

H
Heart failure, 358
Hematologic system, 390–391
Hemivertebrae, 669
Hemivertebra excision, 588
Hepatocyte growth factor (HGF), 714
High-intensity training (HIT), 14
Hoffmann's sign, 41
Hybrid SPECT, 102
Hyperglycemia, 390
Hypertonic saline, 311
Hypophysectomy, 420
Hypothermia, 371

I
Idiopathic scoliosis, 550, 668, 669
Image-guided surgery (IGS), 462, 463
Imaging modalities, 392
Infantile idiopathic scoliosis (IIS), 581
Inflammatory bowel disease, 111
Insulin-like growth factor (IGF), 714
Intensive care unit (ICU)
  cardiovascular system, 383–385
  delirium, 383
  gastrointestinal system, 389–390
  hematologic system, 390–391
  infectious disease considerations, 391–393
  mobility, therapy, and rehabilitation, 383
  neurological exam, 381–382
  pain management, 382
  renal system, 387–389
  respiratory system, 385–387
Interdisciplinary pain management
  biologically adaptive function, 200
  biopsychosocial approach, 200, 201
  interdisciplinary care, 204–206
  neuroscience of pain, 201–202
  psychological impact, 202–203
  psychological treatment, 203–204
Interlaminar epidural steroid injections, 214
International Society for Advancement of Spine Surgery, 317
Interscapular and scapular pain, 122
Intervertebral disc (IVD), 15
Intraoperative cell salvage, 687
Intraoperative enhanced recovery principles
  anesthetic technique, 367–368
  ERAS protocol, 366, 367, 373
  fluid management, 368
  hypotensive anesthesia, 371
  minimization of blood loss, 372
  multimodal analgesia, 369–370
  opioid-free analgesia, 370
  perioperative vision loss, 368
  pharmacologic agents, 371
Intraoperative hypothermia, 371
Intraoperative neurophysiologic monitoring (IONM), 690
Intravenous (IV) iron therapy, 360
Introducer needle, 267
Iron therapy, 689
Ischial bursitis, 130
Ixekizumab, 117

J
JAK inhibitor, 117
Japanese Orthopedic Association Back Pain Evaluation Questionnaire (JOABPEQ), 546
Juvenile idiopathic scoliosis (JIS), 671

K
Kambin's triangle, 440
Kemp's test, 45
Ketamine, 188, 369, 373
Knee meniscus tears, 745
Kyphoplasty, 241, 461
Kyphosis, 600–603, 678, 679
Kyphotic alignment, 470

L
Laminectomy, 478, 503
Laminoplasty, 478
Lateral and medial epicondylitis, 122
Lidocaine, 373
Lifestyle management
  aerobic exercise, 14
  anabolic window, 15
  cervical vertebrae, 1
  chronic LBP patients, 11, 12
  degenerative disc disease, 3
  diarthrodial zygapophyseal joint, 4
  effects from nutrition, 15–19
  effects of overuse, 23–25
  effects of sleep, 19–22
  effects of stress, 22–23
  exercise, 10–15
  intervertebral disc, 3
  lumbar disc herniation, 3, 4
  lumbar strain, 5
  lumbar vertebrae, 2
  MDT method, 12
  posture affects, 6–10
  sinuvertebral nerves, 1
  thoracic vertebrae, 3
Ligament ossification
  epidemiology, 494
  imaging findings, 495
  initial management, 495
  pathophysiology, 493
  physical exam findings, 494
  surgical management, 495, 496
Ligamentous complex, 470
Local anesthesia, 121
Local anesthetics, 183–184, 309
Low back pain (LBP), 6, 35, 211, 502, 546
Lower instrumented vertebrae (LIV), 569
Lumbar bracing, 407
Lumbar discectomies, 330
Lumbar disc herniation, 3
Lumbar interlaminar epidural steroid injections, 212–214
Lumbarization, 36
Lumbar multifidus muscles, 127
Lumbar neural flossing, 300, 301
Lumbar osteoarthritis (OA), 18
Lumbar radiculitis, 743
Lumbar segmental instability (LSI), 76
Lumbar spinal stenosis, 68, 743
  anatomy, 500
  classification, 499
  clinical presentation, 501, 502
  epidemiology, 501
  management options, 502, 503
  pathophysiology, 500
Lumbar spine instability, 742
Lumbar strain, 5
Lumbar vertebrae, 2
Lumbosacral spine, 36
Lumbosacral transitional vertebra (LSTV), 75

