Orthopaedic Knowledge Update®

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Spine

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Preface

Welcome to the sixth edition of *Orthopaedic Knowledge Update®: Spine*. Here, we sought to maintain the tradition of excellence fostered by previous editors, learn from and expand on our experience editing the *OKU®: Spine 5*, and provide readers the latest developments from the rapidly evolving world of spine care. As we noted with *OKU®: Spine 5*, the pace of change and the explosion of new data sources, both geographically and in terms of the numbers of journals covering spine-related topics, have continued their logarithmic pace.

For both learners and specialists working to maintain up-to-date knowledge, the challenge is not finding information, but rather how to sift through and prioritize the mountains of data available. As editors, our job was to assemble a great team of field experts. Currently, optimal spine care requires an interdisciplinary approach with invaluable input from our colleagues in physical medicine and rehabilitation, anesthesia, radiology, neurology, neurosurgery, rheumatology, and internal medicine. We sought to reflect that spectrum of caregivers in our selection of contributors. The more than 90 authors not only represent various disciplines and subspecialty interests but are also diverse in background and geography.

We are indebted to the section editors (each is a recognized expert in the field) for helping select topics and authors and for shepherding those chapters through to completion: Christopher Chaput, Charles H. Cho, Harold A. Fogel, Mitchel B. Harris, Scott R. Laker, Ronald A. Lehman, Charles A. Reitman, Andrew J. Schoenfeld, and Jeffrey C. Wang. Of course, we would also like to thank the authors, many of whom reprised and updated their work from *OKU®: Spine 5*.

The tried-and-true OKU® format allowed us to organize this information with an ultimate goal of answering the questions: "Where are we in spine care?" and "Where are we going?" Most topics begin with a review of critical background information, followed by an update of the past 5 years' literature. Each chapter offers an annotated bibliography to guide readers' further exploration of a topic. Although each chapter stands on its own, the book is also organized with a logic that allows it to be read cover to cover or section by section.

Our thanks go to the project management and editorial teams at Wolters Kluwer (Brian Brown, Stacey Sebring, Emily Buccieri, and Sean Hanrahan) and AAOS (Anna Salt Troise, Hans Koelsch, Lisa Claxton Moore, and Steven Kellert). To ensure timeliness, this book had very tight deadlines. The staff were instrumental in moving the project forward. We would also like to thank the AAOS for the honor of editing another edition of *OKU®: Spine*. We must also acknowledge our practice and life partners. We thank them for their patience with our many volunteer efforts and the time invested in this book.

Eeric Truumees, MD, FAAOS
Heidi Prather, DO

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SECTION 1

Spine Anatomy and Biomechanics

Section Editor:

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CHAPTER 1

Musculoskeletal Anatomy and Physiology of the Spine

ISAAC L. MOSS, MDCM, MASc, FRCSC • SCOTT S. MALLOZZI, MD

ABSTRACT

The vertebral column is a complex three-dimensional structure whose function in health and disease is determined by the anatomy and physiology of the spine and its supporting structures, including the vertebrae, the disks, and the intimate connections with the surrounding soft tissues. To understand, diagnose, and safely treat patients with spinal pathology, it is helpful for surgeons to review the basic anatomy of the spine and be aware of recent developments in understanding how the anatomy of the vertebrae and the surrounding tissues affect function.

Keywords: anatomy; applied anatomy; intervertebral disks; vertebrae

INTRODUCTION

A detailed knowledge of spine anatomy is a prerequisite for safe and effective nonsurgical and surgical treatment of patients with spine pathology. The vertebrae, intervertebral disks, and surrounding ligaments and muscles are important determinants of spinal function, both in health and disease. The evolving body of knowledge on spine anatomy, function, and the complex interactions between the various elements that make up the spine allows a deeper understanding of the pathogenesis of disease and the potential development of future novel treatments.

BASIC ANATOMY

The spinal column consists of 24 vertebral segments. Except for the first cervical level, all individual vertebrae share similar basic morphologic characteristics, including a vertebral body, pedicles, a lamina, and a variety of bony projections that serve as attachments for ligaments and muscles. The mobile spine is traditionally divided into 3 regions consisting of 7 cervical vertebrae, 12 thoracic vertebrae, and 5 lumbar vertebrae. The sacrum consists of five fused vertebrae, with no motion between the vertebrae. Despite important similarities, substantial anatomic variation exists between the vertebrae of each region, with the vertebrae being adapted to the varying functional demands throughout the spine. A thorough understanding of these variations is essential for the safe and effective management of spinal pathology.

The functional spinal unit consists of two adjacent vertebrae and their intervening intervertebral disk and facet joints. The facet joints are true synovial joints with characteristics similar to those of other synovial articulations in the body. The intervertebral disk, however, is the major load-bearing structure of the spine and has unique characteristics. Each intervertebral disk is composed of an inner gelatinous nucleus pulposus consisting primarily of type II collagen and proteoglycans and surrounded by a highly organized collagenous anulus fibrosus, which primarily consists of type I collagen in concentric lamellae, with fibers lying in alternating directions (**Figure 1**). These components are confined cranially and caudally by the vertebral end plates, resulting in a confined hydraulic system with biphasic viscoelastic

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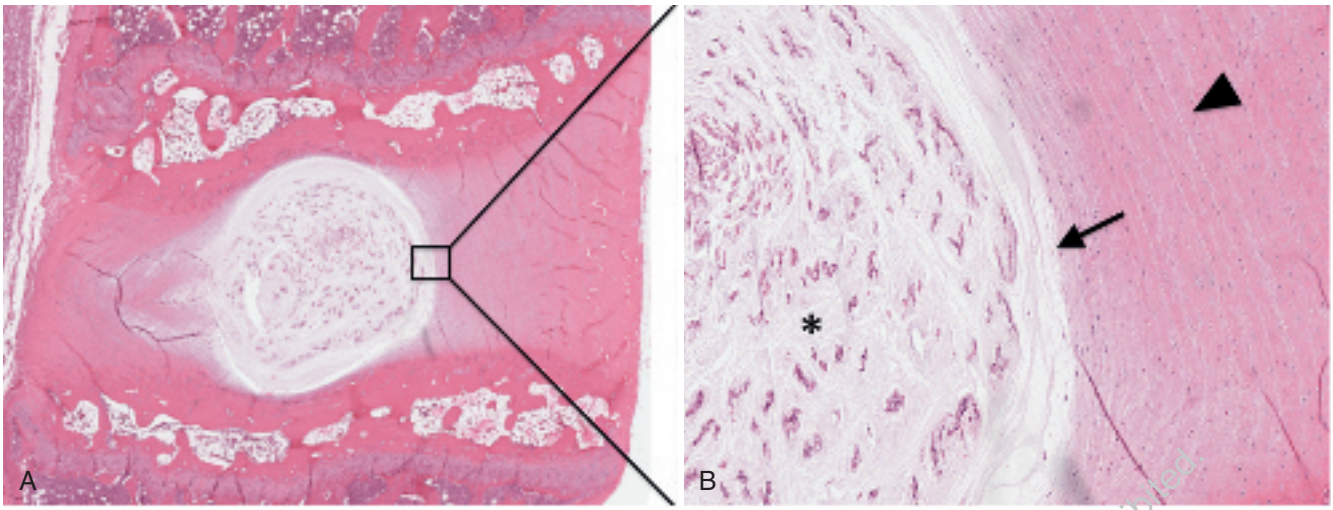

FIGURE 1 Hematoxylin-eosin–stained histologic sections of an intervertebral disk at low (**A**) and high (**B**) power. The nucleus pulposus (NP) (*) is populated by clusters of cells within a gelatinous matrix. A clear border (arrow) between the NP and anulus fibrosus (AF) is evident. The AF demonstrates organized fibrocartilage lamellae (arrowhead). (Reprinted from Moss IL, An HS: Form and function of the intervertebral disc, in O'Keefe RJ, Jacobs JJ, Chu CR, Einhorn TA, eds: *Orthopaedic Basic Science: Foundation of Clinical Practice*, ed 4. Rosemont, IL, American Academy of Orthopaedic Surgeons, 2013.)

biomechanical properties capable of withstanding considerable compressive loads.

Discogenic pain resulting from degeneration and disk herniation is mediated by proinflammatory cytokines at the systemic and local levels. The authors of a 2016 study reported that interleukin (IL)-1β, IL-6, IL-10, and tumor necrosis factor alpha play a particularly important role in the acute period of disk herniations and could be targets for future therapy.[1] A recent microstructural analysis demonstrated evidence of structural connectivity across the cartilaginous–end plate junction secondary to the intermingling of fibrillar components. This may be the underlying basis of osteocartilaginous disk herniations seen clinically.[2]

Ligaments

The spine is stabilized by several ligamentous structures. The anterior longitudinal ligament (ALL) is found on the ventral aspect of the vertebral body, extending from the skull to the sacrum. The ALL has several layers, with its deepest and strongest attachments being to the articular lip at the margins of each vertebra and its more superficial layers spanning multiple vertebrae. The posterior longitudinal ligament (PLL) also spans from the skull to the sacrum, but runs within the spinal canal on the dorsal aspect of the vertebral body. Unlike the ALL, the PLL has attachments only at the disk level, and it is bowstrung across the concavity of the vertebral bodies. The PLL can be elevated by pathologic processes, including disk herniations, trauma, hematomas, infections, and tumors. The location of the PLL reinforces the central anulus fibrosus, with most posterior disk herniations occurring at the lateral margin of the PLL. The ALL is innervated by the recurrent branches of the rami communicantes, whereas the PLL is innervated by the sinuvertebral nerves. These are branches from the spinal nerves in proximity to the origin of the anterior and posterior rami, and therefore may contribute to back pain.

The ligamentum flavum is an important anatomic structure to consider during surgical decompression because it is a major contributor to spinal canal stenosis. In contrast with the ALL and PLL, the ligamentum flavum is a noncontiguous structure, with attachments to the ventral surface of the cranial lamina and superior surface of the caudal lamina of each individual functional spinal unit. When entering the canal with a Kerrison rongeur or burr, surgeons often exploit the fact that the ligamentum flavum extends halfway to two-thirds up the ventral surface of the cephalad lamina because this natural anatomic barrier can help prevent inadvertent durotomy.[3]

Development

The spinal column is formed from the paraxial mesoderm in a process called somatogenesis.[4] As the body axis elongates, individual somites are added on either ventral portion of the somite, which becomes the mesenchymal sclerotome and is responsible for the formation of the vertebrae and the anulus fibrosus. The nucleus pulposus is formed from the remnant of the notochord and is populated by cells of notochordal origin in early life. These cells are subsequently replaced by cells similar to chondrocytes by the end of the first decade of life. Each

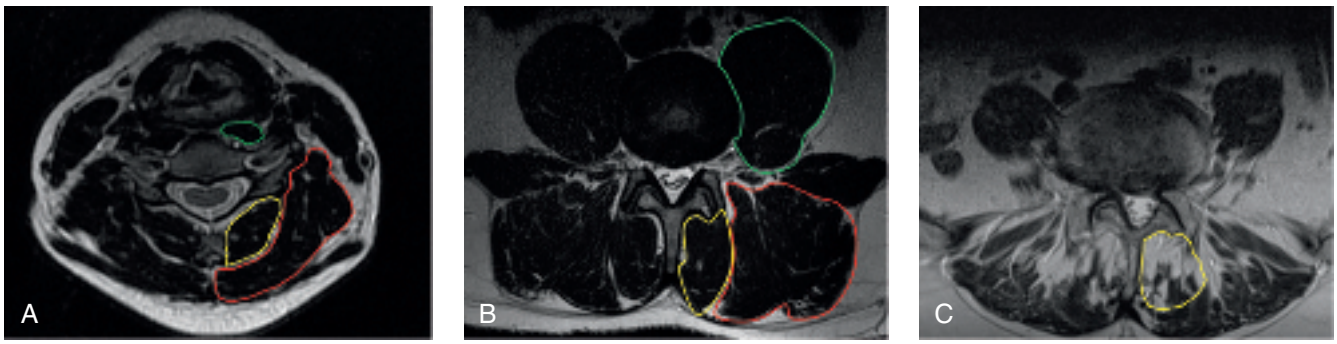

FIGURE 2 Axial T2-weighted magnetic resonance images of (**A**) normal midcervical spine musculature demonstrating the anterior flexor longus colli (green), the deep extensors semispinalis cervicis and multifidus (yellow), the superficial extensors semispinalis capitis, splenius capitis and longissimus (red); (**B**) normal lumbar spine musculature at the L4-L5 disk space demonstrating the psoas (green), the deep extensor, multifidus (yellow), and superficial erector spinae (red); and (**C**) the lumbar spine musculature after open decompression demonstrating significant fatty atrophy of the multifidus muscle (yellow).

vertebra is formed by three primary ossification centers—the centrum, neural arch, and a costal element. Failure of one or more of these ossification centers to develop can result in the formation of a hemivertebra, which often causes substantial deformity (referred to as congenital deformity).[5] Failure of the somite to fully segment results in the formation of block vertebrae or unsegmented bars. The combination of a hemivertebra and a contralateral unsegmented bar leads to the most progressive form of congenital scoliosis.

