Vikas V. Patel
Alpesh Patel
James S. Harrop
Evalina Burger
Editors

Spine Surgery Basics

Springer

Spine Surgery Basics

Vikas V. Patel • Alpesh Patel
James S. Harrop • Evalina Burger
Editors

Spine Surgery Basics

Springer

Editors
Vikas V. Patel
Department of Orthopaedics
University of Colorado, Denver
Aurora, CO
USA

James S. Harrop
Department of Neurosurgery
Jefferson Medical College
Philadelphia, PA
USA

Alpesh Patel
Department of Orthopaedic Surgery
Northwestern University
Chicago, IL
USA

Evalina Burger
Department of Orthopaedics
University of Colorado, Denver
Aurora, CO
USA

ISBN 978-3-642-34125-0 ISBN 978-3-642-34126-7 (eBook)
DOI 10.1007/978-3-642-34126-7
Springer Heidelberg New York Dordrecht London

Library of Congress Control Number: 2013946428

Printed on acid-free paper

Springer is part of Springer Science+Business Media (www.springer.com)

To our families for all the support and to Heidi Armbruster for all the organization to make this project possible.

Preface

The stronger the foundation, the grander the possibilities!

The purpose of this textbook is to lay the foundation of spine education for medical students, residents, fellows, and junior attendings. It provides a concise and accurate methodology to understanding and diagnosing different spine conditions followed by the basics of treatment. This textbook will, thus, provide the link between education, optimized clinical evaluation, and evidence-based decision making.

This is especially relevant today as with an enlarging and aging population, spinal diseases will become more prevalent. Already back pain is the second most common complaint for which patients seek care after the common cold. Concurrently, the past two decades have seen an explosion of technology utilization in patient care. This has led to students and naive practioners diagnosing by exclusion-based testing as opposed to using sound clinical judgement. As healthcare funding becomes limited, this current trend in utilization of special high-end testing and imaging will be unsustainable. The skills to obtain an accurate, real-time clinical diagnosis and treatment plan will be in high demand. In the ever growing plethora of diagnoses, adhering to basics will provide the knowledge and framework necessary to make the correct decision for the patient.

Aurora, CO — Vikas V. Patel
Chicago, IL — Alpesh Patel
Philadelphia, PA — James S. Harrop
Aurora, CO — Evalina Burger

Contents

Part I General

Part II Degenerative Spine

Part III Pediatric Spine

Part I

General

Anatomy

1

Christopher M.J. Cain

1.1 Overview

The normal vertebral column, excluding the coccyx, is made up of 25 segments, 7 cervical, 12 thoracic and 5 lumbar vertebrae, and the sacrum. The sagittal profile of the spine is curved with lordosis in the cervical and lumbar regions and kyphosis in the thoracic region (Fig. 1.1). The normal curvature of the spine results in C1, C7 and T12 being aligned vertically over the sacrum. This represents normal spinal balance.

1.2 Embryology

Vertebral formation begins around 3–5 weeks after fertilization of the egg, with segmentation and chondrification occurring around 6–8 weeks. Each vertebra forms from two adjacent sclerotomes. The caudal or posterior half of one sclerotome fuses with the rostral or anterior half of the adjacent one to form each vertebra, making each vertebra an inter-segment structure.

Chondrification centres appear in each mesenchymal vertebra in the 5th to 6th week. The cartilaginous centrum forms with components from each sclerotome, which usually fuse by the end of the embryonic period (from the 15th day to around the 8th week). The centrum ultimately becomes the vertebral body, and defects in formation or fusion of the centrum may result in hemi- or butterfly vertebrae.

Ossification of the vertebrae occurs in three parts, the centrum and the right and left halves of the neural arch. This process begins at the end of the embryonic period.

The two centres in the neural arches usually fuse with each other at the end of the first year and with the centrum in the third to sixth year. Costal elements form separately as the ribs in the thoracic region and articulate with the neural arches. In all other parts of the spine, costal elements become fused to the neural arches and become integrated as morphological parts of the vertebrae, principally the transverse process.

Five secondary centres of ossification appear around puberty in the upper and lower vertebral body and in the tips of the transverse and spinous processes. Ossification is usually complete by 25 years.

1.3 General Anatomy

Each mobile segment, excluding the articulation between the occiput and C1, and C1 and C2, articulates via a three-joint complex. Anteriorly, there is a fibrous articulation via the intervertebral disc, comprised of an outer tough fibrous *annulus fibrosus* and the central *nucleus pulposus*, and posteriorly two synovial facet or *zygapophyseal* joints.

C.M.J. Cain
Department of Orthopaedics,
University of Colorado School of Medicine,
12631 East 17th Avenue, MS B202 Room 4611,
Aurora, CO 80045, USA
e-mail: christopher.cain@ucdenver.edu

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_1, © Springer-Verlag Berlin Heidelberg 2014

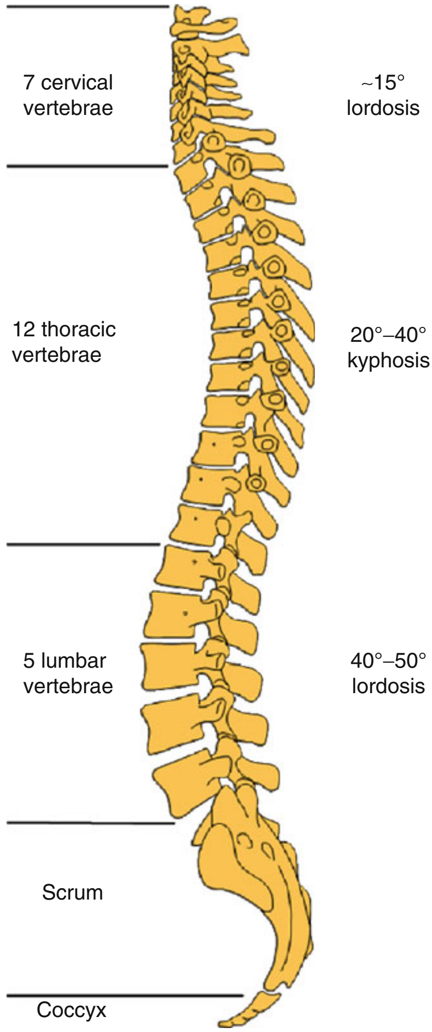


Fig. 1.1 The sagittal profile of the spine showing the normal range of lordosis in the cervical and lumbar regions, and kyphosis in the thoracic region

Ligamentous elements are vital in relation to the maintenance of intervertebral stability, the intervertebral disc being the most significant of these structures, but other elements such as the anterior and posterior longitudinal ligaments extend from the occipital bone to the coccyx. The ligamentum flava extends from the lower half of the anterior aspect of one lamina to the upper border of the lamina below and blends laterally with the facet joint capsule. Thus, for the inferior half of the lamina, the ligamentum flavum sits between the lamina and the dura. The interspinous and supraspinous ligaments are important, along with the posterior longitudinal ligament and facet capsule, in providing a posterior tension band to resist excessive distraction of the posterior elements in flexion. The anterior annulus fibrosus and anterior longitudinal ligament are the principal structures resisting hyperextension. Just about all ligaments act in some way to resist torsion, but it is the annulus and the orientation of the facet joints along with their capsule that are the primary structures resisting this movement.

Muscular elements cannot be ignored when considering both the stability and function of the spine. Details of the origins, insertions, function and relevance in relation to surgical approaches to the spine is beyond the scope of this chapter. Suffice to say that without the maintenance of balanced, toned and appropriately coordinated muscle activity, the function and stability of the spine may be significantly compromised.

1.3.1 Upper Cervical Vertebrae (Occiput to C2)

The first and second cervical vertebrae are atypical in both their structure and function compared to the other cervical vertebrae. Weight bearing between them and the base of the skull is not via the vertebral bodies and intervening disc, like the other vertebrae, but rather via articulations that enable greater movement than other individual motion segments of the spine.

1.3.2 The Atlas (C1)

This vertebra lacks a centrum, or 'body', since it is fused with the centrum of C2 to form the odontoid process or 'dens'. The neural arch on each side is thick and strong and articulates with the occipital condyles of the skull (Fig. 1.2).

The atlanto-occipital joint is a synovial joint between the convex occipital condyle and concave lateral mass of the atlas. This joint allows very little lateral bending or rotation, but a reasonable range of flexion and extension (Table 1.1). In fact, a significant proportion of cervical flexion and extension motion comes from the occiput–C1 junction.

1.3.3 The Axis (C2)

This vertebra is characterized by three main features, the odontoid process or 'dens', the lateral masses with broad superior articular surfaces for articulation with the inferior aspect of the atlas and a large and strong spinous process (Fig. 1.3).

The atlantoaxial joints are synovial joints, one on each side of the dens between the lateral masses of each vertebra and one between the anterior surface of the dens and the posterior aspect of the anterior arch of C1. This articulation provides approximately half of the rotation possible in the entire cervical spine (Table 1.1).

Accessory stabilizing ligaments provide support to the articulation between the occipital and the C1 and C2 vertebra. The *membrane tectoria* is continuous with the posterior longitudinal ligament of the spine and attaches to the back of the body of the Axis and extends up to attach to the anterior half of the foramen magnum. The *cruciform ligament* lies just anterior to the membrane tectoria with the vertical arm extending from the anterior aspect of the foramen magnum to the posterior body of C2. The transverse band attaches to the arch of inner aspect of the arch of C1 behind the dens (Fig. 1.2). The *apical ligament* lies just in front of the superior limb of the cruciform ligament, attaching the tip of the dens to the anterior margin of the foramen magnum. *Alar ligaments* pass obliquely on either side of the apical ligament to the margin of the foramen magnum (Fig. 1.4a–c). Thus, from the standpoint of ligamentous stability, the occiput C1 and C2 act as a motion segment and disruption requires treatment of all three.

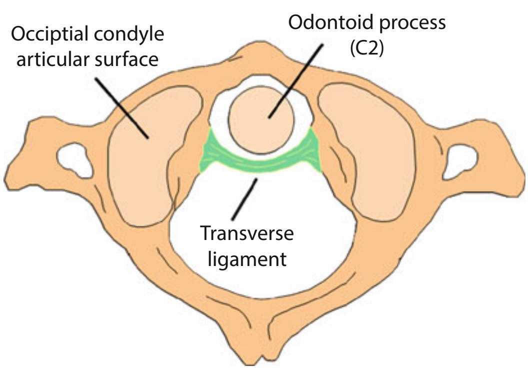


Fig. 1.2 Superior view of the Atlas vertebra (C1) showing the odontoid process of C2 contained by the transverse ligament

1.3.4 Typical Cervical Vertebrae (C3–6) and C7

An image of a typical cervical vertebra is illustrated in Fig. 1.5. During development, the costal elements form the anterior tubercle, the costotransverse bar and the tip of the posterior tubercle produce the vertebral foramen. The vertebral artery typically passes up through the vertebral foramen from C6 to C1, while the vertebral foramen in the lateral mass of C7 contains only the vertebral venous plexus. The vertebral artery passes anterior to the lateral mass of C7.

The spinous processes of the typical cervical vertebrae are usually bifid and relatively short. Spinous processes elongate in the lower segments with the C7 level being transitional between the cervical and thoracic region. The spinous process of C7, the vertebra prominens, can be easily palpated posteriorly at the base of the neck and is not typically bifid. [4]

Table 1.1 Representative values of the range of motion at each motion segment in the cervical spine [1]

Motion segment	Combined flexion/ extension (degrees)	One side lateral bending (degrees)	One side axial rotation (degrees)
C0–C1	25	5	5
C1–C2	20	5	40
C2–3	10	10	3
C3–4	15	11	7
C4–5	20	11	7
C5–6	20	8	7
C6–7	17	7	6
C7–T1	9	4	2

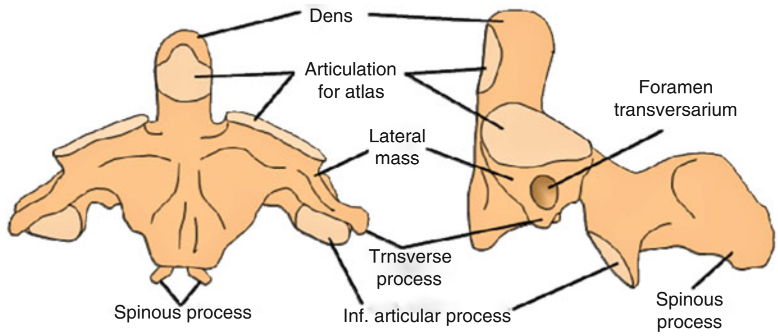


Fig. 1.3 Anterior and lateral view of the Axis vertebra (C2)

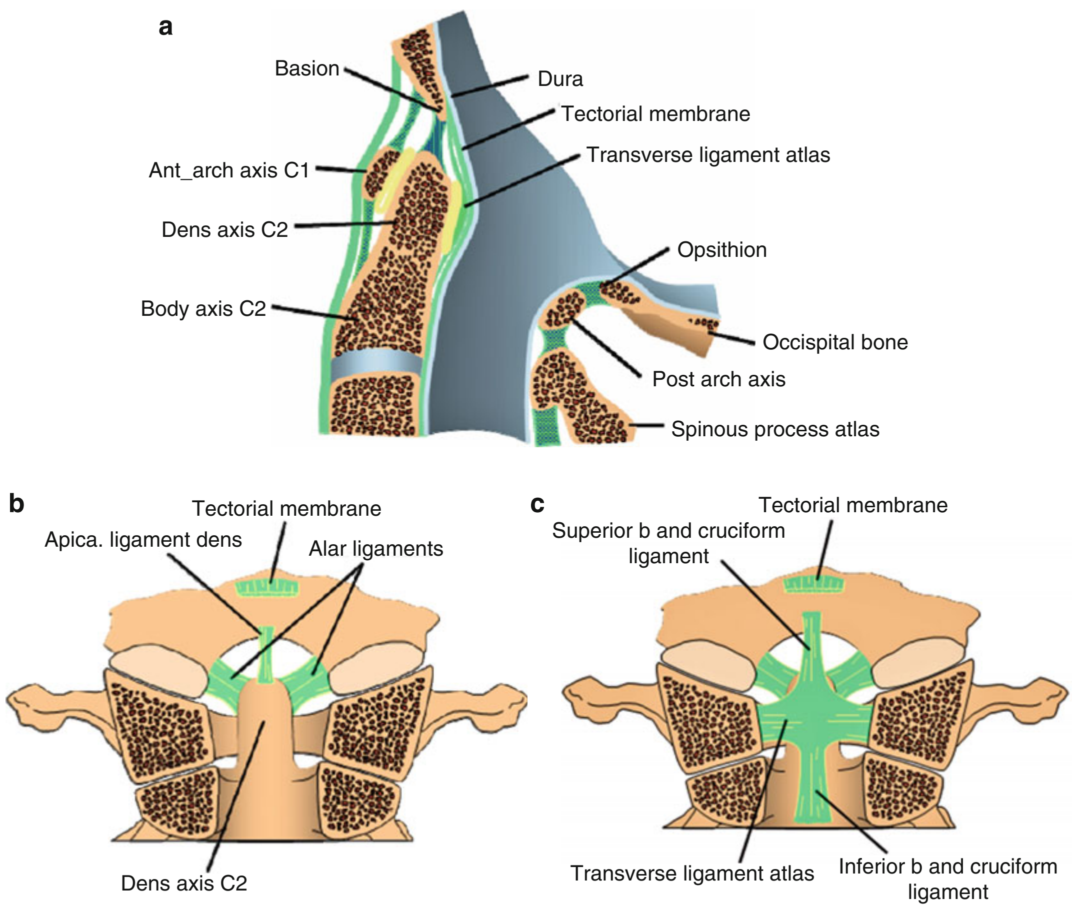


Fig. 1.4 Mid sagittal (**a**) and coronal (**b** & **c**) sectional views of the the upper cervical spine and base of the skull showing the stabilizing ligamentous structures

1.3.5 Thoracic Vertebrae

The typical thoracic vertebra is characterized by the presence of costal facets. There are six of these facets on the vertebrae from T1 to T10. Each has articular facets on each side of the posterolateral inferior and superior aspects of the body for articulation with its like-numbered rib *and* the rib below. There are also costal articular facets on the ventral tips of the transverse processes (Fig. 1.6).

The 11th and 12th vertebrae only articulate with their like-numbered rib via an articulation on the posterior part of the lateral surface of the body. The transverse processes of both the 11th and 12th vertebrae are usually stunted and are projected more directly back dorsally. The 12th thoracic vertebra is transitional between the thoracic and lumbar regions with the superior facet orientated in a way similar to other thoracic vertebrae, but the inferior facet is lumbar in type for articulation with the superior facet of L1.

1.3.6 Typical Lumbar Vertebra

Lumbar vertebra may be slightly wedge shaped, particularly at L5, with greater anterior than posterior height. More often than not, at least the upper four lumbar vertebrae show no wedging; in this case, it is the wedging of the discs that produces the normal lumbar lordosis. The width of the vertebral bodies increase from above down, with progressive widening of the articular processes, and the bodies also become more kidney shaped from proximal to distal. The transverse processes are variable in width, with the 4th usually being the longest, and the transverse process of L5 is shorter, wider and pyramidal in shape, and its base is attached further forward on the base of the pedicle. The L5 pedicle is therefore usually wider. Spinous processes are roughly horizontal (Fig. 1.7).

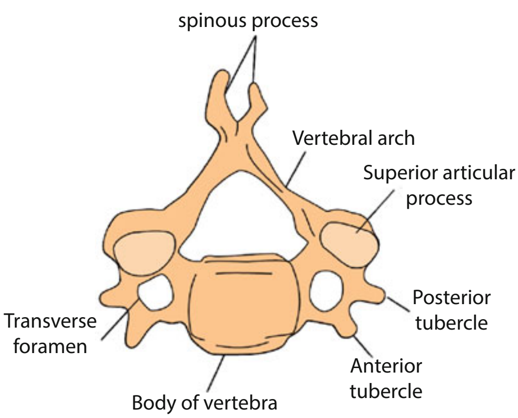


Fig. 1.5 Superior view of a "typical" cervical vertebra

1.3.7 The Sacrum

Five sacral vertebral segments fuse to form the triangular sacrum which is curved to create a concavity facing forward. The sacrum joins the spine to the pelvis and transmits the entire weight of the upper body through to the pelvis and lower limbs. The sacroiliac joint is however not a typical weight-bearing joint, as it is slung on ligaments above and behind the joint. It is these ligaments that carry the weight of the body and transmit this load to the pelvis. The upper surface of the sacrum slopes down at an angle of approximately 30° with the upper posterior surface inclined backwards before the distal portion curves down to articulate with the coccyx (Fig. 1.8).

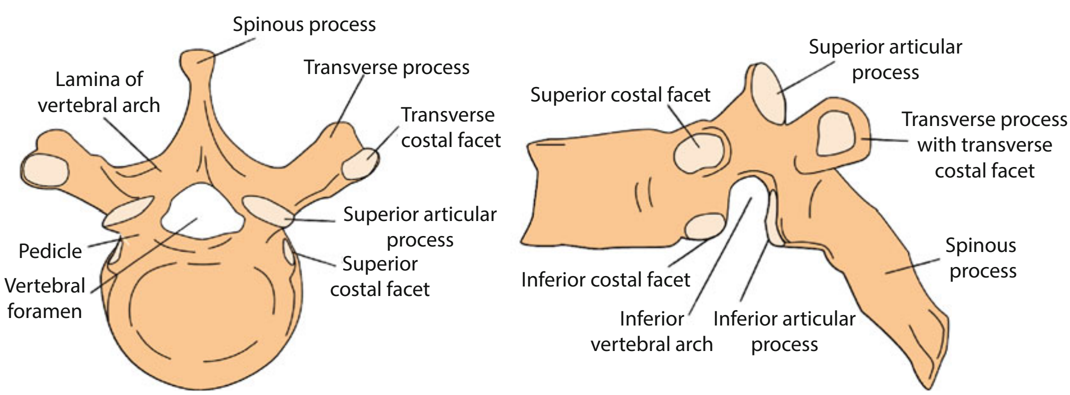


Fig. 1.6 Superior and lateral view of a "typical" thoracic vertebra

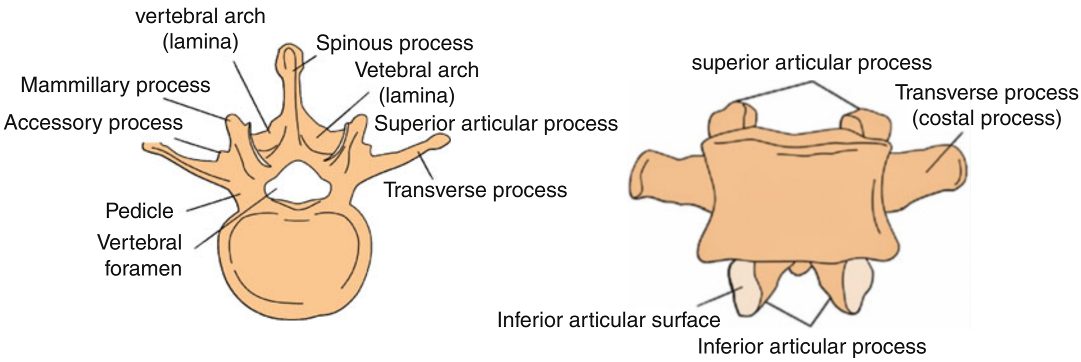


Fig. 1.7 Superior and anterior view of a "typical" lumbar vertebra

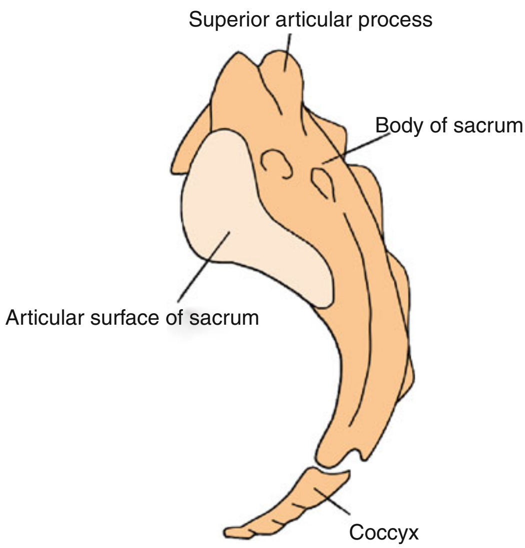


Fig. 1.8 Lateral view of the sacrum and coccyx

1.4 Range of Motion

The orientation and alignment of the facet joints in each region of the spine is a major factor in relation to the range of motion possible in each vertebral motion segment.

1.4.1 Lumbar Spine

Examining the lumbar vertebrae, it is evident that the lumbar facets are fairly vertically orientated and lie in a relatively anteroposterior plane in the upper lumbar region, with rotation into a more coronal plane as you move distally towards the lumbosacral level. These facet joints enable reasonably free flexion and extension and reasonable lateral bending, but resist anterior translation or shear and also limit the rotation that can be achieved at each level (Table 1.3).

1.4.2 Thoracic Spine

Here, the facet joints are inclined at around 60° with the superior facet facing both dorsally and laterally so that the articular surface lies on the circumference of a circle centred in the anterior vertebral body, thus enabling reasonably free rotation in the mid to upper thoracic segments. There is a transition to more lumbar type facet orientation in the lower thoracic spine which limits rotation in this region. The presence of the rib cage limits flexion and extension possible in the upper to mid thoracic region (Table 1.2).

1.4.3 Cervical Spine

Simplistically, around half of the motion in the cervical spine occurs between the occiput and C2 with the remainder distributed throughout the segments C2–T1. Facet joints from C2–3 to C7 T1 are similar to those in the thoracic spine with the exception that they both lie in the same plane, not on the circumference of a circle centred in the vertebral body. While flexion and extension are free, rotation is limited (Table 1.1).

Table 1.2 Representative values of the range of motion at each motion segment in the thoracic spine [1]

Motion segment	Combined flexion/ extension (degrees)	One side lateral bending (degrees)	One side axial rotation (degrees)
T1–T6	4	5–6	8–9
T6–T10	5–6	6	4–7
T10–L1	9–12	6–9	2–4

Table 1.3 Representative values of the range of motion at each motion segment in the lumbar spine [1]

Motion segment	Combined flexion/ extension (degrees)	One side lateral bending (degrees)	One side axial rotation (degrees)
L1–2	12	6	2
L2–3	14	6	2
L3–4	15	8	2
L4–5	16	6	2
L5–S1	17	3	1

1.5 Neuroanatomy

An important part of understanding the spine and assessing spinal disease relates to the contained neural elements and the structures they innovate. Understanding major sensory and motor innervation and basic spinal cord anatomy is paramount in determining the clinical significance of clinical and imaging findings when assessing the level of spinal cord or neurological dysfunction.

Figure 1.9 illustrate the approximate sensory innervation of the upper and lower limbs [2]

Despite the fact that there are only seven cervical vertebrae, there are eight cervical nerve roots, with the C1 root emanating from the spinal canal above the first cervical vertebra and the C8 root emerging through the C7–T1 foramen. There are 12 thoracic, 5 lumbar, 5 sacral and 1 coccygeal nerve roots, all emerging from the spinal canal below the pedicle of the vertebra of the same number.

The myotomal innervation of muscles, a myotome being the amount of muscle supplied by a single segment of the spinal cord, is a little more complicated. Last [3] has simplified, what on the surface appears to be quite complicated, into four facts:

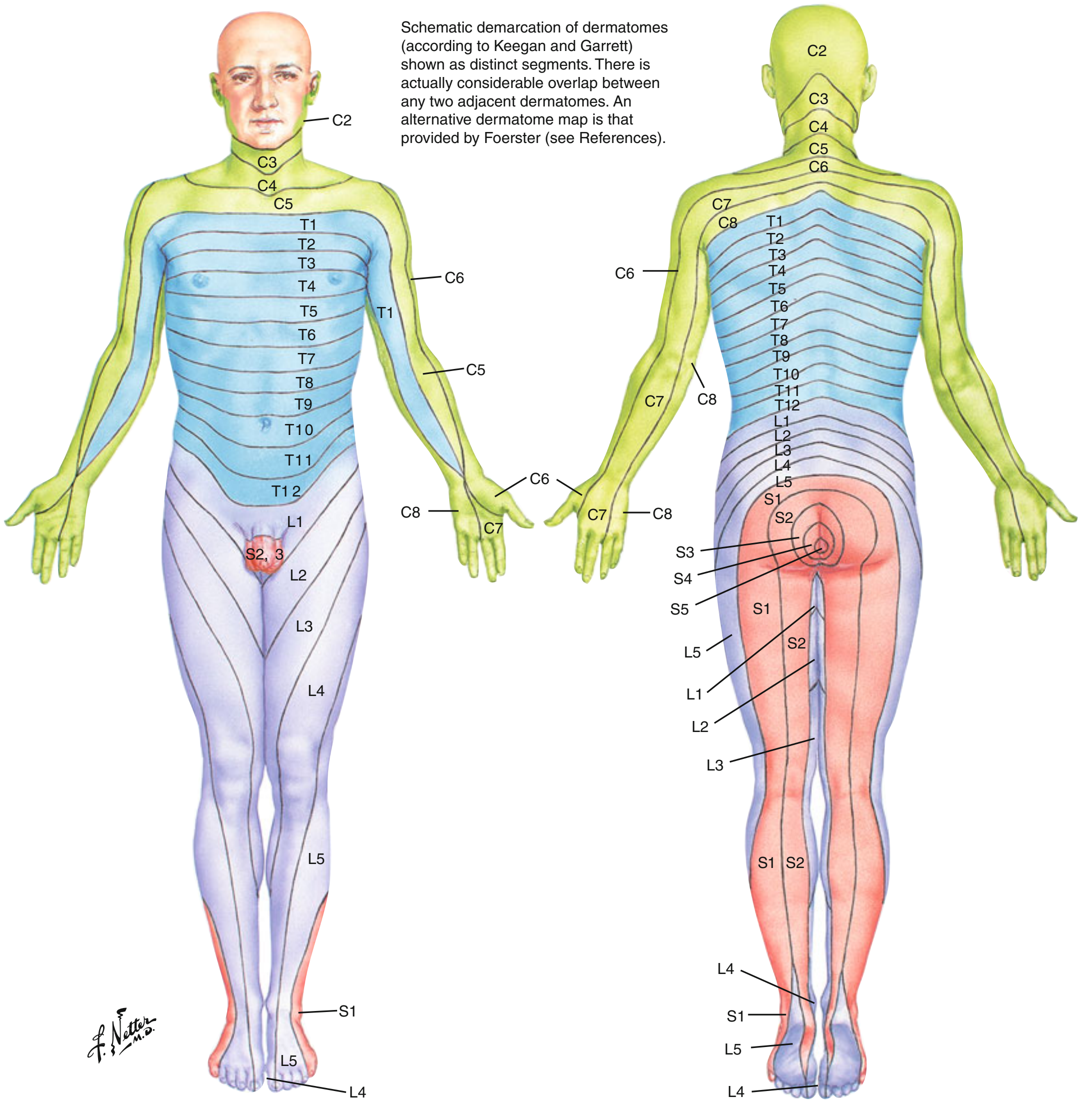
1. Most muscles are supplied equally from two adjacent segments.
2. Muscles sharing a common primary action on a joint, irrespective of their anatomical situation, are supplied by the same, usually two, segments.
3. Their opponents, sharing the opposite action on the joint, are likewise all supplied by the same, usually two, segments, and these segments usually run in numerical sequence with the former.
4. For joints more distal in the limbs, the spinal centre lies lower in the cord. For a joint one segment more distal in the limb, the centre lies, en bloc, one segment lower in the cord. This is summarized in Table 1.4.

1.6 Spinal Cord Anatomy

The spinal cord is the conduit for motor and sensory impulses between the brain and the rest of the body. It is important to have an understanding of basic spinal cord anatomy, as this has relevance in relation to assessing vertebral column and spinal cord pathology.

The spinal cord is divided into segments, corresponding to the relevant exiting nerve root. Anterior and posterior roots emanate from the spinal cord to form a segmental nerve root, with both sensory and motor components. On the dorsal root is the dorsal root ganglion, a junction box where peripheral sensory nerves synapse with spinal nerves to transmit sensory impulses to the brain.

The cross-sectional anatomy of the spinal cord is similar in each region of the cord, with



Levels of principal dermatomes

C5	Clavicles
C5, 6, 7	Lateral parts of upper limbs
C8, T1	Medial sides of upper limbs
C6	Thumb
C6, 7, 8	Hand
C8	Ring and little fingers
T4	Level of nipples
T10	Level of umbilicus
L1	Inguinal or groin regions
L1, 2, 3, 4	Anterior and inner surfaces of lower limbs
L4, 5, S1	Foot
L4	Medial side of great toe
S1, 2, L5	Posterior and outer surfaces of lower limbs
S1	Lateral margin of foot and little toe
S2, 3, 4	Perineum

Fig. 1.9 Diagram of the dermatomal distribution of the sensory innervation of the upper and low limbs. Reproduced from "Aids to the examination of the peripheral nervous system" [2]

some variation in the diameter of the cord, with enlargements in the cervical and lumbar regions of the cord to accommodate additional input and output for the upper and lower limbs.

Figure 1.10 illustrates the cross-sectional anatomy of the spinal cord. There is a central 'H'-shaped grey matter containing spinal nerve cell bodies, short interneurons, dendrites, glia

Table 1.4 Segmental innervation of movements in the upper and lower limb

Upper limb				
Shoulder	Shrug	C3,C4		
	Abduction	C5	Adduction	C6, C7 (C8)
Elbow	Flexion	C5, C6	Extension	C7, C8
Forearm	Pornation	C6	Supination	C6
Wrist	Flexion	C6, C7	Extension	C6, C7
Finger	Flexion	C7, C8	Extension	C7, C8
Intrinsic muscles	Abduction/adduction	T1		
Lower limb				
Hip	Flexion	L2, L3	Extension	L4, L5, S1
	Adduction	L2, L3	Abduction	L4, L5 (S1)
Knee	Extension	L3, L4	Flexion	L5, S1
Ankle	Dorsiflexion	L4, L5	Plantar flexion	S1, S2
	Inversion	L4	Eversion	L5, S1
Toe	Flexion	L5, S1	Extension	L5, S1
	Small muscles of foot	S1, S2		

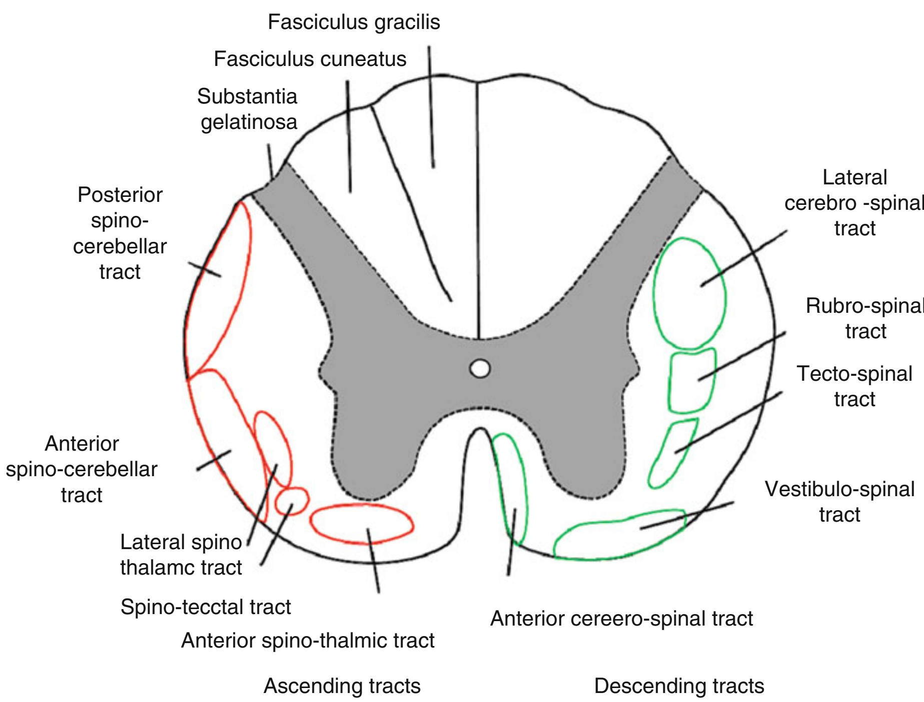


Fig. 1.10 Cross-section of the cervical spinal cord showing the approximate locations of the ascending and descending spinal tracts

and blood vessels, and an outer white matter made up of bundles of mostly myelinated longitudinal spinal tracts, glia and blood vessels. The white matter contains ascending, descending and intersegmental or connecting fibres and is divided into three main columns. The posterior column lies between the posterior grey horn and posterior median septum and contains ascending sensory fibres. The lateral column lies between the anterior and posterior grey horns and contains predominantly descending, but also some ascending tracts, and the anterior column which lies between the anterior grey horn and the anterior median fissure contains descending motor tracts.

The posterior white columns convey normal sensation, warmth and coolness and joint position or proprioception. Their cell bodies lie in the dorsal root ganglia of the spinal nerves and more distal fibres, from the sacrum, lie medially, with fibres from the lumbar, thoracic and cervical regions layered more laterally. Sensory fibres synapse in the nucleus gracilis and cuneatus near the base of the fourth ventricle in the medulla oblongata and cross to the opposite side of the brain via the sensory decussation.

Anterior white columns contain uncrossed pyramidal fibres whose cell bodies lie in the brainstem near the floor of the fourth ventricle. Motor fibres from the cerebral cortex cross in the motor or pyramidal decussation, also in the medulla oblongata.

Pain and temperature fibres entering the cord via the posterior spinal roots enter the dorsal horn of the grey matter synapse and cross the spinal cord to the lateral spinothalamic tract on the opposite side. As a result of this, hemisection of the spinal cord results in a dissociated sensory loss, with loss of joint position and light touch sensation, along with motor function, on the same side as the cord injury, with loss of pain and temperature on the opposite side of the body below the lesion.

Neurons are also layered in the various tracts, with sensory fibres entering the cord first, distally, lying closest to the midline, and those entering last, in the cervical region, lying more laterally. The same is also true for motor tracts, with those leaving the cord first, cervical fibres, lying more laterally. This arrangement leads to the typical features of a central cord lesion that may result from stenosis and a hyperextension injury, conditions such as a syringomyelia and spinal cord tumours, where motor tracts are affected more than sensory, the upper limb more than the lower limb, and distal parts of the limb more than proximal.

Anterior spinal cord pathology such as anterior spinal artery occlusion, compression due to a kyphotic deformity or a central disc protrusion will result in anterior cord syndrome where there is loss of motor function and pain and temperature below the lesion with preserved posterior column function.

References

1. Panjabi M, White A (1990) Clinical biomechanics of the spine, 2nd edn. J.B Lippincott Company, Philadelphia
2. Medical Research Council (1976) Aids to the examination of the peripheral nervous system. Her Majesty's Stationery Office, London
3. Last R (1984) Anatomy, regional and applied, 7th edn. Churchill Livingstone, Edinburgh
4. Clemente C (1976) Anatomy: a regional atlas of the human body. Urban & Schwarzenberg, Baltimore

2 Physical Examination of the Cervical Spine

Virgilio Matheus and Edward C. Benzel

2.1 Anatomy of the Cervical Spine

The cervical spine is gifted with the capacity to provide a wide range of motions which facilitate head movements. Based on its functions, the cervical spine can be divided into two segments: an upper portion, which involves the occipitoatlantoaxial complex, and a lower portion, consisting of C3–C7.

Due to the unique anatomical features associated with the atlas and the axis, the upper segment of the cervical spine forms an extremely versatile and complex articulation that allows for a wide range of head and neck movements.

The atlas is formed by a ring of bone, which can be divided into a ventral and dorsal arch. It lacks a central vertebral body but displays large lateral masses. The latter serve to accommodate the occipital condyles and form the only weight-bearing articulation between the skull and the spine. A small flattening at the rostral border of the dorsal arch represents the trough through which the vertebral artery passes over C1 on its trajectory toward the intradural space.

The axis resembles the typical cervical vertebra with the peculiarity of having a ventral bony process projecting rostrally from its rudimentary vertebral body known as the odontoid process or dens. This process serves as an anchoring point for several ligaments that provide support between the atlas, axis, and condyles. This ligamentous complex is referred to as the cruciate ligament complex. Added stability is provided by the anterior and posterior longitudinal ligaments, which run ventral to and dorsal to the vertebral bodies, up to the skull base. The C1–C2 segment lacks an intervertebral disc. The most rostral disc is, hence, located between the axis and C3. Usually the spinous processes of C2 through C6 display a bifid appearance. In the majority of the cases, the vertebral artery enters a bony ring on the lateral aspect of C6 known as the transverse foramen. The artery follows this path rostrally until exiting the foramen of the axis and curving over the arch of the atlas - to finally pass between the atlas and the condyle as it passes through the foramen magnum.

In addition to the ligamentous support, the cervical spine relies on muscular support for both support and mobility. A combination of unique features exhibited by these muscles and the cervical spine permits extreme flexion, extension, and tilting of the head, without adverse consequences.

2.2 Palpation

The ventral and lateral aspects of the cervical spine are covered by surrounding structures that can lead or suggest potential underlying pathologies, which in some cases might show no relationship with cervical spine pathology. Prior to laying

V. Matheus • E.C. Benzel, M.D. (✉)
Department of Neurosurgery,
Neurological Institute, Cleveland Clinic,
9500 Euclid Avenue, S4, Cleveland, OH 44195, USA
e-mail: benzele@ccf.org; matheuv@ccf.org

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_2, © Springer-Verlag Berlin Heidelberg 2014

hands on the patient, the examiner must look for points of skin erythema or diaphoresis, which could represent painful areas that must be approached carefully to avoid unnecessary pain [1]. Careful palpation of the sternocleidomastoid muscle and all of the triangles it forms is performed next. One should begin by instructing the patient to turn his head toward one side, after which the examiner proceeds to "travel" with his hand along the full extent of the ipsilateral muscle. The examiner should look for masses and tender points. He should repeat this maneuver on the opposite side. It is important to compare muscle bulk and appearance. Next, the examiner should proceed with palpation of the carotid pulse, using the second and third digits. It is normally located medial to the sternocleidomastoid muscle. The examiner should pay attention to strength and symmetry, and to not forget to auscultate the underlying structures, seeking bruits, etc. It is also important to assess anatomical landmarks relevant for surgical approaches such as the thyroid and cricoid cartilages, hyoid bone, and trachea as well as, if possible, palpate the carotid tubercle which usually is palpable at the C6 level. The examiner should palpate, with the same fingers, the supraclavicular region. Abnormal masses or tenderness may represent lymphadenopathy, apical lung masses, or even clavicular fractures. If the palpable structure seems to be bony, suspect an accessory cervical rib.

When palpating the dorsal aspect of the cervical spine, one must remember that the cervical spine is covered by large muscles, most notably the splenius and trapezius muscles, which have insertions on the suboccipital, scapular, and shoulder regions. It, therefore, is important to begin palpating from the occiput down to the cervicothoracic region as well as lateral over the scapula. It is useful to use a systematic approach, first examining the soft tissue and subsequently the bony structures or vice versa. Inflammation and tenderness can be due to muscle spasm. Potential etiologies include trauma, muscle fibrosis, and fibromyalgia. Reproducible focal points of tenderness, with palpation over the scapula or shoulder joint, may indicate ligamentous damage due to overuse.

Finally, one should proceed to palpating the spinous processes. The patient should sit up and perform gentle flexion of the neck. Palpation of spinous process in the midline is appropriate. Tenderness, masses, absence of processes, or any other abnormalities should be noted. One should pay close attention to alignment and tenderness, so underlying fractures and/or luxations are not missed.

2.3 Range of Motion

The most important step prior to performing a range of motion examination is to obtain a thorough history to assess for instability. If the patient is aware of specific painful movements, elicitation of such movements should be reserved for the latter portion of the examination in order to avoid muscle spasm and the carrying of the pain through the remaining steps of the examination. Flexion, extension, lateral flexion, and head rotation are performed in order to seek sources of pain. One should begin with active movements and follow them with passive movements. Compare among them for differences. While performing these movements, the examiner should assess for resistive isometric muscle testing (resistive strength). Careful attention should be paid to pain associated with specific muscles, as well as weakness and/or atrophy - both of which may indicate a muscle strain or a neurological injury [2].

Flexion: Instruct the patient to bring the chin down to the sternum without flexing the chest.

Extension: Instruct the patient to bring the head back without extending his chest. The mouth can be kept open to avoid traction over the anterior neck structures.

Lateral rotation: Instruct the patient to rotate his head to each side at the time. The chin should be above the shoulder joint at the point of maximal rotation (approx. 80–90°). Asymmetry on rotation should raise concern for an underlying problem.

Lateral bending: Instruct the patient to bend his neck sideways, without performing any neck rotation or raising the ipsilateral shoulder. The ear should touch or almost reach the shoulder joint. Pay careful attention to asymmetry while performing the maneuver.

2.4 Fractures

Fractures can be classified as stable vs. unstable, with or without compromise of the spinal canal. If suspicion of a fracture is present, imaging of the spine should be obtained prior to performing any manipulation. Signs of underlying fracture include pain, muscle spasm, limited range of motion, neurological dysfunction, and any obvious deformity. One should pay close attention to the mannerisms of the patient; with severe injuries, it is not uncommon to observe a patient holding his head with his hands, in an attempt to provide extra support and self-limit the range of motion.

Following observation of the neck by the examiner, gentle percussion of the spine can be performed with the patient in the sitting position (assuming that the spine has been otherwise cleared) with his head gently tilted forward. During this test, the development of pain and/or neurological symptoms can represent an underlying fractured vertebra. This test is very nonspecific and may be positive in cases of a ligamentous sprain or strain. Paraspinal muscle percussion can elicit pain in many cases of muscle strain.

2.5 Instability

Similar to fractures, instability usually arises as consequence of an underlying trauma or a degenerative or infection-related process. Instability may be occult or obvious. It, nevertheless, is imperative that imaging tests be performed prior to attempting the maneuvers that are presented here [3].

2.6 Vascular Assessment

2.6.1 Vertebrobasilar Circulation

It is imperative to assess for normal posterior circulation in a patient with whom cervical traction or manipulation is planned. The posterior circulation is most vulnerable with rotation of C1 over C2. Under normal circumstances, the vertebral artery can be compromised with rotation from 30 to 45°, thus collapsing the contralateral vertebral artery. Provocative or functional testing can compress the circulation at several points between the foramen magnum and the transverse process of C6. This compression can be due to rotation itself but also may be due to underlying spondylotic alterations of the uncinate joints. Auscultation for bruits and palpation for pulses are an integral part of the examination.

After performing any test, it is important to provide an examination free time interval in order to prevent confusing any latent symptoms with symptoms elicited by performing maneuver. Signs and symptoms of posterior circulation insufficiency include vertigo, lightheadedness, diplopia, dysarthria, dysphagia, gait ataxia, nausea, and paresthesias. Many different stress-inducing maneuvers are described, most of them involving head rotation with extension.

2.6.2 Subclavian Artery

Both subclavian arteries, after branching off the aorta, eventually give rise to the vertebral arteries in most people. Compromise of this vessel results in symptoms in the upper extremities that may mimic cervical lesions as well as symptoms of posterior circulation insufficiency. Compression of the subclavian artery may arise from hypertrophy or spasm of the anterior scalene muscle, atherosclerotic plaque, and apical lung masses. Symptoms include arm pain, cold limb, supraclavicular region pain, and paresthesias [4].

With the patient in the seated position, the blood pressure is taken in both arms. There should be no more than 10 mmHg difference between them. If the difference is greater than 10 mm and the radial pulse is weak, subclavian artery compromise should be considered. One should also auscultate the supraclavicular area in search of a bruit. If the index of suspicion for pathology is elevated, one may proceed to imaging of the chest (X-rays, CT, MRI) and/or vascular imaging (US, CTA, MRA).

2.7 Neurologic Assessment

2.7.1 C1–C4

A lesion at this level will compromise the innervations to the diaphragm, often resulting in the need for ventilator support.

2.7.1.1 Motor

Scapular elevation (C3–C4). To assess its integrity, the examiner stands behind the patient and instructs him to shrug his shoulders. He then places his hands over the shoulders – pushing them downward. In normal conditions, one should not be able to force the shoulders downward. One should also pay careful attention to asymmetry during the elevation phase.

2.7.1.2 Sensory

The C1–C4 dermatomes provide sensation to the back of the head and neck (Fig. 2.1).

2.7.2 C5

2.7.2.1 Motor

The most recognized specific function of C5 involves arm abduction through innervation of the deltoid muscle. The examiner should instruct the patient to keep his arm resting lateralized to his body, with the elbow flexed to 90°. The examiner should place his hand over the lateral shoulder region and instruct the patient to bring his arm up to the side away from his body until perpendicular to the chest cavity.

It also provides control over the internal and external rotation of the shoulder. Internal rotation is less reliable since muscles involve in carrying out this function receive also innervations from C6, C7, C8, and T1. Elbow flexion is also supplied by these two roots through the functions of the biceps, brachioradialis, and supinator muscles. Ask the patient to sit down and keep his arm flexed in a 90° posture while you hold it with one hand under the elbow and the other under his wrist. Instruct him to perform an internal and external rotation of the shoulder while you assess his tone and strength. Following this, ask him to flex his arm from the resting position at 90° attempting to reach for his shoulder.

2.7.2.2 Sensory

The C5 root supplies, through the axillary nerve, sensation to the upper lateral arm (Fig. 2.1). The bicipital reflex (C5) also should be tested. Instruct the patient to keep his arm at a 90° flexion, resting over your arm while you gently strike with the reflex hammer over the biceps insertion tendon. Compare both sides always.

2.7.3 C6

2.7.3.1 Motor

Wrist extension is predominantly mediated by the extensor carpi ulnaris and radialis. The examiner should have the patient rest his forearm over the examiner's nondominant hand. With your dominant hand over the dorsum of his hand, instruct him to extend his wrist without and then with resistance from your overlaying hand.

2.7.3.2 Sensory

Through its contributions to the musculocutaneous nerve, it provides sensation to the lateral aspect of the forearm and the two first digits.

2.7.4 C7

2.7.4.1 Motor

Its primary function is to elicit elbow extension through contraction of the triceps muscle. The examiner should instruct the patient to hold his hand at his face level with his elbow flexed as in a boxing position. The examiner should grab the patient's elbow with his nondominant hand to prevent usage of other muscles. The examiner should place his dominant hand over the patient's wrist and instruct him to perform extension of his elbow. The examiner should compare without and with resistance bilaterally.

2.7.4.2 Sensory

This root provides sensation to a narrow area of the hand being most specific over the volar region of the middle finger.

Fig. 2.1 Dermatomes of the cervical and brachial plexus

2.7.5 C8

2.7.5.1 Motor

C8 function refers to the flexion of the fingers. This function is mediated through the flexor digitorum and lumbrical muscles. The examiner should instruct the patient to flex his fingers. Then, the patient should be asked to attempt extension, with and without resistance from the examiner's fingers.

2.7.5.2 Sensory

The dermatome to this root is localized over the fifth digit and lateral aspect of the fourth digit (Fig. 2.1).

2.7.6 T1

2.7.6.1 Motor

T1 controls abduction of the fingers through innervations of the dorsal interossei and adduction through innervations of the palmar interossei. To test for abduction, the patient is instructed to spread apart his fingers. The examiner should pinch together every set of fingers to try to force them together. To test for adduction, the examiner should instruct the patient to keep his fingers together on extension after the examiner places a piece of paper between them and pulls it out.

2.7.6.2 Sensory

Sensation over the medial aspect of the forearm (Fig. 2.1).

2.8 Miscellaneous

"Space-occupying para- and intraspinal lesions" can present in many ways, including neurological deficits. Specific tests can be performed during the physical examination to exacerbate these symptoms and confirm the presence of one of these lesions. Unspecific symptoms patients can complain of include neck pain and paresthesias of the upper and lower extremities.

2.8.1 Valsalva Maneuver

With the patient in sitting position, instruct the patient to hold his breath and bear down as if defecating. Inquire about worsening symptoms. This maneuver will raise the intrathecal pressure and possibly exacerbate any symptoms caused by the compressive intraspinal, particularly intradural, lesion [5].

It is important to evaluate the patient's swallowing function during the physical examination. Patients may complain of dysphagia or odynophagia that could be due to an expansive cervical spine mass compressing the esophagus. These and other pathological findings that are observed during swallowing could be manifestations of cranial nerve compression.

2.8.2 Cervical Neural Compression

Both spinal cord and nerve root compression can lead to neurologic compromise. Such may be the case with herniated discs, osteophytes, fractures, luxations, or tumors. Patients with neural compression and/or irritation may complain of cervicalgia, radicular pain, paresthesias, weakness, and myelopathy. It goes without saying that when suspecting high-grade neural compression, one should complement the history and physical examination with the pertinent imaging studies. The following tests can help clinically localize the offending pathology.

2.8.3 Foraminal Compression Test

With the patient in the sitting position and the head in a neutral position, the application of strong downward pressure with both hands for a few seconds can elicit radicular symptoms. Repeating these steps with the patient's head rotated to each side can increase sensitivity.

By applying axial loading, the intervertebral disc is compressed, the foraminal cross-sectional area should decrease, and pressure will hence be exerted upon the apophyseal joints. If the patient develops symptoms or worsening of the preexisting symptoms, the dermatome should be relatively identifiable based on classical dermatomal distributions.

2.8.4 Extension Compression Test

With the patient in the sitting position, he is asked to extend his neck. The examiner then applies his hands on the forehead and applies downward pressure. Such axial loading in an extended spine results in compression of the dorsal apophyseal joints and thus results in the worsening of existing or the development of localized pain related to joint disease. Simultaneously, it decreases the cross-sectional area of the foraminal space which may result in radicular pain.

2.8.5 Flexion Compression Test

The examiner asks the patient to flex his head while in the sitting position. He then applies downward pressure on the cranial vertex. With the head flexed and with axial loading, the compression of the ventral aspect of the disc induces dorsal displacement of a bulging disc into the central canal, thus potentially causing symptoms related to compression of the spinal cord. At the same time, pressure is taken off the dorsal apophyseal joints. Hence, preexisting facet origin pain may improve.

2.8.6 Spurling's Test

With the patient in a sitting position, the examiner applies downward pressure over the patient's head while maintaining a lateral flexed posture (Fig. 2.2). If radicular pain is elicited, the test is considered positive. If no symptoms are elicited, the patient is asked to assume a neutral position. A moderate blow is delivered to the head vertex (Fig. 2.3). With lateral flexion, pressure is applied over the apophyseal joints, worsening any related pain.

2.8.7 Maximal Foraminal Compression Test

With the patient in the sitting position, he is asked to extend his neck while rotating his head. This test exerts compression over the dorsal apophyseal

Fig. 2.2 Spurling's test: Examiner applies downward pressure over the patient's head while maintaining a lateral flexed posture which applies pressure over the apophyseal joints exacerbating any related pain

Fig. 2.3 Spurling's test: If no symptoms are elicited during the initial step of this maneuver the patient is asked to assume a neutral position. A moderate blow is delivered to the head vertex (*arrow*). Symptoms that might not be elicited by the first step of this maneuver might become evident following this step

joints and compresses the foraminal spaces, thus exacerbating pain related to nerve root encroachment [6].

2.8.8 L'hermitte's Phenomenon

With the patient in a relaxed seated position, he is asked to perform head flexion. This results in stressing of the dorsal ligaments and elements of the spine plus compression of the ventral segment of the intervertebral disc. This in turn displaces dorsal disc bulges into the central canal, while the flexion stretches the spinal cord over the ventral compressive masses (sagittal bowstring effect). A positive test involves the development of sudden electrical tingling or shocks down the spine and/or extremities. Such a finding is consistent with significant stenosis and is a sign of myelopathy. Local cervical pain during the test could represent muscle sprain, meningeal irritation from an underlying inflammatory process, apophyseal joint disease, or radiculopathy [7].

Fig. 2.4 Distraction test: Vertical traction to the head is applied (*arrow*) which removes the pressure from the joints and enlarges the foraminal spaces, resulting in the alleviation of the symptoms in cases of joint disease, foraminal root encroachment, and disc herniation in the case of radicular symptoms

2.8.9 Distraction Test

The patient is asked to assume the sitting position. The examiner places his palms over the mastoid processes of the patient bilaterally. Vertical traction to the head is then applied. This maneuver removes the pressure from the joints and enlarges the foraminal spaces, resulting in the alleviation of the symptoms in cases of joint disease, foraminal root encroachment, and disc herniation in the case of radicular symptoms. If pain arises during the test, suspect muscle strain or facet capsulitis (Fig. 2.4).

Table 2.1 Relevant signs and tests of the cervical spine

Name	System	Action	Pathologic finding	Reasoning
Maigne's test	Vascular	Head rotation and extension	Vertigo, dizziness, fainting	Compression of vertebral artery
Dekleyn's test	Vascular	Head extension and rotation while supine with head off the table	Vertigo, dizziness, fainting	Compression of vertebral artery
Hautant's test	Vascular	Sitting, head extension and rotation with arms extension	Vertigo, dizziness, fainting, arm drift	Compression of vertebral artery, stenosis of subclavian artery, posterior circulation insufficiency
O'Donoghue's maneuver	Muscular	Range of motion passive vs. resisted	Pain during resisted motion	Muscle strain elicits worsening pain with motion against resistance
Rust's sign	Osteomuscular/ ligamentous	Head supported by patient's own hands at all times	Self-attempt to immobilize the neck and support head's weight	Immobilization and weight limitation
Valsalva maneuver	Space-occupying lesion	Force exhalation against closed airway	Pain localized to cervical spine	Maneuver increases intrathecal pressure
Jackson's compression test	Radiculopathy, apophyseal joint disease	Seated, lateral neck flexion, downward pressure	Radicular symptoms +/– local pain	Foraminal compression, disc compression, facet joint loading
Extension compression test	Radiculopathy, apophyseal joint disease	Seated, neck and head extension, downward pressure	Radicular symptoms +/– local pain	Foraminal compression, facet joint loading, decreased loading over dorsal annular disc. Relief of symptoms could represent disc material displacement ventrally
Flexion compression test	Radiculopathy, apophyseal joint disease	Seated, head and neck flexion, downward pressure	Radicular symptoms +/– local pain.	Foraminal compression, disc compression, decreased loading over facet joints. Relief of symptoms could represent joint disease
Spurling's test	Radiculopathy, apophyseal joint disease	Seated, same as Jackson's test. If negative proceed with head vertical blow on neutral position	Radicular symptoms +/– local pain	Foraminal compression, intervertebral disc compression, facet joint loading
Distraction test	Radiculopathy, apophyseal joint disease	Seated, upward traction of patient's head	Relief of radicular symptoms +/– local pain	Expansion of foramen, unloading of disc, unloading of facet joints
L'hermitte's sign	Myelopathy	Seated, passive neck and head flexion	Electrical tingling down the spine and/or extremities	Traction of the spinal cord

2.8.10 Bakody's Sign/Shoulder Abduction Test

With the patient in the sitting position, the examiner instructs the patient to place his hand over his head – thus keeping his arm in abduction. This maneuver reduces stretch on the lower trunk of the brachial plexus and relaxes tension on tethered nerve roots at the foraminal level. Amelioration of the pain usually represents extradural compression of a root around C6–T1 (Table 2.1).

References

1. Albert T, Vaccaro A (2005) Physical examination of the spine, 1st edn. Thieme, New York
2. Buckup K (2004) Clinical test for the musculoskeletal system: examinations-signs-phenomena, 1st edn. Thieme, New York
3. Vaccaro A (2003) Fractures of the cervical, thoracic and lumbar spine, 1st edn. Marcel Dekker, New York
4. Greenberg M (2010) Handbook of neurosurgery, 7th edn. Thieme, New York
5. Fuller G (2008) Neurological examination made easy, 4th edn. Churchill Livingstone, New York
6. Cipriano JJ (2003) Photographic manual of regional orthopaedic and neurological tests, 4th edn. Lippincott Williams & Wilkins, Philadelphia
7. Lin V (2010) Spinal cord medicine, principles and practice, 2nd edn. Demoes Medical, New York

Examination of Back Pain

3

Jennifer Malone, Ravi Madineni, Niti Cooper, Angud Mehdi, and Ashwini Sharan

Low back pain is a common musculoskeletal disorder affecting 60–80 % of people at some point in their lives. In the USA it is the most common cause of job-related disability, a leading cause of missed work. Low back pain is classified as acute (less than 4 weeks), subacute (4–12 weeks), or chronic (greater than 12 weeks) [1].

Understanding the causes of low back pain, performing a thorough history and physical examination, and looking for "red flags" for potentially serious conditions allow health-care providers to accurately classify and treat most causes of back pain [2].

The majority of lower back pain is nonspecific and arises from mechanical soft tissue sprain or strain and can be treated within a few weeks of onset with conservative management. In addition to spinal or mechanical causes, lower back pain can arise from nonmechanical etiologies such as failed back syndrome, visceral pain, and multitude of other non-spinal causes [3, 4]. A comprehensive listing of the various etiologies of lumbosacral pain is included for the clinician's consideration.

J. Malone • R. Madineni • A. Mehdi • A. Sharan (✉)
Department of Neurosurgery,
Thomas Jefferson University Hospital,
909 Walnut Street 2nd floor,
Philadelphia, PA 19107, USA
e-mail: ashwini.sharan@jefferson.edu

N. Cooper
Department of Anesthesiology,
Cooper University Hospital, 1 Cooper Plz,
123 Camden, NJ 08103, USA
e-mail: niti-cooper@cooperhealth.edu

3.1 Physical Examination

Successful evaluation of low back pain begins with a thorough history and physical examination leading to an appropriate diagnosis. In performing a physical exam, one can utilize a process of elimination as dictated by the history to uncover the diagnosis and treat it effectively. The patient history should uncover whether there is systemic disease, neurologic impairment that may necessitate surgery, and social or psychological disease that can intensify or prolong pain.

Ask the patient about the onset of pain (sudden, gradual, fleeting), the location of pain (have the patient point to the area of pain or trace with one finger the pain pattern if the pain radiates), the duration of pain (the length of time the pain has been present), and the characterization of pain (have them use adjectives or descriptive words such as "aching," "burning," "sharp," and "electrical"); ask about alleviating and aggravating factors, timing (constant, intermittent), and history (previous history of current symptoms); and inquire about the mechanical nature of the pain (differences in laying, standing, sitting, pain worse on extension).

As with any other physical examination, vital signs as well as height, weight, and assessment of body mass index (BMI) are essential in evaluating the patient. A survey of the patient's skin may reveal surgical scars, lesions from herpes zoster, injection sites (hinting to history of drug abuse), or even undiagnosed cancer areas. Areas of hair loss, skin and nail changes, erythema, and

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_3, © Springer-Verlag Berlin Heidelberg 2014

cyanosis should be noted, as they may delineate the sympathetic nervous system as the source of pain. Temperature, color, and pulses should be evaluated in the legs to differentiate neurogenic claudication from vascular insufficiencies.

The spine must be palpated midline and laterally. Lateral tenderness implies possible facet disease. In addition, the sacroiliac joint which is a common source of pain can be palpated for tenderness.

3.1.1 Motor Examination

It is always imperative and helpful to evaluate baseline muscle mass and tone. The examiner must look for areas of muscle wasting, increased tone, contractures, fasciculation, and postural abnormalities. This will suggest the chronicity effects of pain and compensation mechanisms. Muscles of both the upper and lower extremities should be tested for strength and graded accordingly. This maintains a very good objective baseline on function and needs to be accurately maintained in cases of compressive spinal problems.

Grade	Clinical signs
0	No evidence of contractility
1	Slight contractility, no movement
2	Full range of motion, gravity eliminated
3	Full range of motion with gravity
4	Full range of motion against gravity, some resistance
5	Full range of motion against gravity, full resistance

3.1.2 Sensory Examination

The sensory exam should be focused to information detailed by the patient in the history. Use of tools differentiating sharp (pinprick), light touch (von Frey filament), and vibration (tuning fork) sensations may further delineate the extent of the lesion. Correlations should be made between the sights of numbness or allodynia and the dermatomal or non-dermatomal nature of the pain. In our practice, we find it particularly helpful to use pain maps filled out together with the patient as an aid to categorizing the neuropathic versus radicular components of pain (Fig. 3.1).

3.1.3 Neurological Examination

This portion of the physical examination of the spine is the most objective. The physician should check deep tendon reflexes of the biceps (C5–6), triceps (C7–8), patellar (L3–4), and Achilles (S1) and grade them accordingly.

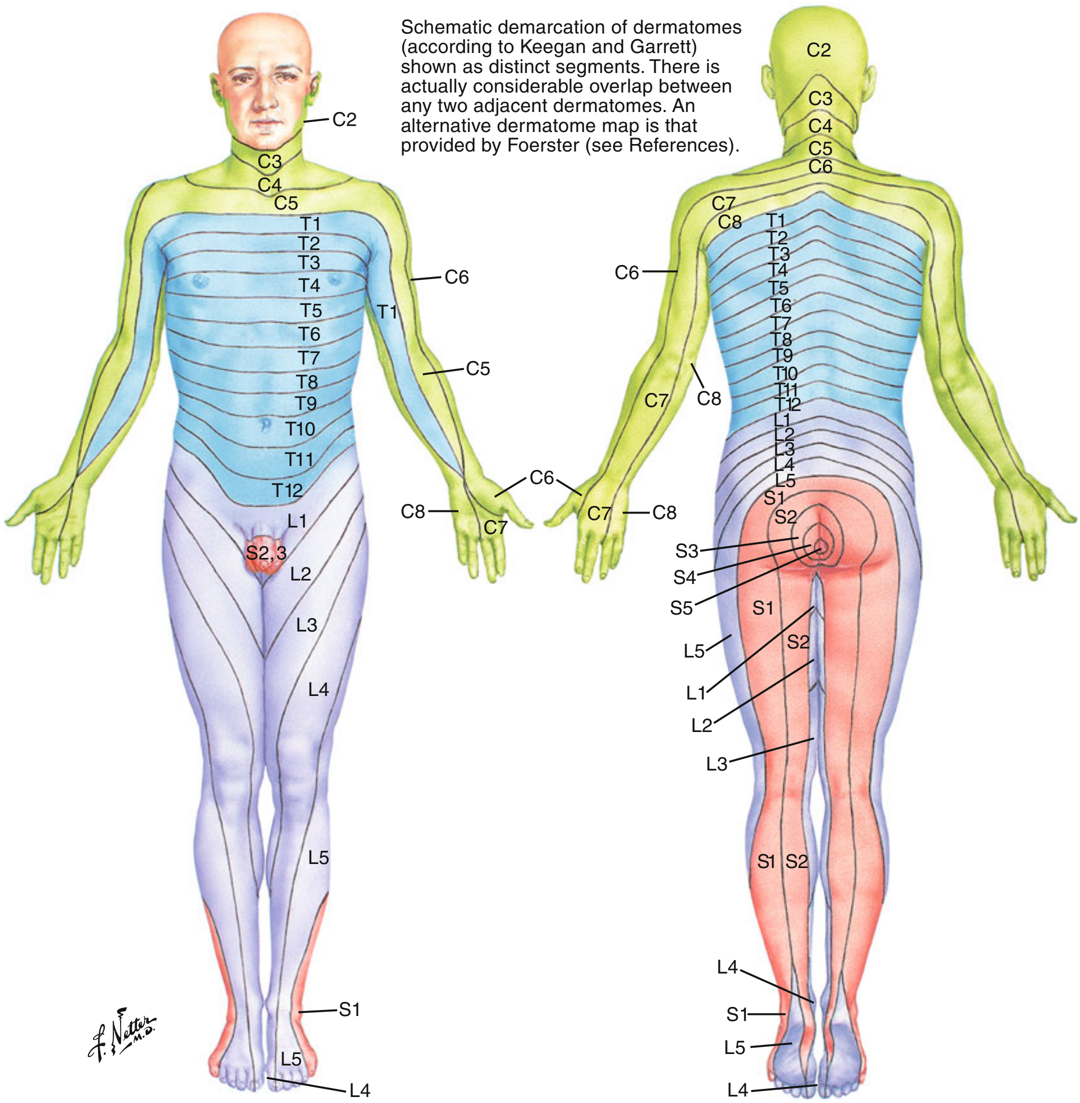
Grade	Clinical signs
0+	No response
1+	Sluggish
2+	Active or normal
3+	Brisk, hyperactive
4+	Abnormally hyperactive, with clonus

Clonus is tested by dorsiflexing the foot and watching for repetitive involuntary plantar flexion and dorsiflexion at the ankle. Clonus, hyperactive reflexes, and a positive Babinski sign (dorsiflexion of the toes, especially the big toe, with stimulation of the lateral aspect of the sole) may suggest upper motor neuron injury. It is important that these clinical signs be further evaluated with more advanced spinal imaging with consideration of magnetic resonance imaging. Diminished or absent reflexes imply lesions present in the peripheral nerve, nerve root, or spinal cord.

3.1.4 Range of Motion and Gait

Flexion, extension, and lateral rotation should be tested in the cervical, thoracic, and lumbar regions. Mechanical versus painful limitations should be documented as they can provide clues to the diagnosis. For example, pain elicited with extension and lateral rotation of the spine along a facet joint implicates the facet joint as the etiology of the pain.

In addition, a straight leg raising test, also known as Lasegue's test, will give information as to whether a radicular type of pain is caused by a dysfunctional disc. This test is performed by



Levels of principal dermatomes

C5	Clavicles
C5, 6, 7	Lateral parts of upper limbs
C8, T1	Medial sides of upper limbs
C6	Thumb
C6, 7, 8	Hand
C8	Ring and little fingers
T4	Level of nipples
T10	Level of umbilicus
L1	Inguinal or groin regions
L1, 2, 3, 4	Anterior and inner surfaces of lower limbs
L4, 5, S1	Foot
L4	Medial side of great toe
S1, 2, L5	Posterior and outer surfaces of lower limbs
S1	Lateral margin of foot and little toe
S2, 3, 4	Perineum

Fig. 3.1 Diagram of the dermatomal distribution of the sensory innervation of the upper and low limbs. Reproduced from "Aids to the examination of the peripheral nervous system"

having the examiner passively raise the patient's leg as the patient is lying supine. A positive test is when the pain is reproduced as the leg is raised between 30° and 70° [5]. Similarly, a Spurling's test can be performed for the cervical spine. A positive test occurs when radicular pain is felt with extension, lateral rotation, and compression of the head [6].

A positive FABER test, also known as Patrick's test, will lead to the sacroiliac joint as the cause of pain [7]. The patient is positioned supine, and the affected lower extremity is flexed at the knee with the ankle placed over the opposite knee, and abducted and externally rotated at the hip. Pain in the region of the sacroiliac joint with compression of the affected knee and opposite anterior superior iliac spine is a positive test and indicates degenerated joint disease, malalignment, or inflammation at the joint.

It is important to have the patient walk and observe their gait. The examiner must note if the patient favors one side, has a wide-based gait, or demonstrates some of the typical patterns of movement disorders (i.e., shuffling gait of Parkinson's disease). The patient should be asked to perform tandem gait and heel-to-toe movements and assess impairment in proprioception or position sense.

If suspicions are raised, check for malingering by observing for overreaction and Waddell's nonorganic signs [8].

Tenderness	Superficial and diffuse and/or nonanatomic tenderness on palpation
Simulation	Pain produced by axial loading (pressing down on the top of the head) or when the patient is asked to passively rotate side to side with the shoulders and pelvis in the same plane
Distraction	Positive tests, such as a positive straight leg raise, are rechecked while the patient is distracted. A nonorganic sign may be present if the finding disappears with distraction
Regional disturbance	Regional weakness or sensory changes not consistent with neuroanatomy. Cogwheel or "giveaway" weakness
Overreaction	Disproportionate verbalizations, facial expression, guarding, tremor, collapse, or sweating

When three or more nonorganic signs are discovered, there is clinical significance as this was found to correlate positively for depression, hysteria, and hypochondriasis. The presence of nonorganic signs should alert the clinician to the need for additional psychological testing [8].

In conclusion, the pain medicine physical examination should be focused on information delineated by the patient in the history. Appropriate skin survey, motor, sensory, neurologic, range of motion, and gait examination must be performed. The results of the physical examination will not only give insight into the pathology of the pain but will also dictate treatment.

3.2 Etiologies of Lumbosacral Pain

3.2.1 Spinal Mechanical

3.2.1.1 Lumbosacral Strain/Sprain

Lumbosacral strain/sprain is the most common cause of low back pain. It is defined as a stretch injury to the large muscles of the low back and/or the ligaments and tendons, leading to microscopic tears and inflammation in these soft tissues [9]. It manifests as pain in the lower back and upper buttocks. Low back muscle spasm can also occur, and patients will often feel a stiffness or describe a "locking up" of the lower back.

Lumbosacral strain/sprain typically occurs because of overuse, improper use of muscles, such as lifting a heavy object improperly, twisting the back in an unusual manner, or trauma. Poor conditioning or deconditioned core muscles of the abdomen and lower back, obesity, smoking, employment, or circumstances that require heavy lifting and improper lifting technique are common risk factors for lumbosacral strain/sprain [4]. Pain is made worse with activities and generally relieved with rest, ice, and nonsteroidal anti-inflammatory agents [10].

3.2.1.2 Degenerative Spine Disease

Degenerative spine disease is not a specific disease but rather a clinical syndrome referring to any dysfunction of the spinal column resulting from the normal aging process and from degeneration that occurs to the bone, joints, muscles, ligaments, nerves, intervertebral discs, and paravertebral tissues of the spine. It encompasses many types of disorders including herniated disc, spinal stenosis, and spondylosis [9].

Degenerative spine disease of the lumbar spine is a major cause of lower back and lower extremity pain and chronic disability and a common reason for referral for medical treatment.

3.2.1.3 Herniated Disc

A herniated disc refers to localized displacement of the nucleus pulposus through a tear in the annulus fibrosus beyond the limits of the intravertebral disc space [11]. The tear in the annulus fibrosus may result in the release of inflammatory mediators which may cause severe pain even without nerve root compression. Only 1–3 % of people presenting with low back pain have lumbar disc herniation and only 1 % will have a nerve root symptom. Most common level for lumbar disc herniations occurs between L4–5 and L5–S1 [12].

Risk factors for herniated lumbar disc include age (between 30 and 50), male gender, lifting a heavy object especially when using a twisting motion of the spine, occupation in a physically demanding job, cigarette smoking, and obesity [13].

Symptoms of lumbar disc herniation often include acute lower back pain and muscle spasm, followed by sudden or gradual radicular leg pain, then typically a reduction in the degree of lower back pain. Bladder symptoms such as urinary urgency, frequency, and hesitancy may be present in up to 18 % of patients with acute disc herniations without cauda equina syndrome [14]. Outright urinary retention or overflow incontinence may however be seen in cauda equina syndrome (see below).

When lumbar disc herniation is associated with heavy lifting, pulling, pushing, or twisting, some patients state that they hear and/or feel a "pop" in their back. They may describe the initial pain as "searing" or "hot" [14]. Patients with radicular leg pain may find relief flexing the knee or the thigh of the affected leg because full extension creates nerve root tension. Patients will generally avoid excessive activity, and yet they cannot remain in one position (sitting, standing, lying) for too long either, prompting them to frequent position changes. Valsalva maneuvers (coughing, sneezing, and straining at the bowel or bladder) may also worsen radicular pain.

3.2.1.4 Cauda Equina Syndrome

Cauda equina syndrome, CES, is a serious neurologic condition involving impairment of the nerves of the lumbar plexus from compression of the cauda equina. CES frequently necessitates emergent or urgent surgical decompression to prevent permanent deficits and/or incontinence.

CES is prevalent in only 0.04 % of all patients with low back pain and only 1–2 % of all patients with lumbar disc herniations [14]. CES is caused by any compressive lesion causing pressure on the nerve roots in the lumbar spinal canal below the conus medullaris; the most common cause of this problem however is a central disc herniation. Other causes include metastatic disease; intrinsic spinal tumors; burst fractures; direct trauma from anesthetic, diagnostic, or therapeutic lumbar puncture; spinal epidural hematoma; penetrating trauma such as knife or bullet injuries; compressive abscess; and spinal stenosis [12].

Spontaneous low back and radiating lower extremity pain with severe or progressive weakness usually involving more than one nerve root may be the most prominent symptom in CES. Pain in a radicular distribution is more prominent than back pain in these cases. Saddle anesthesia is the most common sensory deficit in CES with a distribution involving some or most of the anus, genitals, perineum, buttocks, and posterior-superior thighs. This can be unilateral and asymmetric as well. A patient with CES or developing CES will often describe symptoms of sphincter disturbance; urinary retention is the most common, but other symptoms include bladder frequency, urgency, overflow incontinence, fecal incontinence, and diminished anal sphincter tone. Retention can be evaluated and documented by performing post-void residual measurements either by catheterization or ultrasound measuring residual. Diminished or absent patellar and Achilles reflex may be found [2, 12]. No single sign or symptom defines a CES, but it is a constellation of findings instead.

3.2.1.5 Spinal Stenosis

Spinal stenosis refers to a condition of narrowing in and around the spinal canal, causing compression on the neural elements. It is classified as central canal stenosis, foraminal stenosis, or lateral recess stenosis. Central canal stenosis refers to narrowing of the anteroposterior dimension of the spinal canal, causing compression of the spinal cord or cauda equina. Foraminal stenosis refers to narrowing of the neural foramen, causing compression on spinal nerves, and lateral recess stenosis, a type of lumbar spinal stenosis, arises from hypertrophy of the superior articular aspect of the facet joint. In the lumbar spine, lateral recess stenosis most commonly affects the L4–5 facet [15].

The most common cause of stenosis is related to degeneration and the aging process, osteoarthritis, disc degeneration, and thickened spinal ligaments. Other causes include spinal trauma, previous spinal surgery, spinal tumors, Paget's disease, and having a congenitally small central canal as seen in achondroplasia [12].

Symptoms of spinal stenosis depend on the location of the narrowing and resultant impingement or compression. Any one or combination of the following symptoms of stenosis can be present: low back pain; numbness, paresthesias, cramping, weakness, and pain in the buttocks, legs, and feet; radiating leg pain; and bowel and/or bladder dysfunction.

Symptomatic lumbar stenosis is most common at L4–5 then L3–4 followed by L2–3 and then L5–S1. Lumbar stenosis is classified as stable, facet hypertrophy, thickening of the ligamentum flavum, and disc degeneration. Unstable stenosis is marked by the addition of degenerative spondylolisthesis or degenerative scoliosis [16].

Neurogenic claudication is a common symptom of lumbar spinal stenosis. It can be unilateral or bilateral buttock, hip, leg, or foot discomfort, pain, or weakness that is aggravated by standing and walking and alleviated by sitting or lying. Patients with neurogenic claudication may develop "anthropoid posture," an exaggerated flexion at the waist which possibly creates a reduced lumbar lordosis and opens the facet joints [12]. The patient may reveal that they are also more comfortable leaning forward, for example, on a counter at home or, classically, a grocery cart in the supermarket.

3.2.1.6 Fracture

Most fractures of the spine occur in the thoracic and lumbar spines and most commonly at the thoracolumbar junction, T12–L1. These fractures are typically caused by major trauma such as motor vehicle accidents, falls, or sports accidents, but even minor trauma in a compromised spine can lead to fracture. Persons with bone weakened by osteoporosis, long-term corticosteroid use, substance abuse, or systemic disease and spinal tumors can suffer a nontraumatic fracture during normal daily activities [2, 4].

Types of spinal fractures include compression, burst, flexion/distraction, seatbelt, or Chance fracture, transverse process, fracture dislocation, pathologic fracture from infection or tumor, and osteoporotic fracture [12].

Plain radiography is recommended in patients with persistent pain, history of trauma, fever, unexplained weight loss, cancer, substance abuse, and age greater than 50. Computed tomography (CT) and magnetic resonance imaging (MRI) are more useful for the detection of infection and fracture caused by cancer [4].

Nonsurgical treatment includes 6–8 weeks of bracing, activity modification, analgesics, physical therapy and a gradual return to normal physical activity, and treatment of the underlying systemic disease if present. Surgery is typically reserved for unstable and comminuted fractures, the presence of neurologic deficit, progressive spinal deformity, and pain refractory to nonsurgical management.

3.2.1.7 Spondylolisthesis

Spondylolisthesis is slippage of the superior vertebra over inferior vertebra. This condition most commonly affects the lumbar spine and is less common in the cervical spine. There are five types of lumbar spondylolisthesis:

1. Dysplastic spondylolisthesis – this is caused by a congenital defect in the facet that allows the vertebra to slip forward.
2. Isthmic spondylolisthesis – this results from a defect in the part of vertebra called pars interarticularis. This defect is thought to be caused by repetitive trauma and is more common in athletes due to hyperextension motion.
3. Degenerative spondylolisthesis (DS) – this type occurs due to arthritic changes in the facet joints of vertebra. It is more common in older patients and represents the most common form of spondylolisthesis.

4. Traumatic spondylolisthesis – this occurs secondary to direct trauma and can include a fracture of the pedicle, lamina, or facet joints
5. Pathological spondylolisthesis – this type is caused by bony defect due to tumor which causes bone to be abnormal.

Spondylolisthesis is graded based on extent of slippage of lateral radiograph by Meyerding. This measurement is distance from the posterior edge of upper vertebra to posterior edge of lower vertebra and is reported as a percentage of total upper vertebral body. There are five grades of slippage: grade1, 0–25 %; grade 2, 25–50 %; grade 3, 50–75 %; grade 4, 75–100 %; and grade 5, spondyloptosis when upper vertebra is completely fallen off in relation to lower vertebra.

Among these five types of spondylolisthesis, DS is most commonly seen in patients over 50 years and a common cause of low back pain (LBP). It also commonly involves L4–L5 level and to lesser extent L5–S1. DS is approximately four to five times more common in females than in males, due to greater ligamentous laxity and hormonal effects [17, 18].

LBP is the most common presentation in patients with DS; however, some of them may be asymptomatic. LBP may be mechanical type to pain and relieved with rest. This condition is also associated with neurogenic claudication. Leg pain can be radicular or diffuse and involving dermatomal distribution of L4, L5, and S1 nerve roots, although single nerve root, most commonly L5, involvement may also be seen. These symptoms are seen in 42–82 % of patients who see a spine surgeon for help. Bladder and bowel dysfunctions due to DS can occur but less profound than cauda equina syndrome from disc herniation. This can be seen in severe stenosis in 3 % of patients [19].

Patients with mechanical type of LBP and neurogenic claudication should be investigated with standing lumbar spine x-rays including flexion and extension films. Supine films may not demonstrate the slippage. CT scan and MRI of lumbar spine add to diagnosing this condition accurately with degree of slippage and extent of stenosis causing neural compression.

3.2.1.8 Kyphosis

Kyphosis is a term to describe the natural forward curve of the thoracic and lumbosacral spines where the lumbar and cervical spines have a natural lordosis or lordotic curve. When kyphosis is used to describe a spinal deformity, it refers to an exaggeration of the forward curve in a portion of any part of the spine, also called a kyphotic deformity. Kyphotic deformities can create symptoms that vary from pain and neurologic deficit to compensatory and cosmetic deformities [13].

Kyphotic deformity has a multitude of causes including degenerative (osteoporotic compression fracture, Paget's disease), traumatic, developmental (scoliosis), iatrogenic (following spinal decompressive laminectomy or radiation to the spine), neoplastic (primary spinal tumor or metastatic disease), congenital (achondroplasia), infectious (Pott's disease, osteomyelitis), inflammatory (ankylosing spondylitis, rheumatoid arthritis), and neuromuscular (cerebral palsy) or from Scheuermann's disease [13].

A careful history and exam will reveal presence of deformity, underlying or contributing conditions, neurologic impairment (from spinal cord or spinal nerve compression), and the development of compensatory deformities. Standing or upright x-rays of the entire spine in one view, commonly referred to as "scoliosis x-rays" with anterior-posterior and lateral views, are used to evaluate the structure of the spine and measure the degree of kyphosis or other abnormal curves. When neurologic deficits are discovered, worsening pain or spinal instability is suspected; further imaging with CT and/or MRI is warranted.

3.2.1.9 Scoliosis

The term scoliosis originates from Greek word skoliosis meaning obliquity or bending. Adult scoliosis is a de novo development of curved spinal architecture after completion of skeletal maturity. It is also seen in children and adolescents; however, adult scoliosis differs from child or adolescent scoliosis in terms of curve types and patterns, rate of deformity progression, rigidity of deformity, patient comorbidities, and clinical symptoms and presentation [20]. Some of adolescent scoliosis can be asymptomatic and get detected during adult life due to progression of curvature. Other patients may develop scoliosis after spine surgery for disc degeneration or spinal fusion surgery as adjacent-level degeneration with scoliosis.

The prevalence of adult scoliosis is probably on the rise due to increasing life expectancies. The most common types of scoliosis encountered in adults are idiopathic and degenerative scoliosis. The former condition starts in childhood or adolescence and progresses over a period of time with added degeneration of disc and facets. Degenerative scoliosis is a de novo development of scoliosis secondary to asymmetric involvement of disc degeneration, facet arthrosis, and disc collapse [21].

Patients with scoliosis may present to spine surgeons with symptoms of back pain due to spine deformity or symptoms of neural compression unrelated to deformity. Adult patients with scoliosis present with axial low back pain, neurogenic pain, as well as changes in gait and posture. Physical examination of the back while palpating spine will reveal abnormal curvature of spine and asymmetry of the pelvic crests. Patients with stooped posture may have sagittal imbalance forcing them to walk with walker or cane. Imaging studies begin with plane x-rays of the spine standing, and scoliosis films which determine the severity of sagittal and coronal imbalance and pelvic tilt.

3.2.2 Spinal Nonmechanical

3.2.2.1 ⚐ Neoplasia: Intradural or Vertebral Tumors/ Pathologic Fracture

Diagnosis of spinal neoplasia begins with the history. Patients who complain of subacute back pain that is worse at night or with rest should raise the clinician's suspicion for a possible neoplastic process especially if the patient reports unintentional weight loss and general malaise.

3.2.2.2 ⚐ Infections: Osteomyelitis, Discitis, and Epidural Abscesses

Spinal infections arise from bacteria carried through the bloodstream to the spine from a site of infection elsewhere in the body, urinary and respiratory tract infection, soft tissue (infections on the skin), dental flora, or through intravenous drug use, surgery, injection treatments, or as a result of direct trauma. Infections are most frequently seen in the lumbar spine, followed by thoracic, cervical, and sacrum.

Back pain may be a result of an infection in the bone (osteomyelitis), in the disc (discitis), or on the spinal cord (epidural abscess). Vertebral body collapse with kyphotic deformity is common. Necrotic bone and disc fragments as well as abscess formation can cause spinal cord or cauda equina compression [12]. Presenting symptoms can be as generalized as back pain, low-grade temperatures, malaise, and anorexia. Unexplained back pain following a recent infection or iatrogenic procedure with a strong mechanical component should be considered for a spinal infection.

3.2.3 Inflammatory Arthropathies

3.2.3.1 Ankylosing Spondylitis

Ankylosing spondylitis (AS) is a chronic, systemic inflammatory disease of the joints and the axial skeleton characterized by back and neck pain and progressive stiffening, or ankylosing, of the spine. Pain and stiffness is typical in the thoracic spine or sometimes the entire spine, with referred pain to the buttocks and hamstrings. Pain is often present in the morning, is severe at rest, and improves with physical activity. Sacroiliitis is present in greater than 95 % of persons with AS.

The onset is gradual, typically beginning in late adolescence and early adulthood, and is slightly more common in men than in women in whom the disease evolves more slowly. Approximately 90 % of AS patients express the HLA-B27 genotype, meaning there is a strong genetic association. However, only 5 % of individuals with the HLA-B27 genotype contract the disease [13, 22].

3.2.3.2 Rheumatoid Arthritis

Rheumatoid arthritis (RA) is a systemic inflammatory autoimmune disease which is chronic and progressive in nature. RA affects multiple tissues and organs but predominantly attacks synovial joints, leading to destruction of articular cartilage and ankylosis. It occurs in 0.3–1.5 % of the population, with women affected 2–3 times more often than men. Onset occurs most frequently between the ages of 40 and 50, but people of any age can be affected [22]. Often a disabling and painful condition, it has an insidious onset marked by fatigue, anorexia, weight loss, and generalized aching and stiffness (especially morning stiffness). RA can lead to substantial loss of function and mobility if not adequately treated.

3.2.3.3 Reiter's Syndrome

A reactive arthritis usually occurring 1–3 weeks following certain bacterial infections (commonly Chlamydia, Shigella, Salmonella, Yersinia, and Campylobacter) with involvement of at least one other non-joint area, specifically urethritis, uveitis/conjunctivitis, skin lesions, and mucosal ulcerations. Between 75 and 90 % of patients are also HLA-B27 positive [13].

3.2.3.4 Paget's Disease

Paget's disease (PD) is a metabolic disorder with abnormal bone remodeling, causing spinal stenosis and facet arthropathy. In this disorder, there is excessive breakdown of bone and formation of weak bone, causing pain, fracture, and joint arthritis. Etiology of Paget's disease remains unclear, and it has been thought to be caused by viral infection [23]. Paget's disease also can be inherited as autosomal dominant trait with high penetrance [24]. Reported incidence of back pain in PD patients ranges from 11 to 42 % [25]. Several mechanisms have been described in neural symptoms in patients with PD: (1) compression of neural elements by pagetic process, (2) neural ischemia, and (3) pagetic sacromatous degeneration [26]. Diagnosis of PD is by x-rays, bone scan, and CT scan and MRI. Once PD is confirmed as underlying pathology, treatment is initiated with biphosphonates with goal of relieving bone pain and arrest progression of disease. In addition to biphosphonates, other drugs used are calcitonin and mithramycin.

3.2.3.5 Sacroiliitis

Inflammation of the sacroiliac joint, sacroiliitis is a frequent initial manifestation of one of the seronegative spondyloarthropathies. Sacroiliitis most commonly presents in young people who are HLA-B27 positive and/or have ankylosing spondylitis, psoriatic arthritis, or Reiter's disease. The pain of sacroiliitis most commonly occurs in the lower back and tops of the buttocks, but it can also radiate to incorporate the groin, legs, and feet.

3.2.3.6 Scheuermann's Kyphosis

Scheuermann's kyphosis most commonly affects the vertebral bodies of the thoracic spine but can also occur at the thoracolumbar junction. It is best described as a growth abnormality of one or more vertebral bodies where the anterior portion of the vertebrae stops growing, yet the posterior portion continues to grow, creating an abnormal amount of kyphosis at the apex of the deformity. It is most often seen in males and typically occurs in the final growth spurt of adolescence. Pain and discomfort are common along the site of the kyphotic deformity [13].

3.2.4 Arachnoiditis

Arachnoiditis is a neuropathic disease caused by inflammation of the arachnoid membrane of the spinal cord and spinal nerves. It can be caused by chemical irritation, infection, injury, previous spinal surgery, or other invasive spinal procedures. Arachnoiditis can lead to adhesions, causing the spinal nerves to "clump." The presenting symptoms of arachnoiditis may include constant chronic low back pain unrelated to and unrelieved by specific positions, radiating leg pain, perineal pain, and pseudoclaudication. Treatment is insufficient with the main focus on medication and intrathecal steroid injections [9].

3.2.5 Failed Back Syndrome

Failed back surgery syndrome (FBSS) and post-laminectomy syndrome are terms used to describe a type of a chronic and persistent back and/or leg pain condition that occurs following lumbar spinal surgery. FBSS does not include persistent lower extremity weakness, sensory changes, or reflex abnormalities [13].

Patients with FBS often demonstrate frustration and anger and often have an accompanying diagnosis of depression. These patients frequently require long-term pain management, specifically with narcotic agents. The clinician should be astute to the possibility of malingering for a variety of secondary gain issues which can be found to coexist in patients with FBS.

The causes of FBS include incorrect initial diagnosis; improper preoperative patient selection; continued nerve root compression caused by recurrent disc herniation, scar tissue, pseudomeningocele, hematoma, spinal instability, or stenosis at the surgical or junctional level; permanent nerve injury; technical error during surgery; adhesive arachnoiditis; infection (discitis, osteomyelitis); spondylosis; other non-spinal causes of back pain; and nonanatomic factors/malingering [12].

3.3 Visceral Causes of Back Pain

Occasionally, back pain is the single presenting symptom of a variety of serious medical conditions or possible emergency. Clinicians must be astute to possible medical or visceral causes of back pain when the patient describes severe, refractory, or atypical pain. Examination of the abdomen with palpation of the visceral organs is a necessary step of diagnosis. This chapter will not cover the atypical presentations but will just report a laundry list of differential diagnoses which also may present with back pain: dissecting aortic aneurysm, urinary tract infection, pyelonephritis, prostatitis, pelvic inflammatory disease (PID), endometriosis, ovulation, pregnancy, ectopic pregnancy, acute pancreatitis, duodenal ulcer, cholecystitis, nephrolithiasis, and visceral cancers.

3.4 Non-spinal

3.4.1 Piriformis Syndrome

Piriformis syndrome refers to sciatic symptoms (low back pain radiating in the buttock, thigh, calf, and foot) that do not originate from the lumbosacral plexus and/or from disc herniation but rather by pressure from the piriformis muscle on the sciatic nerve [9]. Pain is made worse with activity such as prolonged sitting and walking.

The piriformis muscle originates at the anterolateral aspect of the sacroiliac region, transverses the sciatic nerve, and inserts on the greater trochanter of the femur. It is innervated by the ventral rami of S1 and S2 and abducts and laterally rotates the femur. The sciatic nerve passes between the two bellies of the piriformis [27]. Trauma or overuse can lead to irritation, inflammation, and spasm of the piriformis, which can compress the transversing sciatic nerve and mimic a herniated lumbar disc or spinal nerve compression.

Performing a straight leg raise (SLR) test can differentiate between piriformis syndrome and sciatica caused by lumbosacral nerve compression. In piriformis syndrome the SLR is only mildly positive or negative unless the examiner flexes, adducts, and internally rotates the proximal leg. Adding these maneuvers places tension on the irritated piriformis and stretches the inflamed nerve root [9].

3.4.2 Bursitis

Trochanteric bursitis and ischiogluteal bursitis are types of pelvic pain that is often misdiagnosed as herniated lumbar disc or sciatica from the lumbosacral spine especially in the elderly patient population. The symptoms of trochanteric bursitis are pain in the hip region with activity and point tenderness over the greater trochanter. Patients will often report that they are unable to lie on the affected side. With ischiogluteal bursitis, pain is localized deep in the center of the buttock. Patients will describe the pain as “unrelenting,” aggravated by sitting or walking, and accompanied by radicular leg pain that is unrelieved by rest. Treatments include rest, anti-inflammatory agents, physical therapy, cortisone injections, or in extreme cases bursectomy [28].

3.4.3 Fibromyalgia

Fibromyalgia is a chronic pain syndrome characterized by widespread musculoskeletal pain, fatigue and heightened tenderness to tactile pressure, general fatigue, and sleep disturbance. The most common sites of pain include "tender points" of the neck, back, shoulders, bony pelvis, and hands. Over 6 million Americans are diagnosed yearly, 90 % of which are women between the ages of 20 and 55 years old. Pain is described as a deep ache, sometimes shooting, and burning.

3.4.4 Spasticity

Spasticity is often found in people with cerebral palsy, traumatic brain injury, stroke, multiple sclerosis, and spinal cord injury. Spasticity results from upper motor neuron lesions creating an imbalance of inhibitory influence on alpha and gamma motor neurons. Spasticity is clinically manifested as a hypertonic state of muscles with clonus and involuntary movement. Often painful and debilitating, spasticity creates challenges with self-care, hygiene, posture, and balance. Back pain is a cardinal feature of spasticity [12].

3.4.5 Degenerative Joint Disease (DJD) of the Hip

DJD of the hip is most often caused by osteoarthritis. It is characterized by pain and stiffness from the breakdown of joint surface cartilage. DJD of the hip creates ipsilateral groin and medial thigh pain. At times the pain can radiate to the knee on the same side, creating confusion as to whether the problem is from the hip, from the knee, or from a lumbar radiculopathy. Hip dysplasia and avascular necrosis of the hip can cause these symptoms as well.

When pain originates from the hip, walking and prolonged activity worsen the pain and motion in the joint is limited, often realized when trying to go from sitting or lying to a standing position. Initially, pain is relieved by rest, but once the DJD progresses, minimal activity such as slight movements in bed can worsen the pain. Treatment includes rest, ice, anti-inflammatory agents, cortisone injections, and, if necessary, joint replacement surgery [29].

3.4.6 Leg Length Discrepancy/Pelvic Level/Gait Abnormality

A leg length discrepancy can be due to a mild variation between two sides of the body and is a normal variation when the difference is 3/5 of an inch or less. There are a variety of causes for leg length discrepancy including previous injury or fracture to the leg (especially in children who are fractured at the growth plate), bony diseases, inflammation and osteoarthritis, and neurologic conditions.

The correlation between patients with a clinically significant leg length discrepancy and the incidence of low back pain is controversial. Leg length discrepancy can lead to a pelvic obliquity which changes the coronal balance of the sacrum, leading to a segmental scoliosis and potential for increased low back pain [9].

Leg length discrepancy can lead to gait abnormalities requiring patients to exert more effort with ambulation and hasten degenerative joint disease. Treatment includes orthotics and surgery for inhibiting growth, lengthening, and shortening.

3.4.7 Posture

The position of the body in both the sitting and standing positions can have considerable effect on the development or prevention of lower back pain. Swayback posture (lumbar hyperlordosis) in standing and a slouched or slumped (thoracic hyperkyphosis) posture in sitting can impact the health and function of the muscles of the abdomen and lower back. When daily activities require prolonged sitting or standing and a balanced seated or standing posture is not achieved, the stabilizing muscles of the spine fatigue and lead to pain and stiffness [9].

3.4.8 Obesity

More than half of the adult population of Americans is categorized as being overweight or obese. Overweight and obesity is a contributing factor to back pain and a significant cause for seeking medical care. Being overweight or obese can contribute to the symptoms of osteoporosis, osteoarthritis, degenerative spine disease, and spondylolisthesis. The addition of excess weight and a deconditioned core translate extra strain and stress to the spine, specifically the lower back [30].

3.4.9 Mood Disorders

Pain, anxiety, and depression are commonly experienced together. In patients with chronic and disabling pain syndromes such as fibromyalgia, low back pain, and nerve pain, this is particularly evident. Patients with diagnosed anxiety and depression report higher incidences and greater severity of pain in addition to increased disability and dysfunction due to pain compared to patients without depression and anxiety [31]. Treatment of both pain and mood disorder is challenging but can be accomplished with a comprehensive plan including cognitive behavioral therapy, relaxation techniques, hypnosis, exercise, antidepressants, and mood stabilizers.

3.4.10 Secondary Gain/Malingering

Secondary gain is an external psychological motivator that may drive a patient to report certain symptoms. If a patient's pain or illness allows them to miss work or gain extra sympathy and attention, these would be examples of secondary gain. With secondary gain, a patient is unconsciously seeking these "rewards." If he or she is deliberately exaggerating symptoms for personal gain, that is, to win a legal dispute, then he or she is malingering [32]. In either case, it is in the patient's best interest to remain debilitated or in pain in order to continue to receive the rewards.

References

1. National Institute of Neurological Disorders and Stroke; National Institute of Health. Low back pain sheet. Updated 15 June 2011. http://ninds.nih.gov/disorders/backpain/detail_backpain.htm
2. Bigos S, Bowyer O, Braen G et al (1994) Acute low back problems in adults, vol Clinical practice guideline no.14. AHCPR publication no. 95-0642. Agency for Health Care Policy and Research PHS, U.S Department of Health and Human Services, Rockville
3. Hart LG, Deyo RA, Cherkin DC (1995) Physician office visits for low back pain. Frequency, clinical evaluation, and treatment patterns from a U.S. National survey. Spine (Phila Pa 1976) 20(1):11–19
4. Deyo RA, Weinstein JN (2001) Low back pain. N Engl J Med 344(5):363–370
5. Speed C (2004) Low back pain. BMJ 328(7448): 1119–1121
6. Tong HC, Haig AJ, Yamakawa K (2002) The spurling test and cervical radiculopathy. Spine (Phila Pa 1976) 27(2):156–159
7. Ross MD, Nordeen MH, Barido M (2003) Test-retest reliability of Patrick's hip range of motion test in healthy college-aged men. J Strength Cond Res 17(1):156–161
8. Scalzitti DA (1997) Screening for psychological factors in patients with low back problems: Waddell's nonorganic signs. Phys Ther 77(3):306–312
9. Cailliet R (1988) Low back pain syndrome, 4th edn. FA Davis, Philadelphia
10. Malanga G, Dunn K (2010) Low back pain management: approaches to treatment. J Musculoskel Med 27:305–315
11. Fardon DF (2001) Nomenclature and classification of lumbar disc pathology. Spine (Phila Pa 1976) 26(5): 461–462
12. Greenberg MS, Arredondo N (2006) Handbook of neurosurgery, 6th edn. Thieme Medical Publishers, New York
13. Vaccaro AR (2005) Spine: core knowledge in orthopaedics. Mosby, St. Louis
14. Deyo RA, Rainville J, Kent DL (1992) What can the history and physical examination tell us about low back pain? JAMA 268(6):760–765
15. Epstein NE, Maldonado VC, Cusick JF (1998) Symptomatic lumbar spinal stenosis. Surg Neurol 50(1): 3–10
16. Duggal N, Sonntag V, Dickman CA (2001) Fusion option and indications in the lumbosacral spine. Contemp Neurosurg 23(1):1–8
17. Rosenberg NJ (1976) Degenerative spondylolisthesis: surgical treatment. Clin Orthop Relat Res 117:112–120
18. Bird H, Eastmond C, Hudson A, Wright V (1980) Is generalized joint laxity a factor in spondylolisthesis? Scand J Rheumatol 9(4):203–205
19. Kostuik JP, Harrington I, Alexander D, Rand W, Evans D (1986) Cauda equina syndrome and lumbar disc herniation. J Bone Joint Surg Am 68(3):386–391
20. Herkowitz HN, Rothman RH, Simeone FA (2011) Rothman-simeone, the spine, 6th edn. Saunders Elsevier, Philadelphia
21. Simmons ED Jr, Simmons EH (1992) Spinal stenosis with scoliosis. Spine (Phila Pa 1976) 17(6 Suppl): S117–S120
22. Klippel J (2001) Primer of rheumatic disease, 12th edn. Arthritis Foundation, Atlanta

23. Mee AP (1999) Paramyxoviruses and Paget's disease: the affirmative view. Bone 24(5 Suppl):19S–21S
24. Hocking L, Slee F, Haslam SI et al (2000) Familial Paget's disease of bone: patterns of inheritance and frequency of linkage to chromosome 18q. Bone 26(6):577–580
25. Rosenkrantz JA, Wolf J, Kaicher JJ (1952) Paget's disease (osteitis deformans); review of one hundred eleven cases. AMA Arch Intern Med 90(5):610–633
26. Herkowitz HN (2004) The lumbar spine, 3rd edn. Lippincott Williams & Wilkins, Philadelphia
27. Saladin K (1998) Anatomy & physiology: the unity of form and function, 1st edn. McGraw-Hill, WCB
28. Dougherty CD, Dougherty JJ (2008) Evaluating hip pathology in trochanteric pain syndrome. J Musculoskel Med 9:428–436
29. Hoaglund FT, Steinbach LS (2001) Primary osteoarthritis of the hip: etiology and epidemiology. J Am Acad Orthop Surg 9(5):320–327
30. Shiri R, Karppinen J, Leino-Arjas P, Solovieva S, Viikari-Juntura E (2010) The association between obesity and low back pain: a meta-analysis. Am J Epidemiol 171(2):135–154
31. Means-Christensen A, Roy-Byrne P, Sherourne C, Craske M, Stein M (2008) Relationships among pain, anxiety, and depression in primary care. Depress Anxiety 25:593–600
32. McDermott BE, Feldman MD (2007) Malingering in the medical setting. Psychiatr Clin North Am 30(4): 645–662

Radiologic Imaging of the Spine

4

Angela Valladares-Otero, Brian Christenson, and Brian D. Petersen

4.1 Imaging Option Overview

4.1.1 Radiography

4.1.1.1 Physics

Radiography is the first-line technique in the evaluation of the spine. It is relatively inexpensive and widely available. X-rays are produced by rapidly moving streams of electrons from the cathode to the anode. The produced ionizing radiation (photons) penetrates an object and the differences in X-ray attenuation, which depends on differences in tissue density, are registered on a sensor plate. Bones absorb the radiation more than soft tissues; denser tissues attenuate the X-ray beam to a greater degree, lessening the number of X-ray photons sensed on the detector plate. The greater the number of X-ray beams to reach the detector, the darker the image. This is why bones are bright and lungs are dark on a conventional radiograph.

4.1.1.2 Radiography as a Screening Tool in Spine Trauma

The standard method of screening the cervical spine is a conventional radiographic series, which typically includes lateral, anteroposterior, and odontoid views. Other views include swimmers lateral (to clear the shoulders and allow visualization of the cervical thoracic junction), oblique views, and flexion/extension views in the lateral projection. Particular difficulty in positioning high-mechanism, poly-trauma patients may lead to a large number of inadequate radiographic examinations.

Cervical spine radiography, although relatively cheap, adds substantially to health-care costs because of the high volume of its use. Emergency departments annually treat millions of patients with trauma who are at risk for cervical spine injury, and the total cost of cervical spine radiography is therefore substantial and judicious use of cervical spine radiography in the emergency trauma is necessary.

Several decision algorithms have been developed. The Canadian C-spine Rule (CCR) [1] and the National Emergency X-Radiography Utilization Study (NEXUS) low-risk criteria (NLC) [2] were developed independently. Both rules are sensitive for detecting acute C-spine injury which allows the emergency department physicians to be more selective in the use of radiography in alert and stable trauma patients [3].

A. Valladares-Otero, M.D. • B. Christenson, M.D.
Department of Radiology,
University of Colorado School of Medicine,
12401 East 17th Avenue, Room 1618, Aurora,
CO 80045, USA

B.D. Petersen, M.D. (✉)
Associate Professor of Radiology and Orthopaedics,
Chief of Musculoskeletal Radiology,
University of Colorado,
Mail Stop L954, 12401 East 17th Avenue, Room 5-006,
Aurora, CO 80045, USA
e-mail: brian.petersen@ucdenver.edu

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_4, © Springer-Verlag Berlin Heidelberg 2014

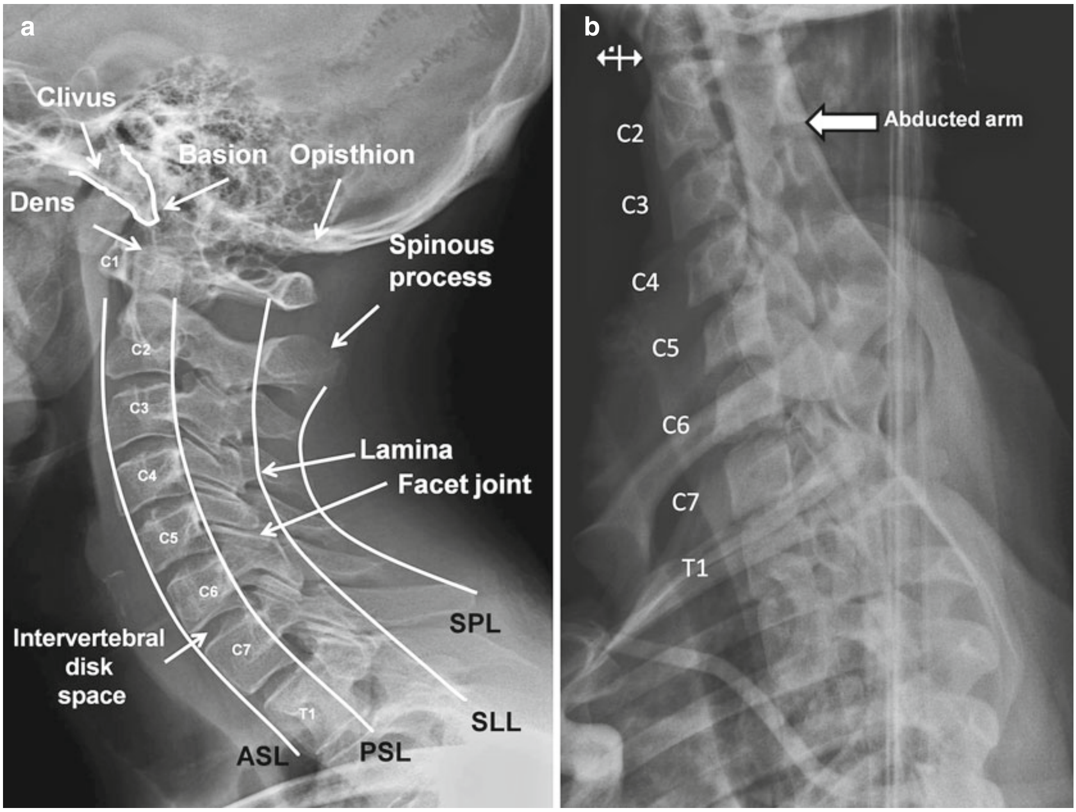


Fig. 4.1 Normal lateral (**a**) and swimmer's lateral (**b**) projections of the cervical spine with labeled pertinent anatomy. Anterior spinal line (*ASL*), posterior spinal line (*PSL*), spinolaminar line (*SLL*), and spinous process line (*SPL*)

4.1.1.3 Normal Radiographic Anatomy of the Spine

When evaluating spinal radiographs, a firm understanding of normal anatomy is necessary to allow one to detect pathology.

Cervical Spine

Radiographic assessment of the cervical spine requires adequately exposed images to allow visualization of the bone trabeculae, as well as adequate patient positioning. On a true lateral radiograph of the cervical spine, the facet joints are superimposed on each other. A frontal view of the cervical spine should have similar coverage as a lateral view. On a true frontal view, the spinous processes are midline. Open-mouth odontoid view should be centered on the C1–C2 articulation and the teeth and occiput should not be superimposed over the area of interest.

Lateral View (Fig. 4.1)

The lateral view (Fig. 4.1a) is the most important view in the routine trauma series. The lateral view must include all seven cervical vertebrae, as well as the C7–T1 intervertebral space. Several methods have been used to include the vertebral bodies and posterior elements of the cervical thoracic junction. On a swimmers view (Fig. 4.1b), one of the arms is raised above the head to avoid superimposition of the shoulders over the cervical thoracic junction. Disadvantages to the swimmers view include higher radiation dose, high scatter, and difficult positioning. Although bony details are usually suboptimal on the swimmer's lateral view, gross alignment can be confirmed.

Lateral view of the cervical spine allows evaluation of the vertebral bodies and intervertebral disks height, as well as the cervical spine alignment which physiologically has a gentle lordotic

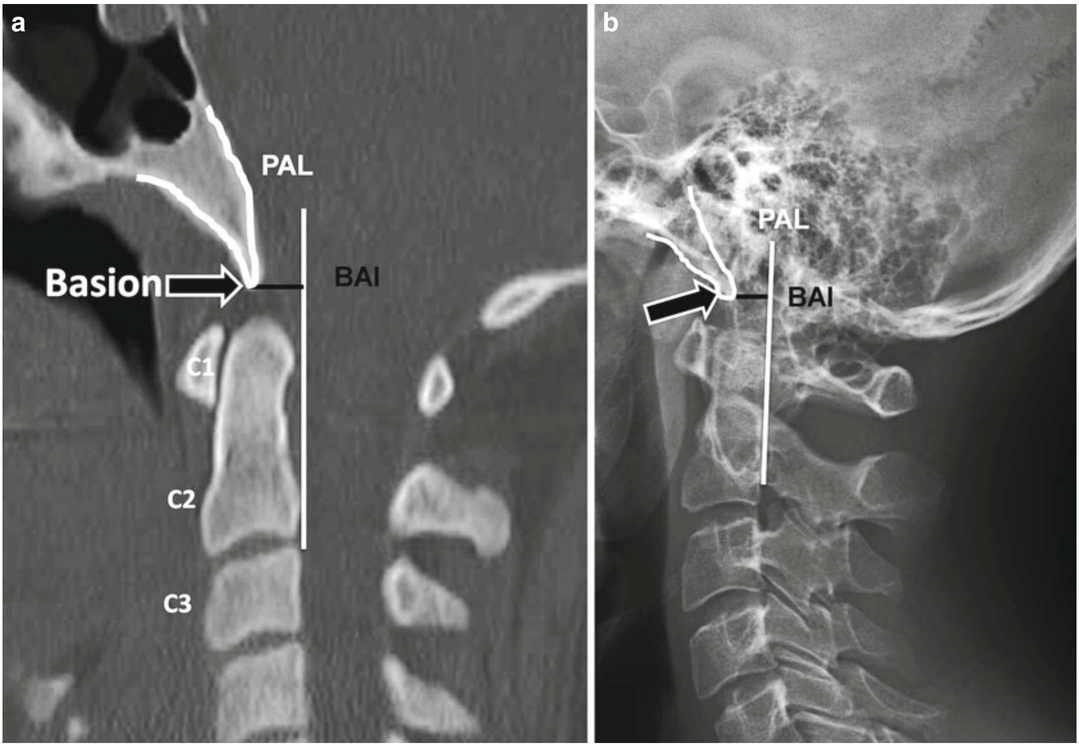


Fig. 4.2 Sagittal CT reconstruction (**a**) and lateral cervical spine radiograph (**b**) demonstrating the basion-axial interval (*BAI*). Basion-axial interval is the distance between basion (*black arrows*) and a line extending superiorly, tangential to the posterior cortex of the C2 vertebral body, the posterior axial line (*PAL*)

curve. Vertebral bodies should be rectangular in shape and similar in size to the adjacent ones.

The articular facets connect the posterolateral aspect of vertebral bodies and combine to form the facet joints. On the lateral view, the lateral masses appear as rhomboid-shaped structures projecting posteroinferiorly.

The anterior longitudinal ligament (ALL) and the posterior longitudinal ligament (PLL) are major stabilizers of the intervertebral joints, helping to maintain the vertebral body alignment [4]. When assessing spinal alignment, it is helpful to evaluate the integrity of the anterior spinal line (Fig. 4.1a, ASL) which extends along the anterior margin of the vertebral bodies; the posterior spinal line (Fig. 4.1a, PSL) along the posterior margin of the vertebral bodies; the spinolaminar line (Fig. 4.1a, SLL) along the posterior margin of spinal canal; and a line connecting the tips of C2–C7 spinous processes, the spinous process line (Fig. 4.1a, SPL). Misalignment of ASL, PSL, SLL, or SPL can suggest ligamentous injury or occult fracture. Although any spinal offset should be scrutinized, there are common nonpathologic reasons for minimal offset including pseudosubluxation of C2 in the pediatric population [5] and the slight offset of the SLL between the posterior elements of C1 and C2.

Radiographic assessment of the craniocervical and atlantoaxial articulations is difficult but crucial (Figs. 4.2, 4.3, and 4.4). Craniocervical and atlantoaxial biomechanical continuity depends on the integrity of the skull base (occipital condyles), atlas (C1), and axis (C2) and their stabilizing ligaments. On lateral view, the craniocervical relationship can be assessed visually by several methods. The basion is the caudal tip of the clivus and can be a critical bony landmark for assessment of the craniocervical relationship. Harris et al. [6, 7] described a simple and reliable method that used the basion-axial interval (BAI) and basion-dens interval (BDI) for accurate assessment of occipitovertebral relationships on initial lateral radiographs in the supine position.

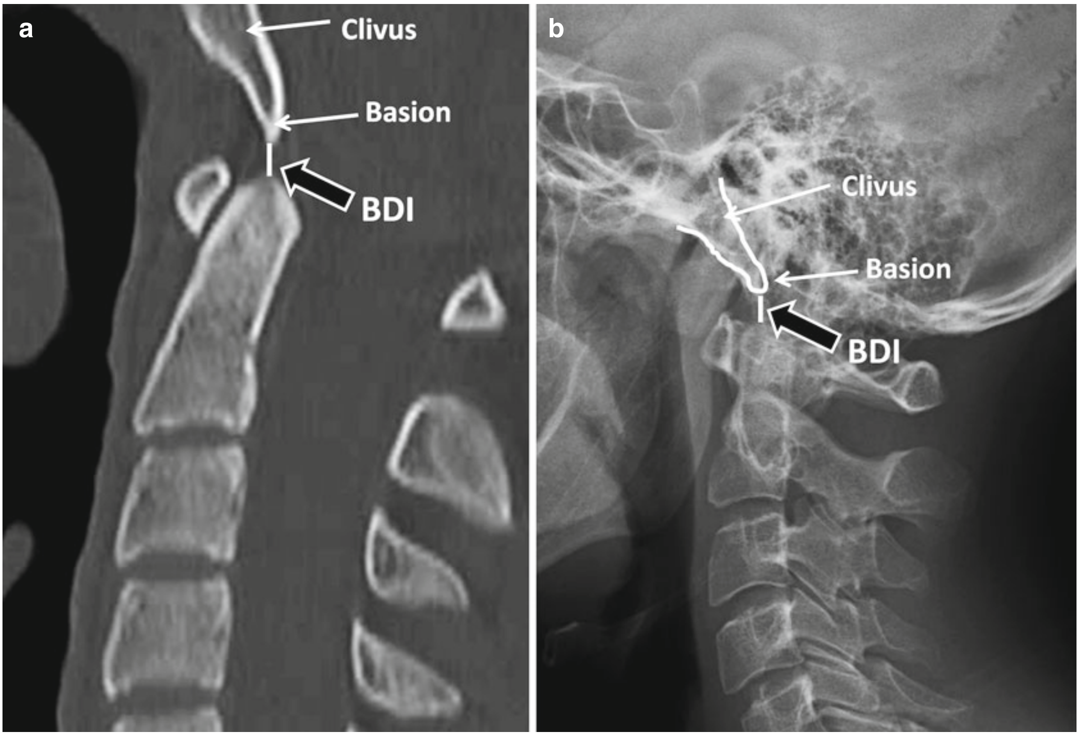


Fig. 4.3 Sagittal CT reconstruction (**a**) and lateral cervical spine radiograph (**b**) demonstrating the basion-dens interval (*BDI*). The BDI is the distance from most inferior portion of basion to closest point of superior aspect of dens

The BAI (Fig. 4.2a, b) is the distance between the basion and a line extending superiorly from the posterior cortical margin of the body of the axis, that is, the posterior axial line (PAL). The BAI should not exceed 12 mm [7].

The basion-dens interval (BDI) (Fig. 4.3) is the distance between the basion and the tip of the dens and should not exceed 12 mm [6]. Abnormal increase in this distance can indicate craniocervical dissociation. An inverted BDI where the tip of the dens is superior to the basion indicates cranial settling/basilar invagination.

The atlantodental interval (ADI) (Fig. 4.4) is a measurement used to evaluate the atlantoaxial relationship. This distance, described by Hinck et al., is considered normal when it is less than 3 mm [8].

The prevertebral soft tissue thickness (Fig. 4.5) should be measured at C3 and should be ≤7 mm in adults [9]. Below C4 the thickness is variable related to variable location of the esophageal takeoff. The neck position in children is pivotal in the assessment of prevertebral soft tissue to prevent false-positive findings.

The facet joints are normally symmetric and uniformly superimposed, with minimal physiologic movement during flexion and extension. The supraspinous and interspinous ligaments, the ligamentum flavum, and the facet joint capsule maintain this anatomic relationship. In the cervical region, the articular facets are small, flat, coronally oriented, and angled approximately 45° from the horizontal plane. This alignment helps to prevent excessive anterior vertebral body translation and is important in weight bearing. This orientation explains the great degree of motion allowed, as well as the relative ease with which cervical facets sublux, dislocate, and lock [10].

Anteroposterior (AP) and Open-Mouth Views

As previously discussed, patient's position is important when evaluating the cervical spine radiograph. On AP view (Fig. 4.6a), the spinous processes should be midline to accurately assess alignment. It is important to make sure all pedicles are present and equidistant from the vertebral body margins. The intervertebral disk spaces

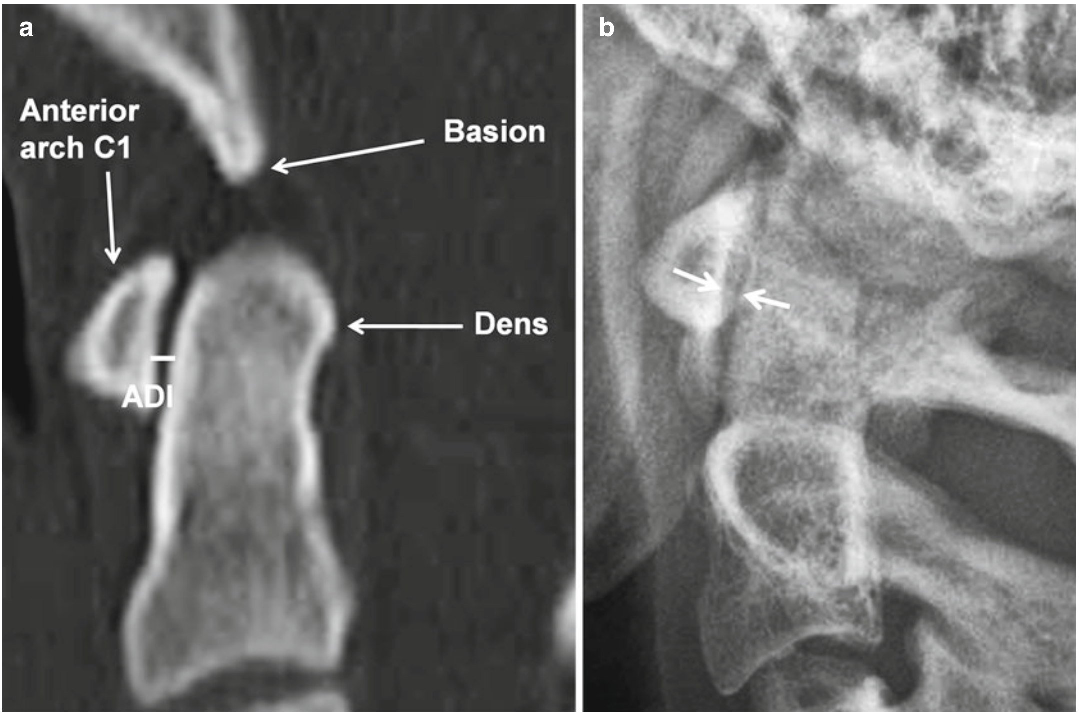


Fig. 4.4 Sagittal CT reconstruction (**a**) and lateral cervical spine radiograph (**b**) demonstrating the atlantodental interval (*ADI*). The atlantodental interval is the distance from the posterior aspect of anterior arch of the C1 vertebra to the anterior aspect of dens

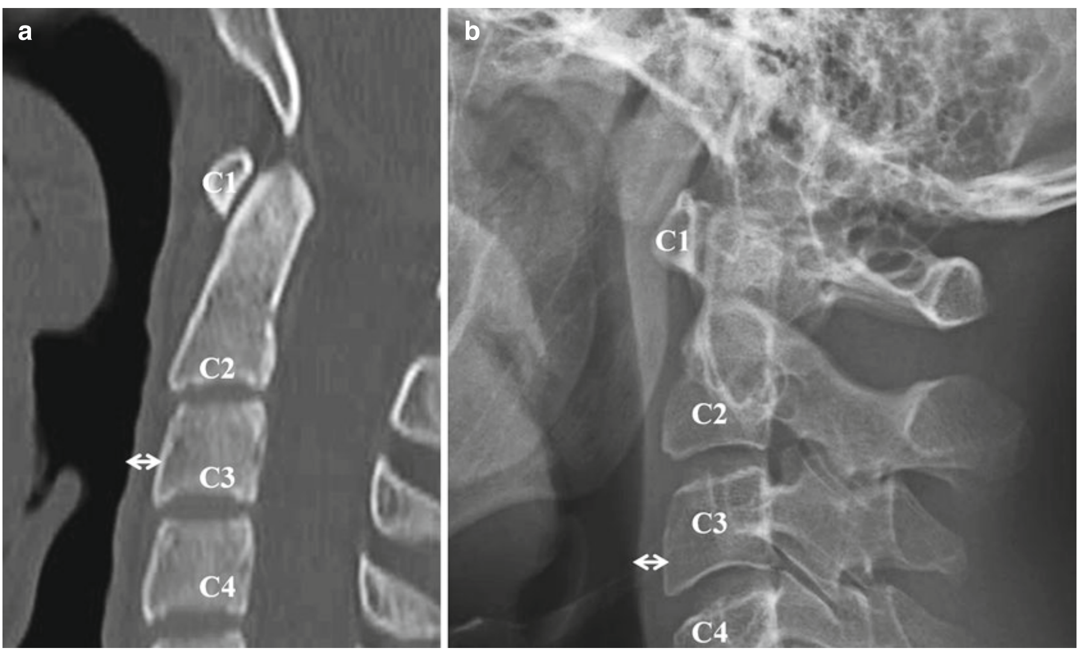


Fig. 4.5 Sagittal CT reconstruction (**a**) and lateral cervical spine radiograph (**b**) demonstrating normal prevertebral soft tissue (*PVST*) thickness. PVST thickness should be measured at C3 and should be ≤7 mm in adults

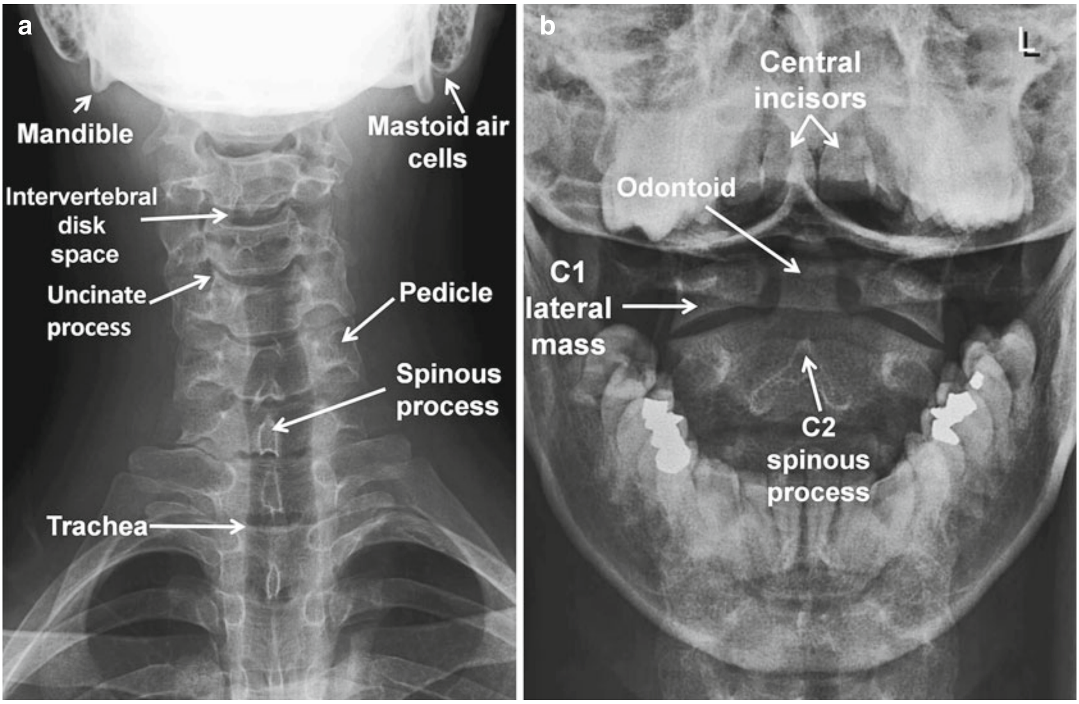


Fig. 4.6 Normal radiographic anatomy on AP cervical spine (**a**) and open-mouth odontoid (**b**) radiographs with labeled pertinent anatomy

should be maintained. Tracheal morphology and lung apices should be assessed for abnormality.