M
Machine learning, 751
MacNab criteria, 448
Magnetically controlled growing rod (MCGR), 618, 623
Magnetic resonance imaging (MRI), 88–97, 230
Major adverse cardiac events (MACEs), 356, 384
Major spine surgery, 723
  benefits, 724
  enhanced recovery protocols, 728
  infusion solutions, 726, 727
  post-operative period, 723
  risks, 724, 725
  surgical procedures, 726
  techniques, 727
Malabsorption syndromes, 335
Manipulation, 150
Marfan syndrome, 611, 677, 678
Massage therapy, 344
Mattress, 21
McGill Pain Questionnaire (MPQ), 542, 544
McKenzie method, 11, 12, 147–148
Mean arterial pressure (MAP), 686, 687, 693
Mechanical diagnosis and therapy (MDT), 12
Medial branch nerve blocks, 221, 222
Medial thalamotomy, 419–420
Mesencephalotomy, 420
Metabolic imbalances, 335
Mexiletine, 183
Microendoscopic discectomy (MED) system, 456
Microvascular decompression (MVD), 430
Midline myelotomy (MM), 417, 418
Midline thoracolumbar pain, 122

Mimickers, 67
Minimally invasive approaches, 489
Minimally invasive laminoforamintomy, 460
Minimally invasive spine surgery
  cervical spine surgery, 459–460
  chronic low back pain, 461
  image-guided surgery, 462, 463
  thoracic spine surgery, 458–459
  thoracolumbar spine surgery
    biplanar fluoroscopic guidance, 455
    extracted large disc fragment, 457
    intended disc space, 457
    lumbar disc disease, 455
    METRx system, 456
    operative microscope, 457
    pedicle screw fixation, 457
    tubular retractor, 457
MiniPM technique, 68
Moderate-intensity training (MIT), 14
Modified Schober test, 111
Monitoring, 683
Motor evoked potentials, 691
Motor unit action potentials (MUAP) morphology, 61
Movement control exercises (MCE), 148, 149
MR neurography (MRN), 98
MR perfusion-weighted imaging (PWI), 101
Multidisciplinary, 754
Multimodal analgesia, 369–370, 723
Multi-modal pain management, 693–696
Myelodysplasia, 669, 675, 676
Myelopathy, 4, 51
Myelopathy disability index (MDI), 545
Myocardial infarction (MI), 357, 384
Myofascial pain, 123, 124
Myofascial pain syndrome (MPS), 49

N
Neck disability index (NDI), 542
Neck pain, 144, 211, 469
Needle EMG, 59
Nerve Conduction Studies (NCS), 57
Neural mobilization (NM), 343, 344
Neuraxial anesthesia, 369
Neurogenic bowel dysfunction, 390
Neurogenic claudication, 501
Neuroleptics, 402
Neuromatrix theory, 23
Neuromuscular scoliosis, 673
Neuropathic back pain, 130
Neurosurgical management
  ablative procedures
    anterolateral cordotomy, 414–416
    DREZ lesioning, 416, 417
    DRGectomy, 419
    midline myelotomy, 417, 418
    neurotomy/neurectomy, 418
  deep brain stimulation, 421–423
  direct drug administration, 427–429
  intracranial ablative procedures, 419–421
  motor cortex stimulation, 421–423
  occipital nerve decompression, 432–433
  occipital nerve stimulation, 433–434
  occipital neuralgia, 432
  spinal cord stimulation
    anterior/posterior radiograph, 425, 427
    complications, 426
    lateral radiograph, 426, 427
    neurotransmitters, 424
    outcome, 426
    patient selection, 424–425
  trigeminal neuralgia, 429–432
N-methyl-D-aspartate (NMDA) receptor antagonists, 188–189
Nociceptive pain, 413
Nociplastic pain, 413
Non pharmacological treatment
  acupuncture, 157
  bracing, 156
  chronic neck pain, 153
  low level laser therapy, 155–156
  manipulation and modalities, 150–158
  physical therapy, 145–150
  psychological treatments and therapies, 158–161
  spinal traction modality, 155
  TENS, 154
  therapeutic exercise
    acute neck pain, 138–139
    aerobic exercise, 142
    chronic spine pain, 139–140
    exercise based exercises, 141
    low back pain, 138
    pilates, 143
    prevention of spine pain, 141
    Qigong, 145
    stretching exercises, 142
    Tai chi, 144–145
    walking, 142
    yoga, 143
  therapeutic modalities, 152
  thermal modalities, 153–154
  ultrasound therapy, 156
Nondystrophic scoliosis, 608
Non-radiographic axial spondyloarthritis (nr-axSpA), 109
Nonsteroidal anti-inflammatory drugs (NSAIDs), 47, 117, 382, 402
Normothermia, 688