Muscles

The paraspinal musculature plays an important role in stabilizing the spine and maintaining upright posture. In the cervical spine, the paraspinal muscles are divided into deep and superficial groups, with the deep musculature mainly responsible for spinal stability and the superficial musculature mainly responsible for movement (**Figure 2**). An increased cross-sectional area in the deep cervical extensor muscles is associated with a higher rate of bony union after anterior cervical fusion.[6] In the thoracolumbar spine, the paraspinal musculature is generally divided into the deep multifidus muscles and the more superficial erector spinae muscles. The multifidus is considered the major posterior stabilizing muscle of the spine. Its large cross-sectional area and sarcomere orientation allow it to generate large forces with small changes in length.[7] The multifidus muscle originates from the spinous process of a single level and typically inserts three levels caudal (on the mammillary process in the lumbar spine). At each level, the multifidus is innervated by the medial branch nerve of the posterior ramus of the spinal nerve, which exits the spinal canal superolateral to the facet joint. Multifidus atrophy is seen after traditional open approaches to the spine and results from a combination of denervation, thermal injury, and pressure necrosis caused by prolonged retraction.[7] Medial branch nerve ablations, which are often performed in the management of back pain, may lead to multifidus atrophy as well.[8]

Recently, the health and function of the paraspinal musculature has been investigated as it relates to back pain and surgical success.[9-11] Paraspinal muscle atrophy and fatty infiltration, most prominently affecting the multifidus, has been associated with chronic low back pain; however, it is unclear if this change is causative or related to disuse in patients with long-term pain.[10] Paraspinal atrophy and fatty infiltration also have been associated with an increased risk of adjacent-segment degeneration after lumbar fusion.[11] Many minimally invasive approaches to the lumbar spine have been designed to preserve the medial branch nerve and minimize trauma to the multifidus.[9]

Spinal Balance

Positioning of the C7 vertebrae over the sacrum is essential for the maintenance of upright posture and efficient locomotion. Proper positioning is achieved by balancing the curvatures of the various anatomic regions of the spine, including lordosis of approximately 60° in the lumbar region and approximately 20° in the cervical region, and kyphosis of approximately 40° in the thoracic and sacral regions[12] (**Figure 3**).

The sagittal vertical axis (SVA) is measured as the distance between the posterior corner of the S1 superior end plate and a vertical plumb line from the midpoint of the C7 vertebral body. An increase in the SVA is linearly correlated with more pronounced symptoms and disability.[13] Similarly, an increase in the C2-C7 SVA, measured as a plumb line from mid-C2 vertebral body to the posterior corner of the superior end plate of C2 (C2-C7 SVA), has been correlated with increased functional disability[14] (**Figure 4**). Lumbar lordosis is not evenly distributed, with two-thirds of overall lumbar lordosis contributed

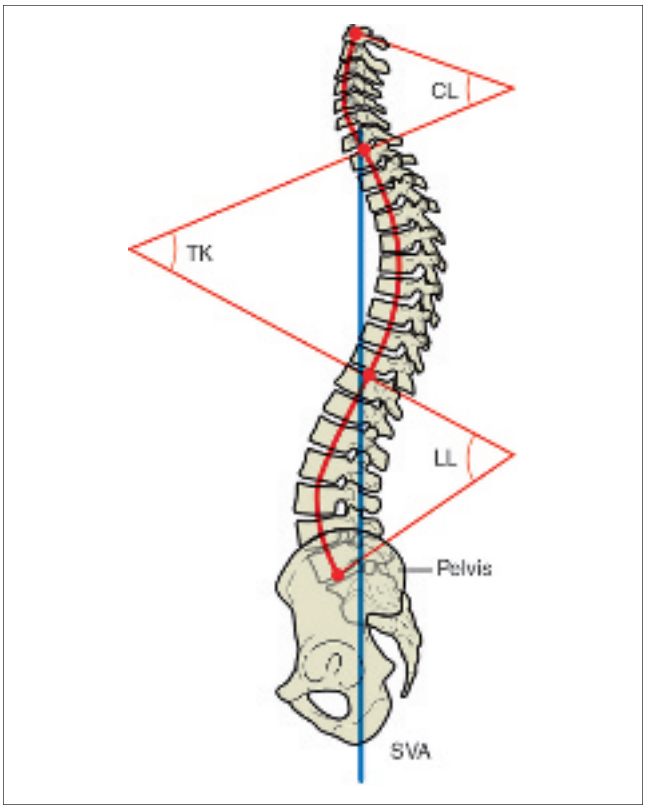

FIGURE 3 Illustration demonstrating normal global sagittal balance as measured by the sagittal vertical axis (SVA), a line drawn vertically from the center of the C7 vertebral body, and this is due to a balanced combination of cervical lordosis (CL), thoracic kyphosis (TK), and lumbar lordosis (LL). (Adapted with permission from Berthonnaud E, Dimnet J, Roussouly P, Labelle H: Analysis of the sagittal balance of the spine and pelvis using shape and orientation parameters. *J Spinal Disord Tech* 2005;18:40-47.)

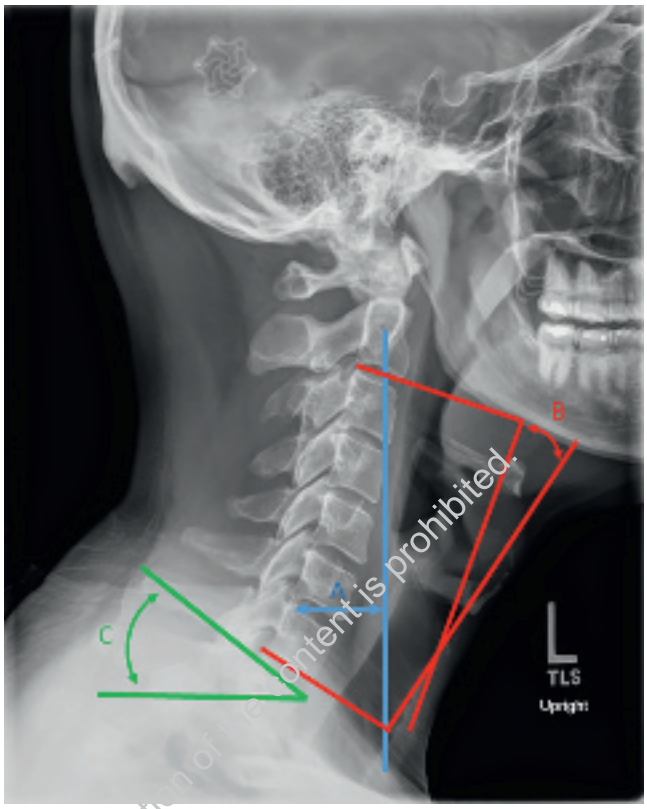

FIGURE 4 Lateral digital radiograph demonstrating parameters used to assess cervical spine sagittal alignment including C2-C7 sagittal vertical axis (A), cervical lordosis (B), and T1 slope (C).

by L4-S1. Optimal lumbar lordosis is closely related to an individual's pelvic incidence, which is an important anatomic parameter specific to each individual to consider when planning corrective surgery for spine deformity. For the cervical spine, the T1 slope minus cervical lordosis (TS-CL) has been proposed in a recent study as a measurement to quantify the severity of cervical sagittal deformity.[15] Additionally, evidence from a 2015 study has shown that an individual's cervical lordosis is related to his or her cranial incidence, an anatomic parameter specific to an individual's skull[16] (**Figure 5**). As demonstrated in a 2017 study, variation occurs in both sagittal balance and pelvic parameters as a result of shifting from a standing to a sitting position, with a reduction in both lumbar lordosis and thoracic kyphosis and a forward shift in the SVA.[17] The relevance of this information when planning spine deformity correction has yet to be determined. With aging, the regional curvatures often change, often with an increase in thoracic kyphosis and decrease in cervical and lumbar lordosis. However, asymptomatic individuals may maintain a stable global balance by compensation in other areas of the spine.[18]

APPLIED ANATOMY BY REGION

Cervical Spine

Occipitocervical Stability

The occipitocervical complex, which extends from the occiput to the C2-3 disk space, consists of specialized bony and ligamentous structures to stabilize this area of vital anatomy while also acting as the major contributor to cervical range of motion. The tectorial membrane, once thought to be the primary stabilizer of the occipitoatlantal articulation, is an extension of the PLL and runs from the anterolateral edge of the foramen magnum to the posterior surface of the C2 body and odontoid process. A 2015 study performed using modern biomechanical techniques demonstrated that the primary stabilizers of the craniocervical junction are the transverse and alar ligaments.[19]

The cruciate ligament, the key structure in atlantoaxial stability, consists of vertical and transverse

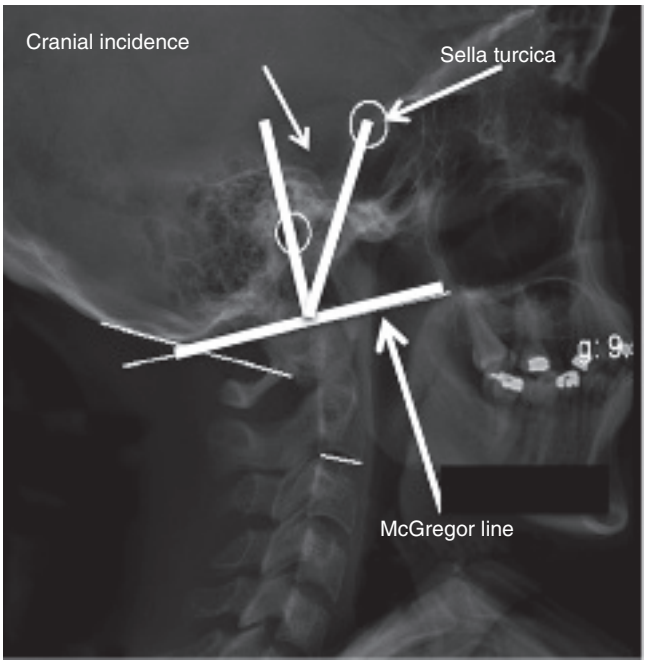

FIGURE 5 EOS image of the head and cervical spine demonstrating the cervical incidence as the angle between a line drawn perpendicular to the center of the McGregor line and a line from the sella turcica (approximate center of rotation of the skull) to the center of the McGregor line. (Reprinted with permission from Springer Nature: Le Huec JC, Demezon H, Aunoble S: Sagittal parameters of global cervical balance using EOS imaging: normative values from a prospective cohort of asymptomatic volunteers. *Eur Spine J* 2015;24[1]:63-71. Copyright 2015.)

components, which stabilize the odontoid to the occiput and atlas, respectively (**Figure 6**). Disruption of the occipitocervical complex, which can result from high-energy trauma, can lead to occipitoatlantal or atlantoaxial dissociation. The sensitivity of plain radiography to detect these often-fatal injuries has been questioned. Efforts have been undertaken to define parameters predictive of ligamentous injury based on CT and MRI, which are commonly obtained imaging studies in trauma settings. On CT, a basion-dens interval of greater than 10 mm and a C1-C2 lateral mass interval of 4 mm or greater are highly sensitive measurements for the detection of occipitocervical complex injury.[20] MRI studies have defined two patterns of occipitocervical complex injury based on the integrity of the occipitoatlantal capsular ligaments.[21] Atlantoaxial dissociation occurs when occipitoatlantal capsular ligaments are preserved, but the cruciate ligament is disrupted. In patients with combined occipitoatlantal and atlantoaxial dissociation, both the occipitoatlantal capsular ligaments and the cruciate ligaments are disrupted.[21] It may be easier to recognize a true dissociation by evaluating not only the midline structures (eg, basion-dens interval), but also the congruency and the displacement of the occiput-C1 articular surfaces.

Vertebral Artery

The foramen transversarium is a key distinguishing anatomic feature of the cervical vertebrae from C2-C7. The vertebral artery, which is a branch of the subclavian artery, usually enters the foramen of C6, runs cranially to exit at C2, and then proceeds around the lateral mass of C1 to the superior surface of the posterior C1 arch and enters the foramen magnum (**Figure 7**). Frequent variations exist in the size and position of the foramen transversarium and the artery contained within.[22] In rare instances, the artery can run through the lateral aspect of the vertebral body and entirely outside the foramen. Thus, a careful review of axial imaging studies is essential when planning cervical instrumentation. The vertebral artery can be injured when using a burr to remove the uncovertebral joint. Fibrous bands, which connect the nerve root to the vertebral artery, can tear this vessel even when the burr remains medial. The vertebral artery is most at risk for injury during the posterior instrumentation of C1 and C2. In addition, a fine-cut CT scan or CT angiogram is helpful when planning instrumentation at C1 and C2. The C2 pedicle has substantial anatomic variation in up to 18% of individuals, which can put the vertebral artery at risk for injury.[23] The Harms technique for C1-C2 fixation (with C1 lateral mass and C2 pedicle screws) has gained popularity over the Magerl transarticular screw technique because it provides greater freedom for screw trajectory and potentially reduces the risk of vertebral artery injury.[24]

Subaxial Cervical Spine

The subaxial cervical spine is most commonly instrumented from an anterior approach that takes advantage of an anatomic corridor to the spine and osseous anatomy for safe instrumentation. The cervical vertebrae and neural foramina of males are typically larger than those of females.[25] With advancing age, cervical vertebrae become wider and more elongated.[23] The average depth of the cervical vertebral bodies ranges from 15 to 17 mm and increases caudally. Subaxial cervical vertebrae have uncinate processes extending from the edges of the superior end plates, which form the lateral borders of the intervertebral disk. The uncinate processes form an important landmark for the lateral extent of anterior decompression procedures. Posteriorly, the cervical vertebrae are characterized by bifid spinous processes and large lateral masses, but not the elongated transverse processes found in the thoracic and lumbar regions. Lateral mass instrumentation is most commonly used for posterior fixation from C3 through C6 because of its technical ease and safety.[26] The starting point for these screws is 1 mm medial to the center of the lateral mass. The screws are angulated approximately 15° cephalad and 30° lateral to limit the risk of injury to the vertebral artery and