The AP "open mouth" or "odontoid" (Fig. 4.6b) is a coned-down view of the craniocervical junction, obtained with the X-ray beam directed through the patient's open mouth. True AP positioning is necessary as rotation can simulate C1–2 misalignment. On an optimal open-mouth view, the central incisors and occiput are not superimposed over the dens. The lateral margins of the C1 ring should align exactly with the lateral masses of C2 when degenerative spurring is ignored [11]. Displacement of C1 lateral masses by more than 2 mm laterally is abnormal. The open-mouth view is also helpful when evaluating an odontoid fracture or incomplete fusion of the dental ossification center to the C2 vertebral body, an os odontoideum.

Oblique Views

On the oblique projection (Fig. 4.7), the patient's neck is 45° angle relative to the detector plate. The oblique views are typically obtained bilaterally and profile the neural foramina to evaluate bony foramina encroachment. Oblique views can also be useful to confirm appropriate alignment of the facet articular processes.

It is important to identify which neural foramina are being profiled, as oblique projections can be obtained in both AP and PA views. The film should be clearly labeled and the visualized neural foramina are contralateral to the direction the mandible is turned. For example, if the patient's chin is turned to the left, the right neural foramina are profiled.

Flexion/Extension Views

Flexion/extension radiographs can theoretically be diagnostic of ligamentous injury in the acute setting, although muscle spasm commonly stabilizes an otherwise unstable spine resulting in false-negative flexion/extension radiography. Delayed radiographs after a period of time in a soft collar, allowing resolution of muscle spasm, have been shown to unmask an otherwise occult ligamentous instability [12].

In nontraumatic situations, flexion/extension radiographs are used to assess the degree of

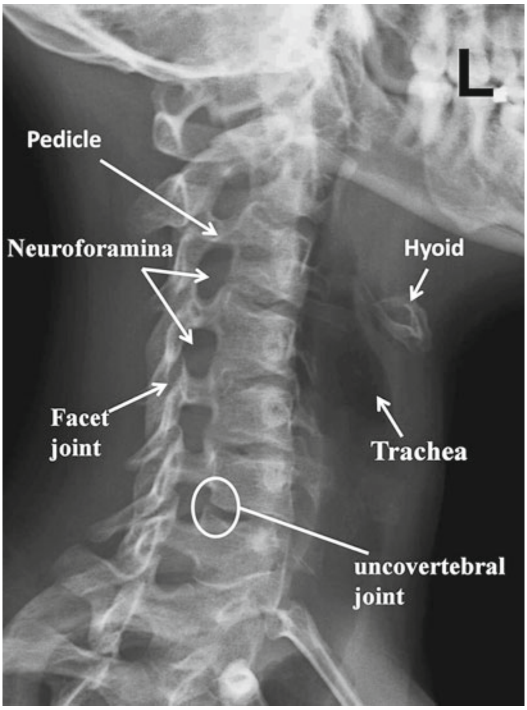


Fig. 4.7 Radiographic anatomy of an oblique projection of the cervical spine. Note that the right neural foramen is profiled as the mandible is turned toward the patient's left (*L*)

instability associated with ligamentous laxity (inflammatory arthropathies, Down's syndrome) or degenerative spondylolisthesis. This can provide valuable information to guide surgical management of chronic cervical spine conditions.

Thoracic Spine

Evaluation of the thoracic spine with radiography allows the assessment of vertebral height, alignment, intervertebral disk spaces, and the presence of fracture and swelling of soft tissues.

Lateral View

The lateral view of the thoracic spine (Fig. 4.8a) should include the seventh cervical vertebra to evaluate the cervicothoracic junction and the first lumbar vertebra to assess the thoracolumbar junction. Evaluation of the high thoracic vertebral bodies commonly requires a swimmer's lateral projection identical to the technique used in complete evaluation of the cervical spine.

On lateral view of the thoracic spine, the anterior margin of the vertebral bodies is slightly concave and the posterior height of the vertebral bodies is greater than the anterior height resulting in a physiologic thoracic kyphosis. The thoracic facets are more vertically oriented in the coronal plane than the cervical facets.

In the traumatic setting, Denis [13] and McAfee et al. [14] independently developed a three-column classification system used to explain the radiographic pattern of injury and subsequent surgical treatment (Fig. 4.9). The system is based on radiographic evaluation of the anterior column (the anterior two-thirds of the vertebral body and intervertebral disk), the middle column (the posterior one-third of the vertebral body and intervertebral disk, as well as the posterior longitudinal ligament), and the posterior column (the osseous neural arch, the facet joints and capsules, the ligamentum flavum, and the supraspinous and interspinous ligaments). Traumatic injury to more than one column of the spine has implications to spinal stability and treatment algorithms.

It is important to evaluate the intervertebral disk spaces in conjunction with the adjacent vertebral endplates. The endplates should be well corticated and distinct (Fig. 4.8). Disk height loss with associated endplate destruction suggests diskitis with associated osteomyelitis, while the association of intervertebral disk height loss with disk vacuum phenomenon, endplate sclerosis, and productive changes is most consistent with common degenerative spondylosis.

Anteroposterior (AP) View

As described on AP view of the cervical spine, adequate patient's position is crucial to the thoracic AP radiograph (Fig. 4.8b). To reduce the radiation dose, collimation is used, although the entire transverse processes and a small portion of the medial ribs should be included.

Thoracic spine AP radiograph is useful for evaluating vertebral body height and alignment. The spinous processes should be aligned and midline. The presence and integrity of the vertebral pedicles is evaluated in the thoracic spine. The interpedicular distance is between the pedicles on AP view and is increased in the setting of a burst fracture relative to adjacent levels. Osseous metastatic disease to the spine commonly involves

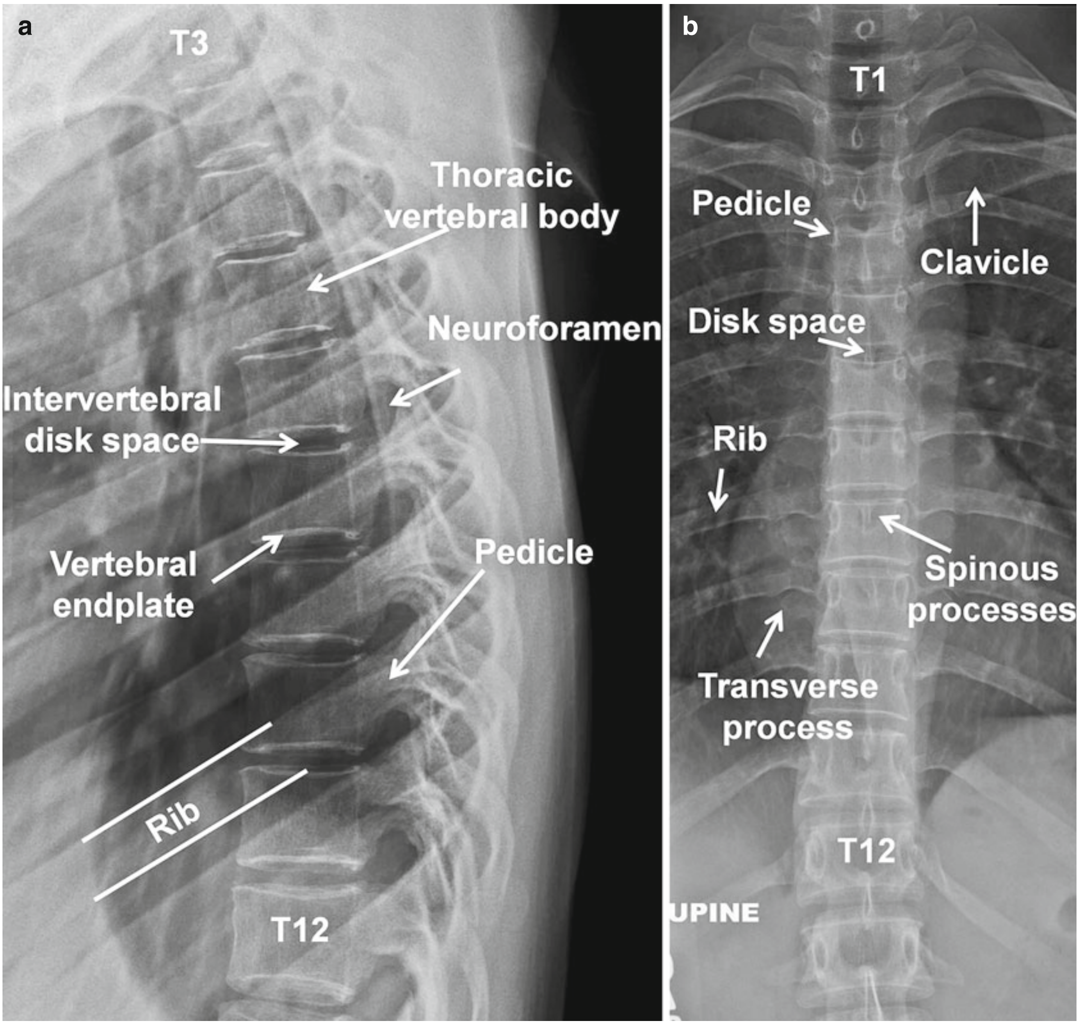


Fig. 4.8 Lateral (**a**) and frontal (**b**) views of the thoracic spine, with pertinent labeled normal radiographic anatomy

the pedicle and can result in an absent pedicle if lytic or a sclerotic pedicle if blastic.

Lumbar Spine

Lateral View

On a lateral view, the lumbar spine characteristically demonstrates a smooth lordotic curvature (Fig. 4.10a). The combination of the thoracic spine kyphosis and lumbar spine lordosis maintains the center of gravity at the central sacrum. The intervertebral disk spaces should appear open, with the L4–L5 disk space typically being of greatest caliber. The neural foramina can be visualized, from T12 through S1. Facet joint orientation transitions to a more sagittal plane in the lumbar spine and allows more flexion and extension than in the thoracic spine but remains limited compared to the cervical spine. A spot film may be used to include the entire sacrum and coccyx for trauma imaging or coned to the lumbosacral junction for routine imaging.

Anteroposterior (AP) View

Optimal radiographic technique for the AP view of the lumbar spine (Fig. 4.10b) is crucial to penetrate the abdominal soft tissues appropriately to obtain adequate exposure and

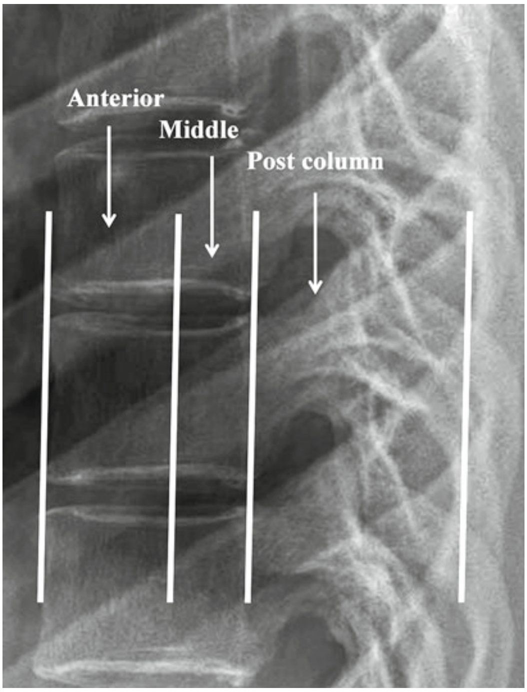


Fig. 4.9 Magnified lateral view of the thoracolumbar junction with lines demarcating the 3-column theory of spinal division. Disruption of more than one column can indicate instability and has implications with regard to treatment

demonstrate vertebral bodies, facet joints, and the spinous processes. An AP radiograph of the lumbar spine should include from the thoracolumbar junction to the coccyx.

The number of non-rib-bearing vertebral bodies should be counted, as transitional and additional vertebral bodies are common at the thoracolumbar and lumbosacral junctions. The most common transitional vertebral body consists of sacralization of the fifth lumbar vertebral body, consisting of a partial or solid, unilateral or bilateral, bony union between an abnormally large L5 transverse process and the sacrum [15]. The spinous processes should be midline of the vertebral bodies and equidistant relative to the pedicles.

Oblique Views

Oblique projections of the lumbar spine are infrequently performed as their utility is controversial and the radiation dose to the patient is significant. However, in young patients with back pain, oblique views can be helpful to confirm the presence of spondylolysis (pars defects) [16]. There is usually no need for routine oblique views in older adults as spondylolysis is much less important in this age group, and there is doubt as to whether it is a significant cause of symptoms in older individuals [17]. Oblique views may be safely omitted in the initial examination for low back pain in the typical patient.

Flexion/Extension Views

Flexion/extension views of the lumbar spine are uncommonly of significant utility in the acute traumatic setting. However, these significantly affect treatment planning with regard to surgical intervention of more chronic back pain. Significant translation (≥3 mm) between flexion and extension can exclude particular treatments related to lumbar stenosis and can shed light on the etiology behind dynamic lumbar radiculopathy.

Whole-Spine Imaging

A brief mention is necessary related to large-format radiography and its use in the spine. Large-format radiography can be performed manually in the absence of digital radiography (radiographic hard copy films shot at different stations and manually connected to include the entire spine). Digital radiography has made large-format imaging much easier with fiducial markers allowing accurate synthesis of multiple acquisitions. The benefit is an overall impression of the spinal balance (Fig. 4.11). In addition, lateral bending, traction, and bolstered films can give further information about the flexibility (and by extension, correctibility) of the curvature, all critical to the surgical management of spinal deformity. Detailed explanation of the evaluation of scoliosis radiographs is, unfortunately, beyond the scope of this chapter.

4.1.2 Computed Tomography (CT)

4.1.2.1 Background and Physics

Computed tomography, which is available since the early 1970s, utilizes an X-ray beam spinning in a circle within a gantry. The detectors, which

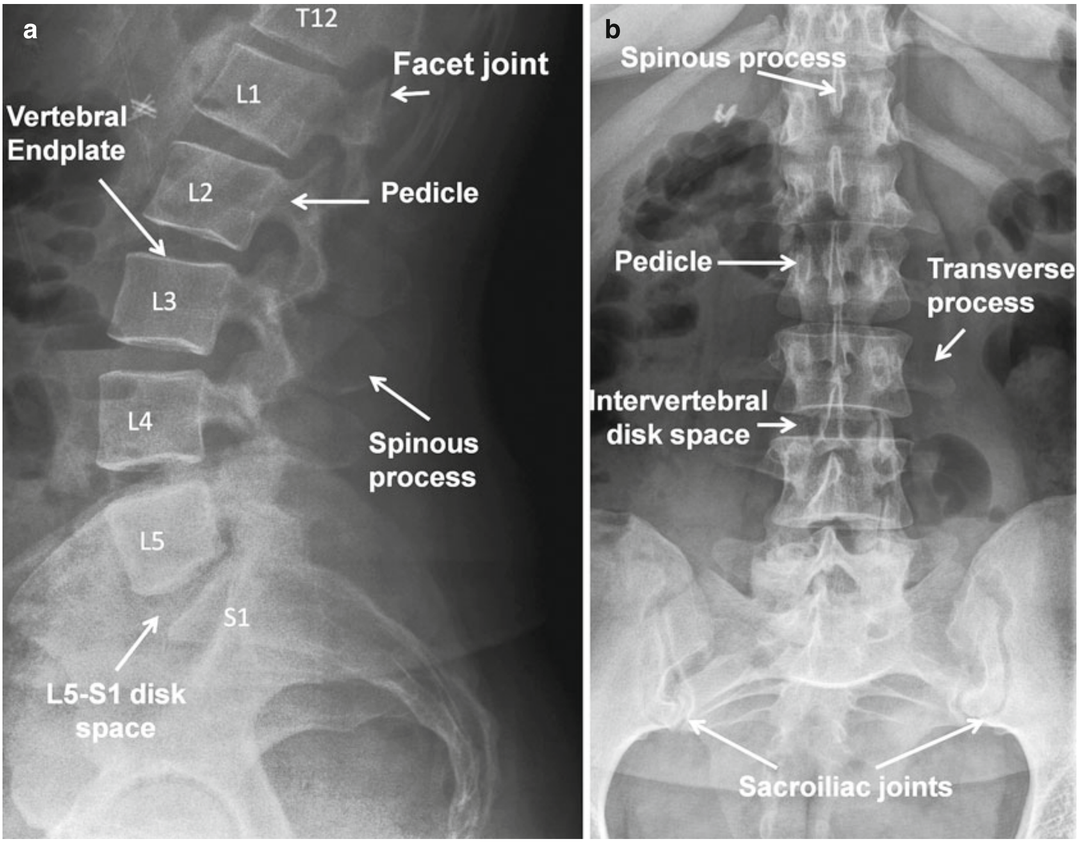


Fig. 4.10 Lateral (**a**) and frontal (**b**) radiographs of the lumbar spine with pertinent radiographic anatomy labeled

are opposite in position relative to the X-ray source, record the strength of the exiting X-ray after passing through the patient; this information is then processed by a computer to produce a detailed two-dimensional cross-sectional image of the body. Early CT scanners had a single X-ray source and single detector. This required a full 360° acquisition followed by the table moving to a new position, where the process was repeated. Early scanners required 30 min or more to do a single CT of the brain. With current technology there are scanners that contain 256 (or more) individual detectors, allowing the table to move quickly through the X-ray beam. This enables acquisition of a tremendous amount of data, resulting in higher-quality images that are easily reconstructed into various multiple planes, thought to be more useful clinically than axial images alone. We can now scan an entire body in a few seconds!

Computed tomography is a noninvasive, painless, and fast imaging diagnostic technique and is the modality of choice for imaging bony detail. Because of the accuracy and ease of CT, there has been a marked increase in usage in the last 30 years. It is estimated that more than 62 million CT scans are currently obtained each year in the USA, as compared with 3 million in 1980, over a 20-fold increase [18]. While CT is a widely used diagnostic technique, several disadvantages need to be considered, such as higher direct medical costs, ionizing radiation, and availability. It is estimated that medical CT scanning contributes approximately 45 % of the US population's collective radiation dose from all medical X-ray examinations [19] and must be used judiciously.

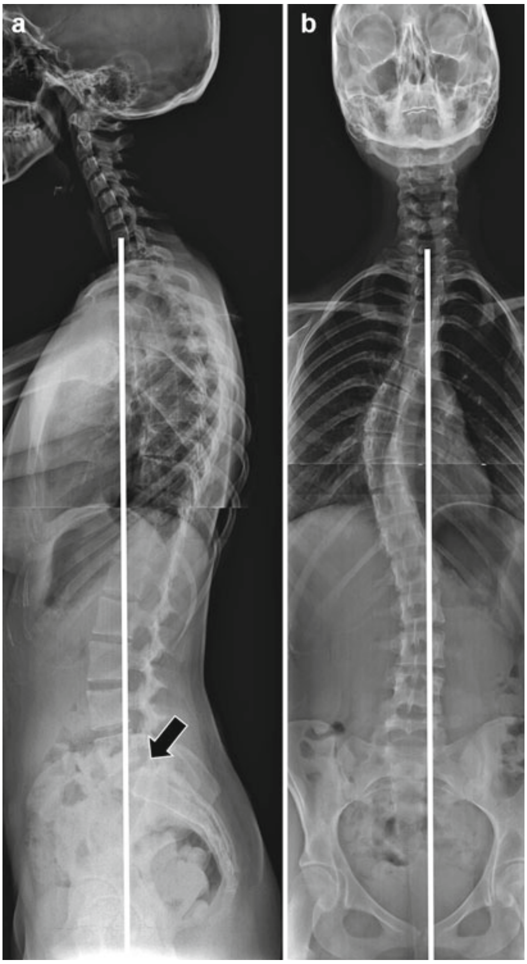


Fig. 4.11 A 19-year-old female with idiopathic scoliosis. Large-format scoliosis radiographs in lateral (**a**) and frontal (**b**) projections. These large-format views allow for evaluation of the overall sagittal balance (**a**, *white line*) and coronal balance (**b**, *white line*). Appropriate sagittal balance is a plumb line that extends from the center of the C7 vertebral body inferiorly to intersect the dorsal margin of the S1 endplate (**a**, *arrow*). Coronal balance is measured with a plumb line that extends from the C7 spinous process inferiorly through the pubic symphysis/midsacrum. This patient demonstrates minimal anterior and left coronal imbalances of a degree that would not likely be considered clinically significant

4.1.2.2 CT for Spine Evaluation

CT demonstrates exquisite bony detail and spatial resolution. The current technology allows reformation of the data in multiple planes. The bony anatomy visible is identical to that seen on radiograph, but the tomographic depiction allows much improved visualization of fractures and bony lesions as discussed above (Fig. 4.12a, b).

CT is commonly used for evaluation of the spine following trauma. Care should be taken to select patients carefully, to minimize radiation dose and medical cost. However, the cost of any diagnostic test needs to be considered with regard to the diagnostic efficacy, the appropriate and rapid work-up of trauma patients, and the risk of misdiagnosis. When considering these factors in the setting of acute cervical spine trauma, C. Craig Blackmore et al.'s cost-effective analysis indicated that screening CT of the cervical spine should be adopted for the initial evaluation of *high-risk* patients [20]. The sensitivity of screening cervical spine CT is higher than that of radiography [21] for fractures of all types. Several other studies have shown CT to be far superior in evaluation of cervical spine trauma [22–25].

The 2007 American College of Radiology Appropriateness Criteria emphasizes this and recommends that "thin-section CT, and not radiography, is the primary screening study for suspected cervical spine injury" [26].

CT is excellent for accurate bony evaluation and is commonly used for assessment of primary or metastatic neoplasms involving the spine. In the evaluation of diskitis and osteomyelitis, CT allows excellent visualization of the characteristic endplate destructive changes (Fig. 4.13a, b).

Radiographic evaluation of the postoperative spine can be challenging. Although the effectiveness of conventional CT can be limited by severe beam-hardening artifacts, the evolution of multichannel CT has made available new techniques that can help minimize these artifacts [27]. Postoperative imaging is typically performed to assess the progress of osseous fusion, to confirm the correct positioning and the integrity of instrumentation, to detect suspected complications (infection, nonunion, or hardware loosening), and to detect

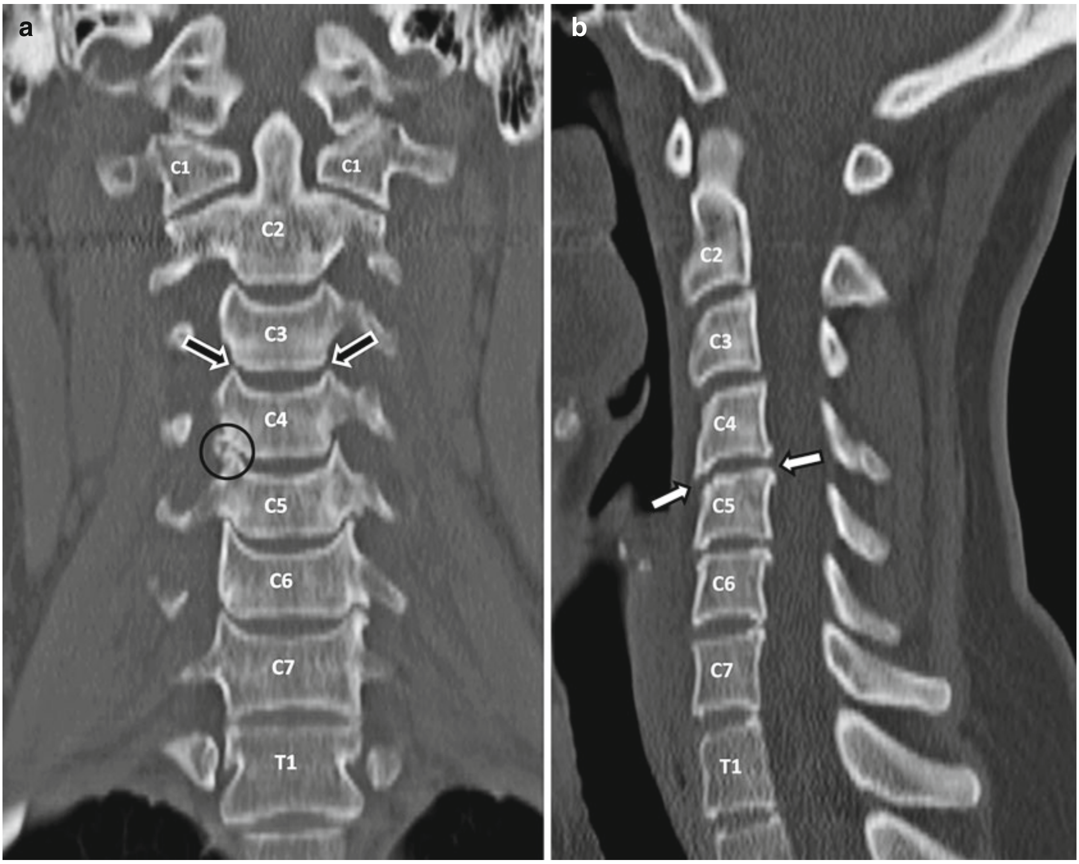


Fig. 4.12 CT coronal (**a**) and sagittal (**b**) reconstructions of a 43-year-old female with chronic neck pain demonstrate the tomographic anatomy afforded by current CT techniques. Coronal reconstruction (**a**) shows normal uncovertebral joints (*black arrows*) at C3–4, with typically degenerative appearance to the C4–5 uncovertebral joints, right greater than left (*circle*). Sagittal reconstruction (**b**) shows great bony detail and in this patient shows degenerative disk disease with endplate productive change greatest at C4–5 (*white arrows*)

new disease or disease progression (Fig. 4.14) [28]. Radiography is the noninvasive modality most commonly used for the assessment of fusion, although CT has been reported to be more accurate [29].

Evaluation of the intraspinal soft tissues in patients who are unable to undergo MRI can be challenging. CT myelography can be used in these situations for the evaluation of nerve root impingement-related spinal degenerative disease. Following the injection of iodinated myelographic contrast material into the thecal sac, CT is performed. The opacification of the thecal sac filled with contrast affords more accurate evaluation of central spinal canal and neural foraminal narrowing.

4.1.3 Magnetic Resonance

4.1.3.1 Background and Physics

MR uses a powerful static magnetic field (commonly referred to as the field strength of the magnet, measured in Tesla (T)) to align the magnetization of atoms in the body. Current clinical MRI systems range from 0.2 to 3.0 T. Once the patient has been placed in this powerful static magnetic field, radio-frequency pulses systematically alter the alignment of these magnetized protons. The frequency at which the protons realign along the static magnetic field is tissue specific, and this information is used to construct an image of the scanned area of the body. A more extensive

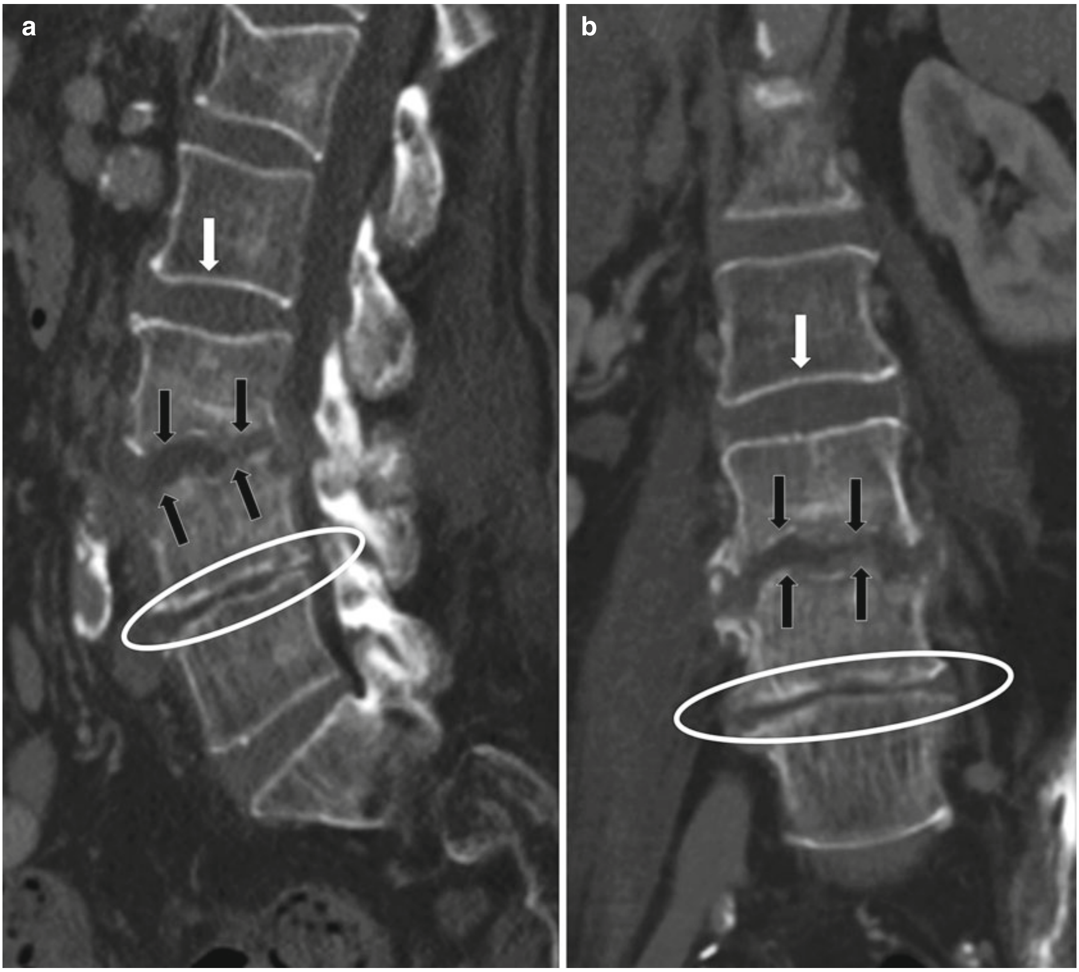


Fig. 4.13 A 69-year-old male 2 months following posterior interspinous fusion surgery. Patient had subsequent postoperative infection with Staphylococcus aureus with ongoing and worsening low back pain and radicular leg pain. Sagittal (**a**) and coronal (**b**) reconstructed CT images show the typical endplate destructive changes of diskitis/osteomyelitis (*black arrows*). Contrast the destructive endplate appearance with the normally corticated L2–3 endplates (*white arrows*). L4–5 disk space is narrowed and there is marginal bony production, but the endplates remain well corticated, typical of degenerative changes (white ellipse)

discussion of MR physics is beyond the scope of this chapter, but the different appearances of the same tissue between different sequences (T1, T2, STIR, fat-suppressed sequences) take advantage of these physical properties, and altering the time at which the protons are measured can allow the different properties of the tissues to be accentuated.

Producing a high-quality MR image is dependent on a number of factors. Jarvik et al., in analyzing the quality of lumbar spine MR images, showed that field strength was the strongest predictor of quality, with the higher magnetic field strength producing higher-quality images [30]. The individual parameters of the sequences, the gradient strength, the receiver coils used to pick up the signal information, the software platform, and many other factors differ between MR systems and contribute greatly to image quality as well. Not all MRI are equal!

MR imaging advantages are multiple, including better spatial and contrast resolution of soft tissue and muscle relative to other imaging modalities. In addition, unlike CT scans or

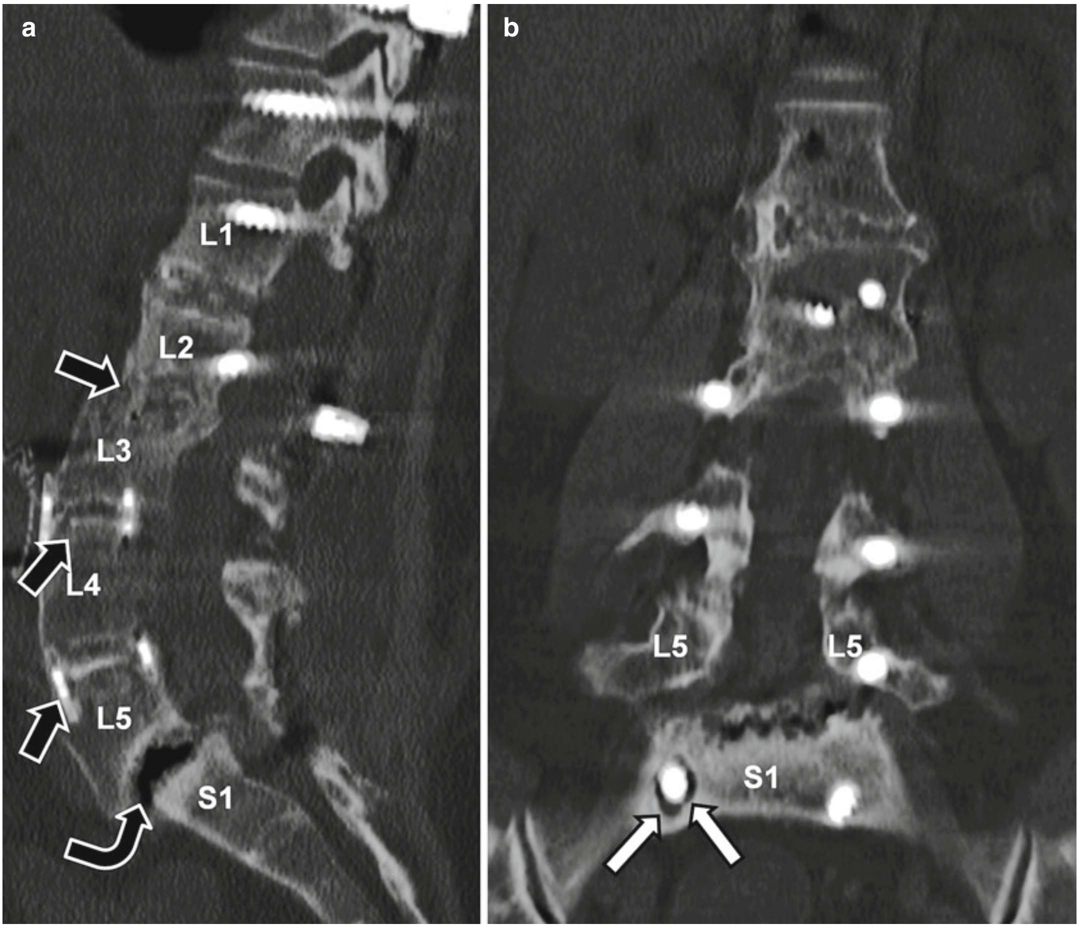


Fig. 4.14 Sagittal (**a**) and coronal (**b**) reconstructed CT images in a 57-year-old female who underwent prior T10–S1 revision spinal fusion 22 months prior to this CT scan. The patient has posterior spinal instrumentation with pedicle screws at all levels and interbody grafts as the solidly fused L2–4 levels (*black arrows*). Findings at L5–S1 are typical of nonunion with vacuum disk phenomenon (**a**, *curved arrow*, indicative of micromotion) and S1 pedicle screw loosening (**b**, *white arrow*). CT is excellent at evaluating the postoperative spine

conventional radiograph, MRI uses no ionizing radiation. MR has become the imaging modality of choice for the evaluation of most spinal pathologies.

Disadvantages of conventional MR imaging include long imaging times and high cost. The prolonged image acquisition time makes MR acutely susceptible to motion artifact and the patient must have the ability to lie still in the bore of the magnet for long periods of time. Unlike CT or radiography, there are many more potential contraindications to MRI. These include claustrophobia, obesity, and metal and implanted devices that are not MRI compatible (pacemakers, spinal stimulators, ferromagnetic material in high-risk areas of the body, and many others). The compatibility of implantable devices is a significant concern and much effort is spent by imaging professionals to confirm the MRI safety of patients prior to scanning (MRIsafety.com is a website where MR compatibility of most implantable devices can be found). Due to the clear advantages of MRI over other imaging modalities, research and development is ongoing to create MR-"safe" implantable devices, such as pacemakers.

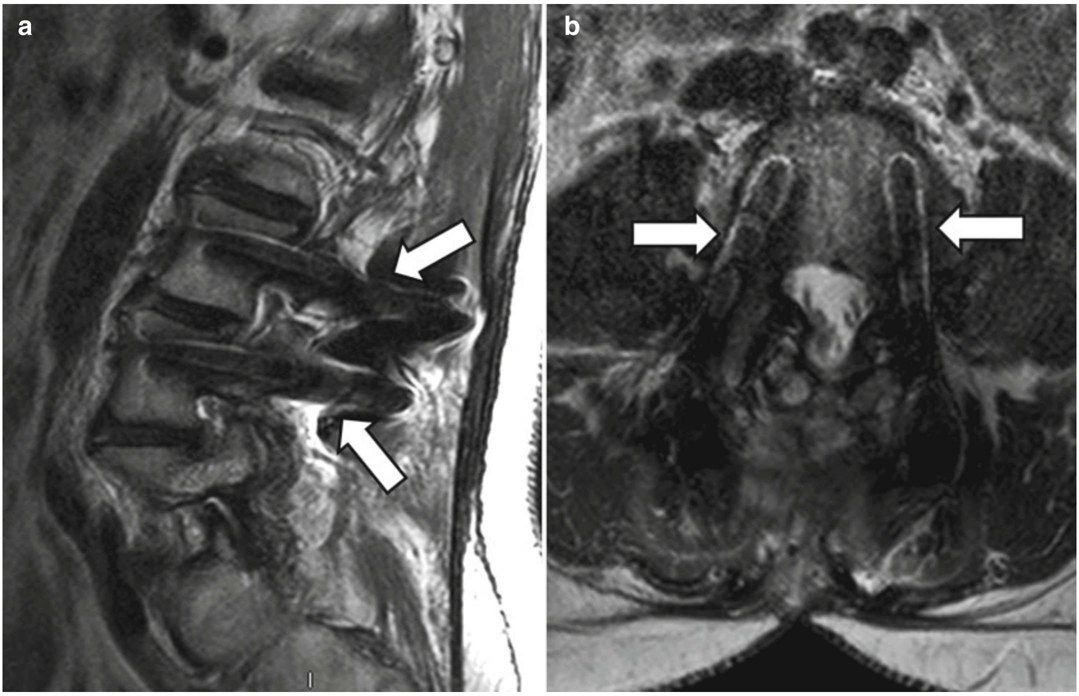


Fig. 4.15 Sagittal (**a**) and axial (**b**) T2-weighted sequences at the level of posterior spinal instrumentation demonstrate susceptibility artifact typical of present-day titanium spinal hardware (*arrows*). Titanium implants are non-ferromagnetic and produce far less artifact compared to older stainless steel hardware. Even so, titanium produces enough artifact to obscure detailed evaluation of adjacent tissues and distorts their appearance

Most metals are MR safe if they are not located in critical anatomic areas; however, it can significantly degrade MRI imaging quality. Metal alters the local magnetic field producing magnetic susceptibility artifact, distorting the normal physical properties of the adjacent tissues. The effect is most severe with ferromagnetic stainless steel implants or foreign bodies. Modern implants made of titanium alloys are less ferromagnetic and thus produce less severe magnetic susceptibility artifacts, but these artifacts remain a significant obstacle to visualization of areas in close proximity to metallic hardware (Fig. 4.15).

Gadolinium-based chelates can be used in MRI as IV contrast agents. The gadolinium produces a paramagnetic effect that shortens the T1 relaxation time of adjacent protons. This creates high T1 signal in tissues with increased vascularity. The appearance of enhancement can be further accentuated by fat suppressing the post-contrast images (Fig. 4.16).

4.1.3.2 MR for Spine Evaluation

MR imaging of the spine can depict alterations in both the anatomy and tissue properties, but these findings clearly need to be considered within a clinical context [31]. MRI is the study of choice for most spine pathology. MRI is helpful in the evaluation of intervertebral disk signal (dehydration) and contour abnormalities, such as disk bulge and herniation, evaluation of the bone marrow, neural foraminal patency, spinal canal narrowing, and facet joints in patients with nontraumatic spine pathology. In a traumatic setting, MRI is highly accurate for evaluation of vertebral body fracture and is the method of choice for imaging ligament injury, traumatic disk protrusion, and posttraumatic spinal cord compression or injury [32].

Full-spine and whole-body MR imaging are useful in the assessment and diagnosis of multifocal lesions of the skeleton. MR imaging, with an accuracy of 90 %, is the diagnostic

Fig. 4.16 Same patient as Fig. 4.13, with diskitis/osteomyelitis of the L3–4 disk space. Sagittal T1-weighted fat-suppressed MR image, following the administration of IV gadolinium, demonstrates the typical T1 paramagnetic effect of gadolinium chelates, with hypervascular tissues demonstrating robust enhancement (*white asterisks*)

imaging procedure of choice for spinal osteomyelitis [33].

Sequences acquired during spine imaging include, at a minimum, sagittal fluid-sensitive T2-weighted sequence, sagittal T1-weighted sequence, and axial T2-weighted sequences. Many centers (including the author's) include a STIR sequence as a sagittal fat-suppressed fluid-sensitive sequence. This allows edema of bone and soft tissue to stand out against a strongly fat-suppressed background (Fig. 4.17). Axial images can be acquired in a stacked fashion, throughout the imaged portions of the spine. Commonly axial images acquired parallel to the disk spaces are used in addition to the stacked axial images. This is particularly useful in the lumbar spine, where lumbar lordosis commonly results in a disk axis that is oblique to the orthogonal axial plane. In the cervical spine, gradient axial images can decrease the T2 artifact associated with flowing CSF and allow a more accurate evaluation of the central canal (Fig. 4.18).

Anatomic structures that are critical to evaluate on MR imaging of the spine are numerous. In imaging any portion of the spine, the bone marrow signal should be critically assessed as diffuse marrow disease commonly involves the spinal marrow. The marrow in an adult should be relatively fatty in content and, as such, be of relatively high T1 signal. With marrow replacement related to diffuse metastatic disease, blood cancers (myeloma, lymphoma, leukemia), or profound anemia (sickle cell anemia, myelofibrosis), the T1 signal drops. The adjacent disk is a useful internal control and the marrow signal should always maintain a T1 signal that is higher than the disk (Fig. 4.19).

In the cervical spine (Fig. 4.17), sagittal images are critically evaluated to confirm spinal segmental alignment, normal cord signal, normal disk height, degree of degenerative disk, facet joint, and uncovertebral joint disease. In the cervical spine, the neural foramina are oriented anterolateral making their assessment on sagittal imaging more difficult as they are not in a true sagittal plane. The axial images are most critical for evaluation of the neural foramina. Neural foraminal narrowing in the cervical spine can be acutely related to disk herniation but more typically is the result of a chronic process related to uncovertebral joint and facet joint arthrosis (Fig. 4.18).

Similar evaluation in the lumbar spine is necessary (Fig. 4.20). Sagittal images are scrutinized for disk hydration and height loss; the conus is assessed for appropriate position (the cord should terminate between T12 and L2); and each spinal level is assessed segmentally. It is important at each level to assess the degree of disk disease or disk herniation, facet arthrosis, and ligamentum

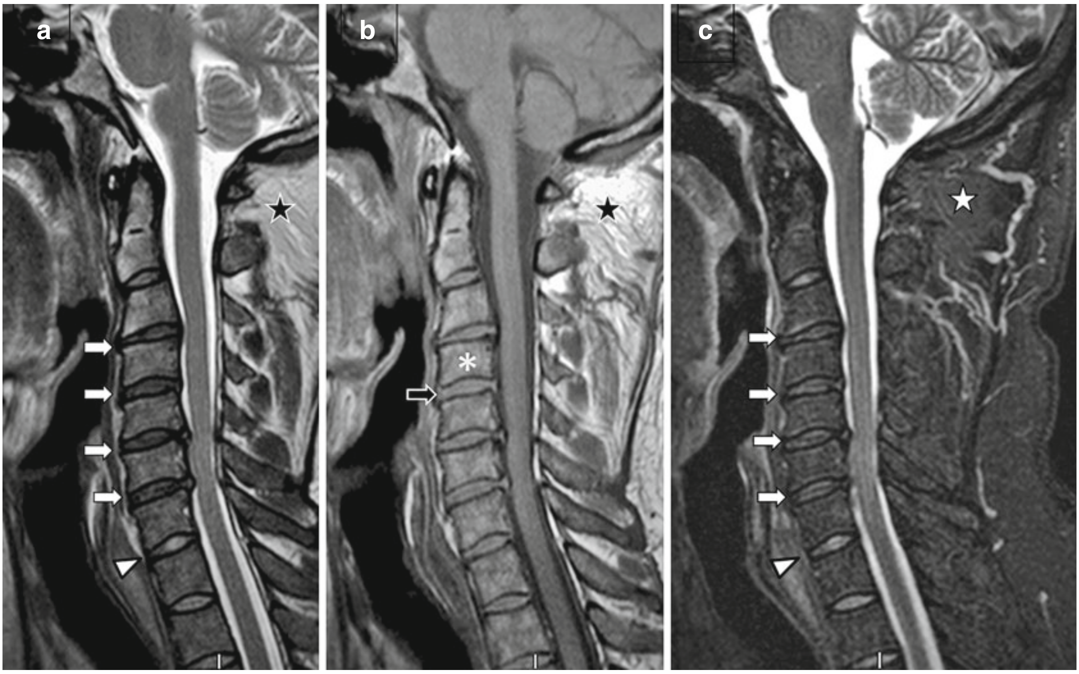


Fig. 4.17 Sagittal MRI sequences of a typical MRI of the cervical spine. (**a**) Sagittal T2-weighted sequence with the typical high signal of cerebral spinal fluid. Disk desiccation is illustrated by dark disks on T2-weighted sequences (*white arrows*), in contrast with normally hydrated disks (*arrowheads*). The cervical cord is well assessed on fluid-sensitive sequences. (**b**) T1-weighted sequence is excellent at evaluating the bony anatomy and overall marrow signal (which should not be hypointense/darker (*asterisk*) compared to adjacent disk (*black arrow*)). (**c**) Sagittal STIR sequence is a fat-suppressed, fluid-sensitive sequence. Note the dark fat on the STIR (*white star*) compared to the bright fat on T2 nonfat-suppressed and T1-weighted sequences (*black stars*)

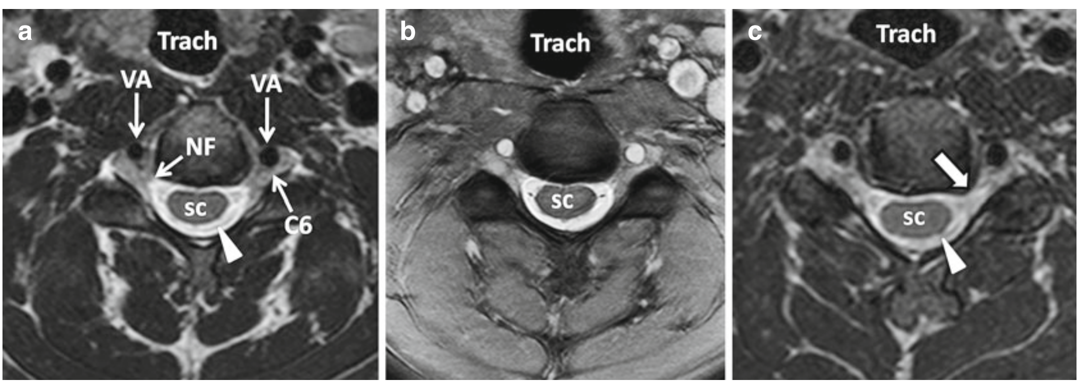


Fig. 4.18 Axial MR images of the cervical spine at C4–5 (**a**, **b**) and C5–6 (**c**). (**a**) Axial T2-weighted image showing normal anatomy, including central spinal canal (containing CSF and spinal cord (*SC*)), neuroforamen (*NF*), exiting C6 nerve roots (*C6*), and vertebral arteries (*VA*). Note the subtle signal heterogeneity of the CSF caused by CSF motion on T2-weighted sequences (*white arrowheads* **a**, **c**). (**b**) Axial gradient sequence does not suffer from the motion artifact of the CSF, with distinct fluid seen surrounding the cord. Additional advantage to gradient echo images is the difference in signal intensity of the disk compared to bone, allowing differentiation of acute herniation from chronic bony spurs. (**c**) Axial T2-weighted sequence at the C5–6 level demonstrating uncinate hypertrophy contributing to mild left-sided neuroforaminal narrowing (*white arrow*, **c**). Trachea (*Trach*)

Fig. 4.19 Sagittal T1-weighted nonfat-suppressed images in two different patients. (**a**) A 57-year-old female with degenerative changes of the spine. Note the hyperintense (*bright*) appearance of her vertebral bodies relative to the adjacent disks. This is typical of adult spinal marrow. (**b**) A 62-year-old male with multiple myeloma. The T1 signal of this patient's marrow is markedly more hypointense (*darker*) compared to the patient in (**a**) and is isointense to adjacent disk. In this patient the abnormal T1 signal is related to the marrow replacement by diffuse myelomatous involvement. Note the pathologic fracture of T10 through a myeloma lesion (*arrow*)

flavum hypertrophy and evaluate the impact of these factors on the traversing and exiting nerve roots at each level (Fig. 4.20c).

4.1.4 Ultrasound (US)

The use of ultrasound in adults for evaluation of the spine is largely restricted to the identification of superficial soft tissue abnormalities, including postoperative fluid collections. Ultrasound is less accurate than CT and MR in detecting soft tissue masses, particularly those arising close to the bony boundaries of the pelvis and sacrum. However, ultrasound is accurate in determining the size of superficial soft tissue masses and is therefore useful and cost-effective in selective circumstances.

US is a well-established method of investigating the spinal canal and cord as well as the

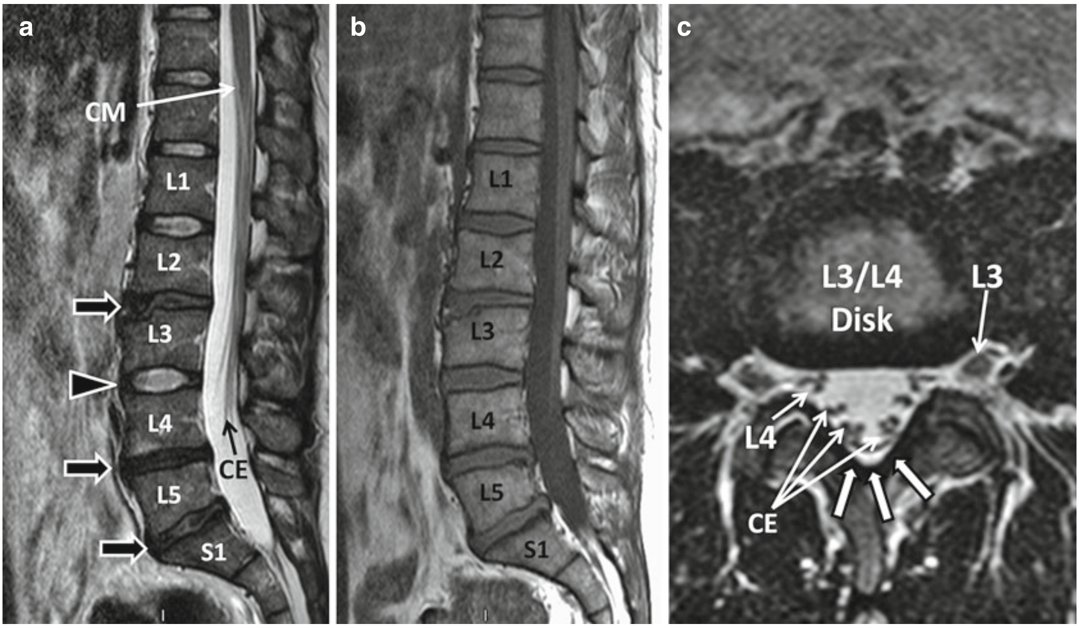


Fig. 4.20 Sagittal T2 (**a**)-, T1 (**b**)-, and axial T2 (**c**)-weighted acquisitions through the lumbar spine. (**a**) Sagittal T2 showing disk desiccation and narrowing of L2–3, L4–5, and L5–S1 (*thick black arrows*). Contrast with normal L3–4 disk (*arrow head*). Conus medullaris (*CM*) and nerve roots of the cauda equina (*CE*). (**b**) Sagittal T1 showing normal marrow hyperintensity compared to disk. (**c**) Axial T2 sequence at the L3–4 level, demonstrating exiting L3 nerve roots (*L3*), traversing L4 nerve roots (*L4*), and the rootlets of the cauda equina (*CE*). Ligamentum flavum (*thick white arrows*)

meningeal coverings in newborns and infants [34–36]. US of the spinal cord is performed in newborns with signs of spinal disease (cutaneous lesions of the back, deformities of the spinal column, neurologic disturbances, suspected spinal cord injury due to traumatic birth, and syndromes with associated spinal cord compression).

4.1.5 Nuclear Medicine

Radionuclide bone imaging is not specific, but its excellent sensitivity makes it useful in screening for many pathologic conditions. Radionuclide bone imaging is quick, relatively inexpensive, widely available, exquisitely sensitive, and valuable in the diagnostic evaluation of numerous pathologic conditions.

Bone scintigraphy with technetium-99m-labeled diphosphonates is one of the most frequently performed of all radionuclide procedures. It serves as a "physiologic" imaging modality with the ability to localize areas of bone turnover. This is particularly useful in evaluating the skeleton for bony metastases or assessing fractures. In the absence of underlying bone abnormalities, three-phase bone scintigraphy can be both sensitive and specific for osteomyelitis [37].

Skeletal single photon emission computed tomography (SPECT) using technetium-99m (^{99m}Tc)-labeled phosphate agents can be used to increase sensitivity and specificity for a number of disorders, most commonly spondylolysis [38]. The advantage of SPECT is the multiplanar capability and allows more precise localization of increased radiotracer bony uptake. More recently SPECT cameras have been incorporated with CT to allow fusion of the exquisite anatomic data of CT with the functional uptake data afforded by scintigraphy.

Positron emission tomography (PET) is widely used for evaluation of malignancy and whole-body imaging to assess disease burden. PET uses compounds labeled with isotopes to target particular tissues in the body and detects the gamma rays emitted by these isotopes within the target tissue. Most widely used is glucose labeled with F^{18} (flourodeoxy-D-glucose) that effectively targets hypermetabolic tissues that metabolize large amounts of glucose (FDG-PET). Although this is most commonly used to image FDG avid tumors, infection and inflammation are FDG avid as well and FDG-PET can be used to effectively image infectious and inflammatory diseases of the spine. However, increased osseous FDG activity has also been observed in inflammatory arthritis, in acute fractures, and in normally healing bone up to 4 months after surgery [39]. It is most common for the metabolic data acquired through PET to be combined with the anatomic data by fusing with CT or MRI images.

A combined study consisting of WBC imaging and complementary bone marrow imaging performed with technetium-99m (99mTc) sulfur colloid is approximately 90 % accurate for diagnosing osteomyelitis [40]. Drawbacks to labeled leukocyte imaging include the labor-intensive leukocyte labeling required, the inconsistent availability of the exam, and the need for direct contact with blood products.

4.1.6 Bone Densitometry

Dual-energy X-ray absorptiometry (DXA) uses the relative attenuation of two different energy X-ray beams to estimate bone density. The estimate of bone density in g/cm^2 is then compared to a normal control population, and the number of standard deviations is reported as the T-score. For example, a T-score of −1.5 indicates bone density that is 1.5 standard deviations below the normal control population mean. The World Health Organization has established cutoff levels that are based on the lifetime risk of fracture of a postmenopausal female. T-scores of −1.0 or above are considered normal bone density, between −1.0 and −2.5 is considered osteopenia, and −2.5 or below qualifying as osteoporosis [41]. Clinically, bone density measurements can be used to predict future skeletal fracture risk, provide serial monitoring, and guide medical treatment. Central DXA has been shown to be most accurate and entails measurement of the spine and hips. Peripheral DXA is less accurate but can be used when hips or spine is not measureable related to orthopedic hardware or significant degenerative arthrosis.

4.2 Case Presentations of Spinal Imaging

4.2.1 Imaging of Trauma

4.2.1.1 Cervical Spine Trauma: Case Presentations and Differential Considerations

Case 1

A 22-year-old man was a passenger in vehicle that was rear-ended while at a stop sign. The speed of the collision was estimated at 10 mph. Airbags did not deploy. The patient is awake, alert, and not intoxicated. He complains of mild neck pain but has no complaints otherwise. On physical exam, the patient has no focal area of tenderness to palpation, and no focal neurologic deficits are found. He is able to actively rotate his neck back and forth without significant discomfort.

Case 2

A 45-year-old man is brought in by his wife after falling off a ladder while cleaning his gutters. He estimates he fell approximately 10 ft, braced himself with his arms, but partially landed on this neck, with a hyperflexion mechanism. His neck and right arm are painful. He is alert and awake with a GCS of 15. He is not intoxicated.

Case 3

A 35-year-old man arrives by ambulance after a high-speed MVC. The patient is sedated and was intubated in the field and a cervical collar is in place. The paramedics report that the patient had severe neck pain and inability to move his upper and lower extremities prior to intubation. Exam is limited by sedation and intubation, but there are clear signs of trauma on initial survey.

4.2.2 Cervical Spine Trauma: Imaging Decision-Making

Trauma patients with potential cervical spine injury (CSI) can be divided into two groups, with different diagnostic pathways. The first group is classified as low risk and consists of patients with a Glasgow Coma Scale (GCS) score of 15, who are awake, alert, cooperative, and non-intoxicated without distracting injury. Distracting injuries are injuries that hinder the reliability of the questioning and examination of the patient [42]. These distracting injuries include long-bone fracture, large laceration, degloving injury or crush injury, large burns, and any other injury producing acute functional impairment.

Within the low-risk group, these patients can be subdivided into patients that require cervical spine radiography for evaluation of their cervical spine and those that do not require cervical spine radiography and can be clinically cleared.

Two large, prospective studies have been performed for developing criteria that rule out significant CSI after blunt trauma based on history and clinical examination. Both studies included only awake and alert patients.

The National Emergency X-radiography Utilization Study (NEXUS) [2] used five criteria to define a low probability of injury (the NEXUS "No's," Table 4.1). The study looked at 34,069 patients who had experienced blunt trauma. In those patients satisfying the NEXUS no-risk criteria, there was a 99.8 % negative predictive value for cervical spine injury. The patient in Case 1 satisfies the NEXUS criteria and does not require any cervical spine imaging.

The Canadian C-spine Rule (CCR) study, which defined three high-risk and five low-risk criteria [43] (Table 4.2), was developed concomitantly with the NEXUS criteria, in a population of 8,773 patients. The CCR relies on the absence of high-risk factors, the presence of low-risk factors, and a pain-free rotational range of motion to exclude the need for radiography. The patient in Case 1 has no CCR high-risk criteria, was involved in simple rear-end MVC (one of the CCR low-risk criteria), and had painless rotational range of motion. Based on CCR evaluation, this patient does not require cervical imaging and can be clinically cleared.

Table 4.1 NEXUS "No's"

1. *NO* posterior midline tenderness
2. *NO* focal neurologic deficit
3. *NO*rmal alertness
4. *NO* evidence of intoxication
5. *NO* painful distracting injury

Table 4.2 Canadian C-spine rule

High-risk factors 1. Age >65 years 2. Dangerous mechanism Fall from >1 m/5 stairs Axial load to head (diving injury) High-speed MVA (>100 km/h), rollover, and ejection Motorized recreational vehicle injury Bicycle collision 3. Paresthesias in extremities	If any high-risk factors are present, proceed to radiography
Low-risk factors (if any low-risk factors are present and high-risk factors are absent, proceed to ROM) 1. Simple rear-end MVC 2. Sitting position in ED 3. Ambulatory at any time 4. Delayed onset neck pain 5. Absence of midline C-spine tenderness	If none of these are present, proceed to radiography
Able to actively rotate neck 45° left or right?	If pain-free, no radiography is necessary

Adapted from [43]

The Canadian study group compared the CCR criteria with the NEXUS criteria in a group of 8,283 patients. Based on the CCR criteria, the number of radiologic studies was reduced, although the methodology of this comparative study was criticized by several authors [44]. Given the absence of large, independent prospective studies that compare both criteria, no clear superiority of either study criterion can be declared.

Imaging decisions in Case 2 are largely based on clinical suspicion of cervical spine injury. If clinical suspicion is low, three-view radiographic cervical spine series (AP, lateral, and open-mouth odontoid views) can be used to assess for injury and, if no injury is demonstrated and the patient has an appropriate exam, the cervical spine can be cleared. In low-risk patients with neurological symptoms and high clinical suspicion, advanced imaging is so commonly indicated that several authors have advised CT scanning of the cervical spine primarily, obviating the time, cost, and radiation dose associated with cervical spine radiographs [45, 46]. In the low-risk patients with a high clinical suspicion of injury (i.e., significant pain and/or paresthesias) and no findings on the radiographs or CT scan, it may be advisable to perform MRI to detect potential soft tissue/ligamentous injury.

When injuries are diagnosed with X-ray imaging, additional imaging is advised for optimal planning of treatment (i.e., CT scan or MRI) [47]. If the three-view series is inadequate for assessment, a CT scan of the cervical spine may be indicated, depending on the risk assessment of the patient.

The second group is high mechanism of injury and consists of unconscious, sedated, intoxicated or noncooperative patients, those with a distracting injury or an altered mental state (GCS < 15), and those with neurological symptoms referable to a cervical spine injury. Case 3 represents this type of patient, where cervical spine injury is clinically probable. In these patients, attempts to obtain diagnostic radiographs are not advocated as they are typically time-consuming and costly, and higher-level imaging is inevitably necessary. CT should be the first-line imaging, with MRI obtained as necessary.

While CT scanning is superior to radiography in detecting injuries, soft tissue injuries such as ligamentous, intervertebral disk, and spinal cord injuries may be missed on CT. Currently, MRI is the most sensitive imaging technique for soft tissue injuries of the C-spine, including spinal cord injuries. However, MRI is less sensitive for osseous injuries (55 %) than CT (90–100 %) [47, 48]. Consequently, MRI as the sole imaging technique is not sufficient to clear the cervical spine and should be used in addition to CT. Furthermore, there are several significant disadvantages of MRI in high-risk patients, most notably the difficulty in monitoring and supporting the vital conditions of the severely injured patients during the transport and imaging procedure itself. In addition, MR imaging may be contraindicated in the presence of ventilation equipment or metal implants (pacer, spinal stimulator, etc.). MRI should primarily be considered in patients with unexplained neurological symptoms or focal neurologic deficits and for evaluation of extensive injuries.

4.2.3 Cervical Spine Trauma: Imaging Findings

Case 1

No imaging was necessary as the patient satisfied both the NEXUS criteria and the Canadian C-spine Rule, obviating the need for imaging.

Case 2

Normal radiographs of the cervical spine were obtained (not provided).

Case 3

CT images of the patient in the high-speed MVC (Fig. 4.21) show a triangular fracture fragment off of the inferior anterior vertebral body of C5, with retropulsion of the C5 vertebral body and marked splaying of the spinous processes. This appearance is consistent with a flexion teardrop fracture.

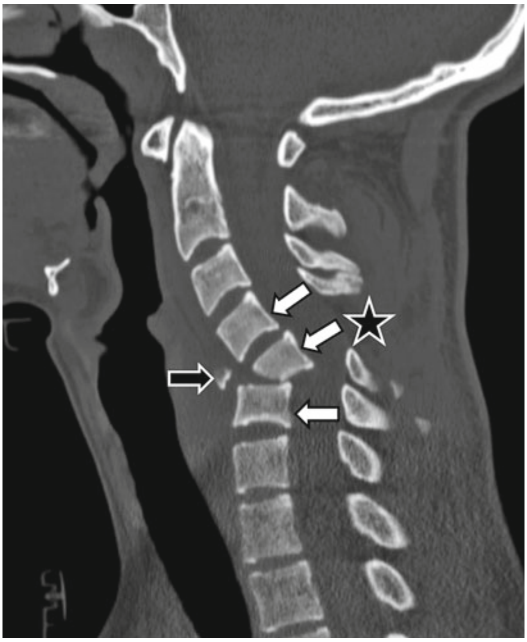


Fig. 4.21 Sagittal reconstruction of a cervical spine CT in a 35-year-old male involved in a high-speed motor vehicle accident demonstrates focal kyphosis at C5 with a fracture fragment donated from the anterior inferior C5 vertebral body (*black arrows*). Note the complete disruption of the posterior longitudinal line (*white arrow*) and the marked splaying of the spinous processes (*star*)

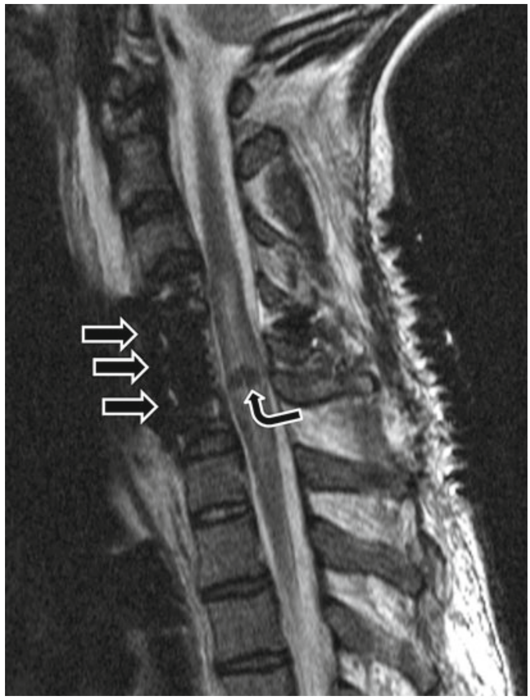


Fig. 4.22 Sagittal T2-weighted image in same patient as Fig. 4.21, following emergent reduction and C4–C6 fusion with anterior fusion plate (*white arrows*). The MRI demonstrates spinal cord hemorrhage (low-signal focus, *curved arrow*) with surrounding edema. Cord hemorrhage connotes a poor neurologic prognosis

4.2.4 Cervical Spine Trauma: Case Discussion

Case 3

A flexion teardrop (burst) fracture is an unstable fracture that results from combined flexion and compression and presents as "acute anterior cord syndrome" characterized by quadriplegia, loss of anterior column senses, and retention of posterior column senses, with greater than 80 % of patients sustaining permanent neurologic injury [49]. In this type of fracture, vertical height of the "teardrop" fragment is often less than or equal to the horizontal caliber (in contradistinction to an extension teardrop fracture fragment, usually a less severe injury). The best way to distinguish a flexion teardrop fracture from an extension-type fracture, however, is by mechanism of injury and the typical three-column involvement of the flexion teardrop injury.

Given the severity of the injury and the patient's associated neurologic abnormalities, an MRI should be performed. In this case, surgical reduction and fusion were performed emergently and the MRI was obtained postoperatively. The most concerning finding on these images is the heterogeneous signal on T2-weighted images in the spinal cord corresponding to spinal cord hemorrhage and edema (Fig. 4.22).

There are three basic patterns of MRI findings in spinal cord injury (SCI). These findings consist of spinal cord hemorrhage, spinal cord edema, and spinal cord swelling. The typical SCI is spindle shaped containing an epicenter of hemorrhage surrounded by a halo of edema with peripheral extension of cord swelling (Fig. 4.22). In acute hemorrhage following injury, deoxyhemoglobin is the most common blood product present, appearing hypointense on T2-weighted and gradient echo sequences. The detection of a sizable focus of blood in the cord (>10 mm in length on sagittal images) is associated with a complete neurologic injury [50]. The anatomic location of hemorrhage closely corresponds to the level of the neurologic deficit.

Spinal cord edema is characterized by increased signal on T2-weighted sequences. The length of edema in the spinal cord is directly proportional to the degree of neurologic deficit [50]. Edema is always found with cord hemorrhage; however, the converse is not always true. Cord edema without hemorrhage connotes a more favorable prognosis.

Spinal cord swelling is defined as a focal increased caliber of the cord. The change in caliber is greatest at the point of trauma and is often secondary to underlying edema or hemorrhage that may not be visible by imaging. Cord swelling alone does not correlate well with the severity of spinal cord injury.

4.2.5 Thoracolumbar Spine Trauma: Case Presentation and Differential Considerations

Case 4

A 45-year-old woman presents with significant back pain after MVC. She was a backseat passenger involved in a high mechanism collision wearing only a lap belt. She was not ejected. On physical exam the patient has tenderness to palpation at the thoracolumbar junction and diffuse abdominal pain. No focal neurologic deficits are elicited. Possible injuries in this patient include lumbar spine fracture, spinal ligamentous injury, intra-abdominal organ injury (including bowel injury), aortic injury, or superficial soft tissue injury such as a contusion or hematoma.

4.2.6 Thoracolumbar Spine Trauma: Imaging Decision-Making and Findings

Initial study in this patient should consist of a trauma CT abdomen/pelvis with thoracic and lumbar spine reconstructions. This imaging study allows the clinician to evaluate for intra-abdominal organ injury as well as spine injury. The CT in this patient shows a transversely oriented fracture through the vertebral body and posterior elements of L1 (Fig. 4.23). This type of injury is characterized as a Chance fracture.

4.2.7 Thoracolumbar Trauma: Case Discussion

A Chance fracture is a transversely oriented fracture through all three spinal columns. Chance fractures are hyperflexion injuries in which there is distraction of the posterior elements and impaction of the anterior components of the vertebrae. The compression component from hyperflexion is usually minor compared to the distraction component. These types of fractures were historically described in the setting of motor vehicle collisions with passengers restrained by lap belts and typically occurred from T10 to L3 [51]. Today, as lap belts have become less common in vehicles, the mechanism tends to be related to falls, where axial load is applied to a flexed thoracolumbar junction [52]. More than 50 % of patients have associated bowel injuries. Occasionally these fractures can be associated with retropulsion of fracture fragments donated from the posterior vertebral body (as is seen in Fig. 4.23 of this case). If these findings are identified, or if the patient has neurologic deficits, a lumbar spine MRI (Fig. 4.24) should follow to evaluate for inferior cord injury and nerve root compromise and to further characterize the degree of ligamentous injury. It should be noted that the hyperflexion mechanism can direct a line of force through the disk space, resulting in a severe, three-column injury to the soft tissue and ligamentous restraints of the spine, with little or no bony involvement.

4.2.8 Imaging of Nontraumatic Back Pain

4.2.8.1 Spondylodiskitis: Case Presentation and Differential Considerations

Case 5

A 25-year-old woman presents with progressive worsening of back pain over the last 3 weeks,

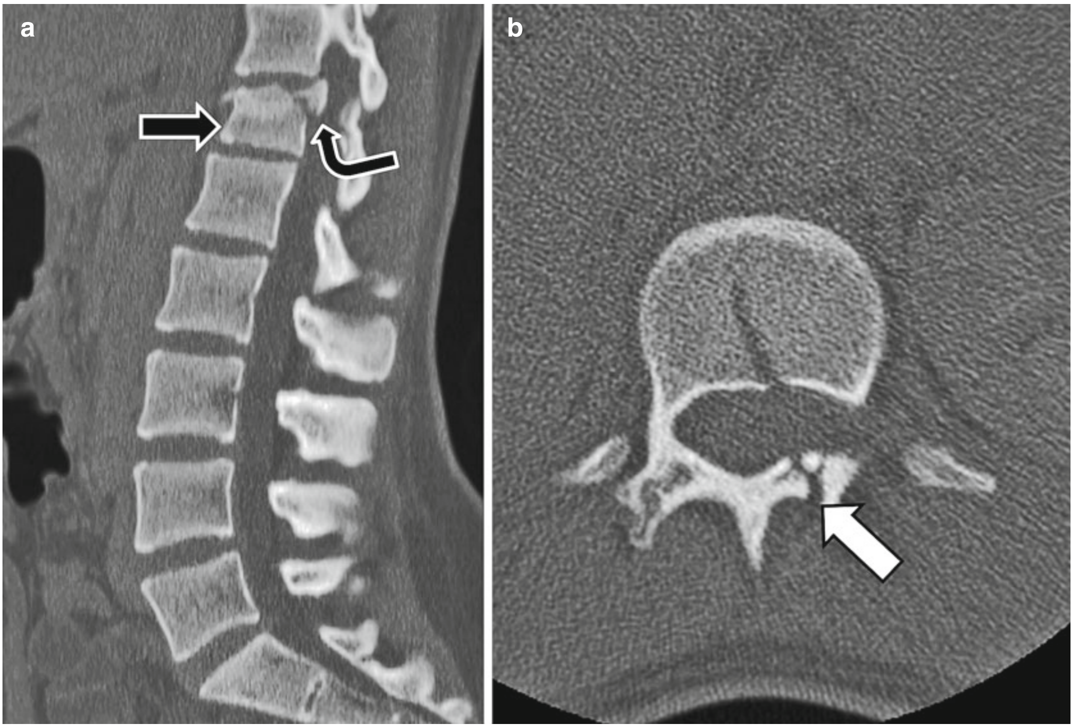


Fig. 4.23 Sagittal (**a**) and axial (**b**) CT images of a T12 Chance fracture (*white arrow*) with retropulsion of the posterior vertebral body (*curved arrow*). The fracture extends through the left lamina on the axial image (*black arrow*)

now severe. She is febrile upon clinical presentation. On questioning, the patient admits to IV drug use. The patient denies any recent trauma or history of chronic back pain. The patient also denies any radiculopathy, and the neurologic exam is intact. Differential considerations in this patient include diskitis/osteomyelitis with or without epidural abscess, pyelonephritis, pancreatitis, soft tissue abscess, muscle strain, and acute disk herniation. Urinalysis and lipase are normal. Although the patient has no fever, inflammatory markers including white blood cell count (WBC), erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) were all elevated.

4.2.9 Spondylodiskitis: Imaging Decision-Making and Findings

Given the progressive back pain, history of IV drug abuse, and elevated inflammatory markers, spondylodiskitis is high on the differential diagnosis. Starting with lumbar spine radiographs in this patient is reasonable, as there are radiographic findings of diskitis (Fig. 4.25). Figure 4.25 shows severe disk space narrowing of L2–3, with endplate destruction of the inferior endplate of L2 and superior endplate of L3. The radiographic findings of diskitis are relatively specific, as there are very few noninfectious entities that are disk centered and cause endplate destruction (aseptic diskitis associated with ankylosing spondylitis, pseudarthrosis in the fused spine, Charcot spine, and dialysis-related disk disease are some rare entities that can simulate infectious diskitis). Occasionally degenerative endplate changes can mimic infection, but consist of endplate spurring and sclerosis, and do not result in true vertebral body endplate destruction, typically easily differentiated from an infectious process by imaging. Early radiographic manifestations of spondylodiskitis are nonspecific and it can take 2–4 weeks to recognize endplate destruction radiographically [52]. In this patient an MRI was subsequently performed to evaluate for soft tissue extension of infection and epidural abscess.

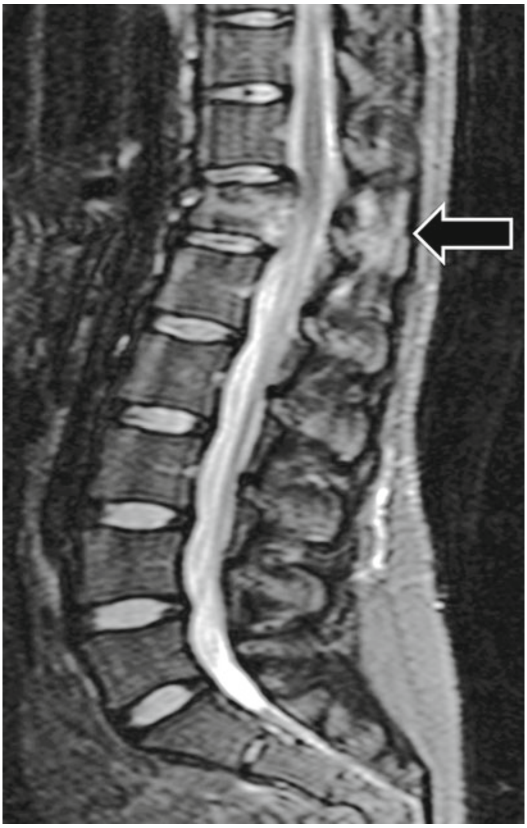


Fig. 4.24 Sagittal short-tau inversion-recovery (STIR) MR image of the T12 Chance fracture shown in Fig. 4.23. The increased T2-weighted signal in the posterior elements (*white arrow*) on the sagittal image is indicative of the distraction injury to the interspinous ligament

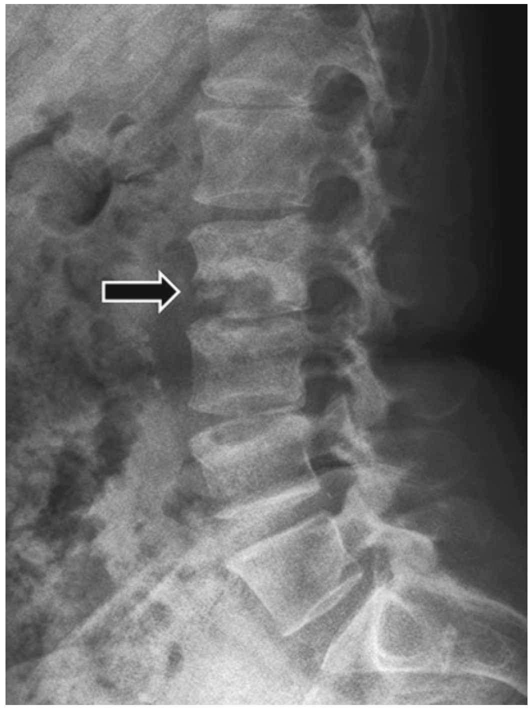


Fig. 4.25 A 25-year-old female with increasing, now severe, low back pain and fever. The L2–3 disk space demonstrates destruction of both adjacent endplates (*black arrow*). This is a very specific radiographic appearance of diskitis as very few entities other than infection cause a disk-centered destructive process

The MRI in this patient confirms a disk-centered destructive process with adjacent endplate destruction, vertebral body marrow edema, and post-contrast enhancement of the L2 and L3 vertebral bodies; MRI findings confirming the suspicion of spondylodiskitis (Fig. 4.26). In spondylodiskitis, the disk can show variable enhancement, while the vertebral endplates characteristically enhance avidly.

4.2.10 Spondylodiskitis: Case Discussion

Spondylodiskitis results from infection of the vertebral body endplates and disk resulting from either hematogenous spread (bacteremia) or iatrogenic (postoperative or postprocedural). Unlike the pediatric population, the disk is avascular in adults. Hematogenous infection involves the vascularized endplates of the vertebral bodies with subsequent and rapid spread to involve the adjacent disk. Distribution of infection within the spine reflects the blood supply, with the majority of infection involving the lumbar spine (58 %), followed by thoracic (30 %) and cervical (11 %) [53]. Lack of blood volume likely accounts for the exceptionally rare hematogenous involvement of the posterior elements.

Epidural abscess results from suppuration of the epidural space and may be due to extension of infection from adjacent tissues (spondylodiskitis, septic facet arthritis) and postoperative or direct hematogenous spread of infection to the epidural space. Predisposing factors include IV drug use, immunocompromised state, diabetes mellitus, chronic renal failure, alcoholism, cancer, or other

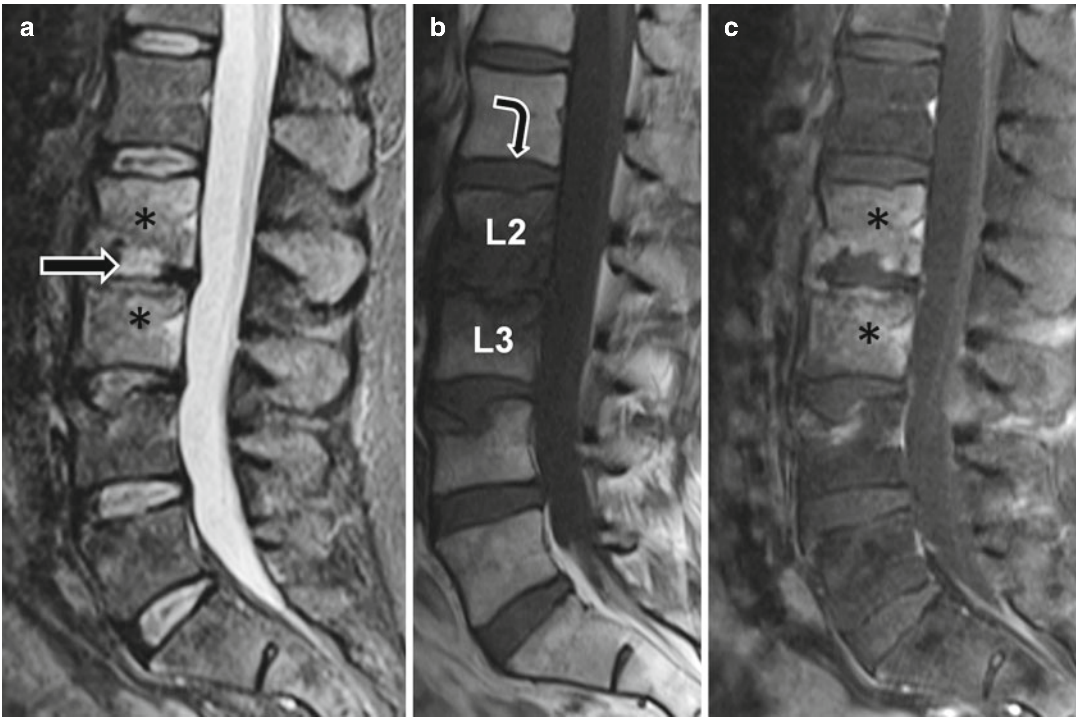


Fig. 4.26 Same patient as in Fig. 4.24 with MRI confirmation of spondylodiskitis. (**a**) Sagittal short-tau inversion-recovery (*STIR*) MR image demonstrates fluid within the disk space (*white arrow*) as well as adjacent marrow edema (*asterisk*). Sagittal T1 (**b**) and T1 fat-saturated post-contrast images (**c**) demonstrate endplate destruction surrounding the L2–3 disk (**b**). Compare to normally corticated endplate of L1 (**b**, *curved arrow*). There is marked marrow enhancement of the L2 and L3 vertebral bodies (**c**, *asterisks*). There was no epidural abscess

chronic diseases. Posterior epidural abscess is more common than anterior epidural abscess (with a prevalence of 80 % vs. 20 %, respectively) [54, 55]. A posterior epidural abscess more commonly arises from hematogenous dissemination of infection from the GU or GI tract, lungs, heart, or mucocutaneous or cutaneous sources. Posterior epidural abscesses are also more common from direct extension of infection from prior surgery or instrumentation. Anterior epidural abscesses often arise from adjacent diskitis/osteomyelitis or from direct extension of intra-abdominal infection. Of note, epidural abscess from diskitis/osteomyelitis tends to smolder, whereas epidural abscess from hematogenous spread tends to progress more rapidly [54, 55].

The most common pathogens responsible for infectious spondylodiskitis vary significantly depending on the part of the world. In the USA, pyogenic organisms are most common with *Staphylococcus aureus*, *Staphylococcus epidermidis*, *Streptococcus*, and *Enterococcus* constituting the most commonly isolated pathogens. Tuberculosis is the most common cause worldwide and brucellosis can be extremely common in some geographic areas.

In lumbar spinal epidural abscess, the patients are at risk for developing cauda equina syndrome due to both compressive effects and ischemic effects from compromised epidural venous plexuses [56]. Patients with epidural abscesses may require emergent surgical decompression with abscess drainage in addition to a prolonged course of antibiotics. Most patients without neurologic compromise can be managed medically with close observation, with prompt surgical decompression reserved for those who develop neurological deterioration [57].

4.2.11 Vertebral Compression Fracture: Case Presentation and Differential Considerations

Case 6

An 85-year-old woman presents to the urgent care clinic with significant low back pain following a fall from standing 2 days prior. She had immediate pain in her back after her fall. The patient does not have any focal neurologic signs on exam, but has focal tenderness to palpation overlying the T12 vertebral body. Most likely diagnosis in an 85-year-old osteoporotic female with acute back pain and focal tenderness following low-mechanism trauma is benign osteoporotic compression fracture. Pathologic fracture from underlying metastatic disease or myeloma should remain in the differential until excluded.

4.2.12 Vertebral Compression Fracture: Imaging Decision-Making and Findings

Initial imaging study in this patient should consist of lumbar spine radiographs to evaluate for a compression fracture, destructive osseous lesion, or degenerative disease. The lumbar spine radiographs in this patient show diffuse osteopenia with vertebral body height loss of T12 (Fig. 4.27a). The acuity of compression fractures can be

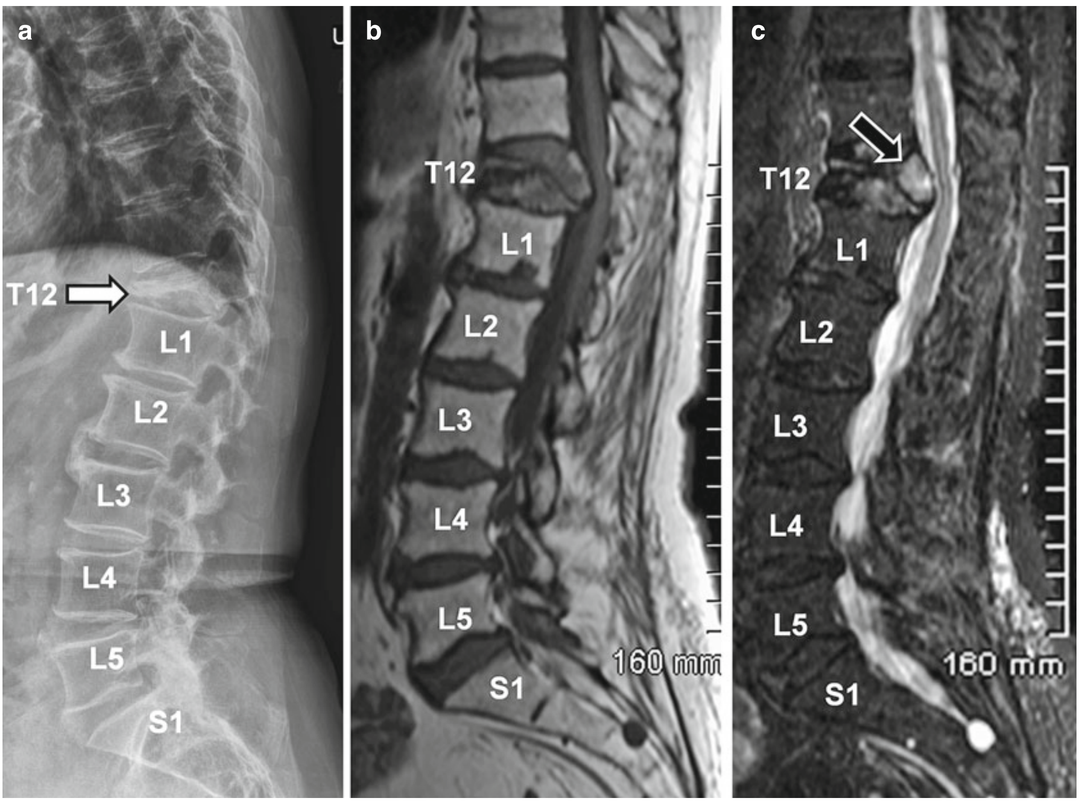


Fig. 4.27 An 85-year-old woman presents to urgent care clinic following a fall from standing 2 days prior. (**a**) Lateral radiograph of the thoracolumbar spine demonstrates severe compression deformity of T12 (*black arrow*) with resultant kyphosis surrounding the fracture. Sagittal T1 (**b**) and sagittal short-tau inversion-recovery (*STIR*, **c**) images demonstrate the severe compression of the T12 vertebral body and the high signal indicative of fracture acuity (**c**, *white arrow*). Note the central spinal canal narrowing related to dorsal cortical bowing. The degree of dorsal concavity was concerning for pathologic fracture, but given the marked degree of height loss, this was thought likely to be a simple osteoporotic compression fracture. Biopsy at the time of kyphoplasty was negative for malignancy

difficult to confirm radiographically, but given the acute onset of symptoms in this patient, the fracture is likely acute. Radiographic signs of old or chronic compression fractures (sclerosis, callous formation) are absent in this case. If there is question as to the etiology of the fracture (insufficiency fracture related to osteoporosis versus pathologic compression fracture related to metastatic disease or myeloma), further imaging should be obtained, with MRI being most useful for differentiating these entities.

An MRI of the lumbar spine was performed in this case (Fig. 4.27b, c), which showed marked compression of the central portion of the vertebral body with visible fracture clefts and surrounding edema on MRI. The dorsal cortex is convex, typically a concerning finding for pathologic fracture. The MRI in this case is more convincing for an evacuative process (osteoporosis) contributing to the fracture, rather than an infiltrative process contributing to fracture (pathologic process) as the degree of dorsal convexity is not more than would be expected for the amount of vertebral body height loss. Compare this patient's MRI to another patients with known breast cancer metastases and pathologic compression fracture of C5 (Fig. 4.28). The degree of dorsal convexity is significantly greater in the pathologic fracture compared to the benign osteoporotic fracture.

Fig. 4.28 Pathologic fracture of the C5 vertebral body in a patient with metastatic breast cancer. Note the degree of convexity dorsally relative to the height loss (*white arrow*). In addition, enhancing tissue is disrupting the dorsal cortex with slight extension craniocaudally (*thin black arrows*)

4.2.13 Vertebral Compression Fracture: Case Discussion

This case illustrates the common conundrum in distinguishing insufficiency fractures from pathologic fractures, as they can often look similar on radiographs. MRI generally can help differentiate these two entities based on morphology and signal characteristics [58–60]. MR imaging findings suggestive of metastatic compression fractures include a convex posterior border of the vertebral body, abnormal signal intensity in the pedicle or posterior elements, an epidural or paraspinal mass, and other spinal metastases [61]. On T1-weighted imaging, pathologic fractures demonstrate rounded or diffuse low-signal-intensity marrow replacement. A low-signal-intensity fracture line may or may not be visible. On T2-weighted imaging, there is typically rounded or diffuse high-signal-intensity marrow replacement, and there tends to be increased conspicuity of lesion by using fat-saturation sequences. On post-contrast T1-weighted sequences, enhancement around the fracture line and underlying lesion is usually more rounded, whereas nonpathologic fractures typically enhance in a more band-like configuration [58–60], although differences in enhancement pattern is not reliable (all bone marrow edema, whether from benign compression fracture or pathologic fracture, will enhance). Occasionally, the differentiation between osteoporotic and metastatic compression fractures cannot be determined by MR imaging, and biopsy may be necessary.

The risk of fracture from tumor depends on tumor type (e.g., lytic lesions confer higher risk than blastic), tumor volume, pedicle involvement, amount of cortical destruction, bowing of vertebral margins, and load on the vertebra.

4.2.14 Inflammatory Back Pain: Case Presentation and Differential Considerations

Case 7

An 18-year-old man presents with worsening, episodic low back pain over a period of 12 months. Pain is worse in the morning and improves throughout the day. The patient is otherwise healthy and denies a history of trauma. The patient denies any radiculopathy or systemic symptoms. Differential considerations in this patient include seronegative spondyloarthropathies, spondylolysis, disk herniation, facet arthropathy, muscular strain, or pathologic fracture.

4.2.15 Inflammatory Back Pain: Imaging Decision-Making and Findings

Chronic back pain in a young individual has a fairly narrow differential to include inflammatory spondyloarthropathies (ankylosing spondylitis, chronic reactive arthritis, and psoriatic arthritis) and spondylolysis (pars defects). This patient's history of episodic back pain, worse in the morning with improvement throughout the day, is very typical of inflammatory back pain (spondyloarthropathies). Initial imaging study in this case should consist of AP and lateral views of lumbar spine and spot lateral view of the lumbosacral spine. A good three-view radiographic evaluation of the lumbar spine allows for evaluation of spondylolysis, early disk space narrowing, and bony changes of spondyloarthropathies (sacroiliitis, early lumbar spine osteitis, syndesmophytes, etc.). Bilateral oblique views of the lumbar spine expose the patient to significant radiation; however, some authors maintain that these views are the most effective way to demonstrate pars fractures [17]. This is controversial, however, as other studies have shown the lateral to be more sensitive [62]. To lessen the radiation exposure in young patients, AP, lateral, and spot lateral views of the lumbosacral spine are sufficient for initial work-up.

Coned-down AP view of the lumbar spine demonstrates erosive changes to the sacroiliac joints bilaterally, with symmetric changes (Fig. 4.29). Differential for inflammatory sacroiliitis includes classic ankylosing spondylitis (AS), enteropathic AS (associated with inflammatory bowel disease), psoriatic spondyloarthropathy, and chronic reactive arthritis (previously known as Reiter's disease). In a young male with relatively symmetric sacroiliitis, the most likely diagnosis would be AS, which was the diagnosis in this case.

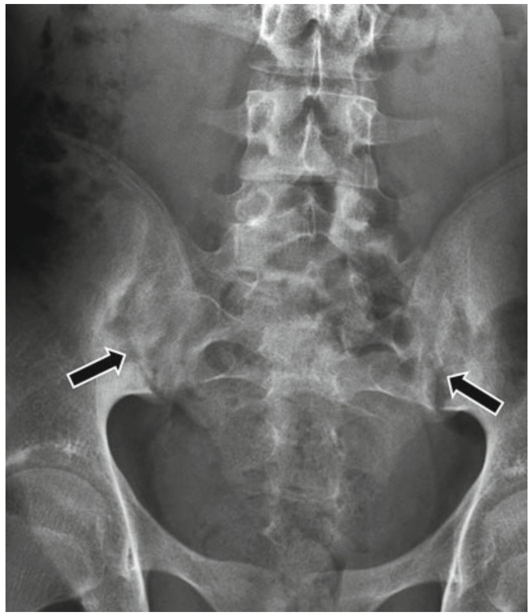


Fig. 4.29 An 18-year-old male presents with chronic, episodic low back pain. Coned-down AP view of the lumbar spine shows bilateral and symmetric erosive changes to the sacroiliac joints (*arrows*)

4.2.16 Inflammatory Back Pain: Case Discussion

Seronegative spondyloarthropathies include classic ankylosing spondylitis, chronic reactive arthritis (previously called Reiter's syndrome), psoriatic arthropathy, and enteropathic spondyloarthropathy (enteropathic ankylosing spondylitis). These arthropathies are characterized by rheumatoid factor-negative inflammatory arthritis and commonly involve the spine and sacroiliac joints.

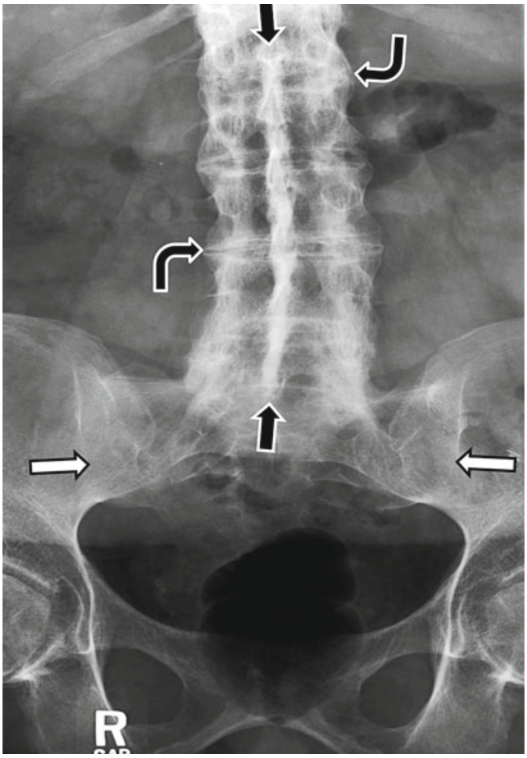


Fig. 4.30 Advanced findings of AS in a different patient. Note the bilaterally fused SI joints (*black arrows*), thin, bridging syndesmophytes at the margins of the disks resulting in characteristic "bamboo spine" appearance (*curved arrows*), and the solid interspinous ossification (*between white arrows*) known as the "dagger spine" sign

The most common imaging finding of ankylosing spondylitis is sacroiliitis. SI joint fusion is associated with long-standing episodic inflammatory flares. The spine is typically involved from the lumbosacral junction superiorly. The entire spine can eventually be involved with thin syndesmophytes spanning all disk spaces (Fig. 4.30). With reactive arthropathy and psoriatic arthritis, the syndesmophytes are typically more bulky and lateral in orientation. The SI joint involvement in AS is usually bilateral and symmetric, and this pattern can also be seen in association with inflammatory bowel disease (enteropathic ankylosing spondylitis). In contrast, the SI joint involvement with chronic reactive arthritis and psoriatic arthropathy is more commonly asymmetric but can be bilateral and symmetric or even, uncommonly, unilateral [63].

Another distinctive finding seen in ankylosing spondylitis consists of focal inflammatory osteitis of the vertebral body corner at the junction of attachment of the annulus fibrosis of the disk (Romanus lesions). These foci of enthesitis can lead to focal radiographic sclerosis, the "shiny corner" sign. This inflammation at the entheses of the disks leads to periostitis of the ventral vertebral body and subsequent infilling of periosteal new bone, resulting in "squaring" of the normal concavity of the ventral vertebral bodies. Ossification of the interspinous and supraspinous ligaments is also known as the "dagger sign" given the resemblance of the long dorsal fusion mass to a dagger (Fig. 4.30). Due to the diffuse ankylosis and rigidity of the spine in these patients, they are more susceptible to fracture. These patients may develop diffuse osteopenia later in the course of the disease, which may limit evaluation for a fracture on spine radiographs, and thus trauma to the spine in these patients may require a CT or MRI to evaluate for a subtle fracture [64].

Findings that can mimic disk infection in patients with AS include aseptic diskitis (Andersson lesion) and pseudarthrosis. Ankylosing spondylitis may also progress to involve other joints of the axial skeleton characterized by erosions progressing to fusion [65, 66].

4.2.17 Imaging of Radicular Pain

4.2.17.1 Radicular Pain Without Degenerative Spine: Case Presentation and Differential Considerations

Case 8

A 30-year-old man with history of back pain presents with new-onset excruciating radicular leg pain. Considerations of cause for the new onset of leg pain in this patient population are large, but the strongest considerations would include acute disk herniation or spondylolisthesis associated with spondylolysis, with other considerations to include synovial cyst (much more common in an older population), neoplasm,

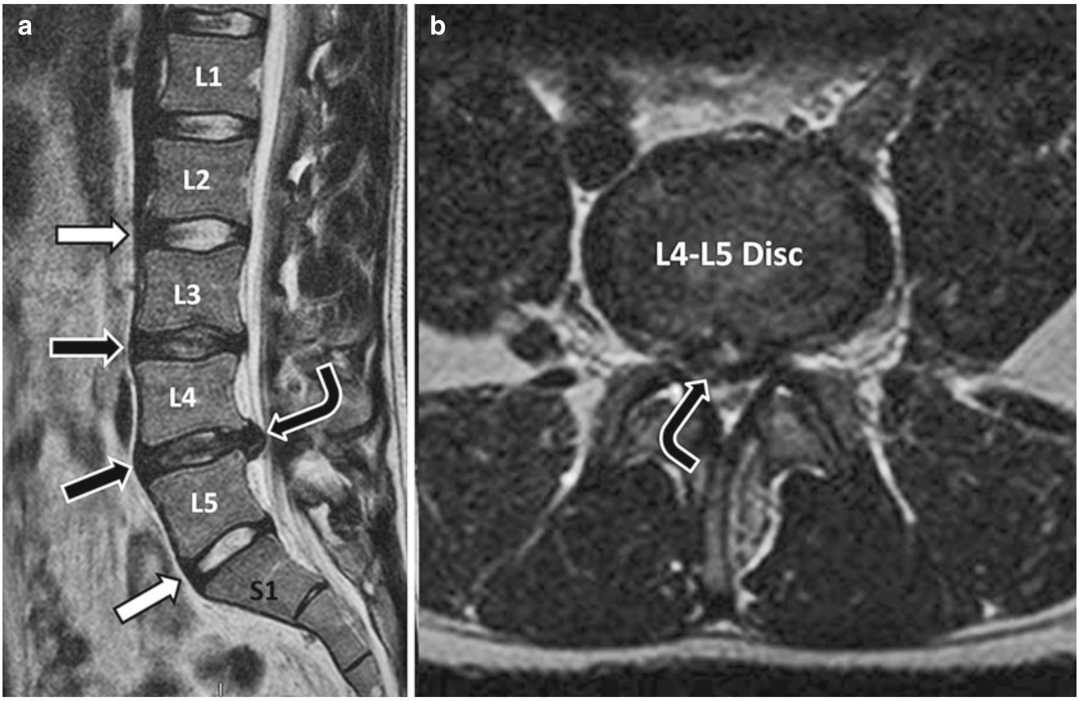


Fig. 4.31 A 30-year-old man with acute right-sided radicular pain. (**a**) Sagittal T2-weighted MRI shows disk desiccation at L3–4 and L4–5 (*white arrows*) compared to normally hydrated disks L2–3 and L5–S1 (*black arrows*). Large disk bulge is narrowing the right paracentral canal at L4–5 (*curved arrow*). (**b**) Axial T2-weighted image at the L4–5 level shows right paracentral protrusion superimposed on the broad-based disk bulge (*curved arrow*), compressing the traversing L5 nerve root and causing moderate to severe central spinal canal narrowing

spinal hematoma, or infectious etiologies being much less likely.

4.2.18 Radicular Pain Without Degenerative Spine: Imaging Decision-Making and Findings

Initial imaging study in this patient consisted of lumbar spine radiographs, which demonstrated mild L4–5 disk space narrowing (not shown). The unremitting, excruciating leg pain in this patient prompted lumbar spine MR (Fig. 4.31a, b). The disks of L4–5 and L3–4 are dark (desiccated) compared to normally bright (well-hydrated) L2–3 and L5–S1 disks. In addition, MRI shows L4–5 right paracentral disk protrusion (curved arrows) superimposed on a broad-based disk bulge. This is compressing the traversing right L5 nerve root at the L4–5 level (Fig. 4.31b).

4.2.19 Radicular Pain Without Degenerative Spine: Case Discussion

Description of disk abnormalities on MRI lumbar spine can be confusing, as many terminologies have classically been used. In 2001 a joint task force gathered to attempt to obtain consensus [67]. A broad-based disk bulge occupies greater than one quadrant of the disk circumference. A disk herniation is defined as occupying less than 90° of the disk circumference. Disk herniation can be characterized as either a protrusion or an extrusion. A protrusion is a herniated disk where the base dimension exceeds the height of the herniation, having a broad interface with the parent disk. An extrusion is defined as a herniation whose height exceeds the width of the base, having a narrow interface with the parent disk. A sequestered or free fragment describes an extruded disk without contiguity to the parent disk. Associated findings with disk herniation on MRI are tears of the posterior annulus fibrosus, also termed high-intensity zones on MRI, given the focal high T2-weighted signal. Of note, asymptomatic patients can often have annular defects or tears, occurring in up to 25 % of normal patients under the age of 20 [68].

4.2.20 Radicular Pain with Degenerative Spine: Case Presentation and Differential Considerations

Case 9

A 55-year-old woman presents with new onset severe right lower extremity radicular pain. Patient has known lumbar facet degenerative arthrosis, previously treated with medial branch blocks with good short-term relief. Chief differential considerations include disk herniation, relatively acute progression of degenerative canal or neural foraminal narrowing, or facet synovial cyst. Infectious or neoplastic processes are less likely.

4.2.21 Radicular Pain with Degenerative Spine: Imaging Decision-Making and Findings

The initial radiographs in this patient showed stable discogenic and facet degenerative changes (not shown). An MRI of the lumbar spine was deemed reasonable in this case, given the new onset of neurologic findings. The MRI shows a high T2 signal mass arising adjacent to the right L4–5 facet joint causing severe compression of the exiting right L5 nerve root (Fig. 4.32). Imaging differential for this mass includes synovial cyst emanating from the facet joint, nerve sheath tumor, and dilation of the normal nerve root sheath (nerve root sheath cyst). Given the MRI appearance, the adjacent degenerative facet, and the new onset of right-sided radicular pain, synovial cyst is far more likely than another cause. In this case a small

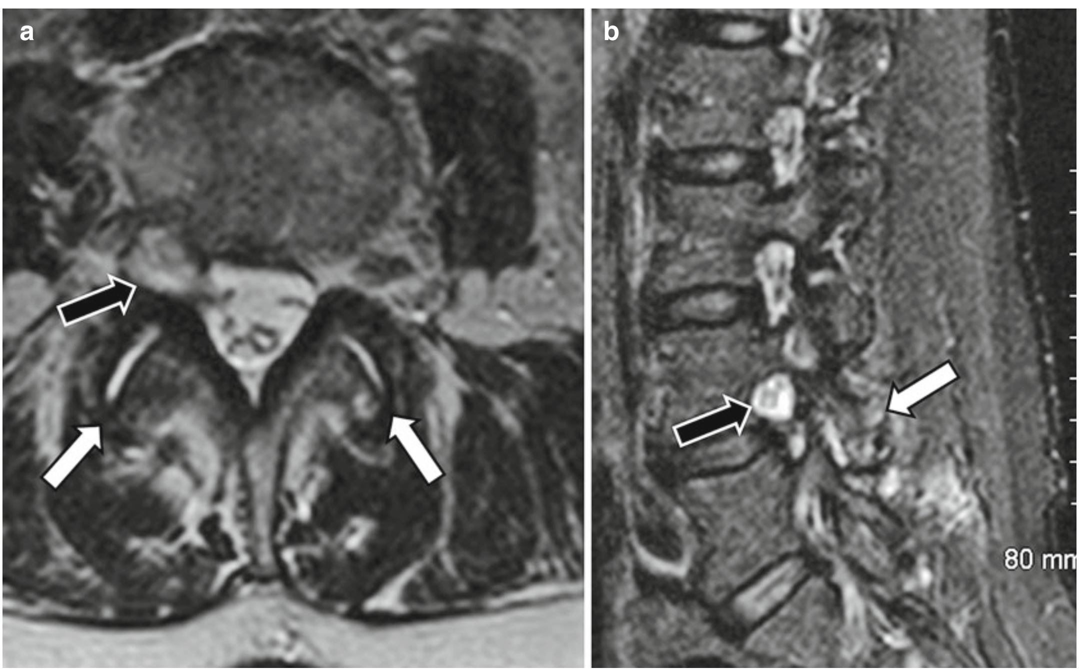


Fig. 4.32 A 55-year-old woman presenting with acute right-sided radicular. Axial T2-weighted (**a**) and sagittal short-tau inversion-recovery (*STIR*, **b**) images demonstrate large high T2 signal lesion filling the right L4–5 neural foramen (*white arrows*). Adjacent L4–5 facets are degenerative (*black arrows*). The exiting L4 nerve root is indistinguishable from this mass indicative of the severe nerve root impingement

neck could be traced back to the degenerative facet, confirming the diagnosis. If the diagnosis is unclear, contrast-enhanced MRI can be performed to exclude solid enhancing nerve sheath tumor.

4.2.22 Radicular Pain with Degenerative Spine: Case Discussion

In the setting of facet osteoarthropathy, joint fluid accumulation and synovial proliferation can lead to cyst formation. 70–80 % of synovial cysts are located at the L4–5 level and can cause mass effect on the L4–5 neuroforamen or spinal canal [69]. This can subsequently result in radicular symptoms, neurogenic claudication, acute pain from hemorrhage, or cauda equina syndrome. Synovial cysts are often T2 hyperintense and T1 hypointense reflecting the nature of the contents (usually simple synovial fluid). The MRI appearance can vary, however, and can be T1 hyperintense if there is a proteinaceous content or presence of hemorrhage. Long-standing cysts may demonstrate calcification on MRI or CT. Treatment may be conservative, as some will spontaneously regress. In the setting of significant or unimproving pain or myopathic symptoms, percutaneous cyst rupture, steroid injection into the cyst, or surgical excision can be performed. Percutaneous rupture is a minimally invasive therapy that can be definitive. Studies have shown that 50–75 % of patients avoid surgery and have long-lasting pain relief following percutaneous therapy [70, 71]. Approximately 20–30 % of percutaneously ruptured cysts will recur. There is a high postsurgical success rate in symptomatic patients as well, with or without fusion with small rates of recurrence (<10 %).

4.2.23 Imaging of the Postoperative Spine

4.2.23.1 Postoperative Spine: Case Presentation and Differential Considerations

Case 10

A 47-year-old woman presents with recurrent low back pain and right-sided radiculopathy following successful L5–S1 microdiscectomy 3 months prior. Possible causes of low back pain in this patient include recurrent disk herniation, perineural fibrosis, diskitis/osteomyelitis, epidural hemorrhage, and myofascial sprain/strain.

4.2.24 Postoperative Spine: Imaging Decision-Making and Findings

MRI of the postoperative spine necessitates knowledge of the timing of the surgery, the reason the surgery was undertaken, and the surgery performed. Post-contrast imaging should be performed if infection is suspected, if there has been partial diskectomy within the last 7 years, and for tumor follow-up. In this patient, the recent microdiscectomy will require post-contrast-enhanced MRI sequences.

Axial T2 and T1 post-contrast images show large recurrent disk extrusion (Fig. 4.33). Note that the disk material is not enhancing (distinguishing characteristic from epidural or peridural postoperative granulation tissue). Thin peripheral enhancement is demonstrated.

4.2.25 Postoperative Spine: Case Discussion

Normal findings 0–6 months after surgery include annular enhancement, disk space enhancement in 67 %, and epidural enhancement with mass effect in 80 %. Also, nerve root enhancement may be seen in 20–62 % of asymptomatic individuals, likely due to disruption of the blood–nerve barrier, but this should be resolved by 6 months after surgery [72, 73]. Paraspinal muscular edema is also common and due to early denervation changes.

Enhancement of the epidural and perineural space can be normal for 5–10 years following surgery. Mass effect from postoperative epidural fibrosis should be minimal by 6 months following surgery. Posterior paraspinal muscle denervation edema and atrophy are common below the level of surgery. Large volume peridural scar following surgery is associated with a poor clinical outcome. Nerve

Fig. 4.33 A 47-year-old female 9 months following L5-S1 microdiscectomy with recurrent right-sided radicular pain. Sagittal STIR **(a)** and axial T2-weighted **(b)** images show a recurrent disk herniation (*white arrows*), confirmed as disk by lack of enhancement on post contrast image (**c**, *black arrow*)

root enhancement should be considered abnormal in the late postoperative period (after 6 months).

Recurrent or residual disk herniation is distinguished from the typical postoperative findings by the lack of enhancement of the herniated disk material. Imaging soon after IV contrast administration is important as peak enhancement of postoperative granulation tissue is approximately 5 min following administration. Disk material can show some mild peripheral enhancement but should not enhance uniformly. Rarely disk material will enhance diffusely if the imaging is delayed too long after contrast is administered.

References

1. Stiell IG et al (2001) The Canadian C-spine rule for radiography in alert and stable trauma patients. JAMA 286(15):1841–1848
2. Hendey GW et al (2002) Spinal cord injury without radiographic abnormality: results of the National Emergency X-Radiography Utilization Study in blunt cervical trauma. J Trauma 53(1):1–4
3. Stiell IG et al (2003) The Canadian C-spine rule versus the NEXUS low-risk criteria in patients with trauma. N Engl J Med 349(26):2510–2518
4. Green JD, Harle TS, Harris JH Jr (1981) Anterior subluxation of the cervical spine: hyperflexion sprain. AJNR Am J Neuroradiol 2(3):243–250
5. Swischuk LE (1977) Anterior displacement of C2 in children: physiologic or pathologic. Radiology 122(3): 759–763

6. Harris JH Jr, Carson GC, Wagner LK (1994) Radiologic diagnosis of traumatic occipitovertebral dissociation: 1. Normal occipitovertebral relationships on lateral radiographs of supine subjects. AJR Am J Roentgenol 162(4):881–886
7. Harris JH Jr et al (1994) Radiologic diagnosis of traumatic occipitovertebral dissociation: 2. Comparison of three methods of detecting occipitovertebral relationships on lateral radiographs of supine subjects. AJR Am J Roentgenol 162(4):887–892
8. Hinck VC, Hopkins CE (1960) Measurement of the atlanto-dental interval in the adult. Am J Roentgenol Radium Ther Nucl Med 84:945–951
9. Rojas CA et al (2009) Normal thickness and appearance of the prevertebral soft tissues on multidetector CT. AJNR Am J Neuroradiol 30(1):136–141
10. Yetkin Z et al (1985) Uncovertebral and facet joint dislocations in cervical articular pillar fractures: CT evaluation. AJNR Am J Neuroradiol 6(4):633–637
11. Deliganis AV et al (2000) Radiologic spectrum of craniocervical distraction injuries. Radiographics 20 Spec No:S237–S250
12. Herkowitz HN, Rothman RH (1984) Subacute instability of the cervical spine. Spine 9(4):348–357
13. Denis F (1983) The three column spine and its significance in the classification of acute thoracolumbar spinal injuries. Spine 8(8):817–831
14. McAfee PC et al (1983) The value of computed tomography in thoracolumbar fractures. An analysis of one hundred consecutive cases and a new classification. J Bone Joint Surg Am 65(4):461–473
15. Castellvi AE, Goldstein LA, Chan DP (1984) Lumbosacral transitional vertebrae and their relationship with lumbar extradural defects. Spine (Phila Pa 1976) 9(5):493–495
16. Gehweiler JA Jr, Daffner RH (1983) Low back pain: the controversy of radiologic evaluation. AJR Am J Roentgenol 140(1):109–112
17. Libson E et al (1984) Oblique lumbar spine radiographs: importance in young patients. Radiology 151(1):89–90
18. IMV (2006) CT market summary report, IMV Medical Information Division. Des Plains
19. Amis ES Jr et al (2007) American College of Radiology white paper on radiation dose in medicine. J Am Coll Radiol 4(5):272–284
20. Blackmore CC et al (1999) Cervical spine screening with CT in trauma patients: a cost-effectiveness analysis. Radiology 212(1):117–125
21. Nunez DB Jr et al (1996) Cervical spine trauma: how much more do we learn by routinely using helical CT? Radiographics 16(6):1307–1318; discussion 1318–1321
22. Diaz JJ Jr et al (2003) Are five-view plain films of the cervical spine unreliable? A prospective evaluation in blunt trauma patients with altered mental status. J Trauma 55(4):658–663; discussion 663–634
23. Holmes JF, Akkinepalli R (2005) Computed tomography versus plain radiography to screen for cervical spine injury: a meta-analysis. J Trauma 58(5): 902–905
24. Mathen R et al (2007) Prospective evaluation of multislice computed tomography versus plain radiographic cervical spine clearance in trauma patients. J Trauma 62(6):1427–1431
25. McCulloch PT et al (2005) Helical computed tomography alone compared with plain radiographs with adjunct computed tomography to evaluate the cervical spine after high-energy trauma. J Bone Joint Surg Am 87(11):2388–2394
26. Daffner RH, Hackney DB (2007) ACR appropriateness criteria on suspected spine trauma. J Am Coll Radiol 4(11):762–775
27. Douglas-Akinwande AC et al (2006) Multichannel CT: evaluating the spine in postoperative patients with orthopedic hardware. Radiographics 26(Suppl 1):S97–S110
28. Rutherford EE et al (2007) Lumbar spine fusion and stabilization: hardware, techniques, and imaging appearances. Radiographics 27(6):1737–1749
29. Andrews CL (2000) Evaluation of the postoperative spine: spinal instrumentation and fusion. Semin Musculoskelet Radiol 4(3):259–279
30. Jarvik JG et al (2000) Variation in the quality of lumbar spine MR images in Washington State. Radiology 215(2):483–490
31. Carrino JA et al (2009) Lumbar spine: reliability of MR imaging findings. Radiology 250(1):161–170
32. Silberstein M, Tress BM, Hennessy O (1992) A comparison between M.R.I. and C.T. in acute spinal trauma. Australas Radiol 36(3):192–197
33. Modic MT et al (1985) Vertebral osteomyelitis: assessment using MR. Radiology 157(1):157–166
34. Cramer BC, Jequier S, O'Gorman AM (1986) Sonography of the neonatal craniocervical junction. AJR Am J Roentgenol 147(1):133–139
35. Zieger M, Dorr U, Schulz RD (1988) Pediatric spinal sonography. Part II: malformations and mass lesions. Pediatr Radiol 18(2):105–111
36. Zieger M, Dorr U (1988) Pediatric spinal sonography. Part I: anatomy and examination technique. Pediatr Radiol 18(1):9–13
37. Schauwecker DS (1992) The scintigraphic diagnosis of osteomyelitis. AJR Am J Roentgenol 158(1):9–18
38. Buscombe JR et al (1993) Clinical high resolution skeletal single photon emission tomography using a triple-headed gamma camera. Br J Radiol 66(789):817–822
39. Meyer M et al (1994) Increased F-18 FDG accumulation in an acute fracture. Clin Nucl Med 19(1):13–14
40. Palestro CJ et al (2006) Combined labeled leukocyte and technetium 99m sulfur colloid bone marrow imaging for diagnosing musculoskeletal infection. Radiographics 26(3):859–870
41. Kanis JA (1994) Assessment of fracture risk and its application to screening for postmenopausal osteoporosis: synopsis of a WHO report. WHO Study Group. Osteoporos Int 4(6):368–381

42. Heffernan DS, Schermer CR, Lu SW (2005) What defines a distracting injury in cervical spine assessment? J Trauma 59(6):1396–1399
43. Canadian C (2002) Spine rule study for alert and stable trauma patients: I. Background and rationale. CJEM 4(2):84–90
44. Saltzherr TP et al (2009) Diagnostic imaging of cervical spine injuries following blunt trauma: a review of the literature and practical guideline. Injury 40(8):795–800
45. Platzer P et al (2006) Clearing the cervical spine in critically injured patients: a comprehensive C-spine protocol to avoid unnecessary delays in diagnosis. Eur Spine J 15(12):1801–1810
46. Hanson JA et al (2000) Cervical spine injury: a clinical decision rule to identify high-risk patients for helical CT screening. AJR Am J Roentgenol 174(3): 713–717
47. Blackmore CC, Mann FA, Wilson AJ (2000) Helical CT in the primary trauma evaluation of the cervical spine: an evidence-based approach. Skeletal Radiol 29(11):632–639
48. Holmes JF et al (2002) Variability in computed tomography and magnetic resonance imaging in patients with cervical spine injuries. J Trauma 53(3):524–529; discussion 530
49. Kim KS et al (1989) Flexion teardrop fracture of the cervical spine: radiographic characteristics. AJR Am J Roentgenol 152(2):319–326
50. Ramon S et al (1997) Clinical and magnetic resonance imaging correlation in acute spinal cord injury. Spinal Cord 35(10):664–673
51. Marsh HO, Bailey D (1970) Seat belt fractures. Chance fractures caused by seat belts: presentation of three cases. J Kans Med Soc 71(9):361–365
52. Manaster BJ, May DA, Disler DG (2007) Musculoskeletal imaging. In: Thrall J (ed) The requisites, 3rd edn. Mosby Elsevier Inc., Philadelphia
53. Mylona E et al (2009) Pyogenic vertebral osteomyelitis: a systematic review of clinical characteristics. Semin Arthritis Rheum 39(1):10–17
54. Varma R, Lander P, Assaf A (2001) Imaging of pyogenic infectious spondylodiskitis. Radiol Clin North Am 39(2):203–213
55. Reihsaus E, Waldbaur H, Seeling W (2000) Spinal epidural abscess: a meta-analysis of 915 patients. Neurosurg Rev 23(4):175–204; discussion 205
56. Eastwood JD, Vollmer RT, Provenzale JM (2002) Diffusion-weighted imaging in a patient with vertebral and epidural abscesses. AJNR Am J Neuroradiol 23(3):496–498
57. Siddiq F et al (2004) Medical vs surgical management of spinal epidural abscess. Arch Intern Med 164(22): 2409–2412
58. Moulopoulos LA et al (1996) MR prediction of benign and malignant vertebral compression fractures. J Magn Reson Imaging 6(4):667–674
59. An HS et al (1995) Can we distinguish between benign versus malignant compression fractures of the spine by magnetic resonance imaging? Spine (Phila Pa 1976) 20(16):1776–1782
60. Shih TT, Huang KM, Li YW (1999) Solitary vertebral collapse: distinction between benign and malignant causes using MR patterns. J Magn Reson Imaging 9(5):635–642
61. Uetani M, Hashmi R, Hayashi K (2004) Malignant and benign compression fractures: differentiation and diagnostic pitfalls on MRI. Clin Radiol 59(2): 124–131
62. Amato M, Totty WG, Gilula LA (1984) Spondylolysis of the lumbar spine: demonstration of defects and laminal fragmentation. Radiology 153(3):627–629
63. Deesomchok U, Tumrasvin T (1993) Clinical comparison of patients with ankylosing spondylitis, Reiter's syndrome and psoriatic arthritis. J Med Assoc Thai 76(2):61–70
64. Mitra D et al (2000) The prevalence of vertebral fractures in mild ankylosing spondylitis and their relationship to bone mineral density. Rheumatology (Oxford) 39(1):85–89
65. Luong AA, Salonen DC (2000) Imaging of the seronegative spondyloarthropathies. Curr Rheumatol Rep 2(4):288–296
66. Braun J, Bollow M, Sieper J (1998) Radiologic diagnosis and pathology of the spondyloarthropathies. Rheum Dis Clin North Am 24(4):697–735
67. Fardon DF, Milette PC (2001) Nomenclature and classificationoflumbardiscpathology.Recommendations of the Combined task Forces of the North American Spine Society, American Society of Spine Radiology, and American Society of Neuroradiology. Spine (Phila Pa 1976) 26(5):E93–E113
68. Munter FM et al (2002) Serial MR imaging of annular tears in lumbar intervertebral disks. AJNR Am J Neuroradiol 23(7):1105–1109
69. Apostolaki E et al (2000) MR imaging of lumbar facet joint synovial cysts. Eur Radiol 10(4):615–623
70. Allen TL, Tatli Y, Lutz GE (2009) Fluoroscopic percutaneous lumbar zygapophyseal joint cyst rupture: a clinical outcome study. Spine J 9(5):387–395
71. Martha JF et al (2009) Outcome of percutaneous rupture of lumbar synovial cysts: a case series of 101 patients. Spine J 9(11):899–904
72. Babar S, Saifuddin A (2002) MRI of the post-discectomy lumbar spine. Clin Radiol 57(11):969–981
73. Van Goethem JW, Parizel PM, Jinkins JR (2002) Review article: MRI of the postoperative lumbar spine. Neuroradiology 44(9):723–739

Diagnostic Evaluations

5

Richard Kendall and Zach Beresford

5.1 Introduction

Neck and lower back pain are two of the most common problems encountered by all physicians, regardless of specialty. While a majority of spinal pain is self-limiting, a significant proportion of individuals have continuous pain and disability. Due to the high prevalence of abnormal imaging findings in asymptomatic patients [31, 32], further diagnostic instruments are necessary to focus effective treatment strategies. This chapter will discuss available interventional diagnostic/therapeutic procedures and physiological studies that are used as an extension of the physical examination and imaging modalities described in Chaps. 2, 3 and 4.