Notalgia paresthetica, 127
Numerical Rating Scale (NRS), 37, 543
Nutrition optimization, 335

O
Oblique lumbar spine anatomy, 271
Obsessive-compulsive disorder (OCD), 419
Obstructive sleep apnea (OSA), 358
Occipital neuralgia (ON), 432
Occupational therapy, 206
Opioid-free analgesia, 370
Opioids, 184–187
Orthostatic hypotension, 385
Os odontoideum, 78
Osseous degenerative changes
  epidemiology, 492
  imaging findings, 493
  pathophysiology, 492
  physical exam findings, 492
Ossification of the posterior longitudinal ligament (OPLL), 493
Ossified posterior longitudinal ligament (OPLL), 479
Osteitis condensans ilii (OCI), 115
Osteophytes, 470
Oswestry disability index (ODI), 542, 546

P
Paget's disease, 123
Paraspinal muscles, 127, 128
Parkinson's disease, 477
Patellofemoral syndrome, 122
Patient Activation Measure (PAM), 322
Patient-controlled analgesia (PCA) pump, 382
Patrick's sign, 46
Peak height velocity (PHV), 553, 560
Percutaneous cementoplasty, 84
Percutaneous facet joint instrumentation, 460
Periaqueductal gray (PAG), 421
Perioperative epidural analgesia, 724
Perioperative visual loss (POVL), 685
Peripheral nerve stimulation (PNS), 414
Peripheral neuropathy, 424
Perivenous counter spread (PVCS), 275, 286
Periventricular gray (PVG) stimulation, 421
Pharmacology
  acetaminophen, 171–172
  anticonvulsants, 176–179
  antidepressants, 174–176
  anti-TNF medications, 189
  capsaicin, 187–188
  corticosteroids, 182–183
  COX-inhibitors, 172–174
  local anesthetics, 183–184
  muscle relaxers, 180–181
  NMDA receptor antagonist, 188
  opioids, 184–187
  PGE1 derivatives, 190
  prostaglandin E1 (PGE1) derivatives, 190
Physical examination, 559
Physical therapy (PT), 145–150, 472
Picture archive and communication systems (PACS), 560
Pilates, 143
Piriformis syndrome, 131–133
Plain radiography, 112
Platelet-derived (PDGF), 714
Poorly-controlled diabetes, 360
Positional MRI (pMRI)-imaging, 96
Positioning, 683, 684
Posterior cervical spine anatomy, 289
Posterior cutaneous nerve entrapment syndrome (PCNES), 131
Posterior fusion, 682
Posterior ligamentous complex injury, 521
Posterior lumbosacral anatomy, 277
Posterior neck pain, 122
Posterior spinal fusion, 587
Posterior superior iliac spine (PSIS) joint distraction, 46
Posteroligamentous complex (PLC), 521
Post-laminectomy deformities, 525
Post-operative care
  activity after discharge, 408–410
  aftercare, 401
  bracing, 406, 407
  complications, 411
  discharge, 407
  DVT prophylaxis protocols, 406
  education, 400
  emergencies after discharge, 411
  expectations, 400
  inpatient nursing, 401–406
  non-trauma spine surgery, 399
  pain management, 410–411
  poly pharmacy, 410–411
Postoperative ileus, 389
Post-operative pain management, 577
Postoperative recovery, 697
Postoperative urinary retention (POUR), 388
Post surgical spinal deformities
  adjacent segment degeneration, 530, 532
  instrumentation, 527–529
  posterior decompression, 526, 527
  proximal junctional kyphosis, 532–534
  surgical correction, 535, 536
Prehabiliation, 331
Preoperative anemia, 359