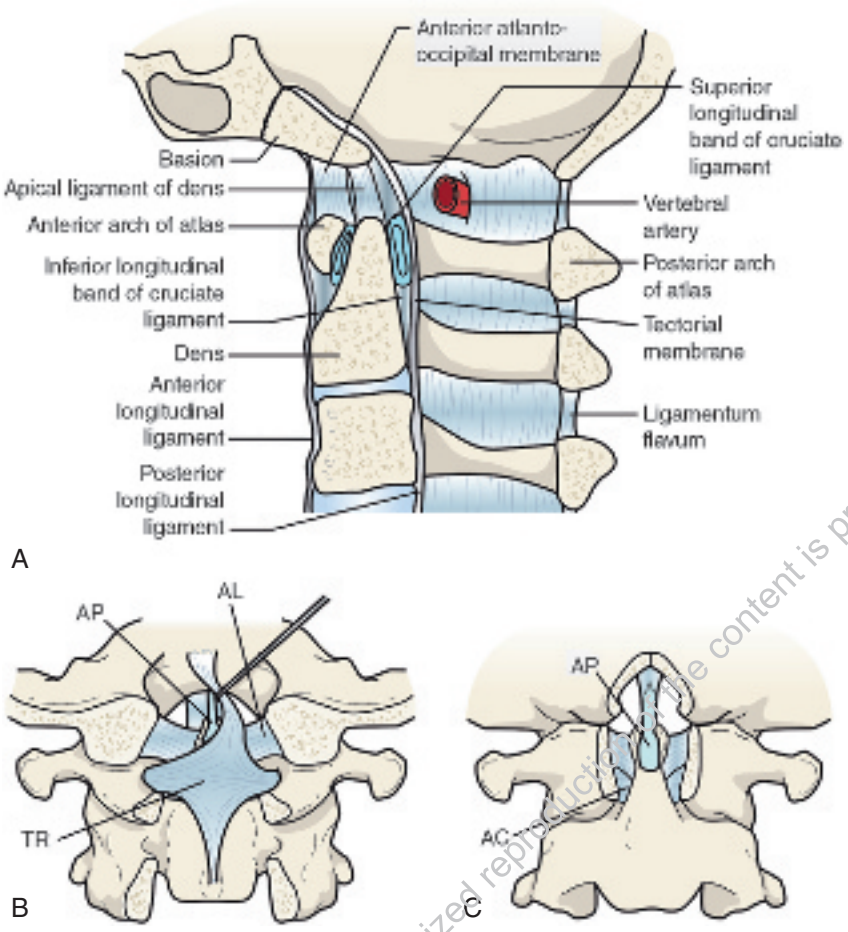

FIGURE 6 **A**, Illustration demonstrating sagittal view of the occipitocervical articulation. Posterior (**B**) and anterior (**C**) illustrations of the atlantoaxial articulation. AC = accessory ligament, AL = alar ligament, AP = apical ligament, TR = transverse atlantal ligament. (Reprinted with permission from Bransford R, Alton T, Patel A, et al: Upper cervical spine trauma. *J Am Acad Orthop Surg* 2014;22[11]:718-729.)

exiting nerve roots, although these parameters may change somewhat depending on the level instrumented and the amount of spinal degeneration.[27] Posterior instrumentation of subaxial cervical pedicles is possible; however, this is associated with a higher risk of neurologic and vascular complications compared with lateral mass fixation.[28] Many surgeons limit the use of this technique to C7, where lateral mass fixation can be poor and there is less risk of injury to the vertebral artery. The starting point for C7 pedicle screw instrumentation is the upper outer quadrant of the lateral mass. The screw trajectory angles medially 25° to 45°. A laminoforaminotomy to palpate the pedicle may improve the accuracy and safety of this procedure.

Thoracic Spine

Several unique anatomic characteristics are important to understand when assessing and treating thoracic spinal pathology. Each thoracic vertebra has a diarthrodial articulation with a rib on each side. This articulation adds to the inherent stability of the thoracic spine and is thought to be a major contributing factor to the decreased frequency of degenerative pathology in the thoracic region compared with the cervical and lumbar regions. The thoracic facet joints are oriented in the coronal plane to allow for axial rotation. The ratio of the canal to the spinal cord is relatively small in the thoracic spine, which increases the risk of neurologic injury even with small amounts of canal incursion.[29] Although the height of the pedicles generally increases from cranial to caudal progression within thoracic vertebrae, CT-based studies have demonstrated that the pedicle width decreases from T1 (9.27 ± 1.01 mm) to T4 (4.5 ± 0.93 mm), and then subsequently increases to T12 (8.31 ± 1.83 mm).[30] As a result, a substantial portion of the pedicles in the midthoracic spine is not wide enough to accommodate a 4-mm screw with

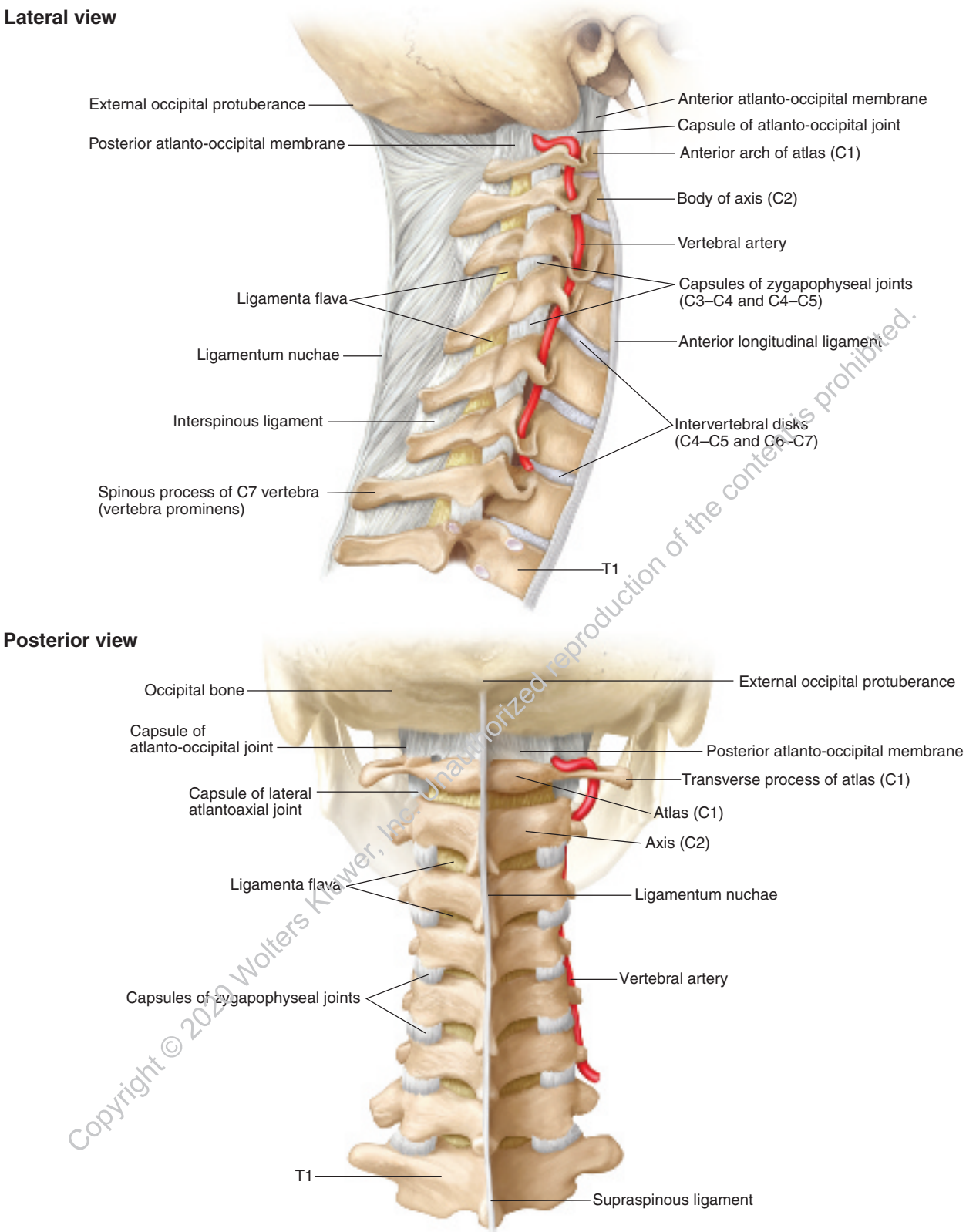

FIGURE 7 Illustration showing the relationship of the vertebral artery to the bony and neurologic anatomy of the cervical spine. (Reproduced with permission from Gest T. *Lippincott Atlas of Anatomy,* ed 2. Philadelphia, PA, Wolters Kluwer; 2019.)

1 mm of clearance, which is considered a satisfactory margin of safety. Close study of thoracic anatomy is mandatory before instrumentation in this region, and alternative techniques should be considered. Such techniques involve the use of hooks, translaminar screws, or an in-out-in trajectory in which the screw intentionally breaches the lateral pedicle wall and abuts the rib head for added stability.

Thoracic pedicles have been shown to expand in diameter to accommodate larger screws placed to maximize pullout strength.[31] Evidence from one study shows that this expansion occurs primarily in the lateral direction; thus, the diameter of the spinal canal is not diminished, and the risk of injury to the spinal cord is not increased.[32]

At T12, the entry point for pedicle screw insertions is the junction of the bisected transverse process and the lamina. Progressing cranially to the midthoracic spine, the starting point shifts to a more medial and cephalad position. In the cranial portion of the thoracic spine, the entry point is at a more cranial and lateral position[33] (**Figure 8**). Alternatively, the funnel and slide techniques can be used to identify the cancellous bone found within the pedicle in contrast to the cortical bone found on the anterior margin of the transverse process.[34] The transverse pedicle angle (TPA) decreases from cranial to caudal, requiring a more medialized trajectory in the proximal thoracic spine (TPA at T1 is approximately 35°) compared with distal vertebrae (TPA at T12 is approximately −10°).[29] Two techniques have been described with respect to sagittal plane angulation. The anatomic technique, with the screw angled caudally in line with the true pedicle anatomy, allows for placement of a longer screw but necessitates the use of a polyaxial screw, which can limit the degree of deformity correction. The straightforward approach with the screw angled in line with the end plate, as opposed to the pedicle, has gained popularity because it allows placement of a fixed-angle screw to obtain more powerful deformity correction.[35]

The position of the great vessels with respect to the spine is an important consideration, especially when instrumenting thoracic vertebrae, because injury to the vessels can have catastrophic consequences. The aorta lies on the left anterolateral aspect of the vertebrae and follows the spine caudally, dividing into the common iliac arteries at or around the L4-L5 disk level. The distance from the margin of the vertebrae to the aorta decreases from the cephalad to caudad thoracic spine (4.8 mm at T1, 1.2 mm at T12).[36] In patients with scoliosis, the relationship between the spine and aorta is altered as a result of both translation and rotation. The aorta is located more posterolateral and closer to the vertebrae at the levels above the apex of the curve. Below the apex of the curve, the aorta is located closer to the midline, which increases the risk of injury at the thoracolumbar junction in patients with Lenke type 1 curves.[37]

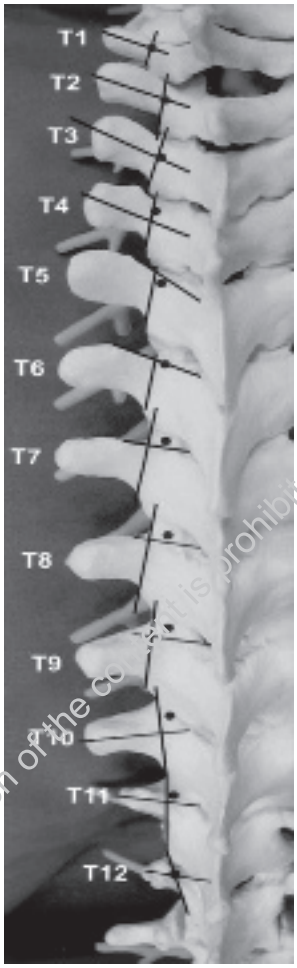

FIGURE 8 Saw bones image depicting starting points for pedicle screw instrumentation of the thoracic spine. (Adapted with permission from Kim YJ, Lenke LG, Bridwell KH, Cho YS, Riew KD: Free hand pedicle screw placement in the thoracic spine: Is it safe? *Spine (Phila Pa 1976)* 2004;29[3]:333-342.)

Navigation systems are used to improve the accuracy of pedicle screw placement, especially in the thoracic spine because the margin for error is small.[38]

Lumbosacral Spine

End Plate Anatomy and Biomechanics

Interbody fusion, including the application of interbody spacers, is used routinely in a stand-alone fashion or as an adjunct to traditional posterolateral fusion. This technique can restore disk height (which in turn provides indirect decompression of the neuroforamen and spinal canal), restores segmental lordosis, and improves spinal alignment. The large surface area within the disk and the compressive environment also provide a favorable setting for bony fusion. The interbody space can be accessed with a variety of approaches, including posterolateral, anterior, or lateral.