5.2 Diagnostic Evaluation: Axial Pain

Chronic axial spine pain is a common problem and can be difficult to treat. There are multiple etiologies of axial low back pain. Common sources of axial spine pain include the intervertebral disc, sacroiliac joint, and zygapophyseal joint. A diagnostic algorithm can be undertaken to investigate this pain as is presented in Fig. 5.1. These diagnostic approaches may be indicated in patients with persistent debilitating axial pain. A thorough history and physical examination must be performed before considering any of these procedures. Also, the patient must be reliable and able to recognize their usual pain and distinguish this pain from other pain, or these procedures have little to no diagnostic value.

R. Kendall, DO (✉) • Z. Beresford, M.D.
Division of PM & R, University of Utah
Orthopaedics Center,
590 Wakara Way, SLC, UT 84108, USA
e-mail: richard.kendall@hsc.utah.edu;
zach.beresford@hsc.utah.edu

5.2.1 Lumbar Discogenic Pain

The intervertebral disc has been thought of as a common cause of back pain since Mixter and Barr's description of disc pathology in 1934 [42]. Intervertebral disc pathology can induce axial pain, as well as radicular symptoms from nerve root compression. This pain may be generated by the annulus as there are abundant nerve endings in the outer one-third of the annulus fibrosis, and free nerve endings can grow into the nucleus pulposus when internal disc disruption is present [15]. Internal disc disruption, however, is difficult to determine based only on history or physical examination [50, 52]. Even advanced imaging of the lumbar spine with CT, myelography, or MRI often does not provide a clear answer on the primary pain generator. There is MRI evidence of disc degeneration in greater than 50 % of asymptomatic individuals over the age of 60 (with 36 % having a disc herniation), and 20 % of asymptomatic individuals under the age of 60 had MRI evidence of disc herniation [2]. Annular tears

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_5, © Springer-Verlag Berlin Heidelberg 2014

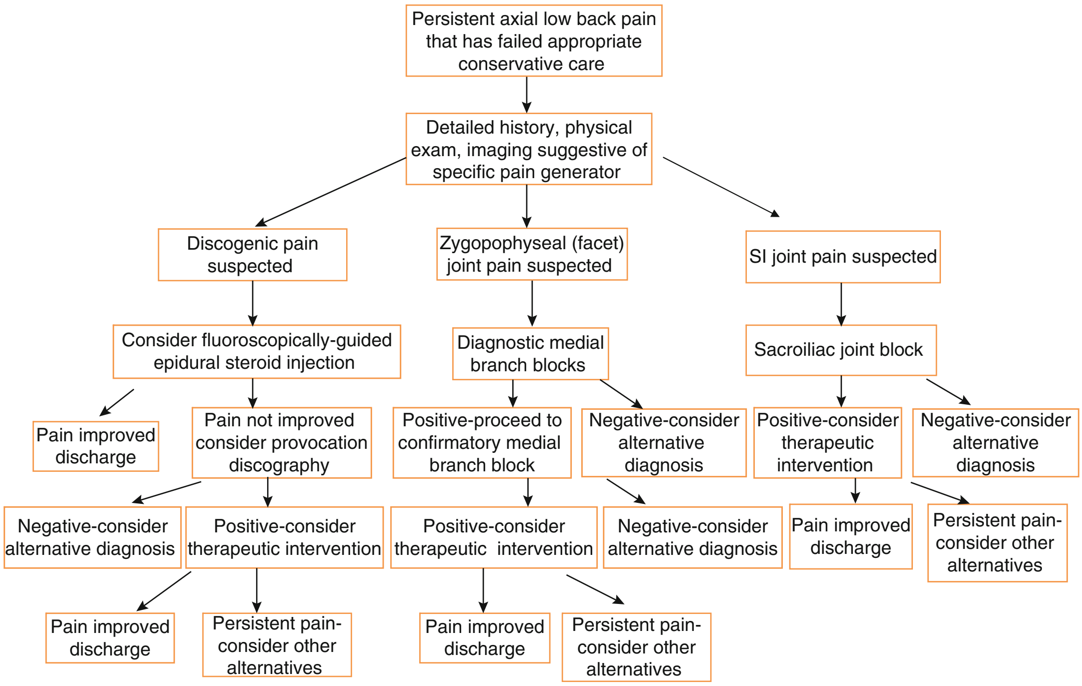


Fig. 5.1 Diagnostic algorithm for axial back pain

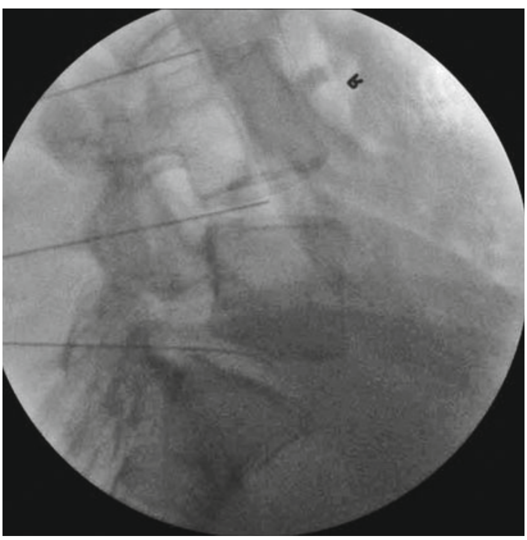


Fig. 5.2 Lateral discogram view prior to injection of contrast

have also been shown to be associated with low back pain but are commonly seen in asymptomatic individuals. Thus, advanced imaging can help to narrow down the source of pain, but it still lacks specificity.

For patients with persistent debilitating axial pain and evidence of disc degeneration on imaging, provocation discography and postdiscography CT can be considered to identify a painful intervertebral disc when treatment is to be targeted at the disc. The North American Spine Society came out with a position statement in 1995 that recommends discography for patients with persistent back pain in whom disc abnormalities are suspected, to assess the discs in patients in whom fusion is being considered, and in postsurgical patients with continued pain [23].

Discography was developed in the late 1940s as a method to diagnose intervertebral disc herniation [39]. It involves precise injection of contrast dye into the nucleus pulposus to increase intradiscal pressure causing a mechanical stimulation of annular free nerve endings in diseased, painful discs [45].

The procedure requires stimulation of discs at adjacent levels as controls (Fig. 5.2). To confirm a diagnosis of discogenic pain, the patient should be blinded to which disc level is being injected and provocation of the target disc should produce concordant, or similar, pain. Provocation of the

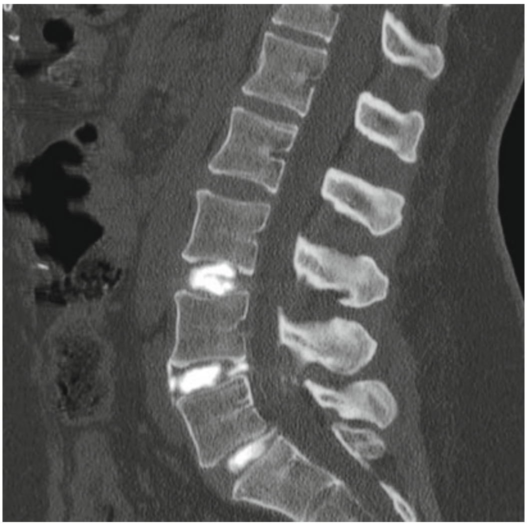


Fig. 5.3 Postdiscogram CT showing annular tears at L4–L5 and L5–S1 and normal disc morphology at L3–L4

adjacent control discs should not reproduce concordant, severe pain. Manometry is used to measure the pressure applied to each disc during the procedure to allow for more controlled testing. The diagnosis of discogenic pain is more likely with reproduction of pain at a pressure that does not produce pain in normal discs. Opening pressure, pressure at onset of pain, and maximum pressure are recorded. In general, discs that are painful at less than 15 psi are thought of as chemically sensitive, and discs that are painful between 15 and 50 psi are defined as mechanically sensitive [3, 56]. Disc pressures above 50 psi are avoided as this can routinely be painful in normal discs.

Computed tomography (CT) is performed immediately following discography to confirm contrast injection into the nucleus pulposus (Fig. 5.3). The degree of disc degeneration and abnormal morphology is measured using the Dallas Discogram Scale [48, 56].

Despite taking the above measures, false-positives exist with provocation discography. In 2006, Carragee et al. followed 32 patients over 5 years who initially presented with low back pain, had a positive single-level discogram, and underwent spinal fusion. The best-case positive predictive value of discography was calculated to be 50–60 %. A high number of asymptomatic patients without chronic low back pain (25 %) had false-positive low-pressure disc injections. Positive injections were correlated with annular disruptions, abnormal psychometric findings, and chronic pain states [6].

Risks of discography include infection and accelerated progression of disc degeneration. The intervertebral disc is avascular, increasing infection risk with injection. Discitis has been reported in up to 1 % of individuals undergoing discography; however, use of prophylactic antibiotics and the double-needle technique can help reduce this risk [3]. Discography may also increase the risk of disc degeneration and disc herniation, even with the use of small gauge needles and limited pressurization. This may limit usefulness of testing normal adjacent levels [6]. Thus, discography is an additional tool to help the surgeon narrow down and possibly identify the source of pain.

5.2.2 Facet Joint Pain

The zygapophyseal joint, or facet joint, is also a common source of axial spine pain. The facet joint was first recognized as a possible source of back pain in 1911 [20]. The facet joint capsule has been found to be richly innervated by the medial branches of the primary dorsal ramus [1, 7, 17, 18]. Common symptoms of facet joint pain include low back pain without radicular referral that is worse with standing, lumbar extension, and axial rotation and improved with sitting and/or lumbar flexion. However, diagnosis of facet-mediated pain is difficult to firmly establish based on history and physical examination [28, 30, 51]. Imaging findings on X-ray, CT, and MRI also are not clearly predictive of facet joint pain [21].

Response to either intra-articular facet injections or medial branch blocks with anesthetic is the best method to provide a specific anatomic diagnosis of facet joint pain [11, 14]. Once a joint is determined to be a source of pain, other treatment modalities can be used to target the facet joint including corticosteroid injection or medial branch radiofrequency.

Medial branch blocks involve injection of a small amount of local anesthetic in the region of the medial branch of the primary dorsal ramus with the use of image guidance. The medial branch is the sensory innervation of the facet joint. In the lumbar spine, the facet joint is innervated by the medial branch at that level and the level above. For example, the right L4–5 facet joint is innervated by the right L3 and L4 medial branches. In the cervical spine, the facet joints are innervated by the medial branches at the same level (i.e., the left C3–4 facet joint is innervated by the left C3 and C4 medial branches of the primary dorsal ramus).

After injection, patients are instructed to record their symptoms in a pain log. A positive response is 80 % or greater pain relief that lasts the duration of the local anesthetic used for the injection. A double-block paradigm using local anesthetics of different durations has been advocated to avoid false-positive diagnostic injections which may be as high as 40 % [41].

Another diagnostic modality used in recent years to identify facet-mediated pain is bone scan with SPECT, also called a "Fire" scan. This involves digital fusion or overlay of a CT scan of the area of interest with a bone scan with SPECT imaging. The fire scan provides the anatomic resolution of the CT scan plus the sensitivity of the bone scan for identifying areas of increased bone turnover [46, 58].

5.2.3 Sacroiliac Joint Pain

The sacroiliac (SI) joint was first described as a possible pain generator in 1905 and accounts for approximately 15–30 % of chronic low back pain [19, 50] and 40 % of postfusion patients [8]. Sacroiliac joint pain often presents as low back and buttock pain and can refer into the lower extremity. History, physical exam, and imaging are all unreliable in consistently diagnosing SI joint pain [13].

Diagnosis of SI joint pain also cannot be diagnosed with nerve blocks [12]. The innervation of the SI joint involves multiple levels of the lumbosacral spine, and the exact pattern of innervation is not established [22, 29].

Controlled diagnostic SI joint blocks are the best method to determine if the SI joint is a source of pain. These involve injection of a local anesthetic into the SI joint in a patient suspected of having SI joint pain. Image guidance is required, as blind injections have been found to only be 22 % accurate [47] (Fig. 5.4). A positive response requires relief of pain with the injection. Further intervention such as steroid injection may be pursued with a positive response to SI joint block.

Fig. 5.4 Sacroiliac joint injection

5.3 Diagnostic Evaluation: Radicular Pain

Before going further, one must define terms that are used interchangeably in discussing pain in the limb. Referred pain is that which originates in one anatomic structure but is perceived in another. For example, pain from the sacroiliac joint may refer posteriorly down the leg to the knee. Radicular pain is a continuous pain from the cervical or lumbar spine down the limb to the hand or foot in the distribution of an affected nerve root. Most commonly this is caused by a herniated nucleus pulposus; however, there are many etiologies that require further diagnostic evaluation than imaging. In the following sections, we will discuss the common etiologies of radicular pain and the diagnostic workup for each.

Table 5.1 Etiologies of radiculopathy

Structural causes	Metabolic causes	Infectious causes	Other
Disc herniation	Diabetes	Lyme disease	Chemotherapy
Facet osteoarthritis	Inflammatory arthritis	Tuberculosis	Radiation plexopathy
Synovial cysts	Vasculitis	Discitis	
Piriformis syndrome		Epidural abscess	
Thoracic outlet			
Tumor mass			

5.3.1 Radiculopathy

Radiculopathy is a dysfunction of a nerve root that causes symptoms of pain, numbness, or paresthesias in a dermatomal distribution, with or without weakness in that myotomal distribution. The most common cause of cervical radiculopathy is osteophytic spurring in the facet and uncovertebral joint causing bony compression of the exiting nerve root. In the lumbar spine, herniation of the nucleus pulposus is by far the most common cause. However, due to normal degenerative changes in the discs and vertebral segments in many individuals, imaging alone is often insufficient to localize the pain generator. In addition to structural causes, radiculopathy may be a result of chemical, infectious, and toxic/metabolic causes (Table 5.1).

Electrodiagnostic studies (EMG) are complementary studies to imaging modalities as they examine the physiologic component of pain via nerve function. The EMG cannot only determine the location of nerve dysfunction but also whether it is axonal or demyelinating. This is important in patients that potentially have overlapping conditions, such as diabetic neuropathy and lumbar spinal stenosis. Additionally, concomitant musculoskeletal problems are common in subjects with spine disorders [5, 55]. An EMG is composed of two parts, nerve conduction studies (NCS) and needle electrode examination (NEE). Each component is able to look at different portions of the nerve to help in localization of the pathology. EMG is most specific if performed by a physician, usually a physiatrist or neurologist, with board certification training in electrodiagnostic consultation. Otherwise, potential confounding diagnoses, such as polyneuropathy, focal peripheral neuropathy, and radiculopathy, are underdiagnosed by podiatrists, physical therapists, chiropractors, and family practitioners who may perform these studies [10, 54]. EMG has shown good correlation with computed tomography, myelography, magnetic resonance imaging, and surgical outcomes with a sensitivity of 80–100 % and good inter-rater reliability between experienced examiners for identifying a lumbar radiculopathy on needle examination [27, 34, 35, 43].

In a patient with unilateral limb pain or paresthesias, a minimum study should include at least one motor and sensory nerve conduction, plus at least five muscles studied on needle examination to investigate a potential radiculopathy. In the upper limb, other concomitant diagnoses include carpal tunnel syndrome, ulnar neuropathy at the elbow, and brachial plexus injuries. An electrodiagnostic consultation will substantially change the clinical diagnosis in 42 % of cases of upper limb pain patients, with confirmation in 37 % [26]. Since the needle electrode examination only investigates the anterior root of the spinal nerve, it is entirely possible that a compressive lesion of the dorsal root would result in a negative study. Therefore, an EMG should be used to confirm the presence of a radiculopathy and exclude other disorders, rather than to "rule out" disease [57].

In cases of suspected radiculopathy where history, physical examination, imaging, and electrodiagnostic testing are inconclusive, the use of diagnostic spinal injections may be undertaken [40]. Spinal injections of anesthetic and/or steroid injected in to the epidural space via a transforaminal approach, an extraforaminal approach, and intralaminar approach all have been correlated with predicting surgical response to radiculopathy from a herniated disc. Young has reported a sensitivity and specificity of 65–100 % and 71–95 %, respectively, for predicting surgical outcome from radiculopathy [60], while others have reported 87–100 % and 99–100 %, respectively [36, 38, 49]. Injection of a small amount of local anesthetic, between 0.5 and 0.75 ml of 2 % lidocaine [44] or 1 ml of 1 % lidocaine [36, 38, 49] around the nerve root in the cervical or lumbar foramen without epidural spread on fluoroscopy, has also been recommended. In one series, subjects with >90 % reduction in VAS after selective nerve root block had a 91 % successful result at surgery for arm/leg pain, whereas in those with <90 % reduction in VAS after selective nerve root block, only 60 % had successful outcomes after surgery [44]. Derby et al. looked at the addition of steroid to the epidural injection and its predictive effects. In subjects with pain less than 1 year in duration, 89 % of subjects had a successful outcome regardless of outcome on the epidural injection. In subjects with pain for >1 year, however, a positive response from the epidural injection resulted in 85 % successful surgical outcome, while in those with a negative epidural response, 95 % had a poor surgical outcome [9]. Caution in interpretation of the result from a reported epidural injection is needed however. The selectiveness of a single nerve root injection has been questioned, due to the volume of medication injected. A transforaminal injection will spread to adjacent nerve roots in 30 % of injections with 0.5 ml of volume, 67 % with 1.0 mls, 87 % with 1.5 ml, and >90 % with 2.5 ml [16]. Unfortunately, the variability of injectate volumes is high with epidural injections, with as little as 0.5 ml to as much as 8 ml of anesthetic injected via the transforaminal route. As such, it would be necessary to know the practice of local interventionalist physicians to determine the selectivity of the injection the patient reports.

5.3.2 Lumbar Stenosis

Lumbar stenosis is a common term that is used to describe both the anatomic dimensions of the spinal canal and a common syndrome of back and leg pain in older adults. An individual with lumbar stenosis may present with a number of clinical scenarios including axial pain alone, axial and unilateral or bilateral radicular pain, bilateral or unilateral radicular pain only, and neurogenic claudication. The anatomic disorder of canal stenosis is a common finding on magnetic resonance scans of older individuals [2, 32]. Care must be taken to correlate clinical findings with imaging findings prior to the initiation of surgical treatment for spinal stenosis [2]. Electrodiagnostic testing, especially paraspinal mapping, has been shown to be useful in delineating clinical spinal stenosis from mechanical back pain and control subjects [24, 59]. While limb EMG can be abnormal in subjects presenting with unilateral or bilateral radiculopathy, abnormal paraspinal mapping scores greater than four have a sensitivity of only 30 % but 100 % specificity in identifying clinically relevant spinal stenosis [25]. A paraspinal mapping score of greater than nine however has sensitivity and specificity of 97 and 92 %, respectively [59]. One caveat to note however is this is in a highly selected population of individuals with more mild to moderate disease and those without other potential neurological conditions, such as polyneuropathy. It is unknown what effect this has on prediction of surgical outcome for spinal stenosis symptoms as no prior studies have investigated this.

Epidural injections for treatment of pain from spinal stenosis have been advocated and shown some short-term to medium-term success [4, 37, 53]; they have not been evaluated to predict outcome from surgical decompression. There is only one prospective study to look at predictive factors of surgical outcome in lumbar spinal stenosis, which found that a patient's own report of good or excellent health and low cardiovascular comorbidity was predictive of improved symptoms and greater postoperative walking ability [33]. In the authors' opinion, in patients with a clinical symptoms which do not correlate with imaging studies, or those with other neurological disorders such as polyneuropathy or diabetes, electrodiagnostic evaluation including paraspinal mapping to detect the presence of fibrillation potentials in the limbs or paraspinal muscles would be helpful in delineating between those with mechanical pain symptoms and clinical lumbar stenosis.

References

1. Beaman D, Graziano G, Glover R (1993) Substance P innervation of the lumbar spine facet joints. Spine (Phila Pa 1976) 18:1044–1049
2. Boden S, Davis D, Dina T, Patronas N, Wiesel S (1990) Abnormal magnetic resonance scans of the lumbar spine in asymptomatic subjects. A Prospective investigation. J Bone Joint Surg Am 72(3):403–408
3. Bogduk N (2004) Practice guidelines. Spinal diagnostic and treatment procedures, 1st edn. International Spine Intervention Society, San Francisco
4. Botwin KP, Gruber RD, Bouchlas CG (2002) Fluoroscopically-guided lumbar transforaminal epidural steroid injection in degenerative lumbar stenosis: an outcome study. Am J Phys Med Rehabil 81(12):898–905
5. Cannon DE, Dillingham MF, Miao H, Andary M, Pezzin LE (2007) Musculoskeletal disorders in referrals for suspected cervical radiculopathy. Arch Phys Med Rehabil 88:1256–1259
6. Carragee E, Don A, Hurwitz E (2009) Does discography cause accelerated progression of degeneration changes in the lumbar disc: a ten-year matched cohort study. Spine (Phila Pa 1976) 34(21):2338–2345
7. Cavanaugh J, Ozaktay A, Yamashita H (1996) Lumbar facet pain: biomechanics, neuroanatomy, and neurophysiology. J Biomech 29:1117–1129
8. Depalma MJ, Ketchum JM, Saullo T (2011) Etiology of chronic low back pain in patients having undergone lumbar fusion. Pain Med 12(5):732–739
9. Derby R, Kine G, Saal JA (1992) Response to steroid and duration of radicular pain as predictors of surgical outcome. Spine (Phila Pa 1976) 17(6):S176–S183
10. Dillingham TR, Pezzin LE (2005) Under-recognition of polyneuropathy in persons with diabetes by non-physician electrodiagnostic services providers. Am J Phys Med Rehabil 84:399–406
11. Dreyer S, Dreyfuss P (1996) Low back pain and the zygapophysial (facet) joints. Arch Phys Med Rehabil 77(3):290–300
12. Dreyfuss P, Henning T, Malladi N (2009) The ability of multi-site, multi-depth sacral lateral branch blocks to anesthetize the sacroiliac joint complex. Pain Med 10(4):679–688
13. Dreyfuss P, Michaelsen M, Pauza K (1996) The value of medical history and physical examination in diagnosing sacroiliac. Spine (Phila Pa 1976) 21(22):2594–2602
14. Dreyfuss P, Dreyer SJ (2003) Lumbar zygapophysial (facet) joint injections. Spine J 3:S50–S59
15. Freemont AJPTGP et al (1997) Nerve ingrowth into diseased intervertebral disc in chronic back pain. Lancet 350:178–181
16. Furman MB, Lee TS, Mehta A, Simon JI, Cano WG (2008) Contrast flow selectivity during transforaminal lumbosacral epidural steroid injections. Pain Physician 11(6):855–861
17. Ghormley R (1933) Low back pain with special reference to the articular facets, with presentation of an operative procedure. JAMA 101:1773–1777
18. Giles L, Taylor J (1987) Innervation of the lumbar zygapophyseal joint synovial folds. Acta Orthop Scand 58(1):43–46
19. Goldwaite GE, Osgood R (1905) A consideration of the pelvis articulations from an anatomical pathological and clinical standpoint. Boston Med Surg J 152(21):593–601
20. Goldwaith JE (1911) The lumbosacral articulation: an explanation of many cases of “lumbago”, “sciatica” and “paraplegia”. Boston Med Surg J 164:365–372
21. Gorbach C, Schmid M, Elfering A (2006) Therapeutic efficacy of facet joint blocks. AJR Am J Roentgenol 186:1228–1233
22. Grob K, Neuhuber W, Kissling R (1995) Innervation of the sacroiliac joint of the human. Z Rheumatol 54(2):117–122
23. Guyer R, Ohnmeiss D (1995) Lumbar discography. Position statement from the North American Spine Society Diagnostic and Therapeutic Committee. Spine (Phila Pa 1976) 20(18):2048–2059
24. Haig AJ, Geisser M, Tong HC, Yamakawa K, Quint DJ, Hoff JT, Chiodo A, Miner J, Phalke VV (2007) Electromyographic and MRI to predict lumbar stenosis, low-back pain and no back symptoms. J Bone Joint Surg Am 89-A(2):358–366
25. Haig AJ, Tong HC, Yamakawa K, Quint DJ, Hoff JT, Chiodo A, Miner J, Choksi VR, Geisser M (2005) The sensitivity and specificity of electrodiagnostic testing for the clinical syndrome of lumbar spinal stenosis. Spine (Phila Pa 1976) 30(23):2667–2676
26. Haig AJ, Tzeng HM, LeBreck DB (1999) The value of electrodiagnostic consultation for patients with upper extremity nerve complaints: a prospective comparison with the history and physical examination. Arch Phys Med Rehabil 80(10):1273–1281. doi:S0003-9993(99)90029-1 [pii]
27. Haldeman S, Shouka M, Robboy S (1988) Computed tomography, electrodiagnostic and clinical findings in chronic workers’ compensation patients with back and leg pain. Spine 13:345–350
28. Helbig T, Lee CK (1988) The lumbar facet syndrome. Spine (Phila Pa 1976) 13(1):61–64
29. Ikeda R (1991) Innervation of the sacroiliac joint. Macroscopical and Histological Studies. Nippon Ika Daigaku Zasshi 58(5):587–596
30. Jackson RP (1992) The facet syndrome. Myth or reality? Clin Orthop Relat Res 279:110–121
31. Jarvik J, Hollingworth W, Heagerty P (2001) The longitudinal assessment of imaging and disability of the back (LAIDback) study: baseline data. Spine (Phila Pa 1976) 26:1158–1166
32. Jensen M, Brant-Zawadzki M, Obuchowski N (1994) Magnetic resonance imaging of the lumbar spine in people without back pain. N Engl J Med 331:69–73
33. Katz JN, Stucki G, Lipson SJ, Fossel AH, Grobler LJ, Weinstein JN (1999) Predictors of surgical outcome in degenerative lumbar spinal stenosis. Spine (Phila Pa 1976) 24(21):2229–2233
34. Kendall R, Werner RA (2006) Interrater reliability of the needle examination in lumbosacral radiculopathy. Muscle Nerve 34(2):238–241

35. Khatri B, Baruah J, McQuillen M (1984) Correlation of electromyography with computed tomography in evaluation of low back pain. Arch Neurol 41:594–597
36. Kikuchi S, Macnag I (1981) Localization of the level of symptomatic cervical disc degeneration. J Bone Joint Surg Br 63:272–277
37. Koc Z, Ozcakir S, Sivrioglu K, Gurbet A, Kucukoglu S (2009) Effectiveness of physical therapy and epidural steroid injections in lumbar spinal stenosis. Spine (Phila Pa 1976) 34(10):985–989
38. Krempen JF, Smith BS (1974) Nerve root injection: a method for evaluating the etiology of sciatica. J Bone Joint Surg Am 56:1435–1444
39. Lindblom K (1948) Diagnostic puncture of intervertebral disks in sciatica. Acta Orthop Scand 17(3–4):231–239
40. Manchikanti L, Boswell M, Singh V, Derby R, Fellows B, Falco FJ, Datta S, Smith HS, Hirsch JA (2009) Comprehensive review of neurophysiologic basis and diagnostic interventions in managing chronic spinal pain. Pain Physician 12(4):E71–E120
41. Manchikanti L, Pampati S, Cash K (2010) Making sense of the accuracy of diagnostic lumbar facet joint nerve blocks: an assessment of the implications of 50 % relief, 80 % relief, single block, or controlled diagnostic blocks. Pain Physician 13(2):133–143
42. Mixter WJ, Barr JS (1934) Rupture of the intervertebral disc with involvement of the spinal cord. N Engl J Med 211:210–214
43. Nardin R, Patel M, Gudas T, Rutkove S, Raynor E (1999) Electromyography and magnetic resonance imaging in the evaluation of radiculopathy. Muscle Nerve 22:151–155
44. Nordman D, Macadaeg K, Sasso R (2002) Selective nerve root injection can predict surgical outcome for a lumbar & cervical radiculopathy: a retrospective review with comparison to MRI. Spine J 2:295
45. Piperno M, Hellio le Graverand M, Reboul P (1997) Phospholipase A2 activity in herniated lumbar discs. Clinical correlations and inhibition by piroxicam. Spine (Phila Pa 1976) 22:2061–2065
46. Pneumaticos S, Chatziioannou S, Hipp J (2006) Low back pain: prediction of short-term outcome of facet joint injection with bone scintigraphy. Radiology 238:693–698
47. Rosenberg J, Quint T, Rosayro A (2000) Computerized tomographic localization of clinically-guided sacroiliac joint injections. Clin J Pain 16(1):18–21
48. Sachs B, Vanharanta H, Spivey M (1987) Dallas discogram description. A new classification of CT/discography in low-back disorders. Spine (Phila Pa 1976) 12(3):287–294
49. Schultz H, Longheed WM (1973) Intervertebral nerve root in the investigation of chronic lumbar disc disease. Can J Surg 16:217–221
50. Schwarzer A, Aprill C, Derby R (1995) The prevalence and clinical features of internal disc disruption in patients with chronic low back pain. Spine (Phila Pa 1976) 20(17):1878–1883
51. Schwarzer A, Derby R, Fortin A (1994) Pain from the lumbar zygapophysical joints: a test of two models. J Spinal Disord 7(4):331–336
52. Schwarzer AC, Derby R (1995) The prevalence and clinical features of internal disc disruption in patients with chronic low back pain. Spine (Phila Pa 1976) 20(17):1878–1883
53. Smith CC, Booker T, Schaufele MK, Weiss P (2010) Interlaminar versus transforaminal epidural steroid injections for the treatment of symptomatic lumbar spinal stenosis. Pain Med 11:1511–1515
54. Sohn MW, Whittle J, Pezzin LE, Miao H, Dillingham TR (2011) Electrodiagnostic consultation and identification of neuromuscular conditions in persons with diabetes. Muscle Nerve 43:812–817
55. Tortolani PJ, Carbone JJ, Quartararo LG (2002) Greater trochanteric pain syndrome in patients referred to orthopedic spine specialists. Spine J 2(4):251–254. doi:S1529943002001985 [pii]
56. Walker J, El Abd O, Isaac Z (2008) Discography in practice: a clinical and historical review. Curr Rev Musculoskelet Med 1(2):69–83
57. Wilbourn A, Aminoff M (1998) AAEM minimonograph 32: the electrodiagnostic examination in patients with radiculopathies. Muscle Nerve 21:1612–1631
58. Willick S, Kendall R, Roberts S (2009) An emerging imaging technology to assist in the localization of axial spine pain. PMR 1(1):89–92
59. Yagci I, Gunduz O, Ekinci G, Diracoglu D, Us O, Akyuz G (2009) The utility of lumbar paraspinal mapping in the diagnosis of lumbar spinal stenosis. Am J Phys Med Rehabil 88(10):843–851
60. Young IA, Hyman GS (2007) The use of lumbar epidural/transforaminal steroids for managing spinal disease. J Am Acad Orthop Surg 15:228–238

6

General Considerations for Spine Surgery Including Consent and Preparation. General Surgical Principles, Guidelines for Informed Consent, Patient Positioning for Surgery, Equipment Needed, and Postoperative Considerations

Evalina Burger

6.1 Introduction

The general surgical principle in spine surgery is to remember that there are no small spine surgeries. These are usually bigger surgeries, and most patients are sick and/or debilitated as well as deconditioned because of their chronic pain. The general surgical principle for spine surgery is "Do no harm." The second rule is to know your limitations, and the third rule is to ensure that the surgeon's and the patient's expected outcomes are synonymous [1].

6.2 Approach

Be cognizant of the patient's comorbidities and plan your approach accordingly. For example, do not plan an anterior approach in a patient where you know there are contraindications such as previous surgery for vascular disease. Tailor your surgery to the patient's comorbidities in terms of diabetes, osteoporosis, immune deficiency, etc. For example, the anterior lumbar approach presents many benefits in terms of restoring the height of the disk and presents a larger area for fusion, but the technical difficulty of the approach with potential blood loss makes candidate selection critical. In the cervical area, the anterior approach is much more the "standard of care." The posterior approach for the cervical spine can present with significant morbidity due to creeping fusion and muscle destruction, while it is the most common approach for lumbar surgery. Though muscle injury is always a concern, it is a relatively safe approach and allows access to the spine and the spinal cord allowing decompression and fusion (Table 6.1).

6.3 Consents

Spine surgery is probably one of the areas that is most litigious. Spine surgery poses a great avenue for lawsuits, due to the complexity of the surgery and the misunderstanding of the complexity of the surgeries by patients. Informed consent is therefore a vital part of communication with your patients. In performing consents, it is best to draw up consents that list most of the common complications listed as a general consent and then add the specific high-risk complications of the particular procedure. It is vital that the patient has

E. Burger
Department of Orthopaedics,
University of Colorado School of Medicine,
12631 East 17th Avenue, MS B202 Room 4602,
Aurora, CO, 80045, USA
e-mail: evalina.burger@ucdenver.edu

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_6, © Springer-Verlag Berlin Heidelberg 2014

Table 6.1 Advantages and disadvantages of anterior and posterior approaches

Anterior spine approach	Advantage	Disadvantage
Lumbar	Disk height restoration	Major vessel injury
	Broader surface area with higher fusion rates	Retrograde ejaculation (7 %)
	Less painful	Difficult in previous surgery or obese patients
Cervical	Easy bloodless dissection	Swallowing difficulties
	Disk height restoration	Potential injury of the recurrent laryngeal nerve
	Decompression of the foramen and cord can be performed adequately	Challenging to reach above C3 and below C7
Posterior spine approach		
	Most procedures can be performed with access to bone and canal and disk	Significant muscle scarring with chronic muscle pain
		Lower fusion rates
	Sagittal balance restoration possible with osteotomies	Painful prominent instrumentation
Cervical	Preferred method for more than four levels of surgery	Muscle dysfunction with neck webbing
		C5 nerve root weakness associated with some procedures
		Creeping fusions

the educational level to understand the explanation. If this is not possible, visual aids and printed material can be very helpful [2, 3].

As a general rule, the consent should include the following: bleeding, which can be acute, late, or subacute; infection which can also be acute or late [4]; and clotting disorders, including DVTs, pulmonary emboli, and, specifically, epidural hematomas.

6.3.1 Pain

As most patients come to spine surgery for pain relief, it is prudent to have the expected outcome in terms of pain relief in the consent, with the understanding that neurogenic pain might stay the same, improve, or even get worse. Even in the best hands, nerve root injury can occur unexpectedly which might render the patient with new pain or worsening pain of a previously compressed nerve root. Neurological complications are devastating and may consist of paralysis, which may be complete, incomplete, or nerve specific. It is vital to add in under neurological complications loss of bladder or bowel function as most patients do not understand that the concept of paralysis also includes the fact that the bladder and bowel functions are lost [5]. If a patient remains with cauda equina syndrome or develops this, the patient could potentially sue for misinformation as it is very difficult for patients to equate bladder or bowel loss as part of paralysis if it is not specifically stated.

6.3.2 Specific Surgical Procedures

6.3.2.1 Cervical Spine Surgeries

When consenting patients for cervical spine surgeries, the specific considerations would be to mention voice changes as this might happen with or without paralysis of the vocal chords. It is also important to point out swallowing difficulty, which may be caused by nerve injury or esophageal injury. Strokes due to carotid occlusion and direct vessel injury should also be discussed as the anterior approach puts the carotid arteries at direct risk for compression, which can cause plaque to dislodge causing strokes. The vertebral arteries can be occluded due to preexisting narrowing and rotation of the head [6].

6.3.2.2 Lumbar Surgeries

It is important to point out pressure points and potential skin injuries. Make patients aware that the surgery is being done prone and/or supine and that due to the length of these surgeries, the risk for pressure points and skin abrasions is always a reality [7]. Additional injury to the anterior cutaneous nerves of the thigh is possible from both ant + posterior approaches.

The next item of importance is instrumentation. It has to be mentioned that screws can be misplaced and that instrumentation can break or disconnect as patients do not understand this. In terms of a fusion, it is important to point out pain from bone grafting sites as well as the long-term complications of nonunion [8].

Although the topic of death is a rare occurrence, the same as paralysis, it still has to be mentioned as well as the complication of blindness as these complications are very rare but devastating. A general note of multiorgan failure and/or complications should be added in as patients can suffer myocardial infarctions, pulmonary emboli, and strokes during surgery [9].

It becomes impossible to list every possible known complication and therefore try to categorize your consent list for informed consent into systems. The intent of a consent form is to point out possible complications and not to scare patients to the point where they do not want the surgery. It is a fine balance between too much information and enough for the patient to feel comfortable [10].

6.4 Patient Positioning for Surgery

Spine patients are mainly positioned in supine, prone, or lateral position. It is therefore important to plan your surgery to decide which approach would be better and ensure all the equipment needed for the position, is available and in a working order [11]. Supine approaches are for anterior interbody work and anterior cervical work. The patient should be positioned in an optimal position where the spine is near the normal alignment, taking into consideration that some of these surgeries are performed for sagittal imbalance [12]. Great care should be taken to never overextend the neck whether this is done as part of a neck surgery or where the patient lies in extension during a lumbar anterior approach. Various aids are available including bumps, gel rolls, and beanbags, and the equipment should be tested and available for the surgery. It is important to position the patient in such a manner that the access is easy as well as to assure visualization with X-rays during the surgery [13]. One of the most frequent mistakes made in spine surgery is wrong level or wrong side due to inadequate X-ray visualization, which in turn can be due to poor positioning. The ideal time to ensure adequate fluoroscopy visualization is during the positioning, before the patient is draped.

In positioning patients in the prone position for posterior cervical, thoracic, or lumbar surgery, it is vitally important to make sure that there is no pressure on the abdomen. Too much pressure on the abdomen may cause venous compression which in turn leads to increased bleeding due to shunting of the blood through the epidural vessels. Older positioning techniques such as the Wilson frame and gel rolls have led to undesired sagittal balance and/or increased bleeding. Newer positioning techniques, with bolstered tables such as the Jackson or Axis table, allow for excellent decompression of the abdominal content and spread the pressure points evenly across the chest while maximizing lordosis. Please make sure that adequate padding is available and remember that even obese patients can get significant skin breakdown. Of further importance in the positioning in the prone position is to make sure that the arms are not kept in abduction or too much elevation of the shoulders. Never place the shoulders in more than 90° of abduction and do not place too much tension on the shoulder joint as this can all cause traction injuries of the brachial plexus [14].

The face is very important in spine surgery. Whether the patient is placed supine or prone, the eyes need to be protected, and patients often will end up with significant skin abrasions in the prone position due to irritation and perspiration. Furthermore, the eyes need to be protected for anterior neck surgery so that sharp instruments are not dropped or placed on the eyes with subsequent eye injury. Of particular importance is the breast in female patients. Make sure that implants are treated appropriately as nipple necrosis can occur in patients that have had implants and are big breasted, and therefore, additional padding with cutout rings around the nipples is desirable when positioning female patients in the prone position.

The lateral position is always a challenge. Depending on whether the lateral position is for thoracic or lumbar approaches, the method to relax the psoas is to keep the bottom leg straight and the upper leg flexed close to 90°. This relaxes the psoas on the side of the approach. Having the legs in this position also stabilizes the pelvis, and beanbags with adequate strapping and padding are needed as well as an axillary roll to protect the long thoracic. In the thoracic area, it is important to take care of the arm if a high thoracic approach is planned so that the arm is placed appropriately in a sling without traction.

After all the positioning is done, it is important to note that neuromonitoring plays a very important role. Neuromonitoring not only is appropriate for the level that we work on but also manages to monitor the extremities to prevent injury to the upper extremities when we are working on the lower extremities and not have your patients wake up with either radial or ulnar nerve palsy. When we are treating patients with severe myelopathy such as in rheumatoid disease with instability or fractures, it is important to have neuromonitoring signal recording before any positioning is attempted. You need a baseline to make sure that the positioning does not paralyze the patient. If this is not done and neuromonitoring shows abnormalities of either the sensory-evoked potentials or motor-evoked potentials, it is hard to discern whether this is due to the surgery when in fact this could have happened during the positioning. It is important to have a succinct preoperative discussion with the anesthesiologist to make sure that they understand the stability of the neck and the desirable position and that endotracheal intubation is sometimes needed awake or fiber-optically.

6.5 Equipment

"A bad craftsman always blames his tools," and there is no other discipline where this is more true than in spine surgery. Dull instruments or inappropriate instruments inevitably will cause irreparable nerve damage, and therefore, this should not be tolerated. Make sure that the sets are appropriate and that all the right sizes of Kerrison and pituitary rongeurs are available. Make sure that the hospital has appropriate sets for your preferences and at least the basic instruments to open a spine and do an emergency laminectomy. Even though you might not be the main spine surgeon doing an emergency laminectomy or diskectomy, it should be part of what is expected from a general neuro- or orthopedic surgeon. Always test the radiology equipment to make sure that it is in a workable condition and that the imaging is satisfactory and always make sure that the sets are appropriately sterilized before the patient is put to sleep. There is nothing worse than waking a patient up with the inability to complete surgery due to equipment malfunction. This always includes the bed: make sure the bed that you use can rise up or change position to allow adequate radiology access and that all the safety mechanisms of the operating bed are in place to prevent iatrogenic patient injury. Make sure that the Mayfield clamps are at the appropriate settings and the same when applying a halo when a Mayfield is not available. Ensure that all the traction is connected to the bed in a sturdy position. This kind of equipment has to be tested by the surgeon. It is the surgeon's responsibility to make sure that it is in working condition before the patient is positioned.

Equipment also includes the availability of an appropriate microscope when needed—always ensure that it is well balanced and that the eyepiece focus is properly adjusted before draping.

Never take a patient to the room without visualizing the X-rays which means that this has to be pulled up on electronic medical records or the hard copies displayed in the room so that during the time of the procedure, the level can be checked again. This is primarily the surgeon's responsibility.

Table 6.2 Equipment needs for special issues

Issue	Need
Osteoporosis	Cement Additional fixations such as cables and hooks
Revision surgery	Screw removal systems Wide variety of new screws Metal-cutting burs and easy-out systems Iliac bolts Additional and various rod screw connectors and rod extenders
Big blood loss surgery	Double equipment such as bovies and bipolars for assistant to facilitate the surgery
Anterior lumbar surgery	Second pulse oximeter to monitor circulation to the leg

It is feasible to have a checklist of all the equipment that may be needed during spine surgeries, and it should be shared, so that all the personnel are aware of what is needed (Table 6.2).

Another word of wisdom: "Always have a backup plan."

6.6 Postoperative Considerations

The postoperative treatment starts in the preoperative planning. A well-performed and well-executed surgery will leave a happy patient who will have relief of their preoperative pain and be able to work through the postoperative recovery as long as they have reassurance and their pain is controlled in the first 24 h. Make sure that orders are written appropriately for neuro- and vascular monitoring, and make sure that the nursing staff understands the importance of and specifically which groups of muscles or distal pulses need to be monitored. Never disregard inappropriate or increasing complaints of pain, as this might be one of the first signs of a possible lurking disaster such as an epidural hematoma. Furthermore, discuss preoperative expectations in the patient whether you want to order a brace and/or bone stimulator or any other equipment that might be needed. It is a great practice to order this preoperatively as this facilitates the discharge. Make sure that the patients are adequately informed about the expectations in terms of wound healing, bandages, and sutures and have the basic knowledge and understanding of how complications may present.

Make sure that postop complications are not dismissed as a small wound infection might lead to a disastrous outcome. Do not dismiss the patient's complaints about leg pain, wound redness, and swelling, and make sure that they come to the office for a visit rather than brushing them off over the phone as drug seeking. Most patients that have postoperative complaints are legitimate, and even though the surgeon might have the knowledge, patients also have reasonable knowledge about their own bodies, and this should not be disregarded [15].

6.6.1 Postoperative Visual Loss (POVL)

A much feared and devastating complication associated with spine surgery is postoperative loss of visual acuity [16]. This can occur with or without associated ocular trauma. The visual deficits can range from blurring to complete blindness, and depending on the etiology, the prognosis for recovery can be poor. There are at least four types of visual loss: central retinal artery occlusion, central retinal vein occlusion, cortical blindness, and ischemic optic neuropathy [16, 18]. Central retinal artery and central retinal vein occlusion are most commonly associated with direct trauma to the globe of the eye (usually direct pressure on the globe resulting in a precipitous increase in intraocular pressure) and less commonly with embolic events. This makes proper positioning of the patient with careful attention to protection of the eyes imperative to prevent this injury. In addition to spine surgery, ION has occurred during cardiac procedures and also robotic intra-abdominal surgery with the patients placed in a steep Trendelenburg position [17]. Most commonly, however, POVL follows prone spine surgery and is caused by ischemic optic neuropathy (ION) [19]. Estimates of the incidence of ION after spine surgery range from 0.01 to 0.2 % [6, 20, 21]. ION can occur in anyone including otherwise young and healthy patients [22].

The causes of ION are currently unknown, but ongoing research into the etiology of the visual loss has provided some insights. It is known that the anatomy of the optic nerve and its blood supply places the nerve at risk within the orbit and at the lamina cribrosa where it penetrates the thick sclera. The blood supply can be variable, and there is also a watershed area of perfusion along the midsection of the nerve between the zones of perfusion from the more posterior hypophyseal branches of the carotid artery and the short posterior ciliary artery anteriorly. The neuropathy is likely caused by a decrease in perfusion pressure to the optic nerve below the autoregulatory threshold, and the extent of the injury will depend on the severity and duration of the ischemia [1]. A recent analysis from the American Society of Anesthesiologists Postoperative Visual Loss (ASAPOVL) registry was conducted using the case – control method [21]. In this study of patients undergoing prone spine surgery, the goal was to identify independent risk factors for the development of ION. Variables found to independently increase the risk of ION were male sex, obesity, use of the Wilson frame, increasing duration of surgery, increasing amount of blood loss, and lower percentage of colloid to crystalloid fluid replacement. The fact that males seem particularly at risk compared to females was also confirmed by an earlier study and may suggest a protective effect of estrogen [20]. Factors and conditions previously postulated to contribute to the risk of POVL but not found to be independent variables included level of anemia, intraoperative blood pressure, and the presence of chronic hypertension, atherosclerosis, smoking, or diabetes. These investigators point out that the findings suggest that intraoperative physiologic changes have a greater influence on the development of ION than preexisting conditions and that venous congestion plays a significant role in precipitating these physiologic perturbations [22].

Currently, there is no known effective treatment for ION, placing prevention at the forefront of patient management strategies. The American Society of Anesthesiologists has published a practice advisory for POVL associated with spine surgery [23]. Review of the literature has led these experts to make a number of recommendations concerning this complication. An abbreviated list of these includes:

1. There are no identifiable preoperative patient characteristics which predispose to ION, but the risk may be increased in patients undergoing prolonged procedures, those with large blood loss, or both.
2. Consider informing these patients that there is a small, unpredictable risk of POVL.
3. Colloids should be used along with crystalloids to maintain intravascular volume in the face of substantial blood loss.
4. There is no documented lower limit of hemoglobin concentration associated with the development of ION; therefore, no transfusion threshold which eliminates the risk of POVL can be established.
5. Direct pressure on the eye should be avoided to prevent central retinal artery occlusion.
6. High-risk patients should be positioned so that the head is above or level with the heart, and the head should be in a neutral forward position.
7. Consideration should be given to the use of staged procedures in high-risk patients.

The practice of staging of prolonged surgical procedures with anticipated substantial blood loss into two or more shorter procedures has been endorsed by the North American Spine Society and the North American Neuro-Ophthalmology Society.

6.7 Pearls

Let common sense prevail.
Pay attention to detail.
Plan well.
Be meticulous.

From inability to let well alone: from too much zeal for the new and contempt for what is old: from putting knowledge before wisdom, science before art, and cleverness before common sense, from treating patients as cases, and from making the cure of the disease more grievous than the endurance of the same, Good Lord, deliver us.
Robert Hutchison

References

1. Lurie JD (2008) Patient preferences and expectations for care: determinants in patients with lumbar intervertebral disc herniation. Spine (Phila Pa 1976) 33(24):2663–2668
2. Ng CY, Gibson JN (2011) An aid to the explanation of surgical risks and complications: the International Spinal Surgery Information Sheet (ISSiS). Spine (Phila Pa 1976) 36(26):2333–2345
3. Mauffrey C et al (2008) The influence of written information during the consenting process on patients' recall of operative risks. A prospective randomised study. Int Orthop 32(4):425–429
4. Czerwein JK Jr et al (2011) Complications of anterior lumbar surgery. J Am Acad Orthop Surg 19(5):251–258
5. Brahams D (1989) Informed consent and 25 % risk of paralysis. Lancet 2(8653):57–58
6. Fountas KN (2007) Anterior cervical discectomy and fusion associated complications. Spine (Phila Pa 1976) 32(21):2310–2317
7. Harimaya K (2009) Increasing lumbar lordosis of adult spinal deformity patients via intraoperative prone positioning. Spine (Phila Pa 1976) 34(22):2406–2412
8. Deyo RA, Nachemson A, Mirza SK (2004) Spinal-fusion surgery – the case for restraint. N Engl J Med 350(7):722–726
9. Schwab F (2010) Adult spinal deformity-postoperative standing imbalance: how much can you tolerate? An overview of key parameters in assessing alignment and planning corrective surgery. Spine (Phila Pa 1976) 35(25):2224–2231
10. Carl A, Kaufman E, Lawrence J (2010) Complications in spinal deformity surgery: issues unrelated directly to intraoperative technical skills. Spine (Phila Pa 1976) 35(25):2215–2223
11. Vanderhave KL et al (2011) Cervical spine trauma in children and adults: perioperative considerations. J Am Acad Orthop Surg 19(6):319–327
12. Tormenti MJ et al (2010) Complications and radiographic correction in adult scoliosis following combined transpsoas extreme lateral interbody fusion and posterior pedicle screw instrumentation. Neurosurg Focus 28(3):E7
13. Bayley E, Clamp J, Boszczyk BM (2009) Percutaneous approach to the upper thoracic spine: optimal patient positioning. Eur Spine J 18(12):1986–1988
14. Uribe JS et al (2010) Brachial plexus injury following spinal surgery. J Neurosurg Spine 13(4):552–558
15. Halpin RJ (2010) Standardizing care for high-risk patients in spine surgery: the Northwestern high-risk spine protocol. Spine (Phila Pa 1976) 35(25):2232–2238
16. Williams EL (2002) Postoperative blindness. Anesthesiol Clin North America 20(3):367–384
17. Nuttall GA, Garrity JA, Dearani JA et al (2001) Risk factors for ischemic optic neuropathy after cardiopulmonary bypass: a matched case/control study. Anesth Analg 93:1410–1416
18. The Postoperative Visual Loss Study Group (2012) Risk factors associated with ischemic optic neuropathy after spinal fusion surgery. Anesthesiology 116:15–24
19. Stevens WR, Glazer PA, Kelley SD et al (1997) Ophthalmic complications after spinal surgery. Spine 22(12):1319–1324
20. Patil CG, Lad EM, Lad SP et al (2008) Visual loss after spine surgery: a population-based study. Spine 33(13):1491–1496
21. Shen Y, Drum M, Roth S (2009) The prevalence of perioperative visual loss in the United States: a 10-year study from 1996 to 2005 of spinal, orthopedic, cardiac, and general surgery. Anesth Analg 109:1534–1545
22. Holy SE, Tsai JH, McAllister RK et al (2009) Perioperative ischemic optic neuropathy: a case control analysis of 126,666 surgical procedures at a single institution. Anesthesiology 110:246–253
23. American Society of Anesthesiologists (2012) Practice advisory for perioperative visual loss associated with spine surgery: an updated report by the American Society of Anesthesiologists Task Force on Perioperative Visual Loss. Anesthesiology 116:274–285

Surgical Approaches

7

Michael C. Gerling and Sheeraz A. Qureshi

This chapter will review the most common surgical approaches to the cervical, thoracic, and lumbar spine with an emphasis on associated indications, anatomy, pearls, and pitfalls. Details not presented herein are easily found in anatomic and dedicated surgical approach textbooks, technique guides, articles, and videos.

Each region has unique anatomic factors that guide the surgical approach selection, though in many cases the choice of approach is dependent upon the surgeon's preference. Influential factors include the pathology type, the regional alignment of the spine, proximity to the spinal cord, goals of surgery, and surgeon preference. Some basic rules include:

1. Spinal cord retraction, elongation, or compression is not safe or permissible during surgery.
2. Laminectomy or posterior decompression without fusion should be performed with caution when kyphosis is present.
3. When possible, it is preferable to approach the spine from the direction where the pathology is found.

M.C. Gerling, M.D. (✉)
Department of Orthopaedic Surgery,
SUNY Downstate Medical Center, Brooklyn, NY, USA

Lutheran Medical Center,
150 55th Street, Brooklyn, NY 11220, USA
e-mail: michael.gerling@lmcmc.com

S.A. Qureshi, M.D., MBA
Department of Orthopaedic Surgery, Mount Sinai Hospital,
Mount Sinai School of Medicine, 5 East 98th Street,
9th Floor, 1188, New York, NY 10029, USA
e-mail: sheeraz.qureshi@mountsinai.org

7.1 Cervical Spine

Surgical treatment of cervical spinal disorders is increasingly common, particularly for the treatment of degenerative disease in patients over 65 years of age. Other indications include traumatic fracture or disk herniations, ossification of the longitudinal ligament (OPLL), deformity, infection, and tumors. The spinal cord is present throughout the cervical spine and can be a source of tremendous disability or occasionally catastrophic injuries that greatly influence surgical decision-making.

7.1.1 Anterior Cervical Approach

The anterior approach to cervical pathology allows access to the cervical disks and vertebral bodies from the anterior aspect. It is commonly used when pathology is anterior, when kyphosis correction is required, and for localized one- or two-level disease. It is combined with the posterior approach for treatment of tumors and multilevel degenerative disease where fusion may be challenging to achieve and especially when combined with cervical deformity.

Positioning

When stenosis is severe or the patient is symptomatic with neck extension, nasotracheal intubation is recommended. Otherwise, in-line stabilization is recommended during orotracheal

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_7, © Springer-Verlag Berlin Heidelberg 2014

intubation. The patient is positioned supine with slight elevation of thorax by placing a bump or rolled towel between the shoulder blades. Head extension is slight and fine-tuned with sheets or cushion under the head. Adequate visualization of the target disk level is confirmed on lateral X-ray or fluoroscopic imaging. Shoulders are gently taped distally with arms tucked at sides and bony prominences protected.

Landmarks

The sternocleidomastoid muscle (SCM) demarcates the lateral aspect of the incision. The medial extent of the incision is catered to the procedure, making longer incisions when more levels are exposed. The sternal notch, thyroid cartilage, and chin define midline. Fluoroscopy can be used to confirm proximity to operative disk levels. Common landmarks for the height of the incision: one finger breadth above the thyroid cartilage is the 3–4 disk level, the thyroid cartilage spans the 4–6 levels, the cricothyroid cartilage is the C5–6 level, and C6–7 is one finger breadth above the clavicle.

Incision

Incision is more cosmetic when transverse, in line with the skin folds. The platysma can be split in line with its fibers or in line with the incision. Care is taken during undermining of the platysma to protect the underlying veins and nerve plexi. The sternocleidomastoid muscle is then identified and gently retracted laterally to expose the carotid sheath that is identified by palpating the carotid pulse. It is carefully protected during the remainder of the surgery.

Planes

The anteromedial approach is most common, defined by passing medial to the carotid sheath that is retracted laterally, and the midline visceral structures are protected medially. Conversely, the anterolateral approach to the cervical spine involves medial retraction of the carotid sheath. This technique is less common and limits the exposure of the prevertebral fascia. It may be a more avascular plane, however. The prevertebral fascia is thus exposed, and the longus colli muscles visualized on either side of the disks that are identified visually and palpably. The disk level is then confirmed using imaging, often by placing a spinal needle into the most likely level. Alternatively, a clamp can be placed on the longus coli. Longus coli are then undermined and self-retaining retractors placed.

Pearls and Pitfalls

Postoperative dysphagia may be improved with careful attention to minimizing trauma of the midline structures. Several techniques are employed, including deflation and reinflation of the endotracheal cuff after placement of self-retaining retractors, placing self-retaining retractors under the longus coli, and periodically moving self-retaining retractors or using handheld retractors alone. The superior laryngeal nerve controls phonation of high notes and is carefully protected from damage in professional singers. It is famous for ending the career of the opera singer, Galli-Curci, after the nerve was severed during surgery. The omohyoid muscle crosses the field at the C5–6 level obliquely and is often sacrificed when interfering with approaches at this level. When possible, it is important to preserve vascular and neurologic structures crossing the field. Cautious identification of vessels and nerves is required, with either retraction or ligation of the vessels when necessary. Cephalad exposures identify the hypoglossal nerve, glossopharyngeal nerve, digastric muscle, superior laryngeal nerve, and the superior thyroid artery and vein. Caudal exposures identify the middle thyroid vein that can be ligated, the inferior thyroid artery and vein, and the omohyoid muscle typically crossing the field at C5–6.

Right-sided approaches distal to C4 require identification of the recurrent laryngeal nerve crossing transversely into the midline viscera. Left-sided approaches distal to C7 may encounter the thoracic duct entering the jugular vein-subclavian vein junction.

7.1.2 Posterior Approaches to the Cervical Spine, Including the Occipitocervical and Cervicothoracic Junctions

Indications

Posterior exposure of the posterior skull base and cervical and upper thoracic spine may be indicated for decompression, biopsy, and for instrumented fusion. Common indications include upper cervical instability, cervical fracture, and multilevel degenerative disease, especially with facet arthrosis.

Positioning

After endotracheal intubation, neuromonitoring including in some cases SSEP and MEP is typically recorded at baseline. Skeletal traction or stabilization is often used and applied at this time in the supine position. Either the surgeon or experienced anesthesiologist is charged with controlling head position during the transition to the prone position. The patient is rolled from stretcher to OR table in a slow controlled fashion, with bolsters to support chest, pelvis, and lower extremities. The traction device is attached to the bed frame before reattaching the ventilator. The arms are gently tucked to side, and shoulders may be taped distally when distal levels of the cervical spine will be exposed or when the neck is short or bulky fat pad is present or obstructs access. A radiolucent table is recommended for optimal imaging in the anterior posterior plane. Mayfield tongs are useful for placing the head in the military tucked position, with chin flexed forward and slight longitudinal traction in patients without dislocation or marked instability. In the presence of instability, it is important that imaging confirmation of bony alignment is obtained during the positioning process prior to commencing the procedure. This position must be suited to the intended operation and typically improves access during decompression of the upper cervical spine. It can be used during screw or wire placement on the C1 and C2 bones, but the head must be extended prior to finalizing instrumentation during fusion in order to lock the spine in anatomic alignment.

Landmarks

The skull is shaved from the posterior hairline to approximately 2 in. above the intended incision. For exposure and instrumentation of the occiput, the incision usually starts at the occipital also known as inion, external occipital protuberance. The exposure usually extends distally in the midline to approximately 1 in. below the spinous process of the distal vertebrae that will be instrumented or exposed. Fluoroscopic imaging or X-ray is useful when extending to the distal cervical levels.

Incision

The incision is carried down sharply through the dermis, and then electrocautery is usually used to expose the spinous process in the midline. The ligamentum nuchum is typically split down the middle, and interspinous ligaments are protected until the operative level is confirmed with intraoperative imaging. Care should be taken as the bleeding can be copious, if one strays off of the midline. After confirmation of operative level, the muscles are stripped off the spinous processes of the operative level only, preserving the ligamentous connections to adjacent levels. Spontaneous fusion can occur between adjacent lamina and at the occipitocervical junction in children when nonoperative levels are dissected. Subperiosteal dissection is performed with elevators to expose necessary anatomy on the occiput and cervical levels, as far laterally as the lateral aspect of the lateral mass of levels distal to C1.

Care is taken at the occipitocervical junction as there are larger gaps between the laminae. It is important to stay on the lamina during dissection as deep penetration adjacent to the C1 arch may injure the atlantooccipital membrane and dura. The C1 arch anatomy is variable and should be studied on preoperative imaging, often using a CT scan or MRI. Dissection laterally on the C1 arch is limited by the course of the vertebral artery as it exits the C1 foramen transversarium and courses posteromedially into its groove on the cephalad aspect of the C1 arch. It is typically safe to dissect up to 3 cm laterally on the C1 arch without injury of the vertebral artery or vein. The blue hue of the vein is first visualized in the groove as a warning to stop dissecting toward the artery. The second cervical ganglion is also encountered during lateral dissection at the inferior aspect of the C1 arch. Further dissection laterally would place the vertebral artery at risk as it passes between the foramen transversarium of C2 and C1. It is also important to note that the C1 arch is often weak and prone to fracture if too much pressure is placed upon it.

Pearls and Pitfalls
Bleeding is minimized with careful attention to midline dissection deep to the incision without violating the paraspinal musculature. Experienced surgeons may inject an epinephrine solution into the paraspinal muscles to decrease bleeding, understanding the risk of dural puncture or cord injury. C1–2 fusion techniques may require subperiosteal dissection of the C2 posterior elements, the isthmus of the lateral arch, and C1–2 facet joint.

7.2 Thoracic Spine

7.2.1 Anterolateral/Lateral Thoracotomy Approach

This approach is most frequently used for burst fractures, disk herniations, anterior release of deformity, infection, and tumors. The approach is amenable to use with traditional retractors or with modern tubular retractor systems.

Positioning
Lateral decubitus position with the most affected side up. A bump is placed under the axilla, and the head is stabilized in the neutral position. For thoracolumbar levels, the diaphragm may need to be retracted anteriorly during the approach. In these cases, the hinge of the table is placed at the approximate level of the twelfth rib or thoracolumbar junction to increase the working distance between the rib cage and iliac crest when the table is extended. The body is carefully padded, especially the fibular head and axilla to avoid peroneal palsies or brachial plexus palsies, respectively. Carefully secure the body to prevent rolling during imaging and exposure where the table may be subsequently repositioned. Tape is used with caution in longer cases as skin breakdown may occur.

Landmarks
Preoperative planning with CT scan or MRI imaging can identify the intercostal space or if necessary, which rib needs to be partially resected. The surgeon can then count the ribs and identify levels for incision. Many surgeons use intraoperative fluoroscopy to identify the optimal plane for incision, especially when tubular retractors are used as they are less forgiving and require more precise alignment with the disk space. In general, a direct lateral approach will require partial resection of the rib that descends from two levels above the involved level. The incision is made in line with the rib to be resected. If tubular retractors are used, fluoroscopy is typically used to identify the incision site, immediately lateral to the site of pathology. Sharp dissection is used to expose the underlying muscle that is split in line with its fibers. Depending upon the targeted level, the layers may include the trapezius, latissimus dorsi, rhomboid major, and the serratus posterior. The rib periosteum is exposed and split in line with the rib followed by subperiosteal dissection of the portion that will be resected. Careful attention should be paid to the costal nerve and vessels that lay beneath the caudal edge of the ribs, both during rib resection and subsequent placement of a rib spreader. The rib is dissected from all soft tissue before the rib cutter is used. If the twelfth rib is sacrificed for approaches to the thoracolumbar junction, then it is disarticulated from its vertebral body and removed in one piece. The ipsilateral lung can be deflated by anesthesia during rib resection and placement of the rib spreader, and it should be directly protected during intrathoracic work. After entering the thoracic cavity, the lung is gently displaced and protected anteriorly, and the ipsilateral aspect of the spine lies posteriorly, invested by pleura. The pleura is gently elevated and split longitudinally, with careful attention to the radicular arteries that run transversely across the body, in parallel with the intervertebral disks. These arteries are sacrificed when necessary for exposure of the vertebral body. A chest tube is

placed and attached to suction at the end of the procedure, usually below the ninth rib.

Pearls and Pitfalls
Positioning is critical to the success of lateral procedures. Proper protection of the prominent bony elements will avoid peripheral complications. Neuromonitoring is critical for early detection of evolving peripheral and central nervous system injuries. The primary arterial supply to the spinal cord at the thoracolumbar junction is the artery of Adamkiewicz most commonly occurring at the level of T10 to L1. It is widely believed that injury to this artery only occurs when dissection extends into the vertebral foramen. Thus, it should be avoided. Avoid injury to the intercostal artery and vein while dissecting the ribs before resection. Open the parietal pleura 1.5 in. from the head of the rib. Segmental vessels are tied off. Use costovertebral articulation, rib head, and pedicle as a landmark to avoid vascular and neurologic injury. Close the parietal pleura and use heavy woven suture to approximate ribs during closure.

7.2.2 Posterior Thoracic Approach

This is a common approach for the thoracic spine and can be adapted to treat the majority of indications in the region. It is used for posterior decompression of stenosis, posterolateral intertransverse process fusions, posterior instrumentation, and resection of posterolateral elements including the facet joints and/or costovertebral articulations when performing anterior decompression/fusions from a posterior approach.

Positioning
After intubation, anesthesia completes their preparation of the patient, possibly including arterial line placement. The surgical team determines the necessity of catheterization of the bladder and neurologic monitoring as indicated depending on magnitude of case. Cord level cases typically require spinal cord monitoring. After intubation, the patient is rolled into the prone position on the operative table. The arms are typically placed overhead and abducted 90° at the shoulder and elbow. Bony prominences are padded, and the face is inspected to ensure there is no pressure on the eyes. The abdomen should be free of compression to reduce intra-abdominal pressure.

Landmarks
The spinous processes lay in the midline. Ribs help identify the thoracic levels and the cervicothoracic and thoracolumbar junctions. Though the iliac crest typically lies at the level of L4–5, preoperative X-rays are used during preoperative planning to confirm the patient's specific anatomy.

Incision
A midline incision is carried down to the spinous processes, with careful attention to hemostasis. Injection of a dilute epinephrine solution often helps reduce bleeding but should be used with caution under the supervision of an experienced surgeon. The fascia is incised the length of the incision, and Cobb Elevators are often used to dissect the paraspinal muscles off the midline. Electrocautery and packing techniques help to control bleeding. The extent of the exposure is related to the procedure. The lamina alone is exposed and stripped of muscle for laminectomies. The facets and transverse processes are exposed during fusion cases, for instrumentation and when anterior structures will be approached via resection of posterior elements, such as interbody fusions, osteotomies, and scoliosis procedures.

Pearls and Pitfalls
Resection of the interspinous ligaments of adjacent levels may lead to adjacent-level degeneration or deformity. Removing the posterior elements in the central thoracic spine may lead to kyphosis, especially when it occurs at the end of a long fusion with rigid instrumentation.

7.3 Lumbar Spine

7.3.1 Anterior Lumbar Approach

Access to the intervertebral disk space and anterior vertebral bodies of the distal lumbar spine, L1–5, can be achieved through the anterior approach. Anterior lumbar interbody fusions with or without anterior or posterior instrumentation are the most common indication, usually in the setting of degenerative disease. The exposure is limited by the presence of the great vessels, the descending aorta and inferior vena cava, which bifurcate somewhere between the third and fifth lumbar vertebrae. Retrograde ejaculation is reported in a small percentage of male patients. The approach avoids disruption of the supporting paraspinous musculature, posterior tension band, and ligamentous structures. The L5–S1 interspace is the easiest to access, and in some cases the L4–5 is feasible.

There are wide varieties of techniques used for the anterior approach that are beyond the scope of this chapter, including retroperitoneal and transperitoneal techniques with or without laparoscopic guidance. Detailed descriptions are available in multiple advanced spine textbooks. The basics of the approach include retraction of the rectus to expose the retroperitoneal space. The peritoneal sac is then retracted medically to expose the psoas muscles, great vessels, and anterior spine. Care is taken to protect the ureters, genitofemoral nerves, and iliolumbar vein.

7.3.2 Lateral Approach to the Lumbar Spine

Sometimes referred to as the lateral transpsoas approach, it was described by McAfee for open treatment of thoracolumbar fractures. This approach is amenable to the use of modern tubular retraction systems.[1] Indications include anterior decompressions of the thecal sac and foramen, diskectomy or corpectomy, lateral interbody fusion for lumbar degenerative scoliosis, disk disease, or adjacent-level degeneration beside a prior fusion.

Positioning
After intubation, the patient is rolled into the lateral decubitus position with their trochanter just distal to the break in the table. Bony prominences are well padded with a roll placed under their axilla. Their body is secured to the table with meticulous attention to the skin, especially in elderly patients where skin tears are more common. Tape or belts are used with padding. An alternative is to place the patient on a Wilson Frame flexed and widened to accommodate the patients size. The upper leg is typically flexed at the hip and knee to allow relaxation of the psoas muscle to decrease both muscle damage during the approach and the tension in the lumbar plexus passing through the psoas muscle. The side of approach is determined by orientation of the vertebrae, local deformity, and perhaps site of pathology. The primary importance is adequate access to the desired level. The bed or Wilson Frame is flexed or jackknifed at the mid-torso to open the interval between the ribs and ilium, with caution in patients with osteoporosis or deformities. More challenging levels will require more break in the bed. This technique is usually performed under the supervision of an experienced surgeon.

Landmarks
Fluoroscopic imaging is vital to ensure adequate and accurate trajectory when using tubular retractors from the lateral approach. The iliac wing caudally and ribs are palpated for reference in the context of preoperative imaging to judge the height of incision.

Incision
Incision is carried down to the external oblique muscle fascia, and the muscle is split in line with its fibers. The internal oblique and transversus abdominis muscle layers are split in similar fashion with self-retainers placed. The transversalis fascia is opened with caution to avoid injury to the peritoneum that lies in the abdominal cavity. Many surgeons introduce an instrument or their gloved finger deep to the transversalis fascia though the membrane on top of the finger to protect the bowel contents. Careful blunt dissection through the retroperitoneal fat will then allow

[1] McAfee PC, Bohlman HH, Yuan HA (1985) Anterior decompression of traumatic thoracolumbar fractures with incomplete neurological deficit using a retroperitoneal approach. J Bone Joint Surg Am 67-A(1):89–104

palpation of the posterior wall of the abdominal cavity and spinous transverse processes (TPs). Just anterior to the TPs, the psoas muscle is palpable overlying the vertebral column. The psoas muscle is then split with dilators, and confirmation of disk level is performed with a guide wire or needle using fluoroscopy or X-ray imaging.

The lumbar plexus lies within the psoas muscle and must be avoided during lateral approach to the lumbar spine. The most dangerous level is at L4–5, where the L5 nerve usually passes the disk in the posterior half of the body. Staying in the anterior half of the disk space or carefully sweeping the psoas posteriorly from the 50 % mark is recommended to avoid damage. Neurologic monitoring is used to detect irritation or early injury to the plexus.

Pearls and Pitfalls

Splitting the psoas bluntly with your finger or a peanut prior to using sharp tools in the area may prevent damage to the plexus. Patients with obesity must be counseled that their weight may prohibit safe technique in which case the procedure may have to be abandoned midway through. Patients would subsequently undergo traditional techniques. The ilium blocks lateral access to the L5–S1 level and in some cases the L4–5 level. Preoperative lateral X-rays help predict the option to use this approach at L4–5. Flexibility in the L4–5 interspace can help in borderline cases, but considerable stress is placed on the rim of the L4 vertebrae, so that caution should be used in patients with poor bone stock or osteoporosis.

7.3.3 Posterior Lumbosacral Approach

This is a common approach for treating disorders of the lumbar spine and lumbosacral junction. It is used for posterior decompression of stenosis, diskectomies, posterolateral intertransverse process fusions, posterior instrumentation, and reconstruction or resection of posterior elements including the facet joints. Anterior decompression with or without fusion and more aggressive osteotomies or tumor surgeries can be performed from a posterior approach.

There are two variations commonly used for the posterior approach, the midline approach and the posterolateral muscle splitting, Wiltse approach. With modern tubular retraction systems, it is possible to perform a wide variety of procedures, including but not limited to the simple diskectomy, laminectomy, or more complex fusions, corpectomies and scoliosis reconstructions.

Positioning

After intubation, anesthesia completes their preparation of the patient, possibly including arterial line placement. The surgical team determines necessity of catheterization of the bladder and neurologic monitoring as indicated depending on magnitude of case. Cord level cases typically require neurologic monitoring. After intubation, the patient is rolled prone or in some cases the lateral decubitus position on the operative table. The arms are placed overhead, abducted 90° at the shoulder, and flexed 90° at the elbow. Bony prominences are padded and the face is inspected to ensure there is no pressure on the eyes. The abdomen should be free of compression to reduce intra-abdominal pressure. This is thought to decrease the backfilling of venous blood into the epidural space and Batson's plexus and thus decrease blood loss during the surgery.

Landmarks

The spinous processes lie in the midline. Ribs help identify the thoracolumbar junction, and the iliac wings can be traced out to identify the pelvis. The top of the iliac crest typically lies at approximately L4–5.

Incision

Midline approach: incision is carried down to the spinous processes, with careful attention to hemostasis. The lumbar fascia is incised along the length of the incision, and Cobb Elevators and electrocautery are often used to dissect the paraspinal muscles off the spinous process. Electrocautery and packing techniques help to control bleeding. The extent of exposure is related to procedure. The lamina alone is exposed and stripped of muscle for laminectomies. The facets and transverse processes are exposed during fusion cases, for pedicle-based instrumentation and when anterior structures will be approached via resection of posterior elements, such as interbody fusions, osteotomies, and scoliosis procedures.

When a posterolateral muscle splitting approach is used, the incision is made off the midline with deference to the goals of the procedure. Though there is no firm rule for the distance off the midline for the incision, patient body habitus plays a large role. In general, diskectomy is performed 1 centimeter (cm) off midline, laminectomy 3 cm off midline, and fusion with instrumentation is typically 4 cm off the midline. Imaging is often used in the OR after positioning to confirm orientation of the spine and to better gauge the location of incision. Incision is carried down to the lumbar fascia which is spit in line with the incision. Deep to the fascia, blunt dissection is used to develop the plane between paraspinal muscle groups when approaching the facet joints followed by placement of retractors. Alternatively, a guide wire is placed percutaneously toward the surgical target followed by blunt dissection using dilators. The retractor size is then selected and placed over the dilators. If exposure is not optimal, the retractor can be shifted, or wanded, under direct visualization of the deep anatomy.

Pearls and Pitfalls

Resection or damage of the posterior ligamentous complex and facet joints at adjacent levels is avoided to prevent acceleration of adjacent-level degeneration or deformity. Injection of a dilute epinephrine solution before incision often helps reduce bleeding but should be used with caution under the supervision of an experienced surgeon. Using tubular retractors, the transverse process (TP) can be mistaken for the facet joint when the trajectory is off. This error is avoided by directly palpating the lateral wall of the facet and following it down to confirm the presence of the TP immediately after retractors are placed.

Bibliography

Bohlman HH, Zdeblick TA (1988) Anterior excision of herniated thoracic discs. J Bone Joint Surg Am 70(7):1038–1047

Bohlman H, Eismont F (1981) Surgical techniques of anterior decompression and fusion for spinal cord injuries. Clin Orthop 154:57–73

Brau SA (2003) Anterior retroperitoneal muscle-sparing approach to L2-5 of the lumbar spine. In: Watkins RG (ed) Surgical approaches to the spine, 2nd edn. Springer, New York, p 165

Davis TT, Bae HW, Mok JM, Rasouli A, Delamarter RB (2011) Lumbar plexus anatomy within the psoas muscle: implications for the transpsoas lateral approach to the L4-5 disc. J Bone Joint Surg Am 93:1482–1487

Hodgson AR, Yau ACMC (1964) Anterior surgical approaches to the spinal column. In: Apley AG (ed) Recent advances in orthopaedics. Williams and Wilkins, Baltimore, pp 289–323

Hodgson AR (1965) Approach to the cervical spine C3-7. Clin Orthop 39:129–134

Jeanneret B, Jeanneret B (2003) Posterior approach to the C1-2 joints. In: Watkins RG (ed) Surgical approaches to the spine, 2nd edn., pp 224–236

Magerl F, Seemann P-S (1987) Stable posterior fusion of the atlas and axis by transarticular screw fixation. In: Kehr P, Weidner A (eds) Cervical spine, Ith edn. Springer, Wien

Ozgur BM, Aryan HE, Pimenta L, Taylor WR (2006) Extreme lateral interbody fusion (XLIF): a novel surgical technique for anterior lumbar interbody fusion. Spine J 6:435–443

Perry J (1977) Surgical approaches to the spine. In: Pierce D, Nochol V (eds) The total care of spinal cord injuries. Little, Brown, Boston, pp 53–81

Robinson RA (1978) Chapter 22: Approaches to the cervical spine C1-T1. In: Schmidek HH, Sweet WH (eds) Current techniques in operative neurosurgery. Grune and Stratton, New York, pp 205–302

Watkins RG (2003a) Thoracotomy approach. In: Watkins RG (ed) Surgical approaches to the spine, 2nd edn. Springer, New York, pp 113–123

Watkins RG (2003b) Approach to the posterior aspect of C1-2. In: Watkins RG (ed) Surgical approaches to the spine, 2nd edn. Springer, New York, pp 217–223

Watkins RG III, Watkins RG IV (2003) Bilateral paraspinous lumbosacral approach. In: Watkins RG (ed) Surgical approaches to the spine, 2nd edn. Springer, New York, p 326

Wiltse LL, Bateman JG, Hutchinson RH, Nelson WE (1963) Paraspinal muscle approach to the lumbar spine. J Bone Joint Surg Am 50:919

Wiltse LL (1973) The paraspinal muscle splitting approach to the lumbar spine. Clin Orthop 91:48–57

Young PH (2003) Posterior and posterolateral approaches to the thoracic disc. In: Watkins RG (ed) Surgical approaches to the spine, 2nd edn. Springer, New York

8 Cervical and Cervicothoracic Instrumentation

Adam Pearson and Todd Albert

8.1 Case Example

A 23-year-old male suffered a C4–5 bilateral facet dislocation in a diving accident and presents with a complete spinal cord injury with no function below C5 (Fig. 8.1a). Closed reduction is attempted but is not successful. A preoperative MRI reveals herniated disk material at C4–5. He is taken to the operating room for anterior cervical diskectomy, followed by posterior reduction and fixation with lateral mass screws and rods and finally anterior fusion with a plate and fibular allograft (Fig. 8.1b). This represents a very strong biomechanical construct that is necessary given the complete ligamentous disruption and severe instability resulting from the injury.

8.2 Anterior Cervical Plating

Anterior cervical plates are the most common form of instrumentation used in the cervical spine, and their use has increased in the past two decades [1]. They have evolved from general trauma plates designed for the extremities to low-profile, contoured plates designed for the cervical spine. They now include both rigid and dynamic devices.

8.2.1 Indications

Anterior cervical plates are used to improve the stability of the cervical spine and increase the likelihood of fusion following diskectomy or corpectomy in the setting of degeneration, trauma, infection, or tumor. The most common use of plating is with anterior cervical diskectomy and fusion (ACDF) or corpectomy. The use of a plate in single-level ACDF is controversial as plating offers only a slight improvement in the fusion rate—from 92 to 97 % according to a recent meta-analysis [2, 3]. Proponents of plating cite the advantages of being able to use allograft and avoid the use of rigid cervical collars when a plate is used [4, 5]. Less controversial is the use of a plate for corpectomy or multilevel ACDF, with the addition of a plate resulting in a significant improvement in the fusion rate (i.e., from 80 to 95 % for two-level ACDF) [2]. Plating is also used regularly in cases of trauma if an anterior or anterior-posterior approach is used.

8.2.2 Relevant Anatomy and Technique

A standard anterior approach to the cervical spine is used to expose the vertebrae to be fused (see Chap. 7). Depending on the number of levels to be

A. Pearson, M.D., M.S.
Department of Orthopaedics,
The Geisel School of Medicine at Dartmouth,
Dartmouth-Hitchcock Medical Center,
1 Medical Center Dr, Lebanon, NH 03756, USA
e-mail: adam.pearson@hitchcock.org

T. Albert, M.D. (✉)
Department of Orthopaedics, The Rothman Institute,
Thomas Jefferson University and Hospitals,
925 Chestnut Street, Philadelphia, PA 19107, USA
e-mail: tjsurg@aol.com

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_8, © Springer-Verlag Berlin Heidelberg 2014

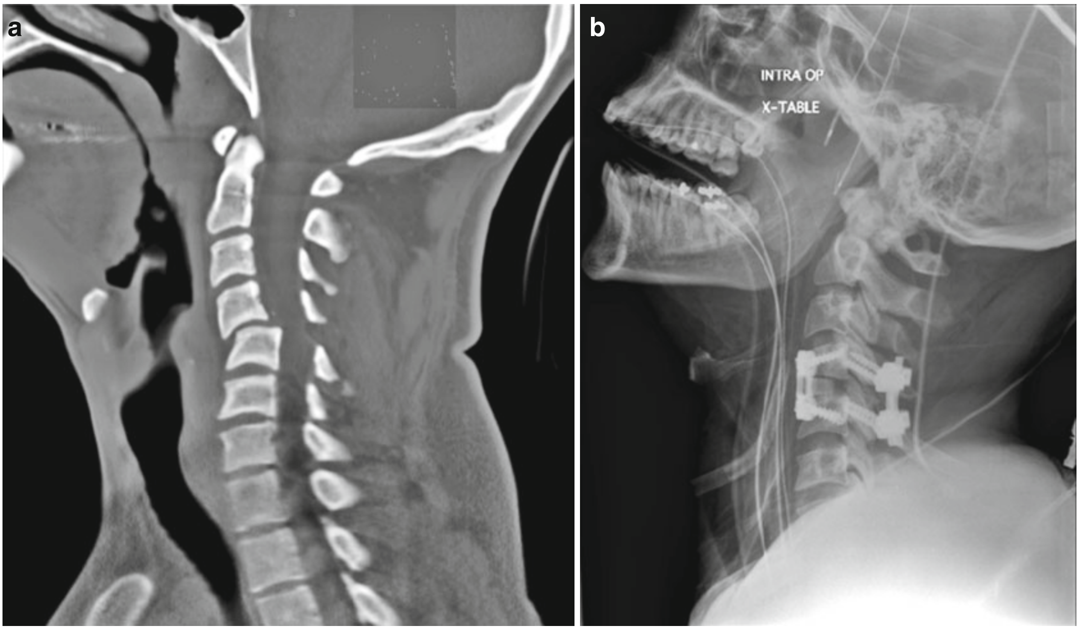


Fig. 8.1 (**a**) C4–5 bilateral facet dislocation. (**b**) Postoperative radiograph demonstrating anterior fixation with a plate and allograft fibula and posterior fixation with lateral mass screws and rods

addressed, either a transverse (usually an acceptable option for three or fewer levels) or oblique longitudinal incision along the medial border of the sternocleidomastoid can be used. The anatomy of the vertebral bodies and disk spaces needs to be considered when placing a cervical plate. The disk spaces are oriented from anteroinferiorly to posterosuperiorly in the sagittal plane, so screw placement must take this into account. The screws going into the superior vertebra must be aimed in a more superior direction, or they are at risk of violating the disk space. The screws in the inferior vertebra can be aimed straight ahead or slightly inferiorly depending on the selected starting point. In general, the shortest plate available should be selected such that the starting points for the screws are adjacent to the disk space as it has been shown that adjacent-level ossification is more common if the plate is within 5 mm of the adjacent disk [6]. Screws should also be aimed slightly medially in order to improve pullout strength through triangulation. Vertebral bodies tend to range from 14 to 20 mm deep in the sagittal plane (i.e., from anterior to posterior), so many surgeons will use a 14-mm screw by default unless the vertebral body appears abnormally small. In general, unicortical fixation is sufficient with modern locking plates; however, bicortical fixation can be considered in cases of osteoporotic bone [7]. Obviously, great care must be taken to avoid injury to the neural elements if bicortical fixation is attempted. Most plating systems are designed with a hand or power drill used to create a pilot hole that can be filled with a self-tapping screw. Attention should be paid to making sure the plate and anterior aspects of the vertebral bodies are contoured appropriately so that the plate is not prominent as this can cause dysphagia. Finally, the plate should be centered on the vertebral bodies in the coronal plane, and the uncovertebral joints, sternal notch, and jaw can be used as landmarks to judge alignment.

8.2.3 Plate Designs

First-generation cervical plates were “unlocked,” meaning that the screws were not fixed to the plate and could toggle within the plate. A major advance was the development of locking plates, in which the screws locked into the plates, creating a fixed-angle device that prevented toggle and backing out of the screws. Biomechanical studies have demonstrated

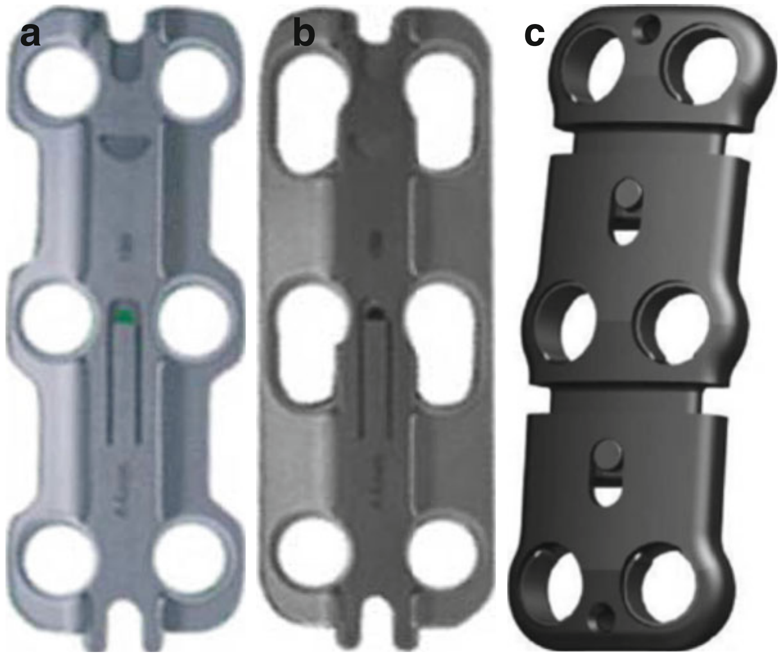


Fig. 8.2 (**a**) Dynamic plate that allows for screw toggle. (**b**) Translational plate that allows for screw translation in a slotted hole. (**c**) Telescoping plate with rigidly fixed screws that allows for shortening of the plate itself

that locked plates maintain rigidity better than unlocked plates during cyclic loading [8]. Rather than loosening, locked constructs fail more frequently with screw or plate breakage. A concern with rigid, locked plates, however, is that they do not allow for load sharing with the graft, which could theoretically result in pseudarthrosis, especially if there is loss of bone graft height [9]. To address this concern, dynamic plates have been developed to allow for graft load sharing and possible subsidence yet prevent screw back out. Three main designs exist: plates that allow for screws to toggle as the graft settles, translational plates that allow for the screws to translate in slotted holes with the vertebra as subsidence occurs, and telescoping plates in which the screws are rigidly locked to the plate and the plate itself shortens (Fig. 8.2a–c) [10]. All three designs should theoretically allow for graft subsidence and better load sharing, yet each has its own limitations. Not surprisingly, plates that allow for screw toggle may be associated with a higher rate of failure at the screw-bone interface, which could lead to nonunion. If subsidence occurs with translational plates that allow for vertical screw translation, the ends of the plate will move closer to the adjacent, unfused disks, raising concerns about adjacent-level ossification [6]. Telescoping plates do not have either of these problems, but they tend to be thicker and actually less rigid due to the telescoping mechanism [11].

Despite the theoretical advantages of dynamic plates, it remains unclear if they lead to measurably better clinical or radiographic outcomes. In the largest randomized trial comparing dynamic and rigid plates, the authors found no significant differences in fusion rates or clinical outcomes at 2 years for patients who underwent one- or two-level ACDF [12]. They did report significantly more implant-related complications for the rigid plate compared to the dynamic plate, yet more loss of lordosis in the dynamic plate group. Similar to this study, a smaller randomized trial comparing dynamic to rigid plates found no clinical or radiographic differences overall but suggested a trend toward better clinical outcomes in the dynamic plate group [13]. At this point, it remains unclear if dynamic plates offer an advantage over rigid plates or if there are specific situations in which they are favored.

8.2.4 Complications

The most common complication following ACDF with plating is dysphagia, which occurs in up to 60 % of patients in the short term, though it is unclear if the addition of a plate actually contributes to it [14]. Voice difficulties are another common complaint, reported by nearly 20 % of patients at long-term follow-up [15]. Neurological, vascular, and esophageal injuries are rare. As discussed above, the addition of a plate to a corpectomy or multilevel ACDF helps to reduce the rate of pseudarthrosis significantly. Implant failures with modern instrumentation occur in approximately 3 % of cases and are associated with osteoporosis and technical errors [16].

8.3 Anterior Cervical Cages

Traditionally, iliac crest autograft was used to fill the disk space following ACDF. However, concerns over harvest site complications and persistent pain have prompted surgeons to evaluate other options [17]. Allograft is a popular option in countries where it can be easily obtained, though it is associated with a small but real risk of disease transmission. Allograft is not available in all nations, so this has stimulated the development of anterior cervical cages that can be used instead of autograft or allograft. Information on bone graft and bone graft substitutes can be found in Chap. 10.

8.3.1 Indications

Cervical cages can be used in degenerative, traumatic, and neoplastic cases when reconstruction of the anterior column is necessary. They can be used in the same fashion as allograft or autograft in ACDF or corpectomy, though it is unclear if they offer any advantage over allograft for these applications, and they tend to cost more. Use of cages in the setting of infection is controversial. Traditionally, autograft has been favored in osteomyelitis due to its resistance to biofilm formation. However, successful use of synthetic cages for reconstruction following corpectomy for osteomyelitis and epidural abscess has been reported [18].

8.3.2 Relevant Anatomy and Technique

Cages rest against the bony end plates of the adjacent vertebrae, so preservation of the end plate is essential to prevent subsidence. On the other hand, fusion can only occur at bone surfaces, so the disk material and cartilaginous end plate overlying the subchondral bony end plate must be completely removed. This is generally performed with careful use of curettes and the high-speed burr. Another important technical issue is achieving the appropriate tightness of fit of the cage. Undersizing the cage in the vertical direction results in a loose fit and the possibility of migration, especially if a plate is not used. Overstuffing the space results in high forces at the end plates and increases the risk of subsidence. Additionally, it may also reduce motion at adjacent levels [19]. Maximizing the footprint of the cage is also important to prevent subsidence as this helps to distribute the load over a greater area.

8.3.3 Cage Designs

Cages can be designed to fill large vertical gaps resulting from corpectomy or the horizontal space following diskectomy. Vertical cages are typically made of titanium, with a mesh being the classic design (Fig. 8.3). Expandable vertical cages are also available and have the advantage of being adjustable to the height of the corpectomy defect [20]. Horizontally oriented cages are used to fill the disk space following diskectomy and have been designed with screw-in, cylindrical mesh, and box-shaped designs. Cages were traditionally made of titanium; however, metal cages reduce the image quality on CT scan and MRI. As a result, carbon fiber and polyether ether ketone (PEEK) cages have been developed. Hybrid devices that incorporate a cage with a plate or screws are the latest development in cage design and provide reconstruction and stabilization in one device [21]. The superiority of one design over another or an advantage over allograft has not been demonstrated clinically [22]. In places

Fig. 8.3 Titanium mesh cage used for anterior reconstruction after corpectomy

where allograft is available, cages are unlikely to be widely adopted for straightforward degenerative conditions until it is demonstrated that they offer an advantage that justifies their cost.

8.3.4 Complications

The most common complications related to cages—and allograft or autograft—are subsidence and pseudarthrosis. Most patients with subsidence remain asymptomatic as long as fusion occurs, though foraminal stenosis and recurrent radiculopathy can occur [23]. Most studies comparing cages to autograft and allograft have found similar rates of pseudarthrosis [22]. More catastrophic complications such as extrusion, cage breakage, and neurological injury are fortunately rare.

8.4 Occipitocervical Instrumentation

Treatment of occipitocervical instability—either congenital, inflammatory, or traumatic—traditionally involved fusion using semirigid wiring techniques. These approaches required the long-term use of a halo postoperatively, accompanied by the complications associated with halo use. Rigid modern instrumentation has been developed in order to increase fusion rates and avoid the complications associated with halo use.

8.4.1 Indications

Occipitocervical fusion is indicated in cases of occipitocervical instability. Down syndrome is associated with less curvature of the occipital condyle, and this can result in congenital instability [24]. Rheumatoid arthritis can lead to atlantoaxial instability with basilar invagination of the dens, and this typically requires C1 laminectomy with occipitocervical fusion. Traumatic occipitocervical dislocation is often fatal and is treated with occipitocervical fusion in surviving patients.

8.4.2 Relevant Anatomy and Technique

Understanding occipital bony anatomy is essential to safely placing occipital screws. The occiput is thickest at the external occipital protuberance (EOP), but the confluence of the transverse sinuses lies anterior to the inner cortex at this level. Going laterally from the EOP is the superior nuchal line, which overlies the transverse sinuses. As such, screws must be placed caudal to the EOP. In the medial-lateral direction, the bone is thickest in the midline and tapers out laterally. At the EOP in the midline, the bone is typically 10–15 mm thick, decreasing to 6–7 mm in the midline 3 cm below the EOP [25].

To place occipital instrumentation, the occiput must be exposed from the EOP to the posterior atlantooccipital membrane. Fixation devices come in a variety of designs (see Sect. 8.5.3), but all modern instruments are attached to the occiput using screws. Screws should be placed close to the midline and the EOP (but not above it due to the risk of injuring the transverse sinus) in order to maximize purchase in the thicker bone found superior and toward the midline. Most plates also have holes slightly off midline in order to provide for rotational control. Screws should be bicortical as this increases pullout strength by 50 % [26]. In general, holes should be created by starting with a drill set at 6–8 mm and gradually increasing depth until the inner cortex is breached. Most screws are in the 6–14-mm range. If a spinal fluid leak occurs, it is simply treated by placing the screw. Following placement of the occipital plate or screws, rods are contoured to allow connection to the cervical fixation.

8.4.3 Construct Designs

Recently there has been significant progress in the development of more rigid and user-friendly occipitocervical instrumentation. Initial techniques for occipitocervical fixation involved wires passed through burr holes and attached to the cervical spine. The first plating techniques used pelvic reconstruction plates contoured to allow for the placement of lateral mass screws and occipital screws and were obviously challenging to use. More recently, occipital plates have been developed. Initially, these plates were designed to be placed bilaterally on the occiput and could be connected to the cervical spine with rods. The latest-generation occipital plates include midline and paramedian screw holes in one plate (Fig. 8.4). These plates include polyaxial rod connectors, allowing for easier connection to the cervical spine. Since rod contouring can be difficult at the occipitocervical junction, hinged rods have been developed.

Fig. 8.4 Occipital plate with polyaxial rod connectors

8.4.4 Complications

Medical complications following occipitocervical fusion are common in the trauma, cancer, and rheumatoid patients. Skin breakdown and subsequent infection is not uncommon, in part due to the prominent occipital hardware, occurring 5 % of the time in one study [27]. Fusion rates are typically over 90 % if autograft is used, though a high rate of fusion failure has been reported with allograft [28]. Not surprisingly, patients typically report a noticeable loss of cervical motion. Complications related to occipital screw placement include cerebrospinal fluid leak and injury to the venous sinuses. The former typically causes no problems if a screw is placed to block egress of the fluid. Venous sinus injury is also treated by screw placement followed by anticoagulation to prevent thrombosis. Neurological injury related to wire placement has been reported, though is uncommon with screw-based constructs.

8.5 Atlantoaxial Instrumentation

Atlantoaxial fixation techniques include wiring, transarticular screw placement, and screw-rod constructs. The unique anatomy of this portion of the cervical spine creates technical challenges for fixation and places neurovascular structures at risk. A thorough understanding of this anatomy and the use of intraoperative imaging is essential to the safe placement of hardware.

8.5.1 Indications

Atlantoaxial instability can result from congenital, traumatic, neoplastic, and inflammatory conditions. Up to 40 % of Down syndrome patients have radiographic evidence of atlantoaxial instability [29]. Dens fractures or transverse ligament ruptures can lead to traumatic instability. Displaced type II dens fractures (involving the base of the dens) should be treated surgically, while the treatment of non-displaced type II dens fractures, particularly in the elderly, is controversial. Rheumatoid arthritis can lead to erosive changes, pannus formation, and destruction of the ligamentous structures that provide stability to the atlantoaxial articulation. Traditional indications for surgical stabilization in rheumatoid patients include an anterior atlantodental interval (AADI) of greater than 9 mm and a posterior atlantodental interval (PADI) less than 14 mm. Cranial settling requires an occipitocervical fusion.

8.5.2 Relevant Anatomy

The unique anatomy of the upper cervical spine needs to be carefully considered when planning C1–2 fixation. Preoperative CT scan and MRI are essential to surgical planning. The atlas (C1) is a ring with relatively flat superior and inferior articular facets allowing for flexion and extension at the atlantooccipital joint and axial rotation at the atlantoaxial joint. The axis (C2) is unique given the presence of the odontoid and its articulation with C1 that is significantly more anterior than the subaxial facet joints. The vertebral artery (VA) is the structure most at risk with C1 and C2 instrumentation. It courses through the transverse foramina of C2 and C1 before going posteriorly and medially along the cranial surface of the C1 ring. It then enters the spinal canal and runs anteriorly to join the contralateral VA anterior to the medulla, forming the basilar artery. When exposing the posterior arch of C1, sharp instruments and Bovie electrocautery should not be used along the superior surface of the ring more than 15 mm lateral to midline in order to avoid VA injury. The VA can also take an anomalous interosseous course within the posterior C1 ring, putting it at risk for injury. The bony ridge forming the posterior aspect of this arcuate foramen is referred to as the "ponticulus-posticus" and has been mistaken for the starting point for a C1 lateral mass screw, resulting in VA injury [30]. The C1 and C2 nerves are unique in that they exit posterior to the C0–1 and C1–2 articulations (subaxial nerves exit anterior to the facet joints). The C2 nerve and a surrounding venous plexus is located posterior to the lateral mass of C1, putting these structures at risk during exposure of the C1 lateral mass. Bleeding from the plexus can be minimized with careful subperiosteal dissection.

8.5.3 Fixation Techniques

Gallie and Brooks wiring were the traditional methods of C1–2 stabilization. Gallie wiring involves passing sublaminar C1 wires that are looped around the C2 spinous process following placement of a structural bone graft. Brooks wiring consists of passing two sublaminar wires at C1 and C2 that are used to secure bilateral bone blocks. While the Brooks technique provides

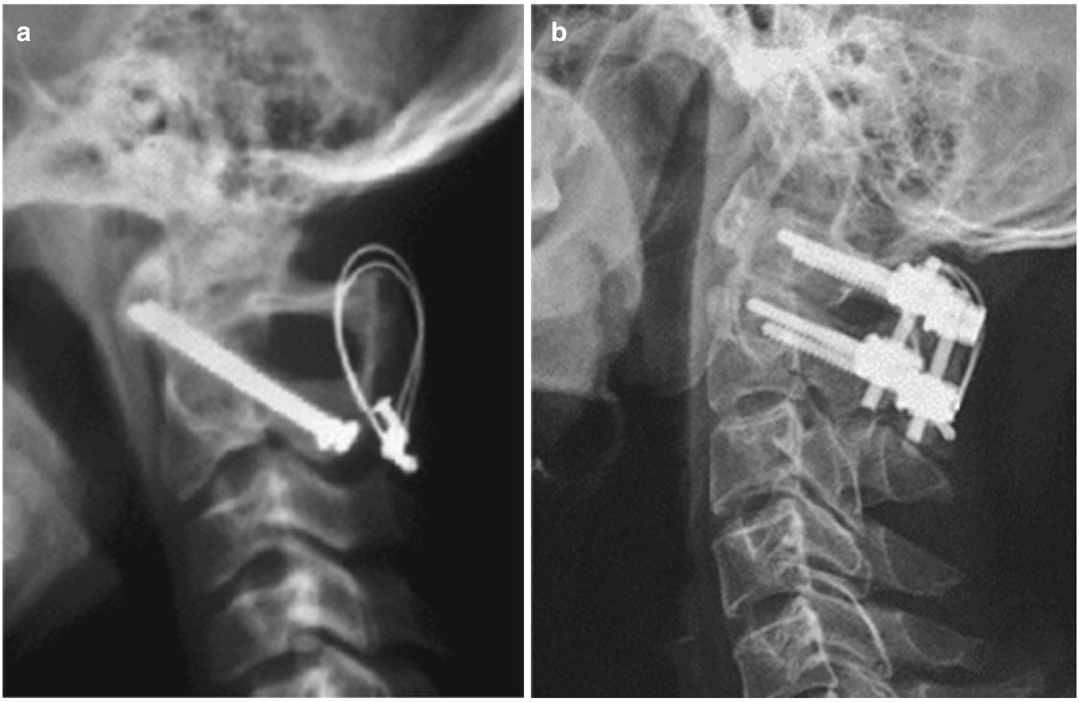


Fig. 8.5 (**a**) Transarticular C1–2 fusion. (**b**) Harms technique of C1–2 fusion. Both constructs shown also include Gallie wiring

greater stability in extension and axial rotation—the Gallie technique essentially resists only flexion—postoperative halo immobilization is still required. For this reason, more rigid fixation techniques were developed.

Magerl first described C1–2 transarticular screws in 1987 (Fig. 8.5a) [31]. This technique involves placing screws across the C1–2 joint. The starting point is just superior to the C2–3 joint, with a steep cranial trajectory necessary in order to traverse the C1–2 articulation. In some cases, a percutaneous drilling technique with a stab incision at the cervicothoracic junction is necessary given the steep angle required. The medial border of the C2 isthmus/pedicle is an essential landmark to guide medial-lateral angulation. In general, the screw trajectory is up to 25° toward the midline in order to avoid the vertebral artery laterally. K-wires are typically placed under mono- or biplanar fluoroscopy prior to drilling. Unicortical fixation in C1 is generally acceptable, especially given the risk of injury to the internal carotid artery anterior to the C1 ring. Some authors have advocated decortication of the C1–2 joint to promote fusion, though this is usually associated with bleeding from the overlying venous plexus. Magerl's initial description also included a Gallie fusion, though high fusion rates have been reported without this [32].

Due to the risk of VA injury and the difficult trajectories associated with transarticular screws, Harms developed a C1–2 fixation technique using C1 lateral mass screws and C2 pedicle screws (Fig. 8.5b) [33]. As discussed above, care must be taken while dissecting out the C1 lateral mass in order to avoid injury to the VA on the superior aspect of the ring and the venous plexus surrounding the C2 nerve that overlies the C1–2 articulation. After exposing the C1 lateral mass, a starting point is selected in the midpoint, and the lateral mass is drilled with 10–15° of medialization and approximately 20° of superior angulation. Fluoroscopy is typically used to guide placement. Some authors advocate bicortical fixation, though this puts the internal carotid artery at risk of injury with anterior perforation. Ideally, a partially threaded screw should be placed to prevent thread forms from contacting the C2 nerve.

Multiple screw techniques have been described for C2 fixation, including pars, pedicle, and translaminar screws. Most descriptions of C2 pedicle screw placement describe techniques which in fact traverse the pars, making the distinction between these techniques somewhat vague [34]. Pars screws typically have a starting point approximately 5 mm caudal to the superior edge of the lamina and 7 mm lateral to the medial border of the pars [35]. They are angulated approximately 10–20° medially and 20–30° superiorly. It has been shown that the most reliable placement method involves entering the spinal canal in the C1–2 interspace and palpating the medial and superior border of the pars to guide screw trajectory. Using a relatively medial and superior starting point has been recommended to avoid lateral violation and VA injury. True pedicle screws require substantial medialization and a more lateral starting point in order to place the screws into the small vertebral body of C2. Due to concerns about VA injury with C2 pars/pedicle screws, a translaminar screw placement technique was developed [36]. To place translaminar screws, a starting point at the base of the spinous process is selected, and screws are placed into the contralateral lamina between the anterior and posterior cortices. If bilateral translaminar screws are being placed, consideration of their trajectories must be considered in order to assure there is sufficient space for both screws. Translaminar screws are a good alternative if anatomical constraints limit pars/pedicle screw placement or if a safe bailout option is needed following VA injury. Biomechanical evaluation of C1–2 constructs demonstrated that pedicle screws provided the stiffest constructs with the highest pullout forces, followed by translaminar and pars screw [37].

Following placement of C1 and C2 screws, the polyaxial heads are connected with rigid rods. Typically, a structural iliac crest autograft is harvested and contoured to overlay the posterior C1 ring and C2 lamina and spinous process, and suture can be used to hold it in place. Decortication of the C1–2 facet joint is possible, though it puts the C2 nerve and surrounding venous plexus at risk, and fusion rates are high with the onlay autograft described.

8.5.4 Complications

Vertebral artery injury is a feared complication of C1–2 fusion, though its occurrence has been found to be under 2 % [38]. If VA injury occurs with bleeding from a screw hole, placement of the screw typically tamponades the bleeding. Placement of screws on the contralateral side should be abandoned, with the exception of translaminar screws that do not put the VA at risk. Uncontained bleeding (i.e., extraosseous) can be more difficult to control and occasionally requires embolization. Cerebrovascular accident is possible but uncommon due to the substantial collateral flow to the brain. Neurological injury from aberrant screw placement is also rare, though it can occur with sublaminar wiring techniques. With the use of autograft, C1–2 fusion rates approach 100 % [39]. Similar to any posterior cervical procedure, infection is the most common complication, with rates likely approaching 5 % [39].

8.6 Subaxial Cervical Instrumentation

Similar to the adoption of rigid instrumentation for the upper cervical spine, rigid implants for the subaxial cervical spine have largely replaced traditional wiring techniques. Lateral mass screws have become the standard for fixation from C3 to C6 due to their ease of insertion, safety, and effectiveness. Subaxial cervical pedicle screws have also been evaluated, though most authors have abandoned them other than for fixation at C7 due to the high risk of neurological or VA injury.

8.6.1 Indications

Subaxial instrumentation and fusion is used in cases of instability in congenital, traumatic, neoplastic, degenerative, and inflammatory conditions. Flexion-distraction injuries (i.e., facet subluxations and dislocations) can be treated with anterior, posterior, or circumferential techniques. Biomechanical investigation has demonstrated that posterior and circumferential fixation is stiffer than anterior plating when a flexion moment is applied, though clinical results were similar for anterior and posterior constructs [40, 41]. Following cervical laminectomy for multilevel spondylosis with myelopathy, many surgeons favor fusion to prevent post-laminectomy kyphosis. Posterior fixation can also improve fusion rates and obviate the need for a plate in multilevel ACDF or multilevel corpectomy.

8.6.2 Relevant Anatomy and Techniques

Modern subaxial fixation techniques include lateral mass and pedicle screw fixation. Due to the ease and safety of lateral mass fixation, this tends to be favored from C3 to C6. Pedicle screw fixation is biomechanically superior but puts the neural structures and VA at risk. As such, pedicle screw fixation is typically used only at C7 due to the absence of an intraforaminal VA in most patients at this level, the larger size of the C7 pedicle, and the smaller C7 lateral mass. The lateral mass is a rectangular-shaped structure that lies between the superior and inferior facets. Anteromedially, it is confluent with the pedicle and the transverse foramen containing the VA lies anterior to its medial border. Directly anterior is the exiting nerve root. This anatomy must be taken into account when placing lateral mass screws.

Multiple techniques of lateral mass screw placement have been described, with different anatomic structures at risk depending on the technique used [42]. Roy-Camille developed a technique in which the starting point was the center of the lateral mass and the trajectory was perpendicular to the lateral mass and 10° lateral to prevent VA injury [43]. This technique tends to put the exiting ventral ramus at risk if an overly long screw is used and also puts the inferior facet joint at risk of violation. Given these concerns, Magerl described a technique in which the starting point is 1–2 mm medial and superior to the center of the lateral mass, with the trajectory 25° lateral and parallel to the facet joint (approximately 45° cephalad). This avoids violation of the inferior facet joint and injury to the ventral ramus but puts the smaller dorsal ramus at risk. An proposed a slight modification of this technique with a starting point 1 mm medial to the midpoint of the lateral mass and a trajectory 30° lateral and 15° cephalad [44]. Anterior violation with this technique puts the ventral ramus at risk. Biomechanical studies have mixed results, with some showing advantages for the Magerl technique [45, 46]. Most surgeons favor the Magerl technique in order to avoid violation of the inferior facet joint. Some surgeons use bicortical fixation as it increases the pullout strength approximately 20 %, though this marginal benefit comes with an increased risk of nerve root irritation [47].

Cervical pedicle screw fixation provides biomechanically superior fixation compared to lateral mass screws, though it is technically much more challenging and puts the VA, spinal cord, and nerve roots at greater risk [48]. Cervical pedicles are bordered by nerve roots cranially and caudally, the spinal canal medially, and the VA laterally. The typical starting point is just lateral to the center of the lateral mass and just below the inferior articular process of the cranial vertebra with a trajectory approximately 45° medial and perpendicular to the lateral mass. However, there is substantial variation in pedicle anatomy, and other techniques such as fluoroscopy and/or palpation of the pedicle following laminoforaminotomy are recommended. As mentioned, most authors suggest subaxial cervical pedicle screws be used only at C7 due to concern about VA injury from C3 to C6. A popular construct used to stabilize a multilevel cervical laminectomy involves lateral mass screw fixation from C3 to C5 with pedicle screws at C7 (Fig. 8.6). Lateral mass screws are often left out at C6 due to difficulty aligning them with C7 pedicle screws and to allow a larger bed for fusion material.

8.6.3 Subaxial Instrumentation

Historically, spinous process wiring techniques were developed to provide stability of the subaxial cervical spine and increase fusion rates.

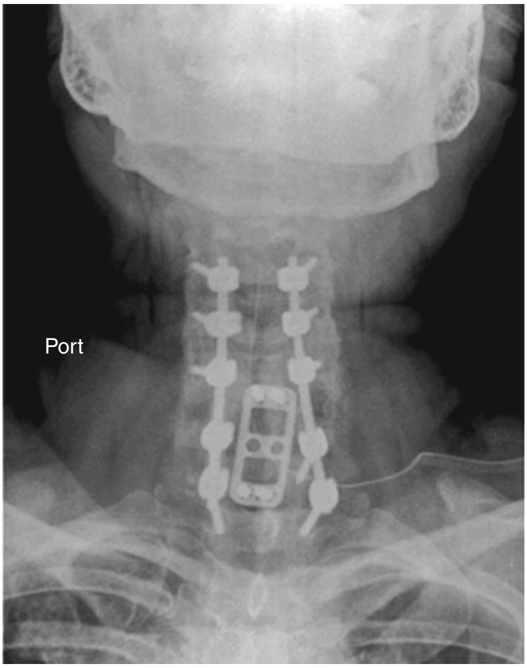


Fig. 8.6 AP radiograph demonstrating a C3–T1 posterior construct with lateral mass screws from C3 to C5 and pedicle screws at C7 and T1. A C5–7 anterior plate was present from a prior operation

Sublaminar wiring was essentially abandoned in the subaxial region due to the risk of spinal cord injury given the smaller canal diameter caudal to C2. More rigid techniques were developed that initially involved lateral mass screws placed through pelvic reconstruction plates; however, there was often a mismatch between the location of the holes and optimal starting points for the screws. The latest generation of instrumentation includes screws with polyaxial heads that can be fixed to contoured rods. Typically, 3.5-mm screws are used in cervical lateral masses and pedicles, with 4-mm screws available as "rescue" screws if the 3.5-mm screws fail to gain purchase. These systems typically allow for connection to occipital plates allowing for occipitocervical fixation.

8.6.4 Complications

Lateral mass screws are relatively safe to insert and are associated with a low complication rate. Injury to the VA has been reported in 1.3 % of cases using the Magerl technique [38]. Clinically significant nerve root injury is quite rare. Radiculopathy can develop if a rod is undercontoured and the foramen is narrowed during reduction of the screw to the rod. Failure of rigid posterior fixation is also uncommon, though this can be seen at the caudal end of the construct with pseudarthrosis or if there is inadequate anterior column support. Cervical pedicle screw placement from C3 to C6 is associated with a higher rate of complications, including vertebral artery and nerve root injury. In one study, 30 % of pedicle screws violated the pedicle, and 5 % had significant (>25 %) transverse foramen violations [49]. When placing C7 pedicle screws, the preoperative MRI should be evaluated to ensure the transverse foramen (if present) does not contain the VA.

8.7 Cervicothoracic Instrumentation

Crossing the cervicothoracic junction with rigid instrumentation is challenging given the marked anatomical differences between the smaller, mobile cervical vertebrae and the larger, less mobile thoracic vertebrae. Due to the unique transitional anatomy, instrumentation techniques have been developed to allow for fixation across the junction.

8.7.1 Indications

The treatment of congenital, traumatic, degenerative, infectious, and neoplastic conditions of the cervical and upper thoracic spine can require stabilization across the cervicothoracic junction as can cervicothoracic deformity. Cervical kyphosis frequently requires lower cervical osteotomy that is stabilized with instrumentation into the upper thoracic spine. Cervicothoracic scoliosis, though uncommon, can also require cervicothoracic fusion. Traumatic instability or instability created by resection of infection or tumor can also require cervicothoracic stabilization. Some surgeons prefer to treat multilevel cervical stenosis with laminectomy and fusion from C3 to T1 to prevent instability at the cervicothoracic junction that can result from a fusion construct ending at C7.

8.7.2 Relevant Anatomy and Techniques

The cervicothoracic junction is unique in that there is a transition from the highly mobile cervical spine to the relatively rigid thoracic spine. The lateral mass of C7 is quite narrow in the anterior-posterior dimension, making lateral mass fixation at this level unreliable. The C7 pedicle is usually relatively large, and the VA is only rarely in the transverse foramen at this level, so C7 pedicle screws are relatively safe to place. The starting point is generally just lateral to the midpoint of the lateral mass and just inferior to the inferior articular process of C6 with a trajectory of approximately 30° medial in the axial plane and perpendicular to the lateral mass in the sagittal plane. In the thoracic spine, pedicle fixation is favored. The starting point for the T1 to T3 pedicles in the cephalad-caudad direction is at the midpoint of the transverse process and in the medial-lateral direction at the junction of the transverse process and lamina. The trajectory is about 20–30° medial in the axial plane and perpendicular to the lamina in the sagittal plane. Given the difficulty of using fluoroscopy at the cervicothoracic junction, laminoforaminotomies can be created to allow direct palpation of the pedicle to help guide screw placement.

8.7.3 Construct Designs

Instrumentation crossing the cervicothoracic junction needs to accommodate the transition from relatively small (3.5 mm) lateral mass and cervical pedicle screws to larger thoracic instrumentation. In cases where instrumentation stops at T1 or T2, 3.5-mm screws can be used throughout the construct, and no special accommodations are needed. However, larger screws and rods are generally used more caudally, and transitional hardware is needed. Rod-to-rod connectors are available to allow for the connection of 3.5-mm rods in the cervical spine to 5.5-mm rods in the thoracic spine. Another option is the use of a transitional rod that tapers from 3.5 mm cranially to 5.5 mm caudally. Comparison of construct stiffness provided by using a 3.5-mm rod throughout the construct, a rod-to-rod connector, and a transitional rod showed that there were no significant differences among the constructs [50]. The use of rod-to-rod connectors requires planning of screw placement so that the rods have the appropriate relationship to each other to allow for placement of the connector. Similarly, transitional rods require planning of screw placement to ensure that a screw head will not be at the tapered portion of the rod.

8.7.4 Complications

Obtaining fusion across the cervicothoracic junction can be challenging due to the high stresses at this region. As such, hardware failure including screw breakage and pullout can occur at the caudal end of the construct, especially if the construct ends at T1. Pedicle screw placement at C7 and in the thoracic spine is associated with a low rate of neurological injury due to pedicle violation and contact with the spinal cord or nerve roots. Lower in the thoracic spine, anterior and lateral screw violations can result in aortic injury.

Questions

1. Use of an anterior cervical plate is not required for:
   (a) Single-level anterior cervical diskectomy and fusion (ACDF)
   (b) Single-level corpectomy
   (c) Multilevel ACDF
   (d) Multilevel corpectomy
   Preferred response: (a) Fusion rates are only marginally improved by using a plate for single-level ACDF. Fusion rates are markedly improved with plating for the other procedures.

2. Placement of occipital screws superior to the external occipital prominence (EOP) puts what structure at risk?
   (a) Spinal cord
   (b) Transverse sinus
   (c) Vertebral artery
   (d) Brainstem
   Preferred Response: (b) The transverse sinus lies just superior to the EOP and is at risk if bicortical screws are placed superior to the EOP.
3. Pedicle screw fixation is preferred at what level in the cervical spine?
   (a) C1
   (b) C3
   (c) C6
   (d) C7
   Preferred Response: (d) Pedicle screws are typically recommended at C2 and C7. They are associated with a high risk of neurovascular injury from C3 to C6.