Preoperative autologous blood donation (PABD), 687
Preoperative optimization
anesthesia preoperative evaluation, 361–362
cardiovascular risk assessment, 356–358
endocrine risk assessment, 360–361
hematologic risk assessment, 359–360
informed consent, 365
preoperative testing, 365
primary care providers, 355
pulmonary risk assessment, 358–359
sample preoperative testing, 366
system-based approach, 362–365
Preoperative planning, 566
Presurgical psychological evaluation
evaluation data, 322–323
feedback session, 324–325
guidelines for, 318
medical chart review, 319–320
psychological clearance, 323–324
psychometric testing, 321
recommendations, 323–324
structured clinical interview, 320–321
Presurgical rehabilitation, 331
Progressive relaxation therapy, 346
Propofol, 373
Protocol-based blood pressure, 384
Proximal Humeral Ossification System (PHOS), 554
Proximal junctional kyphosis (PJK), 532
Pseudarthrosis, 525, 528, 529
Pseudoradiculopathy, 129
Psychometric testing, 321
Pulmonary function tests (PFTs), 359
Pulmonary hypertension, 363
Pulmonary thromboembolism, 387

Q

Qigong, 145
Quadratus lumborum muscle, 128
Quebec Back Pain Disability Scale (QBPDS), 546

R

Racz® catheter, 255, 270
Radiculopathy, 4, 51, 62–64
Radiographic axial spondyloarthritis (r-axSpA), 109
Radiographic (X-ray) imaging, 75
Range of motion (ROM), 734
Referred pain, 133
Regulatory landscape, 712
Reiter's disease, 123
Renal system, 387–389
Respiratory system, 385–387
Rheumatoid arthritis, 479
Robotic-assisted navigation (RAN), 462
Roland-Morris Disability Questionnaire (RMDQ), 546
Rotational injury, 519
Rotational thromboelastometry (ROTEM), 370
ROTEM-guided transfusion, 371
RX-Coude needle, 255, 257, 259, 261, 270, 272, 274, 279–284, 290–292

S

Sacral hiatus, 256
Sacroiliac (SI) joint
compression test, 46
distraction test, 46
injections, 222
Saddle anesthesia, 37
Schober's skin distraction test, 40
Scoliosis, 667
anesthetic intraoperative management, 683, 685, 687
anterior procedures, 682, 683
cerebral palsy, 592–597
congenital kyphosis, 588
congenital scoliosis, 583, 585, 588
Duchenne muscular dystrophy, 603–605
early-onset scoliosis, 612–614, 618, 624, 625, 672, 673
idiopathic scoliosis, 550, 552, 554, 557–561, 565, 566, 568, 574, 577–580, 668–671
infantile idiopathic scoliosis, 581
intraoperative neuromonitoring changes, 693
juvenile idiopathic scoliosis, 581
Marfan syndrome, 611, 612
neurofibromatosis, 608
neuromuscular, 673–677
non-surgical treatment, 681
posterior fusion, 682
postoperative recovery, 697, 698
screening, 555
selecting anesthetic agents, 691, 692
spinal cord monitoring, 689, 690
spinal muscular atrophy, 606, 607
syndromic scoliosis, 677, 678
Seated slump test, 45
Secukinumab, 117
Segmental spinal dysgenesis, 591, 592
Selective serotonin reuptake inhibitors (SSRIs), 174