Regardless of the approach used, an understanding of the anatomy and biomechanics of the vertebral end

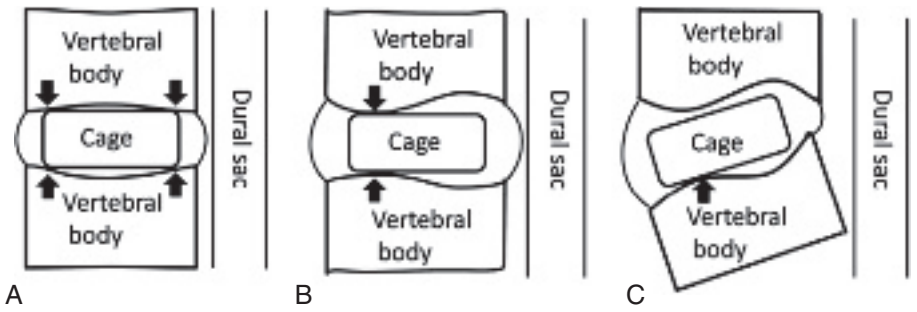

FIGURE 9 Schematic representation of the difference between a common disk shape (**A**), a pear-shaped disk type (**B**), and a pear-shaped disk with greater disk angle type (**C**) with regard to stability of the cage. In the sagittal plane, the cage makes maximal contact with the end plates at all four corners of the interbody device, minimizing the risk for cage migration. (Reprinted with permission from Kimura H, Shikata J, Odate S, Soeda T, Yamamura S: Risk factors for cage retropulsion after posterior lumbar interbody fusion: Analysis of 1,070 cases. *Spine (Phila Pa 1976)* 2012;37[13]:1164-1169.)

plates is essential for the success of the procedure. The bony end plate lies between a layer of hyaline cartilage adjacent to the intervertebral disk and the trabecular bone of the vertebral body. The strength and stiffness of the lumbar end plates increase in the more caudal vertebrae. Within an individual end plate, both strength and stiffness increase from the center to the periphery of the plate. Overall strength and stiffness of end plates decrease by up to 30% and 46%, respectively, at higher grades of disk degeneration, with the most substantial changes in the periphery of the end plates.[39] Injury to the end plates has been shown to reduce pressure within the adjacent nucleus pulposus and increase compressive stress concentration within the posterior anulus fibrosus.[40] This is thought to accelerate the degenerative cascade of the spinal motion segments. The motion segments above L4 and those in older patients are especially vulnerable to motion segment degeneration.[40] The end plates most commonly form a biconcave disk in the sagittal and coronal plane, with the apex of the concavity near the center of the disk. In some patients, a more dorsal apex of concavity is noted and has been associated with retropulsion of posteriorly inserted interbody devices[41] (**Figure 9**). Careful assessment of end plate morphology and the degree of degeneration are important for decreasing interbody cage subsidence and migration rates. A recent study demonstrated that the most significant risk factors for cage migration/retropulsion after transforaminal lumbar interbody fusion are osteoporosis, end plate injury, a pear-shaped disk, posterior positioning of the cage, and use of a single unilateral cage.[42]

Instrumentation

Transpedicular screw fixation from an open posterior approach has become the standard technique for lumbar spine instrumentation because it provides increased strength and stiffness secondary to three-column vertebral fixation. Recently, alternative methods of fixation have been described.[43,44] These methods attempt to decrease the morbidity associated with dissection of the paraspinal musculature required in traditional transpedicular fixation methods or to overcome problems associated with fixation in vertebrae with poor bone quality.

Cortical bone of the pars interarticularis and a medial-to-lateral trajectory can be used to obtain fixation while reducing the exposure required for instrumentation compared with a traditional trajectory. The starting point for the screws in a cortical bone trajectory is 2 to 3 mm medial to the lateral border of the pars interarticularis and caudal to the facet joint. After an initial pilot hole is made with a burr, the trajectory is drilled in an approximate 15° medial-to-lateral direction and a 20° to 25° caudal-to-cranial direction, generally under fluoroscopic guidance. The medial starting point and the medial-to-lateral trajectory limits the required dissection to an area no further than the lateral edge of the facet joint[43] (**Figure 10**). This can preserve innervation to the multifidus muscle and decrease intraoperative blood loss. The cortical bone trajectory also can potentially minimize disruption of the facet joint immediately cranial to the fusion level. This trajectory can be used for all lumbar vertebrae; however, caution should be used at higher levels because the pars interarticularis becomes thinner and the pedicle diameter is smaller, which substantially increases the technical difficulty of screw placement and theoretically increases the risk of pars fracture or inadvertent cortical perforation.[43]

Techniques using facet and translaminar screws were originally described decades ago; however, the use of these techniques in posterior fixation has recently received renewed attention. Although facet and translaminar screws do not provide rigidity equivalent to that of pedicle screws, successful outcomes have been reported when these alternative screw techniques were used as adjuncts in anterior interbody fusion.[44]

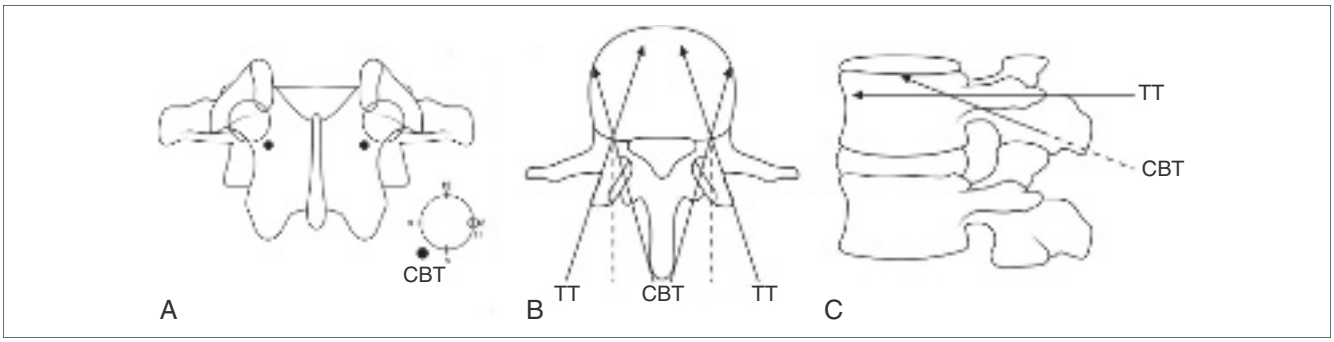

FIGURE 10 Coronal (**A**), axial (**B**), and sagittal (**C**) illustration demonstrating the trajectories for medial to lateral cortical bone trajectory (CBT) screws and traditional trajectory (TT) pedicle screws. (Reprinted from Tortolani PJ, Stroh DA: Cortical bone trajectory technique for posterior spinal instrumentation. *J Am Acad Orthop Surg* 2016;24[11]:755-761.)

Iliac Fixation

Obtaining stable fixation and successful fusion across the lumbosacral junction in long multilevel constructs historically has been challenging for spine surgeons because of the largely cancellous nature of the S1 and S2 pedicles and the substantial forces concentrated on the transition zone from the mobile spine to the relatively rigid pelvis. The addition of pelvic fixation overcomes this challenge by placing fixation across the center of rotation of the pelvis and out of the plane of the remainder of the instrumentation. Initially, the Galveston technique involved the placement of an L-shaped rod between the tables of the ilium.[45] With the advent of modern segmental instrumentation, the technique evolved to use screw fixation within the ilium.

Currently, multiple techniques are available to achieve fixation to the pelvis. Classic iliac fixation uses a starting point in or just medial to the posterior superior iliac spine. A long, large diameter screw is then inserted between the tables of the ilium in a caudal (20° to 45°) and lateral (30° to 45°) trajectory. Although this technique is relatively straightforward, it requires lateral connectors to join to the medial pedicle screw construct. Iliac screws are commonly removed because of symptomatic prominence. A more medial starting point on the posterior superior iliac spine can reduce implant prominence but makes connection of the remainder of the construct more difficult. An S2 starting point that is 2 to 4 mm lateral and 4 to 8 mm caudal to the S1 foramen and a trajectory proceeding through the sacral ala into the pelvis has gained popularity[46] (**Figure 11**). In this S2-alar-iliac (S2AI) technique, the screw tulips are aligned with the remainder of the construct and are unlikely to be symptomatically prominent;

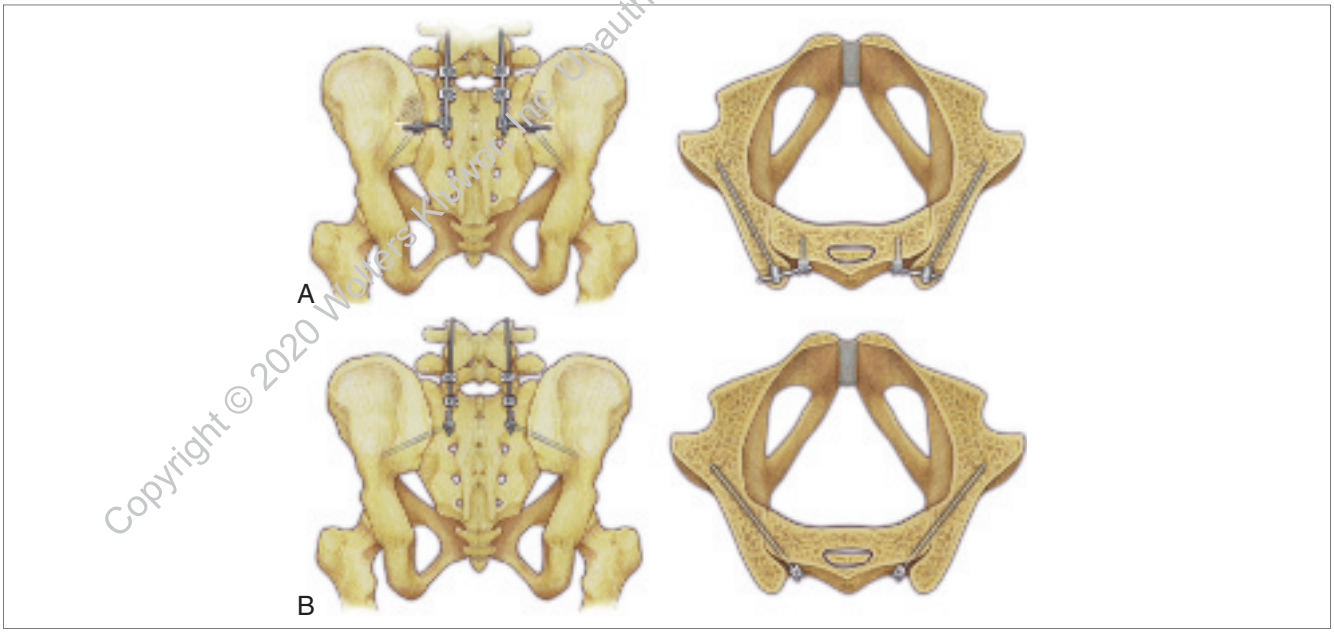

FIGURE 11 Illustration demonstrating posterior and cross-sectional views of traditional iliac screw fixation (**A**) and the S2-alar-iliac trajectory (**B**). (Reprinted from Burns CB, Dua K, Trasolini NA, et al: Biomechanical comparison of spinopelvic fixation constructs: Iliac screw versus S2-alar-Iliac screw, *Spine Deform* 2014;4[1]:10-15. Copyright © 2014, with permission from Elsevier.)

however, because the screws cross the sacroiliac joint, irritation or degeneration of the sacroiliac joint can result. Recent evidence suggests that the technique is associated with a significantly lower revision rate for S2AI screws compared with traditional iliac fixation; particularly a lower rate of infection and hardware prominence.[47,48] This is likely secondary to the extensive soft tissue undermining and paraspinal muscular damage associated with placing traditional iliac screws. Biomechanical evaluation of both traditional iliac fixation or S2AI screw fixation has not demonstrated significant differences in stiffness or load to failure.[49]

SUMMARY

Although the anatomy of the spine has not changed, the understanding of the relationships between the structures that make up the spine and the changes in these structures caused by aging, degeneration, and injury has advanced considerably in recent years. This understanding has implications in the diagnosis and treatment of spinal pathology and is essential knowledge for any surgeon treating patients with common and often debilitating spinal disorders and injuries.

KEY STUDY POINTS

- A detailed knowledge of spine anatomy is a prerequisite for safe and effective nonsurgical and surgical treatment of patients with spine pathology.
- Growing evidence exists that the health and function of the multifidus muscles has an effect on clinical function in the lumbar spine, indicating potential benefits of less invasive surgical approaches.
- The freehand technique for thoracic pedicle screw instrumentation is safe and effective. In patients with spine deformity, the relationship of the great vessels to the spine may be altered.
- The morphology and degenerative state of the vertebral end plate is an important consideration when applying interbody instrumentation.
- Techniques for lumbar and lumbosacral instrumentation continue to evolve. Increasing evidence supports the safety and efficacy of the cortical bone screw trajectory and S2AI fixation.

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This retrospective study analyzed 223 patients who underwent three-column osteotomy for thoracolumbar deformity and correlated cervical sagittal parameters, with the development of cervical sagittal deformity (CSD). In patients with compensated deformities (TS-CL of <17°), there was reciprocal change in cervical and thoracolumbar alignment. Positive predictors of postoperative CSD included baseline TS-CL > 17°, longer fusion, and baseline cervical-thoracic pelvic angle. Level of evidence: III.

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CHAPTER 2

Spine Neuroanatomy and Physiology

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ABSTRACT

A basic knowledge of spine neuroanatomy and physiology is required for the effective evaluation of spine pathology. It is helpful to be familiar with the applied neuroanatomy and physiology of the spine and understand the correlation of clinical signs and symptoms with relevant pathology. A stepwise approach to the diagnosis of spine pathology can aid in providing optimal patient care.

Keywords: neuroanatomy; neurophysiology; spinal cord; spine

INTRODUCTION

The spinal cord plays a fundamental role in sensory, motor, and autonomic control of the human body. Physicians who treat patients with spine disorders must have a basic understanding of the neuroanatomy and physiology of the spine to recognize, accurately diagnose, and manage pathologic spinal conditions.

Dr. Kurd or an immediate family member serves as a paid consultant to or is an employee of K2M Spine and Spinal Elements; has stock or stock options held in DuraStat LLC; has received research or institutional support from Innovative Surgical Designs; and serves as a board member, owner, officer, or committee member of ISASS. None of the following authors or any immediate family member has received anything of value from or has stock or stock options held in a commercial company or institution related directly or indirectly to the subject of this chapter: Mr. Ahern, Ms. Welch-Phillips, and Dr. Butler.