References

1. Zeidman SM, Ducker TB, Raycroft J (1997) Trends and complications in cervical spine surgery: 1989–1993. J Spinal Disord 10:523–526
2. Fraser JF, Hartl R (2007) Anterior approaches to fusion of the cervical spine: a metaanalysis of fusion rates. J Neurosurg Spine 6:298–303
3. Wang JC, McDonough PW, Endow K, Kanim LE, Delamarter RB (1999) The effect of cervical plating on single-level anterior cervical discectomy and fusion. J Spinal Disord 12:467–471
4. Kaiser MG, Haid RW Jr, Subach BR, Barnes B, Rodts GE Jr (2002) Anterior cervical plating enhances arthrodesis after discectomy and fusion with cortical allograft. Neurosurgery 50:229–236; discussion 36–38
5. Campbell MJ, Carreon LY, Traynelis V, Anderson PA (2009) Use of cervical collar after single-level anterior cervical fusion with plate: is it necessary? Spine (Phila Pa 1976) 34:43–48
6. Park JB, Cho YS, Riew KD (2005) Development of adjacent-level ossification in patients with an anterior cervical plate. J Bone Joint Surg Am 87:558–563
7. Ryken TC, Clausen JD, Traynelis VC, Goel VK (1995) Biomechanical analysis of bone mineral density, insertion technique, screw torque, and holding strength of anterior cervical plate screws. J Neurosurg 83:325–329
8. Spivak JM, Chen D, Kummer FJ (1999) The effect of locking fixation screws on the stability of anterior cervical plating. Spine (Phila Pa 1976) 24:334–338
9. Brodke DS, Klimo P Jr, Bachus KN, Braun JT, Dailey AT (2006) Anterior cervical fixation: analysis of load-sharing and stability with use of static and dynamic plates. J Bone Joint Surg Am 88:1566–1573
10. Rhee JM, Riew KD (2007) Dynamic anterior cervical plates. J Am Acad Orthop Surg 15:640–646
11. Brodke DS, Gollogly S, Alexander Mohr R, Nguyen BK, Dailey AT, Bachus aK (2001) Dynamic cervical plates: biomechanical evaluation of load sharing and stiffness. Spine (Phila Pa 1976) 26:1324–1329
12. Pitzen TR, Chrobok J, Stulik J (2009) Implant complications, fusion, loss of lordosis, and outcome after anterior cervical plating with dynamic or rigid plates: two-year results of a multi-centric, randomized, controlled study. Spine (Phila Pa 1976) 34:641–646
13. Nunley PD, Jawahar A, Kerr EJ 3rd, Cavanaugh DA, Howard C, Brandao SM (2009) Choice of plate may affect outcomes for single versus multilevel ACDF: results of a prospective randomized single-blind trial. Spine J 9:121–127
14. Lee MJ, Bazaz R, Furey CG, Yoo J (2007) Risk factors for dysphagia after anterior cervical spine surgery: a two-year prospective cohort study. Spine J 7:141–147
15. Yue WM, Brodner W, Highland TR (2005) Persistent swallowing and voice problems after anterior cervical discectomy and fusion with allograft and plating: a 5- to 11-year follow-up study. Eur Spine J 14:677–682
16. Shapiro SA, Snyder W (1997) Spinal instrumentation with a low complication rate. Surg Neurol 48: 566–574
17. Silber JS, Anderson DG, Daffner SD (2003) Donor site morbidity after anterior iliac crest bone harvest for single-level anterior cervical discectomy and fusion. Spine (Phila Pa 1976) 28:134–139
18. Aryan HE, Lu DC, Acosta FL Jr, Ames CP (2007) Corpectomy followed by the placement of instrumentation with titanium cages and recombinant human bone morphogenetic protein-2 for vertebral osteomyelitis. J Neurosurg Spine 6:23–30
19. Hwang SH, Kayanja M, Milks RA, Benzel EC (2007) Biomechanical comparison of adjacent segmental motion after ventral cervical fixation with varying angles of lordosis. Spine J 7:216–221
20. Auguste KI, Chin C, Acosta FL, Ames CP (2006) Expandable cylindrical cages in the cervical spine: a review of 22 cases. J Neurosurg Spine 4:285–291
21. Samandouras G, Shafafy M, Hamlyn PJ (2001) A new anterior cervical instrumentation system combining an intradiscal cage with an integrated plate: an early technical report. Spine (Phila Pa 1976) 26:1188–1192
22. Miller LE, Block JE (2011) Safety and effectiveness of bone allografts in anterior cervical discectomy and fusion surgery. Spine (Phila Pa 1976) 36(24): 2045–2050
23. Gercek E, Arlet V, Delisle J, Marchesi D (2003) Subsidence of stand-alone cervical cages in anterior interbody fusion: warning. Eur Spine J 12:513–516

24. Browd SR, McIntyre JS, Brockmeyer D (2008) Failed age-dependent maturation of the occipital condyle in patients with congenital occipitoatlantal instability and Down syndrome: a preliminary analysis. J Neurosurg Pediatr 2:359–364
25. Ebraheim NA, Lu J, Biyani A, Brown JA, Yeasting RA (1996) An anatomic study of the thickness of the occipital bone. Implications for occipitocervical instrumentation. Spine (Phila Pa 1976) 21:1725–1729; discussion 9–30
26. Haher TR, Yeung AW, Caruso SA (1999) Occipital screw pullout strength. A biomechanical investigation of occipital morphology. Spine (Phila Pa 1976) 24:5–9
27. Deutsch H, Haid RW Jr, Rodts GE Jr, Mummaneni PV (2005) Occipitocervical fixation: long-term results. Spine (Phila Pa 1976) 30:530–535
28. Stabler CL, Eismont FJ, Brown MD, Green BA, Malinin TI (1985) Failure of posterior cervical fusions using cadaveric bone graft in children. J Bone Joint Surg Am 67:371–375
29. Tredwell SJ, Newman DE, Lockitch G (1990) Instability of the upper cervical spine in Down syndrome. J Pediatr Orthop 10:602–606
30. Young JP, Young PH, Ackermann MJ, Anderson PA, Riew KD (2005) The ponticulus posticus: implications for screw insertion into the first cervical lateral mass. J Bone Joint Surg Am 87:2495–2498
31. Grob D, Magerl F (1987) Surgical stabilization of C1 and C2 fractures. Orthopade 16:46–54
32. Wang C, Yan M, Zhou H, Wang S, Dang G (2007) Atlantoaxial transarticular screw fixation with morselized autograft and without additional internal fixation: technical description and report of 57 cases. Spine (Phila Pa 1976) 32:643–646
33. Harms J, Melcher RP (2001) Posterior C1-C2 fusion with polyaxial screw and rod fixation. Spine (Phila Pa 1976) 26:2467–2471
34. Ebraheim N, Rollins JR Jr, Xu R, Jackson WT (1996) Anatomic consideration of C2 pedicle screw placement. Spine (Phila Pa 1976) 21:691–695
35. Xu R, Nadaud MC, Ebraheim NA, Yeasting RA (1995) Morphology of the second cervical vertebra and the posterior projection of the C2 pedicle axis. Spine (Phila Pa 1976) 20:259–263
36. Wright NM (2005) Translaminar rigid screw fixation of the axis. Technical note. J Neurosurg Spine 3:409–414
37. Dmitriev AE, Lehman RA Jr, Helgeson MD, Sasso RC, Kuhns C, Riew DK (2009) Acute and long-term stability of atlantoaxial fixation methods: a biomechanical comparison of pars, pedicle, and intralaminar fixation in an intact and odontoid fracture model. Spine (Phila Pa 1976) 34:365–370
38. Neo M, Fujibayashi S, Miyata M, Takemoto M, Nakamura T (2008) Vertebral artery injury during cervical spine surgery: a survey of more than 5600 operations. Spine (Phila Pa 1976) 33:779–785
39. Aryan HE, Newman CB, Nottmeier EW, Acosta FL Jr, Wang VY, Ames CP (2008) Stabilization of the atlantoaxial complex via C-1 lateral mass and C-2 pedicle screw fixation in a multicenter clinical experience in 102 patients: modification of the Harms and Goel techniques. J Neurosurg Spine 8:222–229
40. Coe JD, Warden KE, Sutterlin CE 3rd, McAfee PC (1989) Biomechanical evaluation of cervical spinal stabilization methods in a human cadaveric model. Spine (Phila Pa 1976) 14:1122–1131
41. Brodke DS, Anderson PA, Newell DW, Grady MS, Chapman JR (2003) Comparison of anterior and posterior approaches in cervical spinal cord injuries. J Spinal Disord Tech 16:229–235
42. Xu R, Haman SP, Ebraheim NA, Yeasting RA (1999) The anatomic relation of lateral mass screws to the spinal nerves. A comparison of the Magerl, Anderson, and An techniques. Spine (Phila Pa 1976) 24: 2057–2061
43. Levine AM, Mazel C, Roy-Camille R (1992) Management of fracture separations of the articular mass using posterior cervical plating. Spine (Phila Pa 1976) 17:S447–S454
44. An HS, Gordin R, Renner K (1991) Anatomic considerations for plate-screw fixation of the cervical spine. Spine (Phila Pa 1976) 16:S548–S551
45. Barrey C, Mertens P, Rumelhart C, Cotton F, Jund J, Perrin G (2004) Biomechanical evaluation of cervical lateral mass fixation: a comparison of the Roy-Camille and Magerl screw techniques. J Neurosurg 100: 268–276
46. Choueka J, Spivak JM, Kummer FJ, Steger T (1996) Flexion failure of posterior cervical lateral mass screws. Influence of insertion technique and position. Spine (Phila Pa 1976) 21:462–468
47. Muffoletto AJ, Yang J, Vadhva M, Hadjipavlou AG (2003) Cervical stability with lateral mass plating: unicortical versus bicortical screw purchase. Spine (Phila Pa 1976) 28:778–781
48. Johnston TL, Karaikovic EE, Lautenschlager EP, Marcu D (2006) Cervical pedicle screws vs. lateral mass screws: uniplanar fatigue analysis and residual pullout strengths. Spine J 6:667–672
49. Kast E, Mohr K, Richter HP, Borm W (2006) Complications of transpedicular screw fixation in the cervical spine. Eur Spine J 15:327–334
50. Eleraky M, Setzer M, Baaj AA, Papanastassiou I, Conrad BP, Vrionis FD (2010) Biomechanical comparison of posterior cervicothoracic instrumentation techniques after one-level laminectomy and facetectomy. J Neurosurg Spine 13:622–629

Lumbosacral Instrumentation

9

Joanne Elston and Nelson Saldua

The goals of lumbosacral instrumentation include:
1. Stabilization
2. Correction of deformity
3. Reconstruction or replacement
4. Facilitation and enhancement fusion

Internal fixation provides immediate stability; however, the devices are inadequate to withstand prolonged periods of stress and are likely to fail unless a fusion is performed at the time of the instrumentation [1]. Despite all the advances and new developments in spinal instrumentation, fixation failure and pseudoarthrosis continue to be a challenge to spine surgeons, especially at the lumbosacral junction [2].

Prior to the development of modern instrumentation techniques, the only way to maintain spinal alignment and stability after fusion for patients with deformity or fracture was with body casting (see Fig. 9.1). This method was the method of choice before the 1960s [2]. The large number of associated complications and inadequate fusion rates, reported as high as 50 %, led to the development of the Harrington instrumentation (see Fig. 9.2a, b), which quickly became the gold standard for the surgical treatment of scoliosis [2]. The Luque instrumentation system (see Fig. 9.3a, b) was introduced to address some of the shortcomings of the Harrington rod system [3, 4], specifically at the lumbosacral junction. This system consisted of sublaminar wires attached at multiple levels to ¼-in. rods, which was eventually modified to an L-shaped rod to prevent caudal and cephalad migration through the wires [2]. This system permitted for better coronal and sagittal balance [2–4].

A decade later, two new techniques were developed, the Cotrel-Dubousset (see Fig. 9.4a, b) instrumentation and the Galveston technique. The Cotrel-Dubousset system allowed for three-column fixation using pedicle screws with numerous points of fixation proximally in combination with hooks [2, 5]. Later versions added sacral

Fig. 9.1 Risser body casting for treatment of a child with early onset scoliosis

J. Elston • N. Saldua (✉)
Department of Orthopaedic Surgery,
Naval Medical Center San Diego, 34800 Bob Wilson Drive, suite 112, San Diego, CA 92134, USA
e-mail: joanne.elston@med.navy.mil; nelsonsaldua@gmail.com

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_9, © Springer-Verlag Berlin Heidelberg 2014

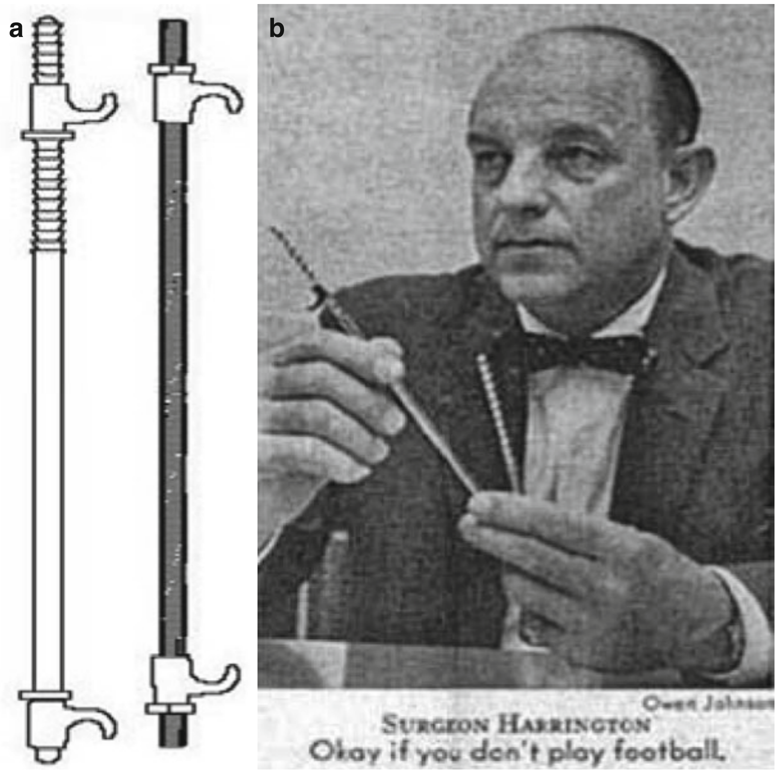


Fig. 9.2 (**a**, **b**) Harrington rod spinal instrumentation with surgeon Paul Harrington pictured on the *right*. The breakthrough was the invention of the Harrington rod by Paul Harrington of Texas in the 1950s, whose stainless-steel rod with a ratchet and a hook at each end allowed the safe placement, on the back of the spine, of a metal strut, which could be lengthened to pull out a C-shaped curve to as near straight as possible

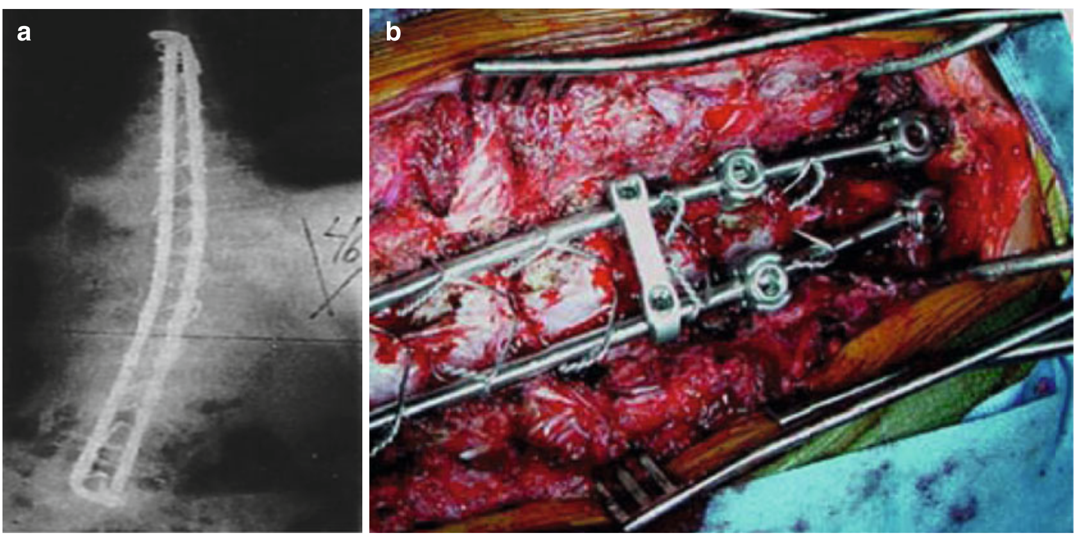


Fig. 9.3 (**a**) Bilateral Luque rods for treatment of scoliosis. Note that the construct failed at the transition of T12 and L1. The Luque instrumentation is a segmental spinal instrumentation system, which uses sublaminar wires attached to the rod. (**b**) Intraoperative illustration of the segmental sublaminar wiring with bilateral rods

fixation for increased biomechanical strength. However, this system still lacked the ability to resist flexion forces at the lumbosacral junction, and pseudoarthrosis rates remained as high as 33 %, particularly for adult deformity correction [5]. The Galveston technique (see Fig. 9.5a, b),

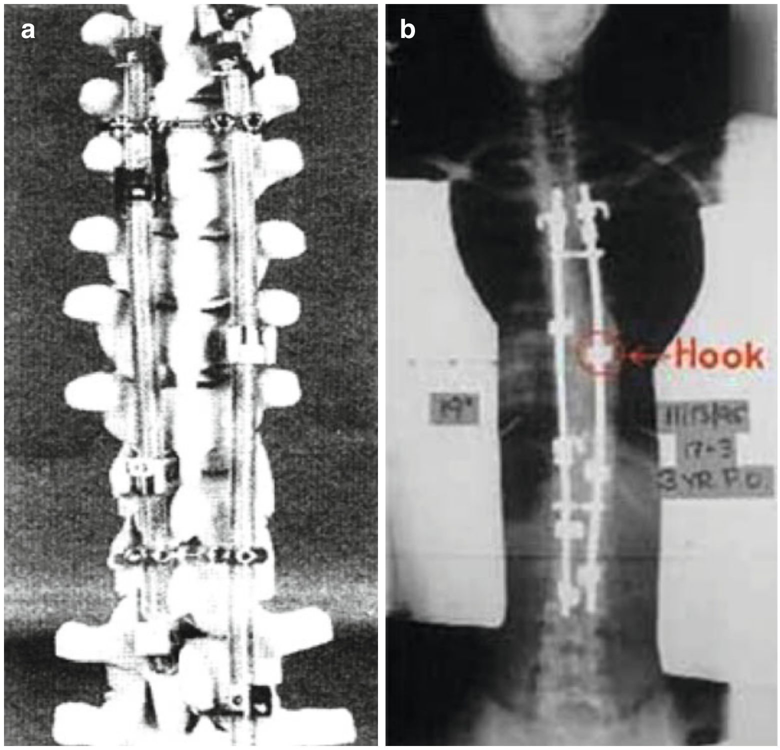


Fig. 9.4 (**a**, **b**) The images are an example of Cotrel-Dubousset instrumentation. This new concept in spinal instrumentation was developed by Drs. Yves Cotrel and Jean Dubousset in France. It uses hooks and rods in a cross-linked pattern to realign the spine and redistribute the biomechanical stress (Images were borrowed from the internet)

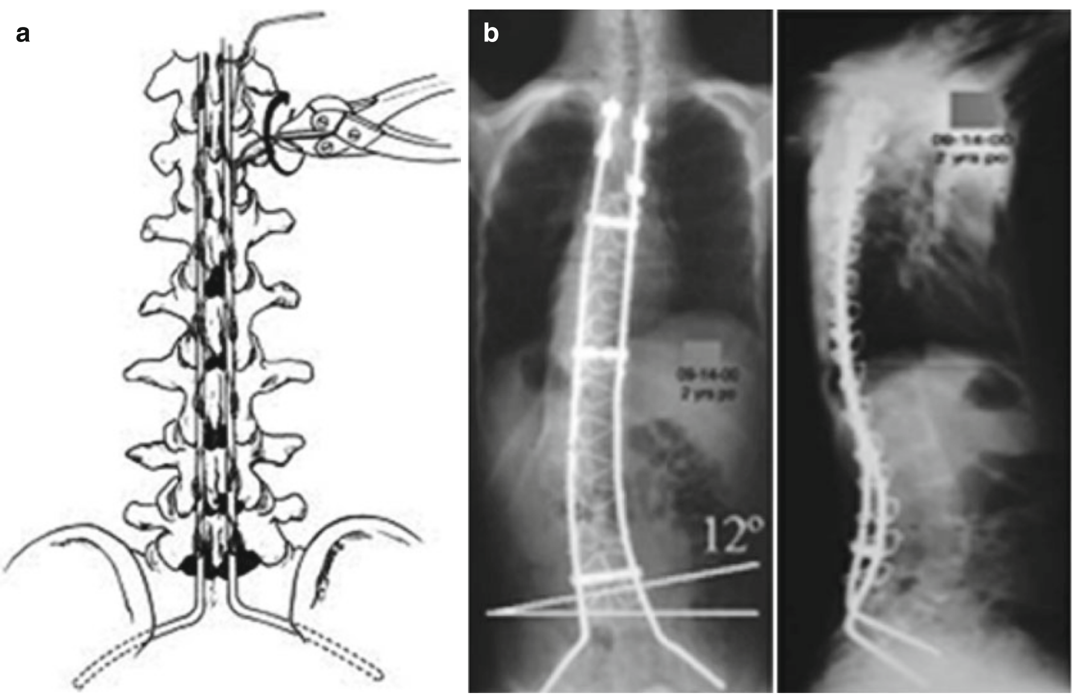


Fig. 9.5 (**a**) The Galveston technique for spinal fixation provides fixation at the lumbosacral junction by introducing a contoured rod into the ilium as shown here. (**b**) Images here demonstrate an AP and lateral radiograph of the Galveston rod spinal instrumentation in a patient with CP. This was used with Luque sublaminar wiring

on the other hand, provided more stable and rigid fixation at the lumbosacral junction by introducing a contoured rod from the posterosuperior iliac spine into the ilium between the inner and outer tables. This increased stiffness in flexion and side bending, which significantly decreased instrumentation failures [2].

Spinal instrumentation has come a long way from the initial hooks and rods used in the 1960s. Numerous techniques developed throughout the remainder of the twentieth century, including various modifications to the aforementioned rod constructs with hooks, wires, and screws. The development of pedicle screws in particular drastically changed spinal instrumentation and lumbosacral surgery. A variety of plates, sacral screws, iliac bolts, cages, and interbody devices continue to be developed. Simultaneously newer forms of instrumentation such as interspinous devices, dynamic stabilization devices, and arthroplasty are changing the face of spine surgery.

We will briefly introduce and review each of the types of instrumentation that are utilized in the lumbosacral spine and discuss the biomechanics, indications, advantages, and disadvantages of each.

9.1 Rods

Rods, either paired or unpaired, are commonly used for both posterior and anterior fixation. Anterior-based rod constructs will be discussed in further detail in the section on anterior instrumentation. Regardless of whether they are used anteriorly or posteriorly, they functionally span a segment of the spine allowing for fixation over multiple levels. They are used with either wires, hooks, screws, or a combination thereof to attach to the spine [6]. Historically, they were first used in 1964 by Knodt and were popularized by Harrington in the 1960s [6]. This method of instrumentation quickly became the gold standard for the surgical treatment of scoliosis over the following decade, as it provided superior stabilization to previous methods of body casting [2]. Classically, this method of instrumentation was non-segmental, implying only two sites of fixation, one proximally and one distally (see Fig. 9.2a). This is rarely used now due to the improved biomechanical strength and deformity correction with segmental correction and multiple points of fixation [1].

McAfee et al. in 1985 biomechanically analyzed three spinal instrumentation constructs in 25 cadaveric spinal segments. Conventional Harrington distraction instrumentation, segmentally wired Harrington distraction rods, and Luque segmental spinal instrumentation were compared in 61 biomechanical tests. Segmentally wired Harrington distraction instrumentation proved substantially advantageous at resisting axial loads in unstable burst fractures, while the Luque segmental spinal instrumentation with L-rods coupled together proved to be the best method of achieving rotational stability in translational injuries (fracture-dislocations) [3]. The biomechanical advantages of spinal instrumentation must always be weighed against the increased operative time, technical expertise required, and potential risks and complications of iatrogenic neurologic sequelae, neurovascular injury, and morbidity of surgery.

9.2 Hooks and Wires

Hooks (see Fig. 9.6) provide only posterior column support but remain an effective and versatile method for stabilizing the spine. Although they are more commonly used in the cervical spine, hooks remain a useful instrumentation tool for the lumbosacral spine as well. They may be anchored to the posterior elements via the lamina, pedicle, or transverse process (see Fig. 9.7). Laminar and pedicle hooks are used with rods to allow compression or distraction forces to be applied to the pedicles or laminae. They come in various sizes and shapes and engage the posterior elements by curving under (up-going hooks) or over (down-going hooks) the lamina (see Fig. 9.8) [6]. These hooks may have blunt or sharp ends or ridges to prevent

Fig. 9.6 Illustration of a pedicle hook, lumbar lamina hook, and thoracic lamina hook

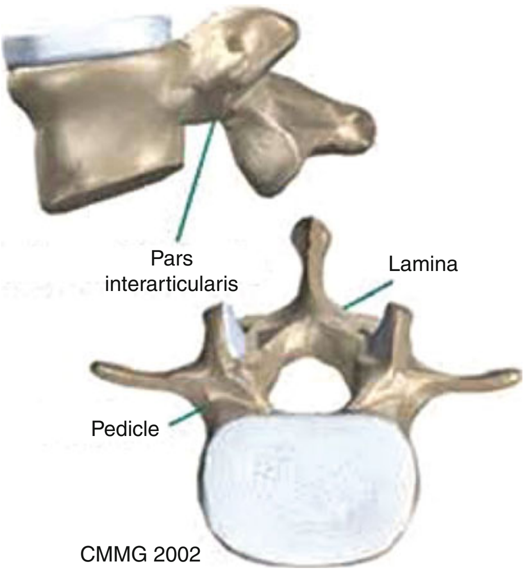


Fig. 9.7 Anatomic illustration of the vertebrae and posterior elements including the pedicle and lamina as well as the pars interarticularis for review (©MMG 2002)

slippage. The hook is held in place by lock washers, bolts, or set screws to the rod (see Fig. 9.9). Laminar hooks have been the workhorse of segmental hook fixation, but may not be used in combination with a laminectomy or for stabilization when there is injury to the lamina secondary to trauma and fracture. Pedicle hooks, however, provide a stronger anchoring point compared to laminar hooks but can only be placed up-going [1]. They are placed inferior to the pedicle at the facet joint. For example, the hook would be placed at the L1–2 facet joint for an L1 pedicle

Fig. 9.8 Radiograph demonstrating posterior spinal instrumentation with pedicle screws and laminar hooks. This lateral radiograph demonstrates the ability for laminar hooks to be placed either up-going or down-going

Fig. 9.9 Image shows a pedicle hook with washer. Also note the sharp ends to help prevent slippage

hook. Sacral hooks may be placed lower than pedicle screws in the sacrum and are often used as an adjunct to pedicle screws for an additional level of stability [6–8].

Sublaminar wiring (see Fig. 9.10) involves passing a wire(s) around the lamina and rod or through a hole in the spinous process (interspinous wiring) [6]. The interlaminar space must be identified after removal of interspinous ligaments. The ligamentum flavum and soft tissue are released from the lamina, and the wire is passed caudad to cranial around the lamina [1]. Cables may be used instead of a wire, which are more pliable and less brittle (see Fig. 9.11) [6]. Sacral foraminal wires may also be used and are more secure than hooks, which have the potential to dislodge. Additionally, they are less bulky than hooks and are less likely to cause discomfort [6]. Nevertheless, these implants are placed dorsal to the mechanical axis of rotation at L5–S1, thus contributing to their high rates of failure [2, 7]. They rely on compressive and distractive forces for their purchase and have inferior torsional stability. Even though wire constructs have good sagittal stability, they have limited torsional stability and cannot provide compression or prevent longitudinal collapse [1].

Fig. 9.10 Illustration demonstrates sublaminar wiring with rod along the posterior spine

Hook and wire constructs are most commonly used to correct deformity, namely, for scoliosis. Scoliosis remains their main indication for use; however, additional uses include treatment of trauma, tumor, degenerative spondylolisthesis and scoliosis, and disc disruption with fusion. Wires may be used without rods for the treatment of certain fractures. Sublaminar wires, hooks, and cables, particularly at the sacral level, lack the biomechanical strength to serve as rigid instrumentation. They have poor pullout strength compared to other constructs [9]. Lack of stability in flexion, rotation, and side bending led to "flat-back syndrome" and complications of sagittal plane imbalance [2]. Over the years, various advances led to the development of locking hooks, to help prevent the common failure mechanism of dislodgement. Up-going hooks and down-going hooks may be used at the same level, a claw mechanism, which helps to reduce dislodgment. Self-adjusting hooks for multilevel

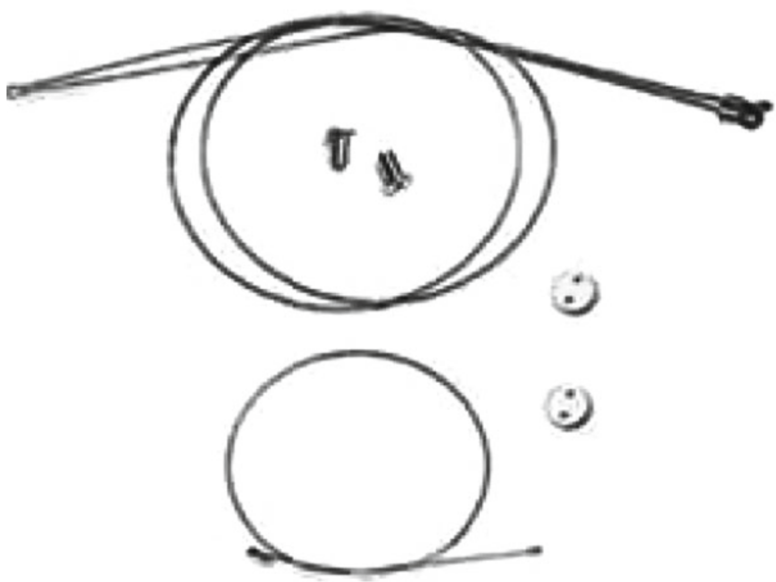


Fig. 9.11 Cable, both single and double loop, designed for sublaminar passage

placement have increased the rigidity of hook rod constructs [1, 6].

Dislodgement is a significant disadvantage of the hook and rod systems, particularly before the development of claw mechanisms and locking hooks. Additionally, hook and wire slippage on the rod has been a commonly reported mechanism of failure in the literature as well as rod, hook, and wire breakage [10]. Another disadvantage of these constructs is that they are intra-canal space-occupying devices that have the potential to cause neural compression or injury. An advantage of hooks and wire systems is that they may be used for either compression or distraction to correct deformity. They have limited multi-planar stability [1]. They may be placed at multiple levels, providing segmental instrumentation, to help increase the rigidity of the construct and provide more precise deformity correction. Additionally, they are easier and quicker to insert than pedicle screws. They are easy to place and are a biomechanically favorable method of fixation over pedicle screws in osteoporotic bone because the anterior aspects of the laminae are the least affected by bone mineral density loss [1]. They are also the least expensive construct for posterior segmental spinal fixation.

9.3 Pedicle Screws

Pedicle screw fixation (see Fig. 9.12a–d) or transpedicle screws are a more recent advancement in spinal instrumentation and have become the workhorse for lumbosacral instrumentation. Pedicle screws are used most commonly in combination with fusion to enhance segmental stability. Other indications include deformity correction, degenerative conditions, fracture fixation, and treatment of tumors and infection. They provide superior biomechanical stability compared with other segmental constructs as they provide fixation in all three-columns of the vertebral bone. Functionally they provide excellent longitudinal (both compression and distraction), torsional, and sagittal stability [1]. They may be used with both plates and rods. Although they were first reported to be used in 1969 by Harrington and Tullos, they did not become popularized until several years later [6]. Various starting points (see Fig. 9.13) have been commonly reported in the literature, but the lateral border of the pars interarticularis and the middle of the transverse process is a great reference starting point. They are angled medially to pass through the pedicle and into the vertebral body. Depending on the vertebral level, both the angle and size of the screw will vary. Additionally, pedicle screws may be used in both open and percutaneous procedures (see Fig. 9.14a–d). They are attached posteriorly to rods or plates with clamps, or set screws. Pedicle screws are able to resist loads in all directions, and this three-dimensional rotational control makes them useful for correcting deformities, much more so than wires and hooks [6, 9, 11]. They provide three-column stability, being anchored to both anterior and posterior vertebral bodies, which is in contrast to the hook systems which are only anchored to the posterior elements [1]. The strength of fixation may be decreased when used in osteopenic bone or when they are inserted too shallow, which is a potential disadvantage in certain situations.

Because of the three-column stability and the segmental fixation, pedicle screws can be used to deliver large corrective forces to the spinal column to treat scoliosis, kyphotic deformities, and other deformities [1, 12, 13]. They are ideal for fracture and dislocation stabilization after trauma. Pedicle screw fixation systems are most useful in correcting degenerative conditions for which the spinous processes and laminae are often removed for neural decompression [14]. Pedicle screw fixation allows stable attachment to a vertebra despite resection of the posterior elements and increases fusion rate when used in combination with bone grafting. Contrary to hooks and wires, they can be used to stabilize vertebrae after laminectomy [1, 15]. In addition, segmental control of the vertebrae is possible, allowing distraction and compression within the length of spinal fusion [14]. Overall, pedicle screws have superior biomechanical stability [1]. However, they are technically demanding to insert with significant potential risk for violation into the spinal canal and potential nerve

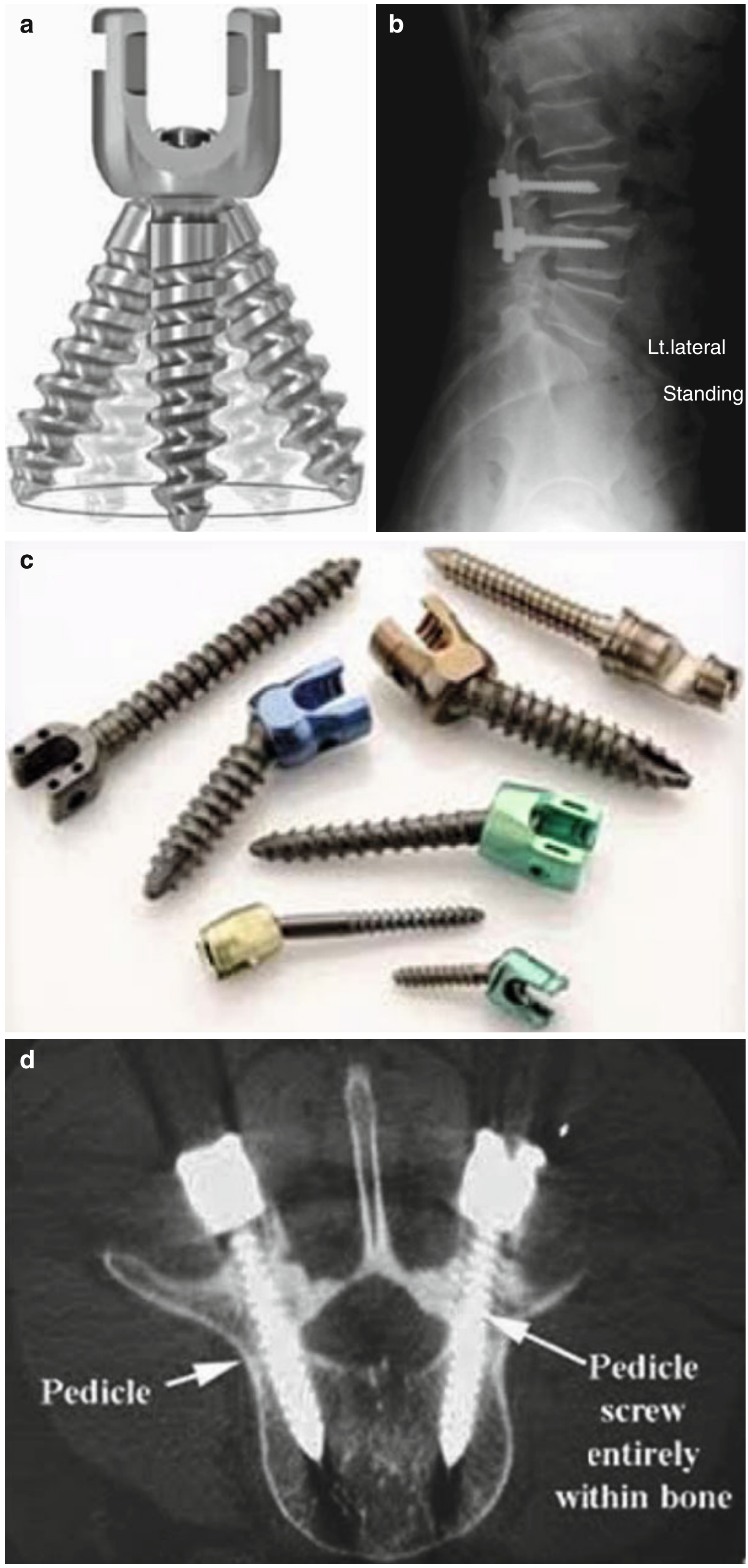


Fig. 9.12 Images (**a**) and (**b**) demonstrate a polyaxial pedicle screw. (**b**) Postoperative lateral radiograph of two-level posterior lumbar pedicle screw instrumentation. Image (**c**) demonstrates various pedicle screw assemblies. (**d**) Axial CT image demonstrating bilateral pedicle screws entirely within the pedicle of the vertebrae without violation of the cortex

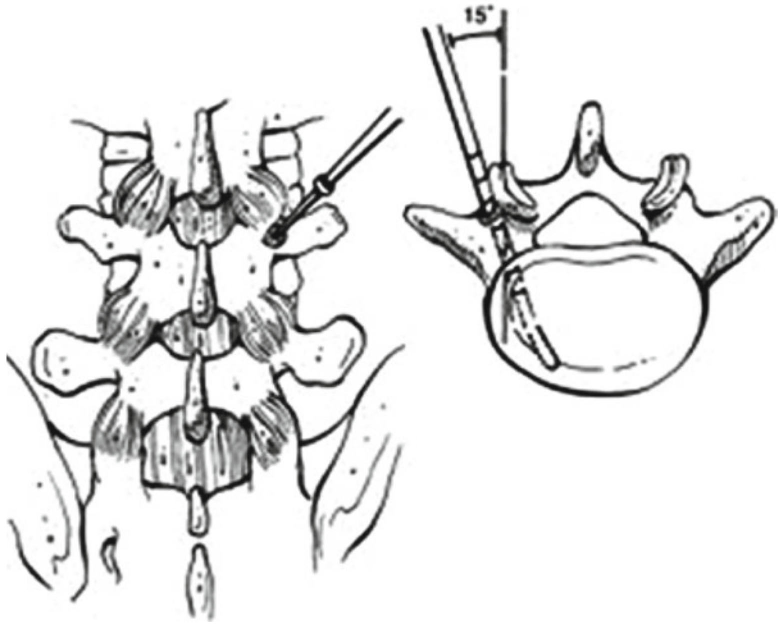


Fig. 9.13 The illustrations demonstrate the proper starting point and trajectory for insertion of a lumbar pedicle screw

injury. Additionally, they provide poor pullout strength in both osteopenic bone as well as the sacrum [1, 9].

Lumbosacral fusions may utilize pedicle screws; however, they are limited to S1 and S2. Sacrum pedicles contain less supportive cancellous bone. In order to improve biomechanical strength and pullout force, screws may be placed through two or even three cortices (sacral promontory) [6, 16]. However, long fusions ending at the sacrum with pedicle screws continue to pose problems for spine surgeons due to the forces on the lumbosacral junction. Failure rates have been reported to be as high as 44 % [2].

In a prospective randomized study, Zdeblick showed that the use of rigid pedicle screw instrumentation increased the chances of a successful fusion [14, 17]; however, the quality of the bone influences screw pullout strength and there is a considerable influence of various geometric variables of screw design on screw performance as well. Skinner et al. compared the relative performance of four different common pedicle screw designs on the market. Important principles from this study are:

1. Improvements in pullout strength can be achieved by an increase in the outer diameter of the screw.
2. Screw displacement before failure appears related to the screw pitch such that an increase in the pitch of the screw will increase the amount of displacement before failure.
3. Screw angulation was found to have little effect on the pullout strength but may impact the screw displacement and energy absorption before failure [12].

9.4 Facet Screws

Translaminar facet joint screw fixation (see Fig. 9.15a–c) provides an alternative to pedicle screw fixation for spine fusion. Similar biomechanical performance has been shown between translaminar facet joint fixation with screws and pedicle screw fixation [14, 18]. The facet joint is the only true articulation in the lumbosacral spine. Thus, translaminar lumbar facet screws provide posterior stabilization of a single lumbar motion segment. They may be placed like pedicle screws either open or percutaneously. The screw is placed at the junction of the spinous process and contralateral lamina through the ipsilateral lamina across the articular surface of the facet joint [1]. This is most commonly used as supplemental fixation with ALIFs. The lamina must be left partially intact to

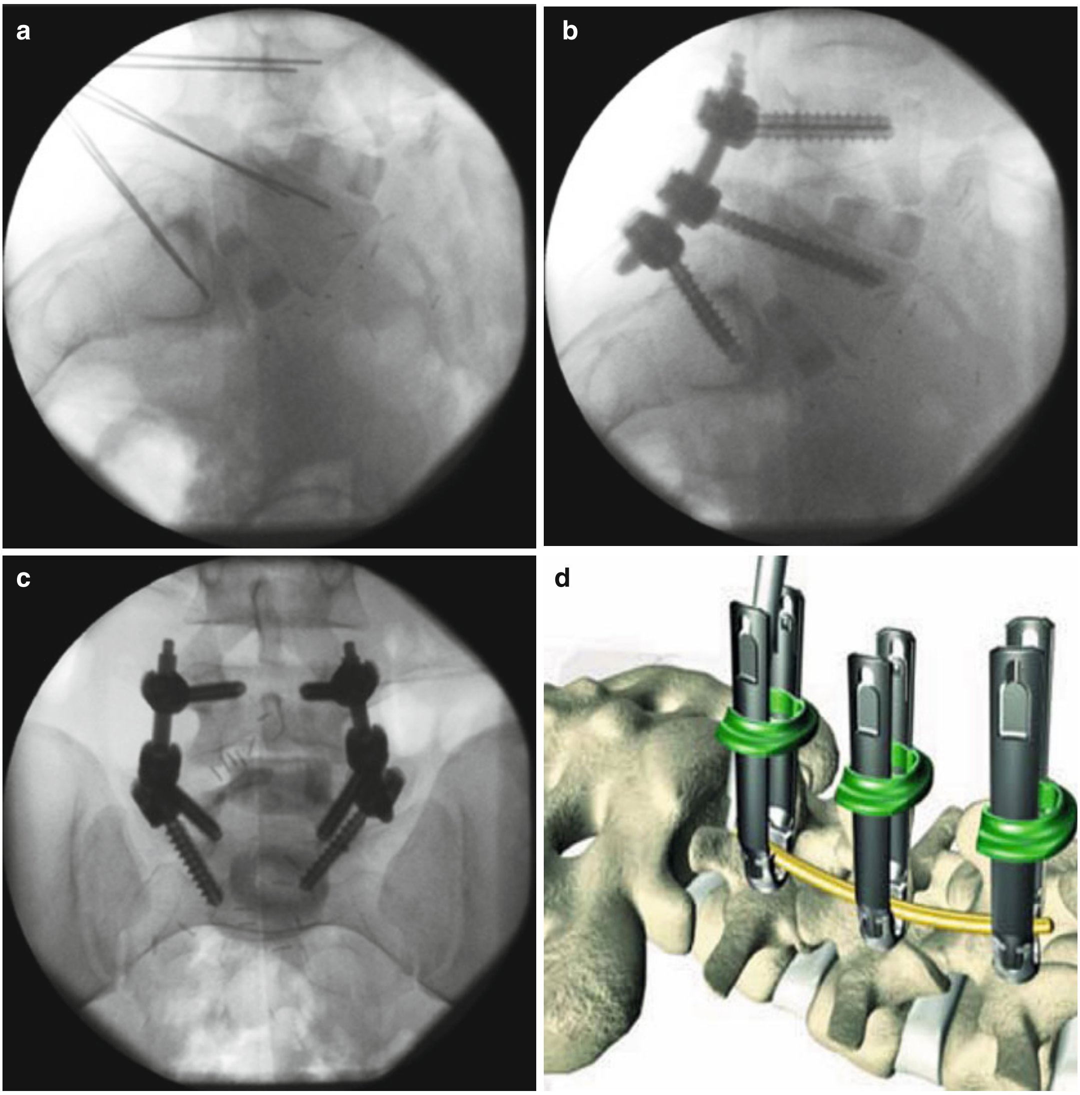


Fig. 9.14 (**a**) Percutaneous wires for placement of lumbosacral pedicle screws. (**b**) Lateral fluoroscopic image of lumbosacral pedicle screws. (**c**) AP fluoroscopic image of lumbosacral pedicle screws. (**d**) Depicting the placement of percutaneous pedicle screw and rod instrumentation in the lumbar spine

perform this fixation; thus, decompression is usually a limited foraminotomy. Although this method of instrumentation is much less commonly used compared to other methods, it is the lowest profile construct that achieves stabilization when posterior bony elements are preserved [14, 19].

Historically, this method dates back to 1944 when Kin preformed Hibbs fusions along with supplementary facet screw fixation and reported a 91 % fusion rate in 44 cases. In 1984, Magerl described translaminar facet joint screw fixation, using a much longer screw through the entire lamina ending at the base of the transverse process [14]. This method is currently widely accepted and has been examined biomechanically and clinically with excellent results as an alternative to other spinal instrumentations [18, 20, 21]. There are few biomechanical performance comparison studies of translaminar facet joint screw fixation and pedicle screw fixation. Vanden Berghe et al. found that pedicle screw fixation and facet fixation perform similarly when tested biomechanically [14, 22].

9.5 Trans S1 Screw

Minimally invasive spine surgery continues to emerge. This growing trend continues to lead to advances for new surgical techniques as well as spinal devices to help decrease the morbidity and complications of spinal surgeons. Technologic advances have now allowed surgeons to perform L5–S1 fusions by posterolateral or anterior approaches through less invasive techniques. The AxiaLIF system (TransS1) (see Fig. 9.16a–c) allows the application of minimally invasive techniques to attain fusion at either L5–S1 or L4–S1 levels with a novel corridor of approach, described as the presacral "safe zone" [23] (see Fig. 9.16d). Anterior access to the L5–S1 disc space can be technically challenging and frequently requires assistance from a general surgeon for adequate exposure. Percutaneous paracoccygeal approach to the L5–S1 interspace is a minimally invasive corridor. Through this safe corridor, diskectomy and interbody fusion can be performed. It may provide an alternative route of access to

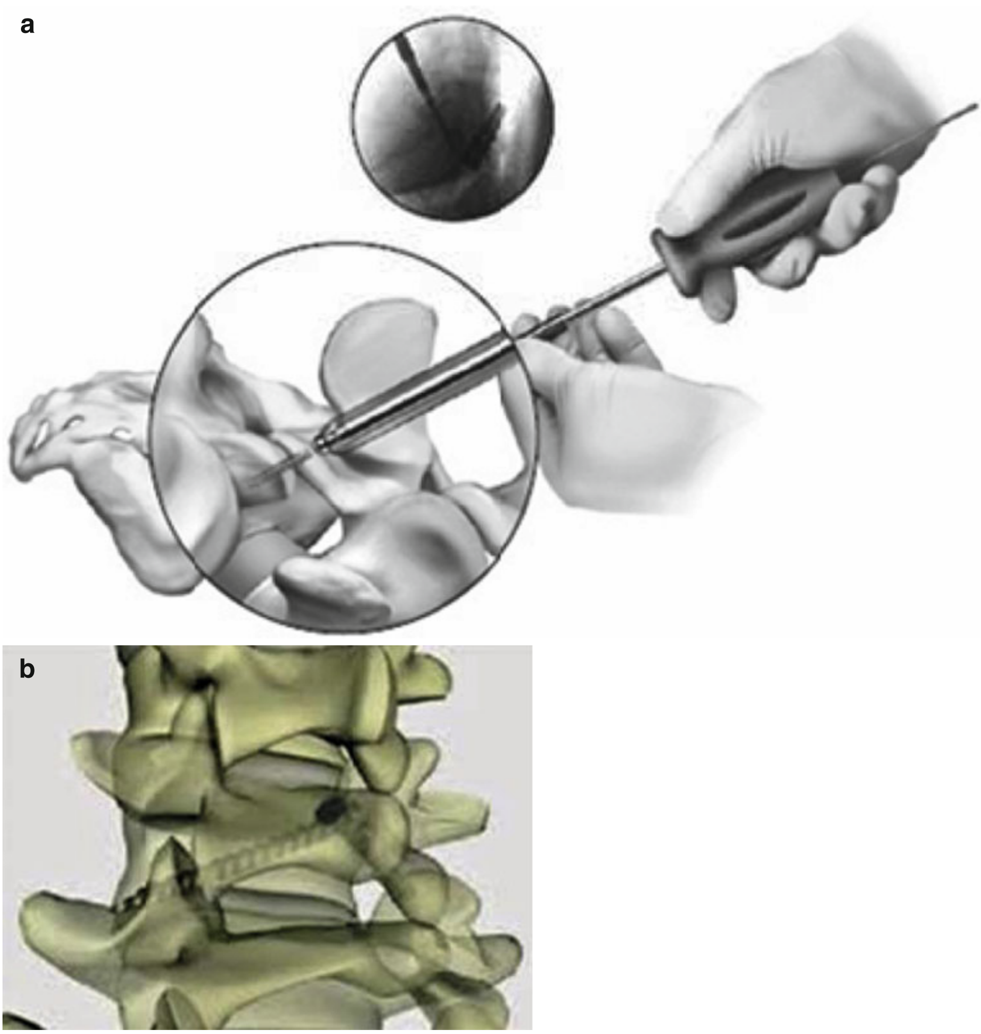


Fig. 9.15 (**a**) Image demonstrates the proper technique for facet screw insertion. (**b**) Illustration demonstrates the trajectory of a translaminar screw. (**c**) AP and lateral radiograph demonstrating a one-level lumbar fusion utilizing a hybrid technique with two pedicle screws on the *left* at L4–L5 and a translaminar facet screw on the *right* at the L4–L5 facet joint

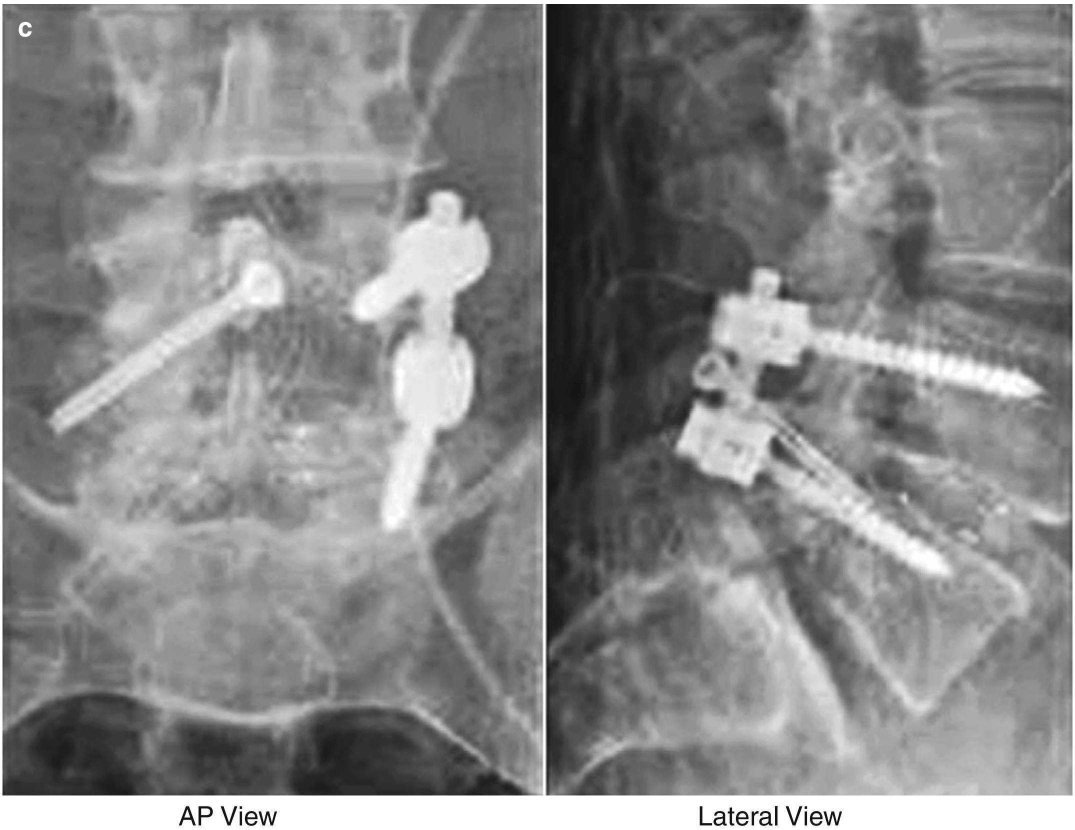


Fig. 9.15 (continued)

the traditional open fusion procedures in patients with unfavorable anatomy [24] (see Fig. 9.16e).

A transsacral rod may be applied through a paracoccygeal approach. Initially, this technique was utilized for single-level axial lumbar interbody fusion; however, recently it has been extended to perform a two-level fusion at both L4–L5 and L5–S1 levels. Indications vary but include back pain secondary to lumbar degenerative disc disease, degenerative lumbar scoliosis, and spondylolisthesis. Early clinical results and biomechanical stability are promising. Various studies have shown radiographic evidence of fusion to be 91 % [24]. The standalone rod reduced intact ROM significantly; however, supplementary fixation with facet screws or pedicle screws is required to achieve higher construct stability for successful fusion [25].

9.6 Interspinous Process Devices

Several interspinous spaces are currently available in the market. Though they vary in design and composition [26], their common mechanical goal is distraction between adjacent spinous processes, thus blocking intervertebral extension at that level. This theoretically provides an indirect decompression of the neural elements. Interspinous process decompression theoretically relieves narrowing of the spinal canal and neural foramen in extension and thus reduces the symptoms of neurogenic intermittent claudication. There are many proposed indications for their use including lumbar canal stenosis, grade I degenerative spondylolisthesis, discogenic low back pain, non-traumatic instability, lumbar disc herniations, and facet syndrome. However, there is limited evidence to support this wide use. The largest number of studies has been with the

X-STOP device, a titanium alloy device that is placed between the spinous processes to reduce the canal and foraminal narrowing that occurs in extension [27] (see Fig. 9.17a–c).

Biomechanical studies show that there is a beneficial effect on the kinematics of the degenerative spine. Other studies show satisfactory outcome to varying degrees. Anderson et al. compared the efficacy of interspinous process decompression with nonoperative treatment in patients with neurogenic intermittent claudication secondary to degenerative spondylolisthesis. The X-STOP device was more effective than nonoperative treatment in the management of

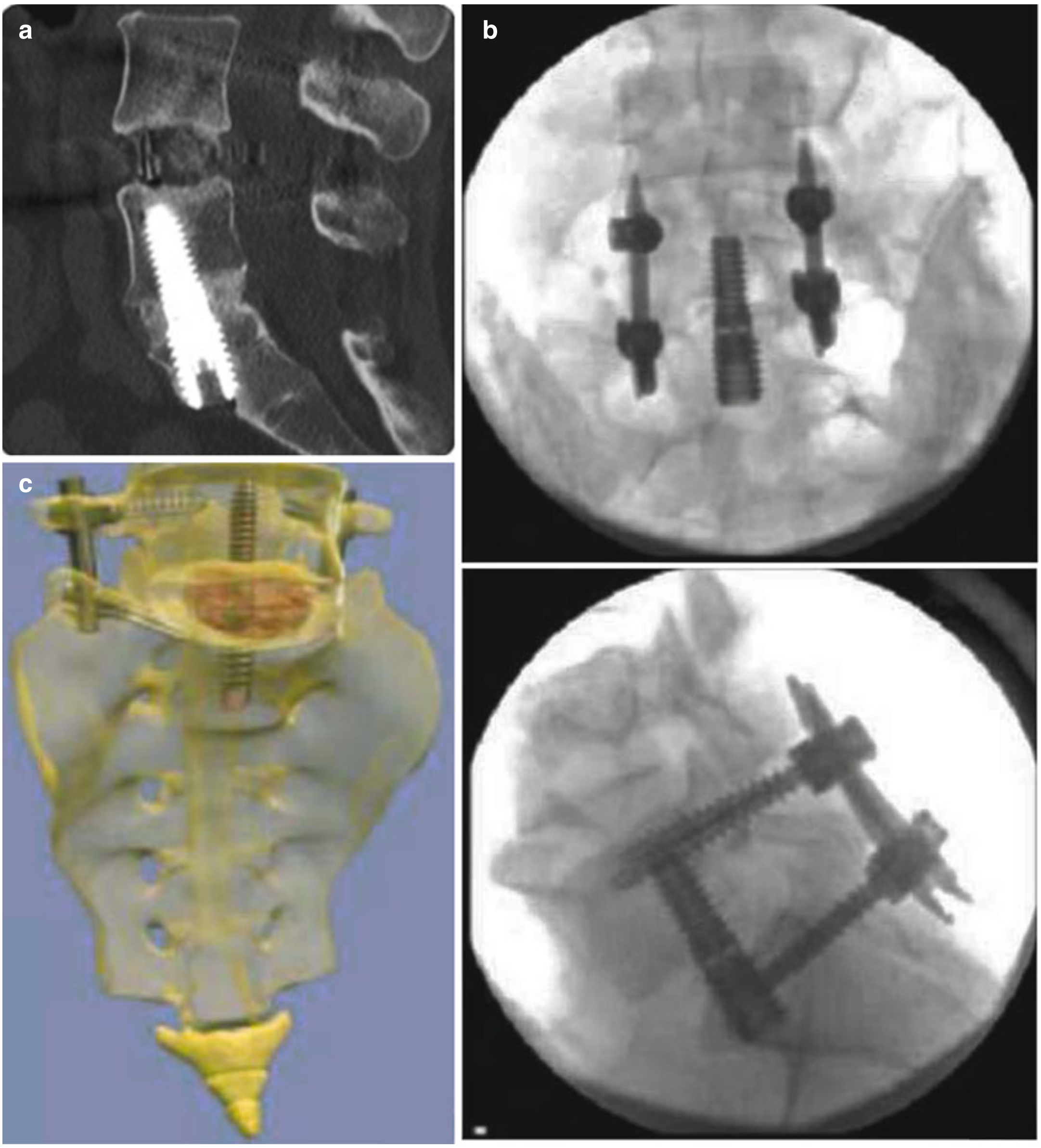


Fig. 9.16 (**a**) Demonstrates a sagittal CT scan of AxiaLIF instrumentation. (**b**) Demonstrates intraoperative AP and lateral fluoroscopic images of AxiaLIF interbody fusion in combination with percutaneous pedicle screw fixation. (**c**) Image depicts a schematic of an axiaLIF interbody fusion in combination with percutaneous pedicle screw fixation. (**d**) The illustration demonstrates access through the presacral fat for AxiaLIF interbody fusion. (**e**) The illustration depicts the procedural steps for an AxiaLIF interbody fusion

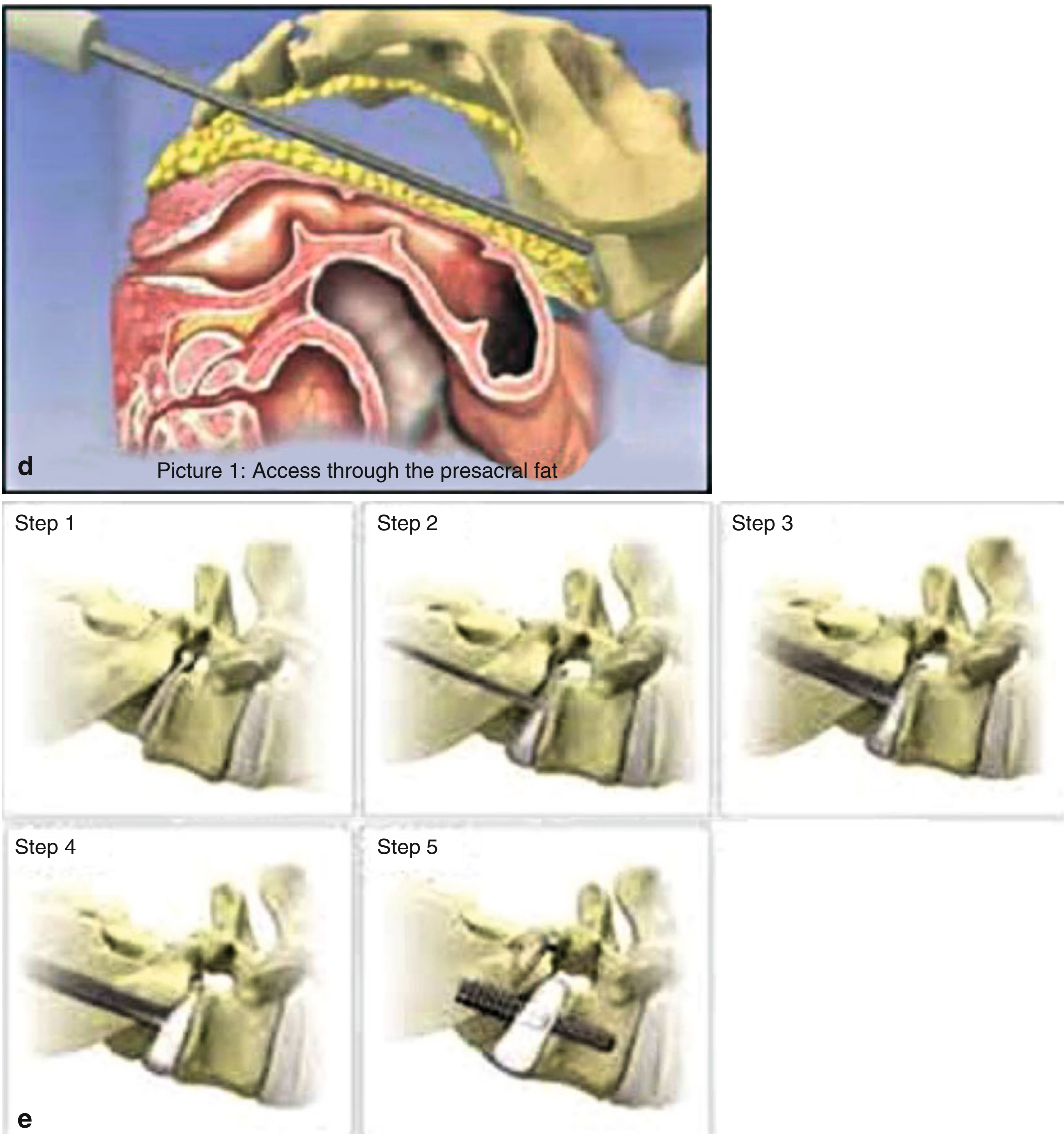


Fig. 9.16 (continued)

neurogenic intermittent claudication secondary to degenerative lumbar spondylolisthesis [27].

9.7 Iliac Bolts

Recent advances and newer spinal instrumentation allow for insertion of screws into the ilium, independent of other points of fixation (see Fig. 9.18). Offset connectors are sometimes used to connect the iliac screws, or bolts, to the longitudinal rods. These screws are very long and provide fixation with pullout strength that has been shown to be three times higher than that of Galveston rods [2]. High fusion rates have been reported at the lumbosacral junction for both high-grade spondylolisthesis and long fusions [28].

Disadvantages include soft tissue dissection, which may potentially increase the risk of infection, reported to be 4 % over a 2-year period in one series of 81 patients [2]. Additionally, care must be taken to avoid violation of the greater

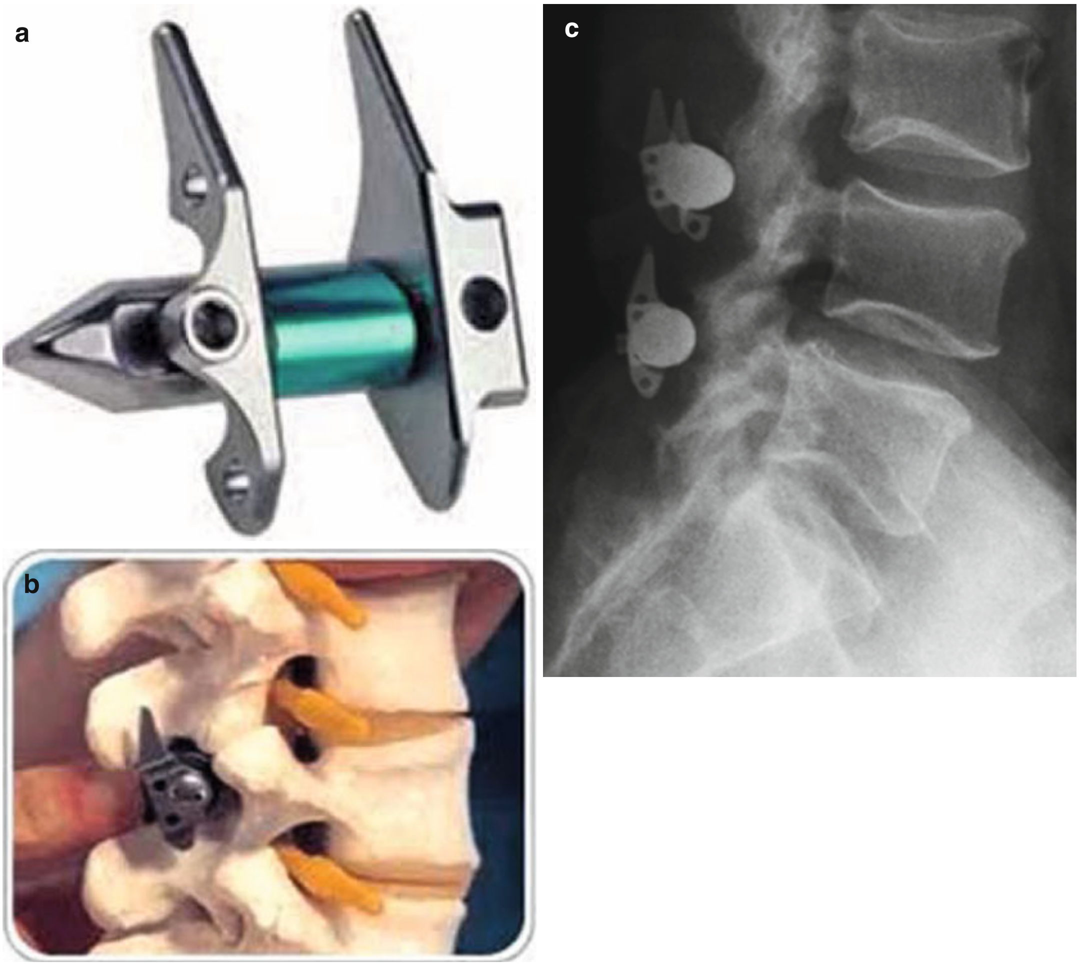


Fig. 9.17 (**a**) The titanium prosthetic X-STOP device. (**b**) Image demonstrates the placement of the prosthetic titanium X-STOP device between the spinous process. (**c**) Shown here is a postoperative lateral radiograph with a two-level interspinous process decompression with an X-STOP device

sciatic notch and all the neurovascular structures there within; however, no injury to any of these structures has been reported in any major case series. The most common complications are instrumentation prominence and pain, which could require eventual removal. The main advantage is increased rigidity with increased fusion rates and decreased rates of pseudoarthrosis at the lumbosacral junction. Kuklo et al. reported a fusion rate of 95.1 % in 81 patients undergoing treatment for high-grade spondylolisthesis or long fusions to the sacrum with bilateral sacral screws, although 14 % of the patients reported discomfort over the iliac screw [29]. The main purpose of adding iliac screws is to decrease the risk of loosening and failure of the S1 screws, where there can be high stresses in longer constructs. They also offload the sacrum as a whole by transferring forces directly to the ilium.

Iliac bolt fixation was found to significantly decrease the flexion-extension moment on the ipsilateral S1 screw by 70 % and the contralateral screw by 26 % in a biomechanical study done by Alagre et al. in 2001 comparing fixation across the lumbosacral junction. Four different L2-sacrum constructs were evaluated with the following findings: (1) There is a significant decrease in the flexion-extension moment on the S1 screw when extending long posterior constructs to either the ilium or S2 sacral screw.

Fig. 9.18 Shown here is an AP radiograph with bilateral iliac bolts in combination with multiple level posterior fusion with pedicle screws and bilateral rods

(2) There is no biomechanical advantage of the iliac bolt over the S2 screw in decreasing the moment on the S1 screw in flexion and extension. (3) Adding anterior support such as an ALIF to long posterior constructs significantly decreases the moment on the S1 screw. Adding distal posterior fixation to either the ilium or S2 decreases the moment on the S1 screws more than adding anterior support [30].

9.8 Interbody Devices

During the past decade, interbody cages have grown in popularity as a useful device to obtain fusion. They may be used alone but typically need supplemental fixation. A variety of surgical approaches allow removal of the diseased disc and degenerative osteophytes, followed by correction of deformity with a cage that is inserted between the vertebral bodies. Thus, distraction of the disc space permits safer, indirect decompression of the intraforaminal zone, which is a common area of stenosis. These devices also theoretically tension the lax spinal ligaments, thus again indirectly decompressing the neural elements [31, 32].

Various interbody device materials have been used, including femoral ring allograft, carbon fiber or polyetherketone structural grafts, titanium mesh, or threaded interbody cage constructs [31, 33] (see Fig. 9.19a–d).

Titanium mesh cages (see Fig. 9.20a–e) are utilized between vertebral bodies for fusions. These mesh cages are porous to promote and facilitate peripheral bone and vascular in growth. Typically, the cage is filled with morcellized bone graft [32]. Delloye et al. evaluated non-perforated cortical bone graft compared with perforated cortical bone graft in sheep models for incorporation. Although there was no statistical difference between both groups for union and bone density, the cortical bone graft porosity and the amount of new bone within the cortical bone differed significantly. Thus, porosity significantly improved the amount of newly formed bone by the host. The channels increased the interface between the host and the allograft and allowed for wider endosteal callus, which resulted in enhanced incorporation [34].

This procedure may be approached from many anatomical locations. As the push toward minimally invasive spinal surgery continues to grow, many new approaches have been invented to help minimize morbidity and complications. These various procedures are named based on the approach but all have the same conceptual goal of removing as much of the disc as possible and placement of graft and a structural spacer in the disc space. This facilitates correction of deformity and helps to optimize lordosis.

1. Anterior lumbar interbody fusion, ALIF (see Fig. 9.21a–d)
2. Posterior lumbar interbody fusion, PLIF (see Fig. 9.22a–d)
3. Transforaminal lumbar interbody fusion, TLIF (see Fig. 9.23a–c)
4. Direct lateral interbody fusion, DLIF (see Fig. 9.24a, b)

Each of the different approaches offers its own advantages and disadvantages. Biomechanical studies indicate that anteriorly placed interbody devices significantly stabilize the motion segment in all directions except extension. Anterior fusion in primary disc lesions produces good results but may be of limited value for spinal stenosis, in which posterior procedures may allow for direct decompression [35]. Posteriorly placed devices provide less stability secondary to the required facetectomy for placement [36]; however, posterior lumbar interbody fusion and segmental pedicle-based plate fixation allow wide decompression and increased exposure for disc space preparation while maintaining stability with the screws. Placement of a PLIF cage does require a significant amount of neural retraction to gain access to the disc space (see Fig. 9.25). By moving the posterior trajectory more diagonally, the transforaminal approach preserves the interspinous ligaments as well as the contralateral laminar

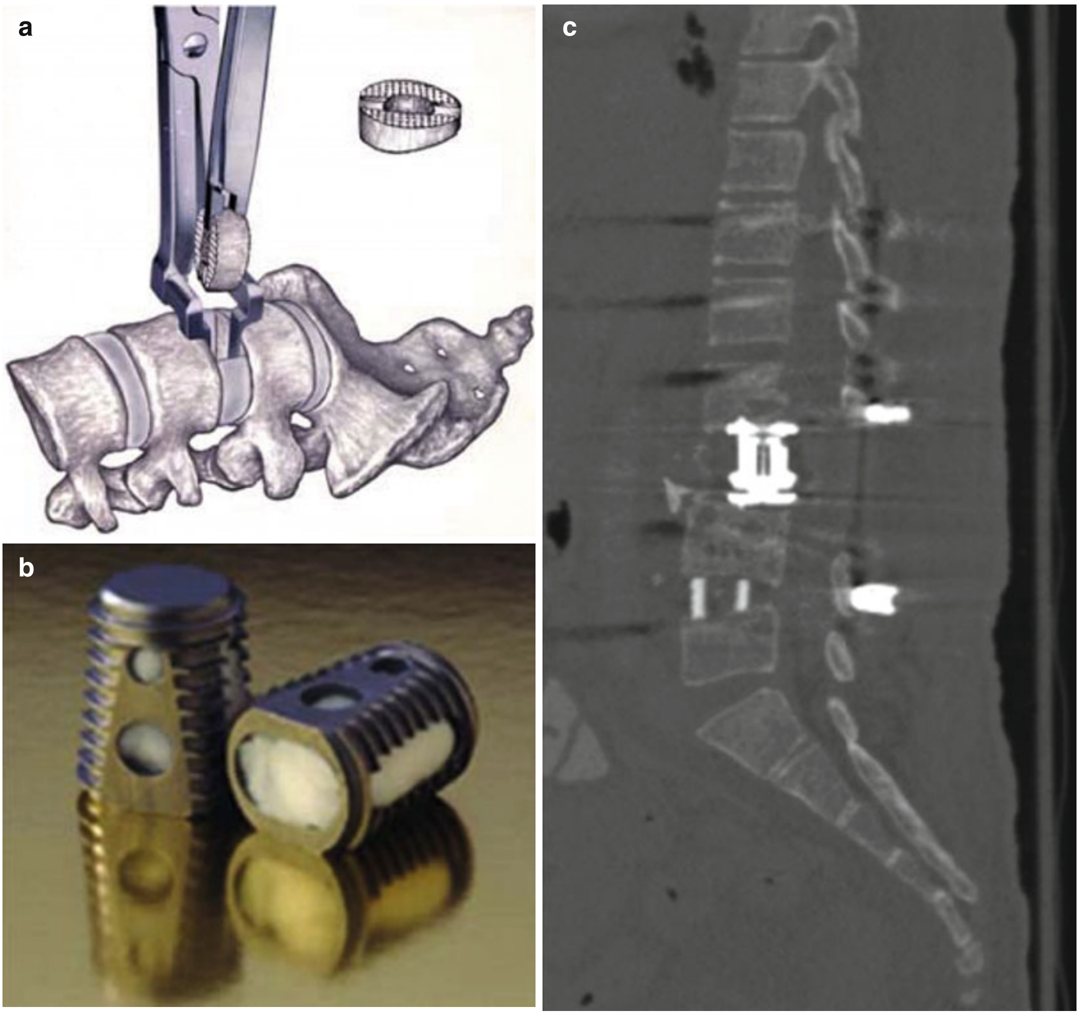


Fig. 9.19 (**a**) Illustration here demonstrates an anterior lumbar interbody fusion with femoral ring allograft. (**b**) After the disc material is removed, the surgeon inserts bone graft material into the disc space, such as autograft or INFUSE® Bone Graft contained in an LT-CAGE® Lumbar Tapered Fusion Device, shown here, to restore the normal anatomic condition of the spine. (**c**) Viewed here is a CT scan with an interbody cage of the lumbar spine. (**d**) The schematic shown here illustrates an expandable cage inserted between two vertebral bodies. Autogenous bone graft is contained within the metal cage

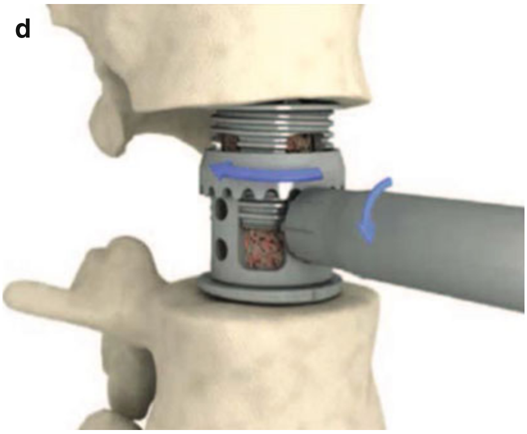


Fig. 9.19 (continued)

surfaces. Additionally, the transforaminal approach, which involves a full unilateral facetectomy, avoids significant retraction of the neural elements (see Fig. 9.26). Oftentimes, fusion surgery is approached from a "360° approach" with both anterior and posterior instrumentation to increase rigidity of the construct [31, 37]. Restoration of anterior column support prolongs instrumentation life, increasing fusion rates irrespective of the number of levels fused [38]. Most failures are the result of poor patient selection or technical difficulties with implantation [36].

Satisfactory outcome relies on a combination of discectomy, decompression, and deformity correction, in addition to achieving a solid fusion [38]. However, adding supplementary fixation reduces spinal motion and increases stiffness compared to stand-alone lumbar interbody fusion (see Fig. 9.27). Gerber et al. found that compared to stand-alone interbody cages, supplementary screw-plate fixation reduced the ROM by a mean of 41 % and supplementary pedicle screw-rod fixation reduced the ROM by a mean of 61 %. Anterior screw-plate fixation of L5–S1 ALIF had only a slightly larger ROM and slightly lower stiffness than L5–S1 ALIF with pedicle screws-rods [31].

Interbody cages have shown to successfully promote fusion in a variety of animal studies and have shown acceptable clinical success rates [36]. Review of the literature concerning long-term results of decompressive procedures indicates short-term failure rates of 15–20 % and about 50 % failure rate by ten or more years after the index procedure. The surgical technique is demanding, fusion rates have been reported up to 96 %, and clinical success up to 86 % patient satisfaction [38].Comparison studies between posterior lumbar interbody fusion and transforaminal lumbar interbody fusion have shown similar operative times, blood loss, and duration of hospital stay in single-level fusions, but more complications were associated with the posterior approach [39].

	Advantage	Disadvantage
ALIF	Allows for large interbody graft to be placed	May need access surgeon
	Can be used as stand-alone device	Must reposition patient if posterior procedure is needed
		Risk of iliac vessel injury
		Risks to lumbosacral plexus leading to retrograde ejaculation
PLIF	No need for access surgeon	Significant amount of dural retraction to place cage(s)
	No need to reposition patient	
TLIF	No need for access surgeon	Can lead to local kyphosis if interbody graft is not placed in anterior ½ of disc space
	No need to reposition patient	Relatively smaller graft compared to DLIF and ALIF
	Less dural retraction than PLIF	
	Facetectomy decompresses exiting and traversing nerve root	
DLIF	No need for access surgeon	Approach is trans-psoas, so can lead to hip flexion weakness
	Allows for large interbody graft to be placed	

9.9 Cages and Plates (for Anterior Fixation)

Anterior instrumentation has its roots in scoliosis surgery. In 1960, Dwyer placed anterior vertebral body screws which were connected by a tensioned wire. This wire was later replaced by a rod that could be locked rigidly to the screws to provide better rotational control and correction [1]. Cadaver biomechanical studies have shown that this system is one of the strongest constructs for anterior spine stabilization [1, 40]. Since the 1960s, the indications for anterior instrumentation have broadened to include fracture stabilization, and with this came the developments of plates. Plates were adapted for use on the spine from their original design for use on the extremities.

Plates (see Fig. 9.28a–c) today are most commonly used anteriorly in combination with cages

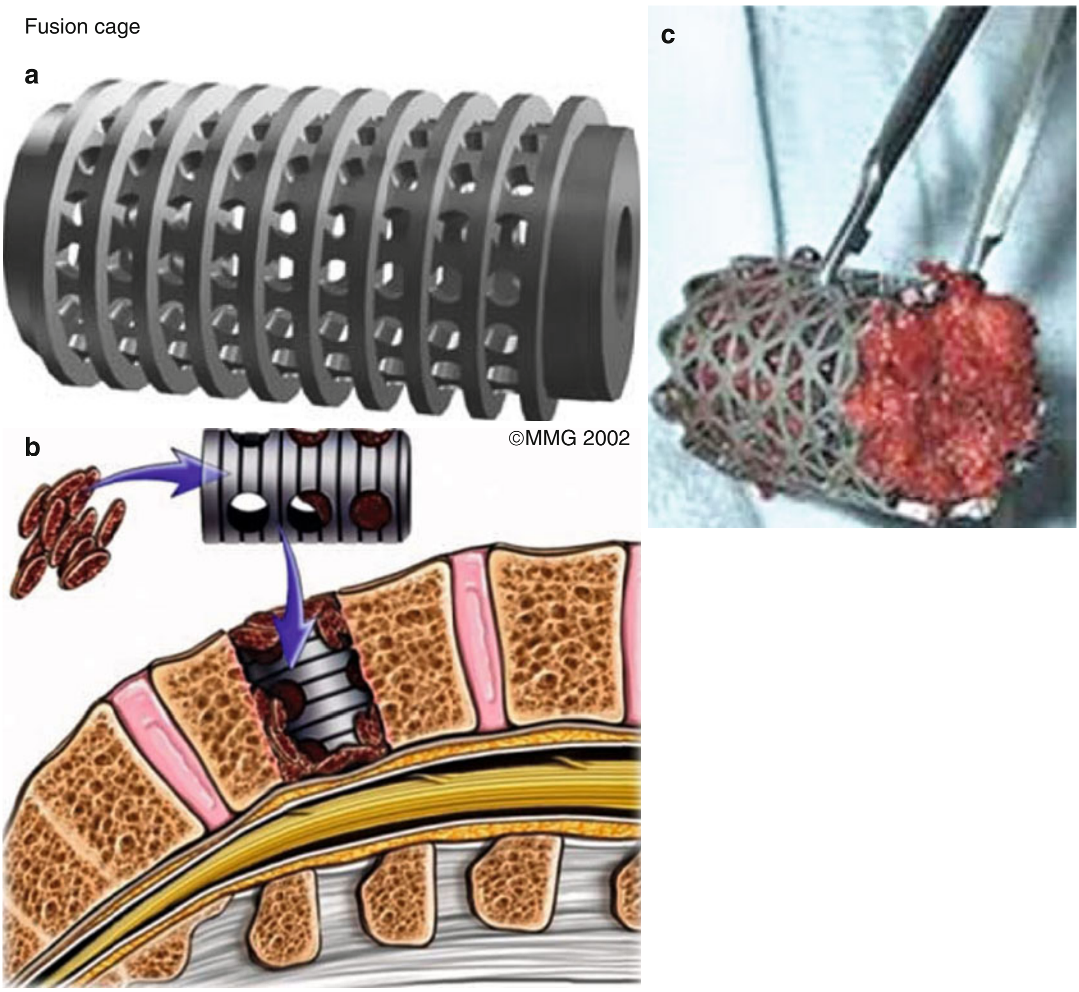


Fig. 9.20 (**a**) The picture shown here is an example of a metal mesh cage used for spinal fusions (©MMG 2002). (**b**) This illustration demonstrates the placement of a mesh cage for anterior interbody fusion. Note that bone graft material is placed within the cage. (**c**) Here is a clinical photograph of a metal cage with autogenous bone graft contained within the cage. (**d**) Shown here is an AP and lateral radiograph of an L2/3 fusion, which was performed using a titanium mesh cage with an autogenous iliac bone graft and Z plate. (**e**) The image shown here is yet another example of a commonly used mesh cage for lumbar spinal fusion. Note the porosity within the cage to promote peripheral bone growth and vascular invasion

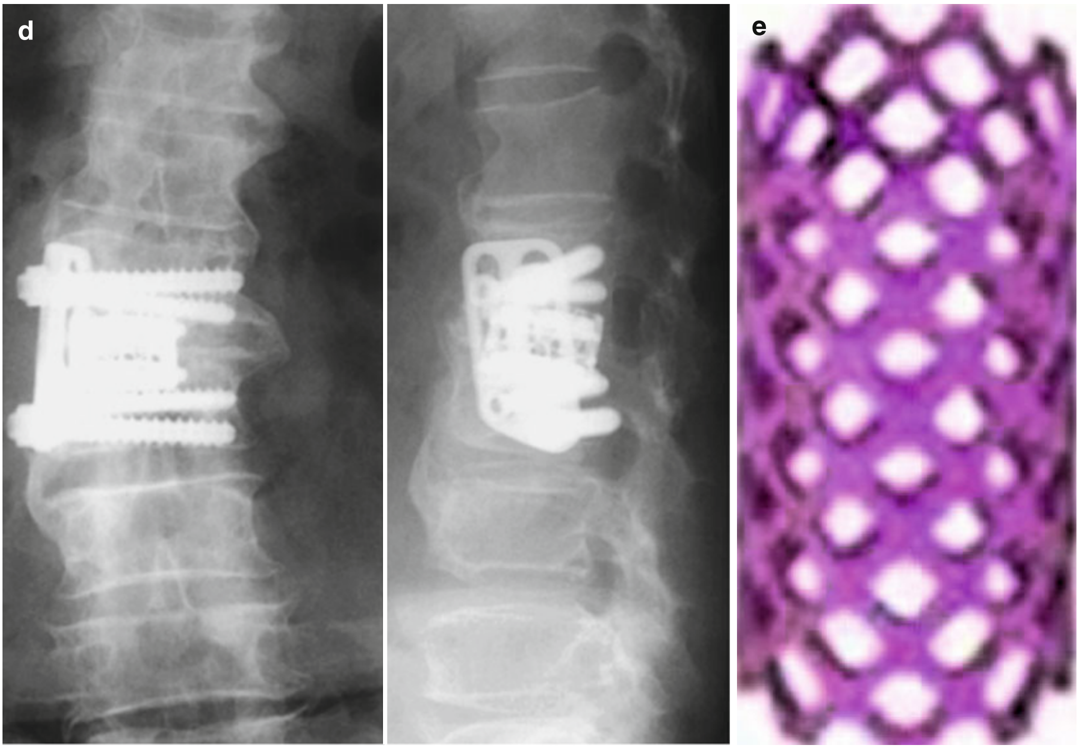


Fig. 9.20 (continued)

for fusions in which the anterior plate places the graft material in compression. Plates can span multiple levels and maintain position and stability of the spine. They do not, however, provide a means to reduce or correct deformity the way pedicle screws do. Indications for use include kyphosis, flat-back syndrome, pseudoarthrosis, and failed posterior surgery. Care must be taken to avoid damaging adjacent soft tissue structures. Screws used in combination with plates should ideally be placed transcortically, which improves their holding power.

Rods connected together can be used in a similar fashion anteriorly. If a single rod is used, the screw (see Fig. 9.29a–c) should be centered longitudinally and transversely within the vertebral body, optimally being parallel to the adjacent end plates in the coronal plane. If a double-rod system or plate is used, two screws should be placed in each vertebral body. Cross-linking with the double-rod Kaneda system enhances mechanical stability [1, 40].

Advantages of anterior plate systems are that they are low profile. However, plates are less stable as a construct than anterior rod systems [40]. Single-rod systems are easy to apply especially when correcting multiple scoliosis levels but are relatively weak biomechanically. Double-rod systems provide superior biomechanical stability but are quite bulky. Any vertebral bone that was removed may be reused and packed into mesh cages. These devices are a weight-bearing device that provides structural anterior column support and increases the surface area of the bone graft that may hasten incorporation [1].

9.10 Lumbar Arthroplasty

Lumbar arthroplasties (see Fig. 9.30a–c) are designed to treat the early stages of degenerative disc disease, which is one of the most common spinal disorders in the population under 65 years of

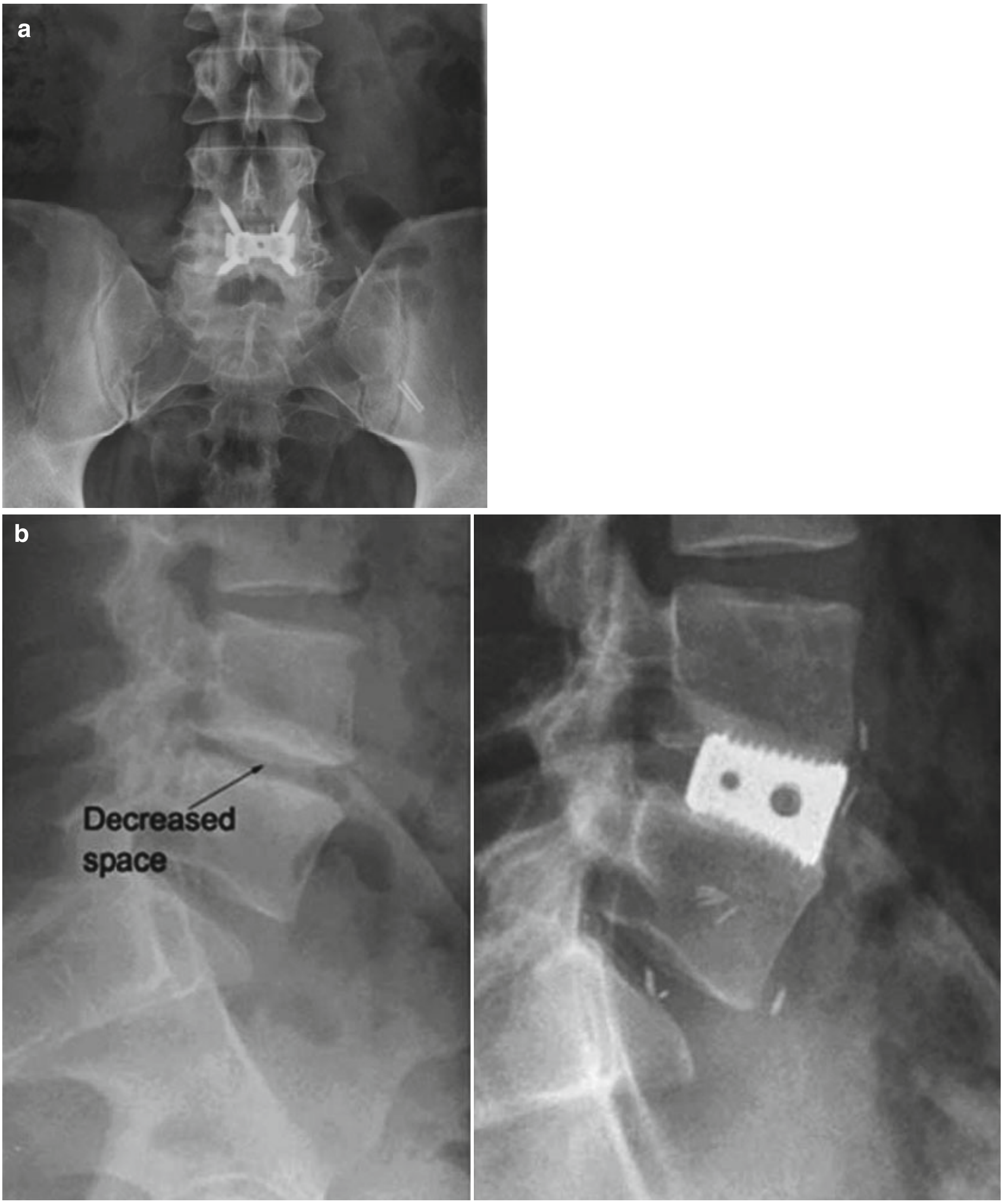


Fig. 9.21 (**a**) This is a postoperative radiograph showing instrumentation for anterior lumbar interbody fusion with an intervertebral cage. (**b**) The image on the *left* is a lateral preoperative radiograph showing decreased disc space between L4 and L5 vertebrae. The image on the *right* is a postoperative radiograph of an interbody cage inserted with an ALIF procedure. The bone graft is contained within the metal cage and cannot be seen. (**c**) Viewed here is another example of an interbody fusion device that is inserted anteriorly with screw fixation into the superior and inferior vertebral bodies. (**d**) Shown here is a postoperative radiograph demonstrating a similar interbody fusion device to the one shown in (**c**)

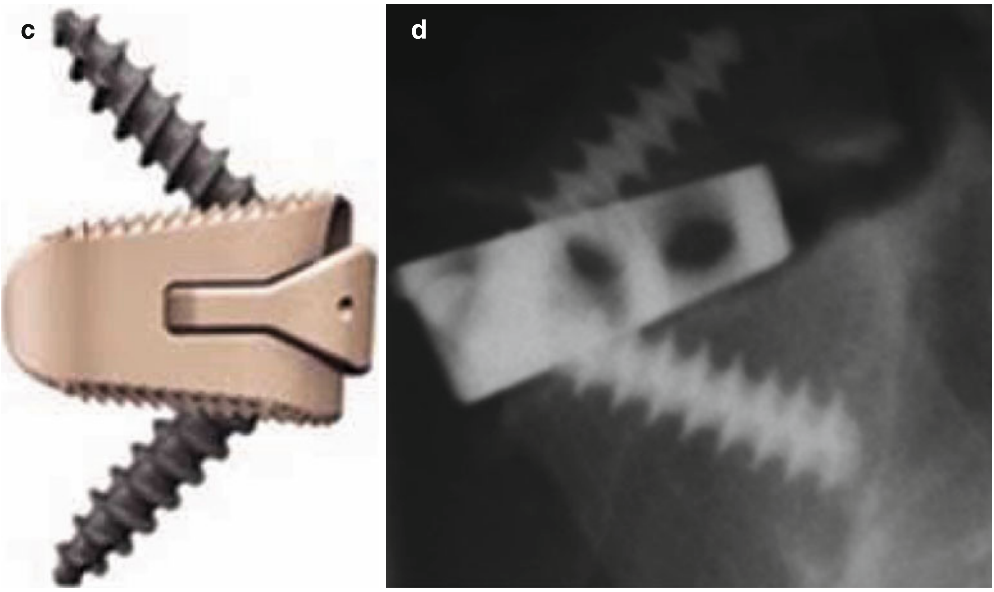


Fig. 9.21 (continued)

age [41, 42]. Despite the excellent short-term results of spinal fusion for traumatic and degenerative spinal disorders, several long-term studies have shown that alteration of the biomechanical environment leads to degenerative changes at adjacent mobile segments [43]. To avoid this problem, an artificial intervertebral disc replacement has been proposed as an alternative to spinal fusion.

Bertagnoli et al. in 2002 prospectively evaluated 134 prosthetic discs using Prodisc II (see Fig. 9.31) in 108 patients for degenerative disc disease. They found that 90.8 % of patients had excellent results and no one had a poor result. Postoperatively, the average vertebral motion was increased in all patients at the operated level; however, degenerative at adjacent levels was noted in ten patients. Patients were able to resume their activities of daily living unaided at an average of 2.3 weeks. They encountered no implant failures or complications due to surgery [44].

Nucleus arthroplasty is an emerging technology that could potentially fill part of the gap in treatment of degenerated discs. Prosthetic devices may be considered an additional therapeutic tool that can be used in selected cases of low back pain secondary to degenerative disc disease [38, 41, 45]. Many various types of arthroplasty devices have been developed to theoretically restore the normal kinematics and load-sharing properties of the natural intervertebral disc [45–47]. Optimal indications for use are disc height >5 mm and degenerative disc changes at an early stage (Pfirmann 2, 3), single-level disease, maintained integrity of posterior facet joints and lack of local anatomical contraindications, and failure of at least 6 months of conservative treatment [41]. Implant migration or dislocation has been one significant disadvantage of these devices [38]; however, few studies have focused on the ideal method of fixation between the prosthetic device and the vertebral body end plates.

NUBAC (see Fig. 9.32) is the first articulating nucleus disc prosthesis, designed to optimally respect the lumbar anatomy, kinematics, and biomechanics. It is constructed in a two-piece manufactured construct from polyetheretherketone (PEEK) with an inner ball/socket articulation. Balsano et al. reported on a 2-year follow-up study on 39 patients who underwent nucleus disc arthroplasty with the NUBAC device. Preliminary results are encouraging. There were no major intraoperative and postoperative complications. Both VAS and ODI scores at 2 years significantly decreased, which provide support that this may be a viable treatment option [41].

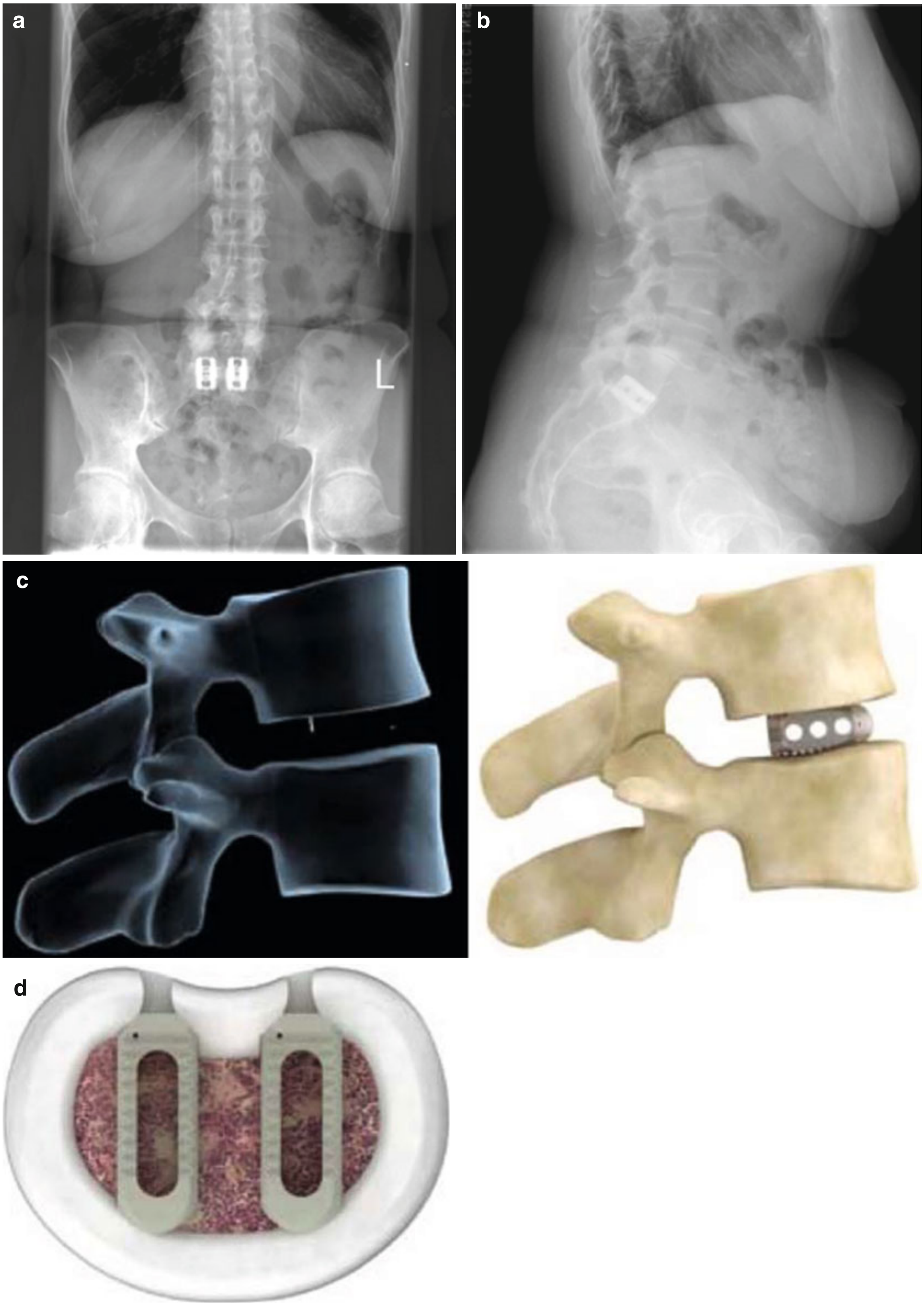


Fig. 9.22 (**a**, **b**) Shown here are postoperative AP and lateral radiographs of a posterior lumbar interbody fusion. (**c**) This image depicts the placement of an interbody device between two lumbar vertebrae in a posterior lumbar interbody fusion procedure. (**d**) Two interbody cages may be placed posteriorly during a lumbar interbody fusion, side by side, to provide biomechanical stability for fusion as shown here in this schematic

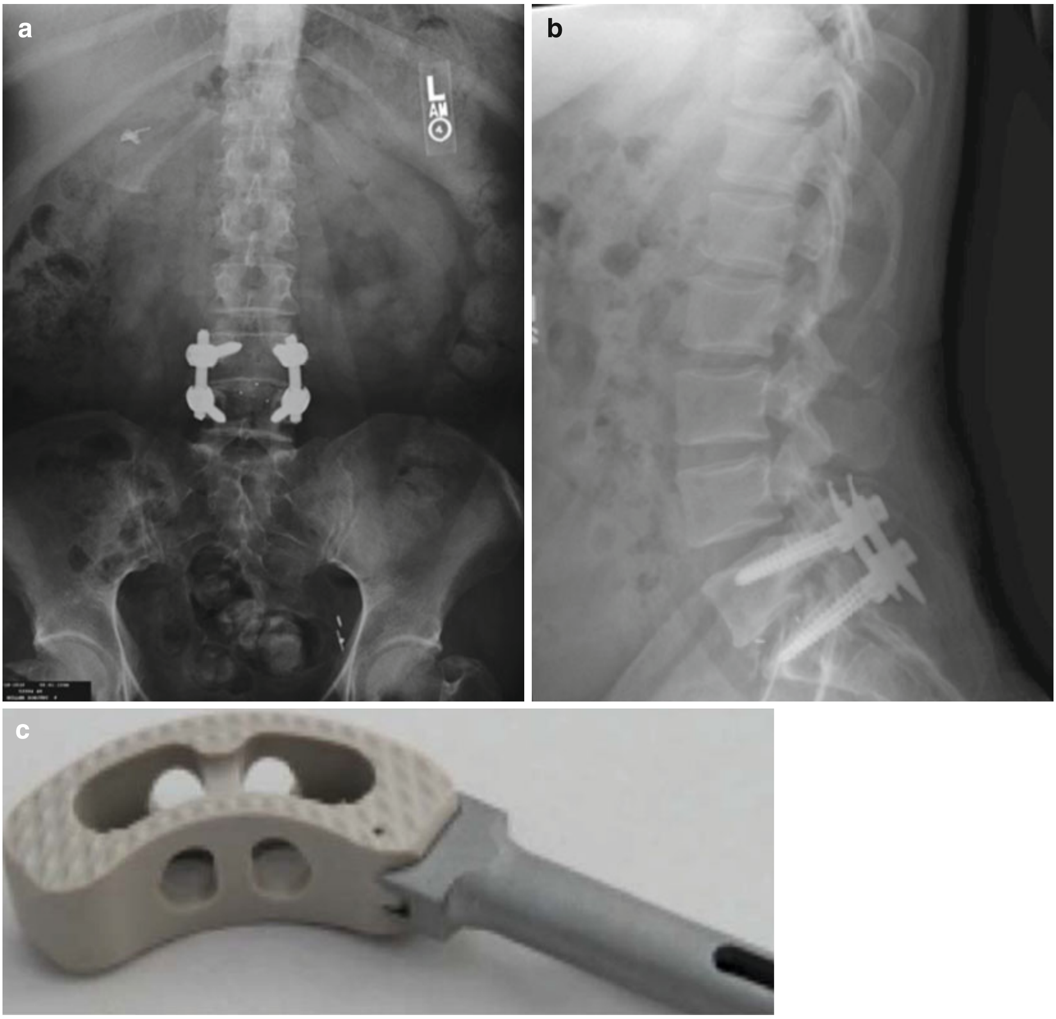


Fig. 9.23 (**a**, **b**) Shown here are an AP and lateral radiograph of a transforaminal lumbar interbody fusion. Note the unilateral facetectomy on the *right*. (**c**) An example of an interbody cage that is used for transforaminal lumbar interbody fusion

9.11 Dynamic Stabilization Devices

Recent developments have been made in posterior pedicle fixation to provide stabilization without fusing vertebral levels for the treatment of degenerative diseases of the lumbar spine. The goal of non-fusion stabilization is to reduce the mobility of the spine segment to less than that of the intact spine while retaining some residual motion. This may be beneficial since preservation of motion theoretically decreases the increased stresses at adjacent levels after fusion, thus potentially minimizing the accelerated rates of adjacent level disease. Pedicle-based posterior dynamic stabilization systems are relatively new; thus, long-term data are lacking in clinical studies, and short-term and midterm data are available for only some of these devices. There is a wide array of posterior dynamic stabilization systems available on the market, and indications for their use have varied substantially, ranging from degenerative disc disease to reconstruction after laminectomy for spinal stenosis. Conclusions cannot yet be drawn regarding the use of posterior dynamic devices compared to fusions [48].

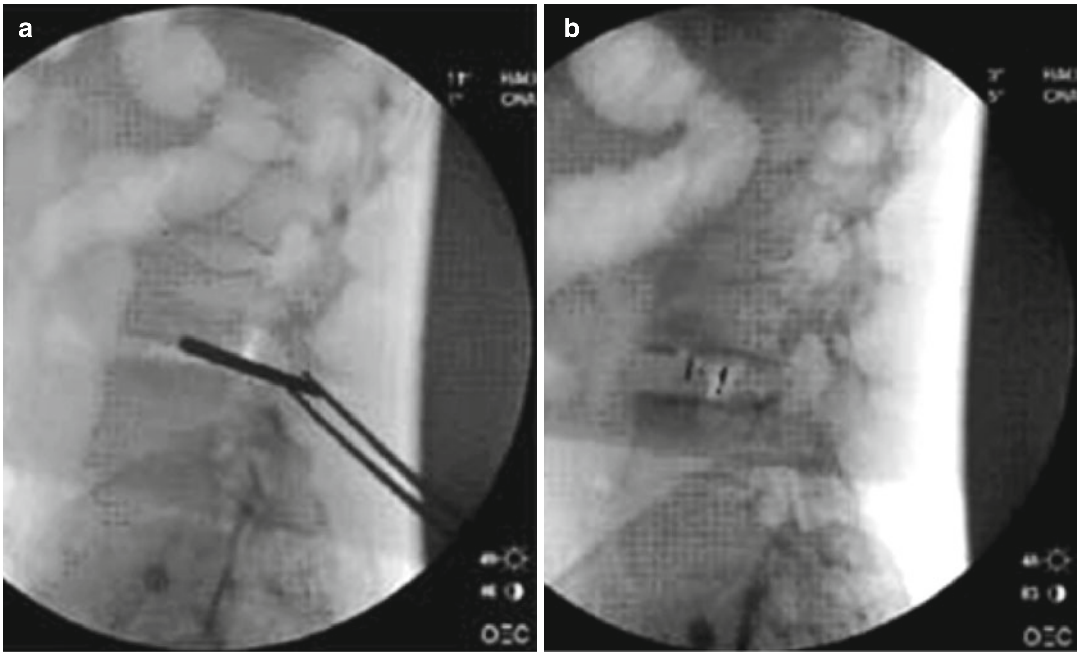


Fig. 9.24 (**a**) The image shown here is an intraoperative fluoroscopy with blunt dilator over the target disc space. The preferred entry location is just anterior to the midline of the vertebral body. (**b**) This intraoperative lateral radiograph shows the placement of an interbody device. Anterior or posterior instrumentation may be added for improved stabilization if needed

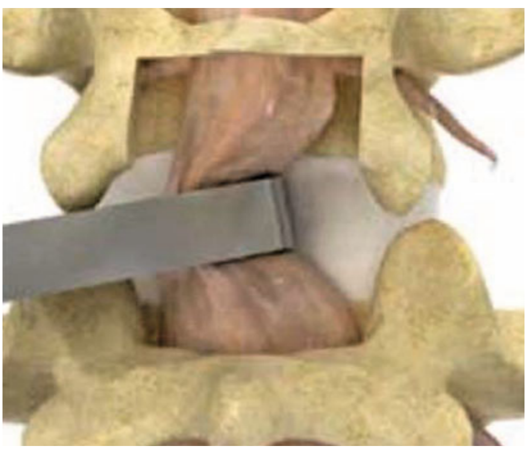


Fig. 9.25 Shown here is the dural retraction that is required during posterior lumbar interbody fusion

Several in vitro studies have been conducted on a dynamic system that is currently available for use, Dynesys (see Fig. 9.33). Segmental ROM was reduced for flexion (less than 20 %), extension (approximately 40 %), and lateral bending (less than 40 %). In torsion, the total ROM was not significantly different from that of the intact level. There are theoretical biomechanical concerns about this device concept. The device is placed at a location posterior to the natural center of rotation of the intervertebral joint, which may preclude this dynamic compliant device from allowing substantial intersegmental motion [49]. Prospective case series have evaluated the radiologic changes in the intervertebral discs after dynamic stabilization. Disc degeneration at the bridged and adjacent segment continued despite dynamic stabilization [50]. Additionally screw loosening is not an uncommon problem. However, the early clinical outcomes of treatment with Dynesys are promising, with decreased pain and disability found at 1-year follow-up. Dynamic stabilization may be preferable to fusion for treatment of degenerative spondylolisthesis and stenosis by decreasing back and leg pain and avoiding the greater tissue destruction and morbidity associated with fusion [51]. However, larger, long-term clinical trials are required before definitive conclusions may be drawn.

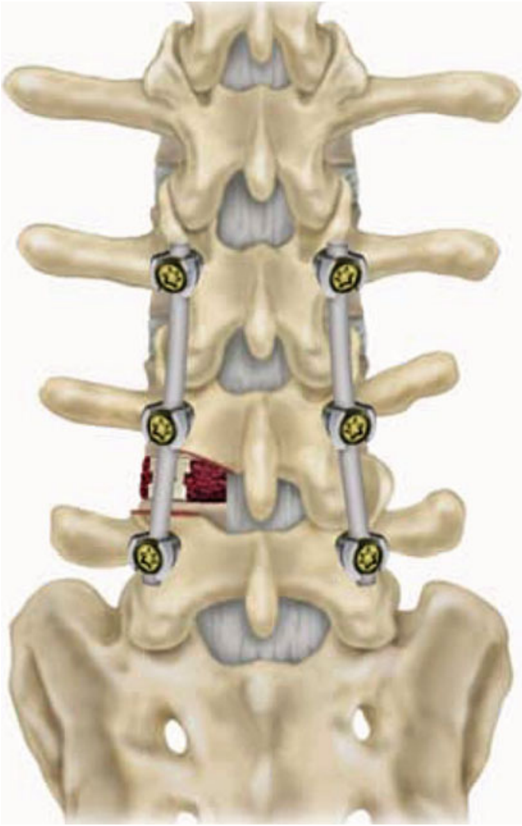


Fig. 9.26 Shown here is a schematic of an open transforaminal interbody fusion and the associated unilateral facetectomy that is part of the procedure. The facetectomy allows placement of the interbody cage without significant retraction of the neural elements

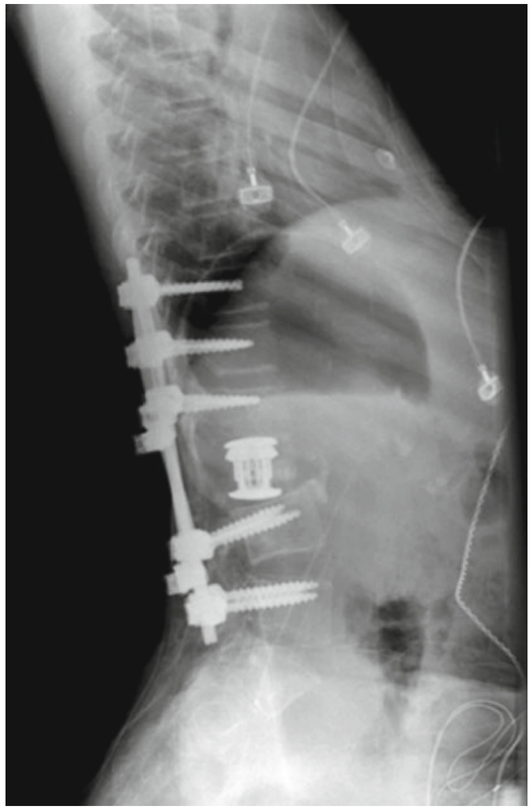


Fig. 9.27 Shown here is a postoperative radiograph of a corpectomy with interbody cage. Note that supplemental posterior instrumentation with pedicle screws and bilateral rod fixation

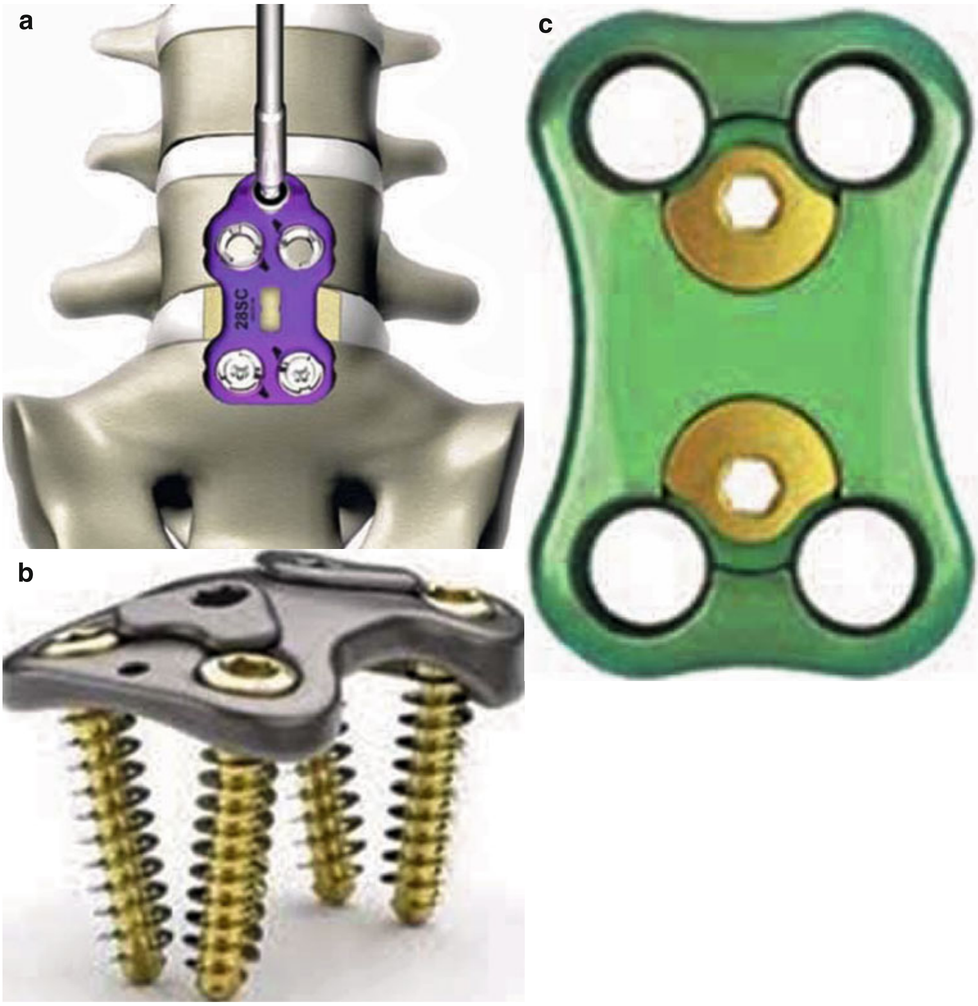


Fig. 9.28 (**a**) Shown here is a schematic of an anterior plate. (**b**) Shown here is another example of an anterior plate that is used for instrumentation in the lumbar spine. (**c**) Viewed here is a four-hole anterior plate

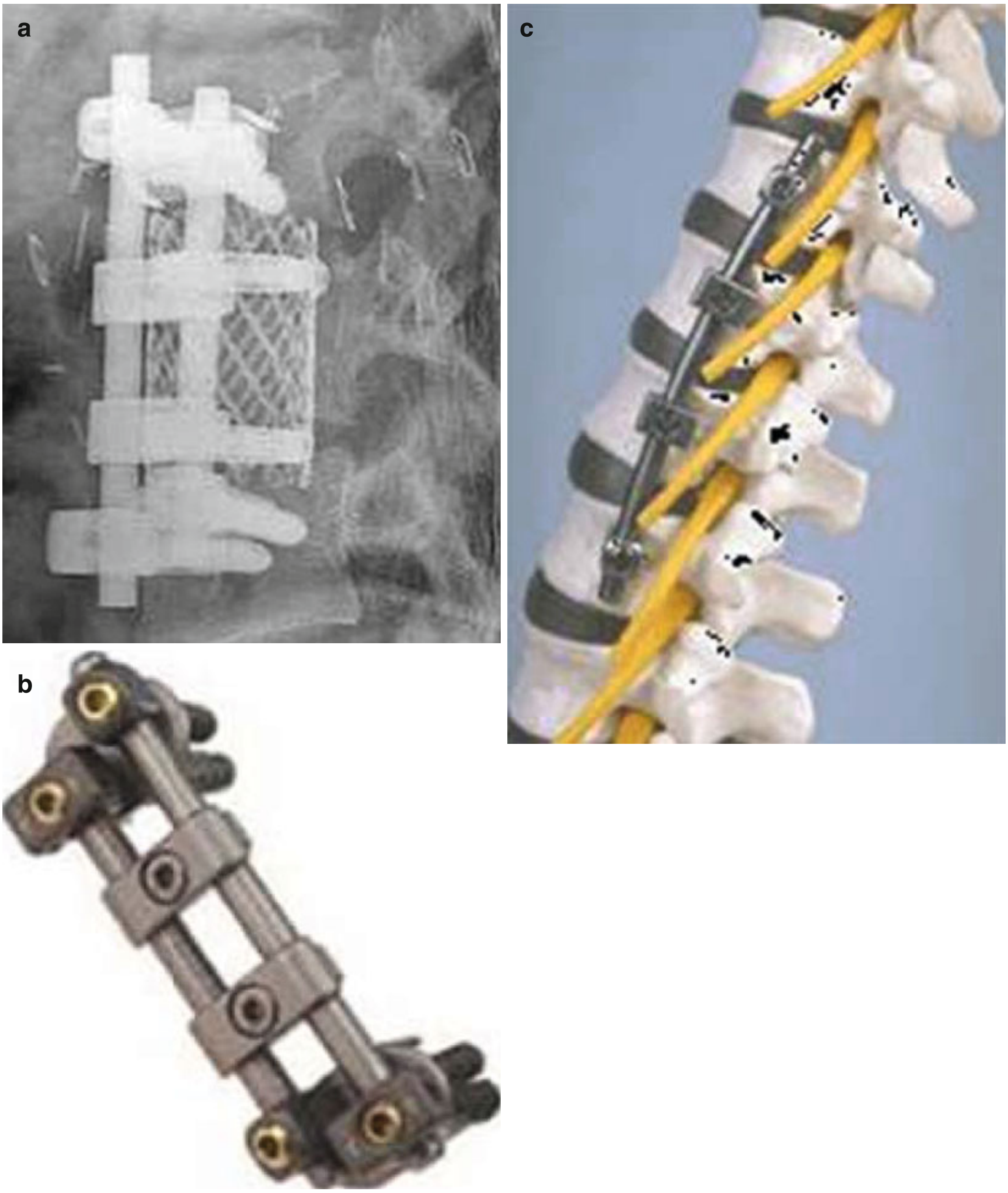


Fig. 9.29 (**a**) The image shown here is a radiograph of an interbody cage with supplemental anterior stabilization with bilateral rods and screw fixation. Note the trajectory of the screws within the vertebral bodies. There are additional cross bars interconnecting the two rods. (**b**) The image here depicts the Kaneda system that is used for anterior scoliosis correction. (**c**) Shown on this spine model is a single rod that is placed laterally along the vertebrae through an anterior approach

Fig. 9.30 (**a**) Here is an AP postoperative radiograph of a lumbar arthroplasty. (**b**, **c**) Here are two more lateral radiographs of two different arthroplasty devices with differing methods of fixation to the vertebrae

Fig. 9.31 The above image is a schematic depicting a lumbar arthroplasty. The two images below it are lateral flexion and extension radiographic images showing the preservation of motion

Fig. 9.32 Shown here is an image of NUBAC, an articulating nucleus disc prosthesis used in the lumbar spine for optimal preservation/restoration of the normal anatomy, kinematics, and biomechanics

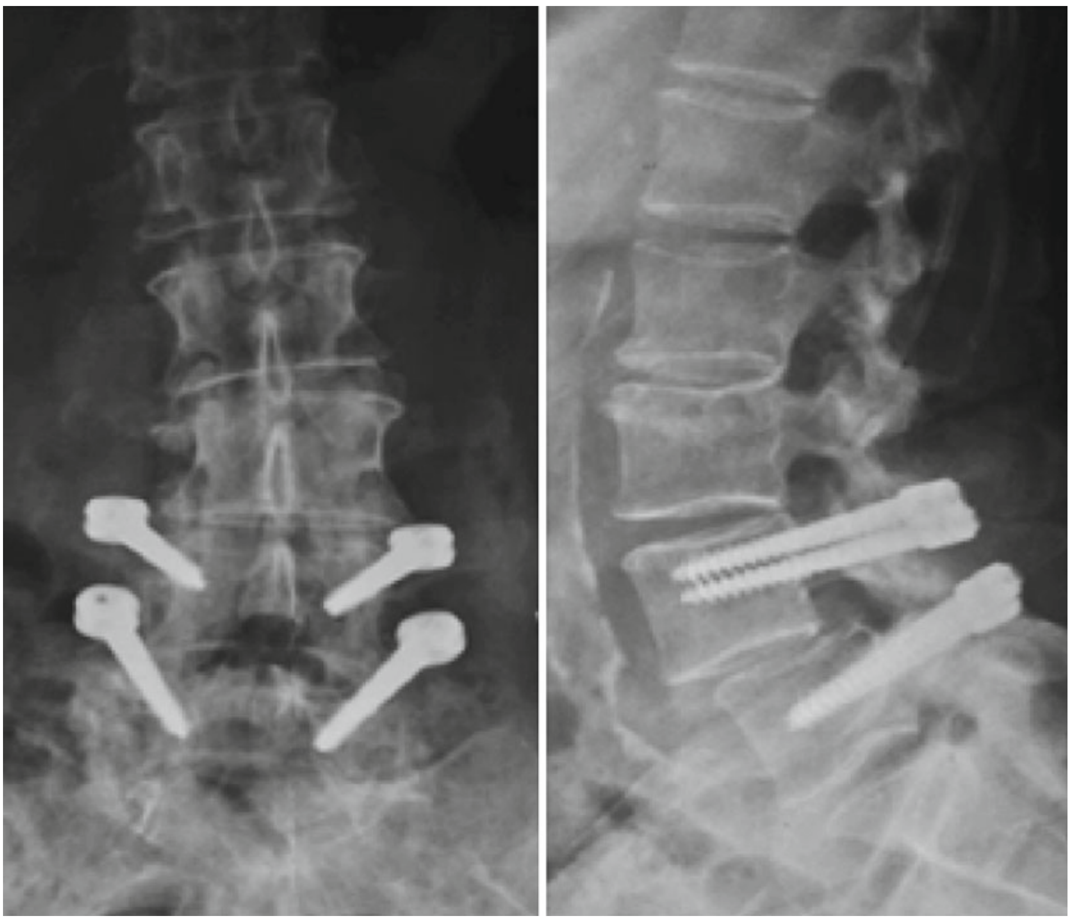


Fig. 9.33 AP and lateral radiograph demonstrating the Dynesys system for lumbar posterior spinal fixation

Conclusion

Spinal fixation devices are used in the lumbar and sacral spine primarily for stabilization, reduction of deformities and fractures, and replacement of vertebral elements affected by tumors or infections. Spinal instrumentation of the lumbosacral spine is necessary for the treatment of many different conditions. The goals of lumbosacral instrumentation include stabilization, deformity correction, reconstruction or replacement, and facilitation and enhancement of fusion.

Internal fixation provides immediate stability; however, the devices are inadequate to withstand prolonged periods of stress and are likely to fail unless a fusion is performed at the time of the instrumentation. Since the development of the first rod, hook, and wire constructs in the early 1960s, various developments and new devices have been invented to help minimize complications and hardware failure and enhance stability and fusion. However, there is a growing trend for instrumentation devices that preserve motion without fusion while avoiding stability, correcting deformity, and decompressing the neural elements. There are many devices utilized for lumbosacral instrumentation, each with their own benefits and drawbacks and indications for usage.