Self-directed exercise, 140
Sensation, 735
Sensory testing, 41
Serial casting, 681
Serotonin-norepinephrine reuptake inhibitors (SNRIs), 174
Short form questionnaire, 543
Shoulder pain, 740
Simons' hypothesis, 123
Single/double inclinometry, 40
Skin puncture, 257
Smoking cessation, 333
Sodium tetradecyl sulfate (STS), 510
Soft tissue pain
  bursitis, 122
  cervical dystonia, 127
  cervical whiplash, 126–127
  differential diagnosis, 122–123
  enthesitis, 122
  FMS, 125
  gluteal bursitis, 129
  greater trochanteric pain syndrome, 128, 129
  ischial bursitis, 130
  myofascial pain, 122–124
  nerve entrapment, 131
  neuropathic back pain, 130
  non-specific low back pain, 121
  notalgia paresthetica, 127
  paraspinal muscles, 127, 128
  piriformis syndrome, 131–133
  quadratus lumborum muscle, 128
  referred pain, 133
  sacroiliac joint, 121
  tendinitis, 122
  thoracolumbar fascia, 128
  torticollis, 127
  treatment recommendations, 133
Somatosensory evoked potentials, 690
Spinal anatomy, 35
Spinal cord injury (SCI), 381, 519, 520
Spinal cord monitoring, 572
Spinal cord stimulation (SCS), 414, 423
Spinal deformity, 597
Spinal DREZ procedures, 416
Spinal dysraphism, 95
Spinal muscular atrophy (SMA), 605
Spinal stabilization, 148
Spinal stenosis, 4, 52
Spine care
  Covid 19, 758
  electronic medical records, 751
  ERAS programs, 754
  guidelines, 752
  healthcare system changes, 755
  injections, 750
  multimodal analgesia, 751
  neuroaugmentation, 751
  new models of care, 755
  opioids, 756, 757
  patient satisfaction, 758
  self management, 754
  surgical innovation, 751
Spine imaging
  chemical shift imaging, 100
  history of, 73–74
  imaging evaluation
    CT, 85, 86, 88
    DVF, 79, 82, 83, 85
    MRI, 88–91, 93, 94, 96, 97
    radiographic evaluation, 75–79
  morbidity, 75
  MRN, 98
  PWI, 101
  VENC, 99
Spine injections
  cervical epidural steroid injections
    cervical interlaminar approach, 217
    selective nerve root blocks, 218–219
  epidural steroid injections
    caudal approach, 214–216
    interlaminar epidural injection, 213
    lumbar epidural space, 212, 214
    mechanism of action, 212
  facet joint injections, 220–222
  medial branch nerve blocks, 220–222
  sacroiliac joint injections, 222
Spine patient evaluation
  arthropathy, 49–50
  bony spine, 35
  central and peripheral components, 36
  discogenic pain, 51
  lumbosacral spine, 36
  myelopathy, 51
  myotendinous pain, 49
  patient history, 37
  physical examination
    functional testing, 42
    inspection, 39
    neurological testing, 40–42
    palpation, 39
    range of motion, 39
  radiculopathy, 51
  red flag symptoms, 37–38
  special maneuvers and provocative tests
    cervical distraction, 43
    lumbosacral region, 44–46
    nonspecific spine pain, 47
    radiculopathy and spinal stenosis, 47–48

Spine patient evaluation (*cont.*)
specific diagnosis, 48
Spurling test, 43
ULTT, 44
spinal stenosis, 52
thoracic spine, 36
vertebral fractures, 52
Spine prehabilitation program
alcohol cessation, 334
aquatic exercises, 341
bone healing, 334
cardiovascular conditioning, 333
centralization of pain, 336
early mobilization, 339, 340
epidemiological considerations, 329–330
ERAS framework, 336–338
manual therapy, 344
mind body exercises, 346–347
neural mobilization, 343, 344
nutrition optimization, 335
patient education, 331, 332
postoperative cardiovascular exercise, 340–341
postoperative rehabilitation, 338–339
preoperative spine patient, 330–331
psychologic intervention, 336
psychosocial considerations, 345–346
reducing fall risk, 333
smoking cessation, 333
soft-tissue mobilization, 344–345
strength training, 341–343
timing and frequency, 340
Spondyloarthritides
algorithm, 113, 115
clinical manifestations, 110, 111
differential diagnosis, 115–116
imaging of enthesitis, 113
lab testing, 112
MRI findings, 112
nonpharmacologic treatment, 116–117
pharmacologic treatment, 117–118
physical exam, 111
plain radiography, 112
treatment options, 116–118
Spondyloarthritis International Society (ASAS), 109
Spondylolisthesis, 679
Spondylolysis, 679
Spondyloptosis, 76
Spondylosis, 469, 471
Spondylotic myelopathy, 469
Spurling test, 43
Standing lumbar maneuver, 302
Stereotactic radiosurgery (SRS), 420, 432
Steroid injections, 472
Straight leg raise, 44
Stretching exercises, 142
Stromal vascular fraction (SVF), 718
Subacromial bursitis, 122
Subscapularis tears, 740
Syndromic scoliosis, 677
cardiac evaluation, 680, 681
Down syndrome, 678
Marfan syndrome, 677, 678
respiratory evaluation, 680
Systemic glucocorticoids, 118