SPINAL CORD STRUCTURE

The spinal cord provides both motor and sensory function. The cerebral cortex, internal capsule, corticospinal tracts, pyramidal tracts, and anterior horns cells control motor function. It is believed that 90% of the pyramidal tract crosses at the medulla to the contralateral lateral corticospinal fasciculus, where the tracts controlling the upper extremities lie medial to those controlling the lower extremities.[1] An understanding of this neuroanatomy has relevance for central cord syndrome, a clinical condition that predominantly affects the medial tracts of the lateral corticospinal fasciculus, which controls motor function to the upper extremities, with relative sparing of the most lateral tracts, which primarily control motor function to the lower extremities.[1]

The posterior columns, the lateral spinothalamic fasciculus, and the anterior spinothalamic fasciculus control sensory function. The sensory tracts carried in the posterior columns are responsible for fine touch, proprioception, and vibration sense and decussate to the opposite side in the medulla oblongata on their pathway to the sensory cortex. Sectioning of a posterior column results in loss of these sensory functions ipsilateral to the lesion. This is in contrast to pain and temperature, which are carried by the lateral spinothalamic tracts. The lateral spinothalamic tracts decussate with the spinal cord, resulting in sensory loss contralateral to damage of one of these tracts.

The anterior spinal artery is the primary vessel supplying vascularity to the anterior and central aspects of the spinal cord in the cervical region.[1] At the brainstem, the vertebral arteries give rise to two medullary feeder vessels. The thoracolumbar region receives its vascular supply from the anterior spinal artery and two posterior spinal arteries.[2] In the cervicothoracic junction region, blood is supplied to the spinal cord by the superior intercostal artery, which is a feeder vessel of the deep cervical

artery arising from the right subclavian artery.[2] Several segmental vessels provide a tenuous blood supply for the upper thoracic cord, with a watershed critical zone existing between T4 and T10.[1] A dominant artery, the artery of Adamkiewicz, arises from the left posterior intercostal artery at T10 in 80% of cases. In other cases, its origin ranges from T5 to L5.[1]

The azygous and hemiazygous venous systems provide the primary venous drainage for the spinal cord. The spinal cord veins lie anterior and posterior to the cord. These vessels, in turn, drain into the extradural, valveless Batson venous plexus. This plexus plays an important role in the etiology of pyogenic spinal infection and the dissemination of metastatic disease.[1]

The termination of the spinal cord is at the L1-L2 level in the tapering conus medullaris (**Figure 1**). The spinal cord is 45 cm in length, with 25 cm of filum terminale.[1] It has a mean diameter of 10 mm and increases in length by approximately 10% with flexion. The spinal cord has two areas of enlargement—one area to supply innervation to the upper extremities and one area for the lower extremities.[3] It increases in size at each level caudal to C1 until approximately the level of C5, where it attains maximal cross-sectional area. At the thoracolumbar junction, it enlarges again before rapidly decreasing in size in the region of the conus medullaris.

The spinal cord is made of central gray matter and peripheral white matter (**Figure 2**). The gray matter comprises efferent neuron cell bodies. Somatosensory function is controlled by the posterior horns; somatomotor function is controlled by the anterior horns; and visceral function is controlled by the intermediolateral horns. The gray matter also acts as the center for somatic reflexes. The white matter contains nerve fibers and glia. The posterior column is composed of the fasciculus cuneatus and fasciculus gracilis; the lateral column consists of the descending motor lateral corticospinal and lateral spinothalamic fasciculi; and the anterior funiculus carries the ascending anterior spinothalamic tract and several descending tracts[1] (**Figure 3**).

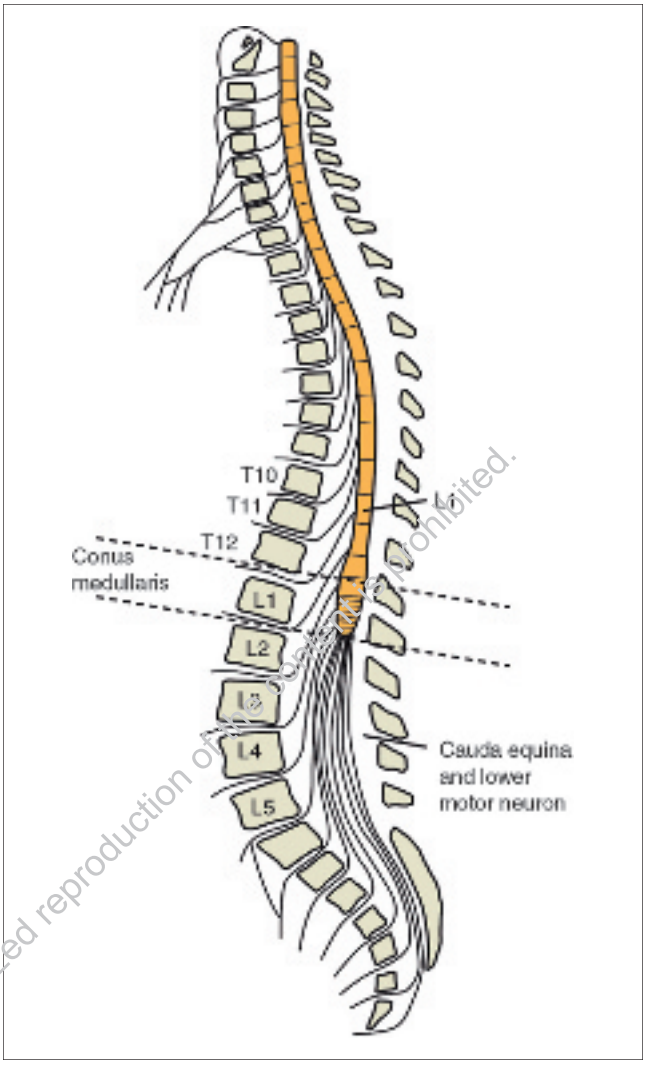

FIGURE 1 Illustration of the spinal cord and nerve roots. The spinal cord emerges from the foramen magnum as a continuation of the medulla oblongata and ends in a cone-shaped structure known as the conus medullaris. The location of the conus medullaris is usually at the L1-2 intervertebral disk in adults.

SPINAL MENINGES

The outermost protective layer surrounding the spinal cord is the dura mater. The leptomeninx is made up of the pia mater and the arachnoid mater, which is a transparent sheet containing cerebrospinal fluid (**Figure 4**). The dentate ligaments connect the spinal cord to the dura. The space in the spinal canal between vertebral bone and dura mater is known as the epidural space. The average diameter of this space is 2 mm at the level of L3-L4, 4 mm at L4-L5, and 6 mm at L5-S1.[1] The dura/arachnoid mater terminates between S1-S2 and S2-S3, where it surrounds the filum terminale and has a coccygeal attachment.

SPINAL NERVES

There are 31 pairs of spinal nerves—8 in the cervical region, 12 in the thoracic region, 5 in the lumbar region, 5 in the sacrum, and 1 in the coccyx. The structure of each spinal root consists of motor and sensory rootlets, a dorsal root ganglion, and a spinal nerve. The sympathetic nervous system is supplied by myelinated preganglionic

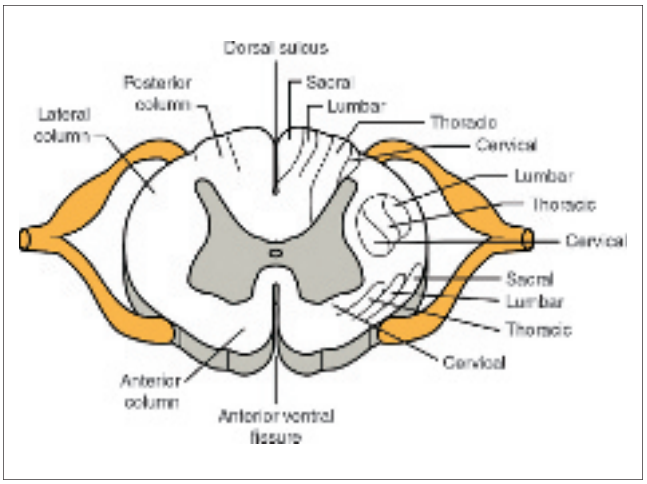

FIGURE 2 Cross-sectional illustration of the spinal cord with the outer white matter and the inner gray matter. The white matter of the spinal cord contains nerve fibers and glia and is divided into three columns: posterior, lateral, and anterior.

rami and unmyelinated postganglionic rami. Important branches of the spinal nerve include the sinuvertebral nerve, which supplies the anulus fibrosus of the intervertebral disk, and the dorsal ramus, which supplies the facet joints and posterior paraspinal musculature. The intervertebral disk in the lumbar region receives innervation from the sympathetic fibers anteriorly and the sinuvertebral nerve posteriorly. The sinuvertebral nerves innervate the posterior longitudinal ligament, the posterior portion of the anulus fibrosus, the ventral aspect of the dura mater, and the superior portion of the intervertebral disk. The dorsal primary rami give off medial, lateral, and, occasionally, intermediate branches. The facet joints, interspinous ligaments, and segmental muscles are supplied by the medial branch, with the iliocostalis and longissimus muscles supplied by the lateral and intermediate branches, respectively.

When considering the anatomic pathway of the spinal nerves, it is important to be aware of regional variations. In the cervical spine, the C1 nerve arises above the C1 vertebra, whereas the C8 nerve arises above the T1 vertebra. Conversely, in the thoracic and lumbar spine, the spinal nerve passes under the pedicle of the same numbered vertebra.[1] The spinal nerves emerge within the intervertebral foramina. In the cervical spine, no intervertebral foramina exist for C1 and C2; in the remaining subaxial spine, the C3-C8 nerve roots emerge through the corresponding intervertebral foramina to occupy approximately 75% of their respective foramen.[2] Thoracic nerves are much smaller, occupying only 20% of their foramen. Lumbar spinal nerves are larger, occupying approximately 33% of the neural foramen, and emerge obliquely under their respective pedicles.[1] In the sacral region, the anterior and

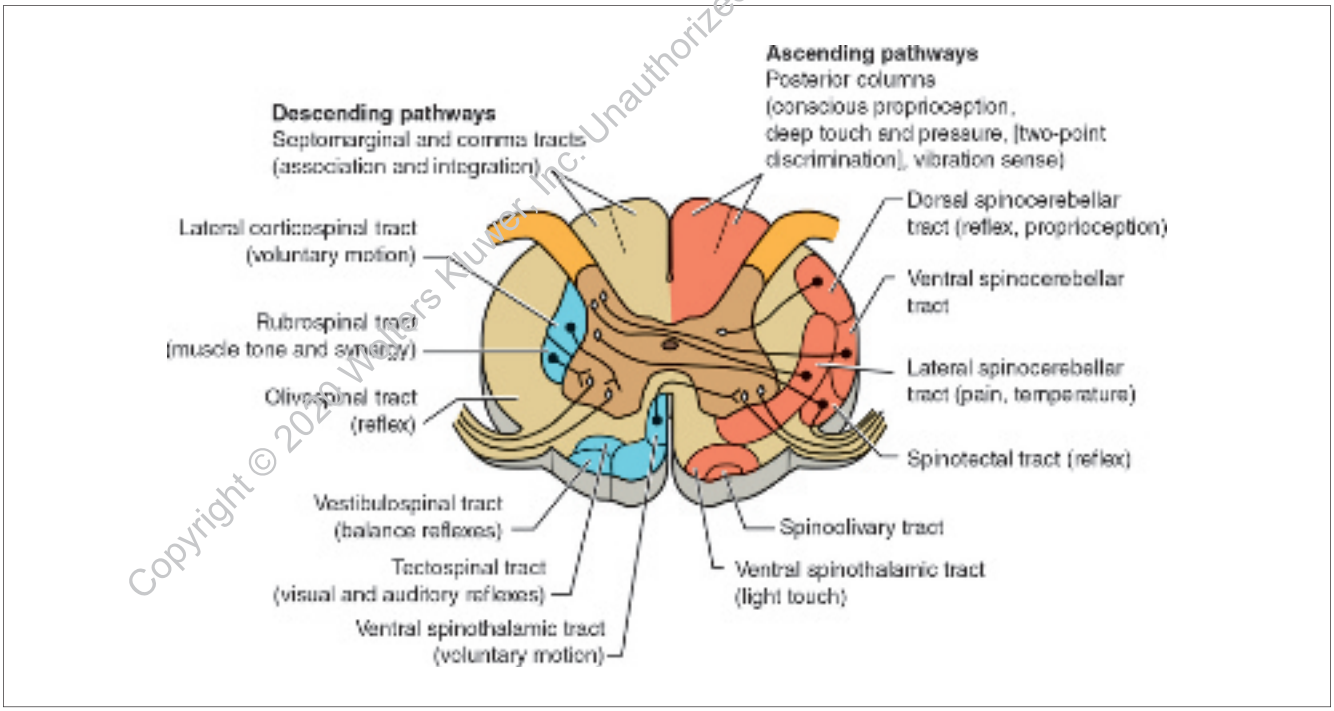

FIGURE 3 Illustration showing the ascending and descending pathways in the spinal cord.