References

1. Bono CM, Garfin SR (2004) Spine, Orthopaedic surgery essentials. Lippincott Williams & Wilkins, Philadelphia, p 343, xiii
2. Kebaish KM (2010) Sacropelvic fixation: techniques and complications. Spine 35(25):2245–2251
3. McAfee PC, Werner FW, Glisson RR (1985) A biomechanical analysis of spinal instrumentation systems in thoracolumbar fractures. Comparison of traditional Harrington distraction instrumentation with segmental spinal instrumentation. Spine 10(3): 204–217
4. Davies AG, McMaster MJ (1992) The effect of Luque-rod instrumentation on the sagittal contour of the lumbosacral spine in adolescent idiopathic scoliosis and the preservation of a physiologic lumbar lordosis. Spine 17(1):112–115
5. Lenke LG et al (1993) Ability of Cotrel-Dubousset instrumentation to preserve distal lumbar motion segments in adolescent idiopathic scoliosis. J Spinal Disord 6(4):339–350
6. Slone RM (1993) Spinal fixation. Part 2. Fixation techniques and hardware for the thoracic and lumbosacral spine. Radiographics 13(3):521–543
7. McCord DH et al (1992) Biomechanical analysis of lumbosacral fixation. Spine 17(8 Suppl): S235–S243
8. Stovall DO Jr et al (1997) Sacral fixation technique in lumbosacral fusion. Spine 22(1):32–37
9. Liljenqvist U et al (2001) Pullout strength of pedicle screws versus pedicle and laminar hooks in the thoracic spine. Acta Orthop Belg 67(2):157–163
10. Slone RM, MacMillan M, Montgomery WJ (1993) Spinal fixation. Part 3. Complications of spinal instrumentation. Radiographics 13(4):797–816
11. Liljenqvist U et al (2002) Comparative analysis of pedicle screw and hook instrumentation in posterior correction and fusion of idiopathic thoracic scoliosis. Eur Spine J 11(4):336–343
12. Skinner R et al (1990) Experimental pullout testing and comparison of variables in transpedicular screw fixation. A biomechanical study. Spine 15(3): 195–201
13. Rose PS (2009) Pedicle screw instrumentation for adult idiopathic scoliosis: an improvement over hook/hybrid fixation. Spine 34(8):852–857; discussion 858
14. Deguchi M (1998) Biomechanical evaluation of translaminar facet joint fixation. A comparative study of poly-L-lactide pins, screws, and pedicle fixation. Spine 23(12):1307–1312; discussion 1313
15. Guyer DW, Wiltse LL, Peek RD (1988) The Wiltse pedicle screw fixation system. Orthopedics 11(10): 1455–1460
16. Leong JC et al (1998) Comparison of the strengths of lumbosacral fixation achieved with techniques using one and two triangulated sacral screws. Spine 23(21): 2289–2294
17. Zdeblick TA (1993) A prospective, randomized study of lumbar fusion. Preliminary results. Spine 18(8): 983–991
18. Heggeness MH, Esses SI (1991) Translaminar facet joint screw fixation for lumbar and lumbosacral fusion. A clinical and biomechanical study. Spine 16(6 Suppl):S266–S269
19. Margulies JY, Seimon LP (2000) Clinical efficacy of lumbar and lumbosacral fusion using the Boucher facet screw fixation technique. Bull Hosp Jt Dis 59(1): 33–39
20. Jacobs RR, Montesano PX, Jackson RP (1989) Enhancement of lumbar spine fusion by use of translaminar facet joint screws. Spine 14(1):12–15
21. Marchesi DG et al (1992) Translaminar facet joint screws to enhance segmental fusion of the lumbar spine. Eur Spine J 1(2):125–130

22. Vanden Berghe L (1993) Stability of the lumbar spine and method of instrumentation. Acta Orthop Belg 59(2):175–180
23. DeVine JG, Gloystein D, Singh N (2009) A novel alternative for removal of the AxiaLif (TranS1) in the setting of pseudarthrosis of L5–S1. Spine J 9(11): 910–915
24. Aryan HE et al (2008) Percutaneous axial lumbar interbody fusion (AxiaLIF) of the L5–S1 segment: initial clinical and radiographic experience. Minim Invasive Neurosurg 51(4):225–230
25. Erkan S et al (2009) Biomechanical evaluation of a new AxiaLIF technique for two-level lumbar fusion. Eur Spine J 18(6):807–814
26. Bono CM, Vaccaro AR (2007) Interspinous process devices in the lumbar spine. J Spinal Disord Tech 20(3):255–261
27. Anderson PA, Tribus CB, Kitchel SH (2006) Treatment of neurogenic claudication by interspinous decompression: application of the X STOP device in patients with lumbar degenerative spondylolisthesis. J Neurosurg Spine 4(6):463–471
28. Harrop JS et al (2009) Iliac bolt fixation: an anatomic approach. J Spinal Disord Tech 22(8):541–544
29. Kuklo TR et al (2001) Minimum 2-year analysis of sacropelvic fixation and L5–S1 fusion using S1 and iliac screws. Spine 26(18):1976–1983
30. Alegre GM et al (2001) S1 screw bending moment with posterior spinal instrumentation across the lumbosacral junction after unilateral iliac crest harvest. Spine 26(18):1950–1955
31. Gerber M et al (2006) Biomechanical assessment of anterior lumbar interbody fusion with an anterior lumbosacral fixation screw-plate: comparison to stand-alone anterior lumbar interbody fusion and anterior lumbar interbody fusion with pedicle screws in an unstable human cadaver model. Spine 31(7):762–768
32. McAfee PC (1999) Interbody fusion cages in reconstructive operations on the spine. J Bone Joint Surg Am 81(6):859–880
33. Sasso RC, Kitchel SH, Dawson EG (2004) A prospective, randomized controlled clinical trial of anterior lumbar interbody fusion using a titanium cylindrical threaded fusion device. Spine 29(2):113–122; discussion 121–122
34. Delloye C et al (2002) Perforations of cortical bone allografts improve their incorporation. Clin Orthop Relat Res 396:240–247
35. Hutter CG (1985) Spinal stenosis and posterior lumbar interbody fusion. Clin Orthop Relat Res 193: 103–114
36. Zdeblick TA, Phillips FM (2003) Interbody cage devices. Spine 28(15 Suppl):S2–S7
37. Lee SH et al (2006) Revision surgery of the lumbar spine: anterior lumbar interbody fusion followed by percutaneous pedicle screw fixation. J Neurosurg Spine 5(3):228–233
38. Enker P, Steffee AD (1994) Interbody fusion and instrumentation. Clin Orthop Relat Res 300:90–101
39. Humphreys SC et al (2001) Comparison of posterior and transforaminal approaches to lumbar interbody fusion. Spine 26(5):567–571
40. Hitchon PW et al (1999) Biomechanical studies on two anterior thoracolumbar implants in cadaveric spines. Spine 24(3):213–218
41. Balsano M (2011) Nucleus disc arthroplasty with the NUBAC device: 2-year clinical experience. Eur Spine J 20(Suppl 1):S36–S40
42. McAfee PC (2004) The indications for lumbar and cervical disc replacement. Spine J 4(6 Suppl):177S–181S
43. Lehmann TR et al (1987) Long-term follow-up of lower lumbar fusion patients. Spine 12(2):97–104
44. Bertagnoli R, Kumar S (2002) Indications for full prosthetic disc arthroplasty: a correlation of clinical outcome against a variety of indications. Eur Spine J 11(Suppl 2):S131–S136
45. McAfee P et al (2007) Treatment of lumbar spinal stenosis with a total posterior arthroplasty prosthesis: implant description, surgical technique, and a prospective report on 29 patients. Neurosurg Focus 22(1):E13
46. Cunningham BW et al (2003) Biomechanical evaluation of total disc replacement arthroplasty: an in vitro human cadaveric model. Spine 28(20):S110–S117
47. Kotani Y et al (2006) Multidirectional flexibility analysis of anterior and posterior lumbar artificial disc reconstruction: in vitro human cadaveric spine model. Eur Spine J 15(10):1511–1520
48. Bono CM, Kadaba M, Vaccaro AR (2009) Posterior pedicle fixation-based dynamic stabilization devices for the treatment of degenerative diseases of the lumbar spine. J Spinal Disord Tech 22(5):376–383
49. Gedet P et al (2009) Comparative biomechanical investigation of a modular dynamic lumbar stabilization system and the Dynesys system. Eur Spine J 18(10):1504–1511
50. Kumar A et al (2008) Disc changes in the bridged and adjacent segments after Dynesys dynamic stabilization system after two years. Spine 33(26):2909–2914
51. Welch WC et al (2007) Clinical outcomes of the Dynesys dynamic neutralization system: 1-year preliminary results. Neurosurg Focus 22(1):E8

Bone Graft and Bone Substitute Biology

10

Harshpal Singh and Allan D. Levi

10.1 Introduction

Human bone grafting dates back to 1668 when Dutch surgeon Job van Meek'ren used a canine cranial xenograft to fill a soldier's cranial defect. Since that time, bone grafting has evolved significantly and has been utilized for a variety of surgical applications. Worldwide, over 2.2 million fusion procedures are performed annually with approximately half a million of those performed in the United States alone [14]. Bone grafting including the development of bone graft substitutes has also evolved into a billion dollar industry, generating approximately 2.5 billion dollars of sales per year [12].

Bone grafting plays a major role in spinal fusion surgery, as it provides the lattice and conduit for bone formation between adjacent segments of the spine. The number of available bone grafting options has exponentially increased, following an equally impressive increase in the number of spinal fusions performed annually. To better understand the different bone grafting options, one must first understand the basic anatomy and physiology of bone and principles of bone fusion.

H. Singh, M.D.
Department of Neurosurgery,
University of Miami, Miami, FL, USA
e-mail: drharshpalsingh@gmail.com

A.D. Levi, M.D., Ph.D., FACS(✉)
Department of Neurosurgery, Neurospinal Services,
Jackson Memorial Hospital, Miami, FL, USA

Department of Neurosurgery,
University of Miami Miller School of Medicine,
University of Miami Hospital, Lois Pope Life Center,
2nd Floor, 1095 NW 14th Terrace (D4-6), Miami,
FL 33136, USA
e-mail: alevi@med.miami.edu

10.2 Bone Physiology and Principles of Bone Fusion

Bone is a porous mineralized structure composed of cells, vessels, and calcium compounds. On a molecular level, it is composed of an organic matrix made of mainly type I collagen that is hardened by deposition of inorganic carbonated hydroxyapatite crystals. Anatomically, the rigid outer layer of bone is called cortical bone and is composed of compact bone tissue with minimal gaps and spaces. It composes approximately 80 % of total bone mass in an adult skeleton and has a porosity ranging between 5 and 30 %. Trabecular or cancellous bone composes the remaining 20 % of bone mass in the adult skeleton and has a comparative 30–90 % porosity [16].

Bone resides in a dynamic state of deposition, resorption, and remodeling; this is regulated by osteoblastic bone formation and osteoclastic bone resorption. Transition among the different states is influenced by a multitude of factors including biomechanics, hormones, physical activity, and nutritional status. Balance among the states typically allows for healthy bone. Imbalance in any direction may lead to pathologically weaker bone, such as osteopenia and/or osteoporosis.

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_10,

Bone fusion is the process by which a bone bridge forms across adjacent bone segments or across a fracture line and is a natural host response to injury after trauma. Surgically induced bone fusion is called arthrodesis, which is the ultimate goal in spinal fusion surgery. Failure of this bone formation results in what is called pseudoarthrosis or nonunion. It may lead to increased medical expenditures and undesirable clinical outcomes including continued pain, disability, and instability [31]. Rates of pseudoarthrosis have been reported to be between 5 and 35 % [19] and have been attributed to many variables including surgeon technique, construct design, biomechanics, metabolic factors, and host-specific factors.

Host responses regulate activation of the necessary components needed for induction of fusion. The three major physiologic processes involved are osteogenesis, osteoinduction, and osteoconduction. The ideal bone grafting material will possess all three physiologic properties.

Osteoconduction is the process by which the bone graft supports the attachment of new osteoblasts and osteoprogenitor cells, thus serving as a scaffold. This is especially important in the first stage of bone healing: a combination of inflammatory responses and capillary bud invasion into the graft, typically occurring in the first 14 days after surgery [10]. The net result is an interconnected network through which vessels can form and cells can migrate. Properties of graft material such as chemical composition, pore size, degree of porosity, stiffness, and architecture heavily influence osteoconduction.

Osteoinduction refers to the ability of the graft to induce proliferation of nondifferentiated stem cells or osteoprogenitor cells into osteoblasts through stimulation by local growth factors [32]. Although allografts and autografts possess osteoinductive potential [17], the most potent osteoinductive effects come from bone morphogenetic proteins (BMPs) and demineralized bone matrix (DBMs) [24].

Osteogenesis refers to the capability of osteoblasts to mineralize and calcify the collagen matrix at the site of new bone formation or more simply stated as the ability to form new bone. This is determined by the presence of viable osteoprogenitor cells and osteogenic precursor cells embedded within the graft itself.

10.3 Bone Grafts

Bone grafts can be divided into two main categories: autologous bone graft and bone graft substitutes. Autologous bone graft (autograft) is defined as bone graft material harvested from the host. It can either be taken locally from the surgical site or from a distant donor site. Bone graft substitutes are synthetic or allograft derived substrates created to enhance fusion. They can be subdivided into many different categories including allografts, ceramics, demineralized bone matrix, osteoinductive factors, autogenous platelet concentrate, mesenchymal stem cells, and gene therapy.

10.3.1 Autologous Bone Graft

Autologous bone is the ideal graft because it possesses all three biological elements of osteoconduction, osteogenesis, and osteoinduction. It is naturally osteoconductive and contains viable embedded stem cells and osteoprogenitor cells, thus being ready for osteogenesis. Lastly, endogenous BMPs provide its highly osteoinductive potential. Autografts have perfect biocompatibility, with no antigenic potential, and remain the gold standard for bone grafting in fusion procedures [22].

Autograft can be harvested locally or from a distant donor site. Local graft can be taken from the spinous processes, lamina, and joints; the amount of autograft harvested depends on the nature of the surgery and degree of decompression. For example, spinous process, lamina, and joint bone can be harvested in surgical cases needing decompression, whereas only spinous process bone can be harvested in cases needing just arthrodesis and without decompression. Distant donor site autograft is most commonly taken from the iliac crest, fibula, or rib [35].

Autogenous bone graft has many benefits including its availability, lack of disease transmission, and zero implant cost. However, autograft material is limited in its supply and carries a notable associated risk of distant donor site morbidity. Iliac crest autograft harvest has been associated with donor site pain persisting in the postoperative period in as high as 60 % of cases [33]. Also, between 2 and 5 % of patients develop wound complications that require reoperation [29]. Other associated morbidities include neurovascular injury, infection, hematoma, seroma, cosmetic deformity, bowel herniation, and iatrogenic fracture [1,18, 28, 30]. In addition to morbidity, obtaining autograft also requires extra surgical time and can be technically demanding.

10.3.2 Bone Graft Substitutes

10.3.2.1 Allograft

Allograft is one of the two most commonly transplanted tissues, second only to blood [25]. The graft is obtained from a deceased donor and subsequently placed through a processing procedure including decontamination, sterilization, and preservation. Because most of the antigenic stimuli are removed during this processing, human leukocyte antigen (HLA) crossmatching is not required [5]. However, very few growth factors and no cells survive processing of the allograft, leaving no osteogenic potential and little osteoinductive potential. Allograft can thereby be said to have good osteoconductive potential, little osteoinductive potential, and no osteogenic potential.

There are many advantages to the use of allograft. It can be available in an almost unlimited quantity and avoids donor site morbidity. Allograft can also be used alone or in conjunction with autograft. It can be used as a structural component of a construct or simply as a ground substrate for fusion. These attributes make it a desirable graft option in large fusion procedures. Major allograft disadvantages include the remote possibility of disease transfer such as HIV or hepatitis C; the HIV transfer rate from allograft bone is estimated at less than one per million [10]. Processing can also lead to changes in the graft properties such as decreased mechanical strength; previous studies have demonstrated up to 50 % loss in mechanical strength of freeze-dried allograft [5]. Allografts may also stimulate a localized immune response that delays the fusion process.

Cortical and Cancellous Allograft

Cortical allografts provide significant mechanical strength and structural support but incorporate slowly into the fusion. A process of periosteal new bone formation occurs around the allograft, which itself may never fully incorporate into the fusion, remaining a mixture of necrotic and viable bone. Alternatively, cancellous allograft provides little mechanical strength but has a much faster rate of fusion incorporation. Bone formation occurs on trabecular surfaces of the graft followed by complete remodeling and more rapid revascularization [13]. Figure 10.1 depicts a fibular allograft in cross section demonstrating cortical bone circumferentially enclosing trabeculated cancellous bone.

Different surgical procedures necessitate different allograft properties. Cortical allografts can be used in applications that require structural support in compression; the use of a femoral ring allograft in an anterior lumbar interbody fusion (ALIF) is an example. Cancellous allografts are more suited for applications needing little mechanical strength but high osteoconductivity. One such example is cancellous chips for posterolateral lumbar fusions.

10.3.2.2 Demineralized Bone Matrix

Demineralized bone matrix (DBM) is an allograft that has been stripped of its mineral composition. This is accomplished through acid extraction of calcium and calcium compounds, leaving behind an organic substrate composed of collagen matrix, noncollagenous proteins, and a low concentration of growth factors. These growth factors are contained within the organic component of bone and are osteoinductive in nature, particularly bone morphogenetic protein (BMP) which is the primary osteoinductive component in DBM. Demineralized bone matrix therefore possesses osteoconductive and osteoinductive potential, but no osteogenic potential.

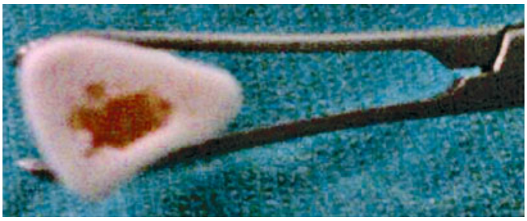


Fig. 10.1 Cross section of a fibular allograft demonstrating cortical shell circumferentially surrounding a trabecular cancellous bone interior. External view of fibular allograft (right demonstrating the dense outer cortical layer)

Advantages of DBM include its various application forms including powder, crushed granules, chips, putty, or gel. It is available in almost limitless quantities, does not require additional surgical time for harvest, and avoids donor site morbidity. DBM also has the least immunogenic potential of the allografts due to its extensive processing but still carries an innate risk of disease transmission. Other disadvantages include questionable quantities of BMP in the graft. High variability in BMP concentrations among different manufacturers of DBM has been demonstrated [2]. Additionally, clinical studies of DBM used for spinal fusion have failed to demonstrate success rates similar to autograft.

10.3.2.3 Osteoinductive Factors (BMP)

Bone morphogenetic proteins (BMPs) are a group of growth factors belonging to the transforming growth factor bet (TGFβ) superfamily of proteins. They play a pivotal role in orchestrating tissue architecture in the body, particularly induction of bone and cartilage (osteoinduction) [26, 27]. BMPs were first discovered in 1965 when Marshall Urist isolated them from demineralized bone extract and implanted them into rabbit connective tissue, resulting in bone formation [34]. However, large amounts of bone and significant costs were required to produce a small amount of BMP until the advent of recombinant DNA technology. Although cost still remains a concern, BMPs are now commercially available in high concentrations and purity. The two most well-studied and most commercially available BMPs are recombinant human BMP-2 (rhBMP-2) and BMP-7 (rhBMP-7).

Recombinant human BMPs are produced in a liquid form that easily dissolves and subsequently inactivates in vivo. Because of this, the clinical application of BMP requires the presence of a carrier vehicle that allows a high concentration capacity and time-controlled release. Multiple carrier vehicles have been used, but the most commonly used and studied carrier is a type I collagen sponge [35].

Bone morphogenetic protein has been FDA approved for use only in titanium cylindrical cages during anterior lumbar interbody fusion (ALIF) procedures. Compared to iliac crest autograft, rhBMP-2 use in cylindrical threaded fusion cages demonstrated a greater reliability in achieving arthrodesis [6]. Significantly higher fusion rates in rhBMP-2 supplemented anterior interbody cages compared with iliac crest autograft have also been shown (94.4 % compared with 89.4 %; $p=0.022$) [8]. A prospective, multicenter, U.S. Food and Drug Administration-approved investigational device study demonstrated a 98 % fusion rate at 6-year follow-up [7]. As described, BMP is a potent osteoinductive agent for arthrodesis and as such has been used frequently in an off-label manner [23].

Disadvantages of bone morphogenetic protein have also been extensively described in the literature [4]. As previously described, rhBMP induces bone formation through chemotactic, pro-inflammatory, and osteogenic pathways. Overactivity of these same pathways is the most commonly reported complication and includes osteolysis, swelling, seroma, heterotopic bone formation, and antibody reaction. Osteolysis was originally recognized as transient radiolucencies surrounding interbody cages or bone dowels and may be attributed to bony end plate violation, construct stability, and BMP dose [9,15]. Heterotopic ossification has been attributed to fragments of rhBMP left in the area of the foramen and has been mostly associated with off-label BMP use in transforaminal (TLIF) or posterior lumbar interbody fusion (PLIF) applications. Radiculitis is another possibly related complication that is seen more frequently in cases where rhBMP has been used [28].

10.3.2.4 Synthetic Grafts

Ceramics

Ceramics are an osteobiologic class of bone graft substitutes that are named after the genera of coral that they mimic. The striking similarity between microscopic structures of marine invertebrate coral and bone was first described in 1975 [11]. Since that time, numerous synthetic grafts have been designed and utilized for grafting purposes. In order to be classified as a ceramic bone graft material, the material must meet the following qualities: tissue and mechanical compatibility, stability in body fluids, ability to withstand sterilization, and capability to be molded into functional shapes. On a molecular level, they can be composed of hydroxyapatite, tricalcium phosphate, bovine collagen, natural coral, calcium carbonate, or a combination of these [10].

Compared to allograft, ceramics do not carry the potential for disease transmission and usually do not induce local inflammatory processes. They are also available in an unlimited quantity, tend to cost less, and can be machined to fit into a variety of shapes and sizes. But because they are synthetic substances, ceramics only function as an osteoconductive scaffold and lack both osteogenic and osteoinductive properties. Ceramics also lack mechanical strength, being brittle and susceptible to shearing forces, thus being better utilized as graft expanders used in conjunction with internal fixation [21].

Cages

Cages are synthetic interbody spacers that have capacity to contain bone grafting materials. Initially, the cage was developed for purposes of interbody fusion in horses [3]. The first cages were cylindrical stainless steel baskets filled with autologous bone graft and placed via an anterior approach. Subsequent generations of the interbody cage have evolved in both composition and shape. Newer cages can be composed of various materials including polyetheretherketone (PEEK), carbon fiber (CF), and titanium. Shapes also vary and include the bullet shape, rectangle, and trapezoid. These different conformations allow the graft to be placed through different approaches including anterior, lateral, and dorsal [20].

References

1. Arrington ED, Smith WJ, Chambers HG et al (1996) Complications of iliac crest bone graft harvesting. Clin Orthop Relat Res 329:300–309
2. Bae HW, Zhao L, Kanim LE (2006) Intervariability and intravariability of bone morphogenetic proteins in commercially available demineralized bone matrix products. Spine 31(12):1299–1306, discussion:1307–8
3. Bagby GW (1988) Arthrodesis by the distraction-compression method using a stainless steel implant. Orthopedics 11(6):931–934
4. Benglis D, Wang MY, Levi AD (2008) A comprehensive review of the safety profile of bone morphogenetic protein in spine surgery. Neurosurgery 62(5 Suppl 2):ONS423–ONS431
5. Berven S, Tay BK, Kleinstueck FS et al (2001) Clinical applications of bone graft substitutes in spine surgery: consideration of mineralized and demineralized preparations and growth factor supplementation. Eur Spine J 10(Suppl 2):S169–S177
6. Boden SD, Zdeblick TA, Sandhu HS, Heim SE (2000) The use of rhBMP-2 in interbody fusion cages. Definitive evidence of osteoinduction in humans: a preliminary report. Spine (Phila Pa 1976) 25(3):376–381
7. Burkus JK, Gornet MF, Schuler TC, Kleeman TJ, Zdeblick TA (2009) Six-year outcomes of anterior lumbar interbody arthrodesis with use of interbody fusion cages and recombinant human bone morphogenetic protein-2. J Bone Joint Surg Am 91(5):1181–1189
8. Burkus JK, Heim SE, Gornet MF, Zdeblick TA (2003) Is INFUSE bone graft superior to autograft bone? An integrated analysis of clinical trials using the LT-CAGE lumbar tapered fusion device. J Spinal Disord Tech 16:113–122
9. Burkus JK, Sandhu HS, Gornet MF (2006) Influence of rhBMP-2 on the healing patterns associated with allograft interbody constructs in comparison with autograft. Spine 31:775–781
10. Cheng I, Oshtory R, Wildstein MS (2007) The role of osteobiologics in spinal deformity. Neurosurg Clin N Am 18(2):393–401
11. Chiroff RT, White EW, Weber KN et al (1975) Tissue ingrowth of replamineform implants. J Biomed Mater Res 9:29–45
12. Desai BM (2007) Osteobiologics. Am J Orthop 36:8–11
13. Ehrler DM, Vaccaro AR (2000) The use of allograft bone in lumbar spine surgery. Clin Orthop Relat Res 371:38–45
14. Giannoudis PV, Dinopoulos H, Tsiridis E (2005) Bone substitutes: an update. Injury 36:S20–S27
15. Glassman SD, Howard JM, Sweet A, Carreon LY (2010) Complications and concerns with osteobiologics for spine fusion in clinical practice. Spine (Phila Pa 1976) 35(17):1621–1628
16. Hall SJ (2007) Basic biomechanics, 5th edn. Mosby-Year Book, St. Louis, p 88

17. Kalfas IH (2001) Principles of bone healing. Neurosurg Focus 10(4):E1
18. Khanna G, Lewonowski K, Wood KB (2006) Initial results of anterior interbody fusion achieved with a less invasive bone harvesting technique. Spine 31(1):111–114
19. Ludwig S, Kowalski J, Boden S (2000) Osteoinductive bone graft substitutes. Eur Spine J 9(Suppl 1):s119–s125
20. McAfee PC (1999) Interbody fusion cages in reconstructive operations on the spine. J Bone Joint Surg Am 81(6):859–880
21. Miyazaki M, Tsumura H, Wang JC, Alanay A (2009) An update on bone substitutes for spinal fusion. Eur Spine J 18(6):783–799
22. Mummaneni PV, Lenke L, Haid RW (2008) Spinal deformity: a guide to surgical planning and management. Quality Medical Pub, St. Louis
23. Ong KL, Villarraga ML, Lau E, Carreon LY, Kurtz SM, Glassman SD (2010) Off-label use of bone morphogenetic proteins in the United States using administrative data. Spine (Phila Pa 1976) 35(19): 1794–1800
24. Pilitsis JG, Lucas DR, Rengachary SS (2002) Bone healing and spinal fusion. Neurosurg Focus 13(6):e1
25. Prolo DJ, Rodrigo JJ (1985) Contemporary bone graft physiology and surgery. Clin Orthop 200:322–342
26. Reid JJ, Johnson JS, Wang JC (2011) Challenges to bone formation in spinal fusion. J Biomech 44(2):213–220, Epub 2010 Nov 10
27. Rihn JA, Gates C, Glassman SD et al (2008) The use of bone morphogenetic protein in lumbar spine surgery. J Bone Joint Surg Am 90:2014–2025
28. Rihn JA, Gates C, Glassman SD, Phillips FM et al (2009) The use of bone morphogenetic protein in lumbar spine surgery. Instr Course Lect 58:677–688
29. Rihn JA, Kirkpatrick K, Albert TJ (2010) Graft options in posterolateral and posterior interbody lumbar fusion. Spine (Phila Pa 1976) 35(17):1629–1639
30. Sawin PD, Traynelis VC, Menezes AH (1998) A comparative analysis of fusion rates and donor-site morbidity for autogenetic rib and iliac crest bone grafts in posterior cervical fusions. J Neurosurg 88: 255–265
31. Schnee CL, Freese A, Weil RJ et al (1997) Analysis of harvest morbidity and radiographic outcome using autograft for anterior cervical fusion. Spine 22(19): 2222–2227
32. Steinmann JC, Herkowitz HN (1992) Pseudarthrosis of the spine. Clin Orthop Relat Res 284:80–90
33. Subach BR, Haid RW, Rodts GE (2001) Bone morphogenetic protein in spinal fusion: overview and clinical update. Neurosurg Focus 10(4):E3
34. Summers BN, Eisenstein SM (1989) Donor site pain from the ilium. A complication of lumbar spine fusion. J Bone Joint Surg Br 71:677–680
35. Urist MR (1965) Bone: formation by autoinduction. Science 150:893–899
36. Vaccaro AR, Chiba K, Heller JG, Patel TC, Thalgott JS, Truumees E, Fischgrund JS, Craig MR, Berta SC, Wang JC, North American Spine Society for Contemporary Concepts in Spine Care (2002) Bone grafting alternatives in spinal surgery. Spine J 2(3): 206–215

11 Neurological Monitoring in Orthopedic Spine Surgery

Tod B. Sloan, Leslie Jameson, Daniel Janik, and Paul Mongan

Abbreviations

APB	Abductor pollicis brevis
AH	Abductor hallucis
CMAP	Compound muscle action potential
CN	Cranial nerve
CST	Corticospinal tract
DEP	Dermatomal evoked potential
ECG	Electrocardiogram
EMG	Electromyography
Hz	Hertz
H reflex	Hoffman reflex
IOM	Intraoperative neurological monitoring
MAC	Minimal alveolar concentration
MEP	Motor evoked potentials
mA	milliamperes
MUP	Motor unit potentials
NMEP	Neurogenic motor evoked potentials
NMJ	Neuromuscular junction
RLN	Recurrent laryngeal nerve
SRS	Scoliosis Research Society
SSEP	Somatosensory evoked potentials
TA	Tibialis anterior
TIVA	Total intravenous anesthetic

T.B. Sloan, M.D., MBA, Ph.D. (✉) • L. Jameson, M.D.
D. Janik, M.D. • P. Mongan, M.D.
Department of Anesthesiology,
University of Colorado School of Medicine,
MS B113 Leprino Office Building (LOB), 12401 E 17th Avenue, Aurora, CO 80045, USA
e-mail: tod.sloan@ucdenver.edu

11.1 Introduction

Intraoperative neurological monitoring (IOM) during orthopedic surgery to correct spinal abnormalities is used in many centers because surgeons have recognized that these techniques enhance their intraoperative decisions thereby improving outcome. In particular, the improvement in outcome with the correction of scoliosis has led to IOM becoming a standard of care in axial skeletal surgery. The value of IOM is that it allows a functional assessment of the nervous system during anesthesia, whereas traditional physiological monitors (e.g., blood pressure and oxygenation) only measure parameters that are supportive of function. As such, IOM can detect an unfavorable surgical or physiological environment that allows corrective maneuvers to reduce neurological risk. This chapter will review the methods commonly used in spinal column corrective surgery; most frequently, several of these methods are combined to provide as much information as possible during a procedure.

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_11, © Springer-Verlag Berlin Heidelberg 2014

11.2 Wake-Up Test and Clonus

Prior to the application of electrophysiological techniques, the "wake-up" test described by Vauzelle was used with Harrington rod distraction in scoliosis to prevent the paralysis resulting from correction of the deformity [131]. In this test, the curvature was corrected and the anesthesia reduced to awaken the patient. The patient was asked to move their hands to signify they understood the command and then asked to move their legs. This procedure worked poorly with patients who were not amenable to such tests (e.g., cognitively impaired individuals). In addition, there were occasional injuries that occurred during the wake-up [12]. This technique was particularly effective in scoliosis since there was a single insult to spinal integrity (e.g., distraction) such that correction could be made if the patient awakened without function.

There is still controversy regarding the question of whether the wake-up test should be performed if sensory or motor evoked response monitoring is also performed [23]. One author suggested that in children, "electrophysiological monitoring has now superseded the wake up test as an index of spinal cord function" [133]. In several studies, the wake-up test usually correlates with the somatosensory evoked potential (SSEP) [12]. In one study of 1,168 cases, the two always correlated, and the authors concluded that "there is probably no need to use wake-up testing" [40]. However, other authors point out the occasional false-negative case and continue to recommend wake-up testing [12–14] particularly for confirming possible injury when the monitoring techniques cannot be acquired or when IOM becomes persistently abnormal during a surgical procedure [17, 37, 68, 98, 115].

An alternative test that does not require active patient participation is the ankle clonus test. It has been conducted similar to the wake-up test in the middle of a procedure as well as at the conclusion of surgery [49]. The ankle clonus reflex is assessed by performing a rapid forced dorsiflexion of the foot and then holding slight tension on the foot in the dorsiflexed position. A positive response is seen as rhythmic contractions of the gastrocnemius muscle resulting in plantar flexion of the foot. It is mediated by the first sacral nerve root which activates the spinal stretch reflex arc. Not present in normal awake individuals, it is seen because of a temporary loss of central inhibition of the spinal stretch reflex because anesthesia wears off such that lower-motor-neuron function returns first, with inhibitory (cortical) impulses returning later. Hence, it can be elicited early during the reversal of anesthesia, before the patient is responsive to verbal stimuli. If, however, there is an injury of the spinal cord, spinal shock causes a diminution of lower motor-neuron impulses, resulting in a loss of the ability to produce ankle clonus. Thus, the presence of clonus during a wake-up test has been used to signal the absence of a cephalad spinal injury and avoid the need to further awaken the patient. It has also been used at the conclusion of surgery to get an early indication of motor integrity before the patient fully awakens. Of note is that clonus may return asymmetrically, and if the patient awakens rapidly, the window of clonus may be missed due to a prompt return of higher center inhibition. One study of 1,006 patients undergoing Harrington rod insertion for scoliosis revealed no false negatives and three false positives (presumed to be missed windows of testing) [49].

One major drawback of the wake-up and clonus tests is that they are conducted at one point in time. As procedures have evolved, multiple potential insults occur rather than the single insult of distraction. As such, a more continuous method of assessment has been considered desirable (e.g., somatosensory and motor evoked potentials and electromyography as below) to identify specific problems, so they can be identified and corrected as they occur.

11.3 Somatosensory Evoked Potentials

One of the first continuous electrophysiological techniques used in spine surgery was the somatosensory evoked potential (SSEP). Among the orthopedic innovators were Clyde Nash, M.D., and Richard Brown, Ph.D., whose seminal work established its value in scoliosis [89]. In this technique, peripheral sensory nerves are stimulated, and the very small electrical responses that are produced are recorded as the signal is transmitted through the spinal cord to the brain. The series of waves that are produced can be used to identify neurological problems occurring along the neurological pathway. This is similar to the use of the electrocardiogram (ECG) identifying problems along the conducting pathway in the heart. Unfortunately, unlike the robust electrical signals of the ECG, the signals of the SSEP are very small and require a digital computer with signal averaging to identify them among the much larger "noise" of the other electrical activity in the body (e.g., electroencephalogram).

The method of signal averaging involves stimulating the nervous system and measuring the response for a set period of time following the stimulus that contains the evoked response of interest. A second stimulation follows, and the electrical activity at each time point following the stimulation is averaged. This is repeated at 2–7 Hz for several hundred times, and the evoked response becomes apparent. This occurs because the random unwanted background activity (noise) averages to zero since it is unrelated to the stimulus, and the desired signal builds because it follows the stimulation by a set time (it is related to the stimulus). Technically, the ratio of the signal to noise builds by the square root of the number of stimulations averaged. The smaller the response is compared to the noise, the larger the number of averages required and the longer the time needed to acquire the response. Other methods, such as differential amplifiers and filtration, are also used to improve the acquisition by removing unwanted noise from the original signal before averaging. The time for averaging the response allows updating of the SSEP every 1–2 min.

The evoked response consists of a plot of voltage over time with a series of peaks and valleys (Fig. 11.1). Peaks may be positive or negative depending on the specific electrodes used for acquisition and are thought to arise from specific neural generators (often more than one neural structure). The information recorded is usually the amplitude (peak to adjacent trough) and the time from the stimulation to the peak (called latency). The SSEP peaks are usually named by polarity and latency, P (positive) or N (negative), and followed by the latency in milliseconds (e.g., N_{20}).

For monitoring, the team will identify the peaks that are cephalad to the surgical site (e.g., the response of the brain during spine surgery). They are then monitored during the surgery to identify the onset of an insult by decreases in amplitude or increases in latency from the initial values (baseline). Usually, both upper and lower extremity responses are monitored in IOM. For cervical surgery, both can be used to indicate a potential problem in the operative area. For thoracic or lumbar surgery, the upper extremity SSEP can be used as a control for factors such as anesthesia as well as monitoring for positioning problems in the arms.

Often multiple recording sites are used to allow diagnosis of which portion of the neural tract involved is affected so as to understand the pathophysiology of the change and help guide corrective measures. As a general principle, amplitude reduction of 50 % or more, or a latency increase of 10 % or more is considered significant for the cortical SSEP peak, although smaller changes may indicate impending compromise. Studies suggest that a slow loss of amplitude (and increase in latency) may be due to diffuse neural ischemia. Fast losses (with minimal latency change) may be due to mechanical injury or focal ischemia, especially in spinal gray matter [93, 122].

The SSEP is produced following stimulation of peripheral sensory pathways using a 200–300-μs square wave impulse of 20–30 mA delivered by pairs of subdermal needles near large, mixed motor and sensory nerves. Typical nerves used include (and their component spinal roots) the median (C6–T1) or ulnar (C8–T1) nerve stimulated at the wrist and the common peroneal (L4–S1) stimulated at the head of the fibula or posterior tibial (L4–S2) nerve stimulated at the ankle. Stimulation activates predominantly the large-diameter, fast conducting Ia muscle afferent and group II cutaneous nerve fibers (i.e., both motor fibers and sensory fibers) [5]. This results in a muscle response seen as a foot or hand twitch

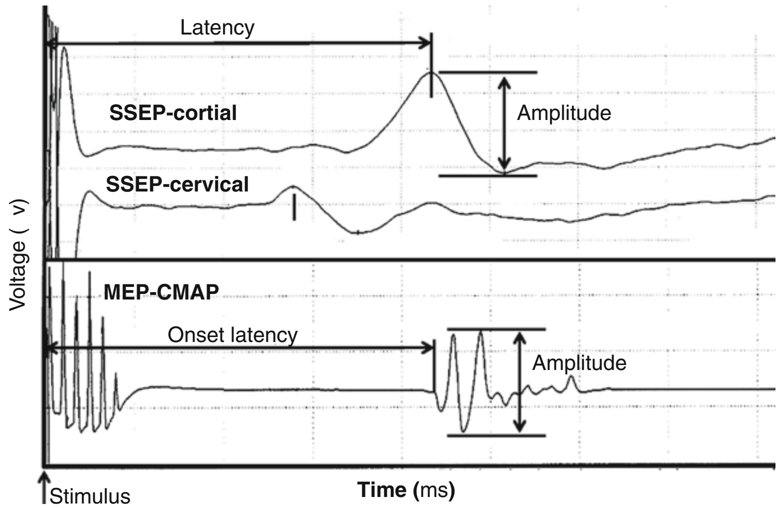


Fig. 11.1 Typical SSEP and MEP tracings. The upper two tracings are typical median nerve SSEP tracings recorded over the sensory cortex (*top*) and over the cervical spine. The peaks recorded are indicated, and the latency (time from stimulus to the peak) and amplitude (from the peak of interest to an adjacent trough) are marked for the cortical peak. Below is a typical muscle recording (*CMAP*) of the MEP in the lower extremity. Shown is the measurement of onset latency (time from stimulation to the first deflection from baseline) and the amplitude (the voltage difference form the highest peak to the lowest trough)

that signifies stimulation and may produce an artifact on the pulse oximeter or ECG but only rarely produces sufficient patient movement to interfere with surgery. The sensory fiber activation produces the response which produces the SSEP.

It is currently thought that the incoming volley of neural activity from stimulation represents activity primarily in the pathway of proprioception and vibration. These responses enter the spinal cord via the posterior nerve roots and ascend in the ipsilateral dorsal column. The first synapse in these pathways occurs near the nucleus cuneatus (for the upper extremities) and the nucleus gracilis (for the lower extremities) at the cervicomedullary junction. The responses then cross the midline and ascend through the brainstem via the contralateral medial lemniscus.

A second synapse occurs in the ventral posterolateral nucleus of the thalamus. From there, the responses ascend to the contralateral sensory cortex. The most commonly recorded responses are those recorded over the sensory cortex; however, the responses can also be recorded at other points along the pathway. For the upper extremity, the evoked responses can be measured from electrodes placed over the antecubital fossa, supraclavicular fossa (brachial plexus), cervical spine, and cortex; for the lower extremity, they can be recorded over the popliteal fossa (for posterior tibial nerve), along the spinal cord (surface or epidural electrodes), and at cervical and cortical locations (Fig. 11.2) [52].

The cortical response is usually recorded using subdermal needles or scalp surface electrodes placed over the primary somatosensory cortex appropriate for the nerve stimulated. Typically for spinal column surgery, the SSEP is monitored over the cervical spine (if not in the operative field) as well as the cerebral cortex. The major cortical peaks recorded after median n. and posterior tibial n. stimulation (N_{20} and P_{38}, respectively) are likely the result of the thalamocortical projections to the primary sensory cortex [5]. Responses recorded posteriorly over the cervical spine probably represent responses of the tracts in the spinal cord or brainstem [5]. In some surgeries, epidural electrodes proximal and distal to the operative site can be used to monitor the SSEP transmission through the spinal cord.

The basis of operative monitoring in humans is founded in studies in animals where generalized insults to the spinal cord (e.g., distraction,

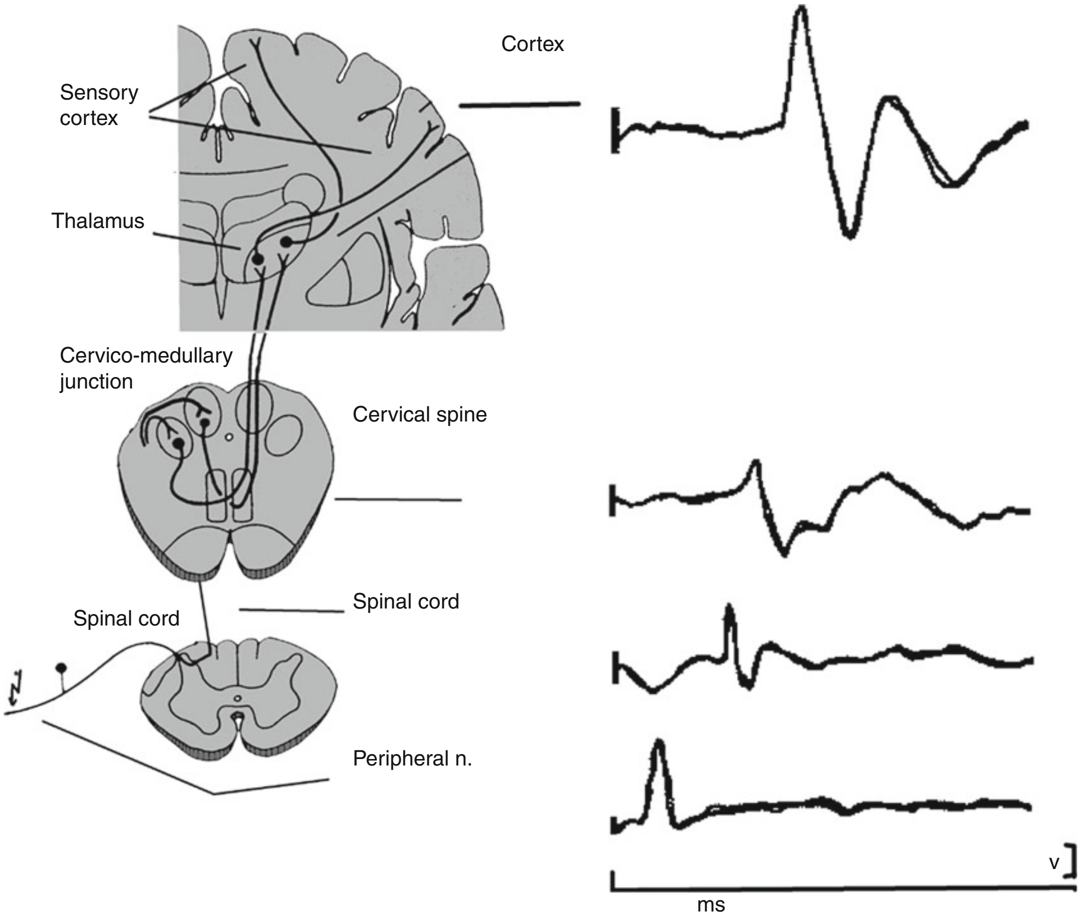


Fig. 11.2 The SSEP is produced by stimulation of a peripheral nerve (*arrow*). The electrical activity enters via the dorsal nerve root and ascends the spinal cord via the dorsal column pathway which mediates the senses of proprioception and vibration. It synapses at the cervico-medullary junction and crosses the midline ascending in the medial lemniscus to the ventral posterolateral nucleus of the thalamus where it has a second synapse. From there, it ascends to the sensory cerebral cortex. SSEP recordings can be made along the pathway; shown are responses from the peripheral nerve, spinal cord, cervical spine, and cerebral cortex (Reproduced from Jameson with permission [52])

graded weights applied directly on the spinal cord [22] or narrowing of the spinal canal anteriorly at C5 using an epidural screw [60]) is associated with SSEP latency and amplitude changes simultaneous to loss of clinical motor function. Thus, these insults impact the functioning of both the posterior SSEP sensory pathway and the anterior motor pathways.

When the SSEP is used during spinal cord or axial surgery, it can identify mechanical or ischemic insults when they result in alteration or loss of transmission through the surgical field (Fig. 11.3) [88]. The current risks of neurological morbidity in spinal surgery without monitoring are not minimal, anterior cervical diskectomy 0.46 %, scoliosis correction 0.25–3.2 %, and intramedullary spinal cord tumor surgery 23.8–65.4 %; an estimated 50–80 % reduction in morbidity is estimated to occur with monitoring [21].

The utility of the SSEP in spinal surgery was shown in a 1995 landmark analysis conducted by the Scoliosis Research Society (SRS) and the European Spinal Deformities Society. They evaluated the results of monitoring during correction of spinal deformity in 51,263 cases (scoliosis, kyphosis, fractures, and spondylolisthesis) by 173 surgeons [92]. In these cases, the overall

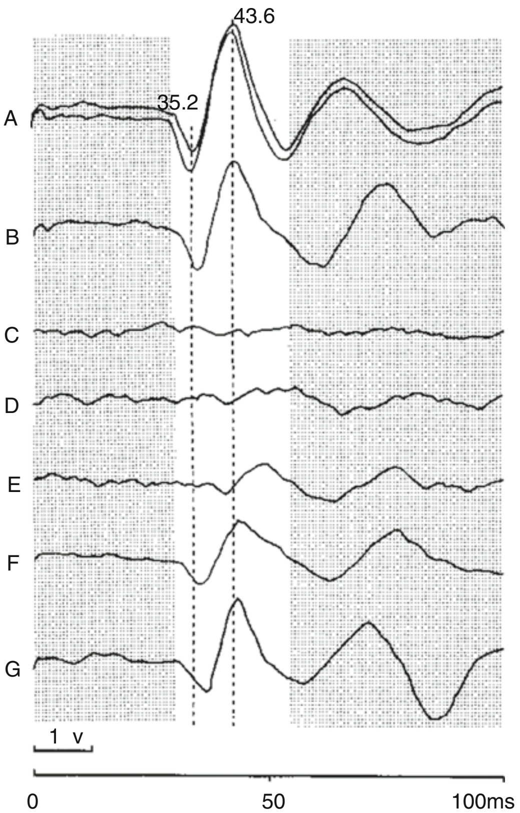


Fig. 11.3 Example of SSEP monitoring during spinal surgery. (*A*) Normal baseline recordings the afternoon prior to operation; (*B*) after induction of anesthesia; (*C*) responses were abolished after passing through the wires around the laminae; (*D*) wake-up test was positive; (*E*) after 15 min, poorly defined potentials reappeared; (*F*) after closure of wound evoked potentials showed a little increased latency P_{40} and N_{50} measuring 4.1 and 2.0 ms, respectively, with normal overall waveform (*G*) (Reproduced with permission from Mostegl et al. [88])

occurrence of paraplegia was 0.55 % (1 in 182 cases), well below the 0.7–4 % historical average expected for these cases with instrumentation without monitoring. The incidence of definite false-negative responses (i.e., in which the patient sustained a major motor injury without SSEP warning) was rare (0.063 % or about 1 case in 1,500 procedures). The economic impact of monitoring was also assessed and revealed that the cost of monitoring enough cases to prevent one major, persistent neurological deficit ($120,000.00 for 200 cases, 1995 dollars) is small compared to the cost of lifelong medical care for that patient [91, 92]. Other authors have conducted similar cost analyses and concluded that properly applied monitoring can be cost-effective [94].

Numerous other studies demonstrate an improvement in outcome with monitoring after spinal surgery such that the SRS developed a position statement that made monitoring a virtual standard of care during axial skeletal and spinal cord procedures [6]. They stated that "neurophysiological monitoring can assist in the early detection of complications and possibly prevent post-operative morbidity in patients undergoing operations on the spine." This was echoed in the British literature by Loughnan: "Today, it is standard practice to conduct some form of monitoring when performing any spinal operation that is associated with a high risk of neurological injury. Generally, operations are considered to carry such a risk when corrective forces are applied to the spine, the patient has pre-existing neurological damage, the cord is being invaded, or an osteotomy or other procedure is being carried out in immediate juxtaposition to the cord" [74]. Hence, SSEP monitoring has become commonplace and almost a de facto standard of care during a wide variety of spinal column procedures.

Studies in humans undergoing spinal surgery indicate that the SSEP is predictive of neural outcome [12, 82, 134]. However, the correlation of SSEP and neural injury is not exact; cases of motor injury without intraoperative SSEP warning have occurred. A major reason for dissociation of the SSEP from motor injury is the fact that the SSEP is predominantly transmitted via the posterior columns where the blood supply is from the posterior spinal artery. The motor tracts are located more anteriorly, and the blood supply is via the anterior spinal arteries. Therefore, spinal corrective surgeries where the SSEP predicts motor deficits are cases where the mechanism of injury affects both the posterior and anterior spinal cord.

The value of the SSEP during surgery is not isolated to procedures on the spinal column. For example, the SSEP technique has been considered indispensable for intraoperative evaluation and monitoring during surgical procedures on peripheral nerves and plexus regions. It has been reported to detect potential nerve injury related to positioning (usually a stretch or pressure injury); ulnar nerve injury, thought to be as high as 4.8 % in the prone position, can be detected by recording the response to ulnar nerve stimulation in the wrist or forearm. The SSEP has also been used to detect cortical ischemia from carotid occlusion during anterior cervical spine surgery [123], and it can identify physiological insults (e.g., hypotension).

Monitoring using the SSEP is possible in children although there are age-related changes due to maturation of the tracts. The conduction velocity increases from birth to age four at which time the growth of body size and nerve length results in increased latencies until adult values are obtained. The effect is more pronounced in the lower extremity response than in the upper extremity response. Since maturation proceeds at different rates in different regions (proceeding more slowly in central regions), the morphology of the SSEP changes from a wide cortical peak to the more defined peak of adulthood by 3–4 years.

11.4 Dermatomal Evoked Potentials

Several variations of the SSEP have been developed in order to overcome some of the anatomic limitations. One problem of the traditional SSEP is that the response travels to the cord via several nerve roots. Hence, the SSEP may not reflect pathology when only one component nerve root is affected. The dermatomal evoked potential (DEP) is produced by stimulation of cutaneous dermatomal regions using surface electrodes [43, 124]. The specific anatomic paths stimulated by the DEP are unknown, but the amplitude of the cortically recorded response appears to be related to the somatotopic representation of the dermatome. Using this method, individual nerve roots can be tested. Unfortunately, controversy surrounds the exact dermatome distribution in some regions (e.g., L5), poor amplitude of the responses in areas of regions with small cerebral representation (such as thoracic region), and marked anesthetic depression limits the application of this technique. As such, electromyographic techniques have replaced the DEP in many surgeries for monitoring individual nerve roots.

11.5 Electromyography

Using muscle responses to monitor neural tracts is referred to as electromyography (EMG). The EMG is generally large enough that averaging is not necessary making it possible to provide immediate feedback. The EMG is recorded by placing two needle electrodes near or in a muscle and displaying the electrical waveforms on a video monitor. The response may also be converted to sound to give immediate audible feedback. Monitoring can be conducted as passive, "free-running" EMG, where continuous activity is observed and recorded, or as "stimulated" EMG, where an electrical stimulus is applied to a nerve and the response of the muscle recorded. EMG electrodes may record activity of only 1–2 % of the muscle fibers in a given muscle. For example, the monitored response in the tibialis anterior is generated by only 18–34 of the 270,000 muscle fibers [69]. The most commonly monitored nerves with spinal surgery are in the cervical (C2–C7) and lumbosacral (L2–S2) regions and in the vocal cords (recurrent laryngeal n.) (Table 11.1).

EMG responses are recordings of the motor unit potentials (MUP) of individual axon-muscle groups. A normal EMG during light general anesthesia has low-amplitude high-frequency activity which disappears as anesthesia is deepened. Hence, activity during anesthesia is abnormal and represents irritation or impending injury of the nerve innervating the muscle. Neurotonic discharges, caused by mechanical or metabolic stimuli, are high-frequency intermittent or continuous bursts of MUPs (Fig. 11.4) [102]. Activity can be bursts lasting less than 200 ms with single or multiple MUPs firing at 30–100 Hz, or they can be long trains of activity lasting 1–30 s or longer [45].

The short bursts (heard as brief "blurps" on a loudspeaker) (Fig. 11.4a) represent relatively synchronous motor unit discharges that result from a single discharge of multiple axons from

Table 11.1 Nerve roots and muscles most commonly monitored

Nerve root		Muscle
Cranial nerve		
Anterior neck	CN. X	Vagus, recurrent laryngeal n.: vocal folds, cricothyroid m.
	CN. X1	Trapezoids, sternocleidomastoid
Spinal cord		
Cervical	C5, 6	Biceps, deltoid
	C6, 7	Flexor carpi radialis
Thoracic	C8–T1	Adductor pollicis brevis, abductor digiti minimi
	T5–6	Upper rectus abdominis
	T7–8	Middle rectus abdominis
	T9–11	Lower rectus abdominis
	T12	Inferior rectus abdominis
Lumbar	L2	Adductor longus
	L2–4	Vastus medialis
Lumbosacral	L4–S1	Tibialis anterior
	L5–S1	Peroneus longus
Sacral	S1–2	Gastrocnemius, abductor hallucis
	S2–4	Anal sphincter

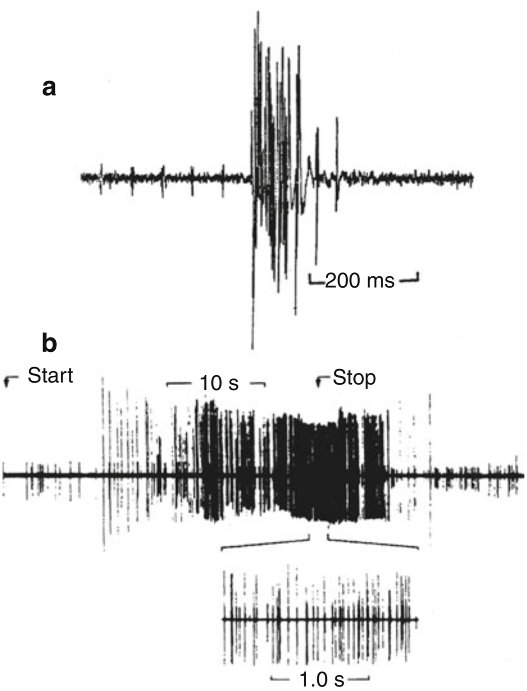


Fig. 11.4 EMG recordings during surgery near motor nerves. The upper trace (**a**) shows a single, brief burst of muscle activity from nerve irritation. The lower trace (**b**) shows sustained "neurotonic" activity from injurious nerve irritation. Note the difference in timescales (Reproduced from Prass with permission [102])

nerve irritation. When these are of sufficient amplitude, they raise concern. Causes of irritation include mechanical stimulation (e.g., nearby dissection, ultrasonic aspiration, or drilling), nerve retraction, thermal irritation (e.g. heating from irrigation, lasers, drilling, or electrocautery), and chemical or metabolic insults. Long trains of continuous, synchronous motor unit discharges (Fig. 11.4b) are associated with impending nerve injury (nerve compression, traction, or ischemia of the nerve). Their audible sounds have a more musical quality and have been likened to the sound of an outboard motor boat engine, swarming bees, popping corn ("popcorn"), or an aircraft engine ("bomber"). These long trains of neurotonic activity are associated with significant stretch and or compression by retractors or surgical position (e.g., spine distraction, dural tear with nerve rootlet herniation). Here prompt action is necessary, or postoperative damage is likely leading to motor dysfunction (motor fibers) or chronic postoperative pain syndromes (sensory fibers) [36, 48].

Electromyography during spinal column surgery is more sensitive for detecting radiculopathy than the SSEP [48]. SSEP may fail to alert the surgeon to individual nerve root damage since the loss of only one of the multiple nerve roots transmitting the SSEP may not alter the SSEP cortical response [69]. As such, free-run EMG monitoring has been recommended for monitoring for impending nerve root injury. The incidence of nerve root injury during lumbar spine surgery is 0.2–31 % usually from retraction or instrumentation leading to mechanical or vascular compromise [76]. During spine surgery, a radiculopathy may also be caused when surgery involves a percutaneous approach to the spine traversing near nerves or through a nerve plexus (e.g., transverse lumbar interbody fusion) and nerves are irritated or stretched. When traversing tissues near nerves, electrified probes can also be used to stimulate nerves whose proximity is nearer than expected.

EMG monitoring has achieved widespread usage as a means for reducing the risk (15–25 %) of injury to nerve roots during the placement of pedicle screws [18]. EMG testing can identify unfavorable screw placement when the pedicle screw or screw hole is stimulated using an electrified monopolar probe. The current intensity that is needed to activate the nearby nerve root and produce a muscle response correlates with the placement of the screw. If the screw is contained entirely within the pedicle, a high current is necessary to activate the nerve and muscle. If the screw has breached the pedicle wall, a lower current will activate the nerve, and if the screw has exited the pedicle and is near or adjacent to the nerve, a very low current will activate the nerve [129]. Hence, the current necessary to activate the nerve and muscle (threshold) is an index of the placement of the screw. It is important to note that stimulation of screws with swivel attachments must be made at the base which is seated in the bone. In addition, some screws are coated and may not conduct the stimulus properly such that the pilot hole needs to be stimulated with a probe before the screw is placed.

Stimulus thresholds vary depending on the circumstance (Table 11.2) [48], but when the screw touches a normal nerve root, the threshold is less than 6 mA; when it has only broken through the pedicle wall, it is 6–10 mA, and when it is entirely within the pedicle, it exceeds 10 mA. Hence, stimulation thresholds less than 6–10 mA usually raise concern that the screw might need to be redirected [129]. It is important to note that abnormal nerves (e.g., nerves that are chronically compressed), through mechanisms of axonotmesis, have a much higher threshold for stimulation than normal nerves. In this case, direct stimulation of the nerve root should be employed to establish a control threshold [47].

Table 11.2 Typical stimulus thresholds

Structure	Stimulus threshold (mA)
Normal nerve root	2.2 (0.2–5.7)
Chronically compressed nerve root	6.3–20
Normal hole	30.4 (16.5–44.3)
Normal screw	24 (12.1–35.9)
Misplaced hole	3.4 (1–6)
Misplaced screw	3.5 (1–6)

From Holland [48]

Data derived from multiple prospective studies and case series performed during cervical, thoracic, and lumbosacral procedures of the spinal column support the sensitivity of EMG monitoring in detecting malpositioned hardware [15, 31, 44, 61, 106, 107, 116]. Unfortunately, the monitoring of screws in the thoracic region is made difficult by the less discrete innervations of intercostal and abdominal wall musculature. As such, techniques have been developed to assess the lower extremity muscle responses which infer current leakage through the pedicle to activate the motor tracts in the spinal cord [32].

The potential for nerve root injuries is particularly significant during surgery on the cauda equina. Procedures such as release of a tethered cord or tumor excision carry the risk of damage to nerve roots which innervate the muscles of the leg, anal sphincter, and urethral sphincter [80, 109]. This is particularly important in children where a moderate percentage of pathology is present in the conus cauda region. Damage to these nerve roots is extremely debilitating (especially loss of bowel and bladder control), and every effort is sought to avoid this complication.

In these cases, EMG monitoring is essential for differentiating neural from nonneural tissue for potential resection. The tissue in question is stimulated, and the EMG used to examine for a muscle response to indicate it is a functional nerve that should be saved; a nonresponse suggests the tissue can be sacrificed [103]. As with many types of surgery, when monitoring tethered cord surgery, other monitoring techniques such as SSEPs and MEPs are also be used, but EMG techniques provide the best guide to the surgeon for tissue evaluation. In essence, the neurosurgeon partners with the neurophysiologist to identify the functional status of tissues to be cut. Often visual clues are distorted as the neural roots may be skewed due to tethering, may pass through a lipoma, and may be involved in a thickened filum terminale. For example, there will be a difference in the stimulation threshold of nonfunctional filum terminale fibers compared to motor nerve fibers of up to 100:1 [103].

Monitoring the peripheral nervous system includes spinal nerve roots as well as the plexus and individual nerves of the limbs. This is typically performed to prevent surgical injury or guide surgical repair. Injury to peripheral nerves may occur during procedures on structures which may cause nerve stretch (e.g., surgery on the pelvis and extremities such as during hip arthroscopy or leg-lengthening procedures) [16] or during procedures on other areas where positioning places the nerves at risk (e.g., ulnar neuropathy during prone spine surgery) [8, 20, 55, 57, 114]. Of note, infants and newborns have an immature neuromuscular junction which may appear as a neuromuscular transmission defect. The maturation process also affects the motor unit potential size and EMG amplitude. Hence, the amplitude may be smaller in the very young than those found in older children and may resemble a patient with a myopathy.

One additional nerve that is often monitored with EMG during anterior cervical spine surgery is the innervation of the larynx via the recurrent laryngeal and superior laryngeal branches of the vagus nerve (c.n. X). The reported incidence of recurrent laryngeal nerve (RLN) injury is 2.3–24.2 % in anterior neck surgery [99]. The incidence of complete nerve injury as reflected by permanent hoarseness is up to 4 % [54]. Symptoms of partial or complete nerve injury which present postoperatively are hoarseness, dysphagia, dysphonia, persistent cough, vocal fatigue, or aspiration. If the injury is bilateral, then severe airway obstruction requiring tracheostomy may occur [41]. It appears that the side of surgical approach has no impact on the incidence of RLN injury [58]. Of all RLN injuries sustained during anterior cervical disk surgery, more than 80 % will recover function within 12 months [87].

Proposed mechanisms of RLN injury include direct surgical trauma, nerve division or ligature, pressure- or stretch-induced neuropraxia, and postoperative edema. The most common cause is reported to be displacement of the larynx against the shaft of the endotracheal tube by the surgical retractor with resultant pressure on the intralaryngeal segment of the RLN [62]. It has been proposed that deflation/reinflation of the cuff of the endotracheal tube after placement of the retractor lowers the incidence of injury, but this is controversial [7, 9]. Jellish and colleagues found increased pharyngeal muscle EMG activity upon retractor insertion in a series of patients and were able to correlate intubation time and endotracheal tube cuff pressure with the occurrence of postoperative dysphonia and odynophagia [53]. They therefore recommended continuous EMG monitoring during anterior cervical disk surgery.

In these cases, EMG monitoring is performed by placing needle electrodes in the cricothyroid or vocalis muscles or more commonly by a specialized endotracheal tube (or one with contact electrodes applied). Note that unless stimulation of the nerve in the operative field is conducted, the absence of EMG activity is considered normal and the nerve is presumed intact. However, if the nerve is severed, there may be no warning EMG response. As such, in critical situations, corticobulbar responses can be used. In this case, the motor cortex is stimulated like the motor evoked potentials described below, and the response of the vocalis muscle recorded signaling continuity in the pathway [28, 29].

11.6 Motor Evoked Potentials

The motor evoked potential (MEP) is the most recent addition to routine IOM in order to more specifically warn of motor pathway compromise. As in the SRS study, a few patients awaken with motor deficits with retained SSEP [23, 97, 136]. Increasing operative complexity with new instrumentation (e.g., sublaminar wires, hooks, screws), diagnostic imaging, and intraoperative imaging has increased the risk of injury to the anterior spinal cord (e.g., motor tract). Since these are better detected by evoked responses in the motor pathway, MEP monitoring facilitates better intraoperative decision making and reduces the risk of surgical intervention where the risk and severity of the potential surgical injury exceeds the functional gain [39]. These factors have increased the desire to independently assess motor and sensory function in a wide range of procedures.

The MEP and SSEP pathways are located in different topographic and vascular regions of the cerebral cortex, brainstem, and spinal cord. The motor pathways in the spinal cord are more sensitive to ischemic insults caused by stretch, compression, vascular disruption, or direct trauma than SSEP pathways because it includes the spinal gray matter [46]. Since MEP monitoring in spinal surgery has a better correlation with postoperative motor outcome than SSEP, many experts advocate MEP monitoring for all surgery for correction of axial skeletal deformity [50, 79, 84, 100] and surgery for intramedullary spinal cord tumors [30, 63, 86, 112].

The earliest attempts to produce specific motor evoked potentials were by transcranial stimulation of the motor cortex. The pioneering work of Merton and Morton in 1980 led to the development of the transcranial electrical motor evoked potential (MEP) monitoring used today [3, 24, 25, 71, 72, 81, 108]. A second method utilizing transcranial magnetic stimulation was pioneered by Barker [10, 24, 25, 34, 81, 117]. Both of these techniques produce sufficient intracortical stimulation to activate the descending motor pathways and produce a muscle response; however, the magnetic technique is more affected by anesthesia, so it is more difficult to use in IOM.

Transcranial electrical stimulation is the standard method used today to generate a MEP response. MEP uses electrical current to stimulate pyramidal cells of the motor cortex resulting in a wave of depolarization that often activates only 4–5 % of the corticospinal tract (CST) (Fig. 11.5) [4, 52]. The motor pathway descends from the motor cortex, crosses the midline in the brainstem, and descends in the ipsilateral anterior funiculi of the spinal cord. When this wave of depolarization is recorded by electrodes in the epidural space, it is termed the "D" (direct) wave. Additional transynaptic activation of internuncial pathways in the cortex results in a series of smaller waves called "I" waves (indirect) that follow the D wave. The electrical activity of the D and I waves summates in the anterior horn cell resulting in activation of peripheral nerves which produce compound muscle action potential (CMAPs) and visible body movements.

The most effective method of motor cortex stimulation utilizes a series of stimulation pulses to produce depolarization at the anterior horn cell [128]. The current method utilizes 3–7 brief, high-frequency (300–500 Hz) electrical pulses of 100–400 V (although up to 1,000 V is possible) through electrodes placed on the scalp over the motor cortex. Manipulation in the number of stimuli, time between stimuli, stimulus duration, and voltage is used to provide an optimal motor response. The multipulse technique helps overcome some of the impediments to producing muscle responses with a single-pulse technique such as anesthetic depression of the anterior horn cell. It is also helpful in patients with preexisting neuropathy, reductions in motor neuron function, and extremes of age. The CMAP response is sufficiently large that averaging is not needed and the time required to obtain a MEP is less than 10 s [128, 130].

Recording from multiple muscles allows differentiation of laterality and therefore localizes neural tissue being affected during skeletal or disk movement. The CMAP recordings are from needle electrodes placed in muscles richly innervated by the CST. These typically include the abductor pollicis brevis in the thenar eminence and the tibialis anterior and abductor hallucis in

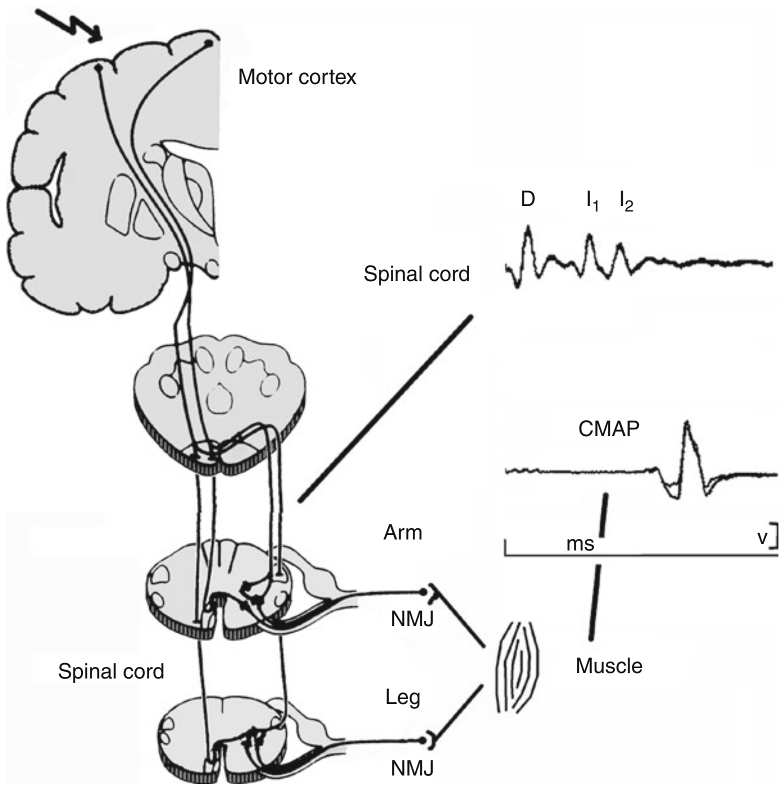


Fig. 11.5 Motor evoked potentials are produced by stimulation of the motor cortex (*). The response can be recorded epidurally over the spinal column as a *D* wave followed by a series of *I* waves. The pathway synapses in the anterior horn of the spinal cord, and the response travels to the muscle via the neuromuscular junction (*NMJ*). The response is typically recorded near the muscle as a compound muscle action potential (*CMAP*) (Reproduced from Jameson with permission [52])

the lower extremity (Fig. 11.6). Other muscles can be used when needed (e.g., anal sphincter). The "best" (largest and most reproducible) response in the lower extremities is selected for IOM throughout the procedure [50]. For cervical surgery, both upper and lower extremity responses are used. In thoracic and lumbar surgery, the upper extremity response can be used as a control (as well as monitoring for an upper extremity positioning injury) similar to the SSEP.

Interpreting the MEP response is similar to SSEP (Fig. 11.1). Onset latency, time from stimulus to response, amplitude, waveform complexity, and stimulus threshold are the standard measures used to assess change. The criteria for a significant change vary to some degree between IOM teams. Since amplitude is usually variable between 200 and 2,000 microvolts, a change in amplitude is usually not considered significant unless markedly reduced or when the response is absent. Since the response is usually polyphasic, some practitioners raise concern when the waveform simplifies into less peaks and valleys. Fortunately, onset latency is usually consistent such that an increase more than 10 % is usually considered significant. Finally, some practitioners raise concern when the voltage needed to produce a response (threshold) is increased significantly.

Other methods to monitor the motor tracts have been attempted. Stimulation of the spinal cord using electrodes in the epidural space or needle electrodes near the lamina of the spine has been used. One popular spinal stimulation technique was pioneered by Owen and is termed neurogenic motor evoked potentials (NMEP) [95, 96]. In this technique, stimulating electrodes were placed percutaneously near the vertebral bodies at adjacent levels cephalad to the level of surgery. Recording electrodes are placed near a peripheral nerve, such as the sciatic nerve, on the other side of the operative field. It is now thought that this technique includes transmission through both sensory and motor tracts with the relative contributions to the response depending on the

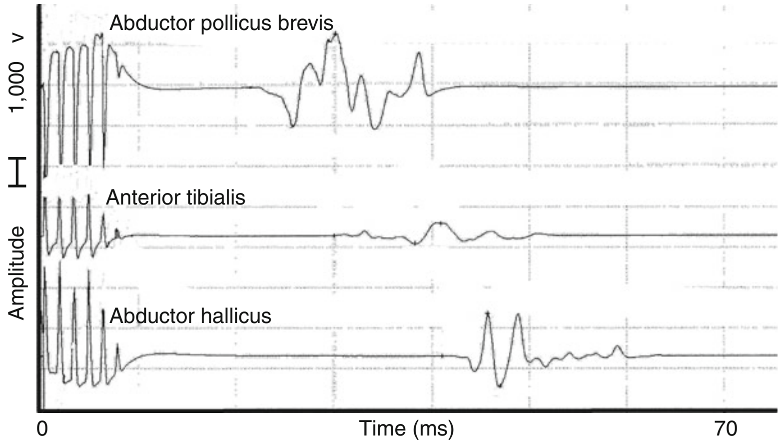


Fig. 11.6 Normal MEP responses. The CMAP response, a large polyphasic wave, is obtained from the upper extremity traditionally using the abductor pollicis brevis (*APB*) and from the lower extremity using tibialis anterior (*TA*) and abductor hallucis (*AH*). Two lower extremity muscle groups are used due to the increased difficulty obtaining a consistent response (particularly in adults). Other upper and lower extremity muscles can be used depending on the needs of the specific patient and procedure

type of anesthesia and the specific type of stimulation employed [27, 65, 96, 101, 115].

Unfortunately, recording a muscle response from spinal stimulation does not guarantee pure muscle tract stimulation because descending (antidromic) sensory tract stimulation can activate the motor pathway at the anterior horn cell through reflex pathways. Despite the uncertainty of the exact tract monitored, NMEP responses appeared useful for monitoring, and it has been advocated as a safe and effective method to perform monitoring in children and young adults with idiopathic or neuromuscular scoliosis [2, 101, 115]. However, studies indicate it is not a specific monitor of the motor tract and it has been replaced by the transcranial stimulation technique for motor monitoring.

Direct spinal cord stimulation has been pioneered in Japan and the United Kingdom with the recording of responses distally along the spinal cord or from peripheral nerves or muscles [12, 75]. Like the NMEP, the actual tracts are not known and do not appear to be specific to the motor tracts. Of note, direct stimulation of the spinal cord can be performed using a specialized handheld device or a strip electrode placed directly on the spinal cord. This technique is used to map or identify the margins between a spinal cord tumor and functional neural tissue. In these cases, the monitoring of the D wave is also used as a quantitative measure of the axons in the CST such that a reduction in D wave amplitude may be used to signal surgical encroachment on the CST and stop the surgery. This has made IOM with MEP an important tool in intramedullary spinal cord tumor surgery where it improves long-term motor outcome [63, 86, 104, 110, 112].

Preexisting medical pathology can decrease the quality of the motor response and the ability to monitor. Adults often have preexisting conditions such as diabetes, spinal cord or nerve root injury, chronic hypoperfusion, and axonal conduction changes that reduce response amplitude. Very young children, particularly those under 6 years, have an immature central nervous system, which makes obtaining a motor response challenging [73, 111]. When scoliosis procedures are performed on children and young adults with substantial neurological deficits (e.g., cerebral palsy) or muscular dystrophies (e.g., Duchene muscular dystrophy, Charcot-Marie-Tooth), monitoring may be extremely difficult [73, 111].

The most frequent use of MEP monitoring is in corrective axial skeletal surgery involving spinal levels that place the spinal cord at risk

(C1 to the termination of the spinal cord). Although the termination is usually L1–L2 in the normal adult, it varies with age and anatomic factors (e.g., tethered cord) [105, 125]. Consensus opinion in the orthopedic literature believes that the evidence supports MEP monitoring in the following specific spine procedures: (1) spinal deformities with scoliosis greater than 45° rotation, (2) congenital spine anomalies, (3) resections of intramedullary and extramedullary tumors, (4) extensive anterior and/or posterior decompressions in spinal stenosis with myelopathy, and (5) functional disturbance of cauda equina and/or individual nerve roots. The evidence is based on large case series and meta-analysis where MEP changes predicted immediate postsurgical neurological findings [1, 38, 112, 126, 127].

When used, MEP has been reported to have 100 % sensitivity, 90–96 % specificity, and a positive predictive value of 96 % [11, 56, 59]. Two recent studies reported a high correlation with outcome in spine surgery with IOM using MEP [64, 79]. In the largest study, five patients with permanent MEP change had partial permanent neurologic injury [64]. In small studies, adults with cervical myelopathy had approximately a 12 % incidence of MEP changes without EMG or SSEP changes. In cervical spine surgery, MEP has become a de facto standard of care and is believed to decrease morbidity [42] in part because it may allow differentiation between cervical cord myelopathy from peripheral neuropathy [19]. Figure 11.7 shows an example of a case where an IOM MEP warning resulted in a change in the procedure.

MEP changes are relatively infrequent. One group reported in 172 pediatric spinal deformity corrective procedures that there were 15 intraoperative MEP alerts, all of which resolved with changes in management and none had new neurological deficits. This group concluded that MEP alone was adequate for spinal deformity surgery with a MEP monitoring sensitivity of 100 % and a specificity of 97 % [50]. In the largest patient series (1,121 procedures) with idiopathic scoliosis, only patients with persistent MEP change had immediate postoperative motor deficits [113, 132].

MEP is also useful when the blood supply to the spinal cord is at risk. This includes surgery on the anterior spine that might interrupt the artery of Adamkiewicz or radicular arteries from the aorta. Because these blood vessels supply the anterior spinal artery which supplies the motor pathways, the MEP is the most reliable monitor of impending cord ischemic damage [26, 78, 90, 100]. The MEP allows rapid detection of ischemia since the gray matter, with its higher metabolic rate, is more sensitive to hypoperfusion than white matter tracts of the SSEP [51, 122]. The blood supply to the spinal cord is particularly vulnerable to damage since the anterior spinal artery may not be continuous, especially in the mid-cervical, upper thoracic, and a narrowed region just cephalad to the lumbar enlargement. The thoracic spinal cord is particularly vulnerable to ischemia, since it is not well vascularly collateralized and may have only one anterior feeding vessel between T4 and L4 [135].

MEP monitoring is not without risk; the U.S. Food and Drug Administration (FDA) approved MEP technology noting several relative contraindications. The most common concern was that the electrical stimulation would cause direct cortical thermal injury (kindling), but over the last 15 years, despite hundreds of patients who underwent MEP monitoring, only two cases have been reported [78]. The more common complications are limited to sore muscles and tongue lacerations from inadequate bite block placement. A survey of the literature published in 2002 noted complications which included tongue laceration ($n=29$), cardiac arrhythmia ($n=5$), scalp burn at the site of stimulating electrodes ($n=2$), jaw fracture ($n=1$), and awareness ($n=1$) [66]. Notably, no new-onset seizures, epidural hematomas or infections from epidural electrodes or movement injuries (e.g., surgical, joint dislocation), neuropsychiatric disease, headaches, and endocrine abnormalities have been reported. Common relative contraindications include epilepsy, cortex lesions, skull defects, high intracranial pressure, intracranial apparatus (electrodes, vascular clips, and shunts), cardiac pacemakers, or other implanted pumps. The absolute number and incidence of even minor

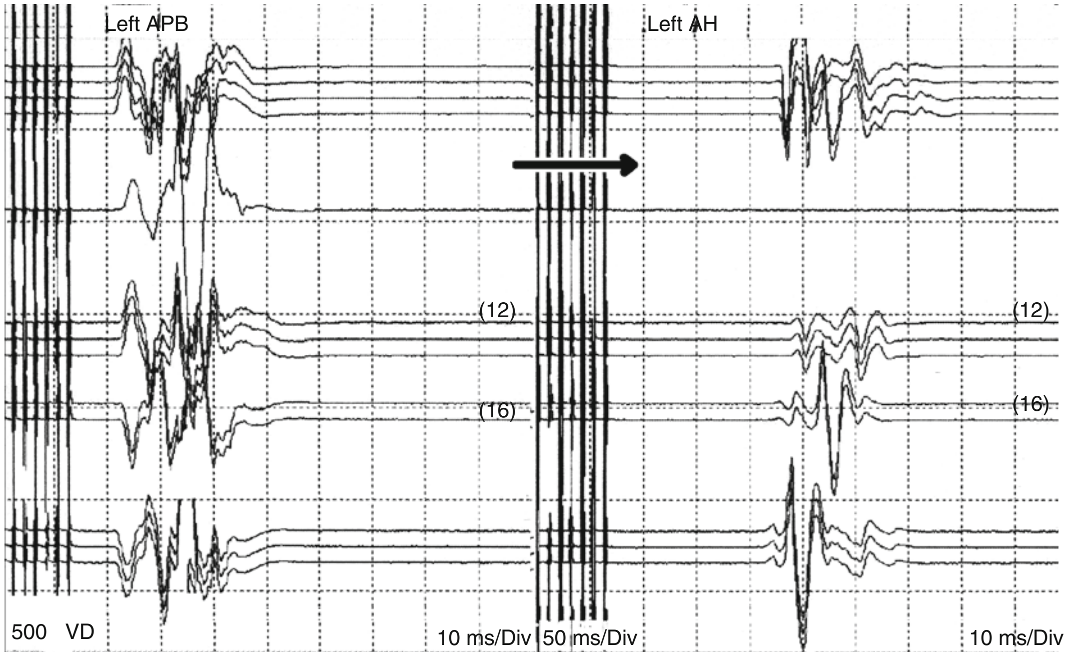


Fig. 11.7 Example of MEP change during spinal procedure. A 70-year-old female was undergoing a L1-S1 posterior spinal decompression and fusion. Shown at the top are baseline responses on the left adductor pollicis brevis (*ABP*) and left abductor hallucis (*AH*). During the procedure (*at the arrow*), the MEP was lost in the AH but not in the APB (the loss was also seen in the left tibialis anterior; no changes were seen on the right-sided responses). The surgery was halted and warm irrigation applied. The systolic blood pressure was elevated 10 mmHg and the response gradually returned. The procedure proceeded uneventfully, and the patient awakened without new neurological deficits

complications is astonishingly low [77]. Very few absolute contraindications exist (e.g., intracortical electrodes), so the practitioner must weigh the benefit of the monitoring with the relative risks in each patient. The movement associated with the MEP means close coordination with the surgeon when performing a MEP is necessary, and as such, the technique is not nearly continuous as SSEP or EMG monitoring.

11.7 Hoffman Reflex

Monitoring of reflex pathways (e.g., Hoffman, H reflex) is occasionally used during surgery to supplement the MEP, where traditional evoked responses are not useful (e.g., cauda equina), when MEP are not recordable, or the risks of MEP exceed the benefits. The H reflex can be monitored in the flexors of the upper extremity and the extensors of the lower extremity, although it has been recorded in over 20 muscles throughout the hand, arm, leg, foot, and jaw [85]. For IOM, it is most often acquired from the gastrocnemius muscle following stimulation of the posterior tibial nerve at the popliteal fossa where the reflex is primarily mediated by the S1 nerve root.

The H reflex is the result of electrical stimulation of the peripheral nerve which activates the lowest threshold Ia fibers. The ascending volley activates motor neurons via synaptic reflex pathways in the spinal cord producing a muscle response (Fig. 11.8) [85]. The H reflex is thus a reflection of transmission in these reflex pathways and the excitability of the anterior horn cells. This is useful to monitor both the peripheral nerves and the gray matter involved in the reflex. In addition, it monitors the more cephalad spinal cord because changes in the descending pathways of the corticospinal, rubrospinal, vestibulospinal, and reticulospinal systems can alter

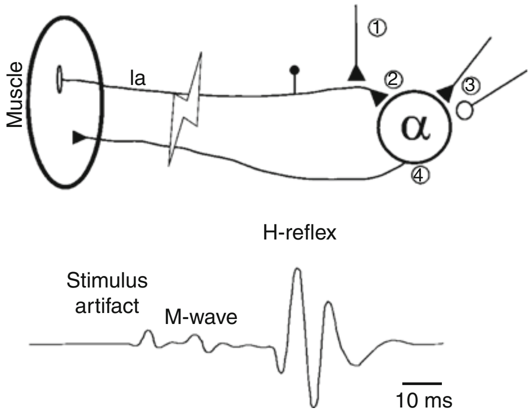


Fig. 11.8 H reflex pathway and typical muscle response. The reflex arc is initiated by stimulation of the peripheral nerve (which produces the M wave) and activates the alpha motor neuron in the spinal gray matter producing the second muscle activity (*the H reflex*). Modulation of the response includes presynaptic inhibition (*1*), homosynaptic depression (*2*), descending spinal tract influences (*3*), and intrinsic alpha motor neuron membrane excitability (*4*) (Reproduced from Misiazek with permission [85])

the anterior horn cell function (e.g., a cephalad spinal injury results in "spinal shock" and the loss of the H reflex) much like described above for clonus [67]. The degree of H reflex suppression has been found to correlate to the degree of spinal injury. A sustained loss of the reflex, or even a 90 % amplitude decrease, has been found to strongly correlate with the onset of a new motor deficit [70].

11.8 Monitoring Strategy

The application of these techniques for IOM is a team effort involving the surgical, anesthesia, and monitoring teams. The monitoring team should confer with the surgical team to design the optimal monitoring strategy given the planned procedure and pathology. Usually, this involves multimodality monitoring with a combination of modalities. In spine surgery, this often includes SSEP and MEP recordings from both the upper and lower extremities and EMG recordings from muscles innervated by nerve roots in the operative field. Similar to the way the anesthesia team confers with the surgical team to provide optimal anesthesia and physiological conditions, the anesthesia team should confer with the monitoring team to design an anesthetic that is supportive of the desired monitoring. As anesthesia is commenced, the monitoring begins, and baseline recordings are obtained. During the case, the recordings are compared to these baselines to assess for possible neural compromise. When an evoked response changes, the physiological, anesthetic, positioning, and surgical environment must be assessed to determine its contribution to the change and determine possible corrective maneuvers to mitigate any adverse mechanical or ischemic factors.

11.9 Effects of Anesthesia and Physiology

Key to the success of surgery and monitoring is the choice of anesthesia and management of the patient's physiology.

The choice of anesthesia agents will depend on the specific monitoring modalities with the technique which is most restrictive guiding the choice. When used, MEP responses are usually the most restrictive because they are the most easily depressed or eliminated, particularly by the inhalational anesthetic agents [118, 119]. Even low concentrations (0.5 minimal alveolar concentration (MAC)) may not be tolerated except in the neurologically normal individual. Hence, they commonly must be avoided with MEP and a total intravenous anesthetic (TIVA) utilized.

The cortical SSEP is slightly less sensitive such that 0.5 MAC is often acceptable, but occasionally the inhalational agents must also be avoided when SSEP is being used. Fortunately, EMG, D wave, and subcortical SSEP are not significantly affected by the inhalational agents. However, since SSEP and MEP are frequently both employed during spine surgery, TIVA is usually chosen. Of note, if a low concentration of inhalational agent is chosen, it is desirable to use an insoluble agent (e.g., desflurane, sevoflurane) such that it can be eliminated if the early assessment of monitoring reveals it is inadequate for

IOM. Thus, it is desirable to establish the desired anesthetic technique early in the procedure and deliver it in ways so that it is unchanging during the procedure (e.g., infusions) lest changes or bolus doses cause monitoring changes that are confused with neural compromise.

TIVA uses infusions of a sedative agent (e.g., propofol, dexmedetomidine) with an opioid (e.g. sufentanil, fentanyl, remifentanil). Often supplementation with ketamine is employed in opioid-tolerant patients (e.g., chronic pain). Benzodiazepines (midazolam) may be used if needed to insure amnesia or to reduce unpleasant dreams with ketamine. Ketamine may also enhance the amplitude of the cortical SSEP and myogenic responses of MEP [119]. Since higher doses of propofol and dexmedetomidine can depress the MEP response, the addition of ketamine can also be helpful by allowing a reduction in their doses.

Since EMG and MEP require muscle responses, neuromuscular blocking agents (NMB) are usually avoided (except for short-term relaxation for intubation) unless they are necessary for selected portions of the procedure (such as during the spine exposure during anterior lumbar procedures). MEP and stimulated EMG (e.g., pedicle screw testing) have been successfully recorded under partial NMB; however, this is not recommended unless absolutely necessary because excessive NMB effect could be confused with a loss of neural function [121]. Since NMB will reduce EMG amplitude, it is also not recommended when mechanically stimulated EMG responses are important (e.g., stretch of a nerve root during retraction). It is also recommended to limit the use of NMB during pedicle screw testing since it can falsely elevate the threshold [83].

As mentioned above, the neurological function of the patient will also determine the monitored responses and the choice of anesthesia. Young adults and adults with robust normal, nervous systems are usually more tolerant of inhalational agents such that low concentrations often are usable, even with MEP. However, very young children with immature nervous systems and adults with neurological compromise may require TIVA (often supplemented with ketamine to enhance cortical SSEP and myogenic MEP).

The physiological management of the patient is also important so as to minimize any contribution to a suboptimal neural environment. Physiological conditions that alter neuronal function include inadequate arterial perfusion (e.g., relative hypotension, raised intracranial pressure), ischemia, anemia, tissue or systemic hypoxia, hypothermia, electrolyte abnormalities, and hypoglycemia [120]. In particular, there is a growing appreciation that the lower limit of autoregulation may be higher than expected, especially in tissues undergoing surgery or injury [33]. This means that reduced blood pressures used to decrease bleeding may be too low to provide adequate spinal cord blood flow. As such, a growing number of practitioners maintain the patient's blood pressure in their normal range ("deliberate normotension") and increase the blood pressure when monitoring suggests neural dysfunction is present (Fig. 11.7) [35].

Conclusion

Neurophysiologic monitoring with EMG, SSEP, and MEP has become an important tool in the operative management of surgery of the axial skeleton or where peripheral nerves or the blood supply to the spinal cord are at risk. In general, multiple monitoring modalities are used with each case to provide the greatest degree of neural vigilance and allow optimal surgical and anesthesiology decision making. In some procedures, IOM has been demonstrated to be cost-effective and improve outcome and has become a standard of care. Hopefully, surgical procedures and IOM techniques will evolve in parallel to continue to provide the best care of our patients as more complex procedures are utilized in patients with challenging neural pathophysiology.

References

1. Abbott R (2002) Sensory rhizotomy for the treatment of childhood spasticity. In: Deletis V, Shils JL (eds) Neurophysiology in neurosurgery. Academic, Boston, pp 219–230
2. Accadbled F, Henry P, de Gauzy JS, Cahuzac JP, Accadbled F, Henry P, de Gauzy JS, Cahuzac JP (2006)

Spinal cord monitoring in scoliosis surgery using an epidural electrode. Results of a prospective, consecutive series of 191 cases. Spine 31(22):2614–2623 [see comment]
3. Amassian VE, Cracco RQ (1987) Human cerebral cortical responses to contralateral transcranial stimulation. Neurosurgery 20(1):148–155
4. Amassian VE, Stewart M, Quirk GJ, Rosenthal JL (1987) Physiological basis of motor effects of a transient stimulus to cerebral cortex. Neurosurgery 20(1):74–93
5. Aminoff MJ, Eisen AA (1998) AAEM minimonograph 19: somatosensory evoked potentials. Muscle Nerve 21(3):277–290
6. Anonymous (1992) Scoliosis Research Society: Position statement on somatosensory evoked potential monitoring of neurologic spinal cord function during surgery. Scoliosis Research Society, Park Ridge
7. Apfelbaum RI, Kriskovich MD, Haller JR (2000) On the incidence, cause, and prevention of recurrent laryngeal nerve palsies during anterior cervical spine surgery. Spine 25:2906–2912
8. Arrington ED, Hochschild DP, Steinagle TJ, Mongan PD, Martin SL (2000) Monitoring of somatosensory and motor evoked potentials during open reduction and internal fixation of pelvis and acetabular fractures. Orthopedics 23(10):1081–1083
9. Audu P, Artz G, Scheid S (2006) Recurrent laryngeal nerve palsy after anterior cervical spine surgery. Anesthesiology 105:898–901
10. Barker AT, Jalinous R, Freeston IL, Barker AT, Jalinous R, Freeston IL (1985) Non-invasive magnetic stimulation of human motor cortex. Lancet 1(8437):1106–1107
11. Bednarik J, Kadanka Z, Vohanka S, Stejskal L, Vlach O, Schroder R (1593) The value of somatosensory- and motor-evoked potentials in predicting and monitoring the effect of therapy in spondylotic cervical myelopathy. Prospective randomized study. Spine 24(15):1593–1598
12. Ben-David B (1988) Spinal cord monitoring. Orthop Clin North Am 19(2):427–448
13. Ben-David B, Allen PW, Waller CS, Edgar MA, Webb PJ, Ransford AO (1991) Spinal cord monitoring in scoliosis surgery. Experience with 1168 cases. J Bone Joint Surg Am 73:487–494
14. Ben-David B, Haller G, Taylor P (1987) Anterior spinal fusion complicated by paraplegia: a case report of a false-negative somatosensory evoked potential. Spine 12:536–539
15. Bose B, Wierzbowski LR, Sestokas AK, Bose B, Wierzbowski LR, Sestokas AK (2002) Neurophysiologic monitoring of spinal nerve root function during instrumented posterior lumbar spine surgery. Spine 27(13):1444–1450
16. Brown DM, McGinnis WC, Mesghali H (2002) Neurophysiologic intraoperative monitoring during revision total hip arthroplasty. J Bone Joint Surg Am 84-A(Suppl 2):56–61
17. Burke D, Hicks RG (1998) Surgical monitoring of motor pathways. J Clin Neurophysiol 15:194–205
18. Calancie B, Madsen P, Lebwohl N (1994) Stimulus-evoked EMG monitoring during transpedicular lumbosacral spine instrumentation. Initial clinical results. Spine 19(24):2780–2786
19. Chistyakov A, Soustiel J, Hafner H, Kaplan B, Feinsod M (2004) The value of motor and somatosensory evoked potentials in evaluation of cervical myelopathy in the presence of peripheral neuropathy. Spine 29(12):e239–e247
20. Chung I, Glow JA, Dimopoulos V, Walid MS, Smisson HF, Johnston KW, Robinson JS, Grigorian AA (2009) Upper-limb somatosensory evoked potential monitoring in lumbosacral spine surgery: a prognostic marker for position-related ulnar nerve injury. Spine J 9(4):287–295. doi:10.1016/j.spinee.2008.05.004
21. Costa P, Bruno A, Bonzanino M, Massaro F, Caruso L, Vincenzo I, Ciaramitaro P, Montalenti E, Costa P, Bruno A, Bonzanino M, Massaro F, Caruso L, Vincenzo I, Ciaramitaro P, Montalenti E (2007) Somatosensory- and motor-evoked potential monitoring during spine and spinal cord surgery. Spinal Cord 45(1):86–91
22. Croft TJ, Brodkey JS, Nulsen FE, Croft TJ, Brodkey JS, Nulsen FE (1972) Reversible spinal cord trauma: a model for electrical monitoring of spinal cord function. J Neurosurg 36(4):402–406
23. Dawson EG, Sherman JE, Kanim LE, Nuwer MR (1991) Spinal cord monitoring. Results of the Scoliosis Research Society and the European Spinal Deformity Society survey. Spine 16(8 Suppl):S361–S364
24. Day BL, Dressler D, Maertens de Noordhout A, Marsden CD, Nakashima K, Rothwell JC, Thompson PD (1989) Electric and magnetic stimulation of human motor cortex: surface EMG and single motor unit responses. J Physiol 412:449–473 [erratum appears in J Physiol (Lond) 1990 Nov;430:617]
25. Day BL, Thompson PD, Dick JP, Nakashima K, Marsden CD (1987) Different sites of action of electrical and magnetic stimulation of the human brain. Neurosci Lett 75(1):101–106
26. de Haan P, Kalkman CJ, Ubags LH, Jacobs MJ, Drummond JC, de Haan P, Kalkman CJ, Ubags LH, Jacobs MJ, Drummond JC (1996) A comparison of the sensitivity of epidural and myogenic transcranial motor-evoked responses in the detection of acute spinal cord ischemia in the rabbit. Anesth Analg 83(5):1022–1027
27. Deletis V (1993) Intraoperative monitoring of the functional integrity of the motor pathways. Adv Neurol 63:201–214
28. Deletis V, Fernandez-Conejero I, Ulkatan S, Costantino P, Deletis V, Fernandez-Conejero I, Ulkatan S, Costantino P (2009) Methodology for intraoperatively eliciting motor evoked potentials in the vocal muscles by electrical stimulation of the corticobulbar tract. Clin Neurophysiol 120(2):336–341
29. Deletis V, Fernandez-Conejero I, Ulkatan S, Rogic M, Carbo EL, Hiltzik D (2011) Methodology for intraoperative recording of the corticobulbar motor evoked

potentials from cricothyroid muscles. Clin Neurophysiol 122(9):1883–1889
30. Deletis V, Sala F (2008) Intraoperative neurophysiological monitoring of the spinal cord during spinal cord and spine surgery: a review focus on the corticospinal tracts. Clin Neurophysiol 119(2):248–264
31. Djurasovic M, Dimar JR 2nd, Glassman SD, Edmonds HL, Carreon LY (2005) A prospective analysis of intraoperative electromyographic monitoring of posterior cervical screw fixation. J Spinal Disord Tech 18(6):515–518 [see comment]
32. Donohue ML, Murtagh-Schaffer C, Basta J, Moquin RR, Bashir A, Calancie B (2008) Pulse-train stimulation for detecting medial malpositioning of thoracic pedicle screws. Spine 33(12):E378–E385
33. Edmonds HL Jr (2002) Multi-modality neurophysiologic monitoring for cardiac surgery. Heart Surg Forum 5(3):225–228
34. Edmonds HL Jr, Paloheimo MP, Backman MH, Johnson JR, Holt RT, Shields CB (1989) Transcranial magnetic motor evoked potentials (tcMMEP) for functional monitoring of motor pathways during scoliosis surgery. Spine 14(7):683–686
35. Edmonds HL, Rodriguez RA, Audenaert SM, Austin EH, Pollock SM, Ganzel BL (1996) The role of neuromonitoring in cardiovascular surgery. J Cardiothorac Vasc Anesth 10:15–23
36. Edwards BM, Kileny PR (2005) Intraoperative neurophysiologic monitoring: indications and techniques for common procedures in otolaryngology-head and neck surgery. Otolaryngol Clin North Am 38(4):631–642
37. Eggspuehler A, Sutter MA, Grob D, Jeszenszky D, Dvorak J, Eggspuehler A, Sutter MA, Grob D, Jeszenszky D, Dvorak J (2007) Multimodal intraoperative monitoring during surgery of spinal deformities in 217 patients. Eur Spine J 16(Suppl 2):S188–S196
38. Eggspuehler A, Sutter MA, Grob D, Jeszenszky D, Porchet F, Dvorak J (2007) Multimodal intraoperative monitoring (MIOM) during cervical spine surgical procedures in 246 patients. Eur Spine J 16(Suppl 2):S209–S215
39. Fehlings MG, Houlden D, Vajkoczy P (2009) Introduction. Neurosurg Focus 27(4):E1. doi:10.3171/2009.8.FOCUS.OCT09.INTRO
40. Forbes HJ, Allen PW, Waller CS, Jones SJ, Edgar MA, Webb PJ, Ransford AO (1991) Spinal cord monitoring in scoliosis surgery. Experience with 1168 cases. J Bone Joint Surg Br 73(3):487–491
41. Fountas KN, Kapalaki EZ, Nikolakakos LG (2007) Anterior cervical discectomy and fusion associated complications. Spine 32:2310–2317
42. Freedman B, Potter B, Kuklo T (2005) Managing neurologic complications in cervical spine surgery. Curr Opin Orthop 16(3):169–177
43. Green J, Gildemeister R, Hazelwood C (1983) Dermatomally stimulated somatosensory cerebral evoked potentials in the diagnosis of lumbar disc disease. Clin Electroencephalogr 14:152–163
44. Gunnarsson T, Krassioukov AV, Sarjeant R, Fehlings MG, Gunnarsson T, Krassioukov AV, Sarjeant R, Fehlings MG (2004) Real-time continuous intraoperative electromyographic and somatosensory evoked potential recordings in spinal surgery: correlation of clinical and electrophysiologic findings in a prospective, consecutive series of 213 cases. Spine 29(6):677–684
45. Harper CM (2004) Intraoperative cranial nerve monitoring. Muscle Nerve 29(3):339–351
46. Hickey R, Sloan TB, Roger JN (1995) Functional organization and physiology of the spinal cord. In: Porter SS (ed) Anesthesia for surgery of the spine. McGraw-Hill, New York, pp 15–39
47. Holland NR (1998) Intraoperative electromyography during thoracolumbar spinal surgery. Spine 23(17):1915–1922
48. Holland NR (2002) Intraoperative electromyography. J Clin Neurophysiol 19(5):444–453
49. Hoppenfeld S, Gross A, Andrews C, Lonner B (1997) The ankle clonus test for assessment of the integrity of the spinal cord during operations for scoliosis. J Bone Joint Surg Am 79:208–212
50. Hsu B, Cree AK, Lagopoulos J, Cummine JL (2008) Transcranial motor-evoked potentials combined with response recording through compound muscle action potential as the sole modality of spinal cord monitoring in spinal deformity surgery. Spine 33(10):1100–1106
51. Jacobs MJ, Mess W, Mochtar B, Nijenhuis RJ, Statius van Eps RG, Schurink GW, Jacobs MJ, Mess W, Mochtar B, Nijenhuis RJ, Statius van Eps RG, Schurink GWH (2006) The value of motor evoked potentials in reducing paraplegia during thoracoabdominal aneurysm repair. J Vasc Surg 43(2):239–246
52. Jameson LC, Sloan TB (2006) Monitoring of the brain and spinal cord. Anesthesiol Clin 24(4):777–791
53. Jellish WS, Jensen RL, Anderson DE, Shea JF (1999) Intraoperative electromyographic assessment of recurrent laryngeal nerve stress and pharyngeal injury during anterior cervical spine surgery with Caspar instrumentation. J Neurosurg 91(2 Suppl):170–174
54. Jung A, Schramm J, Lehnerdt K, Herberhold C (2005) Recurrent laryngeal nerve palsy during anterior cervical spine surgery: a prospective study. J Neurosurg Spine 2:123–127
55. Kamel IR, Drum ET, Koch SA, Whitten JA, Gaughan JP, Barnette RE, Wendling WW (2006) The use of somatosensory evoked potentials to determine the relationship between patient positioning and impending upper extremity nerve injury during spine surgery: a retrospective analysis. Anesth Analg 102(5):1538–1542 [see comment]
56. Kelleher MO, Tan G, Sarjeant R, Fehlings MG (2008) Predictive value of intraoperative neurophysiological monitoring during cervical spine surgery: a prospective analysis of 1055 consecutive patients. J Neurosurg Spine 8(3):215–221. doi:10.3171/spi/2008/8/3/215
57. Kennedy WF, Byrne TF, Majid HA, Pavlak LL (1991) Sciatic nerve monitoring during revision total hip arthroplasty. Clin Orthop 264:223–227

58. Kilburg C, Sullivan HG, Mathiason MA (2006) Effect of approach side during anterior cervical discectomy and fusion on the incidence of recurrent laryngeal nerve injury. J Neurosurg Spine 4:273–277
59. Kim DH, Zaremski J, Kwon B, Jenis L, Woodard E, Bode R, Banco RJ (2007) Risk factors for false positive transcranial motor evoked potential monitoring alerts during surgical treatment of cervical myelopathy. Spine 32(6):3041–3046
60. Kojima Y, Yamamoto T, Ogino H, Okada K, Ono K, Kojima Y, Yamamoto T, Ogino H, Okada K, Ono K (1979) Evoked spinal potentials as a monitor of spinal cord viability. Spine 4(6):471–477
61. Krassioukov AV, Sarjeant R, Arkia H, Fehlings MG (2004) Multimodality intraoperative monitoring during complex lumbosacral procedures: indications, techniques, and long-term follow-up review of 61 consecutive cases. J Neurosurg Spine 1(3):243–253 [see comment]
62. Kriskovich MD, Apfelbaum RI, Haller JR (2000) Vocal fold paralysis after anterior cervical spine surgery: incidence, mechanism, and prevention of injury. Laryngoscope 110:1467–1473
63. Lang EW, Chesnut RM, Beutler AS, Kennelly NA, Renaudin JW (1996) The utility of motor-evoked potential monitoring during intramedullary surgery. Anesth Analg 83(6):1337–1341
64. Langeloo DD, Lelivelt A, Louis Journee H, Slappendel R, de Kleuver M (2003) Transcranial electrical motor-evoked potential monitoring during surgery for spinal deformity: a study of 145 patients. Spine 28(10):1043–1050
65. Laschinger JC, Owen J, Rosenbloom M, Cox JL, Kouchoukos NT (1988) Direct noninvasive monitoring of spinal cord motor function during thoracic aortic occlusion: use of motor evoked potentials. J Vasc Surg 7(1):161–171
66. Legatt A (2002) Current practice of motor evoked potential monitoring: results of a survey. J Clin Neurophysiol 19(5):454–460
67. Leis AA, Zhou HH, Mehta M, Harkey HL 3rd, Paske WC, Leis AA, Zhou HH, Mehta M, Harkey HL 3rd, Paske WC (1996) Behavior of the H-reflex in humans following mechanical perturbation or injury to rostral spinal cord. Muscle Nerve 19(11):1373–1382
68. Lenke LG (1997) The clinical utility of intraoperative evoked-potential monitoring from a spinal surgeon's perspective. Semin Spine Surg 9(4):288–294
69. Leppanen RE (2005) Intraoperative monitoring of segmental spinal nerve root function with free-run and electrically-triggered electromyography and spinal cord function with reflexes and F-responses. A position statement by the American Society of Neurophysiological Monitoring. J Clin Monit Comput 19(6):437–461
70. Leppanen RE (2006) Intraoperative applications of the H-reflex and F-response: a tutorial. J Clin Monit Comput 20(4):267–304
71. Levy WJ Jr (1987) Clinical experience with motor and cerebellar evoked potential monitoring. Neurosurgery 20(1):169–182
72. Levy WJ, McCaffrey M, York DH, Tanzer F (1984) Motor evoked potentials from transcranial stimulation of the motor cortex in cats. Neurosurgery 15(2):214–227
73. Lieberman JA, Lyon R, Feiner J, Diab M, Gregory GA (2006) The effect of age on motor evoked potentials in children under propofol/isoflurane anesthesia. Anesth Analg 103(2):316–321. doi:10.1213/01.ane.0000226142.15746.b2, table of contents
74. Loughman BA, Fennelly ME, Henley M, Hall GM (1995) The effects of differing concentrations of bupivacaine on the epidural somatosensory evoked potential after posterior tibial nerve stimulation. Anesth Analg 81(1):147–151
75. Loughnan BA, Fennelly ME (1995) Spinal cord monitoring. Anaesthesia 50(2):101–102
76. Lyon R, Burch S, Lieberman J (2009) Mixed-muscle electrode placement ("jumping" muscles) may produce false-negative results when using transcranial motor evoked potentials to detect an isolated nerve root injury in a porcine model. J Clin Monit Comput 23(6):403–408. doi:10.1007/s10877-009-9205-9
77. MacDonald D (2002) Safety of intraoperative transcranial electrical stimulation motor evoked potential monitoring. J Clin Neurophysiol 19(5):416–429
78. Macdonald DB (2006) Intraoperative motor evoked potential monitoring: overview and update. J Clin Monit Comput 20(5):347–377. doi:10.1007/s10877-006-9033-0
79. MacDonald DB, Al Zayed Z, Khoudeir I, Stigsby B (2003) Monitoring scoliosis surgery with combined multiple pulse transcranial electric motor and cortical somatosensory-evoked potentials from the lower and upper extremities. Spine 28(2):194–203
80. Marusch F, Hussock J, Haring G, Hachenberg T, Gastinger I (2005) Influence of muscle relaxation on neuromonitoring of the recurrent laryngeal nerve during thyroid surgery. Br J Anaesth 94(5):596–600. doi:aei110[pii]
81. Merton PA, Morton HB (1980) Stimulation of the cerebral cortex in the intact human subject. Nature 285:227–235
82. Meyer PR Jr, Cotler HB, Gireesan GT (1988) Operative neurological complications resulting from thoracic and lumbar spine internal fixation. Clin Orthop 237:125–131
83. Minahan RE, Riley LH 3rd, Lukaczyk T, Cohen DB, Kostuik JP (2000) The effect of neuromuscular blockade on pedicle screw stimulation thresholds. Spine 25(19):2526–2530
84. Minahan RE, Sepkuty JP, Lesser RP, Sponseller PD, Kostuik JP (2001) Anterior spinal cord injury with preserved neurogenic 'motor' evoked potentials. Clin Neurophysiol 112(8):1442–1450 [see comment]
85. Misiaszek JE, Misiaszek JE (2003) The H-reflex as a tool in neurophysiology: its limitations and uses in understanding nervous system function. Muscle Nerve 28(2):144–160
86. Morota N, Deletis V, Constantini S, Kofler M, Cohen H, Epstein FJ (1997) The role of motor evoked potentials during surgery for intramedullary spinal cord tumors. Neurosurgery 41(6):1327–1336

87. Morpeth JF, Williams MF (2000) Vocal fold paralysis after anterior cervical discectomy and fusion. Laryngoscope 110:43–46
88. Mostegl A, Bauer R, Eichenauer M (1988) Intraoperative somatosensory potential monitoring. A clinical analysis of 127 surgical procedures. Spine 13(4):396–400
89. Nash CL Jr, Lorig RA, Schatzinger LA, Brown RH (1977) Spinal cord monitoring during operative treatment of the spine. Clin Orthop 126:100–105
90. Neuloh G, Schramm J, Neuloh G, Schramm J (2004) Monitoring of motor evoked potentials compared with somatosensory evoked potentials and microvascular Doppler ultrasonography in cerebral aneurysm surgery. J Neurosurg 100(3):389–399
91. Nuwer MR (1998) Spinal cord monitoring with somatosensory techniques. J Clin Neurophysiol 15(3):183–193
92. Nuwer MR, Dawson EG, Carlson LG, Kanim LE, Sherman JE (1995) Somatosensory evoked potential spinal cord monitoring reduces neurologic deficits after scoliosis surgery: results of a large multicenter survey. Electroencephalogr Clin Neurophysiol 96(1):6–11
93. Owen J (1997) Cost efficacy of intraoperative monitoring. Semin Spine Surg 9(4):348–352
94. Owen JH (1993) Intraoperative stimulation of the spinal cord for prevention of spinal cord injury. Adv Neurol 63:271–288
95. Owen JH (1997) Applications of neurophysiological measures during surgery of the spine. In: Frymoyer JW (ed) The adult spine: principles and practice, 2nd edn. Lippincott-Raven Publishers, Philadelphia, pp 673–702
96. Owen JH, Bridwell KH, Grubb R, Jenny A, Allen B, Padberg AM, Shimon SM (1991) The clinical application of neurogenic motor evoked potentials to monitor spinal cord function during surgery. Spine 16(8 Suppl):S385–S390
97. Padberg AM, Russo MH, Lenke LG, Bridwell KH, Komanetsky RM (1996) Validity and reliability of spinal cord monitoring in neuromuscular spinal deformity surgery. J Spinal Disord 9(2):150–158
98. Papastefanou SL, Henderson LM, Smith NJ, Hamilton A, Webb JK (2000) Surface electrode somatosensory-evoked potentials in spinal surgery: implications for indications and practice. Spine 25(19):2467–2472
99. Pearlman RC, Isley MR, Ruben GD, Sandler SC, Weisbaum B, Khan MA, Greene BS, Charles V, Shah A, Pearlman RC, Isley MR, Ruben GD, Sandler SC, Weisbaum B, Khan MA, Greene BS, Charles V, Shah A (2005) Intraoperative monitoring of the recurrent laryngeal nerve using acoustic, free-run, and evoked electromyography. J Clin Neurophysiol 22(2):148–152
100. Pelosi L, Lamb J, Grevitt M, Mehdian SM, Webb JK, Blumhardt LD, Pelosi L, Lamb J, Grevitt M, Mehdian SMH, Webb JK, Blumhardt LD (2002) Combined monitoring of motor and somatosensory evoked potentials in orthopaedic spinal surgery. Clin Neurophysiol 113(7):1082–1091
101. Pereon Y, Bernard JM, Fayet G, Delecrin J, Passuti N, Guiheneuc P, Pereon Y, Bernard JM, Fayet G, Delecrin J, Passuti N, Guiheneuc P (1998) Usefulness of neurogenic motor evoked potentials for spinal cord monitoring: findings in 112 consecutive patients undergoing surgery for spinal deformity. Electroencephalogr Clin Neurophysiol 108(1):17–23
102. Prass RL, Kinney SE, Hardy RW Jr, Hahn JF, Luders H (1987) Acoustic (loudspeaker) facial emg monitoring: II. Use of evoked emg activity during acoustic neuroma resection. Otolaryngol Head Neck Surg 97:541–551
103. Quinones-Hinojosa A, Gadkary CA, Gulati M, von Koch CS, Lyon R, Weinstein PR, Yingling CD (2004) Neurophysiological monitoring for safe surgical tethered cord syndrome release in adults. Surg Neurol 62(2):127–133; discussion 133–135
104. Quinones-Hinojosa A, Gulati M, Lyon R, Gupta N, Yingling C, Quinones-Hinojosa A, Gulati M, Lyon R, Gupta N, Yingling C (2002) Spinal cord mapping as an adjunct for resection of intramedullary tumors: surgical technique with case illustrations. Neurosurgery 51(5):1199–1206; discussion 1206–1207
105. Quraishi NA, Lewis SJ, Kelleher MO, Sarjeant R, Rampersaud YR, Fehlings MG (2009) Intraoperative multimodality monitoring in adult spinal deformity: analysis of a prospective series of one hundred two cases with independent evaluation. Spine 34(14):1504–1512
106. Raynor BL, Lenke LG, Kim Y, Hanson DS, Wilson-Holden TJ, Bridwell KH, Padberg AM, Raynor BL, Lenke LG, Kim Y, Hanson DS, Wilson-Holden TJ, Bridwell KH, Padberg AM (2002) Can triggered electromyograph thresholds predict safe thoracic pedicle screw placement? Spine 27(18):2030–2035 [see comment]
107. Reidy DP, Houlden D, Nolan PC, Kim M, Finkelstein JA, Reidy DP, Houlden D, Nolan PC, Kim M, Finkelstein JA (2001) Evaluation of electromyographic monitoring during insertion of thoracic pedicle screws. J Bone Joint Surg Br 83(7):1009–1014
108. Rothwell JC, Thompson PD, Day BL, Dick JP, Kachi T, Cowan JM, Marsden CD (1987) Motor cortex stimulation in intact man. 1. General characteristics of EMG responses in different muscles. Brain 110(Pt 5):1173–1190
109. Sala F, Krzan MJ, Deletis V, Sala F, Krzan MJ, Deletis V (2002) Intraoperative neurophysiological monitoring in pediatric neurosurgery: why, when, how? Childs Nerv Syst 18(6–7):264–287
110. Sala F, Lanteri P, Bricolo A, Sala F, Lanteri P, Bricolo A (2004) Motor evoked potential monitoring for spinal cord and brain stem surgery. Adv Tech Stand Neurosurg 29:133–169
111. Sala F, Manganotti P, Grossauer S, Tramontanto V, Mazza C, Gerosa M (2010) Intraoperative neurophysiology of the motor system in children: a tailored approach. Childs Nerv Syst 26(4):473–490

112. Sala F, Palandri G, Basso E, Lanteri P, Deletis V, Faccioli F, Bricolo A, Sala F, Palandri G, Basso E, Lanteri P, Deletis V, Faccioli F, Bricolo A (2006) Motor evoked potential monitoring improves outcome after surgery for intramedullary spinal cord tumors: a historical control study. Neurosurgery 58(6):1129–1143; discussion 1129–1143
113. Schwartz DM, Auerbach JD, Dormans JP, Flynn J, Drummond DS, Bowe JA, Laufer S, Shah SA, Bowen JR, Pizzutillo PD, Jones KJ (2007) Neurophysiological detection of impending spinal cord injury during scoliosis surgery. J Bone Joint Surg Am 89(11):2440–2449
114. Schwartz DM, Sestokas AK, Hilibrand AS, Vaccaro AR, Bose B, Li M, Albert TJ (2006) Neurophysiological identification of position-induced neurologic injury during anterior cervical spine surgery. J Clin Monit Comput 20(6):437–444. doi:10.1007/s10877-006-9032-1
115. Schwartz DM, Sestokis AK, Wierzbowski LR (1996) Intraoperative neurophysiological monitoring during surgery for spinal instability. Semin Spine Surg 8(4):318–331
116. Shi YB, Binette M, Martin WH, Pearson JM, Hart RA, Shi YB, Binette M, Martin WH, Pearson JM, Hart RA (2003) Electrical stimulation for intraoperative evaluation of thoracic pedicle screw placement. Spine 28(6):595–601
117. Shields CB, Paloheimo MPJ, Backman MH, Edmonds HLJ, Johnson JR (1990) Intraoperative use of transcranial magnetic motor evoked potentials. In: Chokroverty S (ed) Magnetic stimulation in clinical neurophysiology. Butterworths, London, pp 173–184
118. Sloan T (2012) General anesthesia for monitoring. In: Koht A, Sloan T, Toleikis JR (eds) Monitoring for the anesthesiologist and other health professionals. Springer, New York, pp 319–335
119. Sloan TB (1997) Evoked potentials. In: Albin MA (ed) Textbook of neuroanesthesia with neurosurgical and neuroscience perspectives. McGraw-Hill, New York, pp 221–276
120. Sloan TB (2002) Evoked potentials. Anesthesia and motor evoked-potentials monitoring. In: Deletis V, Shills J (eds) Neurophysiology in neurosurgery. Academic, San Diego, pp 451–464
121. Sloan TB, Heyer EJ (2002) Anesthesia for intraoperative neurophysiologic monitoring of the spinal cord. J Clin Neurophysiol 19(5):430–443
122. Sloan TB, Jameson LC (2007) Electrophysiologic monitoring during surgery to repair the thoraco-abdominal aorta. J Clin Neurophysiol 24(4):316–327
123. Sloan TB, Ronai AK, Koht A (1986) Reversible loss of somatosensory evoked potentials during anterior cervical spinal fusion. Anesth Analg 65(1):96–99
124. Sloan TB, Toleikis JR, Koht A (1989) Evaluation of spinal cord function by means of evoked potentials. In: Meyer PR Jr (ed) Surgery of spine trauma. Churchill-Livingstone, New York, pp 121–136
125. Sutter M, Deletis V, Dvorak J, Eggspuehler A, Grob D, Macdonald D, Mueller A, Sala F, Tamaki T (2007) Current opinions and recommendations on multimodal intraoperative monitoring during spine surgeries. Eur Spine J 16(Suppl 2):S232–S237
126. Sutter M, Eggspuehler A, Grob D, Jeszenszky D, Benini A, Porchet F, Mueller A, Dvorak J (2007) The validity of multimodal intraoperative monitoring (MIOM) in surgery of 109 spine and spinal cord tumors. Eur Spine J 16(Suppl 2):S197–S208
127. Sutter MA, Eggspuehler A, Grob D, Porchet F, Jeszenszky D, Dvorak J (2007) Multimodal intraoperative monitoring (MIOM) during 409 lumbosacral surgical procedures in 409 patients. Eur Spine J 16(Suppl 2):S221–S228
128. Taniguchi M, Cedzich C, Schramm J (1993) Modification of cortical stimulation for motor evoked potentials under general anesthesia: technical description. Neurosurgery 32(2):219–226
129. Toleikis JR (2002) Neurophysiological monitoring during pedicle screw placement. In: Deletis V, Shils JL (eds) Neurophysiology in neurosurgery. Academic, New York, pp 231–264
130. Ubags LH, Kalkman CJ, Been HD (1998) Influence of isoflurane on myogenic motor evoked potentials to single and multiple transcranial stimuli during nitrous oxide/opioid anesthesia. Neurosurgery 43(1):90–94; discussion 94–95
131. Vauzelle C, Stagnara P, Jouvinroux P (1973) Functional monitoring of spinal cord activity during spinal surgery. Clin Orthop 93:173–178
132. Wang AC, Than KD, Etame AB, La Marca F, Park P (2009) Impact of anesthesia on transcranial electric motor evoked potential monitoring during spine surgery: a review of the literature. Neurosurg Focus 27(4):E7
133. Whittle IR, Johnston IH, Besser M, Taylor TK, Overton J (1984) Intra-operative spinal cord monitoring during surgery for scoliosis using somatosensory evoked potentials. Aust N Z J Surg 54(6):553–557
134. Wilber RG, Thompson GH, Shaffer JW, Brown RH, Nash CL Jr (1984) Postoperative neurological deficits in segmental spinal instrumentation. A study using spinal cord monitoring. J Bone Joint Surg Am 66(8):1178–1187
135. Wilson-Holden TJ, Padberg AM, Parkinson JD, Bridwell KH, Lenke LG, Bassett GS (2000) A prospective comparison of neurogenic mixed evoked potential stimulation methods: utility of epidural elicitation during posterior spinal surgery. Spine 25(18):2364–2371
136. Wohrle JC, Behrens S, Mielke O, Hennerici MG, Wohrle JC, Behrens S, Mielke O, Hennerici MG (2004) Early motor evoked potentials in acute stroke: adjunctive measure to MRI for assessment of prognosis in acute stroke within 6 hours. Cerebrovasc Dis 18(2):130–134

Part II

Degenerative Spine

Cervical Disk Herniation and Radiculopathy

12

Selvon F. St. Clair and John M. Rhee

12.1 Case Example

A 41-year-old male presents with mild neck pain and persistent left arm pain radiating from the left side of his neck, down the anterolateral aspect of his arm and forearm into his thumb and index finger. He also has associated numbness but no weakness. His symptoms started after carrying a heavy object on his head/neck and progressively worsened over the last 2 months. His examination reveals 5/5 strength in all upper muscle groups, a negative Hoffman's sign, and normal reflexes. His Spurling's test is positive. Plain radiographs (Fig. 12.1) are unremarkable, and MRI images (Fig. 12.2) reveal a foraminal soft disk herniation at C5–6 compressing the C6 nerve root.

S.F. St. Clair, M.D., Ph.D. (✉)
Orthopaedic Institute of Ohio,
801 Medical Drive, Lima, OH 45804, USA
e-mail: stclas01@gmail.com

J.M. Rhee, M.D.
Associate Professor of Orthopaedic Surgery,
Emory University School of Medicine,
Emory Spine Center, Suite 3000,
59 Executive Park South, Atlanta, GA 30329, USA
e-mail: john.rhee@emory.org

12.2 Pathology and Pathophysiology

Although cervical nerve root compression has a variety of causes, the majority of cases can be attributed to ventrally based pathology arising from either "soft" or "hard" disk herniations. Acute soft disk herniations may impinge on the exiting nerve root ventrolaterally at its takeoff from the spinal cord, or intraforaminally as it traverses the neuroforamen (Fig. 12.1). On the other hand, a hard disk results from the spondylotic cascade, which involves chronic disk degeneration, disk height loss, annular bulging, and formation of degenerative osteophytes, which typically arise from the uncinate regions of the posterolateral vertebral body (uncovertebral osteophytes) [1] (Figs. 12.2 and 12.3). Both tend to compress the exiting nerve root as it enters the neuroforamen.

The neuroanatomy of the cervical nerve roots as they exit the spinal cord ventrolaterally at an approximately 45° angle to enter the neuroforamen renders them susceptible to stretch over these ventral lesions. As a result, in some patients, significant pain relief may be obtained by abducting the arm, which presumably decreases the amount of stretch and tension on the compressed nerve root (i.e., shoulder abduction relief sign). These patients describe pain relief when placing their hand on their head. Lastly, the inflammatory local milieu created by herniated nuclear material includes substance P, bradykinin, tumor necrosis factor-α,

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_12, © Springer-Verlag Berlin Heidelberg 2014

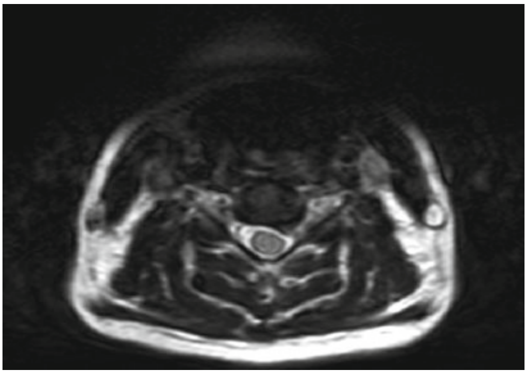


Fig. 12.1 Axial T2-weighted magnetic resonance imaging scan showing left posterolateral soft disk herniation compressing of the exiting root

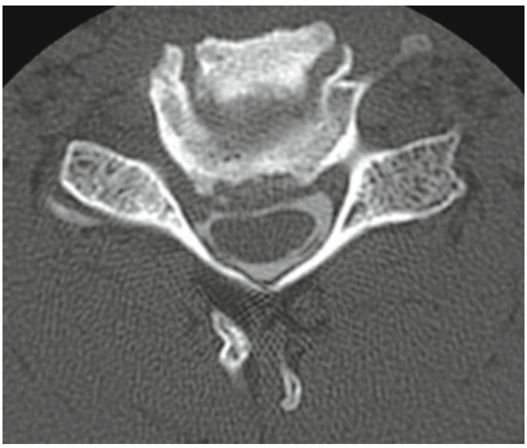


Fig. 12.2 Axial postmyelogram computed tomography scans an uncovertebral spur on the right. The axial slice cuts obliquely through the disk space and through the foramen on the right

prostaglandins, and other pain mediators. These have been associated with radicular symptoms.