T
Tai Chi, 11, 144–145, 346
Team medicine, 754
Tegaderm, 405, 409
Telemedicine, 731
carpal tunnel syndrome, 741
cervical myelopathy, 739
cervical pain, 739
coordination, 736
cranial nerves, 734
gait and station, 737, 738
gluteus medial muscle tears, 744
hip pain, 744
inspection, painful area, 733
lumbar spine instability, 742
lumbar stenosis, 743
opioid withdrawal screen, 733
patellofemoral syndrome, 745
physical exam, 732
psychiatric status screen, 733
range of motion, 734
rotator cuff, 740
sacroiliac pain, 744
sensation, 735
subacromial impingement, 740
subscapularis tears, 740
visualization, 732
vital signs, 732
Therapeutic massage, 151
Thermotherapy, 153
Thigh thrust, 46
Thoracic insufficiency syndrome (TIS), 672
Thoracic spine, 36
anatomy, 485, 486
biomechanics, 486
degenerative diseases, 490, 491
dorsal approaches, 488
dorsolateral approaches, 489
ligament ossification, 493–496
minimally invasive approaches, 489

osseous degenerative changes, 492, 493
surgery, 458–459
ventral approaches, 488
Thoracic vertebrae, 3
Thoracolumbar fascia, 128
Thoracolumbar injury classification and severity score (TLICS), 519, 521
Thoracolumbar spine, 519, 546
Thoracolumbosacral orthoses (TLSOs), 563
Thromboelastogram (TEG), 674
Thromboprophylaxis, 391
Tofacinitib, 117
Topical ketamine, 189
Torticollis, 127
Total intravenous anesthesia (TIVA), 372, 572
Traction, 616, 617
Traditional growing rod (TGR), 672
Tranexamic acid (TXA), 687
Transcutaneous electrical nerve stimulation (TENS), 47, 124, 154
Transforaminal lumbar interbody fusion (TLIF), 446, 447
Transforming growth factor beta (TGF-B), 714
Transfusion-related acute lung injury, 387
Transfusion threshold, 689
Transverse abdominis (TA), 342
Treadmill, 15
Tricyclic antidepressants (TCAs), 174
Trigeminal neuralgia (TN), 429–432
Tropism, 5
Tumor necrosis factor (TNF) alpha inhibitor, 117
Tumor necrosis factor (TNF) inhibitors, 189–190

U
Ultrasound therapy (US), 156
Unilateral buttock pain, 123
Upper end vertebrae (UEV), 569
Upper limb tension test (ULTT), 44
Urinary retention, 388–389
US National Health Interview Survey (NHIS), 12

V
Valsalva maneuver, 44
Valveless venous system, 372
Valvular heart disease, 358
Vascular disorders, 94
Vascular endothelial growth factor (VEGF), 714
Venous thromboembolic (VTE) disease, 383
Ventral approaches, 488
Ventrolateral epidural space, 264
Vertebral column resection, 588
Vertebral compression fractures (VCFs), 52, 115
directed exercise, 238
epidemiology, 227–228
evaluation, 229–231
functional impairments, 227
global effects of, 228–229
medical co-morbidities, 227
medications for pain control, 234, 236
physical therapy, 238
preventative medicine, 240
risk factors, 228
vertebral augmentation, 243–245
Vertebral hemangiomas, 507–509
Vertebroplasty, 84, 241, 461
Vertically expandable prosthetic titanium rib (VEPTR)., 623, 624, 672
Video-assisted thoracoscopic surgery (VATS), 459, 491
Visual Analog Scale (VAS), 37, 542, 543
Visualization, 732

W
Walking, 142
Whiplash-Associated Disorder (WAD), 126
Williams Flexion Exercise (WFE)/Williams Lumbar Flexion Exercise, 149

X
X ray, 73

Y
Yoga, 11–13, 140, 143, 346