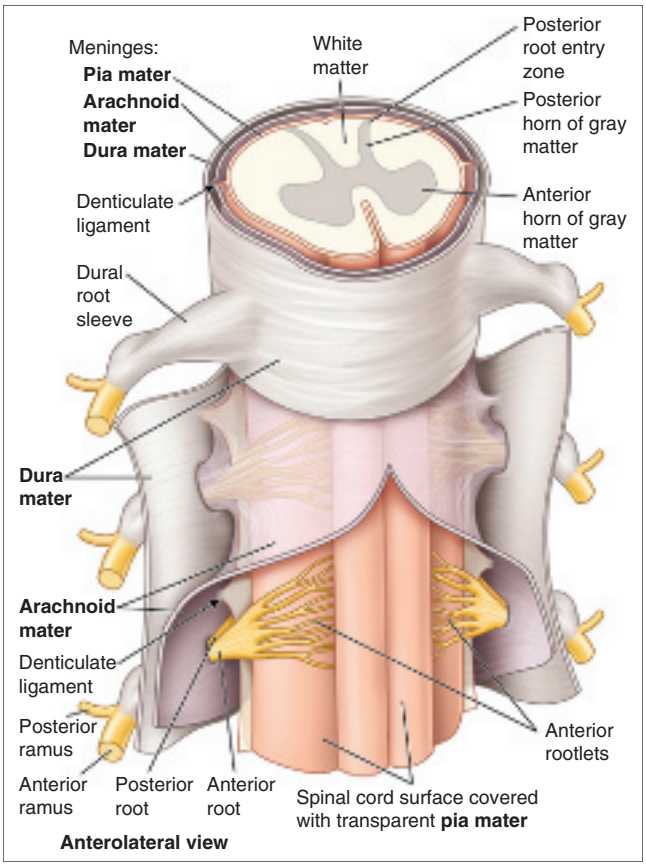

FIGURE 4 Cross-sectional illustration of the spinal cord and meninges. The spinal cord is covered by the pia mater, which is the outer lining of the cord, and transparent arachnoid mater that contains the cerebrospinal fluid. The dura mater is the outer covering of the spinal cord. The spinal cord is anchored to the dura by the dentate ligaments that project laterally from the lateral side of the cord to the arachnoid and dura mater at points midway between exiting spinal nerves. (Reproduced with permission from Agur A, Dalley A. *Moore's Essential Clinical Anatomy,* ed 6. Philadelphia, PA, Wolters Kluwer, 2019.)

posterior rami exit the sacrum through their respective anterior and posterior foramina.[1]

The motor and sensory function of the spinal cord is distributed into distinct dermatomes (sensory) and myotomes (motor) (**Figure 5**). The C4 nerve supplies motor function to the muscles to allow spontaneous breathing and actions such as shoulder shrugging. C5 controls deltoid and biceps function, C6 wrist extension, C7 triceps and wrist flexion, C8 finger flexion, and T1 the hand intrinsic muscles. In the lumbar region, L2 controls iliopsoas function, L3 quadriceps function, L4 tibialis anterior function, and L5 extensor hallucis longus function. In the sacral region, S1 controls the gastrocnemius, S2 the bladder sphincter, and S3 the anal sphincter. When considering sensory function, C5 supplies the upper outer arm, C6 the thumb, C7 the long finger, C8 the little finger, T1 the medial forearm, T10 the periumbilical area, L1 the groin region, L2 the anterior thigh, L3 the knee, L4 the medial malleolus, L5 the great toe, S1 the small toe, S2 the posterior thigh, and S3-S5 the perianal region.

A variety of nerve root anomalies exist, and it is important to recognize them to avoid neural injury during surgery. These anomalies have been divided into type I, involving an intradural anastomosis; type II, involving an abnormal nerve root origin; type III, involving an extradural anastomosis; and type IV, involving an extradural division.[4]

Nerve root vascularization is a complex process. Both the proximal and distal radicular arteries anastomose at the proximal one-third of the spinal nerve root within the foramen, leading to a region of potential vascular insufficiency. Intrinsic vasculature includes interfascicular and intrafascicular vessels. The innermost pia mater permits exchange of metabolites within the cerebrospinal fluid. Mechanical compression of a nerve root can result in vascular compression, which can lead to the development of the classic symptoms of radiculopathy. The cauda equina is an organized structure containing the lumbar and sacral nerve roots, and several of the spinal nerve roots are organized into plexus structures. The cervical plexus is composed of the ventral rami of C1-C4; the brachial plexus is formed by the anterior rami of C5-T1; and the sacral plexus is made up of the lumbosacral trunk (L4, L5) and the S1, S2, S3, and S4 anterior rami.[1]

An in-depth knowledge of the motor and sensory nervous systems is a prerequisite when clinically assessing patients with suspected nerve root pathology or peripheral neuropathies. For example, a C8-T1 and ulnar nerve neuropathy have similar presentations, but subtle differences can aid in differentiating the two. Compression of the ulnar nerve at the elbow (cubital nerve syndrome) leads to anesthesia of the ulnar one and a half digits along with the ventral and dorsal aspects of the hand, but not the forearm. Sensation to the medial forearm is provided by the medial antebrachial cutaneous nerve, a branch of the C8-T1 nerve root via the medial cord of the brachial plexus. In terms of motor function, the ulnar nerve supplies all but five of the intrinsic muscles of the hand, which are supplied by the C8-T1 nerve roots. The abductor (*Ab*) and flexor pollicis brevis (*F*), opponens pollicis (*O*) and lateral two lumbricals (*Law*) are innervated by the C8-T1 nerve roots via the median nerve (mnemonic—AbOF the Law "*Above the law*"). Weakness in these muscles with ulnar-sided dysesthesia suggest a radiculopathy as opposed to cubital tunnel syndrome.[5]

Chronic low back pain has been shown to arise due to disk and end plate degeneration leading to changes in the morphology and biology of the end plate including blood vessel proliferation.[6,7] There is growing evidence

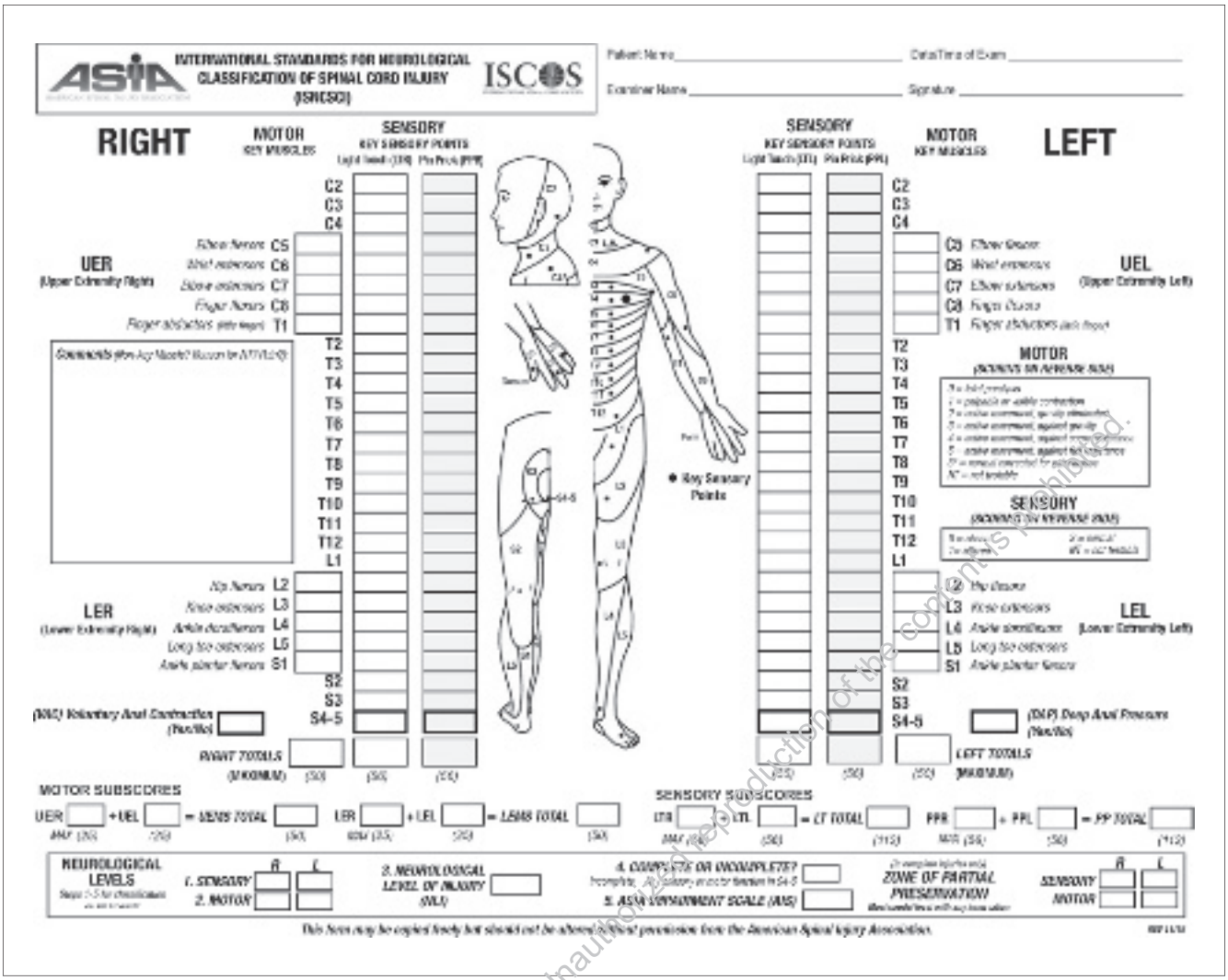

FIGURE 5 Illustration of the International Standards for Neurological Classification of Spinal Cord Injury and the American Spinal Injury Association (ASIA) Impairment Scale. ASIA scores provide a good format for dermatome and myotome assessment. (The American Spinal Injury Association International Standards Committee: *International Standards for Neurological Classification of Spinal Cord Injury*. Available at: https://asia-spinalinjury.org/international-standards-neurological-classification-sci-isncsci-worksheet/. Accessed February 26, 2021. © 2021 American Spinal Injury Association. Reprinted with permission.)

that this pain is transmitted through signals from the basivertebral nerve complex.[7] Damage to the end plate leads to the expression of inflammatory mediators followed by a further signaling cascade, which includes the production of cytokines. MRI displays these changes as Modic changes (three types). The basivertebral nerve complex (first described by the authors of a 1998 study[6]) is a branch of the sinuvertebral nerve and follows the nutrient artery and clusters at the vertebral center before branching to the end plates.[6] Two recent randomized controlled trials have demonstrated intraosseous radiofrequency ablation of the basivertebral nerve as an effective form of treatment for highly selected cohort patients with specific chronic low back pain. This included patients with Modic type 1 or type 2 vertebral changes, those without radicular pain, symptomatic spinal stenosis, or metabolic bone disease. The radiofrequency ablation is delivered via a transpedicular approach to the areas of Modic changes. This method is a potential alternative to nonsurgical measures for the aforementioned specific subtypes of chronic low back pain.[6,7]

AUTONOMIC NERVOUS SYSTEM

The autonomic nervous system is divided into the sympathetic and parasympathetic systems. The preganglionic neurons of the sympathetic system are located between C8 and L4. The sympathetic centers control the cardiovascular and bronchopulmonary systems, sweat gland function, vasomotor activity, anorectal and bladder

continence, and ejaculation. Horner syndrome is a clinical condition that can result from injury to the cervical or first thoracic sympathetic chain. There are three cervical ganglia located along the course of the cervical sympathetic chain, which lies along the longus coli muscles, dorsal to the carotid sheath. The middle cervical chain at the level of the C6 vertebral body is the most common site of injury during anterior cervical surgery. Injury can cause Horner syndrome (ptosis, miosis, anhidrosis, and enophthalmos). Autonomic dysreflexia results from spinal cord injury above the level of the sympathetic splanchnic outflow (T6) and leads to headache, sweating, flushing, and hypertension. Hypogastric plexus injury (often where it lies anterior to the L1-S5 disk) can result in urogenital problems such as retrograde ejaculation. The superior hypogastric plexus is formed by a confluence of lumbar sympathetic chains, branches of aortic plexus (fibers of celiac and inferior mesenteric plexuses), and parasympathetic fibers of ventral roots S2-S4. It is a retroperitoneal structure typically located anterior to the lower third of the fifth lumbar vertebrae and extends to the upper third of the first sacral body (L5-S1).[8,9] Injury to the plexus may cause retrograde ejaculation and dysfunction of abdominopelvic viscera. Retrograde ejaculation or sterility in men can be caused after injury during an anterior surgical approach to the lumbar spine. Two studies proposed different surgical approaches for lumbar interbody fusion which decreases the risk of retrograde ejaculation: one via a lateral approach[8], and the other from right side of the midline with mobilization of the inferior vena cava.[9]

The parasympathetic nervous system controls a variety of visceral functions, including peristalsis and bladder wall contraction. The parasympathetic system also controls relaxation of certain smooth muscles such as those that regulate arterial blood flow and penile erection. Its cell bodies lie in the brainstem and sacral cord. Many visceral parasympathetic functions are carried through the vagus nerve and thus function unopposed by sympathetic outflow in cases of spinal cord injury. Disruption of the sacral signaling pathways can impair crucial autonomic functions such as bladder and defecation control and sexual arousal.[10]

SPINAL CORD TRACTS

The intrinsic pathways of the spinal cord establish connections between various neuronal groups and segments of the spinal cord and serve as relays between intrinsic spinal neurons and descending pathways.[10]

The ascending pathways of the spinal cord are formed by the axons of dorsal root ganglion cells, which enter the spinal cord through the dorsal roots. They subsequently enter an ascending fiber tract, such as the dorsal column pathways, which contain the fasciculus gracilis and fasciculus cuneatus. The fasciculus gracilis controls the lower trunk and lower limbs, whereas the fasciculus cuneatus controls the upper trunk and upper limbs. The dorsal pathways control a variety of discriminative sensory functions, including two-point discrimination, detection of speed, direction of movement, and assessment of cutaneous pressure.[10] Localization of pain and thermal stimuli are controlled by the spinothalamic tract. Its axons decussate to the ventrolateral column and terminate in the ventral posterolateral and central lateral nuclei of the thalamus.[10]