12.3 History and Physical Exam

Patients presenting with pure cervical spondylotic radiculopathy typically complain of pain along a nerve root distribution. A subset of patients may also have frank neurologic dysfunction – i.e., weakness and numbness. The extent of symptoms is unpredictable and can vary from patient to patient. Radiculopathy is characteristically unilateral neck pain, which then radiates in

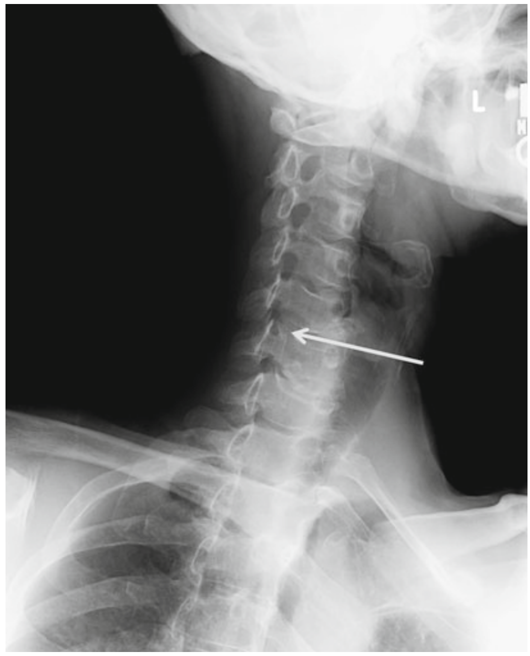


Fig. 12.3 Plain oblique radiograph demonstrating uncovertebral osteophytes causing foraminal narrowing C5–6 (*arrow*)

the distribution of the affected root. In patients with broad herniations or osteophytes with significant cord compression, occipital headaches may be part of their symptomatolgy. In addition, patients may also complain of upper trapezium and interscapular pain. Myelopathy can be seen in association with radiculopathy when there is concomitant spinal cord compression [2].

Table 12.1 lists typical pain and neurologic patterns associated with radiculopathies of the cervical nerve roots. The most common levels of root involvement are C6 and C7, arising from the C5–6 and C6–7 disk levels, respectively. Although C2–4 radiculopathies are less common, they can present with headaches, unilateral upper trapezial pain, and neck pain. Physical examination is performed to help localize the nerve root involved, but doing so can be challenging due to crossover within myotomes and dermatomes. Unlike the thoracic and lumbar spine, cervical nerve roots 1–7 exit above their correspondingly numbered pedicles (e.g., the C6 root exits between C5 and C6), thereby resulting in compression of the exiting root by either posterolateral disk herniations or spondylotic foraminal

Table 12.1 Common cervical radiculopathy patterns

Root	Symptoms	Affected motor function	Reflex
C2	Posterior occipital headaches, temporal pain	None	None
C3	Occipital headache, retro-orbital or retroauricular pain	None	None
C4	Base of neck, trapezius pain	None	None
C5	Lateral arm pain	Deltoid	Biceps
C6	Radial forearm pain, pain in the thumb and index fingers	Biceps, wrist extension	Brachioradialis
C7	Middle finger pain	Triceps, wrist flexion	Triceps
C8	Pain in the ring and little fingers	Finger flexors	None
T1	Ulnar forearm pain	Hand intrinsics	None

stenosis. A large central to mid-lateral disk herniation or stenosis may, however, cause a radiculopathy of the next lower nerve root. Motor strength is graded on a 0–5 scale (5 = normal; 4 = motion against some resistance; 3 = motion against gravity; 2 = motion with gravity eliminated; 1 = muscle contraction without movement; 0 = no evidence of contraction). Sensory testing should include at least one function from the dorsal columns (e.g., joint position sense, light touch) and the spinothalamic tract (e.g., pain and temperature sensation).

Several provocative tests may elicit or reproduce symptoms of radiculopathy. A Spurling's maneuver may reproduce the radicular symptoms in a patient with a foraminal disk or stenosis. The neck is maximally extended and rotated to the side of the pathology, which narrows down a symptomatic neuroforamen. Concomitant adduction of the shoulder with extension of the elbow and wrist may accentuate the Spurling's sign, as these maneuvers not only narrow the foramen but also stretch the root. Improvement of symptoms may occur if the patient subsequently flexes and rotates the neck to the other side and abducts the shoulder with the hand behind the neck, as these maneuvers both open up the foramen and relax the root.

12.4 Differential Diagnosis

The differential diagnosis of cervical radiculopathy is broad. (1) Peripheral nerve entrapment syndromes (e.g., carpal or cubital tunnel syndromes) may mimic the symptoms of radiculopathy but usually do not radiate from the neck. In addition, they may present with positive Phalen's or Tinel's sign, and nerve conduction studies can be helpful to differentiate these pathologies from cervical radiculopathy. (2) Rotator cuff pathology presents with pain in the shoulder or arm, usually above the elbow, made worse with shoulder movements. Impingement signs may be present, and reflex and sensory function should not be affected. (3) Thoracic outlet syndrome is difficult to diagnose. It may present with pain in the shoulder and arm, intermittent paresthesias, often symptoms in a C8–T1 distribution, normal neurological exam, a positive Adson's maneuver, and normal nerve conduction study (NCS). (4) Parsonage-Turner syndrome (acute brachial plexitis) usually presents as a pain in the neck, shoulder, and arm followed within days to weeks by weakness, especially in the C5–6 region as the pain recedes. In contrast, pain and weakness typically coexist in cervical radiculopathy. (5) Herpes zoster usually presents with neuropathic pain in a dermatomal distribution followed by vesicular rash. (6) Visceral disorders, such as coronary artery disease and cholecystitis, both of which cause referred pain to the upper extremity, also should be considered in the differential diagnosis.

12.5 Investigations/Diagnostic Imaging

Plain radiographs are typically the first diagnostic test ordered in patients with clinical suspicion of radiculopathy. It may reveal decreased disk height and osteophyte formation (Fig. 12.3). MRI is the imaging modality of choice in the patient who does not respond well to nonsurgical management or who has severe symptoms. However, clinical correlation is paramount since it is not uncommon to see nerve root compression in patients without symptoms [3]. Although invasive, CT myelography is particularly helpful in delineating bony surgical anatomy and differentiating whether root compression arises from hard versus soft disk pathology (Fig. 12.2). The images obtained with either modality depend in part on the position of the neck at the time of acquisition. MRI and postmyelogram CT images are most commonly obtained with the patient supine, which promotes a neutral or slightly flexed sagittal contour to the neck. This positioning may result in under diagnosis of conditions that are symptomatic in the extremes of flexion (e.g., mild disk herniation) or extension (e.g., mild foraminal stenosis) [4].

12.6 Treatment

The majority of symptomatic patients are initially offered a trial of nonoperative treatment. Operative treatment is typically reserved for the subset of patients who either failed nonoperative treatment or present with moderate to severe symptoms with massive disk herniation or objective signs of muscle weakness and/or numbness.

12.6.1 Nonoperative Treatment

Nonoperative treatment generally consists of nonsteroidal anti inflammatory (NSAID) medications, immobilization, traction, physical therapy, and steroid injections. Narcotics are only recommended in the acute setting as breakthrough treatment to supplement NSAIDs or in patients who cannot tolerate NSAIDs. Although liberally prescribed, the effectiveness of physical therapy, traction, or immobilization for cervical radiculopathy is unclear. Also in the nonoperative armamentarium are epidural steroid injection and cervical selective nerve root injection. In general, they are sparingly employed as they have no proven long-term benefit and are associated with very rare, but potentially devastating complications. Selective nerve root injections can, however, be very useful in identifying the symptomatic root(s) when the level is unclear for the purposes of surgical planning [5–9].

12.6.2 Operative Treatment

Surgery is indicated in patients who have failed a reasonable trial of conservative management or patients with incapacitating pain or progressive neurologic deficits such as muscle weakness and/or numbness. Depending on the pathology, cervical radiculopathy may be surgically addressed either anteriorly or posteriorly. Regardless of the approach, the main goal for surgery is the same: decompress the affected nerve root (s). Currently, there are two major anterior-based surgeries: anterior cervical decompression and fusion (ACDF) (Fig. 12.4a) and the recently approved cervical disk replacement (CDR) (Fig. 12.4b) [3, 10–12]. The attendant advantages of ACDF and CDR are essentially the same and include direct removal of the offending compressive material (disk herniations or osteophytes); restoration of disk space height which opens up the neuroforamen, thereby providing indirect decompression of the nerve root; extremely low rates of infection and wound complications; cosmetically appealing incisions, especially when placed in the creases of the neck; and very little perioperative pain. A unique advantage of ACDF is the associated fusion, which may help to improve any component of neck pain arising from disk degeneration and spondylosis. CDR advantages over fusion include maintenance of motion, avoidance of nonunion, and avoidance of plate-and-screw complications, such as back out, esophageal erosion, and periplate ossification.

Posterior laminoforaminotomy is the posterior-based surgery of choice for cervical radiculopathy, particularly in patients with anterolateral disk herniation or foraminal stenosis [13, 14]. This approach relies on basically unroofing the foramen to adequately decompress the root. The

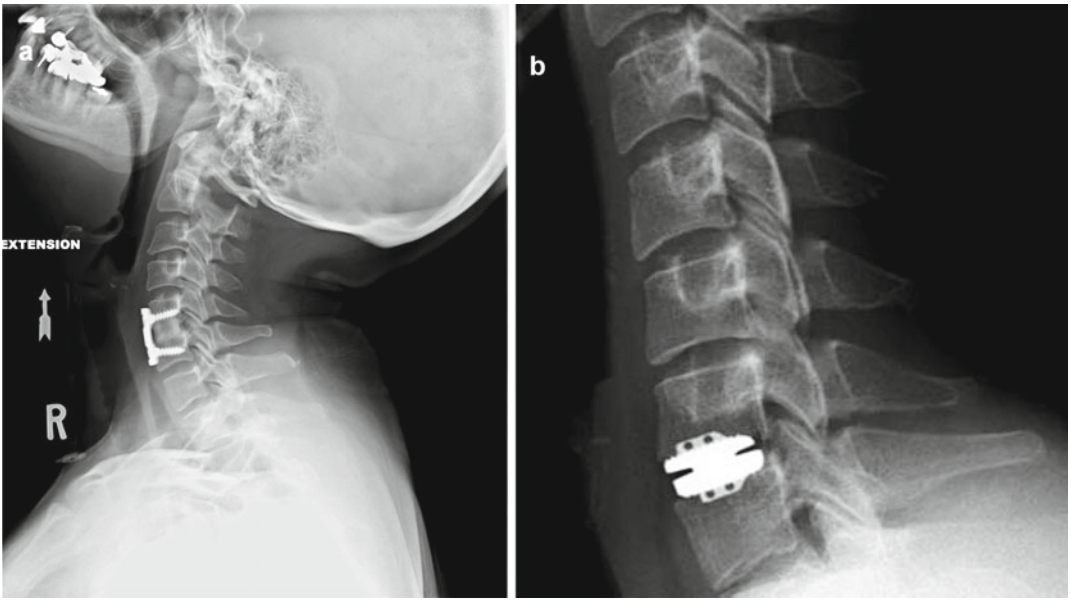


Fig. 12.4 (**a**, **b**) Lateral radiographs after anterior cervical diskectomy and fusion (**a**) and cervical disk replacement (**b**) for soft disk herniation

disk herniation can be removed posterolaterally but typically requires drilling away the superior pedicle of the inferior vertebra to allow safe access to the disk space while at the same time limiting neural retraction. The advantages of laminoforaminotomy are minimal morbidity and avoidance of fusion. The disadvantages of the posterior approach include the potential for incomplete decompression, as uncovertebral osteophytes are typically not removable though this approach; neural traction injury; the inability to restore disk and foraminal height at the diseased level; and the potential for recurrent syndromes over time at the index level.

12.7 Outcomes of Surgical Management

Regardless of surgical procedure used, decompression of cervical nerve roots has been shown to be very successful. About 80–90 % improvement in relief of arm pain as well as improvements in motor and sensory function should be expected [15, 16]. In a randomized trial of 44 patients, there were no statistical differences in outcomes between posterior laminoforaminotomy and ACDF (unplated with autograft), although ACDF yielded better long-term results. Laminoforaminotomy [13] also lends itself to a minimally invasive approach, and microendoscopic laminoforaminotomy has demonstrated good to excellent clinical results in 97 % of patients [14, 17].

12.8 Postoperative Care

Perioperative IV antibiotic is started within 1 h of the incision and continued for 24 h. Precaution against heaving and forceful neck range of motion is discouraged for at least 6–12 weeks. The use of an external orthosis (cervical collar) is sometimes recommended for 6–12 weeks for anterior cervical interbody fusion. Physical therapy is not routinely necessary after ACDF. For patients who either had an ACDF or a CDR, interval radiographic follow-up is needed to monitor fusion healing as well as implant position. Following CDR and posterior laminoforaminotomy procedures, early range of motion may be employed.

12.9 Potential Complications

Severe complications after anterior cervical surgery are relatively uncommon. Acute catastrophic complications include airway obstruction from edema or hematoma, esophageal perforation, and neurologic injury. We recommend overnight observation in the hospital with a deep drain in place due to the potential for devastating airway complications. Temporary dysphagia is universal after anterior cervical approaches, and patients should be counseled preoperatively to expect it to occur. Long-term severe dysphagia is rare, although some degree may persist for 6–12 months. Dysphonia can result from injuries to the superior or recurrent laryngeal nerves. Typically, these are traction or stretch injuries that resolve over time. Long-term complications related to cervical fusion include pseudarthrosis. Data on the longevity of CDR is lacking at present, as this technology remains in its early stages, but there is sure to be a certain failure rate of these devices over time. Although rare in anterior cervical surgery, infection is the most common complication of posterior cervical surgery. Spinal cord and nerve root injury are extremely rare after laminoforaminotomy.

Questions

1. Cervical HNP at C6/7 level causing compression to the C7 nerve root results in weakness primarily of the following:
   (a) Wrist flexors and finger flexors
   (b) Elbow flexors and wrist flexors
   (c) Elbow flexors and finger flexors
   (d) Elbow extensors and wrist flexors
   (e) Elbow extensors and wrist extensors
   Preferred response (d). C7 innervates elbow extensors and wrist flexors. It primarily provides sensation to the middle finger and is associated with the triceps reflex.
2. A 45-year-old man has had spontaneous neck and right arm pain for the past 2 days, and he states that the pain is relieved when he places his hand on the top of his head. Examination reveals decreased sensation on the dorsum of the first web space, weakness in the wrist extensors, and an absent brachioradialis reflex. The remainder of the examination is unremarkable. What is the most likely diagnosis?
   (a) Double crush phenomenon with carpal tunnel syndrome and cervical disk herniation at C5–6
   (b) Cervical disk herniation at C6–7
   (c) Cervical disk herniation at C5–6 with myelopathy
   (d) Acute cervical disk herniation at C5–6
   (e) A shoulder impingement lesion and cervical disk herniation at C6–7
   Preferred response (d). The double crush syndrome with C5–6 herniation is not the right answer since no median nerve compression signs are given. C5–6 herniation with myelopathy is not the correct answer since myelopathy usually presents with generalized weakness (upper > lower), deterioration in gait and manual dexterity, sensory changes, spasticity, and rarely urinary/bowel incontinence. Also look for hyperreflexia and other provocative physical findings, such as the Hoffman's sign (flicking nail of middle finger and abnormal twitching of thumb or index finger). The correct answer is acute cervical disk herniation at C5–6.

References

1. Lees F, Turner JW (1963) Natural history and prognosis of cervical spondylosis. Br Med J 2:1607–1610
2. Boden SD, McCowin PR, Davis DO, Dina TS, Mark AS, Wiesel S (1990) Abnormal magnetic-resonance scans of the cervical spine in asymptomatic subjects: a prospective investigation. J Bone Joint Surg Am 72: 1178–1184
3. Rhee JM, Yoon T, Riew D (2007) Cervical radiculopathy. J Am Acad Orthop Surg 15(8):486–494
4. Rhee JM, Riew KD (2005) Evaluation and management of neck pain, radiculopathy, and myelopathy. Semin Spine Surg 17:174–185
5. Levine MJ, Albert TJ, Smith MD (1996) Cervical radiculopathy: diagnosis and nonoperative management. J Am Acad Orthop Surg 4:305–316
6. Santiesteban AJ (1983) The role of physical agents in the treatment of spine pain. Clin Orthop Relat Res 179:24–30
7. Hurwitz EL, Aker PD, Adams AH, Meeker WC, Shekelle PG (1996) Manipulation and mobilization of the cervical spine: a systematic review of the literature. Spine 21:1746–1759
8. Stav A, Ovadia L, Sternberg A, Kaadan M, Weksler N (1993) Cervical epidural steroid injection for cervicobrachialgia. Acta Anaesthesiol Scand 37:562–566
9. Saal JS, Saal JA, Yurth EF (1996) Nonoperative management of herniated cervical intervertebral disc with radiculopathy. Spine 21:1877–1883

10. Watters WC III, Levinthal R (1994) Anterior cervical discectomy with and without fusion: results, complications. Spine 19(20):2343–2347
11. Rhee JM, Park JB, Yang JY, Riew DK (2005) Indications and techniques for anterior cervical plating. Neurol India 53:433–439
12. Jho HD, Kim WK, Kim MH (2002) Anterior microforaminotomy for treatment of cervical radiculopathy: I. Disc-preserving "functional cervical disc surgery". Neurosurgery 51(5 suppl):S46–S53
13. Herkowitz HN, Kurz LT, Overholt DP (1990) Surgical management of cervical soft disc herniation: a comparison between the anterior and posterior approach. Spine 15:1026–1030
14. Henderson CM, Hennessy RG, Shuey HM Jr, Shackelford EG (1983) Posterior lateral foraminotomy as an exclusive operative technique for cervical radiculopathy: a review of 846 consecutively operated cases. Neurosurgery 13:504–512
15. Bohlman HH, Emery SE, Goodfellow DB, Jones PK (1993) Robinson anterior cervical discectomy and arthrodesis for cervical radiculopathy: long-term follow-up of one hundred and twenty two patients. J Bone Joint Surg Am 75:1298–1307
16. Sampath P, Bendebba M, Davis JD, Ducker T (1999) Outcome in patients with cervical radiculopathy: prospective, multicenter study with independent clinical review. Spine 24:591–597
17. Adamson TE (2001) Microendoscopic posterior cervical laminoforaminotomy for unilateral radiculopathy: results of a new technique in 100 cases. J Neurosurg 95:51–57

Cervical Spondylotic Myelopathy (CSM)

13

Prokopis Annis and Alpesh A. Patel

13.1 Case Example

A 58-year-old female presents with progressive neck pain as well as numbness and weakness in her arms that has worsened over the past 5 months. She also describes a problem with steadiness in her walking and now requires a cane to help with balance. She has had progressive loss of manual dexterity and clumsiness in her hands. Her examination reveals 4/5 strength in the biceps and triceps, hyperreflexia 3+ in her triceps, positive Hoffmann's sign, and dysdiadochokinesia in her upper extremities. Plain radiographs (Fig. 13.1) and MRI images (Fig. 13.2a–c) reveal spondylotic changes at C5–6 and C6–7 with spinal cord compression and spinal cord signal change.

13.2 Pathology/Pathophysiology

The pathophysiology of CSM is likely multifactorial. Spinal canal compression that is the result of age-related degenerative changes of the intervertebral disks and facet joints is one component. A congenitally narrow canal further increases the risk of developing CSM due to the lack of volumetric reserve within the spinal canal [1]. Normal space available for the cervical spinal cord has been estimated to be 17–18 mm (from 13 to 20 mm) [2, 3]. A spinal canal diameter less than 13 mm can cause compression of the spinal cord, while a diameter less than 12 mm is strongly correlated with myelopathy. Additionally, vascular mechanisms likely play a role in symptomatic disease [4]. Furthermore, an underlying genetic predisposition to CSM has been suggested by twin studies as well as population-based reviews [5].

The pathophysiology of CSM results from the sequelae of the aging process on the spine. Disk degeneration is an age-related process that is characterized by disk desiccation and progressive loss of elasticity with final disk height loss and annular bulging. Disk height loss can also cause buckling of the ligamentum flavum. Together these are responsible for canal narrowing. Ligamentum flavum hypertrophy, osteophyte formation, and uncovertebral and facet joint hypertrophy can lead to further central stenosis. In advanced cases, the development of kyphosis may accentuate the degree of stenosis. Finally, ossification of the posterior longitudinal ligament (OPLL) can be another cause of stenosis [6, 7].

All the above-mentioned structural changes of the cervical spine represent static changes that occur in degenerative disease. A dynamic model of spinal canal narrowing has been proposed. In the presence of the static structural changes of the

P. Annis
Department of Orthopaedic Surgery,
University of Utah Neurosciences Center,
590 Wakara Way, Salt Lake City, UT 84108, USA

A.A. Patel, M.D., FACS (✉)
Department of Orthopaedic Surgery,
Northwestern University,
676 N St. Clair St., Suite 1350,
Chicago, IL 60611, USA
e-mail: alpesh2@gmail.com

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_13,

Fig. 13.1 Lateral radiograph of cervical spine. C5–6 and C6–7 degenerative changes (spondylosis) are visualized. *Dashed line* represented measurement of spinal canal diameter

spine that result from the degenerative process, an irreversible damage of the spinal cord can occur during hyperflexion or hyperextension. Translation between vertebral bodies during flexion or extension (motion segment instability) results in narrowing of the spinal canal space available for the spinal cord and contributes to dynamic cord compression [8–10].

In 1924, Barre proposed that vascular factors may also play a role in the development of cervical spondylotic myelopathy [11]. Breig et al. showed that canal narrowing can produce reduction of the blood flow of the anterior spinal artery and radicular arteries. The transverse intramedullary arteries that arise from the anterior sulcal arteries perfuse the gray matter and the adjacent lateral columns [4]. Shinomura et al. published an experimental study of ischemic damage in conjunction with compression of the cord in a dog. They concluded that obstruction of these arteries produces the pathological process of myelopathy [12].

Histologic changes associated with myelopathy include axonal demyelinization followed by cell necrosis and gliosis or scarring [13–15]. Cystic cavitation may occur in the gray matter. Ogino et al. examined pathological specimens and correlated the findings with the degree of cord compression [16]. Mild to moderate compression was associated with degeneration of the lateral white matter tracts. More severe compression led to necrosis of the central gray matter.

While its incidence is unknown, CSM is most prevalent in adults over 55 years of age. Crandall and Batzdorf found CSM to be most common clinically at the C5–6 level, followed by C4–5 [17]. The true natural history of this disorder may be difficult to determine because in the majority of the cases, the diagnosis is made late and symptoms are often attributed to advanced age or other neurological conditions.

Based on studies by Spillane and Loyd in 1952 and Clarke and Robinson in 1956, it has been shown that CSM is a condition of gradual stepwise progression with the following distributions: 75 % have episodic progression, 20 % show a gradual slow progression, and 5 % have rapid onset of symptoms followed by long periods of stability [18, 19]. Also, Lees and Turner reviewed 54 patients who were followed for 3 years [20]. They showed that even if symptoms of CSM remain stable, there was progressive neurological deterioration. They also showed that neither the age of onset of CSM nor conservative treatment with collar immobilization or surgery appeared to influence the prognosis. A multicenter, nonrandomized study by the Cervical Spine Research Society suggested similarly poor results with nonoperative management of CSM [21]. A more recent study from Sadasivan and coworkers followed 22 patients with CSM and found that all patients had slow but progressive worsening of their symptoms [22]. It can, therefore, be concluded that the majority of patients with CSM should expect slow but progressive neurological deterioration.

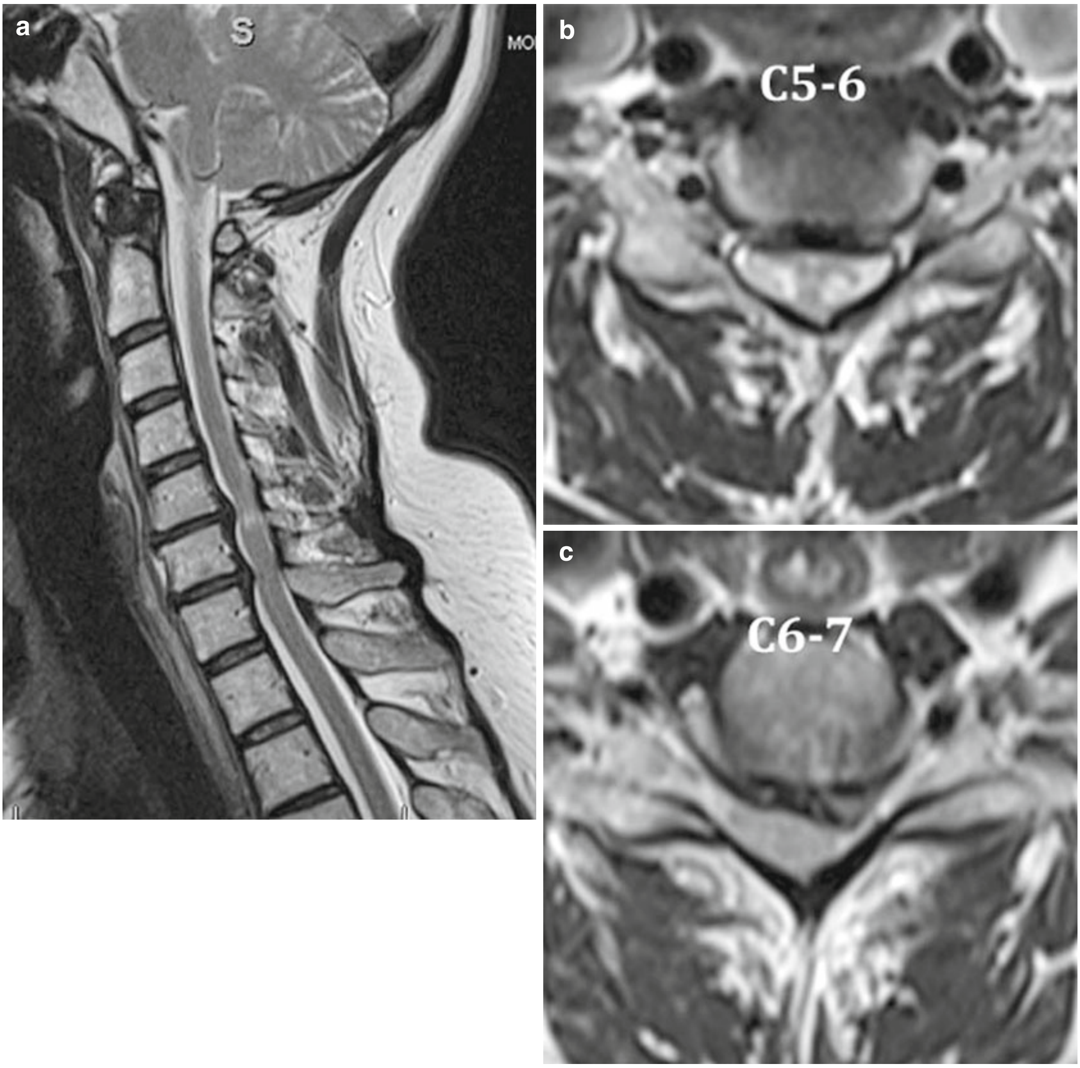


Fig. 13.2 (**a**) Sagittal and (**b**, **c**) axial T2-weighted MRI demonstrates spinal stenosis and cord compression at C5–6 and C6–7. Signal change is visualized within the substance of the spinal cord between C5–6 and C6–7

13.3 History and Physical Exam

The patient with CSM may be asymptomatic or present with an array of symptoms, often present for years, making the diagnosis difficult. Patients with mild symptoms may complain of neck pain and paresthesias without any dermatomal distribution. Subtle gait changes may be present in patients in this stage. In moderate or severe myelopathy, patients present with more severe gait abnormalities or a sense of unsteadiness and balance difficulties. Fine motor control is usually affected, so patients may note difficulty in manipulating small objects (buttons, keys). Numbness, paresthesias, and arm and leg weakness are usually present. Thus, patients notice difficulty in moving their body, going up stairs, etc. In extremely severe cases, bladder and bowel dysfunction may appear, although this is thought to be rare.

The Nurick classification system is commonly used to stratify patients based on the severity of gait disturbance (Table 13.1) [23].

Table 13.1 Nurick classification of disability from cervical myelopathy

Grade I	No difficulty in walking
Grade II	Mild gait involvement not interfering with employment
Grade III	Gait abnormality preventing employment
Grade IV	Able to walk only with assistance
Grade V	Chairbound or bedridden

13.4 Physical Examination

There must be a high degree of suspicion for the diagnosis of myelopathy. Neck ROM is essential to evaluate as many patients with CSM present with painful and restricted neck extension. A detailed neurological examination is required to detect potential motor and sensory changes. In addition to traditional motor/sensory exams (Chap. 3), specific exam findings may be more unique to patients with CSM.

In 1987, Oko et al. described "the myelopathy Hand" [24]. It results in loss of dexterity and is associated with wasting of the intrinsic muscles of the hand and spasticity. They proposed two tests sensitive for its diagnosis. The first is "the finger escape sign" that reveals a deficient adduction and/or extension of the ulnar two or three fingers. The second test is "the grip-and-release test." Normally, one can make a fist and rapidly release it 20 times in 10 s. Patients with myelopathy may be unable to do this that quickly.

Reflex changes are of extreme importance in making the diagnosis. In CSM, upper extremity reflexes are increased below the level of compression (hyperreflexia). In addition to hyperreflexia, pathological reflexes may be present. A positive Hoffmann's sign is elicited by hyperextending the distal interphalangeal joint of the long finger and observing thumb adduction and/or finger flexion. Together with ankle clonus and a positive Babinski sign, these are found in varying degrees in patients with moderate or severe myelopathy.

The gait of the myelopathic patient should also be observed. It is typically a wide-based gait and must be differentiated from that of other neurological diseases, such as peripheral neuropathy or Parkinson disease. A tandem gait (heel-to-toe, single-line walk) may detect early gait disturbances associated with loss of normal proprioceptive function.

13.5 Differential Diagnosis

Cervical spondylotic myelopathy can mimic many other disorders. The most common of these is multiple sclerosis, a demyelinating disease of the central nervous system causing both motor and sensory abnormalities. Amyotrophic lateral sclerosis affects both upper and lower extremity motor neurons of the central nervous system. Tumors of the spinal cord and syringomyelia may cause root weakness combined with spasticity below the involved level. Gait abnormality of the cervical spondylotic myelopathy can be confused with the gait of Parkinson disease or that associated with severe peripheral neuropathy.

13.6 Diagnostic Imaging

Anteroposterior and lateral radiographs are an important part of the diagnostic workup (Fig. 13.2a, b). They may display disk space narrowing, endplate sclerosis, and uncovertebral and facet joint osteophytes that are the proof of the degenerative process. The sagittal diameter of the spinal canal and the presence of congenital stenosis on lateral radiographs can be identified based on the distance between the posterior vertebrae and the spinolaminar line (Fig. 13.1). Cervical alignment (lordosis vs. kyphosis) is also determined on lateral radiographs. Flexion and extension films can also reveal abnormal cervical spine mobility.

MRI is the gold standard diagnostic exam for the evaluation of myelopathy. It clearly defines the extent of cord compression and shows pathological changes of the spinal cord that are important for planning the treatment options. (Fig. 13.2a–c) The MRI signal has been correlated to the severity of spinal cord lesions. A hypointense signal in T1 associated with a hyperintense signal in T2 indicates lesions of microcavitation or gliosis of the spinal cord.

13.7 Treatment

There is controversy about the treatment of CSM. Given the progressive neurological deterioration in a majority of the cases over time, surgical treatment is typically recommended in patients with moderate to severe disease with the hope of avoiding irreversible cord injury.

13.7.1 Nonoperative Treatment

In general, nonoperative treatment is recommended in asymptomatic patients and in patients with mild disease. It consists of observation, intermittent soft collar immobilization for pain, nonsteroidal anti-inflammatory (NSAID) medications for pain, avoidance of high-risk activities, and physiotherapy. Unfortunately there is little evidence to suggest that nonoperative treatment alters the natural history of the CSM. Nonetheless, there are data to suggest that closely observed nonoperative treatment may be an option for selecting patients with CSM.

13.7.2 Operative Treatment

Surgery is indicated in patients with severe or progressive clinical myelopathy with concordant radiographic evidence of spinal stenosis [25, 26]. The decision regarding surgery is often multifactorial, depending on the severity of disease, the clinical signs of spinal cord compression in concordance with the imaging, the duration of symptoms, and the patients' comorbidities. Common operative indications include progressive neurological deficit, progressive gait disturbances, spinal canal narrowing with cord compression, and spinal cord signal change.

Operative treatment is most effective for halting the progression of disease and neurological dysfunction. Though many patients will show some improvement, others may, unfortunately, demonstrate persistent symptoms. Operative treatment results have been showed to be poorest in patients with prolonged spinal cord compression. Suri et al. reported that patients with a duration of symptoms less than a year showed significantly better motor recovery than those with a longer duration of symptoms [27].

13.7.3 Which Approach?

The primary goal of surgery is to decompress the spinal cord and canal in order to improve function. The choice between an anterior, posterior, or combined approach is based on (1) the sagittal alignment of the cervical spine – patients with kyphosis generally require an anterior procedure to both improve alignment and maintain the posterior tension band; (2) extent of the disease – multilevel disease is better treated posterior or combined anterior-posterior approaches; (3) location – ventral compression is more directly treated with an anterior approach; (4) presence of preoperative neck pain – patients with severe neck pain often need fusion to treat the arthritic pain; (5) previous operations – it may be easier and less risky to avoid revision approaches; and (6) surgeon and patient preference – when multiple options are equivalent, the decision should be based on the surgeon and the patient choice [28].

Anterior spinal decompression is commonly performed for one- to three-level disease, with ventral spinal canal compression (Fig. 13.3). Additionally, the presence of radicular symptoms may also indicate an anterior procedure. The presence of significant cervical kyphosis requires an anterior (or combined) procedure to restore lordosis. A combination of anterior discectomy and/or anterior corpectomy and fusion can be performed. Anterior plating is recommended in order to increase fusion rates, especially in multilevel procedures.

In cases of severe developmental stenosis or in extensive involvement of multiple spinal levels (Three or more), posterior cervical decompression provides access to a greater number of levels while decreasing the risk of pseudarthrosis associated with multilevel anterior procedures. Additionally, posterior surgery can directly address dorsal spinal cord compression (congenital stenosis, ligamentum flavum) [29, 30]. Laminectomy decompression and fusion with instrumentation and laminoplasty are the options in posterior surgery. Cervical kyphosis is an absolute contraindication for a

Fig. 13.3 Lateral radiography of cervical spine after anterior cervical decompression (*corpectomy*) and fusion C5–7 with anterior instrumentation

posterior-only approach as insufficient spinal cord migration and decompression may occur, resulting in continued cord compression across the kyphotic segments [31, 32].

13.7.4 Results of Surgery: Anterior Versus Posterior

Laminectomy associated with arthrodesis or laminoplasty has been reported to provide good long-term results in about 80 % of the patients. Anterior decompression improves clinical symptoms in approximately 90 % of patients. Several comparative studies reviewed the clinical course of patients operated for CSM with either anterior or posterior approaches and concluded that the results were without significant difference in neurological recovery. Immediate recovery that was obtained immediately after surgery was followed by a period of stability and then some deterioration of the functional status. For the anterior approach, the main cause of recurrence of symptoms was the adjacent segment disease, while for the posterior approach failure was attributed to kyphosis or instability.

13.8 Postoperative Care

All patients should be maintained on IV antibiotics for 24 h postoperative. Diet following either anterior or posterior cervical fusion procedures and postoperative precautions regarding range of motion, lifting, and work are often recommended. The use of an external orthosis (cervical collar) may be recommended for 6–12 weeks. After a period of immobilization, physical therapy can be directed at improving range of motion and strength of the cervical spine and upper extremities. Frequent radiographic follow-up is needed to monitor fusion healing as well as any potential instrumentation-related complications (plate or screw failure, graft subsidence, or displacement). Following posterior laminoplasty procedures, less precautions and restrictions are needed. Early range of motion and avoidance of cervical orthoses can speed recovery for patients after laminoplasty.

13.9 Potential Complications

The most common complications after anterior cervical surgery include dysphagia, dysphonia, and hoarseness of voice. Spinal cord injury, nerve injury [33], and infection are rare. Other rare complications include injury to the esophagus, carotid sheath, or vertebral artery. The most complication of posterior cervical surgery is infection. Other potential complications include spinal cord injury, nerve injury, and vertebral artery injury. C5 nerve root palsy has been described after both anterior and posterior approaches. Symptoms, which typically begin 24–48 h postoperatively, include pain, numbness, and weakness in a C5 distribution (lateral shoulder/arm and deltoid muscle). While the exact etiology of postoperative C5 root palsy [34] is unclear, the natural history is generally favorable with most patients regaining neurological function over the course of weeks to months.

Questions

1. A 68-year-old male presents with complaints of neck pain with weakness and numbness in the hands. His symptoms have progressively worsened over the past 6 months. Examination reveals hand intrinsic weakness (3/5) in both hands with wasting of the thenar prominence. He is hyperreflexic in his biceps, triceps, and quadriceps with a positive Hoffmann's sign. His radiographs show multilevel spondylosis (C3–C7) with fixed kyphosis. MRI confirms spinal cord compression from C3–7 with spinal cord signal change. The best option for treatment at this time is:
   (a) Bracing and observation
   (b) Posterior laminoplasty C3–7
   (c) Posterior laminectomy and fusion C3–7
   (d) Anterior cervical decompression and fusion C3–7
   (e) Combined anterior and posterior decompression and fusion C3–7

   Preferred Response: The patient's symptoms, exam findings, and MRI confirm the diagnosis of cervical myelopathy. His radiographs demonstrate a fixed cervical kyphosis, making posterior-only surgery less effective, necessitating an anterior approach. Anterior C3–7 decompression and fusion is an option; however, the risk for nonunion is unacceptably high. Therefore, a combined anterior and posterior decompression and fusion accomplishes the goals of kyphosis correction, direct neurological decompression, and optimal fusion outcomes.

References

1. Parke WW (1988) Correlative anatomy of cervical spondylotic myelopathy. Spine 13:831–837
2. Wolf BS, Khilnani M, Malis L (1956) The sagittal diameter of the bony cervical spinal canal and its significance in cervical spondylosis. J Mt Sinai Hosp NY 23:283–292
3. Arnold JGJ (1955) The clinical manifestations of spondylochondrosis (spondylosis) of the cervical spine. Ann Surg 141:872–889
4. Breig A, Turnbull I, Hassler O (1966) Effects of mechanical stresses on the spinal cord in cervical spondylosis: a study on fresh cadaver material. J Neurosurg 25:45–56
5. Ball J, El Gammal T, Popham M (1969) A possible genetic factor in cervical spondylosis. Br J Radiol 42:9–16
6. Tsuyama N (1984) Ossification of the posterior longitudinal ligament of the spine. Clin Orthop 184:71–84
7. Smith MD, Bolesta MJ, Leventhal M, Bohlman HH (1992) Postoperative cerebrospinal-fluid fistula associated with erosion of the dura: findings after anterior resection of ossification of the posterior longitudinal ligament in the cervical spine. J Bone Joint Surg Am 74:270–277
8. Penning L, Zwaag PVD (1966) Biomechanical aspects of spondylotic myelopathy. Acta Radiol 5:1090–1103
9. White AAD, Panjabi MM (1988) Biomechanical considerations in the surgical management of cervical spondylotic myelopathy. Spine 13:856–860
10. Wilkinson M (1960) The morbid anatomy of cervical spondylosis and myelopathy. Brain 83:589–616
11. Barre JA (1924) Troubles pyramidaux et arthrite vertebrale chronique. Medecine, Paris
12. Shimomura Y, Hukuda S, Mizuno S (1968) Experimental study of ischemic damage to the cervical spinal cord. J Neurosurg 28:565–581
13. Bunge RP, Puckett WR, Becerra JL et al (1993) Observations on the pathology of human spinal cord injury. A review and classification of 22 new cases with details from a case of chronic cord compression with extensive focal demyelination. Adv Neurol 59:75–89
14. Mair WGP, Druckman R (1953) The pathology of spinal cord lesions and their relation to the clinical features in protrusion of cervical intervertebral discs: a report of four cases. Brain 76:70–91
15. Taylor AR (1953) Mechanism and treatment of spinal-cord disorders associated with cervical spondylosis. Lancet 1(6763):717–720
16. Ogino H, Tada K, Okada K, Yonenobu K, Yamamoto T, Ono K, Namiki H (1983) Canal diameter, anteroposterior compression ratio, and spondylotic myelopathy of the cervical spine. Spine 8:1–15
17. Crandall PH, Batzdorf U (1966) Cervical spondylotic myelopathy. J Neurosurg 25:57–66
18. Spillane JD, Lloyd GHT (1952) The diagnosis of lesions of the spinal cord in association with 'osteoarthritic' disease of the cervical spine. Brain 75:177–186
19. Clarke E, Robinson PK (1956) Cervical myelopathy: a complication of cervical spondylosis. Brain 79:483–510
20. Lees F, Turner JWA (1963) Natural history and prognosis of cervical spondylosis. BMJ 2:1607–1610
21. Sampath P, Bendebba M, Davis JD, Ducker TB (2000) Outcome of patients treated for cervical myelopathy. A prospective, multicenter study with independent clinical review. Spine 25:670–676
22. Sadasivan KK, Reddy RP, Albright JA (1993) The natural history of cervical spondylotic myelopathy. Yale J Biol Med 66:235

23. Nurick S (1972) The pathogenesis of the spinal cord disorder associated with cervical spondylosis. Brain 95:87–100
24. Ono K, Ebara S, Fuji T, Yonenobu K, Fujiwara K, Yamashita K (1987) Myelopathy hand. New clinical signs of cervical cord damage. J Bone Joint Surg Br 69-B(2):215–219
25. Fujiwara K, Yonenobu K, Ebara S, Yamashita K, Ono K (1989) The prognosis of surgery for cervical compression myelopathy: an analysis of the factors involved. J Bone Joint Surg Br 71:393–398
26. Koyanagi T, Hirabayashi K, Satomi K, Toyama Y, Fujimura Y (1993) Predictability of operative results of cervical compression myelopathy based on preoperative computed tomographic myelography. Spine 18:1958–1963
27. Suri A, Chabbra RP, Mehta VS, Gaikwad S, Pandey RM (2003) Effect of intramedullary signal changes on the surgical outcome of patients with cervical spondylotic myelopathy. Spine J 3:33–45
28. Rao RD, Gourab K, David KS (2006) Operative treatment of cervical spondylotic myelopathy. J Bone Joint Surg Am 88:1619–1640
29. Emery SE, Bohlman HH, Bolesta MJ, Jones PK (1998) Anterior cervical decompression and arthrodesis for the treatment of cervical spondylotic myelopathy:two to seventeen-year follow-up. J Bone Joint Surg Am 80:941–951
30. Epstein JA, Janin Y, Carras R, Lavine LS (1982) A comparative study of the treatment of cervical spondylotic myeloradiculopathy: experience with 50 cases treated by means of extensive laminectomy, foraminotomy, and excision of osteophytes during the past 10 years. Acta Neurochir (Wien) 61: 89–104
31. Hirabayashi K, Satomi K (1988) Operative procedure and results of expansive open-door laminoplasty. Spine 13:870–876
32. Kimura I, Shingu H, Nasu Y, Shiotani A, Oh-hama M, Murata M (1995) Long-term follow-up of cervical spondylotic myelopathy treated by canal-expansive laminoplasty. J Bone Joint Surg Br 77:956–961
33. Flynn TB (1982) Neurologic complications of anterior cervical interbody fusion. Spine 7:536–539
34. Satomi K, Nishu Y, Kohno T, Hirabayashi K (1994) Long-term follow-up studies of open-door expansive laminoplasty for cervical stenotic myelopathy. Spine 19:507–510

Thoracic Disc Herniation

14

Michael Fernandez and Sandeep N. Gidvani

14.1 Case Example

A relatively healthy 55-year-old lady, with a history of chronic low back pain and two prior lumbar surgeries, including a fusion, complained of increasing right knee pain. She was diagnosed with right knee osteoarthritis, and nonoperative treatment was started, which included physical therapy. During her treatment, she had an episode of severe low/mid back pain. She was able to temporarily continue therapy for her knee but began to complain of difficulty walking and unsteadiness in her gait. An MRI of her lumbar spine was performed, which showed expected postoperative changes, but no stenosis. A thoracic spine MRI was performed, which showed a large right paracentral thoracic disc herniation at T10–11 (see Fig. 14.1). She was referred to a spine surgeon for treatment.

Upon further questioning, she had recently experienced several episodes of bladder incontinence but was able to maintain control of her bowels. Her physical exam revealed a clumsy and wide-based, protected gait. Her motor exam revealed mild/moderate proximal lower extremity weakness. On sensory exam, she displayed hypesthesia in her right greater than left lower extremities. She exhibited hyperreflexia but no clonus. Her toes were down going. Rectal exam revealed normal sphincter tone, and she had normal sensation in her perineum. She was admitted for a T10–11 transfacet pedicle-sparing decompression and fusion (see Figs. 14.2 and 14.3). Following her procedure, she experienced

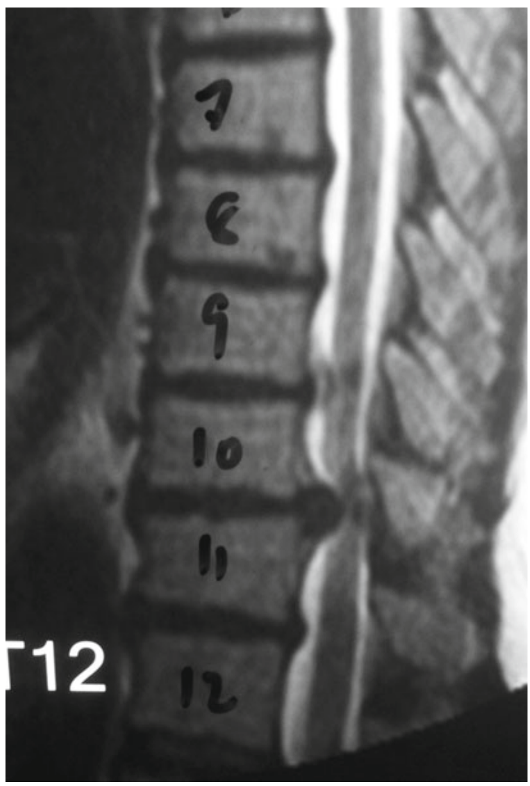
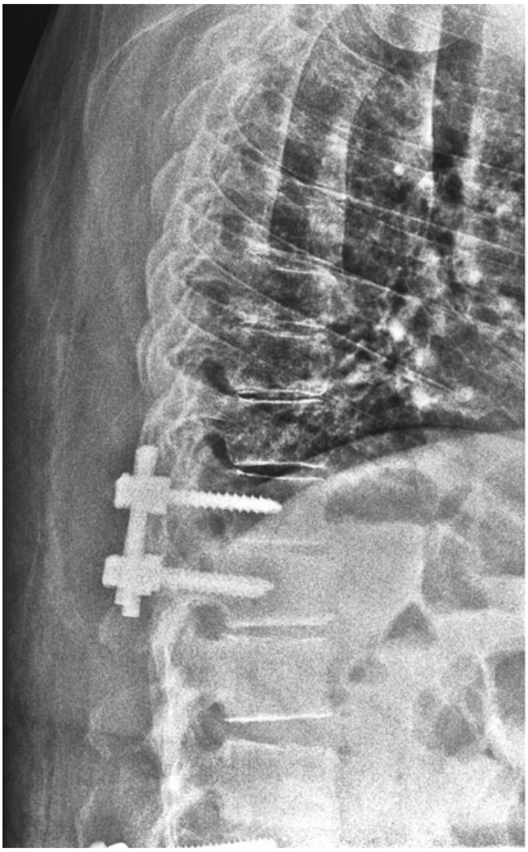
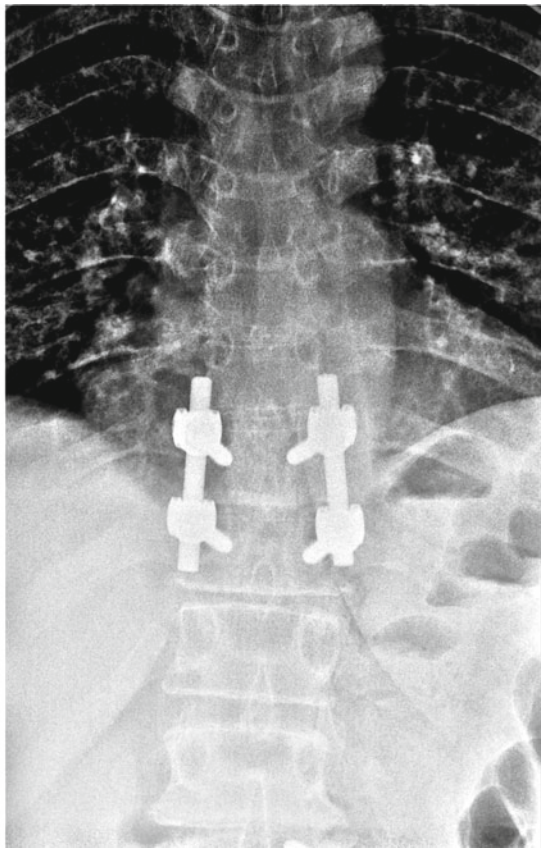


Fig. 14.1 MRI with large thoracic disc herniation, T10–11

M. Fernandez (✉)
Orthopedic Institute of Pennsylvania,
3399 Trindle Road, Camp Hill, PA 17011, USA
e-mail: mfernan@msn.com

S.N. Gidvani, M.D.
Department of Orthopaedic Surgery, Penn State Hershey Medical Center, Bone and Joint Institute,
30 Hope Drive, Building A, Hershey, PA 17033, USA
e-mail: sandeep.gidvani@gmail.com

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_14, © Springer-Verlag Berlin Heidelberg 2014

Figs. 14.2 and 14.3 Postoperative x-rays of T10–11 transfacet decompression and fusion

near-complete resolution of her symptoms and was discharged on postoperative day 3. She has maintained her normal neurologic function for approximately 1 year and has recently undergone a successful total knee arthroplasty.

14.2 Pathology (Basics of Pathology and Pathophysiology)

Key reported the first description of injury to the spinal cord by a thoracic disc herniation in 1838 [14]. Middleton and Teacher identified a TDH lesion on a postmortem exam in 1911 [17]. TDHs occur with a slight male predominance, with most symptomatic cases occurring in the fourth to sixth decade and women presenting later than men [6].

Thoracic disc herniations are classified based on their location and imaging characteristics. Herniations that are midline are classified as central, while those medial to the lateral edge of the dura are centrolateral. Those lateral to the lateral most aspect of the dura are lateral. The majority of TDHs are central or centrolateral. Herniations may be either soft or calcified at the time of presentation, with approximately 30–70 % of herniations showing some form of calcification. Almost 5–10 % of the calcified discs possess intradural extension [20].

Thoracic disc herniations have been found in up to 37 % of asymptomatic patients on MRI [22]. Prior to MRI, CT myelography revealed 11 % of asymptomatic patients possess a TDH [1]. Wood in 1997 and Awwad in 1991 looked at the natural history of asymptomatic TDH and found that small changes may occur in the size of the herniation over time, but for the most part, they remain asymptomatic. Furthermore, no correlation was found between the radiologic characteristics of these lesions and the subsequent emergence of symptomatic disease [23]. Based on these findings, it is important to identify the appropriate indications for surgical intervention.

The natural history of those who are symptomatic has also been studied. In general, these patients are divided between two groups. The first consists of younger patients with acute onset of symptoms, usually related to trauma, which have an acute soft disc herniation. These patients respond well to both nonoperative and operative treatment. The second group is made up of older patients who present with a longer duration of symptoms, degenerated disc pathology, and are likely to have evidence of calcification on advanced imaging.

Although trauma, in some studies, has been found to be a predisposing factor for TDHs, the pathophysiology behind the majority of herniations is felt to be degenerative. Similar to the processes of internal disc derangement that can occur within the discs of the cervical and lumbar spine, a spectrum of pathology exists in the thoracic spine that may lead to an eventual herniation of nucleus pulposus through the annulus fibrosus.

The thoracic spine is a rigid transition zone between the mobile cervical and lumbar regions. Its stability is supplemented by the surrounding thoracic rib cage. There are two facets from each rib head on either side that articulate with vertebral bodies from T2 to T10. In addition, the facets of T1 through T10 are oriented in a shingled fashion, thereby providing further stability during flexion and extension. Most (75 %) symptomatic TDHs involve the lower levels (T8–12) with the highest propensity for occurrence at T11–12. In contrast to the upper thoracic spine, the 11th and 12th ribs are not joined to the sternum and also do not form a true articulation with the transverse process of their own vertebra. Additionally, the facets at these levels transition to a more medial angulation in the coronal plane, allowing for greater movement in flexion and extension and are less effective in resisting of rotational forces. Thus, the lower thoracic spine is more susceptible to normal biomechanical loads and has a higher incidence of degeneration and associated disc herniation. The neurologic dysfunction that can result from the presence of a disc herniation within the thoracic spinal canal is attributed to its compression of the spinal cord, exiting nerve roots, and vascular supply.

Compression of the spinal cord from large central herniations can cause thoracic myelopathy. Dentate ligaments, which run longitudinally between the spinal cord and nerve roots, limit posterior displacement of the cord within the canal, thereby increasing the risk of ventral cord compression from a protruding or herniated disc. In addition, the natural kyphosis of the thoracic spine causes the cord to be draped over the vertebral bodies and the intervertebral discs. Exaggerated kyphosis, as is this case in Scheuermann's disease, has been suggested to be a risk factor for a TDH. In 1995, Wood et al. found that 38 % of patients with an asymptomatic thoracic disc herniation on MRI had Scheuermann-type dystrophic changes of the end plate or frank Scheuermann's disease [4, 21, 22].

Radiculopathy can occur secondary to direct compression of exiting nerve roots by paracentral herniations or via traction on nerve roots as the spinal cord is displaced dorsally by midline herniations.

The thoracic cord has a more tenuous blood supply than other regions of the spine. Vessels that predominately supply this section of the cord are of smaller caliber and are fewer in number than the cervical and lumbar regions. There is limited collateralization present for the single dominant artery of Adamkiewicz, which in turn creates a watershed region for the cord between T4 and T9. Disc herniations that directly compress the cord may exhaust the vasculature's ability to compensate and therefore cause ischemic injury. Neurologically, this may be demonstrated as myelopathy or spinal cord injury syndromes (e.g., Brown-Sequard). Furthermore, paracentral or lateral disc herniations may directly compress the segmental artery between T7 and L4 from which the artery of Adamkiewicz originates and thus create a similar scenario of ischemia.

14.3 History and Physical Exam Findings

Patients may give a history of a true trauma (e.g., fall or MVA) or may relate a subtle history of trauma, such as an acute increase in intrathoracic pressure (e.g., Valsalva maneuver during a sneeze) followed by thoracic back pain. Sometimes, patients will give a history of prior cervical or lumbar disc herniations. Most commonly, however, patients give no real history of trauma. The usual delay between the onset of symptoms and actually presentation to a physician's office lends further support to the notion that the primary etiologic factor is a degenerative process.

Clinical presentation is highly variable and is dependent on multiple factors. These include the location of the TDH (e.g., central, centrolateral, lateral), the size of the herniation, the duration of compression, the degree of vascular compromise, the size of the bony spinal canal, and overall health of the spinal cord and patient. Patients that have become symptomatic from their TDH usually present with one of three complaints: axial back pain, radicular pain, or myelopathy.

Pain is the most common presenting symptom in up to 76 % of patients and may be localized to the middle or lower thoracic spine and can radiate to the lower lumbar spine. Radicular pain, when present, may involve the anterior chest wall in a band-like dermatomal distribution or may radiate to the groin, abdomen, or lower limb. Paresthesias or dysesthesias may accompany the pain, in up to 61 % of patients. Myelopathy, the most severe of the three presentations, can include muscle weakness and paraparesis than can progress to a severe state of complete paraplegia. Stillerman and Chen found that if there was motor impairment at the initial presentation, it was three times more likely to be a paraparesis than a monoparesis [20].

Physical exam findings may also include hyperreflexia, positive Babinski, sustained clonus, wide-based gait, positive Romberg, and/or spasticity. Other upper motor neuron exam findings can include impaired superficial abdominal reflexes and superficial cremasteric reflex. Bowel and bladder dysfunction can be seen, ranging from dysfunctional urination to frank incontinence. Variations in sensation may range from dermatomal paresthesias to complete sensory loss.

14.4 Differential Diagnosis

Because of the variety of patterns in which a patient with a symptomatic TDH may present, the differential diagnosis is quite large. Differential diagnoses can be classified as nonspinal and spinal. Nonspinal etiologies include cardiovascular (i.e., angina, myocardial infarction), pulmonary (e.g., pleuritis), mediastinal (e.g., tumor), hepatobiliary (e.g., cholecystitis), gastrointestinal (e.g., reflux), and retroperitoneal (e.g., kidney stones). Nonspinal diagnoses may also include post-thoracotomy syndrome, polymyalgia rheumatica, fibromyalgia, herpes zoster, rib fractures, and intercostal neuralgia. Differential diagnoses related to the spine include infectious processes (e.g., osteomyelitis, discitis, epidural abscesses), neoplasms (e.g., primary, metastases, neural element origin), trauma (e.g., fracture), degenerative disc disease, spinal stenosis, spondylosis, facet syndrome, osteoporosis, osteomalacia, kyphosis, scoliosis, arteriovenous malformation, ankylosing spondylitis, amyotrophic lateral sclerosis, multiple sclerosis, and demyelinating diseases.

14.5 Investigations

All thoracic disease imaging examination begins with AP and lateral x-rays. These films provide insight into the overall alignment of the thoracic spine and may display any obvious fractures or neoplastic processes. Degenerative changes are identifiable as well, including disc space narrowing, osteophytes, and facet arthrosis. Calcification of the disc is visible in approximately 45–71 % of symptomatic discs versus only 4–6 % of the time in asymptomatic discs on plain radiographs [23]. X-rays are also important to obtain for rib-counting purposes, and comparison in the operating room should surgery be considered.

The imaging modality of choice, however, in those patients that thoracic disc disease and/or herniation is suspected is an MRI. This study is noninvasive, does not expose patients to ionizing radiation, and highlights degenerative disc changes, herniations, and neural element compression with significant detail in both the sagittal and axial planes. Location of the herniation within the canal is easily determined. As mentioned previously, MRI is very sensitive and not necessarily specific in detecting TDHs. Calcification of the disc, if present, is difficult to visualize on MRI, and if surgical intervention is planned, a CT scan should be ordered.

Prior to the advent of MRI, CT myelography was necessary to visualize thoracic disc pathology. In addition to a significant level of radiation exposure to the patient, the procedure is invasive, as it requires injection of an intrathecal contrast agent. It is, however, both sensitive and specific and can be used in patients in whom an MRI is contraindicated (e.g., pacemaker).

14.6 Nonsurgical Treatments

Patients who are not experiencing significant neurologic dysfunction secondary to thoracic disc herniation may be managed nonoperatively. Initial treatment for those with axial back pain may include a brief period of bed rest, activity modification, and the use of over-the-counter or prescription nonsteroidal anti-inflammatory medication. Similar modalities may be used for those with radicular pain. If these regimens fail, oral corticosteroid tapers may also be used. Occasionally, neurologic medications such as gabapentin (Neurontin) or pregabalin (Lyrica) may be beneficial.

Corticosteroid nerve root or intercostal nerve injections may be attempted with hopes of decreasing radicular pain and may also serve a diagnostic purpose. Bracing can be implemented during the acute phase of disc herniation in some patients, as well. These external supports create hyperextension, which may increase canal diameter and therefore relieve the neural elements from compression. Physical therapy may also be of benefit, focusing initially on passive modalities and then transitioning to range of motion, core strengthening, and hyperextension exercises. Per Brown et al., 63 % of symptomatic thoracic disc herniations were successfully treated with nonoperative modalities, and 77 % of patients returned to their previous activity level [6]. Progressive neurological deterioration or myelopathy in the setting of a corresponding thoracic disc herniation on imaging, however, is an indication to abandon conservative management in favor of surgical intervention.

14.7 Surgical Treatments

Candidates for surgery include those patients with myelopathy on presentation; progressive neurologic deterioration; severe, intractable radicular pain; and radicular pain that has not improved after a comprehensive course of conservative treatment.

Several factors must be taken into consideration when selecting the surgical approach to utilize. These variables are divided into patient characteristics, pathology of the disc herniation, and surgeon experience. First, the patient's body habitus, general health, and comorbidities are important to consider when deciding if a patient could medically undergo a large, anterior procedure. Typically, these procedures require single-lung ventilation intraoperatively, result in significant blood loss, necessitate a postoperative chest tube, and usually require a prolonged length of stay. Next, the qualities of the disc herniation itself must be scrutinized. The location, size, consistency, calcification, and possible intradural extension all play a crucial role in deciding which approach to utilize. Finally, from a surgeon experience viewpoint, there is a steep learning curve for certain approaches, especially the less invasive procedures. This can have a profound effect on multiple factors, including the length of the procedure, surgical complications, and postoperative recovery, among others.

There are multiple surgical approaches for a symptomatic thoracic disc herniation. General categories include anterior (transpleural, extrapleural, transsternal, transthoracic), posterior (laminectomy), posterolateral (transpedicular, costotransversectomy, transfacet), lateral (extracavitary), and minimally invasive techniques or video-assisted thoracoscopic surgery (VATS).

Initial attempts at decompression were done via a posterior-based laminectomy. Logue reported in 1952, however, severe complications with this technique, including worsening of neurologic deficits that were seen in the majority of patients. Both mechanical injury and vascular insult to the spinal cord may occur during or after surgery [15]. Kyphosis progression can occur following a laminectomy and may further tether the spinal cord over incompletely removed disc or osteophytes, thereby hastening a neurologic deficit. Given the success with an anterolateral approach to the thoracic spine in treatment of Pott's disease, Hulme et al. began using and publishing successful decompression via this method [13].

The transthoracic, anterior approach is the most versatile approach, allowing for central exposure and decompression from T4 to T12. For mid and lower thoracic levels, a left-side approach is taken to avoid the liver and vena cava, whereas for higher levels, the right side is taken to avoid the heart and subclavian vessels. The patient is placed in a lateral decubitus position. The key to this exposure is following the rib or intercostal neurovascular bundle to the pathologic level. For example, if a T8–9 disc herniation is being targeted, follow the ninth rib to its attachment at the T9 vertebral body. The base of the rib is then excised subperiosteally. The parietal pleura is then reflected, segmental vessels are coagulated, and further subperiosteal dissection is performed to expose the vertebral bodies cephalad and caudal to the disc space. The pedicle of the caudad vertebral body is identified. A Penfield elevator or dissecting tool is used to identify the ventral most aspect of the dura and tease it away from the herniated disc [4].

An extension of this technique may include removal of the posterolateral aspect of the cephalad and caudad vertebral bodies adjacent to the disc herniation. This allows for creation of a trough into which the remainder of the disc material will be delivered and then removed, thereby moving disc material away from the dura and avoiding manipulation of the cord. If indicated and necessary, an arthrodesis is performed [4]. The primary advantage of this approach is the ability to visualize the ventral and lateral aspects of the dura. With this approach, there is generally a lower risk of retained disc fragments and overall, a lower risk of neurologic complications. Disadvantages include the need to violate the chest wall and pleural cavity, the need for extensive soft tissue dissection with retraction/resection of ribs, the potential for development of post-thoracotomy syndrome, and risk of intercostal neuralgia.

The posterolateral (transpedicular) approach is performed via midline or paramedian incision to expose the facet joint posterior to the disc and the pedicle caudal to the disc herniation. The central cancellous bone of the pedicle is then resected to the posterior cortical bone of the vertebral body. Lateral and inferior cortices of the pedicle are left intact. Partial superior and inferior facetectomies are performed, and partial to complete laminectomies may also be required. This allows for entrance to the disc space lateral to the dura and distal to the exiting nerve root. Once the disc herniation is encountered, disc material can then be pushed into the adjacent bony trough created by the removal of the pedicle. Theoretically, simple removal of the disc above T10 does not necessitate fusion because of the stabilizing rib cage [2, 3].

A variation of this technique includes a mini-open technique, which allows for minimal soft tissue disruption. Tubular retractors and dilators are now widely available for this purpose. Chi et al. have reported less blood loss, better early postoperative outcome scores, and equivalent 18-month follow-up outcomes when compared to open procedures [9]. Another variation is to place pedicle screws above and below the TDH, perform a laminectomy from pedicle to pedicle, and then expose the exiting nerve root and the disc space. A lamina spreader may then be placed between the pedicle screws in order to distract the disc space and gain access to the lateral annulus. This technique is referred to as a modified transfacet pedicle-sparing decompression [5].

Posterolateral approaches are ideal mainly for soft disc herniations situated either centrolateral or lateral. Advantages of these approaches include surgeon familiarity with anatomy, limited soft tissue dissection, and preservation of important structural elements (e.g., vertebral body or facets, in some cases). Disadvantages may include difficulty visualizing the herniated disc and subsequently performing a less than complete disc excision.

The costotransversectomy approach offers improved visualization over the posterolateral approaches described above. This approach necessitates the removal of the medial head of the rib and the transverse process of the vertebral body caudal to the TDH. Again, the disc space is entered laterally, and the disc material is removed in a similar manner to a transpedicular approach. Advantages of this method include improved visualization of the ventral and lateral dura and perhaps performance of a more complete discectomy. It can be utilized for paracentral and lateral herniations, soft discs, and for cases in which a transthoracic approach is contraindicated. However, caution should be exercised when targeting calcified central discs using this approach. Disadvantages include the need for lateral dissection away from the midline, removal of stabilizing structures, and potential for pleural violation.

If greater exposure and greater lateral angle of view are required, the extracavitary approach can be utilized. This involves resection of the facet joint, the pedicle of the caudal level, the medial rib head, and the costotransverse articulation. The goal is to stay extrapleural and allow for greater access to the lateral and ventral aspects of the dura. The advantages of this approach are similar to that of the transthoracic approach and include excellent visualization. Disadvantages include extensive bony resection and soft tissue dissection.

Video-assisted thoracoscopic surgery (VATS) has been explored as a minimally invasive alternative to previously described posterolateral and transthoracic approaches. The patient is placed in the lateral decubitus position, with ventilation accomplished via a double lumen endotracheal tube, allowing collapse of the ipsilateral lung. Three endoscopic ports are placed. Once the appropriate level has been confirmed by fluoroscopy, the technique applied is similar to that of a transthoracic approach. Proponents of this approach have cited decreased operative time, less blood loss, lower duration of chest tube use, less use of narcotic pain medication, and shorter length of hospitalization. In addition, a decreased incidence of intercostal neuralgia is noted, as is improved postoperative pulmonary function, when compared to a transthoracic approach. In the upper thoracic spine, shoulder girdle dysfunction is also lessened when compared to open procedures [7, 18, 19].

In a report by Cornips et al., several patients who developed acute myelopathy secondary to a TDH were treated at a referral center between 1 and 9 days after onset of neurologic dysfunction. All patients, even those with a profound deficit, had a remarkable recovery. Therefore, myelopathy secondary to TDH, despite its insidious course in some cases and resultant delays in presentation or referral, should be regarded as a surgically treatable disease [10].

14.8 Postoperative Care

The extent of postoperative care is partly dependent on which approach was utilized. For posterolateral approaches that do not violate the pleural cavity, patients may ambulate out of bed shortly after surgery. In contrast, anterior approaches require the insertion of a chest tube and extensive pulmonary therapy. Similar to lumbar discectomy, the basic postoperative goals include safe ambulation and sufficient oral pain control prior to discharge to a home setting. With significant neurological compromise, placement in acute or subacute rehab may be necessary.

14.9 Potential Complications

Excluding the poor results of laminectomy alone, the overall rates of morbidity and mortality are similar for all the approaches previously mentioned [8, 16]. There are several potential complications encountered with the surgical treatment of TDHs.

A dural tear resulting in a cerebrospinal fluid leak has a reported incidence of 0–15 %. In rare instances, a durotomy is necessary to completely decompress the neural elements in the setting of an intradural TDH. In other cases, the herniated disc may be adherent to the dura and incidental durotomy may occur. Regardless, typical management of the durotomy generally ensues with primary repair, if possible, and the use of a supplemental graft or synthetic material and/or possible fibrin glue application [16, 20].

Postoperatively, some patients may have either worsening or retained neurologic deficit, and one must ensure that a retained disc or disc fragment is not the cause [11]. Neurologic deterioration is rare with all modern approaches. A thorough neurologic exam should be performed pre- and postoperatively. If necessary, advanced imaging may be necessary.

Postoperative instability may manifest as worsening kyphosis, compression fracture, or worsening neurologic status. Kyphosis and compression fractures may be treated with bracing, if appropriate. Repeat surgery may be necessary if bracing fails and/or a worsening neurologic picture develops.

Several methods to counteract wrong-level surgery have been described. These include meticulous counting of ribs on pre- and intra-operative imaging and the use of preoperative localization with interventional radiology methods [11].

Pulmonary complications may occur. This potential increases in those with preexisting pulmonary system comorbidities or decreased physiologic reserve and/or in those patients undergoing a transthoracic approach. Postoperative respiratory therapy is employed in all patients.

Infection reports range for 0–18 %. The severity ranges from superficial wound infection to osteomyelitis/discitis with secondary vertebral body collapse to sepsis. Preoperative antibiotic administration within 1 h of incision and postoperative prophylactic antibiotics for 24 h helps reduce this risk [16].

A specific complication seen with the transthoracic approach is intercostal neuralgia. This can occur with rib resection or rib retraction secondary to either direct mechanical compression from retractors or from stripping of the neurovascular supply to the intercostal nerve during dissection. Ghanayem and Bohlman have discouraged the use of nerve blocks to control intercostal postoperative pain, following their report of two patients who experienced transient paraplegia with this technique. Preoperative counseling regarding this potential complication is important [12].

Questions (Typical In-Training Exam-Type Questions)

1. A 55-year-old woman presents to the emergency room with insidious onset of mid-level back pain and no history of apparent trauma. She has a history of lumbar disc herniation treated with discectomy in the past. She notes a progressive weakness of her lower extremities and over the next few hours after presentation becomes paraplegic. XR and MRI are ordered which show a central, midline disc herniation. What is the next step in management?
   (a) Laminectomy for decompression
   (b) Transthoracic decompression and interbody fusion
   (c) Transthoracic decompression
   (d) CT scan
   (e) Bone scan

   Preferred Response (d): This patient's presentation and progressive neurologic deficit warrant significant concern. Appropriate imaging studies reveal a central, midline thoracic disc herniation. An important step in preoperative planning for patients who require decompression is to obtain a CT scan. This study will delineate the level of calcification that may exist within the herniated disc, if any, and highlight the bony anatomy that may need removal during decompression.
2. Which of the following surgical approaches to thoracic disc herniation is associated with significantly worse outcomes and has been abandoned for the most part?
   (a) Transthoracic
   (b) Posterolateral
   (c) Minimally invasive
   (d) Posterior laminectomy
   (e) Lateral extracavitary

   Preferred Response (d): Initial attempts at decompression were done by a posterior-based laminectomy. Logue reported in 1952, however, severe complications with this technique, including worsening of neurologic deficits, in the majority of patients. Both mechanical injury and vascular insult to the spinal cord may occur [15]. Others have shown that even a minor kyphotic deformity progression can occur from laminectomy and may tether the cord over incompletely removed disc or osteophytes, thereby hastening a neurologic deficit.

3. Which of the following surgical approaches provides optimum visualization of the ventral aspect of the dura and should be utilized, in particular, for those patients with a symptomatic central, calcified thoracic disc herniation?
   (a) Transthoracic
   (b) Transpedicular
   (c) Costotransversectomy
   (d) Posterior
   (e) Lateral extracavitary

   Preferred Response (a): The transthoracic approach allows for resection of the caudad pedicle and then if warranted, cephalad and caudad vertebral bodies, thus affording maximum exposure of the ventral aspect of the dura.

References

1. Awwad EE, Martin DS (1991) Asymptomatic versus symptomatic herniated thoracic discs: their frequency and characteristics as detected by computed tomography after myelography. Neurosurgery 28:180–186
2. Bilsky MH (2000) Transpedicular approach for thoracic disc herniations. Neurosurg Focus 9(4):E3, 1–4
3. Black P (2000) Laminotomy/medial facet approach in the excision of thoracic disc herniation. Neurosurg Focus 9(4):E6, 1–6
4. Bohlman HH, Zdeblick TA (1988) Anterior excision of herniated thoracic discs. J Bone Joint Surg Am 70-A(7):1038–1047
5. Bransford R, Zhang F (2010) Early experience treating thoracic disc herniations using a modified transfacet pedicle-sparing decompression and fusion. J Neurosurg Spine 12:221–231
6. Brown CW, Deffer PA (1992) The natural history of thoracic disc herniation. Spine 17(6):S97–S102
7. Burke TG, Caputy AJ (2000) Treatment of thoracic disc herniation: evolution toward the minimally invasive thoracoscopic technique. Neurosurg Focus 9(4):E9, 1–7
8. Chen TC (2000) Surgical outcome for thoracic disc surgery in the postlaminectomy era. Neurosurg Focus 9(4):E12, 1–5
9. Chi JH, Dhall SS (2008) The Mini-Open transpedicular thoracic discectomy: surgical technique and assessment. Neurosurg Focus 25(2):E5, 1–5
10. Cornips EMJ, Janssen MLF (2011) Thoracic disc herniation and acute myelopathy: clinical presentation, neuroimaging findings, surgical considerations, and outcome. J Neurosurg Spine 14:520–528
11. Dickman CA, Rosenthal D (1999) Reoperation for herniated discs. J Neurosurg Spine 91:157–162
12. Ghayanem AJ, Bohlman HH (1994) Transient paraplegia from intraoperative intercostal nerve block after transthoracic discectomy. Spine 19:1294–1296
13. Hulme A (1960) The surgical approach to thoracic intervertebral disc protrusions. J Neurol Neurosurg Psychiatry 23:133–137
14. Key CA (1838) On paraplegia depending on disease of the ligaments of the spine. Guy's Hosp Rep 3:17–34
15. Logue V (1952) Thoracic intervertebral prolapse with spinal cord compression. J Neurol Neurosurg Psychiatry 15:227–241
16. McCormick WE, Will SF (2000) Surgery for thoracic disc disease. Complication avoidance: overview and management. Neurosurg Focus 9(4):13
17. Middleton GS, Teacher JH (1911) Injury of the spinal cord Due to rupture of an intervertebral disc during muscular effort. Glasgow Med J 76:1–6
18. Perez-Cruet MJ, Kim B (2004) Thoracic microendoscopic discectomy. J Neurosurg Spine 1(1):58–63
19. Rosenthal D, Dickman CA (1998) Thoracoscopic microsurgical excision of herniated thoracic discs. J Neurosurg 89:224–235
20. Stillerman CB, Chen TC (1998) Experience in the surgical management of 82 symptomatic herniated thoracic discs and review of the literature. J Neurosurg 88:623–633
21. Van Landingham JH (1954) Herniation of thoracic intervertebral discs with spinal cord compression in kyphosis dorsalis juvenilis (Scheuermann's disease). Case report. J Neurosurg 11:327–329
22. Wood KB, Garvey TA (1995) Magnetic resonance imaging of the thoracic spine. Evaluation of asymptomatic individuals. J Bone Joint Surg Am 77(11):1631–1638
23. Wood KB, Blair JM (1997) The natural history of asymptomatic thoracic disc herniations. Spine 22(5):525–529

Lumbar Disc Herniation

15

William Ryan Spiker and Brandon D. Lawrence

15.1 Introduction

Low back pain is the second most common presenting symptom of patients seeking medical care in the USA [1] affecting nearly 70 % of the population at some point in time with varying degrees of severity [2]. Due to the complexities inherent in the anatomy of the spine as well as the bio-psychosocial variables in patients afflicted with low back pain, there are numerous treatment modalities marketed toward the treatment of low back pain. The USA spends an estimated 100 billion dollars per year [3] attempting to treat this disorder.

When treating patients with low back pain, it is important to differentiate the nature of the pain as it may be complicated by radiculopathy, defined as pain radiating in the distribution of a single lumbar or sacral nerve root with or without motor or sensory changes. Lumbar disc herniation (LDH) is the most common cause of radiculopathy in adults with an estimated annual incidence ranging from 1.6 % in the general population to 43 % in certain occupations [4]. The symptoms of radiculopathy are due to the *direct mechanical compression* on a nerve root as well as the *inflammatory mediators* present when disc material enters the spinal canal.

The symptoms of radiculopathy that patients experience are variable. Depending on the degree of compression on the nerve root as well as the location of the compression, patients may experience a spectrum of symptoms including pain, numbness, tingling, and weakness. Rarely, cauda equina syndrome may develop secondary to a large disc herniation and is characterized by bowel and/or bladder dysfunction with associated saddle anesthesia, decreased rectal tone, and bilateral lower extremity symptoms. Cauda equina syndrome will be discussed in detail later in this chapter, but it is important to realize that this situation, unlike the other symptoms of LDH, requires emergent evaluation. Most symptoms of radiculopathy resolve with time and nonoperative modalities such as medications, activity modification, physical therapy, and injections. However, if nonoperative treatments are unsuccessful, surgical decompression may be recommended in patients with intractable pain, motor dysfunction, and/or sensory dysfunction.

The surgical techniques for decompressing the offending structure causing radicular symptoms have evolved quite dramatically over the last several decades, but the principles of surgery have been maintained which is to adequately decompress the neural elements. In approximately 90 % of cases, radiculopathy is secondary to a disc herniation, but other causes of nerve root compression such as adjacent level central and lateral recess stenosis, facet cysts, and less commonly tumors may be the underlying cause [5]. Therefore, surgical techniques for addressing radiculopathy secondary

W.R. Spiker, M.D. • B.D. Lawrence, M.D. (✉)
Department of Orthopaedics, University of Utah, 590 Wakara Way, Salt Lake City, UT 84108, USA
e-mail: brandon.lawrence@hsc.utah.edu

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_15, © Springer-Verlag Berlin Heidelberg 2014

to an LDH are referred to as a discectomy. Discectomy remains the most commonly performed lumbar spine procedure with over 250,000 operations performed annually in the USA [6]. Although discectomy can be an effective operation, it carries significant risk and requires careful patient selection and meticulous surgical technique.

In this chapter, we will review the anatomy, clinical presentation, natural history of lumbar spine disc herniations, as well as the role and outcomes of nonoperative and operative treatment modalities.

15.2 Anatomy and Pathology

The intervertebral disc is composed of an outer annulus fibrosis and an inner nucleus pulposus. Each component of the intervertebral disc has unique mechanical and structural properties. The annulus fibrosis consists of an inner and outer layer. The outer component is primarily tightly packed sheets of interlocked type I collagen and the inner layer has a higher proteoglycan content and is less organized. The annulus provides tensile strength to the disc and completely encapsulates the nucleus pulposus. The nucleus pulposus is more gelatinous, composed of type II collagen and proteoglycans that attract water molecules to provide hydrostatic resistance to compression. With aging, the disc dessicates, the compressive strength of the nucleus diminishes, and increased stress is seen by the annulus. This can lead to fissuring of the annulus and eventually frank disc herniation [7].

The intervertebral disc is relatively aneural and avascular. The blood supply to the vertebral body traverses along the periphery of the disc along the end plate, but does not enter the disc itself. The disc obtains nutrition and disposes of waste by diffusional and convective transport through its porous matrix. Similarly, the surface of the annulus fibrosis is innervated, but the central disc is devoid of neural structures [8].

When an LDH occurs, it is classified anatomically as central (Fig. 15.1), paracentral (Fig. 15.2), foraminal (Fig. 15.3), or far lateral (Fig. 15.4). Central disc herniations occur through the midline of the posterior annulus; due to their central location, they less commonly cause a radiculopathy. If central disc herniations are large, they may cause symptoms of neurogenic claudication (Fig. 15.5) or cauda equina syndrome (Fig. 15.6). Paracentral herniations are the most common anatomic variety and occur through the thinner, posterolateral border of the posterior longitudinal ligament. Due to their anatomic location, paracentral herniations more commonly impinge upon the traversing nerve root causing radiculopathy.

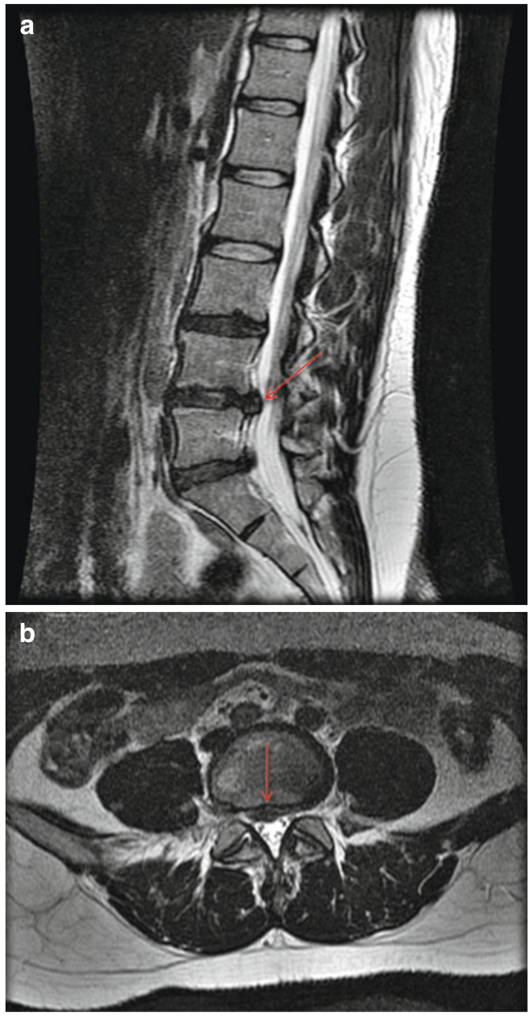


Fig. 15.1 (**a**) Sagittal T2-weighted MRI scan of a 24-year-old female presenting with low back pain exacerbated with flexion. *Red arrow* denotes a central disc herniation at the L4–5 level. (**b**) Axial T2-weighted MRI scan with *red arrow* denoting the L4–5 central disc herniation without significant displacement or compression on the nerves. Nonoperative care was elected

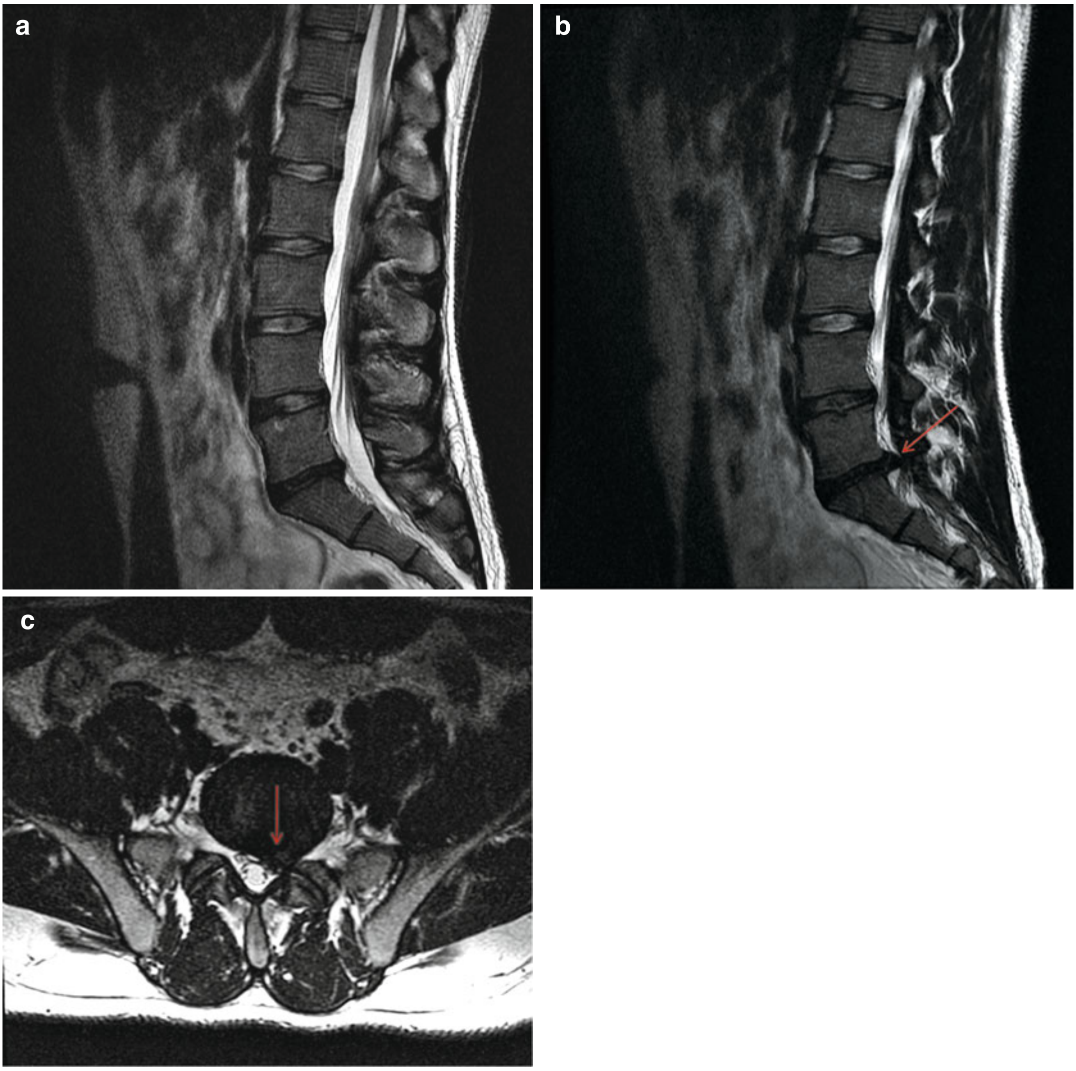


Fig. 15.2 (**a**) Midsagittal T2-weighted MRI scan of a 26-year-old male presenting with left S1 radiculopathy recalcitrant to nonoperative care. Note that there is no nerve compression apparent on this cut. (**b**, **c**) Left-sagittal T2-weighted MRI and axial T2-weighted MRI portraying large left paracentral disc herniation at L5–S1 compressing and displacing the traversing left S1 nerve root denoted by the *red arrow*. Patient underwent a left L5–S1 microdiscectomy

Herniations into the intervertebral foramen and far lateral herniations (lateral to the foramen) more commonly affect the exiting nerve root causing radiculopathy. Lumbar nerve roots exit below the pedicle of the corresponding vertebral body. Thus, a central or paracentral disc herniation at the L5/S1 level will likely compress the traversing S1 nerve root, and a foraminal or far lateral disc herniation will likely impinge upon the exiting L5 nerve root.

Disc herniations are descriptively classified as protrusions, extrusions, or sequestrations. A protrusion is a small defect in the annulus fibers with a portion of the nucleus pulposus displaced through the annular defect. A disc extrusion involves a larger herniation of nucleus material through the annular defect. In a protrusion, the diameter of the annular defect is larger than the diameter of the herniated material, while the diameter of the herniated material is larger than the defect in

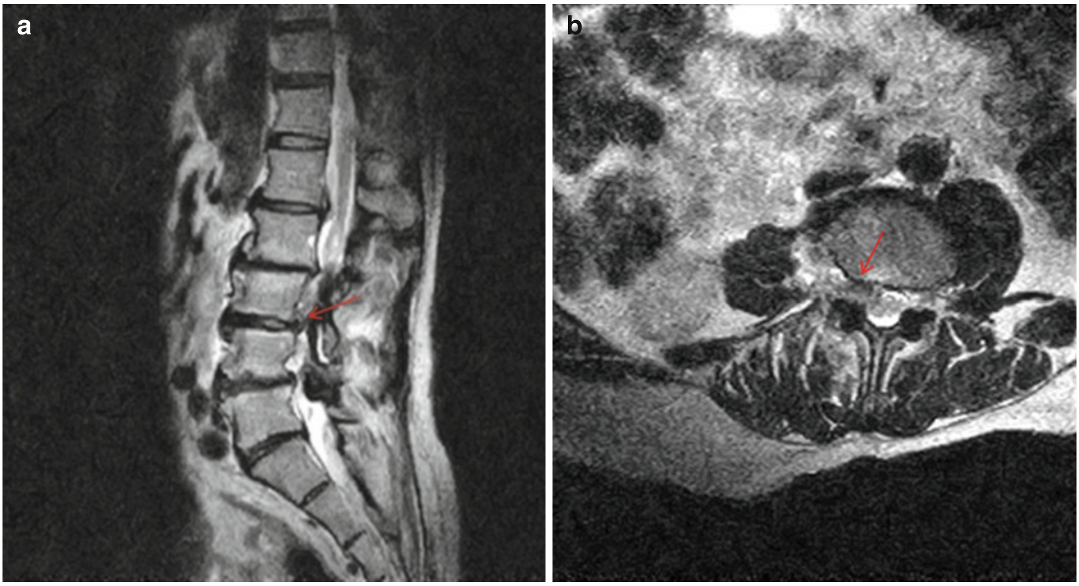


Fig. 15.3 (**a**) Right-sagittal T2-weighted MRI scan of a 73-year-old male presenting with right L3 radiculopathy with associated quadriceps weakness with 3-day relief after a L3–4 transforaminal steroid injection. *Red arrow* denotes right sided L3–4 foraminal disc herniation. (**b**) Axial T2-weighted MRI image portraying right L3–4 foraminal disc herniation compressing and displacing the exiting right L3 nerve root denoted by the *red arrow*. Patient underwent a successful right L3–4 microdiscectomy; note other areas of asymptomatic neural compression that were not addressed

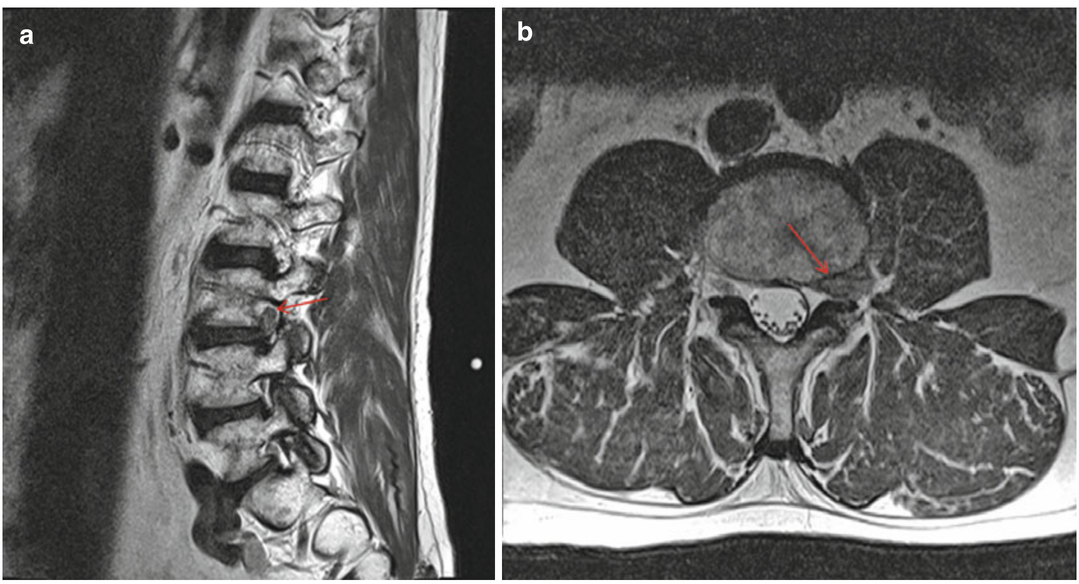


Fig. 15.4 (**a**) Left-sagittal T2-weighted MRI scan in a 57-year-old male portraying a far lateral disc herniation at L3–4 causing a left L3 radiculopathy denoted by the *red arrow*. (**b**) Axial T2-weighted MRI portraying compression and displacement of the exiting left L3 nerve root *red arrow*. Patient underwent a far lateral discectomy

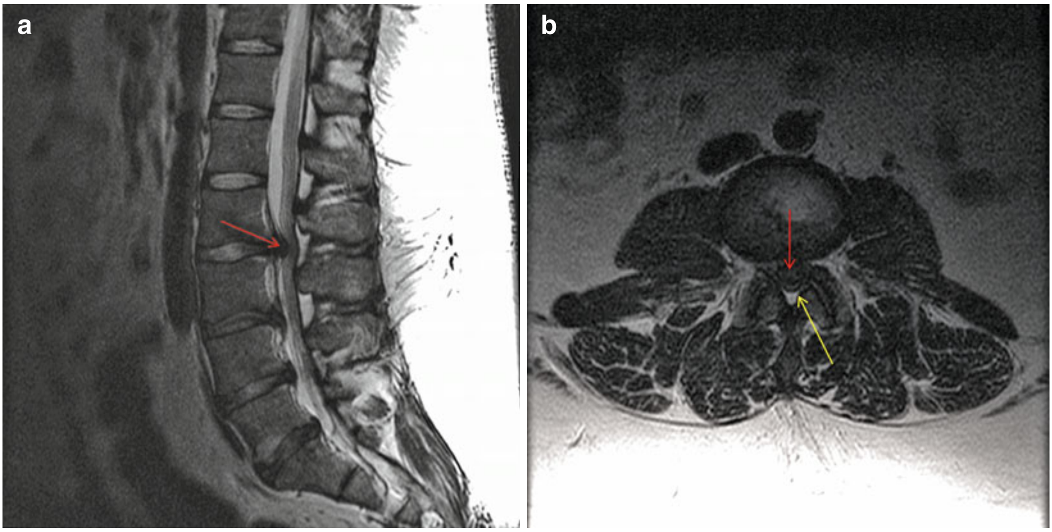


Fig. 15.5 (**a**) Midsagittal T2-weighted MRI in a 28-year-old male at L2–3 with the insidious onset of bilateral lower extremity weakness/numbness/tingling while erect consistent with neurogenic claudication. *Red arrow* denotes large central disc herniation displacing the thecal sac and compressing the rootlets. (**b**) Axial T2-weighted MRI portraying the large central disc herniation (*red arrow*) and the severe central stenosis it causes on the compressed thecal sac (*yellow arrow*). Patient underwent L2–3 laminectomy

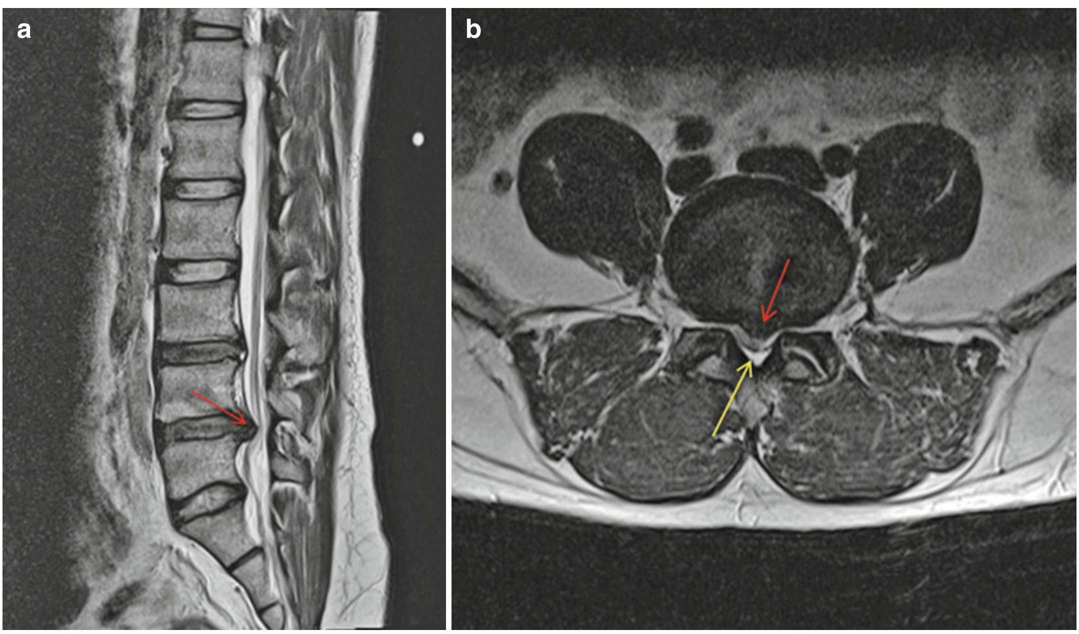


Fig. 15.6 (**a**) Midsagittal T2-weighted MRI in a 32-year-old male at L4–5 after he noted a pop in his back after lifting a 100-lb object while at work. Patient noted immediate pain with progressive bilateral lower extremity weakness, saddle anesthesia, and complete urinary retention over the ensuing 12 h. Patient presented to the emergency department and underwent an emergent L4–5 laminectomy. Disc herniation is denoted by *red arrow*. (**b**) Axial T2-weighted MRI showing severe central stenosis with large disc herniation (*red arrow*) compressing the thecal sac (*yellow arrow*) at L4–5

the annulus in a disc extrusion. A sequestered disc herniation is an unattached fragment of disc material that has been separated from the disc. Finally, disc herniation must be differentiated from a disc bulge, which is a diffuse, prominent bulge of annulus fibrosis into the spinal canal.

In 1–6 % of patients with LDH, the compression of the nerve roots within the spinal canal is severe enough to cause cauda equina syndrome [9, 10]. The pathophysiology of cauda equina syndrome is unknown but likely related to mechanical compression as well as venous congestion and ischemia of the rootlets caudad to the herniation. This syndrome is defined by the presence of bowel or bladder dysfunction, saddle anesthesia, and varying degrees of decreased motor and sensory function (decreased rectal tone is usually a late finding) [11]. Cauda equina is a clinical diagnosis usually made by history and physical exam accompanied by magnetic resonance imaging (MRI) to identify the anatomic location of the space-occupying lesion. Treatment is emergent surgical decompression of the spinal canal.

15.3 Clinical Presentation, Physical Exam, and Imaging

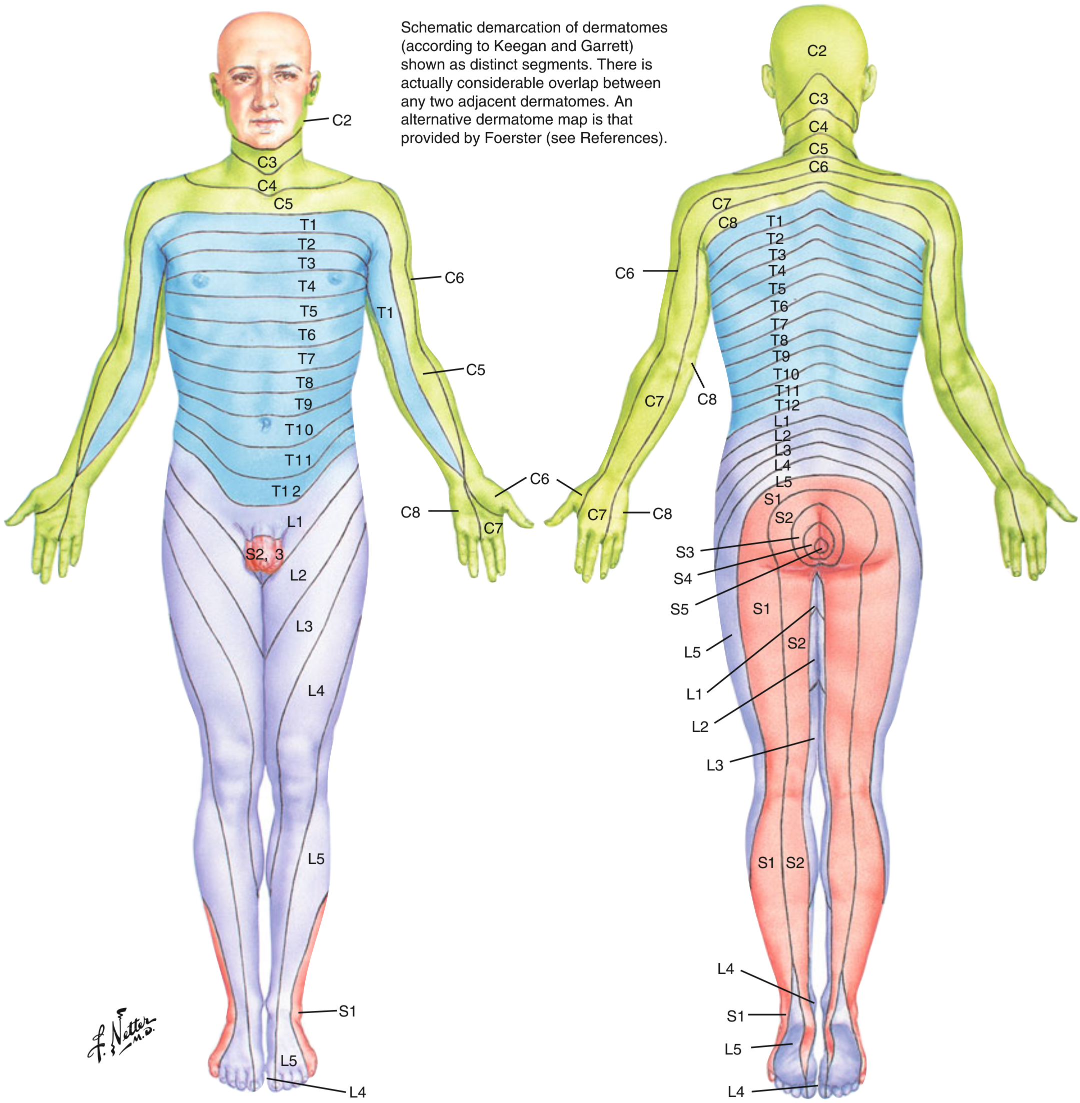
Evaluation of a patient with a suspected lumbar disc herniation begins with a thorough history and physical exam. The primary complaint of most patients with lumbar disc herniations is radicular pain in the lower extremity with or without concomitant back pain. The pain from an LDH should follow the specific dermatome of the involved nerve root (Fig. 15.7). It is important to ascertain the intensity and quality of the pain and the progression of the patient's symptoms over time. Any history of weakness or numbness in the lower extremities may indicate nerve irritation or injury. Physical examination should consist of a thorough neurologic exam to look for sensory deficits, motor weakness, or abnormal reflexes (patellar, achilles, clonus, and babinsky). Any motor weakness or altered reflexes should occur in the corresponding nerve distribution as well. Tension signs should also be attempted to be elicited including the femoral stretch test or the straight leg raise. If patient's symptoms do not correlate with the suspected nerve root and do not follow the anatomic dermatomal pattern, then suspicion should be raised prior to embarking upon surgical decompression.

The history of bowel or bladder dysfunction and/or bilateral lower extremity symptoms suggests a possible cauda equina syndrome and warrants emergent evaluation. Peri-anal sensation and a rectal exam are also indicated in the patient with possible cauda equina syndrome.

Prior to obtaining any imaging studies, it is important that a thorough history and physical exam be obtained. Depending on the level of suspicion and magnitude of presenting signs/symptoms, advanced imaging may be necessary. In the absence of any red flags (tumor, trauma, infection, and/or neurologic injury) (Table 15.1), the acquisition of upright plain radiographs, including dynamic flexion/extension views, is recommended after a course of nonoperative therapy has been attempted, typically for 6 weeks. If red flags are present, advanced imaging (computed tomography and/or magnetic resonance imaging) should proceed without delay.

Plain radiography may reveal evidence of spondylotic changes, demonstrate spinal instability including disc space collapse, facet arthropathy, and vertebral endplate sclerosis. Plain radiography also has the advantage of quickly and easily obtaining standing flexion and extension images that may demonstrate spinal instability not seen in supine MRI and CT imaging. The presence of congenital stenosis, dynamic instability (spondylolisthesis), trauma, tumor, or infection on plain radiographs usually warrants acquisition of advanced imaging.

Magnetic resonance imaging (MRI) remains the gold standard in evaluating the soft tissues of the lumbar spine including the discs, ligaments, and neural elements. Due to the sensitivity of MRI in visualizing abnormalities of the soft tissues, it is important to correlate MRI findings with the patients signs/symptoms, as mentioned previously. MRI will allow excellent visualization and localization of a disc herniation as well as rule out any other pathology.



Levels of principal dermatomes

C5	Clavicles
C5, 6, 7	Lateral parts of upper limbs
C8, T1	Medial sides of upper limbs
C6	Thumb
C6, 7, 8	Hand
C8	Ring and little fingers
T4	Level of nipples
T10	Level of umbilicus
L1	Inguinal or groin regions
L1, 2, 3, 4	Anterior and inner surfaces of lower limbs
L4, 5, S1	Foot
L4	Medial side of great toe
S1, 2, L5	Posterior and outer surfaces of lower limbs
S1	Lateral margin of foot and little toe
S2, 3, 4	Perineum

Fig. 15.7 Diagram of the dermatomal distribution of the sensory innervation of the upper and low limbs. Reproduced from "Aids to the examination of the peripheral nervous system"

When evaluating patients with radiculopathy, computed tomography (CT) scanning with myelography still plays a vital role especially if contraindications to performing an MRI scan exist (pacemaker, foreign bodies, etc.). Even though CT myelography is an invasive procedure, it can delineate osseous sources of compression from soft tissue sources that may not be well visualized on MRI scanning, as seen in the setting of foraminal stenosis.

Table 15.1 Red flags

• Recent trauma
• Night pain
• Systemic signs (fever, night sweats)
• Progressive worsening of back pain
• Cord level symptoms
• Neurologic deficit

15.4 Etiology/Natural History

Although deformity of the spinal column was first associated with sciatic pain over 230 years ago [12], our understanding of the etiology and natural history of lumbar disc herniations remains incomplete. Advanced age and male gender are risk factors for disc herniations [13] while smoking and mechanical stress also contribute to a smaller degree to disc degeneration [13, 14]. The dominant risk factor for disc disease has been shown to stem from an inherited element [15, 16]. However, the underlying genetic cause of this heritable risk remains unknown. Variations in the collagens and other extracellular matrix components likely play a role, but we are only now beginning to scratch the surface of this complex genetic process.

The natural history of lumbar disc herniations is also largely unknown. True natural history studies simply do not exist, as contemporary studies treat nonoperative patients with variable physiotherapy protocols. Research has revealed that disc herniations most commonly occur in the fourth and fifth decades of life, are more common in men, and are clinically silent in approximately 95 % of patients [17]. The lifetime risk of surgical intervention for a disc herniation is estimated between 1 and 3 %. Clinical practice has vacillated over the years between nonoperative modalities and surgical treatment as new data have emerged. Several excellent prospective, randomized studies currently guide treatment of disc herniations and are discussed in detail.

15.5 Nonoperative Treatment Strategies

The nonoperative care of lumbar disc herniations commonly includes bed rest, pharmacotherapy, physical therapy, activity modification, and epidural/transforaminal steroid injections. Recommended bed rest has never been shown to impact functional outcomes, but a randomized trial by Deyo et al. reported that recommending 2 days of bed rest rather than 7 days did decrease the number of days of work missed without impacting any other outcome measure [18]. The most common medications used in the treatment of LDH are nonsteroidal anti-inflammatory drugs (NSAIDs), oral steroids, and muscle relaxants. NSAIDs have been extensively studied for low back pain (LBP), and a Cochrane review of 51 clinical trials concluded that they were beneficial in the treatment of acute LBP. This is logical especially considering a significant proportion of the pain may be caused by inflammation around the nerve. There was no evidence that NSAIDs are more effective than muscle relaxants or steroids, and no specific NSAID was proven superior to another [19].

Another important modality commonly prescribed for patients with LBP/LDH is physical therapy. Unfortunately, physical therapy is inherently difficult to study due to the variation in methodologies performed, timing of treatments, and many other variables. However, in general, it has been shown to reduce self-reported pain scores, decrease time away from work, and result in less disability [20].

When the more conservative approaches to nonoperative treatments fail, many patients with radiculopathy are considered for either epidural and/or transforaminal injections. The goal of injections is twofold: (1) provide therapeutic relief to the patient and (2) provide diagnostic information to the treating physician. The mode of action of epidural and transforaminal injections is to reduce inflammation around the herniated disc material and involved nerve root. Both of these procedures have gained popularity in recent years. Flouroscopically guided transforaminal steroid injections are indicated in patients presenting with predominantly unilateral leg symptoms [21]. In a prospective randomized trial of patients with a symptomatic LDH, patients received either an epidural steroid injection or surgery after a 6-week trial of conservative care. Even though they reported positive results in 42–56 % of patients who received an epidural injection with relief that ranged from weeks to years, these results were overshadowed by those who underwent surgical decompression. In this study, they reported faster resolution of symptoms and a 92–98 % satisfaction rate [22]. With that said, epidural and transforaminal steroid injections play a vital role in the treatment of patients with LDHs and carry a lower risk profile than surgical techniques.

15.6 Operative Treatment Strategies

15.6.1 Patient Selection

Patient selection is integral to obtaining clinical improvement from surgical treatment of lumbar disc herniation. Surgical candidates should have clinical findings consistent with lumbar radiculopathy for greater than 6 weeks despite nonoperative interventions and MRI-documented evidence of a disc herniation at the corresponding level as mentioned previously. Other causes of the patient's symptoms such as abscess, tumor, hematoma, and spinal stenosis must be ruled out by examination and imaging studies. Patients with cauda equina syndrome require emergent surgical decompression to attempt to prevent permanent bladder and bowel dysfunction. Similarly, patients with decreased strength in the appropriate nerve distribution or with a progressive neurologic deficit are indicated for urgent decompression to maximize postoperative neurologic function. However, in the absence of neurologic deficits, conservative treatment should be attempted for a period of 6 weeks to 3 months.

15.6.2 Treatment Outcomes

The surgical treatments for LDH have historically had excellent results when performed on patients meeting the above criteria. Despite the excellent reported results, these Level 3 and higher studies reported small sample sizes, study design limitations, and failure to plan for high crossover rates that limited the strength of these studies. Due to that, there have been several Level-1 studies performed comparing the effectiveness of discectomy versus nonoperative care Table 15.2.

The first such study was performed by Weber [23] and was a prospective randomized controlled trial. In this study, 126 patients with persistent radiculopathy and evidence of LDH at L4/5 or L5/S1 were randomized to operative versus nonoperative treatment. At 1-year follow-up, 92 % of operatively treated patients reported fair to good outcomes, while only 61 % of nonoperatively treated patients could report the same outcomes. This trend of superior functional outcomes in the surgically treated group continued at 4 and 10-year follow-up but was only statistically significant at the 1-year postoperative exam. He concluded that most patients with LDH will improve during the first 3 months, and operative intervention should be reserved for patients with continued symptoms after this trial of conservative therapy.

The Weber study was criticized for the small number of patients randomized, the inclusion of only a single treatment center, and inclusion of patients treated over 30 years earlier. In an attempt to address these issues, the Maine Lumbar Spine Study group [24] completed a

Table 15.2 Indications for surgery

• Cauda equina syndrome
• Severe weakness
• Progressive weakness
• Severe debilitating pain
• Failure of conservative treatment

prospective cohort study in patients with LDH to evaluate surgical and nonsurgical treatment outcomes in a contemporary USA practice. Of 507 eligible consenting patients initially enrolled, 10-year outcomes were available for 400 of 477 (84 %) surviving patients, 217 of 255 (85 %) treated surgically, and 183 of 222 (82 %) treated nonsurgically. At 10-year follow-up, a larger proportion of surgical patients reported that their low back and leg pain were much better or completely gone (56 % vs. 40 %, $P=0.006$) and were more satisfied with their current status (71 % vs. 56 %, $P=0.002$). Change in the modified Roland back-specific functional status scale favored surgical treatment, and the relative benefit persisted over the follow-up period. They concluded that surgically treated patients with a herniated lumbar disc had more complete relief of leg pain and improved function and satisfaction compared with nonsurgically treated patients over 10 years.

Most recently, Weinstein et al. [25] in 2008 completed a multicenter prospective, randomized study with a concurrent observational cohort study to more rigorously study the results of surgical and nonoperative treatment for lumbar disc herniation known as the SPORT trial. Patients were enrolled into prospective, randomized (501 participants), and observational cohorts (743 participants) at 13 spine clinics in 11 US states. Interventions were standard open discectomy versus usual nonoperative care. The main outcome measures were changes from baseline in the SF-36 bodily pain (BP) and physical function (PF) scales and the modified Oswestry Disability Index (ODI) assessed at 6 weeks, 3 months, 6 months, and annually thereafter. At the 4-year follow-up analysis, those receiving surgery demonstrated significantly greater improvement in all the primary outcome measures. The percent working was similar between the surgical and nonoperatively treated groups, 84.4 % versus 78.4 %, respectively. They concluded that patients who underwent surgery for a lumbar disc herniation achieved greater improvement than nonoperatively treated patients in all primary and secondary outcomes except work status.

15.6.3 Complications

Despite the benefits that surgical treatments provide to patients, there are also complications associated with any surgical procedure. The most common risks that are inherent to performing a discectomy include the possibility of recurrent disc herniation, dural tear, and postoperative wound infection. Other rare complications that have been reported include nerve root injury, vascular injury, and bowel injury. Another issue that has arisen recently and one that patients often inquire about, though more applicable after a fusion is performed, is the risk that another spine operation will be required in the future. Atlas et al. [24] followed patients for 10 years who initially presented with radiculopathy, and the rates of lumbar spine reoperation in patients are 25 % after discectomy if followed for 10 years, but the rate of operation in patients never having had lumbar spine surgery are also 25 % if followed for 10 years in this patient population. Thus, surgery does not increase the risk of additional surgery in the subsequent 10 years.

The overall risk of recurrent disc herniation is reported to be between 5 and 15 % [25, 26]. Research has shown that 2 years after discectomy, nearly 25 % of patients will have MRI evidence of recurrent herniation at the same level. However, less than half of these herniations were symptomatic and required any treatment. Initial attempts at treating recurrent disc herniations with nonoperative modalities should be entertained just as they are for primary LDHs, but revision discectomy can be performed with good results, albeit with overall increased health care cost to society [27–29]. However, a third herniation at the same level or subsequent instability of the motion segment may require a fusion to avoid further recurrent symptoms from that diseased disc.

The risk of dural tear has been reported to be between 2.7 and 8.7 % for primary discectomy procedures with higher rates seen in minimally invasive approaches [30]. The risk of dural tear lies not in the meningeal injury that is sustained, recognized, and appropriately treated at the time of surgery but rather in the unrecognized or postoperative tear that can lead to wound dehiscence or pseudomeningocele formation. Pseudomeningocoele can cause recurrent back or leg pain and often requires surgical treatment to excise the mass and repair the meningeal defect [29].

Surgical site infections after a discectomy for an LDH occur in approximately 0.1–0.5 % of patients, with diabetic, immunocompromised, and elderly patients at the greatest risk [31]. Infections are often diagnosed by increasing back pain, fevers, chills, erythema, or drainage about the incision and/or increasing inflammatory markers (WBC, ESR, and CRP). Any deep infection after discectomy should be considered a discitis until proven otherwise and treated with surgical irrigation and debridement with intravenous antibiotics.

Conclusion

Lumbar disc herniation with radiculopathy remains one of the most common clinical entities encountered in spine care today. The diagnosis causes significant disability and distress among patients and has a large economic impact on society. A thorough understanding of the clinical presentation, pathologic mechanism, and treatment algorithm of disc herniations is crucial to treating this common disease. Initial management with nonoperative measures such as NSAIDs, activity modification physical therapy, and steroid injections are often successful, but patients must be screened for red flags that necessitate further workup or emergent surgical intervention. Surgical decompression has proven effective for patients that fail conservative management, but the surgeon and the patient must understand the small but significant risk of complications.

References

1. Frymoyer JW (1988) Back pain and sciatica. N Engl J Med 318(5):291–300
2. van Tulder M, Koes B, Bombardier C (2002) Low back pain. Best Pract Res Clin Rheumatol 16(5):761–775
3. Katz JN (2006) Lumbar disc disorders and low-back pain: socioeconomic factors and consequences. J Bone Joint Surg Am 88(Suppl 2):21–24
4. Konstantinou K, Dunn KM (2008) Sciatica: review of epidemiological studies and prevalence estimates. Spine (Phila Pa 1976) 33(22):2464–2472
5. Valat JP, Genevay S, Marty M, Rozenberg S, Koes B (2010) Sciatica. Best Pract Res Clin Rheumatol 24(2):241–252
6. Taylor VM, Deyo RA, Cherkin DC, Kreuter W (1994) Low back pain hospitalization. Recent United States trends and regional variations. Spine (Phila Pa 1976) 19(11):1207–1212; discussion 1213
7. Lee JK, Amorosa L, Cho SK, Weidenbaum M, Kim Y (2010) Recurrent lumbar disk herniation. J Am Acad Orthop Surg 18(6):327–337
8. Buckwalter JA, Mow VC, Boden SD, Eyre DR, Weidenbaum M (2000) Intervertebral disc structure, composition, and mechanical function. In: Buckwalter JA, Einhorn TA, Simon SR (eds) Orthopaedic basic science: biology and biomechanics of the musculoskeletal system. American Academy of Orthopaedic Surgeons, Rosemont, p 547
9. Kostuik JP, Harrington I, Alexander D, Rand W, Evans D (1986) Cauda equina syndrome and lumbar disc herniation. J Bone Joint Surg Am 68(3):386–391
10. Shapiro S (1993) Cauda equina syndrome secondary to lumbar disc herniation. Neurosurgery 32(5):743–746; discussion 746–747
11. Spector LR, Madigan L, Rhyne A, Darden B 2nd, Kim D (2008) Cauda equina syndrome. J Am Acad Orthop Surg 16(8):471–479
12. Pott P (1936) Remarks on that kind of palsy of the lower limbs which is frequently found to accompany a curvature of the spine, and is supposed to be caused by it, together with its method of cure. Med Classics 1:281–297
13. Battie MC, Videman T, Gibbons LE, Fisher LD, Manninen H, Gill K (1995) Volvo award in clinical sciences. Determinants of lumbar disc degeneration. A study relating lifetime exposures and magnetic resonance imaging findings in identical twins. Spine (Phila Pa 1976) 20(24):2601–2612
14. McGill SM (1997) The biomechanics of low back injury: implications on current practice in industry and the clinic. J Biomech 30(5):465–475
15. Videman T, Battie MC, Ripatti S, Gill K, Manninen H, Kaprio J (2006) Determinants of the progression in lumbar degeneration: a 5-year follow-up study of adult male monozygotic twins. Spine (Phila Pa 1976) 31(6):671–678
16. Patel AA, Spiker WR, Daubs M, Brodke D, Cannon-Albright LA (2011) Evidence for an inherited predisposition to lumbar disc disease. J Bone Joint Surg Am 93(3):225–229
17. Awad JN, Moskovich R (2006) Lumbar disc herniations: surgical versus nonsurgical treatment. Clin Orthop Relat Res 443:183–197
18. Deyo RA, Diehl AK, Rosenthal M (1986) How many days of bed rest for acute low back pain? A randomized clinical trial. N Engl J Med 315(17):1064–1070
19. van Tulder MW, Scholten RJ, Koes BW, Deyo RA (2000) Nonsteroidal anti-inflammatory drugs for low

back pain: a systematic review within the framework of the Cochrane Collaboration Back Review Group. Spine (Phila Pa 1976) 25(19):2501–2513
20. Saal JA (1996) Natural history and nonoperative treatment of lumbar disc herniation. Spine (Phila Pa 1976) 21(24 Suppl):2S–9S
21. Young IA, Hyman GS, Packia-Raj LN, Cole AJ (2007) The use of lumbar epidural/transforaminal steroids for managing spinal disease. J Am Acad Orthop Surg 15(4):228–238
22. Buttermann GR (2004) Treatment of lumbar disc herniation: epidural steroid injection compared with discectomy. A prospective, randomized study. J Bone Joint Surg Am 86-A(4):670–679
23. Weber H (1983) Lumbar disc herniation. A controlled, prospective study with ten years of observation. Spine (Phila Pa 1976) 8(2):131–140
24. Atlas SJ, Keller RB, Wu YA, Deyo RA, Singer DE (2005) Long-term outcomes of surgical and nonsurgical management of sciatica secondary to a lumbar disc herniation: 10 year results from the maine lumbar spine study. Spine (Phila Pa 1976) 30(8):927–935
25. Weinstein JN, Lurie JD, Tosteson TD et al (2008) Surgical versus nonoperative treatment for lumbar disc herniation: four-year results for the Spine Patient Outcomes Research Trial (SPORT). Spine (Phila Pa 1976) 33(25):2789–2800
26. Swartz KR, Trost GR (2003) Recurrent lumbar disc herniation. Neurosurg Focus 15(3):E10
27. Suk KS, Lee HM, Moon SH, Kim NH (2001) Recurrent lumbar disc herniation: results of operative management. Spine (Phila Pa 1976) 26(6): 672–676
28. Ambrossi GL, McGirt MJ, Sciubba DM et al (2009) Recurrent lumbar disc herniation after single-level lumbar discectomy: incidence and health care cost analysis. Neurosurgery 65(3):574–578; discussion 578
29. Kaar GF, Briggs M, Bashir SH (1994) Thecal repair in post-surgical pseudomeningocoele. Br J Neurosurg 8(6):703–707
30. Teli M, Lovi A, Brayda-Bruno M et al (2010) Higher risk of dural tears and recurrent herniation with lumbar micro-endoscopic discectomy. Eur Spine J 19(3): 443–450
31. O'Toole JE, Eichholz KM, Fessler RG (2009) Surgical site infection rates after minimally invasive spinal surgery. J Neurosurg Spine 11(4):471–476

16 Lumbar Stenosis

Paul D. Kim and Hyun Bae

16.1 Pathophysiology

Simply described, lumbar stenosis is a narrowing of the spinal canal with its bony and soft tissue elements [11]. Lumbar congenital stenosis occurs when anatomic variations in the lumbar spinal anatomy lead to a narrow canal and is typically present in multiple levels in younger patients with less degenerative changes [10]. Achondroplastic dwarfs are also commonly affected by this condition much more than the general population [6]. In degenerative lumbar stenosis during the course of aging, disk degeneration results in loss of disk height. With loss of disk height, the annulus fibrosus bulges, impinging the dural sac anteriorly; posteriorly, the ligamentum flavum buckles also impinging on the neural elements (Figs. 16.1 and 16.2). Laterally, the facet joints may degenerate with osteophytes, thickened capsules, or synovial cysts also causing stenosis most commonly from the superior facet of the inferior vertebral body (Fig. 16.3).