The corticospinal tract is the most developed tract of those in the descending pathways. It originates in the motor cortex, with its axons forming the pyramidal tract, and most of its fibers decussate in the lower medulla to form the lateral corticospinal tract. The remaining fibers remain in the ventral funiculus and subsequently decussate in the ventral commissure. The corticospinal tract exerts refined motor control through its influence on other descending spinal pathways.[11]

SPINAL CORD FUNCTION

The spinal cord plays a central role in sensory, motor, and autonomic control. It controls the sensory processing of pain, temperature, touch, and proprioception. The peripheral sensory receptors are specialized sense organs that connect with axons from the dorsal root ganglion. They are called first-order neurons because they are directly linked with peripheral sensory receptors; their processing of sensory information is determined by their branching pattern (**Figure 6**). These neurons terminate and synapse on neurons in the substantia gelatinosa. In this part of the dorsal horn, the second-order neurons give rise to their processes, which carry signals to other areas of the brain and spinal cord.[2,10] Second-order neurons play an important role in processing sensory information within the spinal cord. Somatic afferent fibers, in addition to the fibers controlling visceral sensation and pain, converge on the neurons of the substantia gelatinosa. Certain regions of the brain also supply substantial input to affect neuromodulation within the substantia gelatinosa.[12]

Specialized organs in the skin and connective tissue and free nerve endings in the dermis can sense light touch. These sense organs send signals along axons arising from the dorsal root ganglia. This signaling pathway conveying the sensation of touch is also responsible for controlling more sophisticated sensory functions such as proprioception and two-point discrimination.[10] Muscle spindles monitor muscle length; Golgi tendon organs monitor tendon stretch; and the Pacini corpuscles monitor the pressure exerted on joints and bony structures.[10] The axons from muscle spindles, which send signals to the spinal cord, are among the largest and fastest

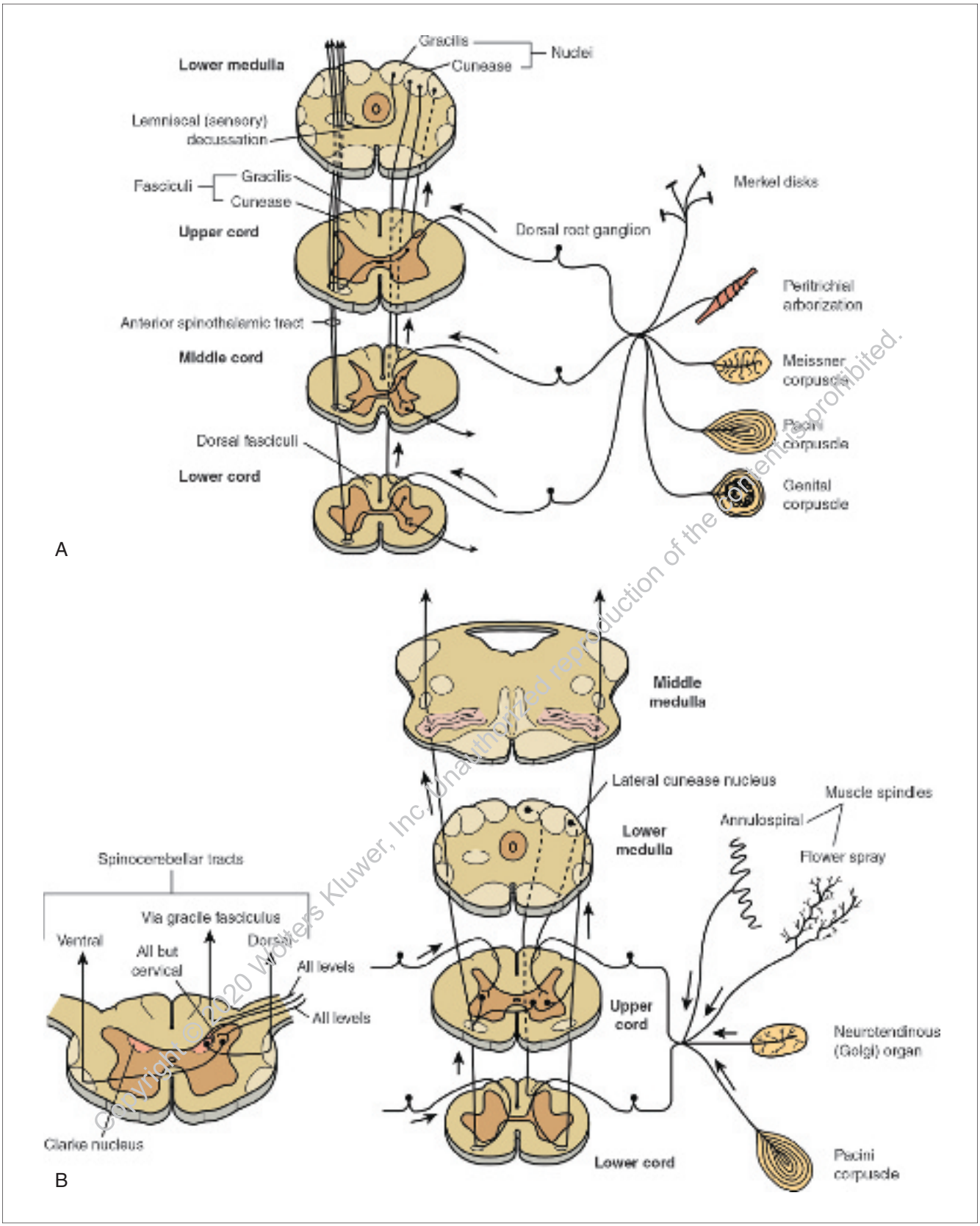

FIGURE 6 **A** and **B**, Illustrations of the types of sensory nerve endings in peripheral tissues innervated by sensory nerves (peripheral processes). The Clarke nucleus is demonstrated in relation to the ventral and dorsal spinocerebellar tract and dorsal columns.

conducting nerves in the nervous system. The central branch, making up the medial division of the dorsal root, splits after entering the spinal cord. These synapses with motor neurons allow monosynaptic reflex activity. These monosynaptic connections have a high degree of specificity, with muscle spindle afferents from a given muscle (in response to changes in muscle length and velocity) making contact only with motor neurons that innervate the muscle of origin of the afferent fiber. The monosynaptic stretch reflex is initiated by activation of the type IA afferent fibers from the muscle spindle, which results in contraction of the synonymous muscle. Muscle contraction strength in response to the same stimulus is not always identical and is influenced by preceding activity of the spinal cord. The variability of reflex activity also is affected by temporal and spatial summation of excitatory inputs and inhibitory influences from other sources.

All other reflexes are considered polysynaptic, with each neuron involved in the reflex potentially contributing to the outcome, which is the motor response to a stimulus (**Figure 7**). Simple spinal reflexes demonstrate that neurons not only are excited but also can be inhibited by certain inputs. This inhibition can be postsynaptic or presynaptic. In postsynaptic inhibition, the membrane potential of the postsynaptic neuron increases, and the same excitatory input is unsuccessful in depolarizing the neuron sufficiently to initiate an action potential. In presynaptic inhibition, a reduced amount of excitatory transmitter is released from the presynaptic terminal.

SPINAL CORD LESIONS

Acute issues in spinal cord injury include both neurogenic and spinal shock. Neurogenic shock results in hypotension and relative bradycardia and can be fatal. It occurs as the result of circulatory collapse from loss of sympathetic tone and is attributable to disruption of the autonomic pathway within the spinal cord. This leads to lack of sympathetic tone, decreased systemic vascular resistance, pooling of blood in the limbs, and hypotension.

Spinal shock is a temporary loss of spinal cord function and reflex activity below the level of a spinal cord injury. It results in a flaccid areflexic paralysis and an absent bulbocavernosus reflex. Spinal shock occurs because of hyperpolarization of neurons that remain unresponsive to brain stimuli, and symptoms of neurogenic shock may be present. It almost always resolves within 48 hours.

A variety of incomplete spinal cord lesions can occur (**Figure 8**). Central cord syndrome is the most common and often affects elderly individuals who sustain a minor extension injury to the cervical spine. It is caused by spinal cord compression and central cord edema and results in selective destruction of the white matter in the central area of the lateral corticospinal tract; the upper extremities are preferentially affected. Although central cord syndrome is associated with a good prognosis, complete functional recovery is rare.

Anterior cord syndrome (also known as ventral cord syndrome and anterior spinal artery syndrome) results from direct or indirect damage, with subsequent loss of function to the anterior two-thirds of the spinal cord but preserved posterior column function. Therefore, anterior cord syndrome results in abrupt onset of pain and associated flaccid paraplegia or quadriplegia (depending on the level of the injury) below the lesion along with alterations in temperature and pain sensation (lateral spinothalamic tract) and autonomic dysfunction. Autonomic dysfunction may include hypotension, sexual dysfunction, and/or bowel/bladder dysfunction.[13] The lower extremity is affected more often than the upper extremity. Injury to the anterior spinal cord is often caused by direct compression (eg, osseous, tumors, abscesses, hematomas) or damage to the anterior spinal artery. The anterior spinal artery is the sole blood supply to the anterior spinal cord. Iatrogenic mechanisms of injury include prolonged clamping of the aorta during vascular repairs, resulting in ischemia or emboli to the artery.[13] Anterior cord syndrome carries the worst prognosis of all incomplete spinal cord lesions, and the effects are most likely to mimic those of a complete spinal cord injury.

Brown-Séquard syndrome results from spinal cord hemitransection, often from a penetrating injury. Hemitransection of the spinal cord leads to an ipsilateral deficit of the lateral corticospinal tract, which controls motor function, and a deficit in the dorsal columns, which control proprioception and vibration sense. A contralateral deficit in the lateral spinothalamic tract, which controls pain and temperature, also occurs. Brown-Séquard syndrome is more commonly seen at the cervical and thoracic levels secondary to traumatic events, which include fractures, stab wounds, and, less commonly, tumors or abscesses. It has also been described secondary to herniated cervical disk disease.[14] The syndrome is associated with an excellent prognosis for functional improvement.

Posterior cord syndrome, which is very rare, results in loss of proprioception, with preservation of motor function, pain sensation, and temperature. Infarction of the posterior spinal artery can rarely lead to neurologic deficits related to the posterior column pathway. This is characterized by loss of vibration, proprioception sensation, and reflexes below level of the lesion. Demyelinating diseases commonly target the dorsal column pathway. These include infective causes such as tabes dorsalis (late manifestation of tertiary syphilis) and subacute combined degeneration of the spinal cord, secondary to vitamin B_{12} deficiency.[14] Tabes dorsalis presentation includes peripheral reflexes loss, impairment of vibration, position sense

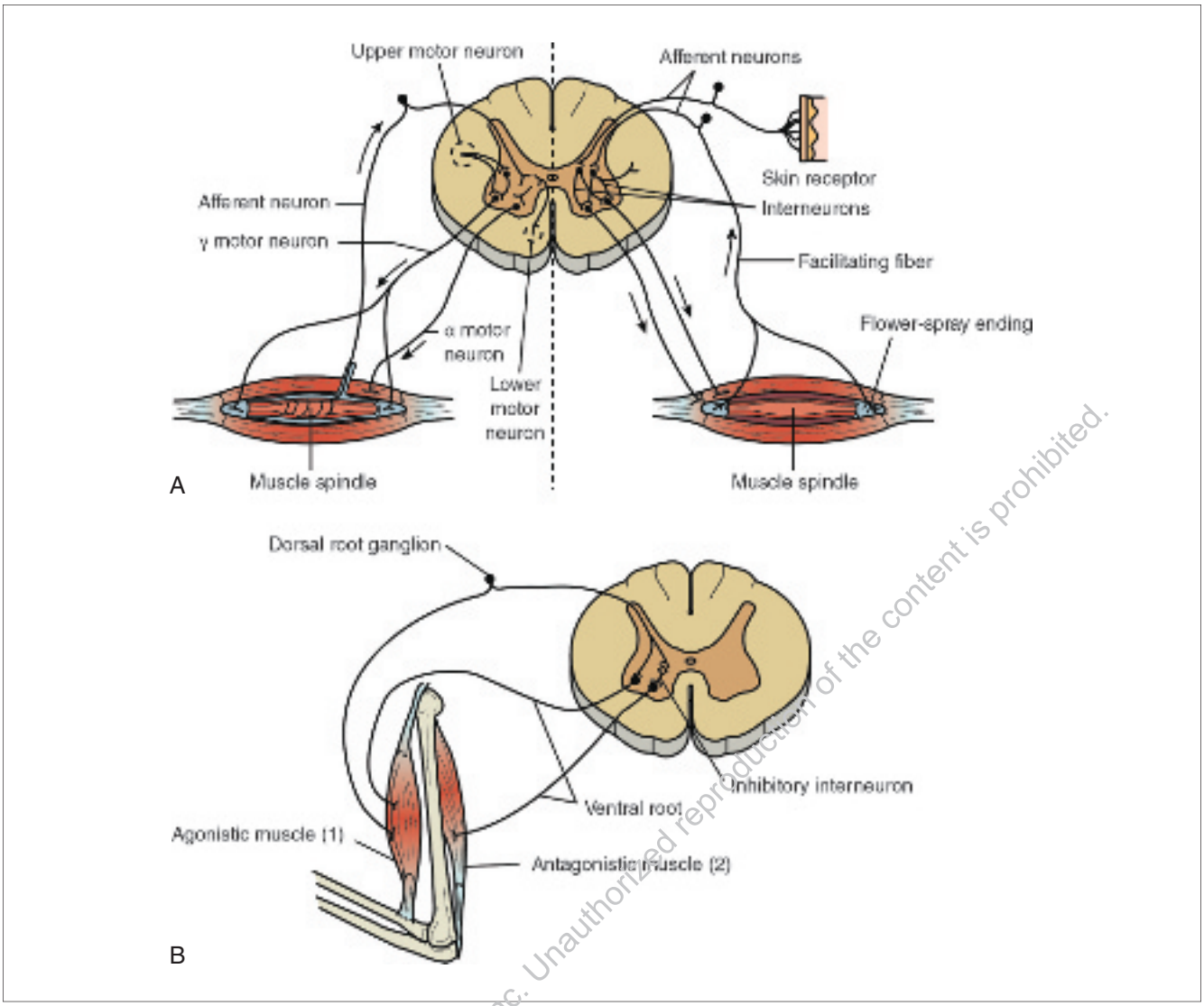

FIGURE 7 Illustrations show the sensory and motor innervation of muscles spindles along with a cross-section of the spinal cord with various inputs (**A**) and the reciprocal inhibition of antagonistic muscles during monosynaptic stretch reflex (**B**).

and progressive ataxia along with sudden onset of severe lightning pain. Some patients will present with Argyll Robertson pupils (loss of ability to constrict with preservation of ability to accommodate). B12 deficiency leads to compromise in nerve transmission due to disruption of myelination process.[14]

The conus medullaris and cauda equina syndromes are complex clinical syndromes, which result from damage or compression of the conus medullaris and cauda equina, respectively. Both have overlapping presentations, which may include back and unilateral or bilateral radicular pain, paresthesia and weakness, in addition to bowel/bladder dysfunction. Anatomically, the conus medullaris is the terminal dilatation of the spinal cord and marks the transition of the central nervous system to the peripheral nerve fibers of the cauda equina. Therefore, patients with conus medullaris syndrome may display a mixture of both upper and lower motor neuron signs, compared with the exclusively lower motor neuron signs seen in cauda equina syndrome.

PHYSIOLOGY OF PAIN

Pain is defined as an unpleasant sensory or emotional experience associated with actual or potential tissue damage.[15] Pain is an experience that is subjective and difficult to quantify, thus separating it from nociception, which is the transmission of noxious stimuli to the brain from peripheral pain receptors (nociceptors).[16] Nociceptors are receptors in tissues that can sense noxious stimuli,

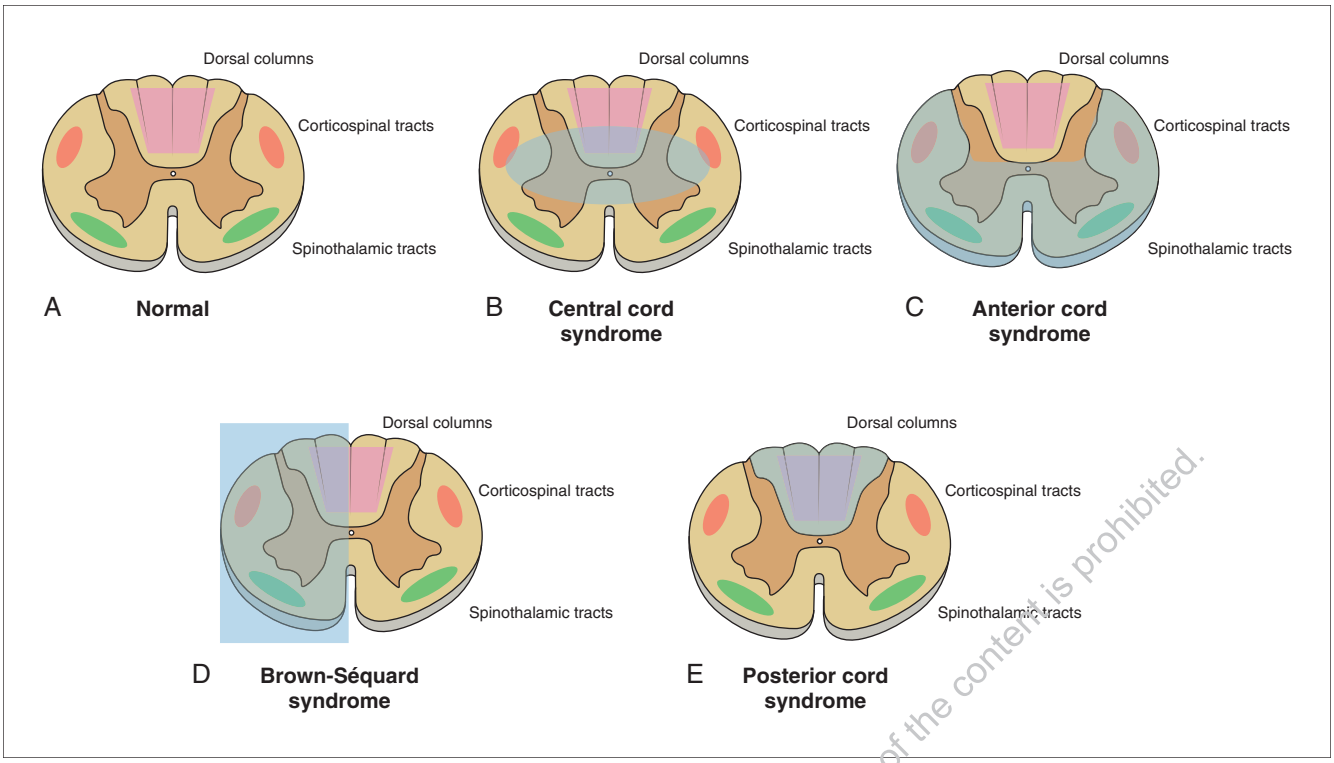

FIGURE 8 Schematic of various cord syndromes. **A**, Normal. **B**, Central cord syndrome. **C**, Anterior cord syndrome. **D**, Brown-Séquard syndrome. **F**, Posterior cord syndrome.

via mechanical deformation and chemical mediation (eg, inflammatory mediators).[16] Therefore, pain is a complex interaction of various signaling pathways and the unique experience of the individual. The pathophysiology[16] of spinal surgery pain is the result of a combination of nociceptive, inflammatory, and neuronal stimuli.[17] Effective pain management regimens target multiple mechanisms of pain transmission.

Perioperative pain originates from a combination of primary (peripheral sensitization) and secondary (central sensitization) hyperalgesia. Peripheral sensitization occurs when damaged nociceptors become sensitized by injury and inflammatory mediators such that they have a lower threshold for firing, an increased response to noxious stimuli and may fire following nonnoxious stimulation. Central sensitization is the persistent state of high reactivity in the spinal cord and brain following peripheral nerve injury and contributes to chronic pain.[17]

The management of postoperative pain is improved when the aforementioned painful stimuli are inhibited either via intraoperative measures or preemptive analgesia. Chronic postsurgical pain is present in up to 50% of patients who have intense tissue trauma during surgery. This trauma results in peripheral nerve damage and a release of nociceptive substances from the nerve endings and tissues, leading to the phenomenon known as wind-up and central sensitization. This leads to peripheral sensitization of nociceptors resulting in allodynia and hyperalgesia. Preemptive analgesics aim to target the nociceptor before injury.[17]

Acetaminophen is part of most analgesic regimens, but despite its widespread use, its mechanism of action is not completely understood. It may modulate pain through a number of pathways, namely the selective inhibition of cyclooxygenase within the central nervous system and the activation of descending serotonergic pathways (**Figure 9**). It may also modulate the endogenous cannabinoid system.

Local anesthetics prevent excitation of peripheral nerve endings and the propagation of noxious stimuli along the peripheral nerve. Their mechanism of action typically involves the reversible binding and inactivation of sodium channels. They are now in widespread use preoperatively throughout all disciplines of surgery to prevent the initial sensitization of the peripheral nerve receptors.

Opioid analgesics are often more successful in treating rest pain, than ambulatory pain. Because of the toxic effects and potential for addiction, they are usually given in combination with NSAIDs and other adjuvants as part of a multimodal pain program.[17] The synergistic effect

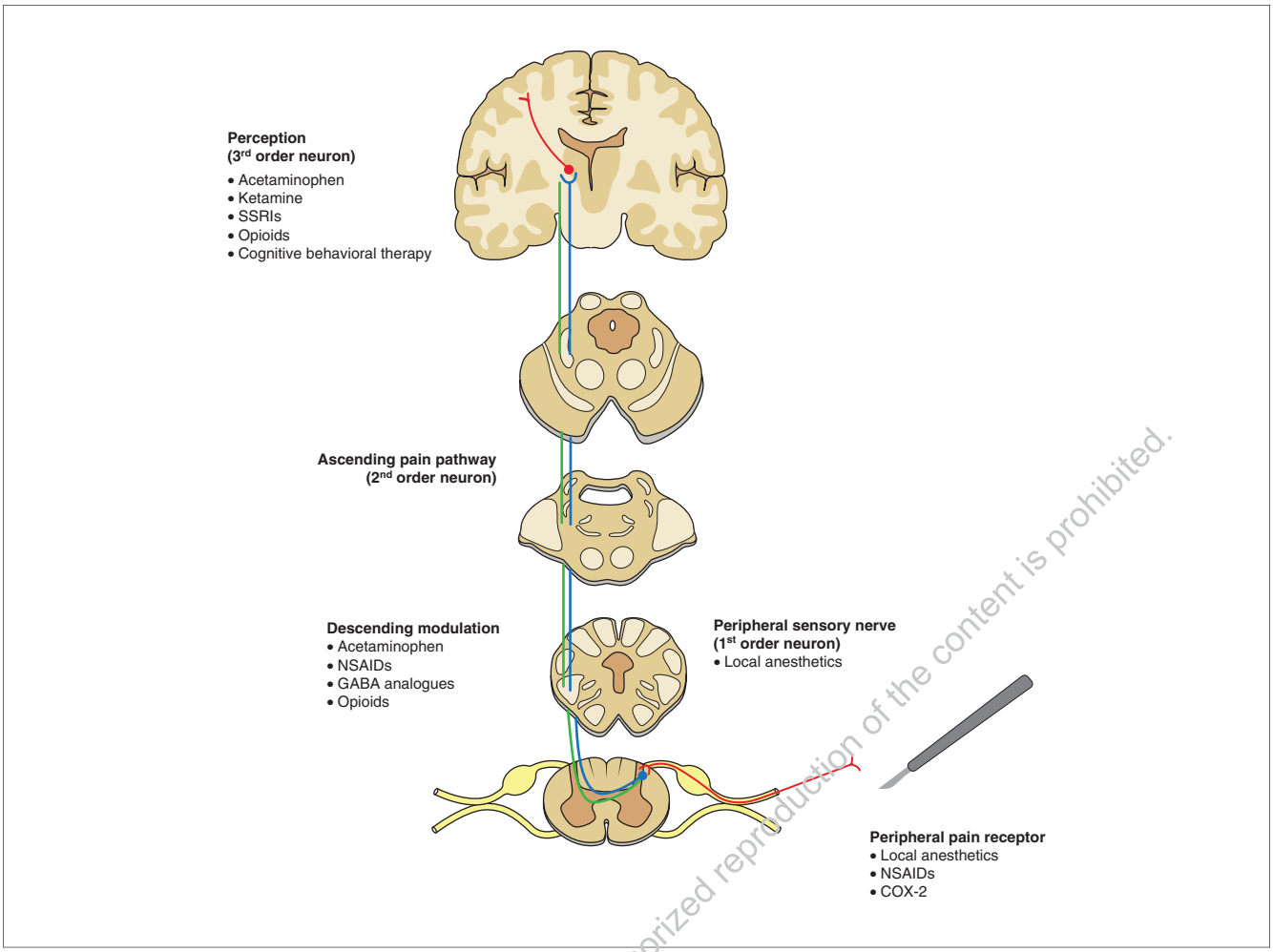

FIGURE 9 Schematic of pain pathway and the targeted areas of various analgesics. COX-2 = cyclooxygenase 2; GABA = gamma aminobutyric acid; SSRIs = selective serotonin reuptake inhibitors.

of these analgesics allows for dose reductions of each individual agent and may help minimize the amount of time patients take narcotics postoperatively.

NSAIDs block prostaglandin secretion, hence reducing the hypersensitivity state and inflammatory mediators which can sensitize the nociceptors. A concern when using NSAIDs in the postoperative management of spinal fusion and fracture repair is the potential inhibitory effect of NSAIDs on bone formation. However, a 2016 randomized study showed that low doses of NSAIDs did not affect fusion rates. In this instance, ketorolac, an NSAID with linear pharmacokinetics, was used at dosages less than 120 mg/d for fewer than 2 days.[17]

The transmission of pain signals from the peripheral nervous system to the central nervous system is controlled by a number of mechanisms, including the opioid and cannabinoid systems, inhibitory amino acids (such as gamma-aminobutyric acid), galanin, cholecystokinin, and nitric oxide.[16] Pregabalin and gabapentin are two frequently used gamma-aminobutyric acid analogs that act to inhibit this neuronal signaling, thereby reducing postoperative pain and opioid requirements. Moreover, NSAIDs and gamma-aminobutyric acid analogs appear to have a synergistic action, thus allowing for the dose reduction of both agents.

SUMMARY

A thorough understanding of the basic principles of neuroanatomy and physiology is crucial for a better appreciation of the mechanisms that cause spine disorders. Knowledge of the anatomic and physiologic processes related to spinal pathology help facilitate an accurate diagnosis of spine disorders and optimal treatment for affected patients.

KEY STUDY POINTS

- There are 31 pairs of spinal nerves, and the structure of each spinal root consists of motor and sensory rootlets, a dorsal root ganglion, and a spinal nerve.
- The spinal cord plays a central role in sensory, motor, and autonomic control.
- Multimodal drug regimens use agents that function on different pathways to improve pain control and limit the amount of narcotics used postoperatively.

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