Paul D. Kim, M.D.
Spine Institute of San Diego,
6719 Alvarodo Road, Suite 308,
San Diego, CA 92130, USA

H. Bae, M.D. (✉)
Director Education, Cedavs Sinai Spine Center,
444 S. San Vicente Blvd, Los Angeles,
CA 90048, USA
e-mail: baemd@me.com

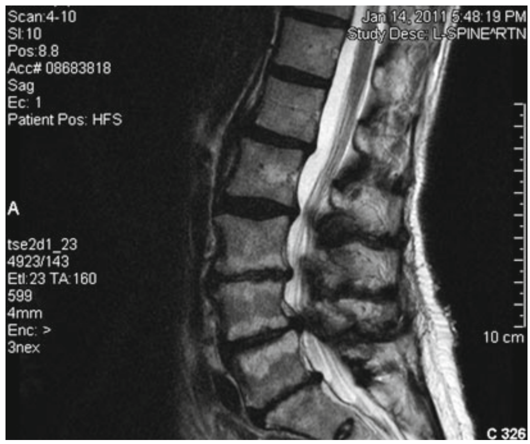


Fig. 16.1 Sagittal T2 MRI demonstrating spinal stenosis

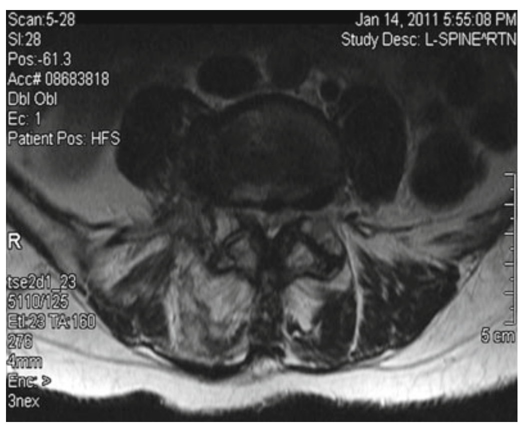


Fig. 16.2 Axial T2 MRI demonstrating spinal stenosis

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_16, © Springer-Verlag Berlin Heidelberg 2014

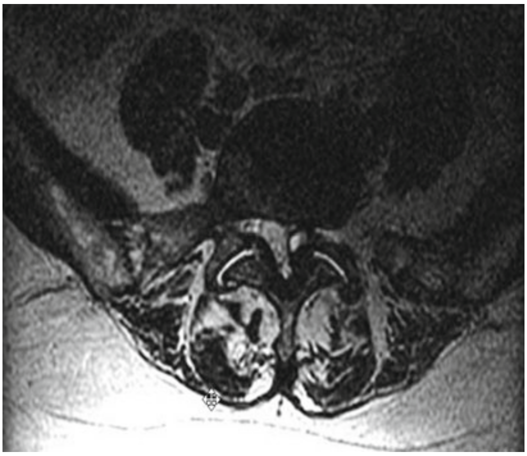


Fig. 16.3 Axial T2 MRI demonstrating a left-sided facet cyst

Fig. 16.4 Lateral radiograph demonstrating spondylolisthesis at L5–S1

More precisely, spinal stenosis may be divided into central stenosis, lateral recess stenosis, and foraminal stenosis. Central stenosis is defined as impingement over the dural sac, and lateral recess stenosis is the area of compression between the dural sac and the beginning of the neural foramen. The foreman lies between the pedicles with the lateral disk anterior and the facet and ears dorsal. Close inspection of preoperative imaging is required for surgical planning. Commonly associated with lumbar spinal stenosis is degenerative lumbar spondylolisthesis which most commonly presents at the L4–5 level in adult females, though L5–S1 spondylolisthesis also commonly occurs with isthmic spondylolisthesis (Fig. 16.4).

16.2 History

Patients typically present in the fifth to seventh decades and symptoms may have an insidious onset. Patients may present in a variety of ways, and there is no feature which is considered pathognomonic; however, a typical symptom is low back pain with radiation down to the buttocks and legs [1]. Patients may describe improvement in their symptoms with a forward-flexed posture (leaning on a shopping cart, cycling, walking up a hill) where the cross-sectional area of the canal increases slightly; conversely, symptoms worsen with activities in an erect posture. This is known as neurogenic claudication. Common in the age group is peripheral vascular disease, causing vascular claudication which must be differentiated from neurogenic claudication. In vascular claudication, the symptoms improve with rest and do not require sitting or flexing forward to relieve the discomfort. Also, in vascular claudication, patients may complain of calf pain and diminished pulses. A history of long-standing diabetes mellitus in a non-dermatomal sensory disturbance may also indicate peripheral neuropathy rather than spinal stenosis. Although cauda equina syndrome is rare with spinal stenosis, it is prudent to question whether there are symptoms of incontinence and perineal numbness (“saddle anesthesia”).

16.3 Physical Examination

The history of spinal stenosis is important in diagnosing lumbar spinal stenosis as there is no one specific physical examination finding which diagnoses someone with spinal stenosis. Most commonly, patients present with a completely normal exam; however there are some subtle findings that may be present. Upon examination, patients may have gait abnormalities and may walk with a forward-flexed posture which may alleviate their symptoms of spinal stenosis. Also, patients may display tenderness in the lower back, buttocks and have a positive straight leg raise if there is nerve root entrapment associated with a herniated disk.

Conversely, sensation may be altered especially in patients with long-standing diabetes mellitus, and a non-dermatomal pain distribution may point to a peripheral neuropathy. Muscle weakness should also be noted. Deep tendon reflex asymmetry should also be tested for, although loss of deep tendon reflexes may occur with aging. Hyperreflexia should be also examined closely in this population as myelopathy commonly occurs in this age group. The hip must also be examined to rule out hip pathologies, and pulses should be taken to rule out vascular etiologies.

16.4 Diagnostic Studies

Standing radiographs with flexion and extension views are essential in the workup of spinal stenosis as it may delineate otherwise subtle instabilities of the spine. Bony lesions, spondylosis, and spinal alignment are shown with radiographs. MRI is the most common modality to visualize the soft tissue and neural elements and must be carefully examined to identify the particular spinal level of and particular area of stenosis. Computed tomography (CT) with myelography is useful in patients with previous surgery and contraindication to magnetic resonance imaging (MRI) scanning (pacemakers, cochlear implants). This is, however, an invasive procedure where contrast medium is injected into the spinal canal, and post-procedure headaches are not uncommon with this procedure. Disk herniations or facet cysts must be identified which may produce radicular symptoms. Patients may also be referred for electrophysiological studies (EP) (EMG, nerve conduction velocity) if nonorganic extremity pain or peripheral neuropathy is suspected.

16.5 Nonoperative Treatment

Nonoperative modalities remain the foundation of initial treatment of the patient. Activity modification and nonsteroidal medication (NSAIDs) have proven to be effective in treating the inflammatory component of pain. NSAIDs should be used carefully as needed because of the side effects associated with long-term use such as ulcers, renal dysfunction, and cardiovascular events [7]. Narcotic and muscle-relaxants should only be used acutely because of the serious side effects such as constipation, drowsiness, and habit-forming potential. Oral corticosteroids have proven to be effective acutely for radicular symptoms however must be used only temporarily because of known long-term side effects such as glucose intolerance and avascular necrosis. Tricyclic antidepressants (TCA) are effective for dysesthetic nerve pain and may help patients achieve sleep if the pain causes sleep disturbances. After the acute phase of pain has been controlled, physical therapy should be initiated to strengthen the core muscles and potentially help to stabilize the spine. Flexion-based exercises can be useful since flexion increases the diameter of the spinal canal with activities such as biking or an inclined treadmill.

Epidural steroid injections may be effective in particular for radicular-type symptoms and may provide temporary relief of symptoms although controversy exists for their effectiveness [4]. It is a relatively benign procedure and not only may treat the patient but may serve as a good predictor as to who will benefit from spinal surgery as well. Thus, the utility of injections may be greatest in their ability to help verify the diagnosis and localize the pathology. Potential complications of epidural include persistent dural leak, infection, and epidural bleeding although rare.

16.6 Operative Treatment

16.6.1 Indications

Operative treatment should be considered when a patient has failed nonoperative treatment, is suffering from intolerable disability, or has a persistent neurological deficit. Medical comorbidities must be taken into consideration when deciding to undergo and decide upon the type of surgical treatment. Careful preoperative planning must be done with the use of proper imaging correlated with the history and physical exam findings. Elderly patients with medical issues may benefit from direct or indirect lumbar decompression [9]. Indications for lumbar fusion in addition to decompression include instability (spondylolisthesis) or extensive decompression causing iatrogenic instability. In addition, an intraoperative pars interarticularis fracture may necessitate fusion.

16.6.2 Surgical Decision Making

The backbone of surgical treatment of spinal stenosis remains adequate decompression at the stenotic levels. There are many techniques used to decompress the neural elements ranging from standard laminectomies to minimally invasive tubular decompressions. Laminectomy is the gold standard and provides the widest and most direct decompression. It includes partial removal of both osseous (lamina, facet joints) and soft tissue elements (posterior ligamentous complex). This must be performed carefully as excess removal may contribute to instability in the lumbar spine. When performing a laminectomy, at least 50 % of each facet-joint complex must be preserved to prevent iatrogenic instability. Alternatives to laminectomy include laminoplasty in which the lamina is undercut and bony elements are preserved [8]. Most of the spinous process and associated posterior soft tissue elements remain intact, and this is the preference at our institution.

Indications for fusion include instability, degenerative scoliosis, associated spondylolisthesis, back pain after previous decompression, or iatrogenic instability. Lumbar fusion may be achieved in a variety of ways including noninstrumented fusion or instrumented fusion. Instrumented fusion results in higher fusion rates; higher fusion rates result in better clinical outcomes [5]. The most common modern instrumentation used for fusion includes pedicle screws and rods in combination with either autogenous bone graft or allograft. Use of interbody devices may also increase fusion rates providing anterior spinal column support with the ability to re-create disk height and resultant increased foraminal height and improved lordosis.

Finally, indirect decompressions can be an option for patients with dynamic stenosis or neurogenic claudication. If the patient is completely better with sitting and symptomatic with standing, a spacer such as an x-stop device can be placed between the spinous processes to block extension of that level with standing and walking. This procedure is relatively new but shows promise in well-selected patients.

16.6.3 Results

Lumbar spinal stenosis is a common condition and the focus of recent high-quality clinical trials. The spine outcome research trial (SPORT) demonstrated that at 2 years, surgically treated patients had less pain and better function than those treated with nonoperative modalities in the as-treated analysis. These patients also had less disability [12]. The Maine lumbar study which prospectively followed up an observational cohort up to 10 years demonstrated that the surgically treated patients did better short term (4 years) while at 10 years, the results were equivocal [2, 3].

16.7 Postoperative Care

For a fusion, postoperative films are taken, and patients are usually hospitalized for 2–4 days postoperatively to manage pain and medical stabilization. Prophylactic antibiotics are given for 24 h. Postoperative closed suction drains may be placed and are usually discontinued when producing less than 30 cc per 12 h shift. Venodynes or compression stockings and sequential compression devices (SCDs) are continued postoperatively until the patient is mobilized, and patients typically do not receive anticoagulation unless they were on it preoperatively (usually started postoperative day 2 or 3 to minimize the risk of epidural hematoma). The foley catheter is discontinued when the patient is mobilized. The wound is inspected before discharge to make sure that there is no erythema, drainage, and fluctuance. Inpatient physical therapy is undertaken to mobilize the patient, and patient is discharged to either home or an acute/subacute rehabilitation facility to continue postoperative rehabilitation. The patient may be braced postoperatively to limit lumbar range-of-motion. Patients are usually followed up at 2 weeks, 6 weeks, 3 months, and 6 months postoperatively.

16.8 Complications

Dural tears have an incidence up to 8 % of the time in primary cases and up to 15 % of the time in revision cases. Patients typically complain of positional headaches postoperatively. The durotomy is repaired primarily, if possible, intraoperatively and placed on 24–48 h of bed rest. Neurological deficits do occur if a nerve root is excessively handled during the decompression or there is misplaced hardware. An epidural hematoma is rare (<1 %) but may also cause potential neurological complications. These patients usually present with severe back pain which evolves into a neurological deficit. Close sequential neurological examinations must be performed and documented to help prevent further injury.

16.9 Summary

Lumbar spinal stenosis is one of the most common spinal disorders today and projected to increase with the aging population with more physical demands at a later age. The treatment decision must be balanced with the patient lifestyle demands and medical comorbidities. Research and new technologies continue to evolve to help elucidate the proper treatment and techniques to treat this common problem.

References

1. Amundsen T, Weber H et al (1995) Lumbar spinal stenosis. Clinical and radiologic features. Spine (Phila Pa 1976) 20(10):1178–1186
2. Atlas SJ, Keller RB et al (2000) Surgical and nonsurgical management of lumbar spinal stenosis: four-year outcomes from the Maine lumbar spine study. Spine (Phila Pa 1976) 25(5):556–562
3. Atlas SJ, Keller RB et al (2005) Long-term outcomes of surgical and nonsurgical management of lumbar spinal stenosis: 8 to 10 year results from the Maine lumbar spine study. Spine (Phila Pa 1976) 30(8):936–943
4. Cuckler JM, Bernini PA et al (1985) The use of epidural steroids in the treatment of lumbar radicular pain. A prospective, randomized, double-blind study. J Bone Joint Surg Am 67(1):63–66
5. Kornblum MB, Fischgrund JS et al (2004) Degenerative lumbar spondylolisthesis with spinal stenosis: a prospective long-term study comparing fusion and pseudarthrosis. Spine (Phila Pa 1976) 29(7):726–733; discussion 733–734
6. Lutter LD, Longstein JE et al (1977) Anatomy of the achondroplastic lumbar canal. Clin Orthop Relat Res (126):139–142
7. Mukherjee D, Nissen SE et al (2001) Risk of cardiovascular events associated with selective COX-2 inhibitors. JAMA 286(8):954–959
8. O'Leary PF, McCance SE (2001) Distraction laminoplasty for decompression of lumbar spinal stenosis. Clin Orthop Relat Res (384):26–34
9. Rosen DS, O'Toole JE et al (2007) Minimally invasive lumbar spinal decompression in the elderly: outcomes of 50 patients aged 75 years and older. Neurosurgery 60(3):503–509; discussion 509–510
10. Singh K, Samartzis D et al (2005) Congenital lumbar spinal stenosis: a prospective, control-matched, cohort radiographic analysis. Spine J 5(6):615–622
11. Verbiest H (1954) A radicular syndrome from developmental narrowing of the lumbar vertebral canal. J Bone Joint Surg Br 36-B(2):230–237
12. Weinstein JN, Lurie JD et al (2008) Surgical versus nonoperative treatment for lumbar disc herniation: four-year results for the Spine Patient Outcomes Research Trial (SPORT). Spine (Phila Pa 1976) 33(25):2789–2800

17 Lumbar Degenerative Spondylolisthesis

Loukas Koyonos and Jeffrey A. Rihn

17.1 Introduction

Spondylolisthesis is the anterior slippage of one vertebra on top of another. Several different types of spondylolisthesis have been described including congenital, isthmic, degenerative, traumatic, pathologic, and iatrogenic. This chapter will focus on degenerative spondylolisthesis of the lumbar spine. Degenerative lumbar spondylolisthesis is a relatively common condition that typically affects persons over the age of 50 and is more common in females and those of African American decent. Patients typically present with a constellation of symptoms that include back pain, radiculopathy, and/or neurogenic claudication. In the absence of progressive neurological deficit and/or symptoms of cauda equina syndrome, treatment begins with a series of nonoperative interventions that include physical therapy, nonsteroidal anti-inflammatory medications, and epidural injections. In those patients who fail nonoperative treatment, surgical intervention in the form of lumbar decompression and fusion has proven to provide effective and durable relief of symptoms.

L. Koyonos, M.D. • J.A. Rihn, M.D. (✉)
Department of Spine Surgery,
The Rothman Institute of Orthopaedics,
Thomas Jefferson University Hospital,
925 Chestnut Street, 5th Floor, Philadelphia,
PA 19107, USA
e-mail: loukaskoyonosmd@gmail.com;
jrihno16@yahoo.com

17.2 Epidemiology

The overall incidence of lumbar degenerative spondylolisthesis is estimated at 8.7 % [7]. It occurs more commonly in females than males, in African Americans than Caucasians, and in individuals greater than 50 years old. Females may be affected more because of increased ligamentous laxity relative to males. It has also been shown that there is a high expression of estrogen receptors in facet articular cartilage, and this may have an implication in the development of this condition in postmenopausal women [5]. The most commonly affected level is L4–5. This may be secondary to the relative sagittal orientation of the facet joint at this level as compared to the L5–S1 articulation, making it less able to resist forward flexion forces.

17.3 Pathophysiology

Lumbar degenerative spondylolisthesis begins with degeneration of the intervertebral disk and facet joints. Disk space narrowing leads to buckling of the ligamentum flavum, which lies posterior to the thecal sac and contributes to the development of stenosis. With progression of disk and facet joint degeneration, instability can develop, leading to slippage (listhesis) of one vertebra on top of another. With the typical anterior slip of the superior vertebra, (i.e., anterolisthesis), arthritic changes can progress at that segment,

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_17, © Springer-Verlag Berlin Heidelberg 2014

such as osteophyte formation, hypertrophy of the ligamentum flavum, sclerosis of the subchondral bone, and hypertrophy of the facet. These changes represent the body's attempt to stabilize the joint. As a result of these compensatory changes, the superior vertebra rarely slips more than half the distance of the inferior vertebral body as seen on a lateral x-ray. The arthritic changes that occur are thought to cause lower back pain. Likewise, as the vertebra slips anteriorly, stenosis of the spinal canal ensues, particularly in the lateral recess and neuroforamen, leading to compression of the neural elements.

17.4 Evaluation

This condition usually presents with lower back pain and symptoms of spinal stenosis, including neurogenic claudication and/or radiculopathy. Less commonly, progressive lower extremity weakness and/or bowel/bladder dysfunction can occur. These more severe symptoms represent a surgical emergency. Patients experiencing neurogenic claudication often complain of buttock or proximal thigh pain and numbness, tingling, or weakness in the lower extremities. Classically, symptoms are worse with walking upright or standing and better with sitting or leaning forward (i.e., flexing at the waist). This phenomenon is exemplified in the "shopping cart sign." The shopping cart sign refers to the ability of people with spinal stenosis to exhibit ample walking endurance when leaning over and pushing a shopping cart. In this position, the lumbar spine is flexed, creating more room for the neural elements within the stenotic spinal canal. Similarly, patients with stenosis will be able to ride a bike with minimal symptoms because they are doing so with their body in a flexed position. It is also typically easier for patients with stenosis to walk up a hill as opposed to down a hill because the body takes a slightly flexed position when walking up a hill as opposed to a slightly extended position when walking down a hill.

Neurogenic claudication must be differentiated from vascular claudication. Vascular claudication is classically relieved by rest as this decreases the oxygen demand of muscles. Neurogenic claudication is classically relieved by a change in position (flexion of the spine) as this decreases compression on the neural elements. The two are often differentiated by having the patient ride a stationary bicycle. A patient who solely has neurogenic claudication will perform better because of the flexed position of the spine and the relative patency of his/her blood vessels supplying the musculature of the lower extremities.

This condition can also cause radiculopathy (pain, parasthesias, motor and sensory loss in the distribution of one specific nerve root). The most commonly affected nerve root is L5, which usually corresponds to an L4–5 anterolisthesis. Symptoms may arise secondary to mechanical compression of the nerve or simply due to local release of inflammatory mediators. When the L5 nerve root is affected from an L4–5 spondylolisthesis, it usually occurs because of lateral recess stenosis caused by osteophytes from the superior articular facet of L5 and ligamentum flavum hypertrophy, in addition to the slippage. A more significant spondylolisthesis can lead to neuroforaminal stenosis and compression of the exiting nerve root (L4 nerve root in the case of a L4–5 spondylolisthesis). Compression can also be compounded by the presence of a herniated disk.

Other presenting symptoms include mechanical lower back pain, unsteady gait, frequent falls, and even bowel or bladder dysfunction with severe stenosis. Low back pain is a relatively common complaint in patients with degenerative spondylolisthesis. The cause of the pain is controversial, but it is thought to arise primarily from the degenerated disk with flexion movements and the arthritic facets with extension movements. Like the lower extremity symptoms, the low back pain is often worse with walking or standing and improved with sitting. This is referred to as "claudicatory low back pain." This is opposed to patients with discogenic (pain originating from the intervertebral disk) low back pain, who typically have increased pain with sitting.

Physical exam should include a thorough neurologic exam including strength, sensation, and reflexes. Likewise, a careful vascular exam should also be performed to rule out vascular causes of

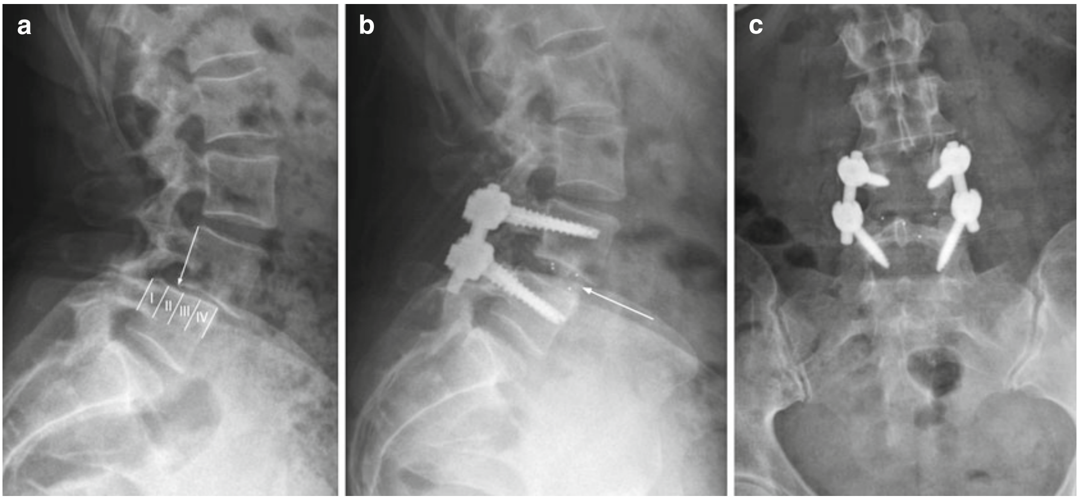


Fig. 17.1 (**a**) Preoperative lateral x-ray showing a Grade II spondylolisthesis. The *arrow* outlines the posterior aspect of the L4 vertebral body and indicates a grade I anterolisthesis. Note the Meyerding classification has been drawn in. (**b**) Postoperative lateral x-ray after transforaminal lumbar interbody fusion. The *arrow* points to radiopaque markers within the cage which identify its position between the vertebrae. Note the improvement of the spondylolisthesis. (**c**) Postoperative AP x-ray showing pedicle screw and rod fixation

claudication. Patients may exhibit a stooped posture, hip flexion contractures, weakness/atrophy of paraspinal muscles, and loss of lumbar lordosis. Range of motion of the spine may reveal increased flexion of the lumbar spine and sometimes a palpable step-off of the spinous process at the affected level. Patients with this condition often have limited lumbar extension. It is not uncommon for the neurological exam to be completely normal in patients with degenerative spondylolisthesis and stenosis, although findings of weakness and/or numbness in a particular nerve distribution can exist.

17.5 Imaging/Classification

Radiographic evaluation begins with standing, weight-bearing anteroposterior (AP) and lateral x-rays. These are used to evaluate bony architecture, degenerative changes, severity of spondylolisthesis, and any associated deformities such as scoliosis. The amount of motion at adjacent vertebral segments can also be assessed by obtaining flexion and extension lateral x-rays in a supine or sitting position. Greater than 3 mm of motion when comparing the flexion and extension radiographs is indicative of spinal instability.

The most utilized classification system for spondylolisthesis was described by Meyerding [9]. It is described as a percentage slip in the anterior direction of the cephalad vertebra on the caudad vertebra. The point of reference is the posterior vertebral body. Grade I is 0–25 %, Grade II is 25–50 % (Fig. 17.1a), Grade III is 50–75 %, and Grade IV is 75–100 %. Listhesis more than 100 % is termed spondyloptosis.

In the past, CT scans were used to evaluate three-dimensional bony architecture, especially in planning an operation. For the most part, they have been replaced by MRIs, which are useful in evaluating disk pathology, nerve root compression, hypertrophy of the ligamentum flavum, and synovial cyst formation. In patients in whom MRI is contraindicated, a CT myelogram (Fig. 17.2) can be useful to further delineate the pathology and to plan an operation.

17.6 Treatment

17.6.1 Nonoperative

Treatment usually begins with a conservative, nonoperative approach. This is successful in the majority of cases, for at least some period of time.

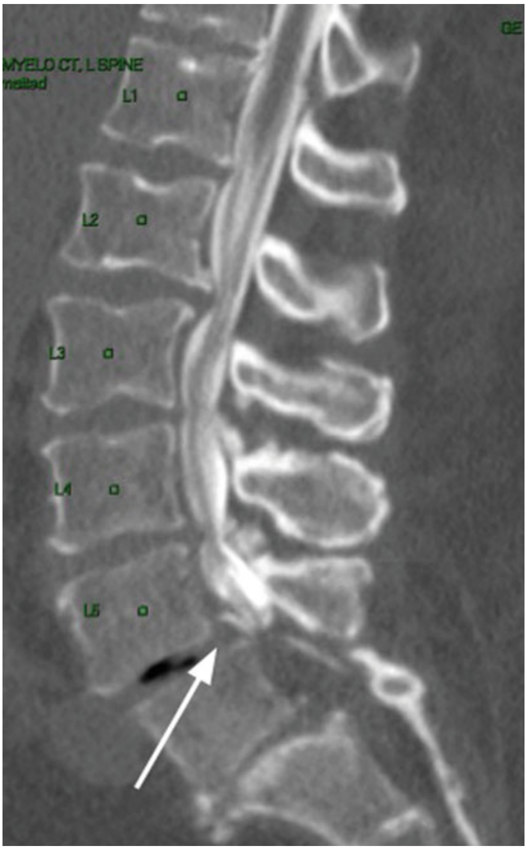


Fig. 17.2 Sagittal CT myelogram depicting anterior spondylolisthesis at L5–6. The *arrow* points to the “cutoff” of intrathecal dye at the level of the spondylolisthesis, indicating the presence of severe spinal stenosis at this level. This patient has six lumbar vertebrae, which is a variant of normal anatomy

First-line treatment includes activity modification, nonsteroidal anti-inflammatory medications, and physical therapy. Second-line treatment entails epidural corticosteroid injections whose theoretical benefit comes as a result of bathing the nerve roots and surrounding area in anti-inflammatory medication. Long-term benefits have yet to be shown, but a subset of patients do find these injections helpful in the short term.

17.6.2 Operative

Indications for operative treatment include the following: (1) neurogenic claudication or persistent back pain combined with leg pain leading to a severe reduction in quality of life despite at least 3 months of conservative intervention, (2) worsening neurologic symptoms, or (3) bowel or bladder symptoms related to neurologic compression [10].

The mainstay of surgical management consists of a spectrum ranging from decompression alone, to decompression and un-instrumented fusion, to decompression with instrumented fusion (Fig. 17.3a, b). Decompression alone has shown good to excellent results [2], but most surgeons avoid this approach because it can further destabilize the spine and lead to progression of the spondylolisthesis. Un-instrumented fusion as compared to decompression alone has shown improved outcomes, so most surgeons advocate fusion at this time [6]. It has been shown that instrumentation does improve the fusion rate but may not improve functional outcome (Fig. 17.2) [3]. Subsequent long-term data though suggests that developing a pseudarthrosis (nonunion) can result in a poorer outcome [8]. This continues to be a controversial topic. In an effort to better delineate the efficacy of surgical vs. nonsurgical management, the Spine Patient Outcomes Research Trial (SPORT) was conceived. In 2009, 4-year data revealed greater pain relief and improvement in function in the surgical group [11].

Transforaminal lumbar interbody fusion (Fig. 17.1b, c) is a more recent technique that uses a posterolateral approach to access the intervertebral disk space in order to place a graft between the two vertebral bodies. The goal is to provide a greater surface area for fusion. This procedure is usually combined with a posterolateral fusion as well. Studies are currently being performed to determine the efficacy of this procedure as compared to posterior fusion alone.

Attempts have also been made to develop motion-sparing devices, which avoid fusion, stabilize the spine, and decrease neural compression. One type of motion-sparing device is the interspinous spacer. Such a device is placed between the spinous processes and is used to distract the posterior elements of the vertebrae at the affected level (Fig. 17.4a, b). This distraction has been shown to increase the area available for the neural elements and reduce the symptoms associated with stenosis [12]. However, degenerative

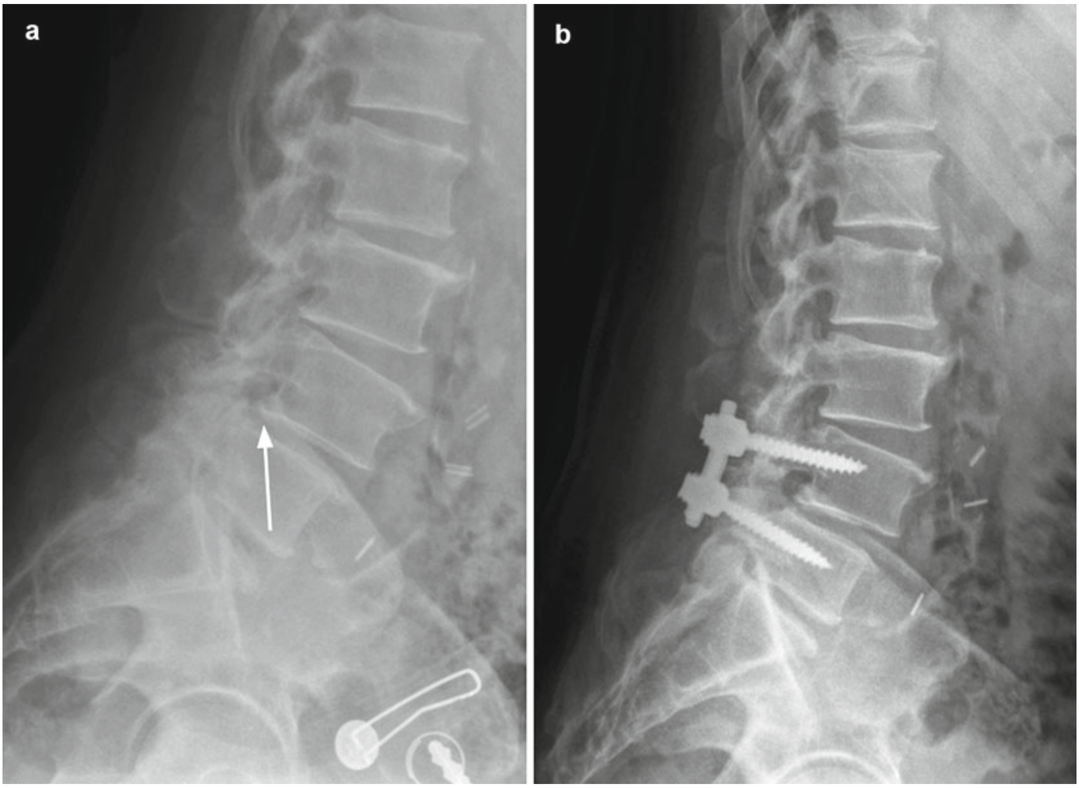


Fig. 17.3 (**a**) Lateral x-ray of lumbar spine. The *arrow* points to a Grade I spondylolisthesis. (**b**) Postoperative lateral x-ray showing pedicle screw and rod fixation after laminectomy and fusion. Note the retrolisthesis at the level above the fusion (i.e., the L3 vertebral body is slipped slightly posterior on the L4 vertebral body)

spondylolisthesis greater than Grade I is currently a contraindication to the use of this device. The long-term efficacy of these devices is unknown, and current investigation is under way to determine the role of these options.

Historically, iliac crest autograft bone has been used for posterolateral and interbody fusion of the lumbar spine. Because of the relatively high incidence of donor site-related complications (e.g., infection, wound complications, persistent pain), alternatives to autograft bone have been utilized, including allograft bone, demineralized bone matrix, synthetic bone graft substitutes (e.g., calcium sulfate, beta-tricalcium phosphate), and recombinant bone morphogenetic protein (BMP). Recombinant BMPs have been popularized in recent years as a way to promote bony fusion in spine surgery. Recombinant human bone morphogenetic protein-2 and protein-7 (rhBMP-2 and rhBMP-7) are the BMPs that have been most rigorously studied in clinical spine surgery. Currently, rhBMP-2 is the only form of BMP that has FDA approval for use in spinal fusion. Furthermore, it is only FDA approved for use in anterior lumbar interbody fusion. Nonetheless, it is used extensively in an "off-label" fashion in posterolateral fusion and transforaminal lumbar interbody fusion. The correct dose of BMP is still being studied for these applications, but multiple investigations have validated the safety and efficacy of its use in these settings. For example, rhBMP-2 has been shown to result in both quicker time to fusion as well as more rapid improvement in functional outcome as compared to local autograft bone [1]. Similarly, when compared to iliac crest bone graft (the gold standard) in patients over 60 years of age, rhBMP-2 has been shown to have comparable safety, clinical efficacy, and cost-effectiveness [4].

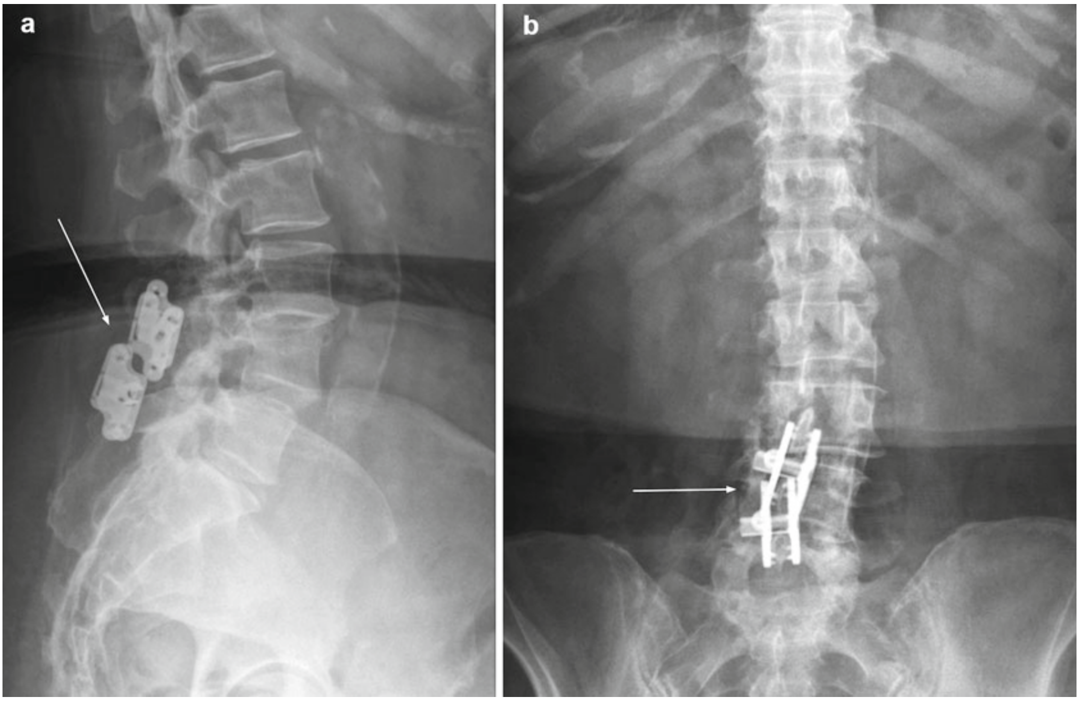


Fig. 17.4 (**a**) Lateral x-ray depicting interspinous spacer *arrows* devices placed between L4–5 and L5–S1. (**b**) AP x-ray of the same devices. Because of the significant spondylolisthesis in this patient, particularly at the L4–5 level, this patient was not a good candidate for this procedure. Postoperatively, the patient's symptoms did not improve, and the patient required reoperation to perform a formal L3–5 laminectomy and fusion

Conclusion

Lumbar degenerative spondylolisthesis is a relatively common disorder as people age. It most commonly affects the L4–5 level and is more common among females and those of African American descent. It can cause debilitating back pain, radiculopathy, and neurogenic claudication. Many patients respond well to nonoperative treatment, at least initially. For those who do not respond or who develop subsequent progression of symptoms, recent evidence suggests that surgical treatment can provide a significant improvement in outcomes.

References

1. Boden SD, Kang J, Sandhu H, Heller JG (2002) Use of recombinant human bone morphogenetic protein-2 to achieve posterolateral lumbar spine fusion in humans: a prospective, randomized clinical pilot trial: 2002 Volvo Award in clinical studies. Spine (Phila Pa 1976) 27:2662–2673
2. Epstein NE (1998) Decompression in the surgical management of degenerative spondylolisthesis: advantages of a conservative approach in 290 patients. J Spinal Disord 11:116–122; discussion 123
3. Fischgrund JS, Mackay M, Herkowitz HN, Brower R, Montgomery DM, Kurz LT (1997) 1997 Volvo Award winner in clinical studies. Degenerative lumbar spondylolisthesis with spinal stenosis: a prospective, randomized study comparing decompressive laminectomy and arthrodesis with and without spinal instrumentation. Spine (Phila Pa 1976) 22:2807–2812
4. Glassman SD, Carreon LY, Djurasovic M, Campbell MJ, Puno RM, Johnson JR, Dimar JR (2008) RhBMP-2 versus iliac crest bone graft for lumbar spine fusion: a randomized, controlled trial in patients over sixty years of age. Spine (Phila Pa 1976) 33:2843–2849
5. Ha KY, Chang CH, Kim KW, Kim YS, Na KH, Lee JS (2005) Expression of estrogen receptor of the facet joints in degenerative spondylolisthesis. Spine (Phila Pa 1976) 30:562–566
6. Herkowitz HN, Kurz LT (1991) Degenerative lumbar spondylolisthesis with spinal stenosis. A prospective

study comparing decompression with decompression and intertransverse process arthrodesis. J Bone Joint Surg Am 73:802–808
7. Iguchi T, Wakami T, Kurihara A, Kasahara K, Yoshiya S, Nishida K (2002) Lumbar multilevel degenerative spondylolisthesis: radiological evaluation and factors related to anterolisthesis and retrolisthesis. J Spinal Disord Tech 15:93–99
8. Kornblum MB, Fischgrund JS, Herkowitz HN, Abraham DA, Berkower DL, Ditkoff JS (2004) Degenerative lumbar spondylolisthesis with spinal stenosis: a prospective long-term study comparing fusion and pseudarthrosis. Spine (Phila Pa 1976) 29:726–733; discussion 733–724
9. Meyerding HW (1956) Spondylolisthesis; surgical fusion of lumbosacral portion of spinal column and interarticular facets; use of autogenous bone grafts for relief of disabling backache. J Int Coll Surg 26: 566–591
10. Sengupta DK, Herkowitz HN (2005) Degenerative spondylolisthesis: review of current trends and controversies. Spine (Phila Pa 1976) 30:S71–S81
11. Weinstein JN, Lurie JD, Tosteson TD, Zhao W, Blood EA, Tosteson AN, Birkmeyer N, Herkowitz H, Longley M, Lenke L, Emery S, Hu SS (2009) Surgical compared with nonoperative treatment for lumbar degenerative spondylolisthesis. Four-year results in the Spine Patient Outcomes Research Trial (SPORT) randomized and observational cohorts. J Bone Joint Surg Am 91:1295–1304
12. Zucherman JF, Hsu KY, Hartjen CA, Mehalic TF, Implicito DA, Martin MJ, Johnson DR 2nd, Skidmore GA, Vessa PP, Dwyer JW, Puccio ST, Cauthen JC, Ozuna RM (2005) A multicenter, prospective, randomized trial evaluating the X STOP interspinous process decompression system for the treatment of neurogenic intermittent claudication: two-year follow-up results. Spine (Phila Pa 1976) 30:1351–1358

18 Adult Spondylolysis and Isthmic Spondylolisthesis

William D. Long III and Peter G. Whang

18.1 Case Example

A 20-year-old male collegiate athlete presents with progressive lumbar pain with occasional numbness and tingling in his buttocks that radiates into his thighs. His pain is worsened with increased physical activity and prolonged standing. Physical examination reveals a palpable step-off at the lumbosacral junction and increased sacral inclination. He exhibits normal strength in the lower extremities and experiences mild pain with lumbar hyperextension. A lateral radiograph of the lumbar spine reveals anterior translation of L5 on S1 (Fig. 18.1). After attempting a number of conservative measures including a prolonged course of physical therapy designed to strengthen the core muscle groups, nonsteroidal anti-inflammatories, and bilateral foraminal steroid injections, he elected to undergo surgical intervention. An anterior lumbar interbody fusion was performed at L5–S1 followed concurrently with an instrumented L5–S1 posterior spinal fusion (Figs. 18.2 and 18.3), with full return to athletics after 6 months.

W.D. Long III, M.D. • P.G. Whang, M.D. (✉)
Department of Orthopaedics and Rehabilitation,
Yale University School of Medicine,
P.O. BOX 208071, New Haven, CT 06520-8071, USA
e-mail: pgwhang@yahoo.com

18.2 Pathology/Pathophysiology

The development of spondylolysis typically occurs after an individual has begun to bear weight; these lesions have not been observed in children who are unable to walk, suggesting that upright posture and bipedal ambulation play a role in its pathogenesis [1]. It has been postulated that multiple anatomic and mechanical factors may act in concert to bring about spondylolytic defects. The most commonly affected vertebral segment is the lumbosacral junction which accounts for approximately 90 % of adolescent cases [2, 3]. Axial compression is primarily absorbed by the intervertebral disc and vertebral body, whereas shear stresses are resisted by the disc and posterior bony elements [3]. Biomechanical studies have confirmed that the load on the posterior arch increases significantly from L1 to L5 during dynamic flexion and extension of the spine, with the greatest stress concentrated at the level of the L5 pars [4]. Thus, the application of significant forces to the lumbosacral spine prior to the ossification of the posterior elements in the setting of an elastic intervertebral disc predisposes the pars to fatigue and fracture [5]. This has been corroborated by subjecting pediatric cadaveric specimens to cyclic shear loading which gave rise to fractures across the isthmus [6, 7]. Based on these findings, it is likely that the shear forces generated by sacral inclination, lumbar lordosis, and a bipedal gait contribute to the pathogenesis of spondylolisthesis [8]. Bilateral pars defects weaken the tension band

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_18, © Springer-Verlag Berlin Heidelberg 2014

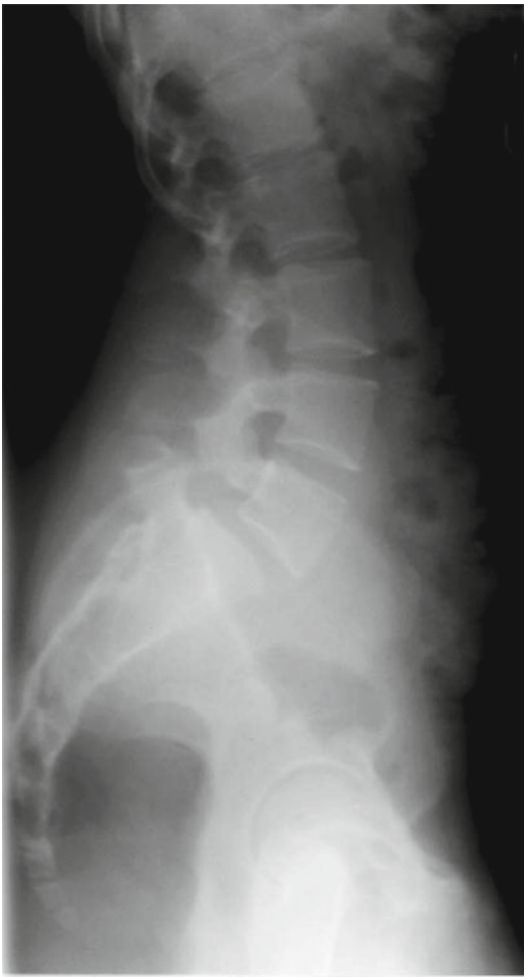


Fig. 18.1 Lateral radiograph of the lumbar spine showing a grade II spondylolisthesis

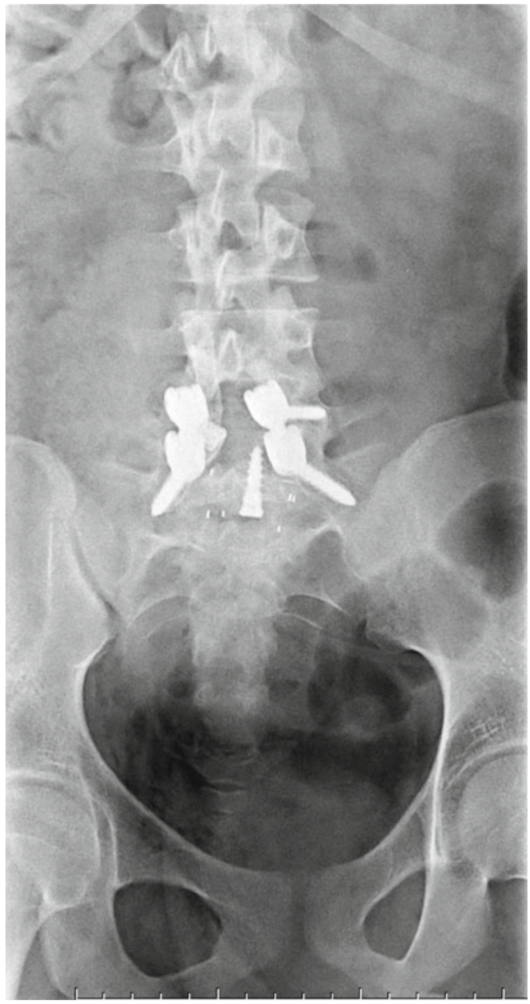


Fig. 18.2 Anterior-posterior radiograph of the lumbar spine showing the instrumentation construct of the spinal fusion

effect of the posterior elements and may allow for the ventral migration of the anterior column characteristic of a spondylolisthesis deformity [9].

Fredrickson and colleagues reported an incidence of lumbar spondylolysis of 4.4 % among 500 first grade students which increased to 6 % once they reached adulthood [10, 11]. The male-to-female ratio is approximately 2:1, and there is some ethnic variability such that it has been described in 2 % of the black population but may be present in up to 54 % of Eskimos [12]. Roche and Rowe also noted similar gender and racial disparities in their examination of 4,200 cadavers and calculated the following frequencies of spondylolysis: white men, 6.4 %; black men, 2.8 %; white women, 2.3 %; and black women, 1.1 % [13]. In Japan, lumbar spondylolysis was identified in 5.9 % of all individuals but this condition was evident in 20–30 % of professional athletes competing in rugby, judo, soccer, and baseball [14]. This association between elite athletes to show a higher incidence has been demonstrated in other cultures as well, with an incidence ranging from 13 to 27 % of throwing athletes, rowers, gymnasts, and weight lifters from Spain [15] as well as 15 % of American college football players [16].

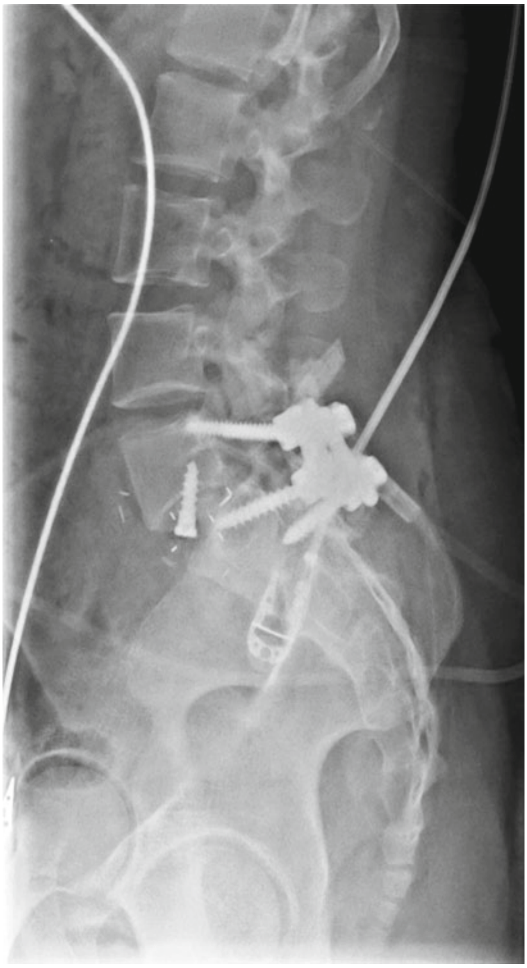


Fig. 18.3 Lateral radiograph of the lumbar spine showing the instrumentation construct of the spinal fusion

A 45-year natural history study of spondylolysis showed that adults with unilateral defects tended not to degenerate and develop spondylolisthesis [11]. Subjects with bilateral defects demonstrated variable progression to listhesis, with half showing no evidence of spondylolisthesis and half slipping a mean of 24 %. The authors found the risk of developing listhesis in asymptomatic patients with bilateral defects to be 5 %, a risk that decreased with age. Others have estimated slip progression in symptomatic adults at 20 %, with disc degeneration at the level of the spondylolisthesis in all patients [17]. Progression is more common in adults compared to adolescents, with translation more than 10 mm occurring in less than 5 % of patients [18].

Wiltse has recognized five types of spondylolisthesis: dysplastic, isthmic, degenerative, post traumatic, and pathological (Table 18.1) [1]. A sixth type has been added to account for mechanical instability occurring in 20 % of spastic children who have undergone laminectomy for rhizotomy [19]. Meyerding described a grading system designed to describe the percentage of anterior displacement of the body of L5 in relation to the sacrum (Table 18.2) [20]. Grade I shows 0–25 % displacement, grade II 26–50 %, grade III 51–75 %, grade IV 76–100 %, and grade V or spondyloptosis for more than 100 % translation. Low-grade lesions are described with less than 50 % anterolisthesis, whereas high-grade lesions demonstrate slips of greater than 50 %.

18.3 History and Physical Examination

Patients suffering from spondylolysis and isthmic spondylolisthesis typically present with mechanical midline lumbar pain. The age at presentation can vary from adolescence through adulthood. Younger patients frequently report pain aggravated by athletics or prolonged standing. Postural spinal deformity and radiculopathy are more commonly seen in high-grade spondylolisthesis, with bowel and bladder dysfunction described in severe cases of listhesis. Palpation of the spinous process may reveal a step-off with the L5 process more prominent than the L4 spinous process. Traumatic etiologies may reveal focal tenderness. Hamstring tightness is found in 80 % of symptomatic patients. The Phalen-Dickson sign is described as a wide-based posture with marked flexion of the hips and knees, a crouching gait with a stiff lumbar spine in significant lordosis, a posteriorly tilted pelvis, a vertical sacrum, and the presence of an abdominal crease [21]. Increased pain with lumbar

Table 18.1 Wiltse classification of spondylolisthesis

Type	Name	Description
I	Dysplastic	Congenital abnormalities of the L5 arch or sacrum
II	Isthmic	Lesions of the pars interarticularis
III	Degenerative	Results from chronic intersegmental disease
IV	Post-traumatic	Fractures located within in the spine other than the pars
V	Pathologic	Systemic or local bone disease

Table 18.2 Meyerding grading system of spondylolisthesis

Grade	Percentage of displacement (%)
I	1–25
II	26–50
III	51–75
IV	76–100
V (spondyloptosis)	>100

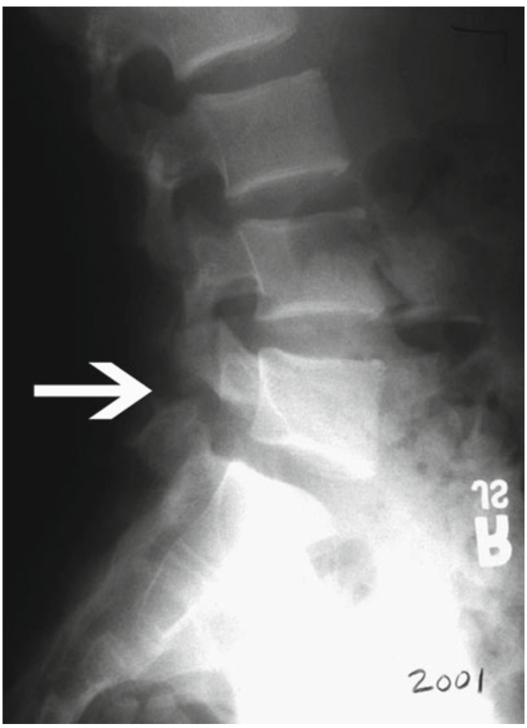


Fig. 18.4 Lateral radiograph of the lumbar spine showing a defect (*arrow*) of the pars interarticularis

hyperextension is a common clinical finding that can be elicited when performing the single leg hyperextension test. As with all patients with possible spinal pathology, a complete motor, sensory, and reflex evaluation should be performed routinely.

18.4 Diagnostic Imaging

Initial radiographic evaluation of patients suspected to have spondylolysis begins with plain x-rays. A lateral lumbosacral spine radiograph can reveal the presence of spondylolysis (Fig. 18.4) and allow for grading of spondylolisthesis as well as measurement of the slip angle, the lumbar index, and the pelvic incidence. Further imaging with AP and 30° oblique lateral/caudal tilt views should also be obtained to check for scoliosis, spina bifida occulta, a trapezoidal L5 vertebra, sacral doming, and loss of disc height [8]. The "Scotty Dog" phenomenon (Fig. 18.5), best seen on oblique views, can demonstrate the pars defect at the neck of the contrived canine. Flexion and extension sagittal views are useful in evaluating dynamic motion at the slipped segment in a patient with a known pars defect. Computed tomography (CT) can be utilized to demonstrate in fine detail the three-dimensional bony architecture and is highly sensitive for detecting spondylolysis (Fig. 18.6). It can be performed serially to monitor for healing following surgical repair or immobilization. Magnetic resonance imaging (MRI) is less useful in detecting clefts within the pars interarticularis compared with CT, but offers improved soft tissue detail and can be useful in evaluating degenerated or herniated intervertebral discs. Axial MRI views can also demonstrate the presence of foraminal stenosis at the level of the listhesis, correlating with nerve root impingement and radicular symptoms. In adolescents with a high suspicion for spondylolysis with negative radiographs and CT, Tc-99m bone scans can be incorporated to identify uptake at the pars, identifying stress reaction or subacute injury prior to fracture (Fig. 18.7).

Fig. 18.5 Oblique radiograph of the lumbar spine demonstrating the "Scotty Dog" and pars defect (*arrow*) through the neck of the canine

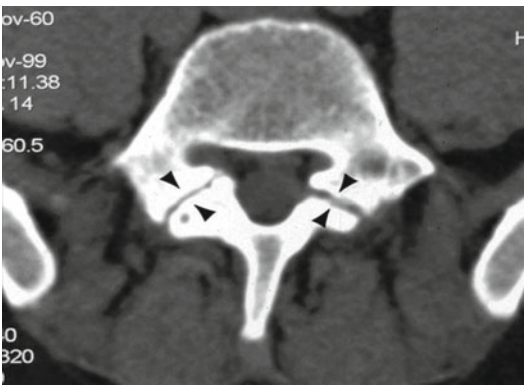


Fig. 18.6 Axial view of a CT scan of the lumbar spine demonstrating bilateral pars defects (*arrowheads*)

18.5 Treatment

Therapeutic options for spondylolysis and isthmic spondylolisthesis depend on patient age, remaining growth, and presence and severity of symptoms. Surgery is indicated for progression of deformity, high-grade listhesis with sagittal imbalance, neurologic deficit, persistent lumbago despite an exhaustive course of conservative therapy, and radicular pain with associated nerve root compression [22]. The majority of patients will improve with conservative measures; however, patients with persistent symptoms after 6 months of therapy should be considered for surgical intervention. Although the principles for surgery in adults and children/adolescents are similar, a number of considerations must be taken into account: Adults are more likely to require direct neural compression as adolescents frequently experience resolution of radiculopathy with fusion of the hypermobile segment alone; adults have more risk factors for pseudarthrosis (smoking, steroid use, comorbid disease), arguing for circumferential fusion as opposed to posterior-alone approaches that may be successful in children; risk of progression is higher in children and adolescents, so skeletally immature patients with high-grade slips and evidence of progression should be considered for surgery more commonly [22].

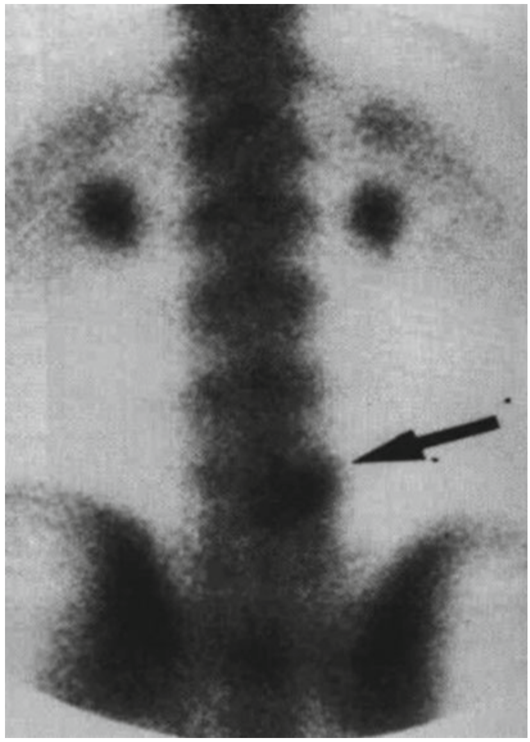


Fig. 18.7 Bone scan image showing increased uptake (*arrow*) in the region of the pars, sensitive to spondylolysis

18.5.1 Nonoperative Treatment

Treatment for patients with spondylolysis is commonly nonoperative, rather focusing on activity modification, rest, and physical therapy

[19]. Strengthening of the abdominal core, trunk stabilization, and hamstring stretching should be the focus of a physical therapy program. Nonsteroidal anti-inflammatories, muscle relaxants, and narcotic medications should be used judiciously. Functional immobilization in adolescents to treat spondylolysis with a thoracolumbar orthosis for 3–6 months has been shown to be effective for the majority of unilateral lesion and half of acute bilateral lesions [23, 24]. Furthermore, bracing has been shown to be effective in alleviating lumbago in up to 80 % of patients with a grade I spondylolisthesis [25]. More than 75 % of adults with grade I or II spondylolisthesis demonstrate improvement with antilordotic bracing and activity modification for 3–6 months [26]. There has been no clear evidence showing the benefits of heat, ultrasound, or massage for the treatment of spondylolisthesis [8]. Symptomatic high-grade spondylolisthesis in children and adolescents runs a high risk of progression and low likelihood of symptom relief, prompting most to forgo conservative treatment and elect for surgical intervention instead. Adults with high-grade listhesis frequently reach a stable anatomic alignment without progression, and some can be successfully treated with physical therapy and nerve root injections if radicular symptoms exist.

18.5.2 Direct Pars Repair

Very rarely indicated in adults, direct fixation of the pars has a role for treating adolescents with spondylolysis and back pain that has been localized to the pars with lidocaine injections [27]. It is most often recommended for patients that have minimal spondylolisthesis, no evidence of radiculopathy or neural deficit, and a normal intervertebral disc on MRI. The most common technique for repair utilizes pedicle screws and sublaminar hooks connected with rods to allow for direct compression across the defect [28, 29]. This requires debridement of the nonunion site and bone grafting concomitantly with a construct that provides stability and compression, without violating the facet joint capsule.

18.5.3 Decompression

Compression of the L5 nerve root at the neural foramen is commonly seen with anterolisthesis of L5 on S1. Further compression can be caused from hypertrophic reparative tissue at the site of the pars defect, as well as from an anterior direction due to bulging degenerated discs. Elderly comorbid patients with radiculopathy and low-grade isthmic spondylolisthesis without evidence of instability on dynamic imaging may benefit from minimizing the extent of surgery with decompression alone. This is never indicated in the pediatric and adolescent population, as this intervention would create iatrogenic instability with the potential to cause listhesis progression [30].

18.5.4 Posterolateral Fusion

The gold standard for grade I and grade II spondylolisthesis stabilization has been the in situ posterolateral L5–S1 fusion. In adolescents, fusion rates greater than 90 % with excellent outcomes have been the norm, whereas the adult literature has shown more variability, ranging from 33 to 100 % fusion rates [22, 31, 32]. Decompression is typically added if significant radiculopathy or neurologic compromise is present, although most adolescents will have resolution of dynamic root irritation with fusion alone. Extension of the fusion construct proximally to include L4 can be considered if a high-grade slip is present, if there is instability of the L4–L5 segment, or if the L5 transverse processes are too small of a mass for acceptable fusion [22]. Long-term follow-up for high-grade slips has found good function and pain relief with posterolateral in situ fusion in adults [33, 34]. Fusion rates in children and adolescents, however, have been lower prompting some to argue for circumferential fusion [35].

18.5.5 Anterior Lumbar Interbody Fusion (ALIF)

Although infrequently performed, stand-alone ALIF for the treatment of isthmic spondylolisthesis has been described, with favorable outcomes.

A retrospective comparison of 20 adults undergoing ALIF with 20 adults who underwent instrumented posterolateral fusion found no difference in clinical outcomes between the groups [36]. This technique runs the risk of destabilizing the posterior column as a result of the pars defect, however, possibly leading to a progression of the slip. For this reason, supplementation with posterolateral fusion and instrumentation should be performed.

18.5.6 Circumferential Fusion

Adult patients with low-grade isthmic spondylolisthesis most reliably achieve fusion and a successful clinical outcome with combined anterior and posterior approaches [37]. This meta-analysis of 35 studies found a fusion rate of 98 % for circumferential stabilization versus 83 % for posterolateral fusion and 74 % for anterior fusion alone. The literature on this topic, however, is limited to mostly retrospective reviews with no well-designed prospective-randomized trials, suggesting that the best treatment option remains unclear [38]. In most cases of low-grade isthmic spondylolisthesis, in situ instrumented posterolateral fusion yields successful clinical outcomes, particularly in the adolescent population. A number of limited studies, however, have suggested that a circumferential fusion overall yields higher fusion rates and improved outcomes [39–41]. Some advocate consideration of a combined anterior-posterior approach for patients with numerous risk factors for pseudarthrosis, and those with large or hypermobile discs that may degenerate over time with a posterior-alone approach [22]. Patients with high-grade spondylolisthesis frequently benefit from the stability that an anterior construct provides, in addition to providing a greater surface area available for interbody fusion and preventing further progression of the slip. A number of methods have been described, including staged anterior and posterior approaches, the transforaminal lumbar interbody approach, and the transsacral approach [8, 22].

18.5.7 Posterior Reduction

The goal of treatment for patients with high-grade spondylolisthesis with significant lumbosacral malalignment due to kyphosis is to restore the sagittal balance of the spinopelvic complex. Since these patients are typically symptomatic at an early age, most patients undergo surgery during the adolescent years. In adults with high-grade spondylolisthesis, the overall sagittal balance is frequently maintained and the deformity is less flexible, negating the need for a reduction. When performed, this maneuver attempts to improve the sagittal alignment by reducing the slip angle and allowing for fusion by reducing the shear force (converting it to compressive force) across the end plates and preventing further progression of deformity. Despite the theoretical benefits that a posterior reduction would offer, a review of the available data found no evidence that reduction improved outcomes compared with fusion alone [42]. Nonetheless, a number of studies have demonstrated good results with reduction and fusion of high-grade slips [43–45]. If a reduction is performed, a circumferential fusion is generally recommended [8].

18.5.8 Spondyloptosis

Treatment of the most severe aspect of the spectrum is challenging. One option for correction of a spondyloptosis deformity includes a transsacral approach first described by Bohlman [46], utilizing a fibular dowel graft across the L5–S1 disc for an in situ fusion without reduction. Gaines devised a more invasive procedure that addresses the lumbosacral deformity via a combined anterior and posterior approach that requires resection of L5 and reduction of L4 onto the sacrum, thereby shortening the spine [47]. This has been associated with a high rate of neurologic complication and is reserved for only the most severe cases.

Questions

1. Which of the following factors is NOT thought to contribute to the development of pars defects?

(a) Bipedal ambulation
(b) Sacral inclination
(c) Lumbar lordosis
(d) Premature birth
(e) Shear forces

Preferred reponse, (d). The results of cadaveric testing have suggested that the shear forces that arise as a consequence of sacral inclination, lumbar lordosis, and a bipedal ambulation may predispose certain individuals to the formation of pars fractures. These abnormalities have never been identified in utero and have not been shown to be associated with prematurity.

2. According to the Meyerding classification system, all of the following are types of spondylolytic lesions EXCEPT for
(a) Congenital
(b) Isthmic
(c) Degenerative
(d) Post traumatic
(e) Pathologic

Preferred response, (a). Based on the Meyerding paradigm, congenital abnormalities of the posterior arch are included in the "dysplastic" category of spondylolytic defects.

3. Which of these patients is most likely to develop a spondylolytic spondylolisthesis?
(a) 12-year-old male with bilateral stress reactions evident on bone scan
(b) 77-year-old female with a unilateral pars defect and diffuse spondylosis
(c) 17-year-old asymptomatic female who is found to have bilateral spondylolysis during a routine physical examination
(d) 30-year-old male with unilateral spondylolysis and moderate disc degeneration
(e) 45-year-old male with bilateral spondylolysis who presents with chronic low back pain

Preferred response, (e). Progression of spondylolytic slips is most commonly seen in adults rather than adolescents and generally does not occur in patients who are asymptomatic or those with unilateral defects.

4. Which of the following clinical findings is not a relative contraindication for a direct pars repair?
(a) Right lower extremity sciatica
(b) Significant low back pain
(c) Grade II spondylolisthesis
(d) Moderate disc degeneration evident on MRI
(e) No relief with lidocaine injections into the pars

Preferred response, (b). Operative intervention including pars repair may be considered for certain individuals with spondylolysis who complain of debilitating axial back pain. However, this procedure is not appropriate for those with compression of the neural elements resulting in sciatic pain, sensory deficits, or weakness (a). Spondylolysis with frank instability or advanced disc degeneration that requires surgery is most often addressed with some type of fusion (c, d). Finally, failure to obtain any pain relief with pars injections raises the possibility that there may be other etiologies contributing to the patient's symptoms other than the spondylolytic lesions (e).

References

1. Wiltse LL, American Academy of Orthopedic Surgeons (1969) Spondylolisthesis: classification and etiology. In: Symposium on the spine. Mosby, St. Louis, pp 143–167
2. Morita T, Ikata T, Katoh S, Miyoke R (1995) Lumbar spondylolysis in children and adolescents. J Bone Joint Surg Br 77-B:620–625
3. Tsirikos AI, Garrido EG (2010) Spondylolysis and spondylolisthesis in children and adolescents. J Bone Joint Surg Br 92-B:751–759
4. Dietrich M, Kurowski P (1985) The importance of mechanical factors in the etiology of spondylolysis: a model analysis of loads and stresses in human lumbar spine. Spine 10:532–542
5. Wiltse LL, Jackson DW (1976) Treatment of spondylolisthesis and spondylolysis in children. Clin Orthop 117:92–100
6. Cyron BM, Hutton WC (1978) The fatigue strength of the lumbar neural arch in spondylolysis. J Bone Joint Surg Br 60-B:234–238
7. Troup JD (1976) Mechanical factors in spondylolisthesis and spondylolysis. Clin Orthop 117:59–67
8. Jones TR, Rao RD (2009) Adult isthmic spondylolisthesis. J Am Acad Orthop Surg 17:609–617
9. Ikata T, Miyake R, Katoh S, Morita T, Murase M (1996) Pathogenesis of sports-related spondylolisthesis in adolescents: radiographic and magnetic resonance imaging study. Am J Sports Med 24:94–98
10. Fredrickson BE, Baker D, McHolick WJ, Yuan HA, Lubicky JP (1984) The natural history of spondylolysis and spondylolisthesis. J Bone Joint Surg Am 66-A:699–707

11. Beutler WJ, Fredrickson BE, Murtland A et al (2003) The natural history of spondylolysis and spondylolisthesis: 45-year follow-up evaluation. Spine 28:1027–1035
12. Simper LB (1986) Spondylolysis in Eskimo skeletons. Acta Orthop Scand 57:78–80
13. Roche MB, Rowe GG (1951) The incidence of separate neural arch and coincident bone variations; a survey of 4200 skeletons. Anat Rec 109:233–252
14. Sakai T, Sairyo K, Suzue N, Kosaka H, Yasui N (2010) Incidence and etiology of lumbar spondylolysis: review of the literature. J Orthop Sci 15:281–288
15. Soler T, Calderon C (2000) The prevalence of spondylolysis in the Spanish elite athlete. Am J Sports Med 28:57–62
16. McCarroll JR, Miller JM, Ritter MA (1986) Lumbar spondylolysis and spondylolisthesis in college football players: a prospective study. Am J Sports Med 14:404–406
17. Floman Y (2000) Progression of lumbosacral isthmic spondylolisthesis in adults. Spine 25:342–347
18. Saraste H (1987) Long-term clinical and radiological follow-up of spondylolysis and spondylolisthesis. J Pediatr Orthop 7:631–638
19. Pizzutillo P, Hummer CS (1989) Nonoperative treatment for painful adolescent spondylolysis or spondylolisthesis. J Pediatr Orthop 9:538–540
20. Meyerding HW (1941) Low backache and sciatic pain associated with spondylolisthesis and protruded intervertebral disc: incidence, significance, and treatment. J Bone Joint Surg Am 23:461–470
21. Phalen GS, Dickson JA (1961) Spondylolisthesis and tight hamstrings. J Bone Joint Surg Am 43-A: 505–512
22. Agabegi SS, Fischgrund JS (2010) Contemporary management of isthmic spondylolisthesis: pediatric and adult. Spine J 10:530–543
23. Sairyo K, Katoh S, Takata Y et al (2006) MRI signal changes of the pedicle as an indicator for early diagnosis of spondylolysis in children and adolescents: a clinical and biomechanical study. Spine 31:206–211
24. Sys J, Michielsen J, Bracke P, Martens M, Verstreken J (2001) Nonoperative treatment of active spondylolysis in elite athletes with normal x-ray findings: literature review and results of conservative treatment. Eur Spine J 10:498–504
25. Steiner ME, Micheli LJ (1985) Treatment of symptomatic spondylolysis and spondylolisthesis with the modified Boston brace. Spine 10:937–943
26. d'Hemecourt PA, Zurakowski D, Kriemler S, Micheli LJ (2002) Spondylolysis: returning the athlete to sports participation with brace treatment. Orthopedics 25:653–657
27. Suh PB, Esses SI, Kostuik JP (1991) Repair of the pars interarticularis defect. The prognostic value of pars infiltration. Spine 16(8 suppl):S445–S448
28. Tokuhashi Y, Matsuzaki H (1996) Repair of defects in spondylolysis by segmental pedicle screw fixation. Spine 21:2041–2045
29. Gillet P, Petit M (1999) Direct repair of spondylolysis without spondylolisthesis using a rod-screw construct and a bone grafting of the pars defect. Spine 24:1252–1256
30. Amuso SJ, Neff RS, Coulson DB, Laing PG (1970) The surgical treatment of spondylolisthesis by posterior element resection: a long-term follow-up study. J Bone Joint Surg Am 52:529–536
31. Pizzutillo PD, Mirenda W, MacEwen GD (1986) Posterolateral fusion for spondylolisthesis in adolescence. J Pediatr Orthop 6:311–316
32. Lenke LG, Bridwell KH, Bullis D et al (1992) Results of an *in situ* fusion for isthmic spondylolisthesis. J Spinal Disord 5:433–442
33. Johnson JR, Kirwin EO (1983) The long-term results of fusion *in situ* for severe spondylolisthesis. J Bone Joint Surg Br 65:43–46
34. Seitsalo S, Osterman K, Hyvarinen J et al (1990) Severe spondylolisthesis in children and adolescents. A long-term review of fusion *in situ*. J Bone Joint Surg Br 72:259–265
35. Molinari RW, Bridwell KH, Lenke LG et al (1999) Complications in the surgical treatment of pediatric high-grade, isthmic dysplastic spondylolisthesis. A comparison of three surgical approaches. Spine 24:1701–1711
36. Kim NH, Lee JW (1999) Anterior interbody fusion versus posterolateral fusion with transpedicular fixation for isthmic spondylolisthesis in adults: a comparison of clinical results. Spine 24:812–816
37. Kwon BK, Hilibrand AS, Malloy K et al (2005) A critical analysis of the literature regarding surgical approach and outcome for adult low-grade isthmic spondylolisthesis. J Spinal Disord Tech 18(Suppl):S30–S40
38. Jacobs WC, Vreeling A, De Kleuver M (2006) Fusion for low-grade adult isthmic spondylolisthesis: a systematic review of the literature. Eur Spine J 15:391–402
39. Swan J, Hurwitz E, Malek F et al (2006) Surgical treatment for unstable low-grade isthmic spondylolisthesis in adults: a prospective controlled study of posterior instrumented fusion compared with combined anterior-posterior fusion. Spine J 6:606–614
40. Ekman P, Moller H, Tullberg T et al (2007) Posterior lumbar interbody fusion versus posterolateral fusion in adult isthmic spondylolisthesis. Spine 32:2178–2183
41. Videbaek TS, Christensen FB, Soegaard R et al (2006) Circumferential fusion improves outcomes in comparison with instrumented posterolateral fusion: long-term results of a randomized clinical trial. Spine 31:2875–2880
42. Transfeldt EE, Mehbod AA (2007) Evidence-based medicine analysis of isthmic spondylolisthesis treatment including reduction versus fusion *in situ* for high-grade slips. Spine 32:S126–S129
43. Ruf M, Koch H, Melcher RP, Harms J (2006) Anatomic reduction and monosegmental fusion in high-grade developmental spondylolisthesis. Spine 31:269–274
44. Fabris DA, Costatini S, Nena U (1996) Surgical treatment of severe L5-S1 spondylolisthesis in children and adolescents. Results of intraoperative reduction, posterior interbody fusion, and segmental pedicle fixation. Spine 21:728–733

45. Smith JA, Deviren V, Berven S et al (2001) Clinical outcome of trans-sacral interbody fusion after partial reduction for high-grade L5-S1 spondylolisthesis. Spine 26:2227–2234
46. Bohlman HH, Cook SS (1981) One-stage decompression and posterolateral and interbody fusion for lumbosacral spondyloptosis through a posterior approach. Report of two cases. J Bone Joint Surg Am 64:415–418
47. Gaines RW, Nichols WK (1985) Treatment of spondyloptosis by two stage L5 vertebrectomy and reduction of L4 onto S1. Spine 10:680–686

Degenerative Disk Disease with and Without Facet Arthritis

19

Kern Singh, Jonathan A. Hoskins, Steven J. Fineberg, and Matthew Oglesby

19.1 Introduction

Degenerative disk disease (DDD), also known as intervertebral disk disease (IVD), is a major cause of musculoskeletal disability in humans [1, 3, 5]. Degenerative disk disease is a common cause of low back pain (LBP); however, the exact relationship between the two remains uncertain [25, 29].

19.2 Case Example

A 44-year-old man presents with a history of chronic low back pain worsened with prolonged sitting and forward flexion. The patient denies any lower extremity dysesthesias. The patient has been treated with physical therapy (core-strengthening and McKenzie back extension exercises) for 3 months. T2-weighted axial and sagittal MRI scans (Figs. 19.1 and 19.2) demonstrate a decreased signal intensity and disk height loss at the L4–5 and L5–S1 levels.

19.3 Normal Intervertebral Disk and Facet Anatomy

The functional spinal unit (FSU) consists of an anterior intervertebral disk and two posterior facet joints. The relationship of this tri-joint complex allows for motion and provides resistance to compressive forces exerted across the vertebral column. While motion occurs at all three components, the intervertebral disk provides the most resistance to compression.

The intervertebral disk lies between the vertebral bodies. It serves as a load-bearing structure comprised of a fibrous outer layer rich in collagen and an inner layer rich in proteoglycans. The outer layer, the annulus fibrosis (AF), provides tensile strength, whereas the hydrostatic properties of the inner layer, the nucleus pulposus (NP), provide resistance against compression. Proper interactions between these two substructures are necessary in order for the disk to absorb and disperse the normal loading forces experienced by the spine [15, 22].

Facet joints are diarthrotic joints with articulations lubricated by synovial fluid. Together with posterior structures such as the pedicle, lamina, and transverse and spinous process, the facet joint contributes to the essential posterior stabilization of the FSU. In addition, the posterior structures act as anchors for the paraspinal musculature further stabilizing the FSU.

K. Singh, M.D. (✉) • J.A. Hoskins, M.D.
S.J. Fineberg, M.D. • M. Oglesby, B.A.
Department of Orthopedic Surgery,
Rush University Medical Center,
1611 W Harrison St., Suite 300,
Chicago, IL 60612, USA
e-mail: kern.singh@rushortho.com;
dr.hoskins@gmail.com;
steve.fineberg@rushortho.com;
matt.oglesby@rushortho.com

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_19, © Springer-Verlag Berlin Heidelberg 2014

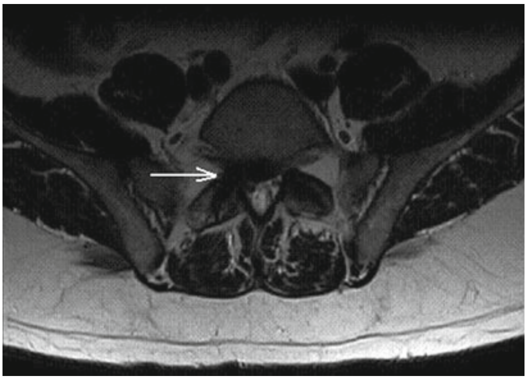


Fig. 19.1 A T2-weighted axial MRI of the lumbar spine demonstrating a posterolateral disk herniation (*arrow*) at the L5–S1 segment completely obliterating the traversing right S1 nerve sleeve

Fig. 19.2 A T2-weighted sagittal MRI of the lumbar spine demonstrating disk disease and moderate narrowing of the spinal canal (*arrows*) at L4–5 and a broad-based disk protrusion which appears to displace the right S1 nerve sleeve posteriorly at L5–S1

19.4 Pathology/Pathophysiology

More than 25 years ago, Kirkaldy-Willis et al. presented the concept of a cascade of spinal motion segment degeneration invoking progressive wear of the IVD and facet joints [14]. The authors emphasized the independence of the disk and facet joints for normal spinal function and described how derangement or injury to either of these articulations leads to abnormal forces and impairment of the other, the so-called tripod effect. While this concept highlighted biomechanical disturbances associated with degeneration, over the decades since, we have come to appreciate that the pathophysiology of DDD is most likely a multifactorial process (Table 19.1).

Table 19.1 Factors associated with disk degeneration

Age
Family history
Smoking
Vibration (prolonged driving)
Heavy repetitive loading of the spine
Diabetes

Degeneration of the spine is an inevitable consequence of aging. Miller et al. reported an increase in disk degeneration from 16 % at age 20 to ~98 % at 70 years based on macroscopic disk degeneration grades of 600 specimens. Interestingly, the authors noted that lumbar disk degeneration was already present in 11- to 19-year-old males and 10 years later in females [17].

Degeneration of the intervertebral disks and facet joints due to repetitive biomechanical loading of the spine has also been suggested. Kirkaldy-Willis et al. postulated that injury or repetitive strain to the facet joint is a cardinal event in the spinal degenerative sequence [14]. Butler et al. suggested that disk degeneration likely predates facet arthrosis based on computed tomography (CT) and magnetic resonance imaging (MRI) study [6]. The authors noted that in 68 patients (330 disks/390 facet joints), there were 144 degenerated disks and 41 levels with facet osteoarthritis. Disk degeneration without facet osteoarthritis was found at 108 levels, whereas all but one of 41 levels with facet degeneration also had disk degeneration [6].

Furthermore, genetic predisposition has emerged as perhaps the strongest factor leading to symptomatic degenerative disk disease. Disk degeneration was found to be much more likely among family members of patients requiring lumbar surgery than in the general population [16]. Siblings of patients with disk degeneration were found to have a higher prevalence of both disk degeneration and osteoarthritis than the general population [4]. Finally, a study of twins found a strong link between heredity and cervical and lumbar disk degeneration [23].

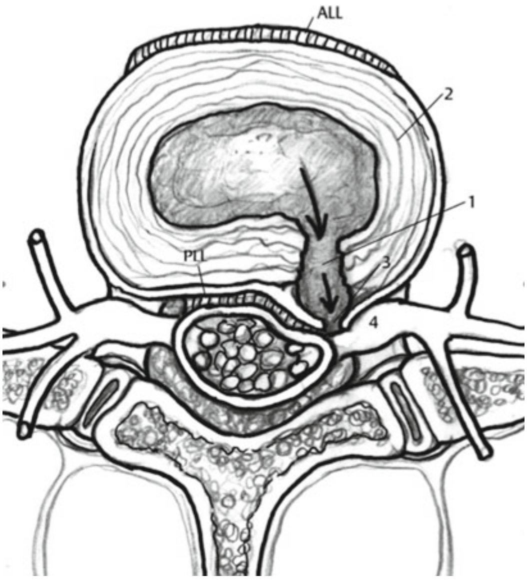


Fig. 19.3 Biology of disk disruption. *1* Nucleus pulposus, *2* annulus, *3* fissure/anular defect, and *4* dorsal root ganglion. *ALL* anterior longitudinal ligament, *PLL* posterior longitudinal ligament

As aging occurs, there is typically a decrease in overall proteoglycan content within the NP, which leads to an inability to maintain disk hydration. This in turn leads to a loss in disk height. In addition, the inner and outer AF takes on a fibrocartilaginous character, making the layers indistinguishable. As this process occurs, the weakened lamellar structure of the outer AF becomes less resilient to applied forces, and it may develop defects that can predispose the disk to herniations of the inner disk material and to disk bulging (Fig. 19.3). The loss of biomechanical integrity within the AF may transfer force to and overstress the facets, contributing to further degeneration [8, 9, 18, 21]. In addition, the superficial layers of the annulus are richly innervated with small nerve fibers (A delta and C fibers) that have been implicated in the generation of pain originating from the intervertebral disk [11, 19, 20]. Stimulation of these fibers has also been implicated in the generation of LBP.

Low back pain secondary to DDD typically affects individuals between 30 and 50 years of age [27]. Most commonly, the L4–5 and L5–S1 levels are affected, although more cephalad levels can also be involved in older populations [12, 24]. The natural history of LBP is that with conservative management, 50–60 % of individuals will experience resolution of symptoms within 1 week and 90 % recover within 3 months.

19.5 History and Physical Exam

The clinical presentation is key to the diagnosis of lumbar degenerative disk diseases. Patients most commonly present with discogenic LBP that is typically axial in nature but may involve the buttocks and some leg symptoms from indirect and direct foraminal stenosis. Classic discogenic pain is exacerbated by activities that load the disk, such as sitting, arising from a seated position, awaking in the morning, lumbar flexion with and without rotation/twisting, lifting, vibration (i.e., riding in a car), coughing, sneezing, laughing, and the Valsalva maneuver.

19.6 Physical Examination

Physical examination is an important adjunct to the history in determining a discogenic etiology of symptoms. Patients may prefer to stand or sit in a reclining position since these positions usually decrease intradiscal pressures.

Range of motion should be evaluated. Pain on flexion of the lumbar spine suggests discogenic pain, while pain on lumbar extension suggests facet disease.

Discogenic stress maneuvers usually reproduce LBP and buttock symptoms in the patient. These maneuvers include sustained hip flexion which is performed with the patient supine raising the patient's extended lower extremities to ~60° in relation to the examination table. Ask the patient to hold the extremities in that position and release. Ask the patient regarding reproduction of LBP and/or buttock pain. Then lower the extremities successively ~15°, and at each point note, the reproduction and intensity of pain. The test is positive if the patient complains of LBP and/or buttock pain of increasing intensity as the extremities are lowered at successive angles.

Motor, sensory, and reflex function should be assessed to determine the affected nerve root level. To help determine whether the problem is root pathology or a focal neuropathy, two muscles should be tested with reflexes elicited, representing each lumbar root.

19.7 Differential Diagnosis

Other disorders may elicit LBP and should be considered (Table 19.2). One rare but serious condition is cauda equina syndrome, a spinal emergency caused by compression of the nerve roots below the level of the spinal cord.

19.8 Investigations/Diagnostic Imaging

Radiographs typically demonstrate loss of disk height and, when severe, sclerosis of the end plates and/or facet hypertrophy.

MRI is the gold standard diagnostic tool for the evaluation of lumbar disk herniation (Fig. 19.3). In addition, Aprill and Bogduk have suggested that a high-intensity zone (HIZ) observed on MRI may be a specific marker of a painful disk [2] (Fig. 19.4). However, Carragee et al. determined that although a HIZ was more prevalent in patients with symptomatic LBP, there was also a high prevalence in asymptomatic patients; thus, the presence of a HIZ was not a reliable indicator for LBP [30].

Discography with or without CT remains an important, albeit controversial tool in establishing discogenic low back pain [10, 26, 28]. The discogram provides information consisting of four components: the morphology of the disk, disk pressure, volume of fluid injected, and the subjective pain response. The reproduction of discogenic pain is essential to confirm a potential intradiscal etiology for the patient's pain.

19.9 Treatment

19.9.1 Nonoperative Treatment

Although low back pain and radiculopathy are common causes of disability, nonsurgical management remains the cornerstone for treatment in this patient population. Conservative management is reasonable as the majority of these patients recover from their symptoms. Conservative management consists of observation, physical therapy (core-strengthening and McKenzie back extension exercises), nonsteroidal anti-inflammatory (NSAID) medications for pain, muscle relaxants, and epidural steroid injections in those patients with a concomitant radiculopathy.

Table 19.2 Differential diagnosis and red flags in the clinical presentation that require further investigation

Differential diagnosis
Muscle strain
Ligament/tendon injury
Hip joint pain
Stress reaction
Fibromyalgia
Red flags
Fracture
Tumor
Infection
Cauda equina syndrome

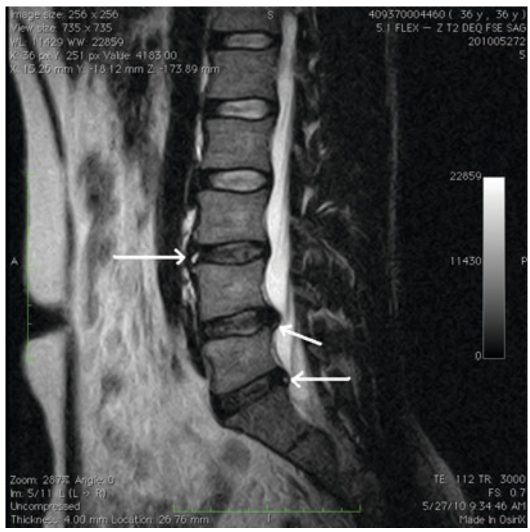


Fig. 19.4 A T2-weighted sagittal MRI scan of the lumbar spine demonstrates three high-intensity zones (*arrows*) visible at L3–4 (*anterior*) and L4–5 and L5–S1 (*posterior*)

19.9.2 Operative Treatment

Surgery is indicated in patients who have failed at least 3 months of conservative management, unremitting pain, or have a massive disk herniation demonstrated on MRI or exhibit progressive neurological symptoms.

19.9.3 Which Approach?

Several surgical options to treat discogenic back pain exist. The most common procedures include fusion and, more recently, artificial total disk replacement (TDR). The primary goal of fusion surgery involves two components. The first, decompression, involves removal of bone or disk material from around the compressed or irritated nerve root(s). The second, spinal fusion with or without instrumentation, involves using bone graft or cages to fuse one or more vertebrae and stop painful vertebral segment motion. Both components may be achieved using an anterior lumbar interbody fusion (ALIF), posterior lumbar interbody fusion (PLIF), or via transforaminal lumbar interbody fusion (TLIF) technique. In addition, these procedures can be performed using the traditional open approach or the minimally invasive approach. It is important to remember that posterior fusion alone without removal of the disk is much less likely to relieve discogenic pain.

The advantages of ALIF compared with other interbody fusion options include minimal or no posterior muscle dissection, especially when used as a stand-alone anterior procedure.

PLIF or TLIF obviate the need for an anterior approach. The TLIF procedure is a modification of PLIF. This procedure offers good exposure with decreased risk of neurological injury, particularly in repeat cases of spine surgery in which the presence of scar tissue makes PLIF very difficult. The procedure can be used to treat single-level and multilevel degenerative disease.

Total disk replacement (TDR) is a technology that offers an alternative to spinal fusion in patients with lumbar discogenic pain. This technology is purported to relieve disk pain while preserving motion of the treated spinal segment, reducing adjacent-segment degeneration.

The decision on what procedure or approach to utilize should be based on the discussion with the patient, the patient's pathology, and the surgeon's experience and comfort with the selected procedure.

19.9.4 Results of Surgery: PLIF, TLIF, and TDR

Early experience with the ALIF technique demonstrated good results and minimal complications. However, one study reported a 30 % nonunion rate, 22 % revision rate, 22 % complication rate, and 70 % fair or poor outcome at 3- to 6-year follow-up [7]. Humphreys et al. compared PLIF versus TLIF in 74 patients with DDD and central disk herniations. In that study, patients who underwent a PLIF procedure suffered far more complications, including four radiculopathies (compared with none in the TLIF patients) [13]. The latter demonstrates the smaller retraction of neural elements required for the TLIF procedure. As a result, TLIF has emerged as a viable alternative to ALIF and PLIF.

Bertagnoli et al., in a 2-year study of patients older than 60 years, showed a 94 % satisfaction rate with TDR therapy. Artificial disk replacement has shown results similar to fusion in the short term, but long-term results are not known.

19.10 Postoperative Care

All patients should receive IV antibiotics for at least 24 h during the postoperative period. In the setting of an ALIF procedure, oral intake is delayed until bowel sounds return or flatus is passed. Patients should receive in-patient physical and occupational therapy as soon as possible. Frequent follow-up imaging is necessary to evaluate fusion as well as any potential instrumentation-related complications (plate or screw failure, cage subsidence, or migration).

19.11 Potential Complications

Complications associated with the ALIF technique include vessel damage (2–5 %), retrograde ejaculation (0.5–1 %), and extreme difficulty of a repeat anterior approach if revision is necessary.

The most common complication associated with the PLIF technique is nonunion. The quoted rate of nonunion after this procedure is 5–10 %. Studies suggest the rate of this complication may decrease with supplementation of posterior instrumentation.

The TLIF procedure has few complications that include cerebrospinal fluid leaks, transient neurological complications, and minor wound infections. In some series, radiographic fusion was demonstrated in 74–93 % of patients. Of these patients, 90 % reported they would have the procedure again.

Reported complications after TDR include unilateral foot drop, implant subsidence, and loss of vibration and proprioception. However, many of these complications were observed in patients with circumferential spinal stenosis.

Questions

1. What is the major constituent (by wet weight) of the intervertebral disk in a 20-year- old patient?
   (a) Proteoglycan
   (b) Aggrecan
   (c) Water
   (d) Type I collagen
   (e) Type II collagen
   Preferred Response (c): The intervertebral disk is composed mostly of water (56–90% wet weight), with significant quantities of collagen (15–65 % wet weight), proteoglycan (10–60 % dry weight), and other matrix proteins (15–45 % dry weight). The outer annulus fibrosis is mostly type I collagen, with the inner annulus and nucleus pulposus consisting mostly of type II collagen. Aggrecan is the large proteoglycan of articular cartilage.
2. Disk herniation has been found in what percentage of asymptomatic individuals over the age of 60?
   (a) 20 %
   (b) 40 %
   (c) 60 %
   (d) 80 %
   (e) 100 %
   Preferred Response (b): In a very often-quoted study, Boden et al. performed MRI on 67 patients that had never had low back pain. They found that 20 % of these asymptomatic people under the age 60 had MRI evidence of disk herniation. In the group that was over 60 years old, 37 % had confirmed disk herniations and 21 % had spinal stenosis despite having no pain.
3. An otherwise healthy 45-year-old woman reports the onset of severe right leg pain. A sagittal MRI scan of the lumbar spine demonstrates a disk herniation at the L4–5 level; an axial MRI scan demonstrates the herniation in the far lateral region of the disk. What nerve root is the most likely source of her pain?
   (a) L3
   (b) L4
   (c) L5
   (d) S1
   (e) S2
   Preferred Response (b): Far lateral disk herniations are more likely to compress the nerve exiting at the same level of the lesion rather than the next most caudal level.
4. The MRI scans of the lumbar spine of a patient who has failed 7 months of conservative management demonstrate a right-sided far lateral disk herniation at L5–S1. Operative treatment should be:
   (a) Anterior retroperitoneal approach with anterior lumbar interbody fusion (ALIF)
   (b) Anterior transperitoneal approach with discectomy only
   (c) Posterior midline lumbar laminectomy, decompression, and fusion with pedicle screw fixation
   (d) Posterior midline hemilaminectomy with discectomy
   (e) Paraspinal muscle-splitting approach to the intertransverse space and discectomy
   Preferred Response (e): A right-sided far lateral disk herniation is not easily treated with a standard midline approach, as this approach does not easily allow access laterally. Therefore, the Wiltse paraspinal approach is

ideal, which preserves segment stability by avoiding injury to the lamina and facet joints. The potential complication to know from the Wiltse approach is potential dorsal root ganglia injury, resulting in dysesthesias.

5. A 45-year-old manual laborer presents to the office with acute onset back pain that radiates to his right leg after carrying a heavy object. He also has mild weakness with ankle dorsiflexion on that side. His MRI demonstrates disk herniation at L5–S1. His initial treatment should involve:
   (a) Microdiskectomy
   (b) Posterior spinal fusion with instrumentation
   (c) Decompression only
   (d) Strict bed rest
   (e) Anti-inflammatory medication and physical therapy

   Preferred Response (e): Fifty to 60 % of patients will recover from their symptoms of low back pain within 1 week with 90 % recovering within 3 months.

References

1. Anderson JA (1986) Epidemiological aspects of back pain. J Soc Occup Med 36(3):90–94
2. Aprill C, Bogduk N (1992) High-intensity zone: a diagnostic sign of painful lumbar disc on magnetic resonance imaging. Br J Radiol 65(773):361–369
3. Battie MC, Videman T, Parent E (2004) Lumbar disc degeneration: epidemiology and genetic influences. Spine 29(23):2679–2690
4. Bijkerk C, Houwing-Duistermaat JJ, Valkenburg HA, Meulenbelt I, Hofman A, Breedveld FC, Pols HA, van Duijn CM, Slagboom PE (1999) Heritabilities of radiologic osteoarthritis in peripheral joints and of disc degeneration of the spine. Arthritis Rheum 42(8):1729–1735
5. Buckwalter JA (1995) Aging and degeneration of the human intervertebral disc. Spine 20(11):1307–1314
6. Butler D, Trafimow JH, Andersson GB, McNeill TW, Huckman MS (1990) Discs degenerate before facets. Spine 15(2):111–113
7. Button G, Gupta M, Barrett C, Cammack P, Benson D (2005) Three- to six-year follow-up of stand-alone BAK cages implanted by a single surgeon. Spine J 5(2):155–160
8. Chen CS, Cheng CK, Liu CL (2002) A biomechanical comparison of posterolateral fusion and posterior fusion in the lumbar spine. J Spinal Disord Tech 15(1):53–63
9. Cinotti G, Della Rocca C, Romeo S, Vittur F, Toffanin R, Trasimeni G (2005) Degenerative changes of porcine intervertebral disc induced by vertebral endplate injuries. Spine 30(2):174–180
10. Cohen SP, Larkin TM, Barna SA, Palmer WE, Hecht AC, Stojanovic MP (2005) Lumbar discography: a comprehensive review of outcome studies, diagnostic accuracy, and principles. Reg Anesth Pain Med 30(2):163–183
11. Coppes MH, Marani E, Thomeer RT, Groen GJ (1997) Innervation of “painful” lumbar discs. Spine 22(20): 2342–2349; discussion 2349–2350
12. Friberg S, Hirsch C (1949) Anatomical and clinical studies on lumbar disc degeneration. Acta Orthop Scand 19(2):222–242, illust
13. Humphreys SC, Hodges SD, Patwardhan AG, Eck JC, Murphy RB, Covington LA (2001) Comparison of posterior and transforaminal approaches to lumbar interbody fusion. Spine 26(5):567–571
14. Kirkaldy-Willis WH, Wedge JH, Yong-Hing K, Reilly J (1978) Pathology and pathogenesis of lumbar spondylosis and stenosis. Spine 3(4):319–328
15. Lotz JC, Hsieh AH, Walsh AL, Palmer EI, Chin JR (2002) Mechanobiology of the intervertebral disc. Biochem Soc Trans 30(Pt 6):853–858
16. Matsui H, Kanamori M, Ishihara H, Yudoh K, Naruse Y, Tsuji H (1998) Familial predisposition for lumbar degenerative disc disease. A case–control study. Spine 23(9):1029–1034
17. Miller JA, Schmatz C, Schultz AB (1988) Lumbar disc degeneration: correlation with age, sex, and spine level in 600 autopsy specimens. Spine 13(2): 173–178
18. Mimura M, Panjabi MM, Oxland TR, Crisco JJ, Yamamoto I, Vasavada A (1994) Disc degeneration affects the multidirectional flexibility of the lumbar spine. Spine 19(12):1371–1380
19. Nakamura S, Takahashi K, Takahashi Y, Morinaga T, Shimada Y, Moriya H (1996) Origin of nerves supplying the posterior portion of lumbar intervertebral discs in rats. Spine 21(8):917–924
20. Nakamura SI, Takahashi K, Takahashi Y, Yamagata M, Moriya H (1996) The afferent pathways of discogenic low-back pain. Evaluation of L2 spinal nerve infiltration. J Bone Joint Surg Br 78(4):606–612
21. Niosi CA, Oxland TR (2004) Degenerative mechanics of the lumbar spine. Spine J 4(6 Suppl):202S–208S
22. Roughley PJ, Alini M, Antoniou J (2002) The role of proteoglycans in aging, degeneration and repair of the intervertebral disc. Biochem Soc Trans 30(Pt 6):869–874
23. Sambrook PN, MacGregor AJ, Spector TD (1999) Genetic influences on cervical and lumbar disc degeneration: a magnetic resonance imaging study in twins. Arthritis Rheum 42(2):366–372
24. Schultz A, Andersson G, Ortengren R, Haderspeck K, Nachemson A (1982) Loads on the lumbar spine. Validation of a biomechanical analysis by measurements of intradiscal pressures and myoelectric signals. J Bone Joint Surg Am 64(5):713–720
25. Vanharanta H, Sachs BL, Spivey MA, Guyer RD, Hochschuler SH, Rashbaum RF, Johnson RG, Ohnmeiss D, Mooney V (1987) The relationship of pain provocation to lumbar disc deterioration as seen by CT/discography. Spine 12(3):295–298

26. Walsh TR, Weinstein JN, Spratt KF, Lehmann TR, Aprill C, Sayre H (1990) Lumbar discography in normal subjects. A controlled, prospective study. J Bone Joint Surg Am 72(7):1081–1088
27. Weber H (1994) The natural history of disc herniation and the influence of intervention. Spine 19(19):2234–2238; discussion 2233
28. Weinstein J, Claverie W, Gibson S (1988) The pain of discography. Spine 13(12):1344–1348
29. Yang KH, King AI (1984) Mechanism of facet load transmission as a hypothesis for low-back pain. Spine 9(6):557–565

20 Adult Degenerative Scoliosis

Joshua Ellwitz and Munish Gupta

20.1 Etiology

There are many forms of scoliosis that affect different patient populations. Idiopathic scoliosis which begins during childhood or adolescence can continue to progress resulting in large, painful curves in adulthood. These deformities typically involve a structural thoracic curve and a smaller, more flexible compensatory lumbar curve. Degenerative scoliosis, also termed "de novo" scoliosis, occurs in the lumbar spine and arises due to the degeneration of spine that occurs as a result of aging.

Initial hypotheses postulated that this type of scoliosis was due to metabolic bone disease and the associated weakening of the bones. Subsequent studies, however, demonstrated no difference in bone density between patients with adult idiopathic scoliosis and degenerative scoliosis. Currently it is believed to develop as a result of asymmetric degeneration and collapse of the intervertebral disks and degeneration of the facets joints with marginal osteophyte formation. This results in the characteristic loss of lumbar lordosis and lateral or rotatory listhesis resulting in coronal deformity.

20.2 Prevalence and Natural History

Scoliosis is a frequent finding in the adult population and has been reported to have prevalence between 2.5 and 15 % of patients receiving routine imaging studies. In patients >70 years of age, recent reports have estimated a prevalence approaching 68 % [23]. The full natural history has not been identified, but radiographic risk factors have been identified. A Cobb angle of 30° or more, lateral vertebral translation of 6 mm or more, apical rotation of grade 3, and prominent or deeply seated L5 vertebrae are factors found to be predictive of progression of spinal curvature.

20.3 Clinical Presentation

Patients presenting for treatment of degenerative scoliosis typically report low back pain that has been present for many years. It is frequently the onset of leg pain, however, that incites them to see a spine surgeon. Back pain that localizes near the convexity of the curve is often diffuse and the result of muscle fatigue or spasm. When the pain occurs on the concavity, however, it can be attributed to disk rupture or facet hypertrophy resulting in lateral recess stenosis compressing the traversing nerve root. When compression of the nerve roots is severe, there may be motor weakness but this is less commonly seen.

J. Ellwitz, M.D.
Bronson Healthcare Midwest,
Western Michigan University School of Medicine,
Kalamazoo, MI, USA

M. Gupta, M.D. (✉)
Department of Orthopaedics,
University of California, Davis,
4860 Y Street, Suite 3800, Sacramento,
CA 95817, USA
e-mail: munish.gupta@ucdmc.ucdavis.edu

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_20, © Springer-Verlag Berlin Heidelberg 2014

20.4 Evaluation

Complete history and physical is essential in creating a complete differential diagnosis for the patient presenting with back pain and radicular pain. Components of the axial back pain that are most helpful include the location of the pain, radiation, aggravating and alleviating factors including positional and activity related, and temporality. Positional pain may be indicative of spinal instability or stenosis. Start-up pain, pain that begins with activity after rest and decreases with prolonged activity, is more consistent with degenerative changes within the joints. Pain that is not relieved when supine or occurs during sleep necessitates the investigation of infection or tumor. Full review of systems is helpful for identifying other medical conditions that can present as back pain from the retroperitoneal space such as pancreatitis, kidney stones, or intra-abdominal malignancy. Medical comorbidities also may limit treatment options and have been shown to correlate with decreased success rates, as perceived by the patient, and have been shown to increase the rate of complications [2]. Finally, family history and social history are relevant because depression, nicotine use, and substance abuse have been associated with decreased improvement in outcome after surgical intervention.

Physical evaluation begins with inspection. The patient should be viewed in the coronal and sagittal plane to evaluate for decompensation (loss of ability to position head over the pelvis). It is important to note as well if the patient is compensating for their loss of lordosis with hip or knee flexion, which can result in a compensated sagittal deformity. In the coronal plane, the most common decompensation is a list toward the apex of the thoracolumbar curve. Skin examination for café au lait spots, nevi, sacral dimpling, and hair patches overlying the spine may be indicative of intraspinal pathology.

Evaluation for mechanical back pain is best evaluated with the patient lying prone, allowing the physician to identify areas of maximal tenderness using deep palpation on different segments of the spine such as the apex of the curve vs. the lumbosacral junction. This position also allows for evaluation of paraspinal muscular tenderness. The hips and pelvis should be evaluated for flexion contractures as well as pain from arthritis, impingement, or the sacroiliac joint. Nerve tension signs should be evaluated utilizing the femoral stretch test for the femoral nerve and straight leg raise for the sciatic nerve. Evaluation of deep tendon reflexes can demonstrate nerve root problems and abnormal reflexes (Babinski, clonus, Hoffmann's) can point to a possible cord compression. Sensory loss can be present when there is nerve root impingement, but the utility may be limited as they frequently do not occur in a dermatome pattern. Motor strength should be evaluated with manual muscle testing utilizing standard scoring as well as functional muscle testing performed against the patient's body weight. One should also test for symmetric pulses as vascular problems can manifest as leg pain, especially vascular claudication.

Radiographic evaluation should begin with full-length posterior-anterior and lateral standing films. It is critical to have full-length films to fully evaluate the balance of the spine in the coronal and sagittal planes. Ideally the lateral film would include the odontoid and the femoral heads to evaluate the pelvic incidence and gravity line as well [13, 17]. The Cobb method should be used to define the severity of the curves. The surgeon should also note levels of disk degeneration, lateral listhesis, anterolisthesis, and retrolisthesis. Additional information can be gained from obtaining lateral bending films to determine the flexibility of the curves, flexion/extension films to evaluate instability, and supine extension film over a bolster to evaluate the flexibility of any kyphotic deformity. In adult degenerative scoliosis, however, the spine is frequently fairly rigid.

Evaluation of spinal balance in the coronal and sagittal plane is critical to successful treatment of adult scoliosis. Placement of the head over the pelvis decreases energy expenditure with ambulation, minimizes pain, and minimizes stress at adjacent segments. Sagittal balance is measured with a plumb line from posterior aspect of the superior endplate of T3. Neutral alignment is defined as passing through the posterior aspect of the S1 endplate. Alternatively, the author likes to evaluate a line from the odontoid that should pass through the center of the femoral heads, but this can be limited by the length of the film and the ability to visualize the odontoid. When the plumb line falls anterior to neutral, it is defined as positive sagittal balance and posterior as negative sagittal balance. Coronal balance is assessed by constructing a line that bisects the sacrum and extends vertically (center sacral vertebral line (CSVL)). The CSVL should pass through the center of the C7 vertebrae and bisect the head. Deviation from this is measured as either left or right coronal decompensation.

Radiographic parameters have been evaluated to determine the correlation with visual analog pain scores [22]. L3 or L4 tilt angle, listhesis, thoracolumbar kyphosis, and loss of lumbar lordosis have been found to correlate with increasing pain scores. Conversely, Cobb angle, pelvic tilt, level of listhesis, and plumb line were not found to correlate with pain scores.

A classification system based upon radiographic parameters has been established by Schwab et al. that is correlated with outcome scores. Curves are classified based on (1) apical level of scoliotic deformity, (2) degree of lumbar lordosis loss, and (3) degree of maximal intervertebral subluxation. Sagittal balance was also later included into the system. These parameters were demonstrated to correlate with the decision to operate as well as the surgical approach. A small group of patients with 1–2 year follow-up demonstrated greatest clinical improvement in patients with the most abnormal radiographs. The greatest clinical improvement was associated with loss of lumbar lordosis [24].

Advanced imaging can be helpful as an adjunct for surgical planning, but is never a substitute for adequate plain radiographs. Magnetic resonance imaging is the best screening tool for evaluating the spinal cord, nerves, neuroforamen, and bone marrow of the vertebral bodies. Spinal stenosis occurs when there is loss of disk height, bulging of the disk, hypertrophy of the facet joints, and ligamentum flavum. If a patient is unable to undergo an MRI due to size or metallic implants or pacemaker, CT myelogram is a good alternative. CT scan can also provide additional information with 3D reformatting for larger curves with greater rotational deformity. Discography is a technique that has been used in the past to locate specific levels that are pain generators, but recent studies have questioned the possibility of accelerated degeneration due to damage to the disk with needle insertion [5].

20.5 Treatment

20.5.1 Nonoperative

Patients who initially present with complaints of back pain, radicular symptoms, or spinal stenosis secondary to degenerative scoliosis should be treated with a trial of conservative measures just like they would if they presented with axial or mechanical low back pain. Nonsteroidal anti-inflammatory medications are the first line of treatment for pain. If a patient fails this, then muscle relaxants and analgesics may be considered. Referral to pain management for epidural steroid injections, nerve root blocks, and facet injections should be considered at this stage as well. In addition to using pharmacotherapy to decrease pain, physical therapy may be useful to increase muscular strength and flexibility and may relieve pain that is secondary to deconditioning. Bracing in adult degenerative scoliosis has not been shown to prevent curve progression, but may provide some pain relief in the short term. Glassman et al. investigated the cost and benefits of nonoperative management of adult scoliosis. Although this study was a retrospective review, they found no improvement in health status with nonoperative treatment. Despite the lack of improvement, health care costs range from $10,000 to $14,000 over a 2-year period depending upon severity of symptoms [10].

20.5.2 Operative

Patient's who have failed conservative management may be candidates for surgical intervention. In adult scoliosis, in contrast to adolescent scoliosis, it is pain and disability more than the risk of curve progression that necessitates surgical correction. The goals of surgical treatment are decompression of the neural elements and a stable balanced spine (Gupta). To determine what procedures may be appropriate, we must consider the severity and extent of the spinal stenosis and spinal deformity. Treatment options range from decompression of a focal segment to combined anterior/posterior approaches with osteotomies.

Patients who have minor deformities and have complaints consisting mostly of radicular pain may be candidates for decompression alone. Ideally these patients would have coronal curves less than 30° and minimal rotary or lateral listhesis (Fig. 20.1). The risks of decompression without instrumentation arthrodesis are twofold. First, while decompression will reliably relieve leg pain, persistent mechanical pain postoperatively can lead to patient dissatisfaction. Secondly, removal of the posterior elements may further destabilize an already abnormal spinal segment and lead to rapid curve progression. Women may be at greater risk for progression due to (1) smaller bony elements with osteoporosis, (2) fewer osteophytes stabilizing the spine, and (3) a thinner pars interarticularis that becomes prone to stress fracture after laminectomy. In older patients without instability, the author has had success performing in situ fusion with decompression when minor back pain is a complaint and instrumentation and correction is not desired due to medical comorbidities and bone quality.

Decompression and posterior spinal fusion with instrumentation is an appropriate procedure for patients that have a significant amount of back pain in addition to radicular symptoms or have larger curves with signs of instability. They must, however, have a coronally and sagittally balanced spine. The amount of lordosis from L1 to S1 should approximate the kyphosis from T2 to T12 (Fig. 20.2). It is important to pay close attention to this as in this elderly population, thoracic kyphosis steadily increases, and even more rapidly in females. Failure to recognize this fact can result in fusing the spine with inadequate lordosis leading to increased pain and flatback deformity. Multiple studies have demonstrated that surgical failures resulting in persistent back pain and pseudoarthrosis are attributable to inadequate restoration of lordosis [2, 8, 22]. The author's clinical experience has been a relative inability to correct lordosis without interbody release and fusion.

When planning posterior spinal fusion for deformity correction, there are many details which must be addressed including cranial level of fusion, caudal level of fusion, bone graft source, and bone graft expanders or adjuncts. When choosing the top level of a fusion construct, it is important to identify the apex of the coronal curve. Even when limited levels are chosen to fuse, it is important to not stop at the apex because this puts additional stress at the transition from fusion to mobile spine in an area that is already unstable from the deformity. As the apex is frequently at L3 or L2 level, consideration must also be given to extending the fusion across the thoracolumbar junction to prevent adjacent segment collapse resulting in sagittal decompensation. Frequently degenerative scoliosis spares L5–S1 possibly because it is seated within the pelvis and has additional ligaments stabilizing the L5 vertebrae. This may allow the fusion to stop L4 or L5. Indications to fuse to the sacrum include (1) the presence of spondylolisthesis or previous laminectomy L5–S1, (2) stenosis requiring decompression, (3) severe degeneration, or (4) oblique takeoff of L5 >15°. When fusion to the sacrum is performed, the surgeon must consider the increased risk of pseudoarthrosis. In constructs that include S1 and are greater than 3 levels, consideration should be made to including iliac fixation as lack of stability with S1 fixation alone is one of several factors thought to contribute to the increased risk of nonunion. In these cases, some authors have advocated interbody fusion via posterior or anterior approach to increase fusion rates. It has been this author's experience, however, that with the use of iliac fixation and the use of bone morphogenic protein 2 (BMP-2) fusion at L5–S1 has not been difficult to obtain. Pelvic fixation has been shown to improve caudal stabilization in patients with 5 years of follow-up [26].

The gold standard for bone graft material is iliac crest autograft. Other materials including allograft, recombinant proteins, and synthetics

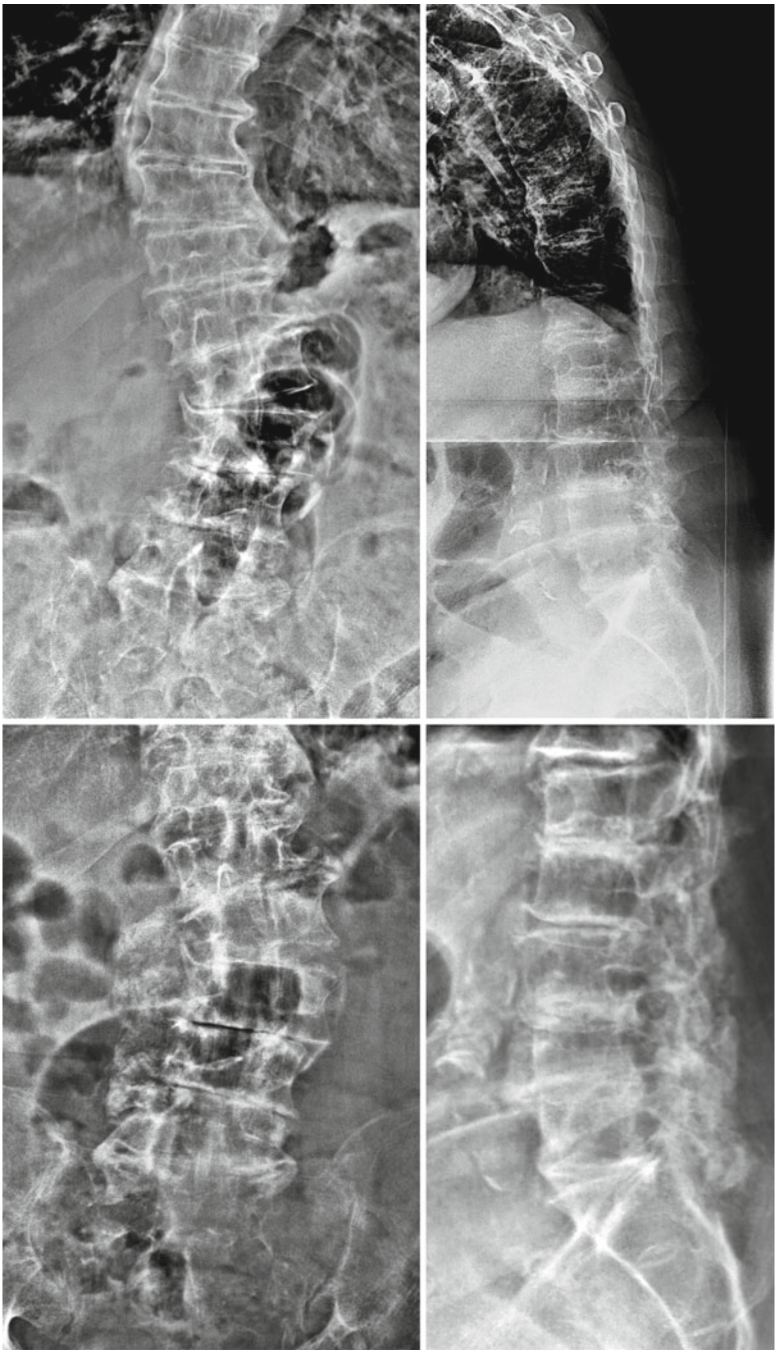


Fig. 20.1 An 83-year old man with severe radicular symptoms. Moderate amount of back pain, but manageable. He underwent decompression alone and is happy with the resolution of his leg pain. Preoperative images are *above* and postoperative *below*

have been developed because of a limited supply of autograft and pain and morbidity associated with iliac crest bone graft harvest. Additionally, autograft harvesting may compromise the ability to obtain stable iliac fixation in long constructs. Recent studies have demonstrated the use of

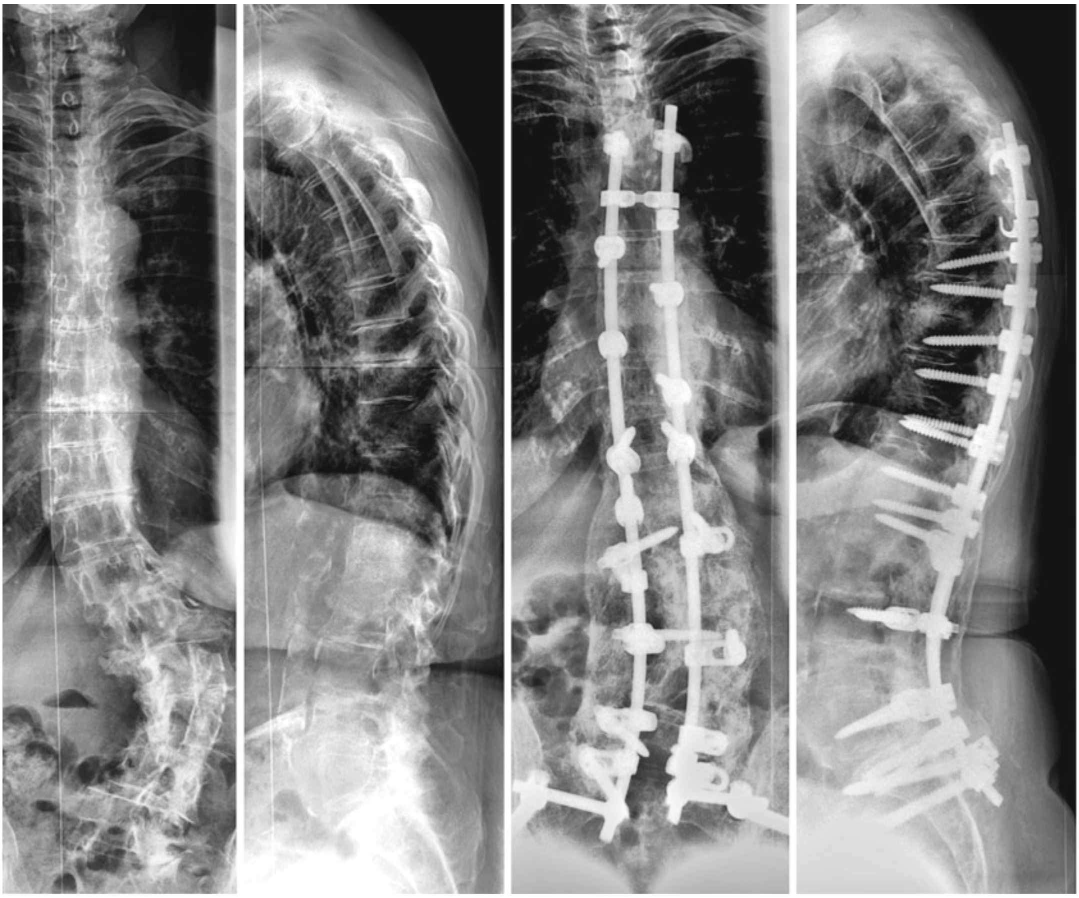


Fig. 20.2 An 83-year old woman with long history of back pain. She has mild, intermittent radicular leg pain. Patient underwent posterior only deformity correction due to good preoperative sagittal balance. Preoperative and postoperative AP and lateral images are shown

BMP-2 with allograft results in fusion rates of 96 % anteriorly and 93 % posteriorly, which approaches the rate obtainable with autograft [19]. Despite the success of BMP-2, its cost is one factor that limits routine use. Recently, Lad et al. performed a cost analysis which demonstrated that the additional cost of BMP is recouped within the first 2 years due to decreased complications, shorter operative and hospital times, and less rehabilitation costs [16]. While there are multitudes of other products available, to date, none have sufficient evidence to support fusion rates near the gold standard of autograft.

When there is coronal or sagittal imbalance and a larger magnitude curve, posterior fusion alone frequently is not sufficient and interbody fusion is necessary. Anterior release results in increased flexibility allowing greater manipulation and correction of the coronal deformity. Restoration of disk height with graft placement increases fusion rate and begins to restore lumbar lordosis and sagittal balance [11]. There are multiple techniques to achieve these goals, but the anterior approach with complete release and structural support is an extremely effective method. There are multiple devices such as titanium mesh cages, expandable cages, and femoral ring allografts that can be inserted in the disk space to provide anterior column support. The author's preference is the use of femoral ring allografts because of the structural support, incorporation into anterior fusion (albeit slowly), and relatively low cost compared to cage alternatives (Fig. 20.3). Allograft chips and iliac crest graft

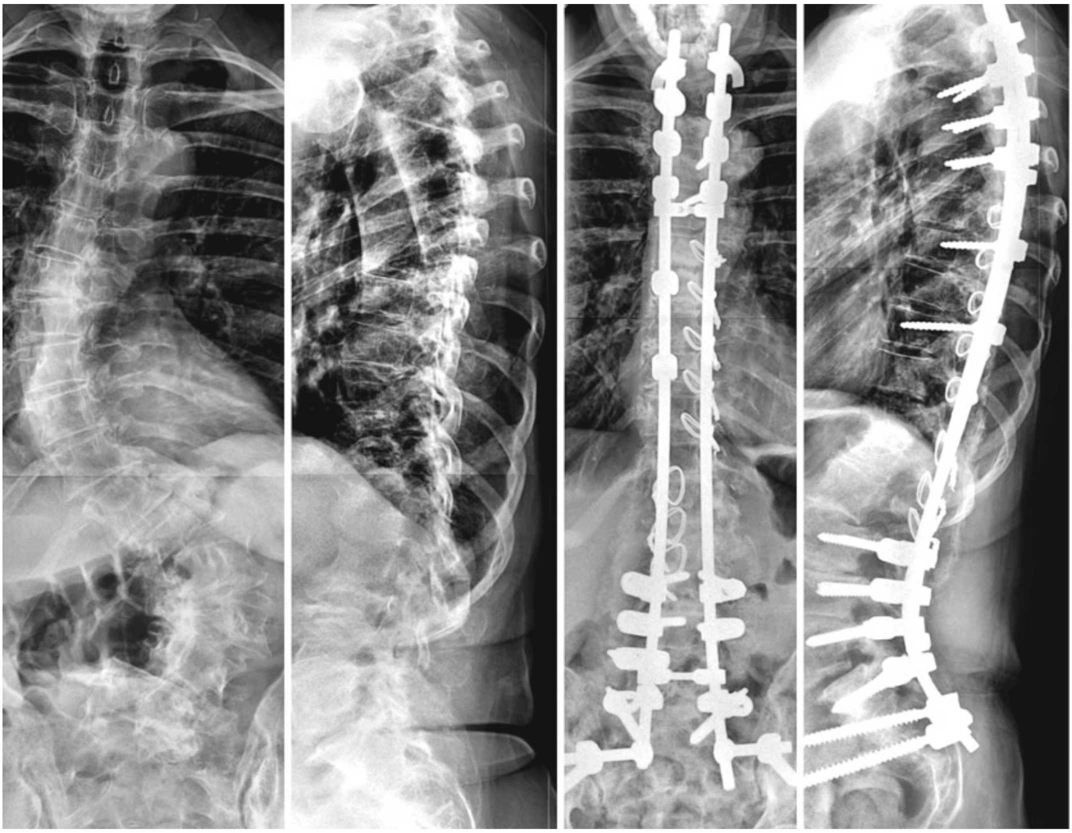


Fig. 20.3 A 60-year old woman with long history of back pain. No significant leg pain. Due to the severity of the curve, inflexibility, and sagittal alignment, ASF T12–L5 and PSF T3-pelvis were performed

appear to not provide adequate structural stability for this purpose.

Recently surgeons have been investigating less invasive techniques besides open thoracoabdominal approach to restore disk height and provide interbody fusion. A lumbar interbody fusion (transforaminal lumbar interbody fusion (TLIF) or posterior lumbar interbody fusion (PLIF)) can be performed through the same incision as the posterior fusion. The advantages of these procedures are there is no need for separate incision or access surgeon, less overall operative time with experience, and lower patient morbidity. The disadvantages include lack of release of anterior longitudinal ligament (ALL), more difficult to perform complete diskectomy, less restoration of disk height, smaller graft surface area, and increased complications from nerve root retraction. The ability of this technique to restore lumbar lordosis is less than seen with anterior approach and is reported as a mean of 36–39° [21]. Another technique that has become increasingly popular is extreme lateral interbody fusion. This procedure involves the use of an expandable retractor placed through a lateral incision into the retroperitoneal space. The advantages of this technique include the ability to manipulate the ALL, increased visualization for diskectomy compared to posterior procedures, no need for access surgeon, less morbid approach, and increased surface area of graft compared to posterior techniques. The disadvantages include reports of injury to the lumbar plexus especially at the L4–5 level, inability to address L5–S1, and a risk of not restoring lordosis if positioning or size of the implant is not correct. Recently presented data demonstrated preoperative lumbar lordosis of 32°, which improved to 49° after far

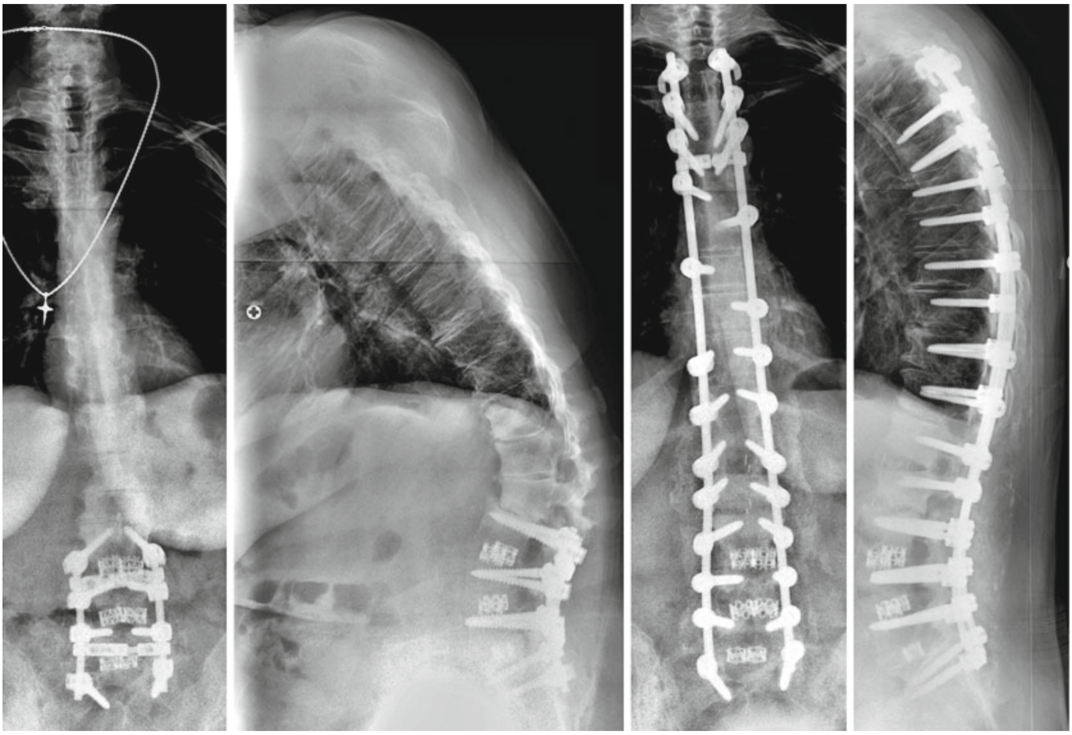


Fig. 20.4 A 54-year old woman who previously had undergone ASF/PSF had severe back pain, severe sagittal imbalance, and left radicular pain. She underwent two levels far lateral interbody fusion at her junctional kyphosis, and PSF was extended to T4

lateral interbody and with final lordosis of 51° after posterior instrumentation [15] (Fig. 20.4). In another study evaluating for complications after lateral interbody demonstrated 33 % incidence of motor weakness, which was transient in 86 % of these cases. There was one patient (0.9 %) that had profound weakness 1/5 attributable to a lumbar plexus injury, which improved to 4/5 by 6 months [14].

In addition to interbody fusion, regardless of technique, posterior fusion and instrumentation is implemented to supplement fixation and provide additional correction. Frequently the decompression is easier after anterior procedure or far lateral procedure due to correction of lateral listhesis and enlargement of the neuroforamen. Pedicle screws have repeatedly been shown to provide greater support and allow for improved segmental correction when compared with hook fixation [1]. When anterior procedures have not adequately restored sagittal and coronal balance, release of the posterior ligaments and Ponte osteotomies (osteotomies of the posterior spinal elements) will often allow for restoration of spine balance. It is the author's preference to perform posterior release along the apex of the coronal deformity resecting the ligament flavum and the facet capsules bilaterally.

20.6 Revision Surgery

There are multiple factors that may necessitate revision surgery. Two main categories of failure are (a) symptoms of radicular pain associated with stenosis and (b) back pain secondary to deformity. The timing of the onset and the location of symptoms may help determine the cause and dictate treatment options. Historically clinical outcomes from revision procedures have been viewed to be less successful than primary procedures. This may be due to a lower preoperative health status as Cho et al. have demonstrated that the clinical improvement (change in health status scores) is the same between primary and revision procedures [7].

When a patient has radicular symptoms after surgery, the timing is paramount to determining the cause [11]. In the immediate postoperative period, the symptoms may be caused by incomplete decompression, misplaced hardware, epidural hematoma, or missed diagnosis. Investigation into the cause of the residual pain is warranted, and treatment can be dictated by the pathology. If the index procedure involved deformity correction, there may be a new area of stenosis that was not present prior to the change in spinal alignment.

In the early postoperative period (days–weeks), recurrent symptoms can be caused by infection, herniation of a nerve root through an iatrogenic durotomy, or BMP-2-related radiculitis. The first step in evaluation is to rule out infection. Lab values may be helpful but can be falsely elevated during this postoperative time frame. Infection warrants irrigation and debridement with implant retention as long as the instrumentation has not been present greater than 3 months. Cultures should be obtained ideally before any antibiotics are given, so appropriate IV antibiotic treatment can be targeted at the inciting organism. BMP-2 radiculitis is a diagnosis of exclusion and can be treated with observation as symptoms have been observed to improve with conservative management.

Midterm recurrence (weeks–months) can be caused by recurrent disk herniation, infection, arachnoiditis, or epidural fibrosis. The mainstay of treatment in this category is nonoperative management once infection is ruled out. If recurrent stenosis is symptomatic after a trial of conservative management, repeat decompression is an option if residual stenosis is identified.

Recurrence of symptoms that occur late in the postoperative period (months–years) is more likely due to recurrent stenosis. This can be the result of degeneration at adjacent segments, or there may be instability from a pars interarticularis stress fracture due to much bone removed at the index procedure. Evaluation and treatment in these cases is similar to the evaluation of a new problem.

After surgery, patients may also have recurrence, worsening, or a new onset of back pain. In the early postoperative period, back pain can become more prominent after decompression alone because the patient is not distracted by the radicular pain. New pain after a decompression can also be caused by too much bony resection resulting in a fracture of the par interarticularis. In the elderly, pain after instrumentation and fusion may be caused by fracture at the transitional zone. In these patients the weak link is the bone implant interface and is prone to failure where the relatively stiff instrumented segments meet the relatively mobile segments. Diagnosis is based upon imaging studies, and treatment can be tailored to the severity of the pain and deformity.

Back pain that develops later after surgery may be due to infection, a failure of fusion, adjacent segment disease resulting in proximal junctional kyphosis, or iatrogenic failure to correct sagittal imbalance resulting in flatback syndrome. Lab studies in addition to a careful history should be used to rule out infection. Any prolonged wound drainage or systemic symptoms should alert the treating physician to the possibility of infection. Failure of fusion, pseudoarthrosis, can be evaluated by careful examination for broken or loosened implants. Additionally, CT scan is useful for evaluating for bridging bone across the segments that were meant to be fused. It is important to note that if there are broken implants even with apparent bridging bone on CT, there must be a high index of suspicion for nonunion. In fact, 30 % nonunions may be found 3–4 years after the index procedure. Treatment involves revision surgery and should consider the use of anterior fusion to increase fusion rates. The preoperative health of the disk immediately proximal to the cephalad aspect of the construct is the greatest predictor of the development of adjacent segment disease [4]. Degeneration at this level can lead to the development of proximal junctional kyphosis (PJK). Treatment of back pain from PJK depends on the severity of the deformity. For patients with mild pain or minimal deformity, nonoperative treatment with bracing is a reasonable first step. More severe deformities often require revision surgery with more proximal extension of the fusion. When

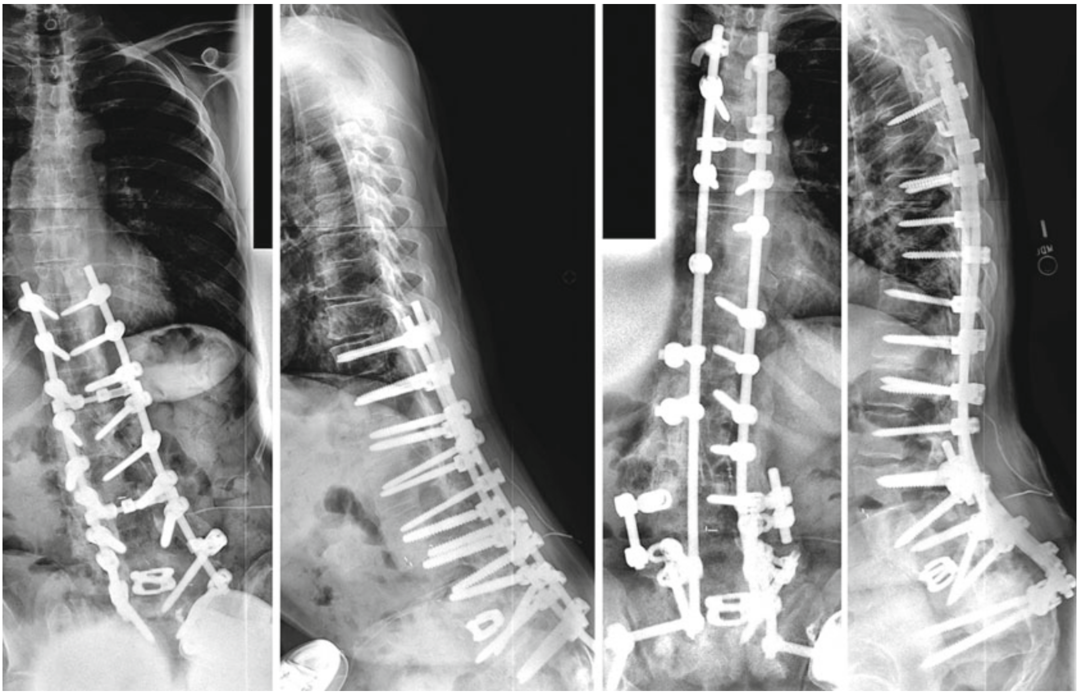


Fig. 20.5 A 52-year old woman with a history of multiple spine surgeries in the last 5 years. She has severe complaints of sagittal imbalance and to a lesser degree coronal imbalance. She has severe back pain and is unable to stand erect. She had extension of her fusion to T4 and pedicle subtraction osteotomy at L3

back pain is secondary to sagittal imbalance due to iatrogenic flatback syndrome, revision surgery is appropriate when pain management modalities have failed. The author's preferred treatment algorithm is determined by the status of the anterior column. If there is no anterior fusion, anterior release and fusion with posterior osteotomies and correction of the deformity is performed. Occasionally this involves a posterior release, followed by anterior procedures, culminating with posterior reinstrumentation and correction of deformity (back, front, back). When the anterior column is solidly fused, revision posterior fusion is performed with pedicle subtraction osteotomy through the anterior fusion mass (Fig. 20.5).

20.7 Complications

Despite an improvement in surgical techniques, anesthesia, and perioperative care surgery for adult, degenerative scoliosis continues to have a high rate of complications. Multiple sources have estimated that the complication rate for adult scoliosis is between 40 and 70 % [3, 20, 25, 27]. The severity of these risks depends on the age of the patient, surgical approach, medical comorbidities, and the number of levels being operated on. It also has been demonstrated that major complications result in a decrease in patients' overall health demonstrated on SF-12 scores 2 years postoperatively [9].

Infection can be a devastating complication because it frequently prevents fusion and requires revision surgery for debridement with long-term antibiotic coverage. Current recommendations from our infectious disease colleagues frequently entail 6 weeks of IV antibiotics despite the paucity of literature to support a particular duration. If the infection occurs in the early postoperative period, spinal implants can be retained as infection resolves quicker when the spine is stable. It is the author's preferred treatment to retain instrumentation if the index procedure is within

3 months, but to perform instrumentation exchange to titanium implants for longer time periods. The incidence of infections has been reported between 3 and 5 % for posterior procedures but is dependent upon the multiple factors including the extent of the fusion, age, and primary vs. revision [2].

Age is another risk factor that has been extensively studied. There are some conflicting reports, but generally it is believed that the incidence of complications is increased in an elderly population [20, 25]. One factor contributing to this is the increase in comorbidities that occur with age which results in an increase in medical complications perioperatively. The two most common mechanisms for construct failure in this population are (1) fracture or screw loosening at the proximal end of instrumentation and (2) late progressive kyphosis at the proximal end of instrumentation. This risk can be minimized by not ending the construct at the thoracolumbar junction or the apex of the kyphosis, but instead extending instrumentation into the proximal thoracic spine. Despite a 4× increase in minor complications and 5× increase major complications, elderly patients have increased improvement in pain and disability scores compared to younger age groups [25]. Additionally, ODI, neck, and leg pain scores are the same 2 years postoperatively in young and elderly patients.

With the rigidity and sagittal imbalance frequently encountered, combined anterior and posterior combined approaches are frequently required. Many authors have examined whether complication rates differ between staged procedures or same day combined approaches. One challenge with staged procedures is malnutrition. Lenke et al. demonstrated a 6–12 week time period to return to nutritional baseline [18].

Postoperative neurologic deficit or paralysis is a feared complication of deformity correction. Fortunately neurologic injury rates are low between 1.0 and 2.5 % [12]. Injuries can be caused by direct injury, tension/compression of the dura, or from ischemia. The risk of ischemia is often underestimated, and the consequences are seen in postoperative patients who undergo cardiac arrest and lose neurologic function after these events. Neuromonitoring with SSEP and MEP is now the standard for deformity surgery. Unfortunately, these modalities are not always reliable and have a relatively low sensitivity but high specificity [12].

A rare complication after spinal surgery is visual loss. Most of the literature consists of case reports, but the incidence is estimated to occur between 0.05 and 1 % after major spinal surgery [6]. The cause of blindness can occur from ischemic optic neuropathy, retinal artery occlusion, and cortical blindness. Unfortunately, most of the reported cases have been permanent visual loss.

20.8 Summary

Adult degenerative scoliosis is a complex, three-dimensional deformity that can be challenging to treat. Symptoms range from back or leg pain to severe deformity with either coronal or sagittal imbalance. Treatment requires complete evaluation of the patient including history, physical, and radiographic components, and must be tailored to each individual patient.

The goals of surgery are to decompress the neural elements; correct sagittal, coronal, and rotational deformity; and provide solid fixation. Small deformities causing radicular symptoms can be managed with decompression alone. Larger deformities without sagittal imbalance may be managed with posterior fusion alone. When sagittal or coronal imbalance is present, larger procedures requiring anterior and posterior surgery with or without osteotomies will be required.

Complicating the surgical management of these patients is the increasing frequency of medical comorbidities with age and incidence of complications associated with these procedures.

References

1. Barr S, Schuette A, Emans J et al (1997) Lumbar pedicle screws versus hooks: results in double major curves in adolescent idiopathic scoliosis. Spine 22(12):1369–1379
2. Birknes J, White A, Albert T et al (2008) Adult degenerative scoliosis: a review. Neurosurgery 63:A94–A103
3. Bradford D, Tay B, Hu S (1999) Adult scoliosis: surgical indications, operative management, complications, and outcomes. Spine 24:2617–2629

4. Bridwell K (2004) Selection of instrumentation and fusion levels for scoliosis: where to start and when to stop. Invited submission from the joint section meeting on disorders of the spine and peripheral nerves, march 2004. J Neurosurg Spine 1:1–8
5. Carragee E, Don A, Hurwitz E et al (2009) Does discography cause accelerated progression of degeneration changes in the lumbar disc. Spine 34(21): 2338–2345
6. Cheng M, Sigurdson W, Tempelhoff R et al (2000) Visual loss after spine surgery: a survey. Neurosurgery 46:625–631
7. Cho S, Bridwell K, Lenke L et al (2012) Comparative analysis of clinical outcome and complications in primary vs. revision adult scoliosis surgery. Spine (Phila Pa 1976) 37:393–401
8. Glassman S, Berven S, Bridwell K et al (2005) Correlation of radiographic parameters and clinical symptoms in adult scoliosis. Spine 30:682–688
9. Glassman S, Hamill C, Bridwell K et al (2007) The impact of perioperative complications on clinical outcome in adult deformity surgery. Spine 32(24): 2764–2770
10. Glassman S, Carreon L, Shaffrey C et al (2010) The costs and benefits of nonoperative management for adult scoliosis. Spine 35(5):578–582
11. Gupta M (2003) Degenerative scoliosis, options for surgical management. Orthop Clin North Am 34:269–279
12. Hamilton K, Smith J, Sansur C et al (2011) Rates of new neurological deficit associated with spine surgery based on 108,419 procedures: a report of the scoliosis research society morbidity and mortality committee. Spine (Phila Pa 1976) 36:1218–1228
13. Hong J, Suh S, Modi H et al (2010) Correlation of pelvic orientation with adult scoliosis. J Spinal Disord Tech 23(7):461–466
14. Isascs R, Hyde J, Goodrich A et al (2010) A prospective, nonrandomized, multicenter evaluation of extreme lateral interbody fusion for the treatment of adult degenerative scoliosis. Spine 35(26S):S322–S330
15. Keshavarzi S, Mundis G, Pekmezci M et al (2011) The utility and limitations of XLIF for adult scoliosis. Spine J 11:S163
16. Lad S, Nathan J, Boakye M (2011) Trends in the use of bone morphogenetic protein as a substitute to autologous iliac crest bone grafting for spinal fusion procedures in the United States. Spine 36(4): E274–E281
17. Legaye J, Duval-Beaupere G, Hecquet J et al (2008) Pelvic incidence: a fundamental pelvic parameter for three dimensional regulation of spinal sagittal curves. Eur Spine J 7:99–103
18. Lenke L, Bridwell K, Blanke K et al (1995) Prospective analysis of nutritional status normalization after spinal reconstructive surgery. Spine 20:1359–1367
19. Luhmann S, Bridwell K, Cheng I et al (2005) Use of bone morphogenetic protein-2 for adult spinal deformity. Spine 30:S110–S117
20. Sansur C, Smith J, Coe J et al (2011) Scoliosis research society morbidity and mortality of adult scoliosis surgery. Spine 36(9):E593–E597
21. Scheufler K, Cyron D, Dohmen H et al (2010) Less invasive surgical correction of adult degenerative scoliosis, part I: technique and radiographic results. Neurosurgery 67(3):696–710
22. Schwab F, Smith V, Biserni M et al (2002) Adult scoliosis: a quantitative radiographic and clinical analysis. Spine 27(4):387–392
23. Schwab F, Dubey A, Gamez L et al (2005) Adult scoliosis: prevalence, SF-36, and nutritional parameters in an elderly volunteer population. Spine 20(9):446–455
24. Schwab F, Farcy J, Bridwell K et al (2006) A clinical impact classification of scoliosis in the adult. Spine 31:2109–2114
25. Smith J, Sansur C, Donaldson W et al (2011) Short-term morbidity and mortality associated with correction of thoracolumbar fixed sagittal plane deformity: a report from the scoliosis research society morbidity and mortality committee. Spine 36(12):958–964
26. Tsuchiya K, Bridwell K, Kuklo T et al (2006) Minimum 5-year analysis of L5-S1 fusion using sacropelvic fixation (bilateral S1 and iliac screws) for spinal deformity. Spine 31:303–308
27. Yadla S, Maltenfort M, Ratliff J et al (2010) Adult scoliosis surgery outcomes: a systematic review. Neurosurg Focus 28:E3

Part III

Pediatric Spine

Scoliosis

21

Prerana Patel and Andrew G.S. King

21.1 Introduction

Scoliosis is usually described as a spinal deformity in which the person's spine is curved from lateral to medial. In fact, the spine is twisted in a complex helix with deformity in all three planes. It is convenient to describe the deformity as visualized on two-dimensional images on posteroanterior (PA) and lateral radiographs. Scoliosis can be categorized in two broad types: nonstructural and structural scoliosis. Nonstructural scoliosis is a secondary curve caused by unequal leg lengths, or by conditions such as herniated lumbar disks, renal anomalies, or spondylolisthesis that cause back muscle spasms. This type of spinal curvature is not built into the spine and will resolve when the primary underlying condition is corrected. A structural, or true, scoliosis is built into the spine and chest wall and involves rotational deformity of the vertebrae, intervertebral disk, ribs, and soft tissues. The appearance of the trunk, shoulders, chest wall, and hips may be altered in a number of recognizable forms, depending on the location of the apex of the curve (Fig. 21.1a, b).

The severity of a scoliotic curve is determined by measuring and recording the Cobb angle. This is the angle formed between the maximally tilted superior and inferior vertebral bodies of the curve as seen on the PA radiograph (Fig. 21.1c). A 10° threshold is usually required before the diagnosis of a structural scoliosis is made, though that does not exclude a nonstructural curve.

There are three broad categories of scoliosis: idiopathic (cause unknown, subclassified as infantile, juvenile, adolescent, or adult according to onset); congenital scoliosis (caused by vertebral anomalies present at birth); and neuromuscular scoliosis (secondary to abnormalities in nervous system and/or muscles, as seen in syringomyelia, spina bifida, cerebral palsy, spinal muscular atrophy, Duchenne muscular dystrophy, paraplegia, and numerous other neuromuscular disorders). The most common form is adolescent idiopathic scoliosis. It affects approximately 2–3 % of the population (about six million people in the USA) [5, 7, 21]. It is commonly diagnosed for the first time in children aged 10–15 years. About 10 % of the adolescent population has some degree of scoliosis, but less than 1 % of people who develop scoliosis require treatment [49].

21.2 Case Example

A 12-year-old girl was referred to an orthopaedic clinic by her pediatrician for evaluation of a back deformity. The deformity had been picked up by a scoliosis screening carried out by a nurse at her school. She was a normal healthy child, active in sports and dance, and had never had any medical complaints related to her back. She lived with her stepmother, who had noticed a significant increase

P. Patel, M.D. • A.G.S. King, M.D. (✉)
Department of Orthopaedics,
Louisiana State University Health Science Center,
1542 Tulane Avenue, New Orleans, LA 70112, USA
e-mail: aking@lsuhsc.edu

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_21, © Springer-Verlag Berlin Heidelberg 2014

in her height in last few months and that she was leaning more to one side. They wondered if this could be related to a very heavy book bag she was required to carry to school. She had begun her menstrual cycle just 3 months previously. Her natural mother had required scoliosis surgery as a teenager.

Her examination standing revealed uneven shoulders with the right higher than the left. The trunk was translated to the right, making her left hip more prominent. On forward bending, there was a rib prominence on the right side, with that side about four centimeters higher than the left (Fig. 21.2). Her neurologic exam revealed 5/5 strength and 2+ reflexes bilaterally in all four extremities with intact sensation to light touch over the extremities. Abdominal reflexes were present and symmetric in all four quadrants. Babinski sign was downgoing. Her plain radiographs revealed a right-sided scoliosis. The Cobb angle, measured between the sixth thoracic and second lumbar vertebrae (T6–L2), measured 51° with significant vertebral rotation. A compensatory lumbar curve (L2–L5) was present, measuring 20° (Fig. 21.3).

21.3 Idiopathic Scoliosis

Idiopathic scoliosis is divided into three categories, based upon the age of the patient when the scoliosis is first detected. Infantile idiopathic scoliosis presents between the ages of birth and 3 years. Juvenile idiopathic scoliosis presents

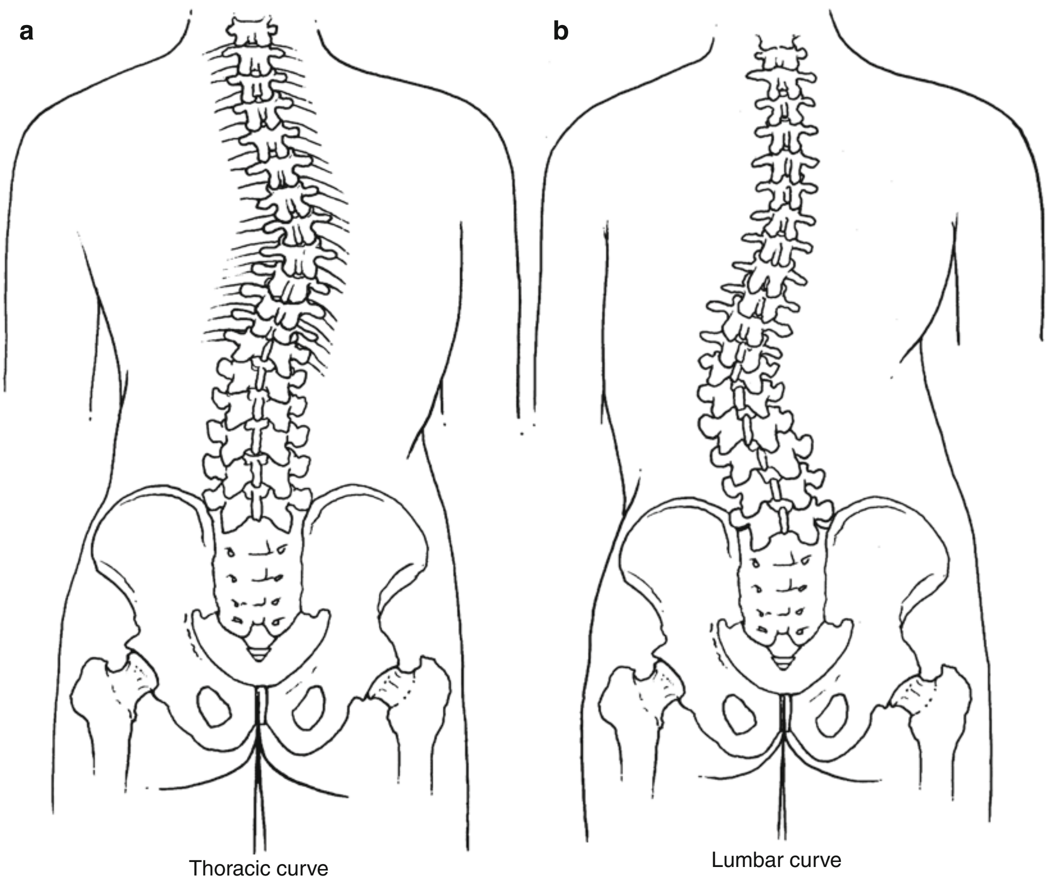
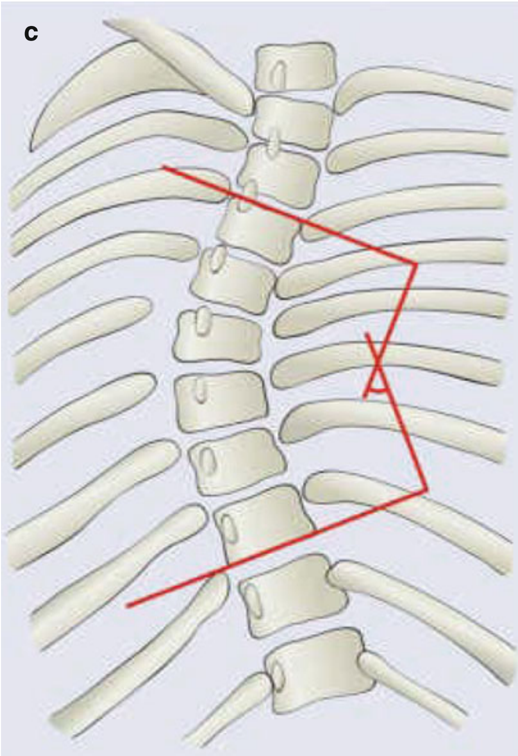


Fig. 21.1 (**a**) Drawing of thoracic scoliosis. Note the trunk asymmetry, or shift, that occurs secondary to the curve. (**b**) Drawing of lumbar scoliosis. This can also cause trunk shift and an asymmetric appearance of the waist. (**c**) Cobb angle: The end vertebrae are the most superior and inferior vertebrae which are least displaced and rotated and have the most tilted end plates. A line is drawn along the superior end plate of the superior end vertebra, and a second line drawn along the inferior end plate of the inferior end vertebra. The angle between these two lines (or lines drawn perpendicular to them) is measured as the Cobb angle



Fig, 21.1 (continued)

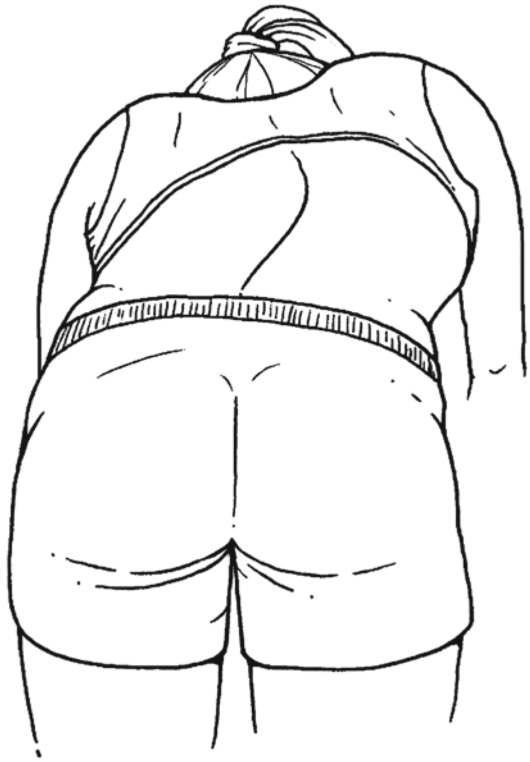


Fig. 21.2 Trunk rotation evident on forward bend is usually seen in AIS

between the ages of 4 and 10 years. Adolescent idiopathic scoliosis (AIS) presents between the ages of 11 and 17 years.

21.4 Adolescent Idiopathic Scoliosis (AIS)

The cause of AIS remains unknown but is widely suspected to involve heredity. Research continues to look at genetic transmission, connective tissue disorders, muscle and platelet disorders, and hormonal imbalance [8, 12, 22, 23, 52, 61]. Genetic studies based on a wide variety of populations have suggested an autosomal dominant or multifactorial inheritance pattern [2, 13]. A similar incidence is seen in nearly all racial and ethnic groups. Population studies involving index patients and their families indicate that 11 % of first-degree relatives are affected, as are 2.4 % and 1.4 % of second- and third-degree relatives, respectively [20, 52]. Twin studies have consistently shown high concordance rate in monozygous twins (73 %) as compared to dizygous twins (36 %) [19, 22]. The ratio of affected females to males is 1:1 in curves smaller than 10° and increases as the curve magnitude increases, with a ratio of 8:1 in curves requiring intervention [1, 14, 32, 55]. There is a relative lengthening of the anterior part of the spinal column with respect to the posterior part, which leads to a flattening of the normal thoracic kyphosis. This is seen in even the smallest curves leading one to believe that it is part of the etiology. Therefore, the term "kyphoscoliosis" is inaccurate in idiopathic scoliosis.

21.5 Natural History/Prognosis

Scoliosis is usually painless and develops gradually. If worsening of the curve is going to occur, it is timed to the upswing of the preadolescent growth spurt. This in turn, most commonly occurs at 1–2 years prior to the onset of a regular menstrual

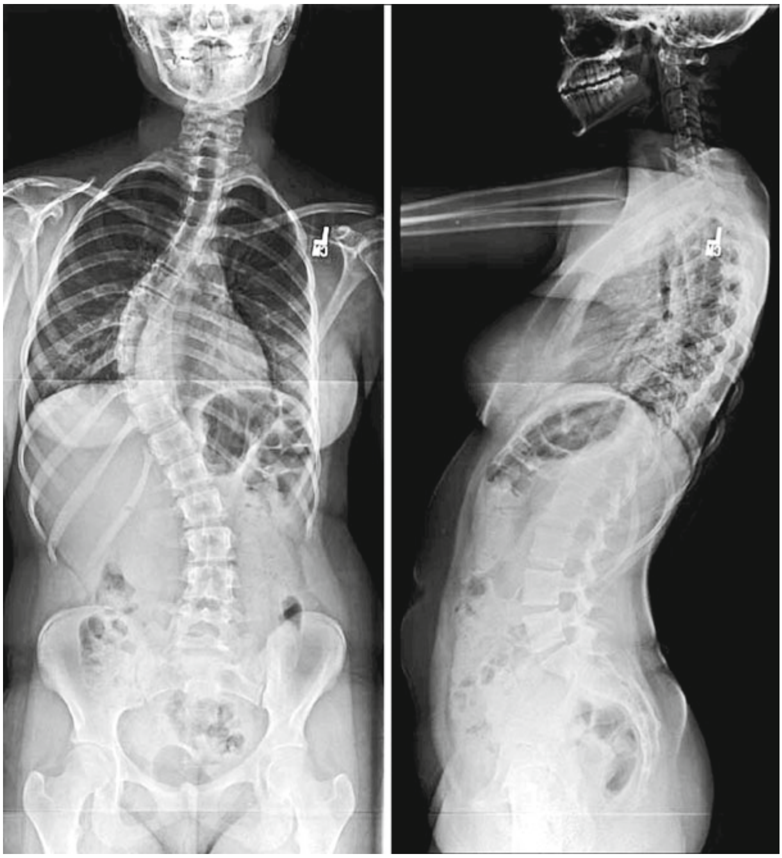


Fig. 21.3 Standing PA and lateral thoracolumbar spine X-rays demonstrating thoracic scoliosis. Asymmetry of the ribcage can be appreciated, demonstrating the rotational component of the deformity

cycle in girls, often around age 10. In boys, it is timed 2–3 years later than usually seen in girls. The main predictors of curve progression are curve magnitude at the time of diagnosis and remaining growth potential. In general, females have an eightfold higher risk of having a curve progress to surgical magnitude. This may be because their growth spurt is superimposed on a spinal column that is over 2 years less mature than that for males. It is clear that assessment of the remaining growth potential of an individual is essential for predicting curve progression, and advising treatment. Clinical signs of puberty (Tanner staging) and radiographic parameters such as triradiate cartilage closure and the Risser sign (iliac apophysis ossification) are helpful in assessing growth potential [64, 65]. The Risser sign is graded from 0 to 5. Grade 0 means no ossification, and grade 5 means complete ossification of the iliac apophysis [53]. Tanner stage 2–3 and Risser sign 0 with open triradiate cartilage indicate high growth potential and greater risk of curve progression. Once the triradiate cartilage has closed, peak growth velocity has passed, and the rate of growth will slow over time. AIS is thought to have its onset during peak growth velocity. Nachemson et al., in a study of untreated female patients who had thoracic scoliosis, suggested that the risk of progression increases with the magnitude of the curve at the time of detection and decreases with increased age at the time of detection. Younger girls (10–12 years old) who had a curve of at least 30° at the time of detection, prior to peak growth velocity, had the highest likelihood of progression, ranging from 90 to 100 % [43]. Spinal growth can be predicted to have stopped in girls who are 2 years out from the onset of regular monthly periods, or in girls and boys who have reached Risser 5. Thoracic curves less than 30° at skeletal maturity are unlikely to progress, whereas curves measuring from 30 to 50°

may continue to slowly progress an average of 10–15° over a lifetime. Curves greater than 50° at maturity tend to progress steadily at a rate of about 1° per year due to settling. For thoracolumbar and lumbar curves, those under 20° at maturity do not progress, while those of 40° or more progress steadily into adulthood. Curves in the midrange are less predictable, but those over 25° slowly increase in some patients past maturity [42, 66]. This data from long-term natural history studies of untreated patients forms the basis of our treatment strategies and recommendations. Long-term curve progression is associated with increased incidence of back pain and problems in adulthood; therefore, surgical treatment is recommended for those curves expected to progress past maturity.

21.6 History and Physical Examination

Idiopathic scoliosis patients are usually asymptomatic. They or their families often seek orthopaedic evaluation for the appearance aspect of their deformity, such as uneven shoulders, rib prominence, or waist asymmetry. These findings are often first appreciated during school screening programs for scoliosis or during back-to-school or sports physicals examinations by a pediatrician or family physician. The presence or absence of severe back pain is important because most patients with idiopathic scoliosis have little or no discomfort. Complaints of persistent or significant back pain warrant careful evaluation to rule out pathology in the spinal column or spinal cord that may be causing both pain and scoliosis. However, a recent study by Ramirez et al. suggested 32 % of AIS patients experience back discomfort at some point (23 % at presentation and 9 % during the period of observation) [50]. Patients without antecedent symptoms may also report pain after the diagnosis is made. The history should also record any family history of scoliosis, and details such as onset of menses that would help in determining growth potential.

The physical examination of a patient with scoliosis includes evaluation of the back deformity itself, a thorough neurologic exam, and examination of the skin, feet, joints, and general appearance. The neurologic examination should exclude long tract signs in both lower extremities. Full lower extremity motor and sensory evaluation, as well as abdominal and lower extremity deep tendon reflexes, should be performed. Scoliosis resembling AIS is often the presenting sign of syringomyelia, and asymmetric abdominal reflexes and other asymmetries of motor function and sensation are often seen with this condition. Height and weight should be obtained on new patients, and serial heights of follow-up patients are useful to assess whether growth is still progressing rapidly.

Non-idiopathic causes of scoliosis can be quickly excluded during the examination. Neurofibromatosis often causes severe scoliosis, and the skin should be carefully evaluated for the presence of café au lait spots. Congenital abnormalities of the spinal cord, such as tethered cord and lipomeningocele, often are accompanied by a lumbosacral nevus, hairy patch, lipoma, or skin dimple. An AIS-like pattern may be seen in patients with collagen disorders such as Marfan syndrome or Ehlers-Danlos syndrome. These curves often require longer follow-up past maturity and do not do as well with limited surgery, so it is important to assess the patient carefully for generalized ligamentous laxity and other characteristic features to diagnose this. Family history of these disorders should also be elicited. Finally, the examination should exclude disorders of the posterior columns of the spinal cord, such as Friedrich's ataxia and Charcot-Marie-Tooth disease (CMT), now known also as hereditary motor-sensory neuropathy (HMSN), by testing balance and assessing deep tendon reflexes. The presence of cavus feet, intrinsic wasting of the hands, and stork-like calves can also assist in the diagnosis of HMSN (Fig. 21.4).

Evaluation of the scoliotic deformity starts with a standing assessment done from behind the patient, with the patient's bare back visible and shoulders and pelvis accessible. Unevenness of the shoulders or pelvis, waist asymmetry, and trunk shift should be noted (Fig. 21.5). Any significant decompensation, where one curve does not appear to be balanced by another, should be noted. The patient should be assessed from the side also to ensure that no significant kyphosis is present. The presence of kyphosis should alert the examiner to potential need for further evaluation.

Fig. 21.4 Right cavus foot. Note the inward tilt, or varus, of the heel, and more visible forefoot compared to the normal left side. This can be associated with an underlying neurologic abnormality

Fig. 21.5 Clinical photo of patient with severe scoliosis. The right shoulder is elevated, while the entire trunk is shifted to the right. The waist is asymmetric, with a straight appearance on the left and wrinkling of the skin on the right

The forward bend test, described by Adams, has the patient bend forward at the waist with the knees straight and palms together. When the flat

Fig. 21.6 Clinical photo of patient with severe scoliosis. Adams forward bend test reveals the significant trunk rotation and rib deformity present in scoliosis

of the back is parallel to the floor, the rotation of the scoliotic spine will be most obvious (Fig. 21.6). Rotation may be measured with a scoliometer to record the rotation for future comparison. This examination should be performed from behind (to assess lumbar and midthoracic rotation) and from the front (to assess upper thoracic rotation), as well as from the side (to assess kyphosis) [6].

21.7 Radiographic Evaluation

The initial examination of the spine should include standing posteroanterior (PA) and lateral radiographs of the entire spine on 36 by 14 in. cassettes. The PA projection is preferred over the AP projection for frontal plane assessment because the former results in significantly less radiation exposure to breast and thyroid tissue [17, 28].

The PA projection is useful to assess the coronal curve pattern, the type of scoliosis (congenital or idiopathic), the overall balance of the spine and trunk, skeletal maturity (as determined by Risser sign), open versus closed triradiate cartilage, and the presence of a lower limb length discrepancy by evidence of asymmetric height of the iliac crests (Fig. 21.7). The Cobb method is used to measure the degree of scoliosis on the PA X-ray [10]. The largest curve by Cobb measurement is termed the “major” curve. Lesser curves above and below are termed minor or compensatory curves. In addition to curve magnitude,

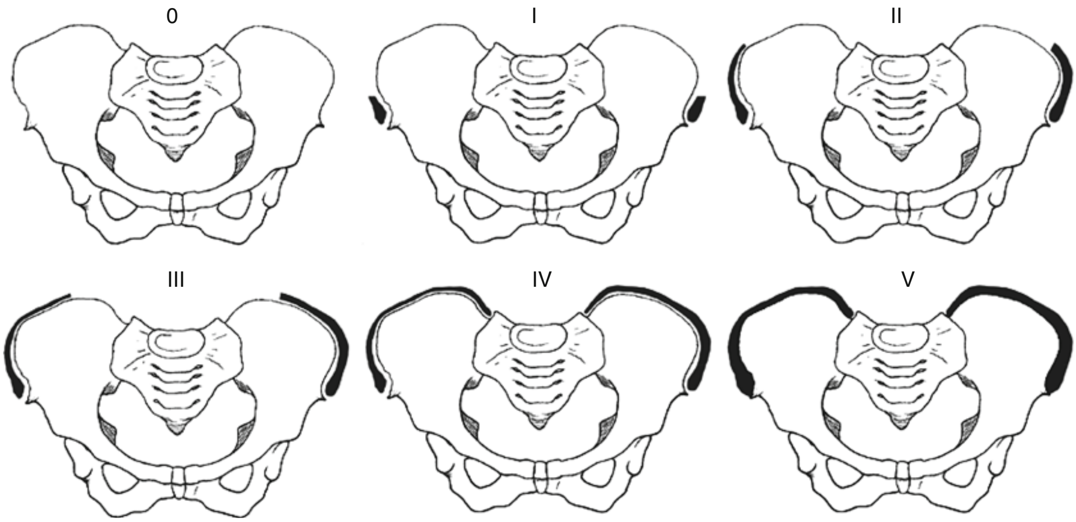


Fig. 21.7 Risser stages of pelvic maturation. Stage 0 demonstrates no capping of the iliac crests, seen in skeletally immature patients. Progressive appearance of the iliac crest apophysis on radiographs correlates with the normal slowing of spinal growth over time. Fusion of the apophysis to the crest, or Risser V, correlates with completion of spinal growth

physicians should describe curves as "right" or "left," based on the direction of the curve convexity. The large majority of idiopathic scoliosis curves will be convex to the right in the thoracic spine and to the left in the lumbar spine. The reverse may indicate an underlying neurologic cause for the scoliosis such as syringomyelia.

The lateral projection is useful to evaluate the global sagittal balance of the thoracic and lumbar spine and determine the presence and severity of the decreased thoracic kyphosis. The decreased kyphosis is not always obvious on the lateral due to rotation of the spine. Thoracic kyphosis of 35° or greater should prompt MRI evaluation [48]. The radiograph should also allow assessment of the presence of spondylolysis and/or spondylolisthesis. Coronal bending scoliosis radiographs allow an assessment of the stiffness of the curves and may be helpful in planning surgical correction, as well as in determining which curves truly require intervention and which may be left alone [9].

Obtaining an MRI of the entire spine routinely in a patient with scoliosis is controversial. Several prospective studies of routine MRI screening for preoperative assessment of all patients with idiopathic scoliosis have shown that MRI should be reserved for patients with an early onset of scoliosis (infant and juvenile age-groups), significant back pain, rapid curve progression, an unusual curve pattern such as a left thoracic curve, abnormal neurologic exam, or cutaneous findings suggestive of dysraphism or neurofibromatosis [15, 18, 35, 40, 46, 57] (Fig. 21.8). Additionally, Ouellet et al. have suggested that an increased kyphosis on the lateral radiograph of the thoracic spine should raise suspicion of syringomyelia and necessitate an MRI [48].

21.8 Treatment

Treatment consists of observation, bracing, and surgical intervention, depending on curve magnitude, progression, and patient age and growth status. An algorithm for the treatment of adolescent idiopathic scoliosis is outlined in Table 21.1.

21.8.1 Observation

This is indicated in patients with a curve less than 20° or in patients whose growth has progressed past the rapid stage (Risser 3 and higher). A follow-up PA X-ray every 4–12 months is advised depending upon the patient's age and remaining growth potential. No restriction of activities is required.

21.8.2 Bracing

Bracing is recommended for curves in immature patients (Risser grade 1 or less) with a minimum magnitude of 20°. Curves of less than 25° should be braced only after there is documented progression of 5° or more in patients who are Risser grade 1 or less [34, 56]. The theory behind bracing for scoliosis is that the increased pressure on the vertebral growth plate on the concave side of a curve will cause decreased growth and lead to worsening of the curve via the Hueter-Volkmann principle (Fig. 21.9a). A well-constructed brace should relieve this pressure by partially straightening the spine during the time the patient is wearing the brace, relieving the Hueter-Volkmann forces (Fig. 21.9b). A brace-wearing schedule may vary from full time to nighttime only depending on the type of brace used. Traditionally, full-time (23 h per day) brace wear was advised; this continues to be recommended by many who prescribe scoliosis braces. Over the last two decades, many centers reduced the wearing schedule to 16 h per day to improve patient compliance and allow the child to go to school without the brace. The brace most commonly used for full-time or 16-h wear is the Boston type underarm thoracolumbosacral orthosis (TLSO). It is able to control curves with an apex of T9 or below, though it has been used up to T6 if the push pads are effective. The Charleston bending brace was developed on the concept that part-time use may be effective. This brace holds the patient in maximum side-bending correction. The side-bending force exerted by the brace does not allow its use in the upright position, thus making wear feasible only when the patient is recumbent. This brace is best used in lumbar or thoracolumbar curves, as the higher the curve apex, the less bend will be achieved. The main appeal of this brace is the limited number of hours of daily wear, all of which are accomplished during sleep. Many other

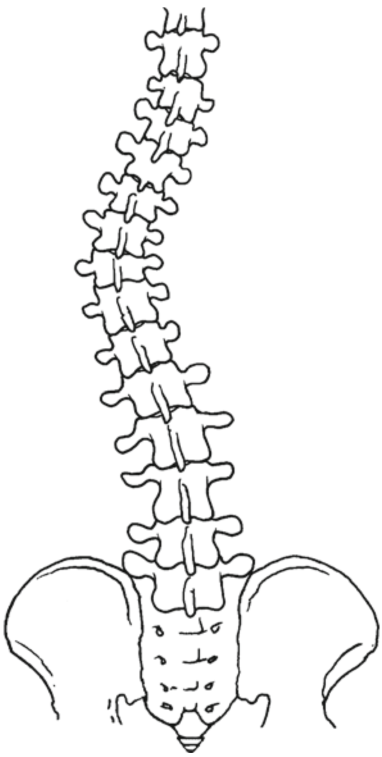


Fig. 21.8 Drawing of left thoracic curve. This atypical appearance can be a sign of underlying neurologic or other abnormality and usually warrants further investigation with an MRI

Table 21.1 A general outline of treatment for idiopathic scoliosis

	Risser sign		
Curve magnitude (degrees)	Grade 0/premenarchal	Grade 1 or 2	Grade 3, 4, or 5
<20	Observation	Observation	Observation
20–40	Brace therapy (begin when curve is >20° with documented progression >5°)	Brace therapy	Observation/bracing
>45	Surgery	Surgery	Surgery (when curve is >50°)

The treatment recommended may vary from one individual to another, as curve patterns and individual needs vary [33, 34, 56, 66]

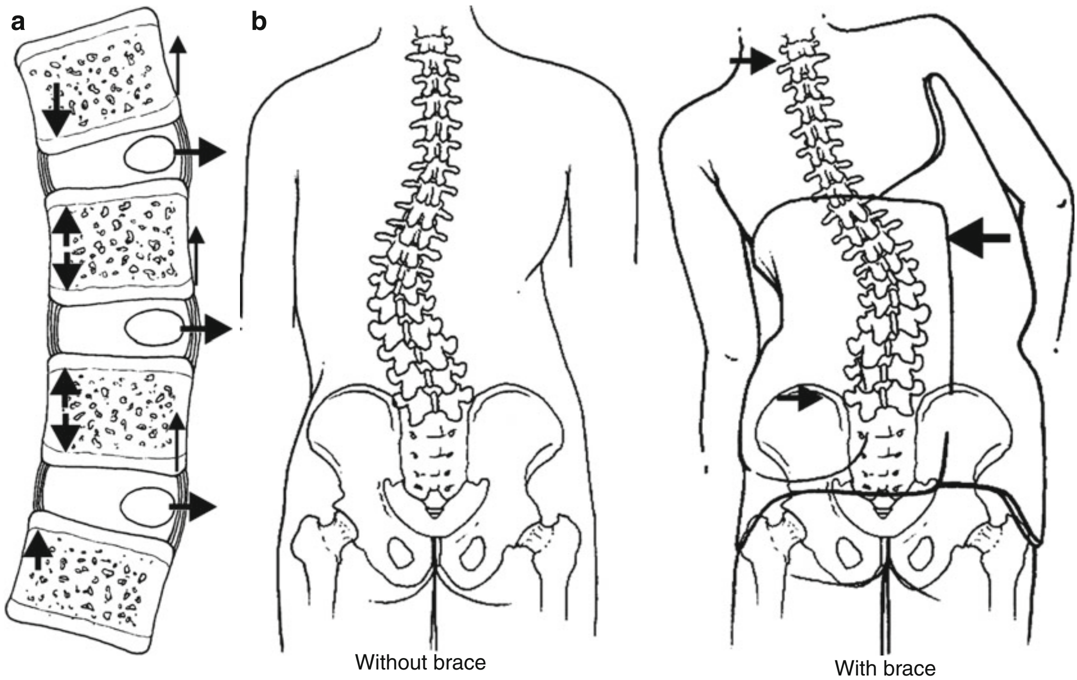


Fig. 21.9 (**a**) Hueter-Volkmann principle. Spinal curvature worsens during growth due to growth suppression on the concavity (*left*) and continued growth on the convexity of the curve. (**b**) Bracing during growth is thought to relieve the compressive forces and allow the spine to grow in a balanced fashion without worsening the curve. The brace demonstrated here is used during sleep because of the extreme bend placed on the lumbar curve

brace types exist, but these two are most widely used (Fig. 21.10a, b).

The effectiveness of bracing for idiopathic scoliosis has been debated. Controlled blinded trials are difficult to perform due to the variability of the curves, the difficulty in measuring compliance, and the reluctance of some patients and parents to accept a nontreatment arm of a prospective study. In 1995, the results of a prospective, controlled (but not randomized) study of bracing by the Scoliosis Research Society were published. Results were compared in 286 patients, aged 10–15 years, with an initial curve of 25–35°: 129 patients were observed but received no treatment, 111 were treated with an underarm brace, and 46 were treated with nighttime electrical stimulation. The percentages of curve progression greater than 5° were 26 % in bracing, 66 % in observation, and 67 % in electrical stimulation; these findings demonstrate a statistically significant effect of bracing in this population over observation and electrical stimulation [44]. Most centers have accepted these results and continue to advise brace treatment for progressive curves in skeletally immature adolescents. Contraindications to brace treatment include large curves greater than 45°, skeletally mature patients, patients with large body habitus, and noncompliant patients. Some studies have shown compliance in boys to be so poor that bracing should not be recommended. This is controversial.

Once a brace is obtained, it should be assessed for effectiveness. The brace should be able to be worn comfortably but snugly, and when worn correctly, should reduce the curve magnitude while in brace by 50 % if possible. This may not be possible with stiffer curves and does not necessarily mean that bracing should be abandoned, but may indicate less likelihood of curve control with the brace. The night bending braces may reduce the curve to zero while in brace, as they are most effective in lumbar and thoracolumbar curves, which tend to be more flexible. It is

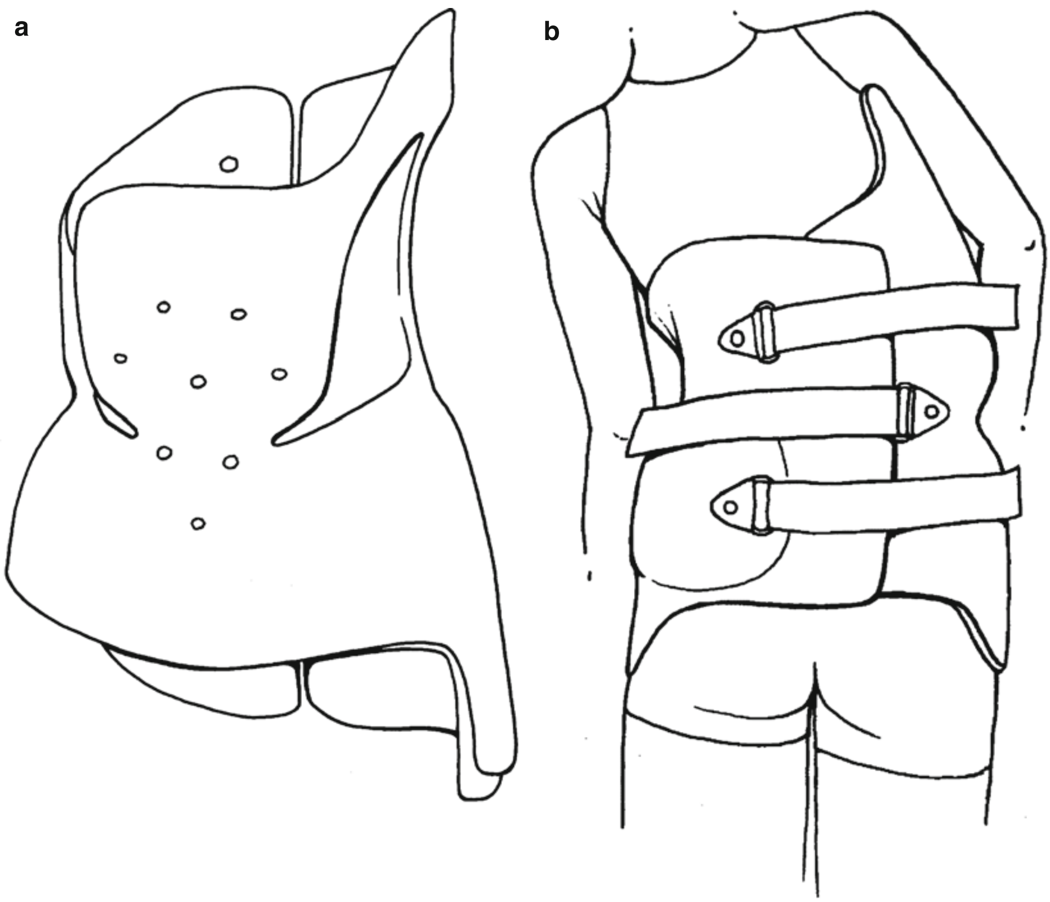


Fig. 21.10 (**a**) Boston-type underarm brace for scoliosis. Push pads and rotational molding are used to apply corrective forces to the thoracic curve. (**b**) Charleston-type night bending brace. This is useful for more distal curves such as lumbar and thoracolumbar curves. The brace bends the patient against the major curve, applying corrective force to allow growth without curve progression

important to note to the families that bracing does not make the existing curve go away permanently, but is simply designed to prevent curve progression. Not all curves will respond to bracing.

One challenge in the nonoperative treatment of scoliosis is that we have not had a way to tell which curves are likely to progress and which ones could simply be observed, preferably even without radiographs. Even with the diminished exposure from digital radiography, patients with scoliosis will be subjected to several X-rays throughout their teenage years. If we knew which curves were less likely to progress, in theory, those patients could be followed less and examined clinically instead of radiographically until a change was detected. Conversely, curves that were expected to reach surgical magnitude might not require bracing, as surgery would be an expected outcome. Or, if the family preferred, they might be braced more aggressively and for longer. Genetic testing in AIS has recently become available (ScoliScore, Axial Biotech), with improving validity. It is currently only validated for the Caucasian population and was found to not be successful in determining curve progression in Asians or Hispanics. The test involves obtaining a sample of saliva in the clinic, which is processed for a set of 50 genetic markers that correlate with likelihood of curve progression. A score is obtained between 1 and 200 that predicts the likelihood of curve progression to surgical range. Clinical application of the results has been mixed among surgeons, and testing continues to evolve.

A recent alternative to bracing for patients with small curves that progress is operative vertebral body stapling, in which Nitinol staples are applied across the end plates and disk at several levels through the convexity of the curve. The principle behind this is similar to guided growth treatment of other growth centers in the body with staples, but the validity of this comparison is controversial, as the stapling occurs across the end plates and the intervertebral disks. The long-term effects of crossing the disk space with a compressive device are unknown, leading many surgeons to be reluctant to embrace this technique. Vertebral body stapling has only been found to be effective at reversing or controlling scoliosis in a small number of curves with magnitude less than 35° and sufficient growth remaining, limiting the number of patients for whom it is a viable option. The treatment remains investigational [3, 24, 47].

21.8.3 Surgical Treatment

The goals of surgical treatment of idiopathic scoliosis include improved spinal alignment and balance, prevention of the curve progression, and improvement of trunk appearance. Operative treatment aims to fuse the spine in a balanced position with the patient's shoulders, head, and trunk centered over the pelvis in both the coronal and sagittal planes. Although various considerations enter into the decision for surgery, curve magnitude remains the primary factor. Thoracic curves and double major curves that exceed 50° at skeletal maturity have a significant probability of worsening over time and warrant operative intervention. Thoracolumbar and lumbar curves of lesser magnitude, when associated with marked apical rotation or translatory shift, also have a propensity to worsen over time in mature patients. In these cases, surgery should be considered when the curves exceed 40–45°. Corrective instrumentation techniques have evolved in past few decades from Harrington rod instrumentation to today's dual rod and segmental pedicle screw fixation. The use of pedicle screw fixation at multiple spinal levels allows control of all three columns of the spine from the back, and therefore allows placement of greater corrective forces on scoliotic spines. Controversy exists whether thoracic pedicle screw fixation is worth the additional cost of the implants over the prior generation of implants, hooks, and wires, which could fix the posterior column of the spine alone and could not allow axial rotational correction. Pedicle screw fixation has been demonstrated to result in about 10° better coronal curve correction, and significant axial plane correction. However, the sagittal plane correction appears to be compromised with the use of screws, as the derotation of the thoracic curve produces lordosis of the thoracic spine. Stiffer rods, different placements of the screws along the rods, and use of significant soft tissue releases have all been employed to prevent this lordosing tendency and restore appropriate thoracic kyphosis. Restoration of the sagittal plane appears to be the most important factor in long-term spine health. Patients treated with Harrington instrumentation, which was distraction based and resulted in a loss of lumbar lordosis or "flatback," often had later pain and decompensation and required complex reconstruction to restore lumbar lordosis and appropriate sagittal balance (Fig. 21.11). The advent of dual rod, nondistraction-based instrumentation that could preserve sagittal contour, beginning with Cotrel-Dubousset instrumentation in the early 1980s, appears to have solved this problem, but our newer and more powerful methods of correction have the potential to bring it, or other issues, to the forefront again.

21.8.4 Preoperative Planning

Preoperative workup includes routine blood work, urinalysis to rule out urinary tract infections, and often a set of pulmonary function tests as a baseline. Most centers take side-bending radiographs in the coronal plane for use in operative planning. Discussion of preoperative risk should include infection, both acute and delayed; blood loss requiring transfusion; neurologic injury, both transient and permanent; injury to adjacent structures; failure of fusion; failure of

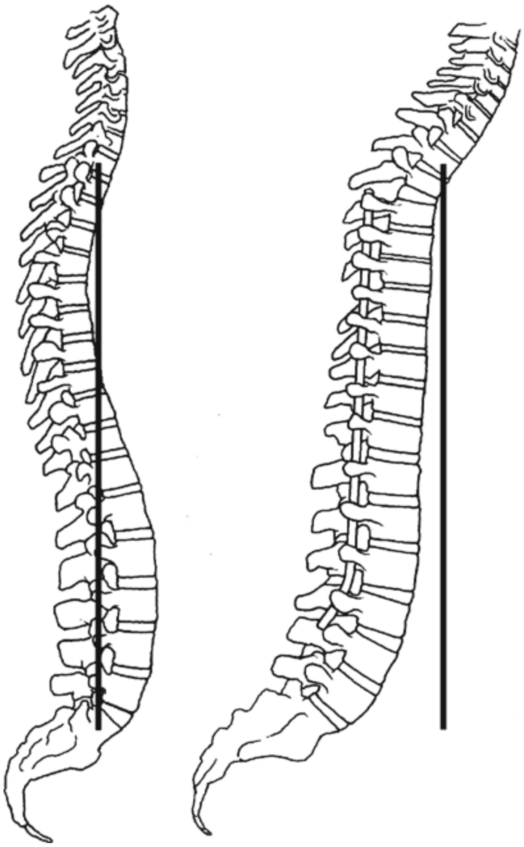


Fig. 21.11 Flatback deformity and its effect on sagittal balance are illustrated in this drawing. On the *left*, the normal sagittal alignment of the spine is demonstrated. Flattening of the lumbar lordosis after posterior distraction changes the sagittal balance, which can lead to problems in the future

instrumentation; dural leak; need for further procedures; late decompensation or curve progression; medical risks of surgery; anesthetic risks; and possible death. Cadaveric allograft bone is used for fusion mass instead of autologous iliac crest graft in primary cases; though this carries a small risk of infection, fusion rates are high and the often long-standing pain of iliac crest bone harvest is eliminated. Transfusion is often required, and cell-saver machines are popular during surgery to reduce the allogeneic transfusion need. Preoperative autologous donation is employed at some centers. Noncosmetic scarring is likely from a posterior approach even with a cosmetic closure. A very rare but possible risk is blindness from ocular hypoperfusion during surgery if the blood pressure is allowed to remain very low for too long. This is more common during adult spine surgery. The use of motor evoked potential monitoring during surgery may make this less likely, as it requires the maintenance of a higher perfusion pressure during the procedure. The patient is instructed to have a shower with a chlorhexidine-based soap 1 day prior to and on the day of surgery. Some centers advocate nasal swabs to detect methicillin-resistant *Staphylococcus aureus* (MRSA). Controversy exists over the appropriate preoperative antibiotic and over the duration of use postoperatively. Cefazolin was traditionally employed, but with the rise of MRSA infection, some surgeons are choosing vancomycin or clindamycin as their prophylactic antibiotic of choice.

21.8.5 Curve Patterns

Levels of fusion are decided after analyzing the primary and compensatory curve magnitudes and flexibility as well as the coronal and the sagittal alignment of the spine. A classification of the curve pattern is made, and commonly accepted rules of fusion levels applied to each curve type. Currently most surgeons use the classification system described by Lenke and coworkers in 1995 [26]. This classification is comprehensive (there are 42 curve patterns) and details of the classification can be found elsewhere. Despite the presence of a classification system, studies have demonstrated that experienced deformity surgeons use multiple other factors to determine which curves to fuse and which levels to include, demonstrating that a classification system alone is insufficient to guide treatment [16, 25, 27, 45] (Fig. 21.12a–d).

21.8.6 Goals of Surgery

It is useful to remember that the first goal of treatment is to stabilize the curve and prevent progression. Because our powerful instrumentation techniques give us the ability to restore a more

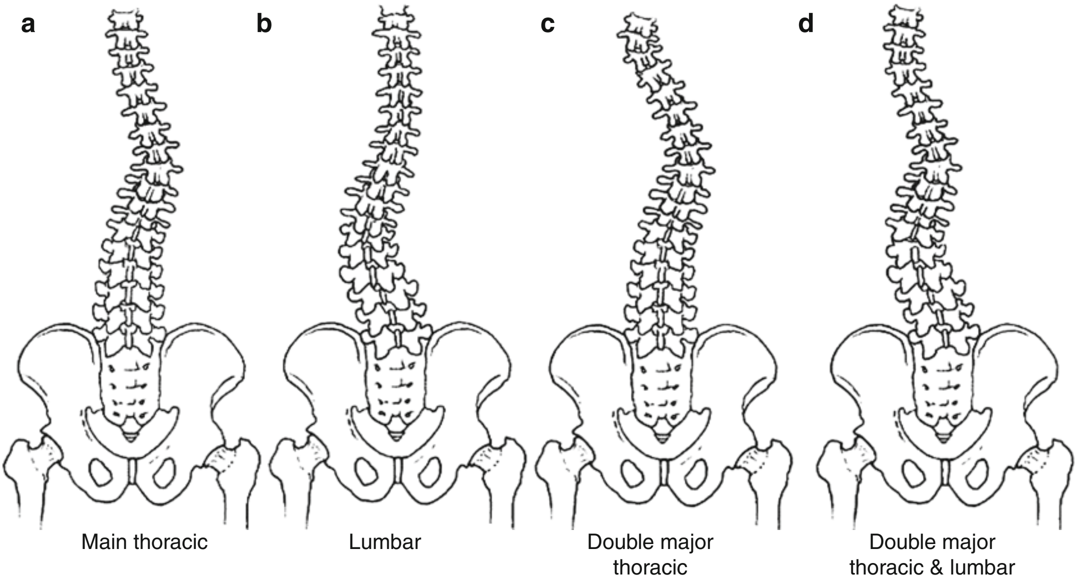


Fig. 21.12 (**a**–**d**) Drawing of four basic curve types seen in adolescent idiopathic scoliosis. Curves are defined based on the location of the apical vertebra. This vertebra is the most rotated and usually the least tilted within the curve

normal back appearance and correct the curve radically, we often focus on this. However, the risk incurred by the patient is proportionate to the amount of time spent in surgery and the amount of correction sought, so keeping the overall goal in mind is useful.

21.8.7 Surgical Approach

Two general approaches are employed: a posterior approach (from the back of the spine), or an anterior approach (through the side of the body, to reach the vertebral bodies and disks). The posterior approach is the most commonly used. This approach is performed through a longitudinal incision over the posterior midline. Subperiosteal dissection of the paraspinous muscles is performed out to the transverse processes at each level to expose the spine adequately for instrumentation and fusion. The inferior articular facet is harvested at each level, and the cartilage of the superior facet cleared. This both loosens the spine for correction and promotes fusion. Locally harvested bone is reserved. The spine is then instrumented and custom-contoured rods are used to reduce the amount of curvature and push it into the desired plane (Fig. 21.13). Once correction is satisfactory, the external laminae are decorticated to promote fusion. Autologous harvested facet and spinous process bone is chopped up and mixed with cadaveric allograft cancellous bone, and this is packed on both sides of the midline, around the rods and implants, to provide fusion mass. Closure is performed, often over a deep or subcutaneous drain, and the patient is not braced after surgery and encouraged to sit up and walk soon afterward as long as no dural leak is encountered.

The anterior approach is less commonly used today. Indications include especially large and rigid curves (greater than 90°), syndromic curves that require additional area for fusion, and potentially curves in young children with risk of crankshaft deformity (progression of the curve from anterior growth even after posterior fusion). It is not clear whether crankshaft deformity is still an issue with pedicle screw instrumentation. The approach may require the services of a general or thoracic surgeon, and if instrumentation below L1 is required, the diaphragm may need to be taken down and later repaired. The vertebral bodies and disks are accessed through a thoracotomy (Fig. 21.14). Thoracic anterior instrumentation

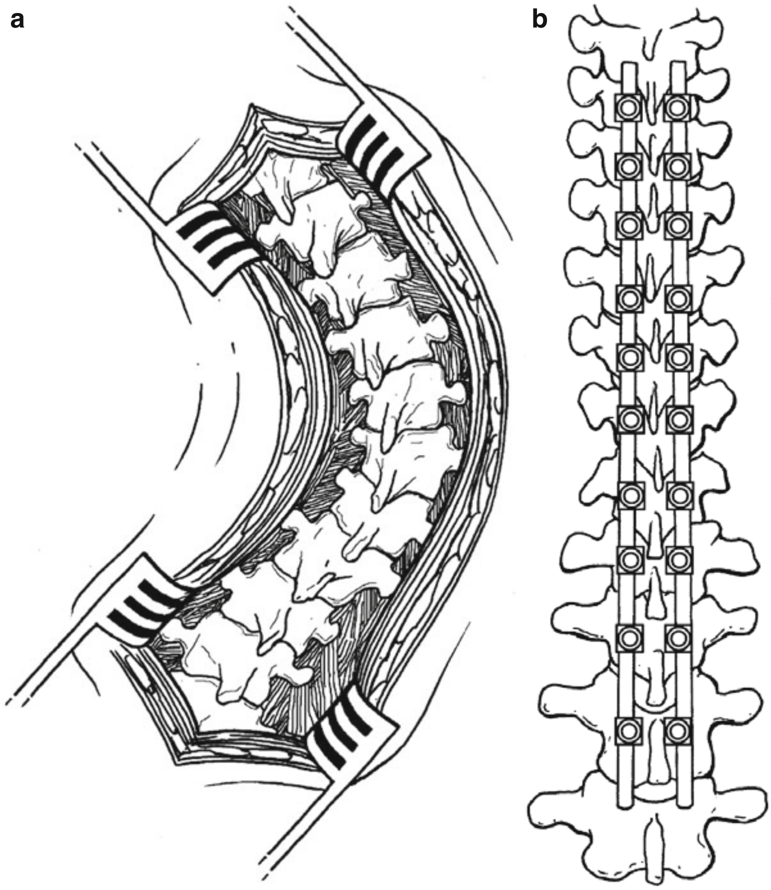


Fig. 21.13 (**a**) Posterior exposure of the spine in scoliosis surgery. (**b**) Segmental posterior spinal instrumentation is used to correct scoliosis

requires the ability to tolerate single-lung ventilation. The anterior approach has a much lower infection rate and less average blood loss than the posterior approach. However, violation of the chest wall appears to result in a decrease in pulmonary function of up to 13 % as measured by PFTs at 2-year follow-up [31]. The significance of this long-term is unclear. This, along with the inconvenience of needing a second surgeon and the more catastrophic risks of vascular injury and blood clots, has reduced the use of anterior surgery. Video-assisted thoracoscopic surgery, or VATS, allows anterior diskectomy and release with minimal injury to the chest wall and no significant permanent decrease in pulmonary function [31]. Instrumentation is also possible through this method, resulting in a very cosmetic correction for small thoracic curves and preserving the function of the paraspinous muscles posteriorly. This became popular briefly for these advantages, but the extreme technical difficulty in performing the procedure caused it to primarily be performed at only a handful of centers, and even these have largely abandoned it in favor of posterior surgery.

21.8.8 Potential Risks and Complications with Scoliosis Surgery

Other than the very unlikely risk of death, the most devastating risk with scoliosis surgery is paraplegia. It is very rare, with a rate of 0.05 % in the latest publication of complications from the Scoliosis Research Society, but it is a permanent harm to a previously healthy patient. Transient neurologic injury and nerve root injury are more common, with a rate of 0.3 %. Overall rate of all neurologic injury for AIS was 0.8 % [51]. To minimize this risk, multimodal spinal cord monitoring is used

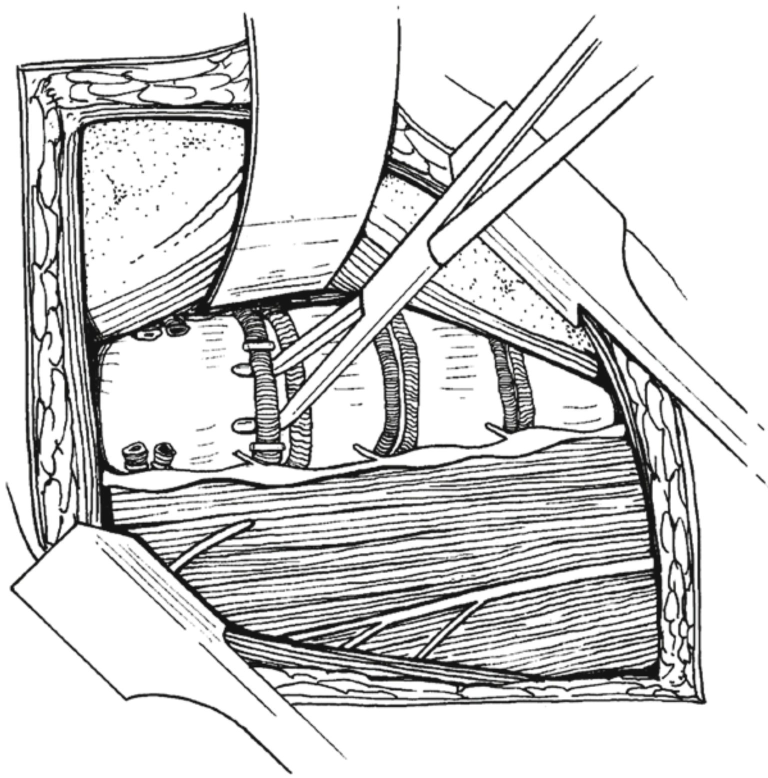


Fig. 21.14 Drawing of anterior exposure of the thoracic spine. Vertebral bodies are exposed through a thoracotomy

throughout the surgery. Current recommended monitoring modalities include somatosensory evoked potentials (SSEPs) and transcranial motor evoked potentials (TcMEPs). Continuous EMG monitoring is also useful, with monitoring of the anal sphincter for potential injury. It is also used for screw stimulation to detect potential for nerve root injury. These modalities can help detect malpositioned implants, stress on the spine from excessive correction, and insufficient spinal blood flow, often from hypotensive anesthesia.

Other potential risks and complications include excessive bleeding, infection, dural leak, failure of spine to fuse, instrumentation failure, and worsening of the curve above or below the instrumentation.

21.8.9 Postoperative Care and Long-Term Follow-Up

Following uncomplicated scoliosis surgery, patients may sit up without restriction and be up walking as soon as the next day. Early mobility is encouraged to prevent respiratory complications and to encourage patient confidence. Total hospital stay is usually about 4–7 days. Patients can return to school about 2–4 weeks after surgery. Walking is the only permitted exercise for the first 6 weeks. Usually after that, running on level surfaces and swimming may safely begin. At 3 months postoperatively, many surgeons allow resumption of noncontact sports. Any physical contact or significantly jarring activities are restricted for about 6 months after surgery to allow fusion. Generally the patient will be monitored with intermittent examinations and X-rays for 2–5 years after the surgery. Once the bone is solidly fused, no further treatment is required. Late infection with *Propionibacterium acnes* or *Staphylococcus epidermidis* may be encountered about 1 year postoperatively or later. By this time, the fusion is usually solid and implants may be removed and left out. Reports from experienced surgeons of creeping or settling of the curve despite fusion have led some to employ early

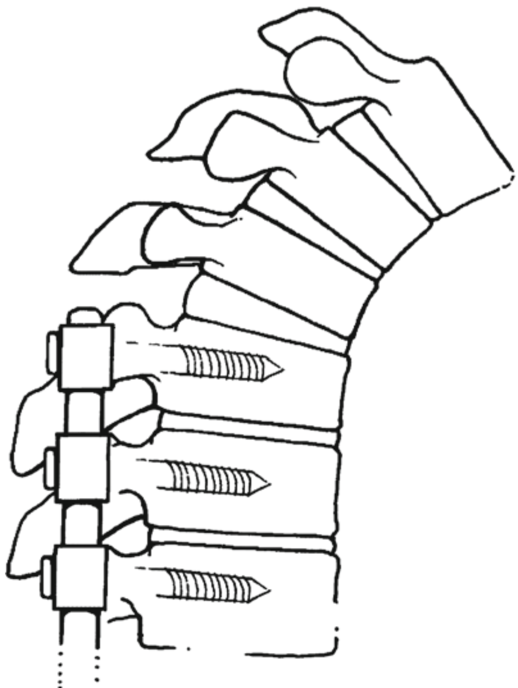


Fig. 21.15 Drawing of proximal junctional kyphosis. This condition can cause pain and necessitate revision of the construct to a higher level

reinstrumentation with titanium implants to prevent the curve from progressing. Some surgeons have switched to use of titanium instrumentation primarily, as it has the ability to resist glycocalyx formation from *Staphylococcus*.

Late decompensation, kyphosis at the ends of the construct (junctional kyphosis), pseudarthrosis, implant failure, irritation from prominent implants, and unexplained pain can all lead to need for revision or implant removal (Fig. 21.15). A workup for infection should always be performed in cases of unexplained pain, implant irritation, and implant failure/suspected pseudarthrosis.

21.8.10 Case History

The patient mentioned in the above case example underwent instrumented posterior spinal fusion from T4–L2. She remained inpatient during the postoperative period and was discharged on the 4th postoperative day. She was advised rest at home and prescribed pain medications. She was followed in the clinic after 2 weeks for surgical wound check. Subsequently she was followed at 6 weeks, 3 months, 6 months, and a year postoperatively with PA and lateral standing thoracolumbar spine X-rays (Fig. 21.16). At 1 year out from her surgery, she is reporting no pain and resumption of all her presurgical activities.

21.9 Infantile Idiopathic Scoliosis

Infantile idiopathic scoliosis is a condition that affects children before the age of 3. Infantile scoliosis is a rare condition, accounting for less than 1 % of all cases of idiopathic scoliosis, but recently it appears to be increasing in incidence. The condition is seen more frequently in boys than girls. For unknown reasons, the curve in the spine tends to bend to the left in infants with scoliosis. These curves may either resolve spontaneously or progress to more severe deformity [30]. The progressive form needs to be treated aggressively, as it can cause severe medical problems including pulmonary insufficiency, leading to early death. Distinguishing between the benign form and the progressive form has become possible radiographically. Mehta's rib-vertebral angle difference (RVAD) purports to classify the curve as either resolving or progressive. The RVA is formed by a line drawn perpendicular to the end plate of the apical vertebra and a line drawn along the center of the rib. The RVAD is calculated by subtracting the angle value of the convex side from that of the concave side [39] (Fig. 21.17). Mehta reported that 83 % of infantile curves with RVAD less than 20° tend to resolve and 84 % curves with RVAD more than 20 showed progression. The progressive curves are treated with serial Mehta body casting under anesthesia, in which carefully applied and molded derotation casts allow control of the curve [38]. These casts must be replaced approximately every 3–4 months for growth of the patient. The best results are obtained when this process can be started by age 12 months. This requires early diagnosis, which

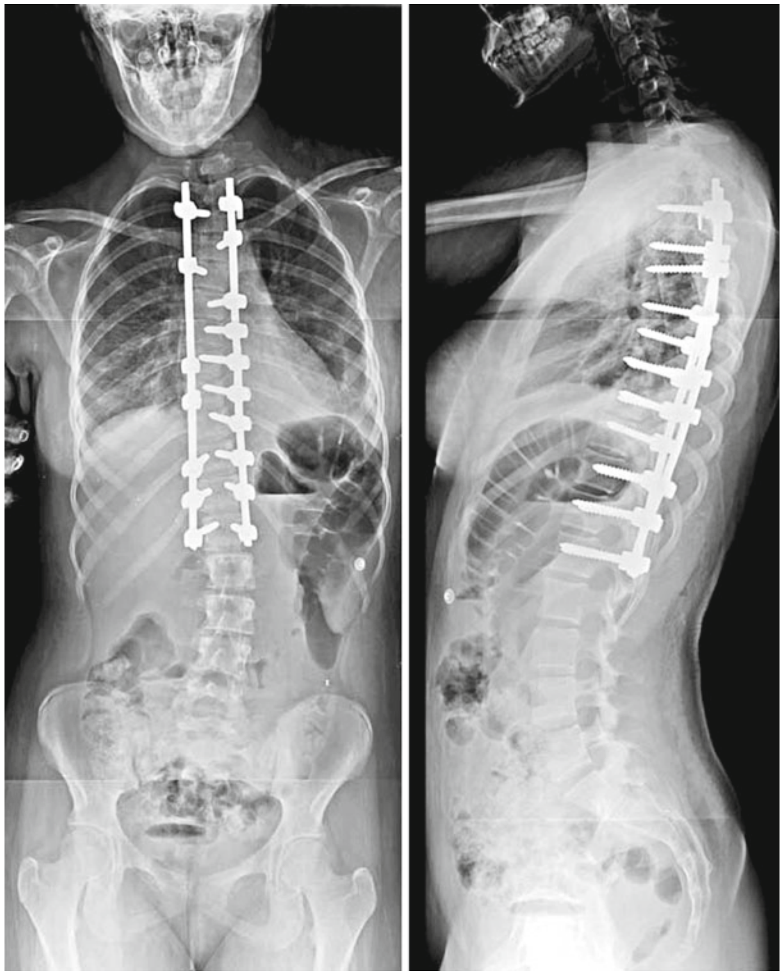


Fig. 21.16 Postoperative follow-up radiographs for patient from Fig. 21.3. Excellent curve correction and balance have been achieved in both planes

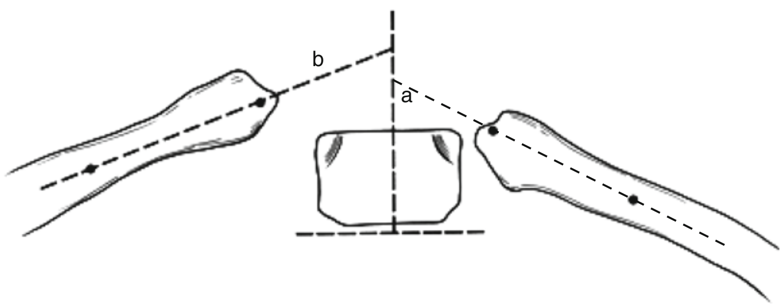


Fig. 21.17 Rib-vertebral angle of Mehta for infantile idiopathic scoliosis. This angle (formed between a and b on the drawing) is measured on both sides of the curve at the apical vertebra. The concave is subtracted from the convex. If the ensuing rib-vertebral angle difference is less than 20°, the curve is likely to spontaneously resolve. Differences greater than 20° are likely to progress and necessitate treatment

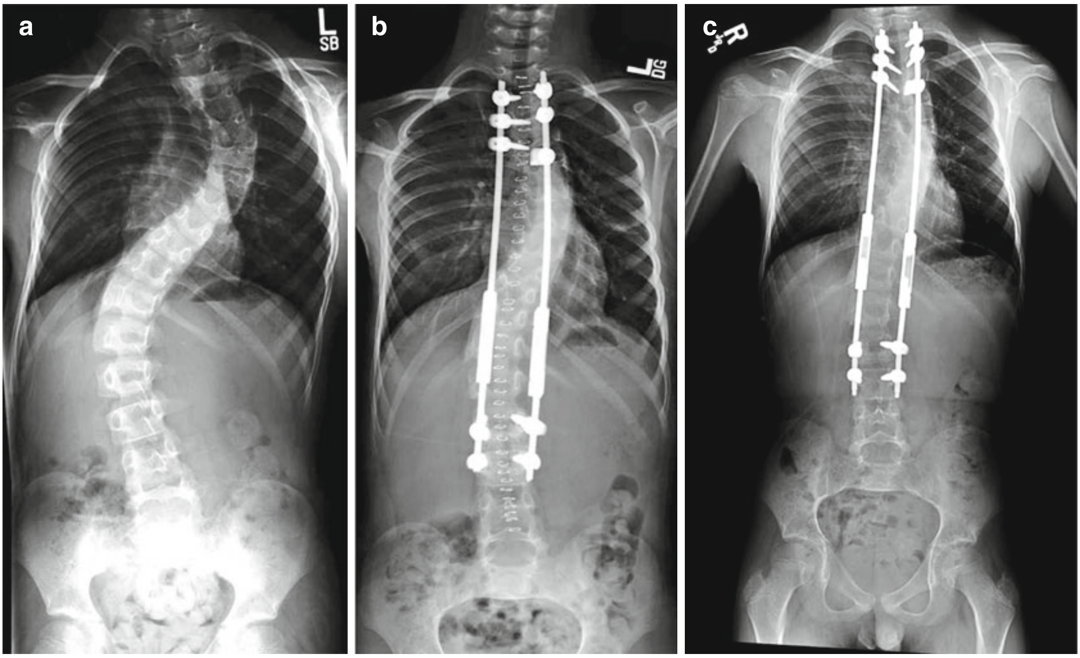


Fig. 21.18 (**a–c**) Pre- and postoperative radiographs of patient treated with "growing rod" construct for juvenile scoliosis. The boxlike structures in the lower part of the construct are lengthening boxes. The third image demonstrates the radiographic appearance of the box, with the ends of the rods distinguishable, after subsequent lengthening procedures

often may not occur. The process of treatment results in significant anesthetic exposure at a young age, though for short procedures, and can be very stressful for families. Once the curve can be brought down to an RVAD of less than 20, or the curve is made as small as it will go on serial casts, the patient may be switched to a well-molded body jacket type TLSO, allowing normal bathing. Careful follow-up every 4–6 months to ensure that control of the curve is not being lost and that the brace is being used 23 h a day is vital, and a noncompliant family will not be able to employ this treatment. The brace will require replacement every 6–12 months because of growth. If control of the curve is lost with bracing, serial casting can be attempted again.

21.9.1 Growth-Sparing Surgery

Curves that are not able to be controlled with casting or bracing may necessitate instrumentation. The best outcomes are obtained when nonoperative treatment can be maintained as long as possible, but when the curve cannot be controlled and continues to progress despite the appropriate treatment, operative strategies that do not promote fusion of the spine are employed. One method involves the placement of minimal implants, at the apex of the curve and at the proximal and distal extents, with fusions of two vertebrae at the top and bottom to form a stable anchor, joined by dual contoured submuscular rods with lengthening boxes or devices. The rods are lengthened against one another as much as possible at the first surgery, employing distraction for curve control and improvement of trunk height, and then the rods are re-lengthened through a small incision every 6 months to push spinal growth and keep the curve from significant progression (Fig. 21.18). The rods may require replacement when no further lengthening can be achieved on them. Infection is common, as are loss of fixation, prominent implants with skin complications, and, most concerningly, premature fusion. The length gained appears to decrease with each successive lengthening until no significant lengthening can be achieved. The hope is that, by the time this occurs, the patient will have had sufficient trunk growth to support adult respiration. The goal of treatment is to support the patient through peak growth velocity without allowing relentless curve progression that would otherwise occur.

Another strategy that has emerged is the use of titanium prosthetic rib expansion devices, originally designated for treatment of Jeune's syndrome and other malignant chest wall deforming diseases that caused early death from pulmonary complications. Uses have been expanded to employ these as distraction devices for the growing spine. Anchors are placed on the ribs proximally and the spine or pelvis distally, and distraction is performed (Fig. 21.19). Lateralization of the implants away from the spine promotes stability, and there is theoretically less risk of premature fusion because no spinal dissection need occur. Long-term follow-up is needed to determine whether this method is better than the dual growing rod method. This method also requires repeat lengthening every 6 months and carries the same associated complications aside from premature fusion, the risk of which is not yet defined. Other issues associated with this method are problems with the anchors, particularly the cradles cutting through the ribs and migrating, and cutting through the pelvis and becoming embedded. For both methods, there is a high rate of unplanned return to the operating room for these various complications.

The oldest strategy to allow growth was the Luque trolley, in which sublaminar wires were used to anchor the spine to paired rods that were left long at the top. The patient was able to grow with the rods merely guiding the spine to remain straight, until the wires came off the top of the rod. No further surgery was required to promote growth. This resulted in problems with proximal junctional kyphosis, but did allow verifiable growth. A version of this employing screws and dual rods, known as the Shilla technique and developed by Dr. Richard McCarthy, was temporarily available in the USA and remedied some of the challenges of the Luque trolley. However, it has currently been taken off the market by the manufacturer.

21.10 Juvenile Idiopathic Scoliosis

Juvenile idiopathic scoliosis is reported to occur among 12–21 % of patients with idiopathic scoliosis [54]. The juvenile form may resemble either of the adolescent and infantile idiopathic scoliosis forms. The reported overall female to male ratio for juvenile idiopathic scoliosis ranges from 2:1 to 4:1. In the early age group, 3–6 years, the female to male ratio is 1:1 with left-sided curve predominance. Among children between the ages of 6 and 10 years, the ratio of affected females to males ranges from 8:1 to 10:1, with right-sided curve predominance. Magnetic resonance imaging (MRI) has shown an association between juvenile scoliosis and intraspinal pathologic changes, most commonly Arnold-Chiari malformation and syringomyelia (4–30 %) [18, 57, 62]. Lewonowski et al. recommended routine MRI examination of all children with scoliosis who are younger than 11 years [29]. Approximately 70 % of curves among patients with juvenile scoliosis progress and necessitate treatment; about one half of patients with curve progression need surgery.

21.11 Congenital Scoliosis

Congenital scoliosis results from abnormally developed vertebrae or ribs, and these changes occur in utero at 4–7 weeks of gestation. As this is the same period in which the heart and kidneys are developing, associated abnormalities are common. Intraspinal anomalies have been reported to occur approximately 30 % of the time with congenital scoliosis [49, 63, 67]. Hence, all the patients at some stage need an MRI of the entire spine, cardiac evaluation, and possible renal ultrasound if the renal anomalies cannot be adequately seen on the spine MRI. Abnormal vertebral development is usually due to sporadic or environmental causes rather than genetic

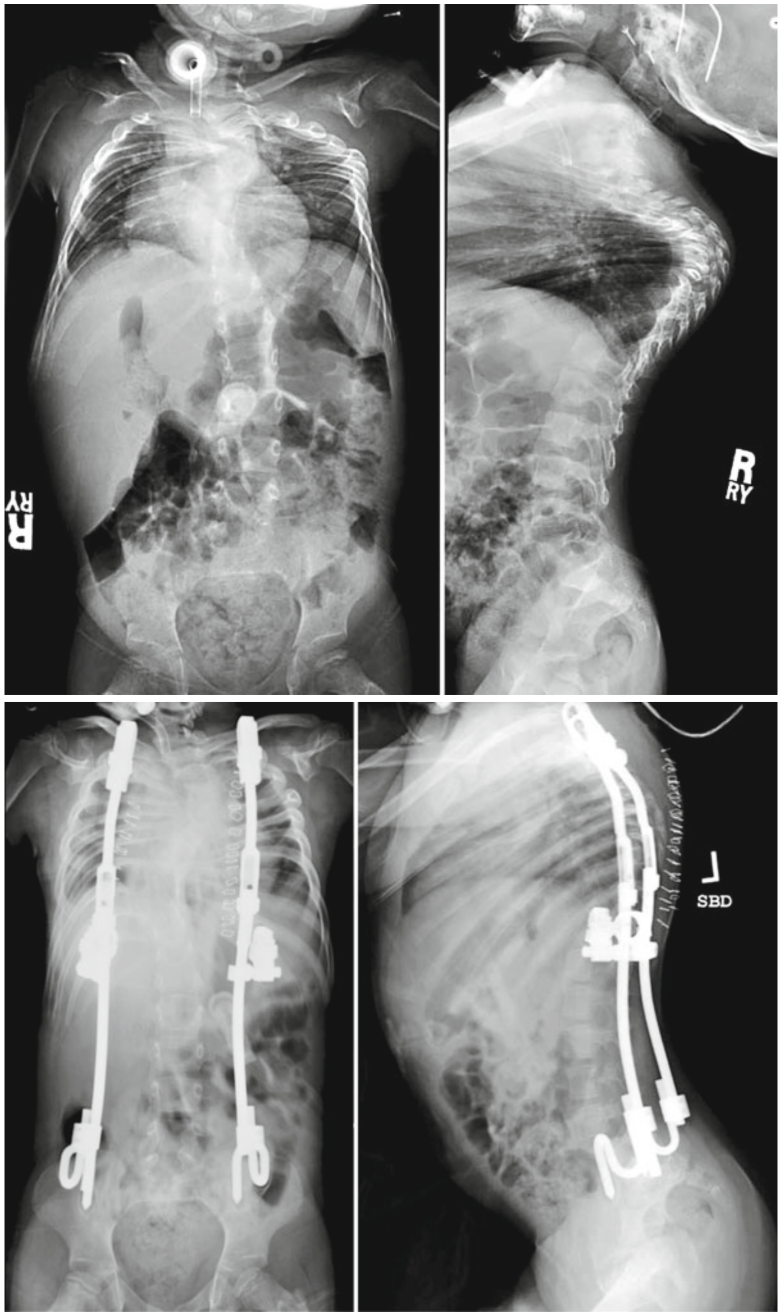


Fig. 21.19 Pre- and postoperative PA and lateral radiographs. Patient with severe scoliosis treated with vertebral expansion prosthetic titanium rib (VEPTR, Synthes) device. Note the improvement of the deformity in both planes

factors. Congenital scoliosis has three classifications: failure of formation of vertebra (hemivertebra), failure of segmentation (unilateral bar/block vertebra), or a combination of both [36, 37, 67] (Fig. 21.20a–c). Recognition of the full extent of the vertebral and rib malformations can be difficult using plain radiographs alone. A CT scan can be helpful, and a 3D reconstruction should be asked for. A CT scan is important in planning surgical intervention.

In congenital scoliosis, curve progression can be predicted by the type of vertebral anomaly, site of anomaly, and age of the patient at the time of diagnosis. The type of anomaly that causes the

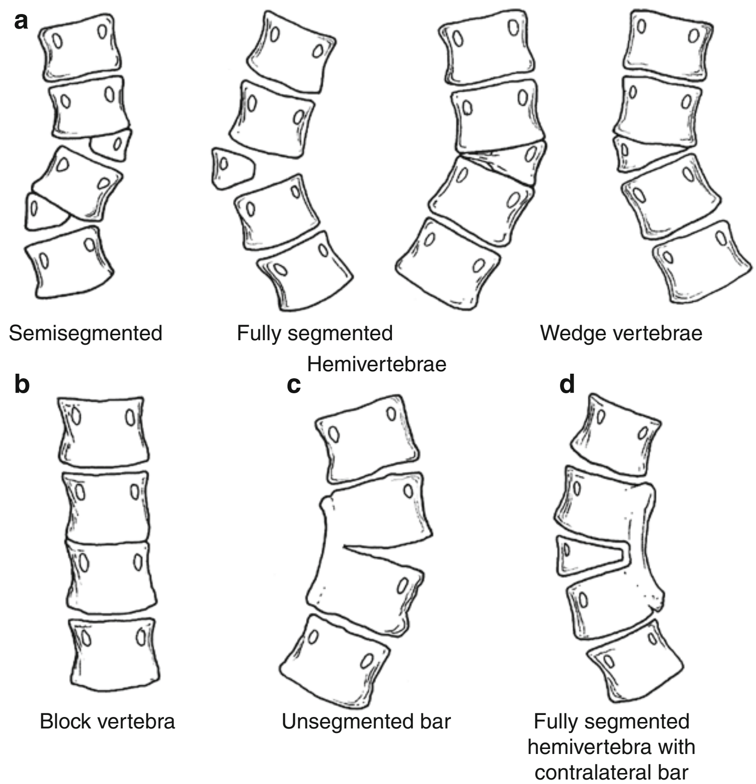


Fig. 21.20 (**a–d**) Types of hemivertebrae are demonstrated in (**a**), representing failure of formation. The failure of segmentation defects are demonstrated in (**b**) and (**c**). The combination of a failure of formation with a failure of segmentation, with the particular pattern of a fully segmented hemivertebra opposite a contralateral bar is the most likely to cause significant scoliosis (**d**)

most severe scoliosis is a unilateral unsegmented bar with contralateral hemivertebra at the same level [36, 37, 67] (Fig. 21.20d). Open disk spaces above and below the hemivertebra indicate the potential for growth imbalance and subsequent scoliosis. The rate of deterioration of the resulting scoliosis is most severe in the thoracic and thoracolumbar regions as compared to upper thoracic and lumbar region.

The objective of treatment in congenital scoliosis is to produce a spine that will be as straight as possible at the end of growth. The treatment plan is made based on the risk of curve progression during growth. Treatment options include observation and surgical intervention. Bracing is usually not helpful. Hemivertebra excision or fusion in situ is often employed when a progressive deformity is present. Early diagnosis while the curve is small with anticipation of growth imbalance may provide an opportunity for prophylactic surgery to prevent a severe deformity. A simple operation to balance the growth of the spine and prevent increasing curvature is preferable to a hazardous multistage surgical procedure to correct a severe and rigid deformity at a later stage, though this is sometimes necessary.

21.12 Neuromuscular Scoliosis

Neuromuscular scoliosis is the curvature of the spine occurring concomitantly with a disorder of the neuromuscular system such as cerebral palsy, spina bifida, muscular dystrophy, or intraspinal pathology [4, 58–60]. The Scoliosis Research Society classified these deformities into neuropathic and myopathic conditions. Neuropathic scoliosis is derived from such disorders as spinal cord injury, syringomyelia, spina bifida, and degenerative neurologic disorders. Myopathic scoliosis is derived from disorders such as Duchenne muscular dystrophy, spinal muscular atrophy, and severe cerebral palsy. Development of scoliosis is extremely variable depending upon the neuromuscular disorder and the ambulatory status of patient. Poor balance and poor

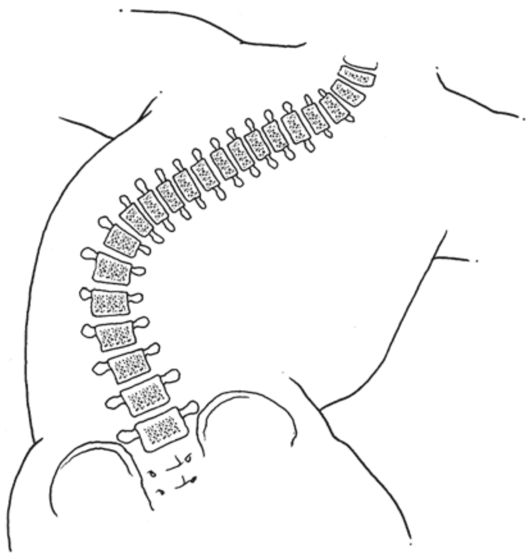


Fig. 21.21 Neuromuscular scoliosis often features a large C-shaped curve without the ability to compensate for the primary deformity

coordination of their trunk, neck, and head; disturbed proprioception; and the pulling action of gravitational forces on the spine predispose them to the development of scoliosis. The curvature of the spine is most rapidly progressive during the preadolescent growth spurt, but continues to progress after skeletal maturity. Many patients require a wheelchair due to underlying neuromuscular conditions. As the trunk muscles weaken, the spine collapses into a C-shaped curve, and the pelvis becomes oblique in the coronal plane making it difficult for the patient to sit upright (Fig. 21.21). Progressive curves may affect the child's ability to be seated comfortably, thereby affecting their quality of life and function. Pressure sores may result when a wheelchair-bound patient with pelvic obliquity is balancing his weight on one ischial tuberosity instead of it being distributed between both. This is most common in insensate patients, but patients with severe cerebral palsy can also be at risk. Their often limited ability to communicate means that caregivers and physicians must be aware of the risk and monitor the curve and the area at risk carefully. The long-term effects of spinal deformity in these neuromuscular conditions are more disabling to the patient than are those of idiopathic scoliosis. Neuromuscular curves can affect digestion, respiration, and even urination [11].

The treatment of neuromuscular scoliosis is individualized. Options include observation, nonoperative treatment such as bracing and seating support, and surgical intervention. Bracing may provide support for the trunk in the seated position while the patient is young and the curve is flexible, but is usually not effective at stopping progression of the curve over time. Seating modifications such as inserts into wheelchairs may help with positioning the child, but are also not corrective in terms of the scoliosis. Seating modifications can help prevent pressure lesions. Surgery is considered in progressive curves to keep upright posture of the patient over a level pelvis (Fig. 21.22). Decision-making is complex and based on expected survival of the child, risks of a large surgery with significant blood loss in a patient with limited reserve, and expectations from the family of how the surgery would affect their ability to care for their child. Surgeons prefer to treat progressive curves when small, 30° in Duchenne muscular dystrophy, for example, because the child's risk of morbidity and mortality from surgery goes up in the teenage years as there is a predictable deterioration of cardiac and respiratory function. However, parents may not see the concern with a curve of 30° that they might not be able to appreciate clinically, and may choose to delay treatment until it may be too late to accomplish the goals of surgery safely for the child. It is important that the surgeon be able to articulate clearly to the family the risks over time, which necessitates knowledge of the underlying disorder. Consultation with the patient's cardiologist, neurologist, or pulmonologist is very helpful in decision-making with the family.

Once surgery has been chosen as the treatment, medical considerations must be taken into account. Patients with neuromuscular scoliosis, across all subtypes, have a much higher baseline risk of infection, wound breakdown, need for ICU care, risk of aspiration, and risk of death. The cost of care and the expected length of stay for these patients are much higher than in the AIS population. Nutritional deficits place patients at the highest risk for infection, so prealbumin and

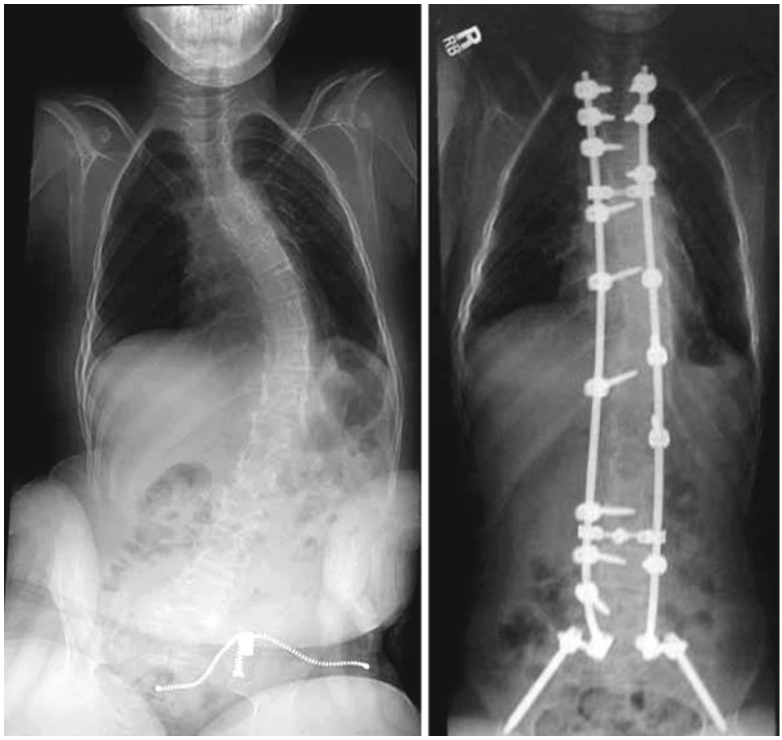


Fig. 21.22 Pre- and postoperative radiographs of patient with neuromuscular scoliosis. A long fusion is usually required, with use of pelvic fixation to restore sitting balance when excess obliquity is present. Here, the pelvis is balanced nicely with excellent positioning of the head over the midline, enabling better seating, posture, and respiratory function

albumin levels should be optimized preoperatively. Antiseizure medications can cause osteomalacia and resultant loss of fixation, so the number and type of implants must be chosen to reflect this [41]. Patients often have bleeding disorders, also likely related to medication, that make the potential for blood loss during surgery profound. The presence of a baclofen pump means higher infection risk in posterior spinal fusion, and if the delivery catheter is damaged or removed during surgery, a severe systemic response can be seen. Oral baclofen has to be resumed carefully after surgery, as it may be metabolized differently in the postoperative period and lead to overdose, which can cause coma. Children who do not swallow safely at baseline have a risk of aspiration after extubation, which may lead to pneumonia and death. Children with cardiopulmonary compromise, such as in the muscular dystrophies, may not be able to be extubated for a prolonged period after surgery, and may even become permanently ventilator dependent. Also, children with neuromuscular disorders may have significant anesthetic risks, such as a risk of malignant hyperthermia in patients with myopathies.

Given the serious medical considerations for these children, a team approach is often helpful, particularly in the postoperative phase. Patients usually spend the first night in the ICU, where a team can assess whether extubation is safe over time. Once extubated, step-down care with a favorable nurse to patient ratio is often used to keep a watchful eye on these children. Medical team consultants are very helpful in safely resuming medications and guiding treatment to restore patients to baseline function. Physical therapy is of limited use, as these children are usually fully dependent on caregivers for mobility, and this does not change after surgery, though it becomes easier.

Questions

1. A 10-year-old premenarchal girl is referred to orthopaedic clinic for evaluation of scoliosis. She is a normal healthy child, active in sports with no previous back complaints. Her physical exam reveals a right thoracic prominence with no neurologic deficit or abnormal reflexes. Her X-rays show right thoracic scoliosis of 28° with a compensatory lumbar curve of 13°. She is

Risser 0, and her triradiate cartilage is still open. What would be the next step in management?
   (a) Observation with repeat radiographs in 6 months
   (b) Bracing with a thoracolumbosacral orthosis
   (c) Posterior spinal fusion with instrumentation
   (d) Magnetic resonance imaging (MRI)
   (e) Do nothing and have her return to the office when she has pain
2. An 8-year-old male presents with a left thoracic rib prominence. Physical exam shows absent abdominal reflexes in the upper and lower quadrants on the left side. Radiographs show a 24° left thoracic curve. What is the next step in management?
   (a) Observation with repeat radiographs in 3 months
   (b) Bracing with a thoracolumbosacral orthosis
   (c) Posterior spinal fusion with instrumentation
   (d) Magnetic resonance imaging (MRI) of the whole spine
   (e) Growing rod instrumentation to allow spine growth
3. Which of the following anatomic patterns of congenital scoliosis is associated with the worst prognosis?
   (a) Fully segmented hemivertebra
   (b) Wedge vertebra
   (c) Block vertebra
   (d) Unsegmented bar
   (e) Unsegmented bar with contralateral hemivertebra
4. A 14-year-old girl is referred to orthopaedic clinic for evaluation of scoliosis. 'Her physical exam reveals a thoracic prominence on the right side and trunk asymmetry. What X-rays you will order?
   (a) Anteroposterior and lateral X-rays of the whole spine
   (b) Right and left bending X-rays in addition to above X-rays
   (c) Anteroposterior and lateral X-rays of the thoracic spine
   (d) Posteroanterior and lateral X-rays of the whole spine
   (e) No X-rays needed

References

1. Asher M, Green P, Orrick J (1980) A six-year report: spinal deformity screening in Kansas school children. J Kans Med Soc 81(12):568–571
2. Bell M, Teebi AS (1995) Autosomal dominant idiopathic scoliosis? Am J Med Genet 55(1):112
3. Betz RR, Ranade A, Samdani AF, Chafetz R, D'Andrea LP, Gaughan JP, Asghar J, Grewal H, Mulcahey MJ (2010) Vertebral body stapling: a fusionless treatment option for a growing child with moderate idiopathic scoliosis. Spine 35(2): 169–176
4. Bradford DH (1995) Moe's textbook of scoliosis. In: Lonstein JE, Bradford D, Winter R, Ogilvie JW (eds) Neuromuscular spinal deformities. WB Saunders, Philadelphia, p 295
5. Brooks HL, Azen SP, Gerberg E, Brooks R, Chan L (1975) Scoliosis: a prospective epidemiological study. J Bone Joint Surg Am 57(7):968–972
6. Bunnell WP (1984) An objective criterion for scoliosis screening. J Bone Joint Surg Am 66(9):1381–1387
7. Burwell RG, James NJ, Johnson F, Webb JK, Wilson YG (1983) Standardised trunk asymmetry scores. A study of back contour in healthy school children. J Bone Joint Surg Br 65(4):452–463
8. Cheng JC, Guo X, Sher AH (1999) Persistent osteopenia in adolescent idiopathic scoliosis. A longitudinal follow up study. Spine 24(12):1218–1222
9. Cheung KM, Luk KD (1997) Prediction of correction of scoliosis with use of the fulcrum bending radiograph. J Bone Joint Surg Am 79(8):1144–1150
10. Cobb J (1948) Outline for the study of scoliosis. Instr Course Lect 5:261
11. Comstock CP, Leach J, Wenger DR (1998) Scoliosis in total-body involvement cerebral palsy: analysis of surgical treatment and patient and caregiver satisfaction. Spine 23:1412–1425
12. Cowell HR, Hall JN, MacEwen GD (1972) Genetic aspects of idiopathic scoliosis. A Nicholas Andry Award essay, 1970. Clin Orthop Relat Res 86:121–131
13. Czeizel A, Bellyei A, Barta O, Magda T, Molnar L (1978) Genetics of adolescent idiopathic scoliosis. J Med Genet 15(6):424–427
14. Daruwalla JS, Balasubramanian P, Chay SO, Rajan U, Lee HP (1985) Idiopathic scoliosis: prevalence and ethnic distribution in Singapore schoolchildren. J Bone Joint Surg Br 67(2):182–184
15. Do T, Fras C, Burke S, Widmann RF, Rawlins B, Boachie-Adjei O (2001) 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 83-A(4):577–579
16. Donaldson S, Stephens D, Howard A, Alman B, Narayanan U, Wright JG (2007) Surgical decision making in adolescent idiopathic scoliosis. Spine 32(14):1526–1532

17. Frank ED, Stears JG, Gray JE, Winkler NT, Hoffman AD (1983) Use of the posteroanterior projection: a method of reducing x-ray exposure to specific radiosensitive organs. Radiol Technol 54(5):343–347
18. Gupta P, Lenke LG, Bridwell KH (1998) Incidence of neural axis abnormalities in infantile and juvenile patients with spinal deformity. Is a magnetic resonance image screening necessary? Spine 23(2): 206–210
19. Inoue M, Minami S, Kitahara H, Otsuka Y, Nakata Y, Takaso M, Moriya H (1998) Idiopathic scoliosis in twins studied by DNA fingerprinting: the incidence and type of scoliosis. J Bone Joint Surg Br 80(2):212–217
20. Kane WJ (1977) Scoliosis prevalence: a call for a statement of terms. Clin Orthop Relat Res 126:43–46
21. Kane WJ, Moe JH (1970) A scoliosis-prevalence survey in Minnesota. Clin Orthop Relat Res 69:216–218
22. Kesling KL, Reinker KA (1997) Scoliosis in twins. A meta-analysis of the literature and report of six cases. Spine 22(17):2009–2014; discussion 2015
23. Kindsfater K, Lowe T, Lawellin D, Weinstein D, Akmakjian J (1994) Levels of platelet calmodulin for the prediction of progression and severity of adolescent idiopathic scoliosis. J Bone Joint Surg Am 76(8):1186–1192
24. Lavelle WF, Samdani AF, Cahill PJ, Betz RR (2011) Clinical outcomes of nitinol staples for preventing curve progression in idiopathic scoliosis. J Pediatr Orthop 31(1S):S107–S113
25. Lenke LG, Betz RR, Clements D, Merola A, Haher T, Lowe T, Newton PO, Bridwell KH, Blanke K (2002) Curve prevalence of a new classification of operative adolescent idiopathic scoliosis: does classification correlate with treatment? Spine 27(6):604–611
26. Lenke LG, Betz RR, Harms J, Bridwell KH, Clements DH, Lowe TG, Blanke K (2001) Adolescent idiopathic scoliosis: a new classification to determine extent of spinal arthrodesis. J Bone Joint Surg Am 83-A(8):1169–1181
27. Lenke LG, Edwards CC, Bridwell KH (2003) The Lenke classification of adolescent idiopathic scoliosis: how it organizes curve patterns as a template to perform selective fusions of the spine. Spine 28(20): S199–S207
28. Levy AR, Goldberg MS, Mayo NE, Hanley JA, Poitras B (1996) Reducing the lifetime risk of cancer from spinal radiographs among people with adolescent idiopathic scoliosis. Spine 21(13):1540–1547; discussion 1548
29. Lewonowski K, King JD, Nelson MD (1992) Routine use of magnetic resonance imaging in idiopathic scoliosis patients less than eleven years of age. Spine 17(6 Suppl):S109–S116
30. Lloyd-Roberts GC, Pilcher MF (1965) Structural idiopathic scoliosis in infancy: a study of the natural history of 100 patients. J Bone Joint Surg Br 47:520–523
31. Lonner BS, Auerbach JD, Estreicher MB, Betz RR, Crawford AH, Lenke LG, Newton PO (2009) Pulmonary function changes after various anterior approaches in the treatment of adolescent idiopathic scoliosis. J Spinal Disord Tech 22(8):551–558
32. Lonstein JE, Bjorklund S, Wanninger MH, Nelson RP (1982) Voluntary school screening for scoliosis in Minnesota. J Bone Joint Surg Am 64(4):481–488
33. Lonstein JE, Carlson JM (1984) The prediction of curve progression in untreated idiopathic scoliosis during growth. J Bone Joint Surg Am 66(7):1061–1071
34. Lonstein JE, Winter RB (1994) The Milwaukee brace for the treatment of adolescent idiopathic scoliosis. A review of one thousand and twenty patients. J Bone Joint Surg Am 76(8):1207–1221
35. Maenza RA (2003) Juvenile and adolescent idiopathic scoliosis: magnetic resonance imaging evaluation and clinical indications. J Pediatr Orthop B 12(5): 295–302
36. McMaster M (1994) Congenital scoliosis. In: Weinstein SL (ed) The pediatric spine: principles and practice. Raven Press, New York, p 227
37. McMaster MJ (1998) Congenital scoliosis caused by a unilateral failure of vertebral segmentation with contralateral hemivertebrae. Spine 23(9):998–1005
38. Mehta MH (2005) Growth as a corrective force in the early treatment of progressive infantile scoliosis. J Bone Joint Surg Br 87(9):1237–1247
39. Mehta MH (1972) The rib-vertebra angle in the early diagnosis between resolving and progressive infantile scoliosis. J Bone Joint Surg Br 54(2):230–243
40. Mejia EA, Hennrikus WL, Schwend RM, Emans JB (1996) A prospective evaluation of idiopathic left thoracic scoliosis with magnetic resonance imaging. J Pediatr Orthop 16(3):354–358
41. Modi HN, Suh SW, Yang JH, Cho JW, Hong JY, Singh SU, Jain S (2009) Surgical complications in neuromuscular scoliosis operated with posterior-only approach using pedicle screw fixation. Scoliosis 4:11
42. Montgomery F, Willner S (1997) The natural history of idiopathic scoliosis. Incidence of treatment in 15 cohorts of children born between 1963 and 1977. Spine 22(7):772–774
43. Nachemson A (1968) A long term follow-up study of non-treated scoliosis. Acta Orthop Scand 39(4): 466–476
44. Nachemson AL, Peterson LE (1995) 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 77(6): 815–822
45. Newton PO, Faro FD, Lenke LG, Betz RR, Clements DH, Lowe TG, Haher TR, Merola AA, D'Andrea LP, Marks M, Wenger DR (2003) Factors involved in the decision to perform a selective versus nonselective fusion of Lenke 1B and 1C (King-Moe II) curves in

adolescent idiopathic scoliosis. Spine 28(20): S217–S223
46. O'Brien MF, Lenke LG, Bridwell KH, Blanke K, Baldus C (1994) Preoperative spinal canal investigation in adolescent idiopathic scoliosis curves > or = 70 degrees. Spine 19(14):1606–1610
47. O'Leary PT, Sturm PF, Hammerberg KW, Lubicky JP, Mardjetko SM (2011) Convex hemiepiphysiodesis: the limits of vertebral stapling. Spine 36(19): 1579–1583
48. Ouellet JA, LaPlaza J, Erickson MA, Birch JG, Burke S, Browne R (2003) Sagittal plane deformity in the thoracic spine: a clue to the presence of syringomyelia as a cause of scoliosis. Spine 28(18):2147–2151
49. Prahinski JR, Polly DW, McHale KA, Ellenbogen RG (2000) Occult intraspinal anomalies in congenital scoliosis. J Pediatr Orthop 20(1):59–63
50. Ramirez N, Johnston CE, Browne RH (1997) The prevalence of back pain in children who have idiopathic scoliosis. J Bone Joint Surg Am 79(3): 364–368
51. Reames DL, Smith JS, Fu KM, Polly DW, Ames CP, Berven SH, Perra JH, Glassman SD, McCarthy RE, Knapp RD, Heary R, Shaffrey CI, Scoliosis Research Society Morbidity and Mortality Committee (2011) Complications in the surgical treatment of 19,360 cases of pediatric scoliosis: a review of the Scoliosis Research Society Morbidity and Mortality database. Spine 36(18):1484–1491
52. Riseborough EJ, Wynne-Davies R (1973) A genetic survey of idiopathic scoliosis in Boston, Massachusetts. J Bone Joint Surg Am 55(5):974–982
53. Risser JC (1958) The iliac apophysis; an invaluable sign in the management of scoliosis. Clin Orthop 11:111–119
54. Robinson CM, McMaster MJ (1996) Juvenile idiopathic scoliosis. Curve patterns and prognosis in one hundred and nine patients. J Bone Joint Surg Am 78(8):1140–1148
55. Rogala EJ, Drummond DS, Gurr J (1978) Scoliosis: incidence and natural history. A prospective epidemiological study. J Bone Joint Surg Am 60(2):173–176
56. Rowe DE, Bernstein SM, Riddick MF, Adler F, Emans JB, Gardner-Bonneau D (1997) A meta-analysis of the efficacy of non-operative treatments for idiopathic scoliosis. J Bone Joint Surg Am 79(5):664–674
57. Schwend RM, Hennrikus WL, Hall JE, Emans JB (1995) Childhood scoliosis: clinical indications for magnetic resonance imaging. J Bone Joint Surg Am 77(1):46–53
58. Shapiro F, Bresnan MJ (1982) Orthopaedic management of childhood neuromuscular disease. Part I: spinal muscular atrophy. J Bone Joint Surg Am 64(5):785–789
59. Shapiro F, Bresnan MJ (1982) Orthopaedic management of childhood neuromuscular disease. Part II: peripheral neuropathies, Friedreich's ataxia, and arthrogryposis multiplex congenita. J Bone Joint Surg Am 64(6):949–953
60. Shapiro F, Bresnan MJ (1982) Orthopaedic management of childhood neuromuscular disease. Part III: diseases of muscle. J Bone Joint Surg Am 64(7): 1102–1107
61. Skogland LB, Miller JA (1980) Growth related hormones in idiopathic scoliosis. An endocrine basis for accelerated growth. Acta Orthop Scand 51(5):779–780
62. Sponseller PD (1992) Syringomyelia and Chiari I malformation presenting with juvenile scoliosis as sole manifestation. J Spinal Disord 5(2):237–239; discussion 239–244
63. Suh SW, Sarwark JF, Vora A, Huang BK (2001) Evaluating congenital spine deformities for intraspinal anomalies with magnetic resonance imaging. J Pediatr Orthop 21(4):525–531
64. Tanner JM, Whitehouse RH, Takaishi M (1966) Standards from birth to maturity for height, weight, height velocity, and weight velocity: British children, 1965. I. Arch Dis Child 41(219):454–471
65. Tanner JM, Whitehouse RH, Takaishi M (1966) Standards from birth to maturity for height, weight, height velocity, and weight velocity: British children, 1965. II. Arch Dis Child 41(220):613–635
66. Weinstein SL, Ponseti IV (1983) Curve progression in idiopathic scoliosis. J Bone Joint Surg Am 65(4): 447–455
67. Winter R (1994) Moe's textbook of scoliosis. In: Lonstein J, Winter R, Ogilvie J (eds) Congenital spinal deformity. WB Saunders, Philadelphia, p 257

22 Pediatric Kyphosis

Michael J. Kramarz, Steven W. Hwang,
Amer F. Samdani, and Phillip B. Storm

22.1 Introduction

This chapter will discuss our current understanding and provide a general overview of various causes of thoracic kyphosis in the pediatric population. We will discuss the development of the thoracic sagittal profile and then introduce fundamental principles surrounding the more commonly encountered causes of thoracic hyperkyphoses such as Scheuermann kyphosis, postural kyphosis, iatrogenic kyphosis, and congenital kyphosis. Although we will discuss various etiologies of hyperkyphosis, Scheuermann kyphosis will be highlighted as it is the most commonly encountered reason for surgical intervention.

Scheuermann kyphosis, historically called idiopathic kyphosis, is a pathologic developmental process in which vertebral wedging causes increased kyphosis [1, 3, 31]. The condition originally was described as a rigid thoracic kyphosis with wedging of vertebral bodies. Type I involves thoracic kyphosis with an apex at T6–8 (subtype a) or an apex at the thoracolumbar junction (subtype b). In Type II, lumbar subtype of Scheuermann disease, the predominant deformity is in the thoracolumbar or lumbar spine, typically with thoracic hypokyphosis or even lordosis. These conditions are most commonly seen in adolescent boys who are physically active or do heavy lifting [9]. Patients initially present with localized back pain, with or without a clinical or cosmetic deformity. Radiographs reveal vertebral end plate irregularities and Schmorl nodes at and below the thoracolumbar junction, with a loss of lumbar lordosis. The etiology is unknown, although the epidemiology suggests a mechanical cause. Despite their radiographic similarity, thoracic (type I) and lumbar (type II) Scheuermann disease may be different pathophysiologic entities. Lumbar Scheuermann disease appears to be nonprogressive, and its symptoms usually resolve over time with the use of nonsteroidal anti-inflammatory drugs and activity modification [33].

22.2 Normal Spine Kyphosis and Development

Infants are born with a mild degree of spinal kyphosis. As children develop and begin to elevate their heads and ambulate, thoracic kyphosis increases and lumbar and cervical lordosis develops. The overall curvature of the spine in the

M.J. Kramarz, B.S.E. • P.B. Storm, M.D. (✉)
Division of Neurosurgery,
Children's Hospital of Philadelphia,
Wood Center, 6th Floor, 34th and Civic Center Blvd,
Philadelphia, PA 19104, USA
e-mail: mkramarz@mail.med.upenn.edu;
storm@email.chop.edu

S.W. Hwang, M.D.
Department of Neurosurgery and Orthopedic Surgery,
Tufts Medical Center,
Boston, MA, 02111, USA

A.F. Samdani, M.D.
Department of Pediatric Spine,
Shriners Hospitals for Children,
Philadelphia, PA, 19140, USA

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_22, © Springer-Verlag Berlin Heidelberg 2014

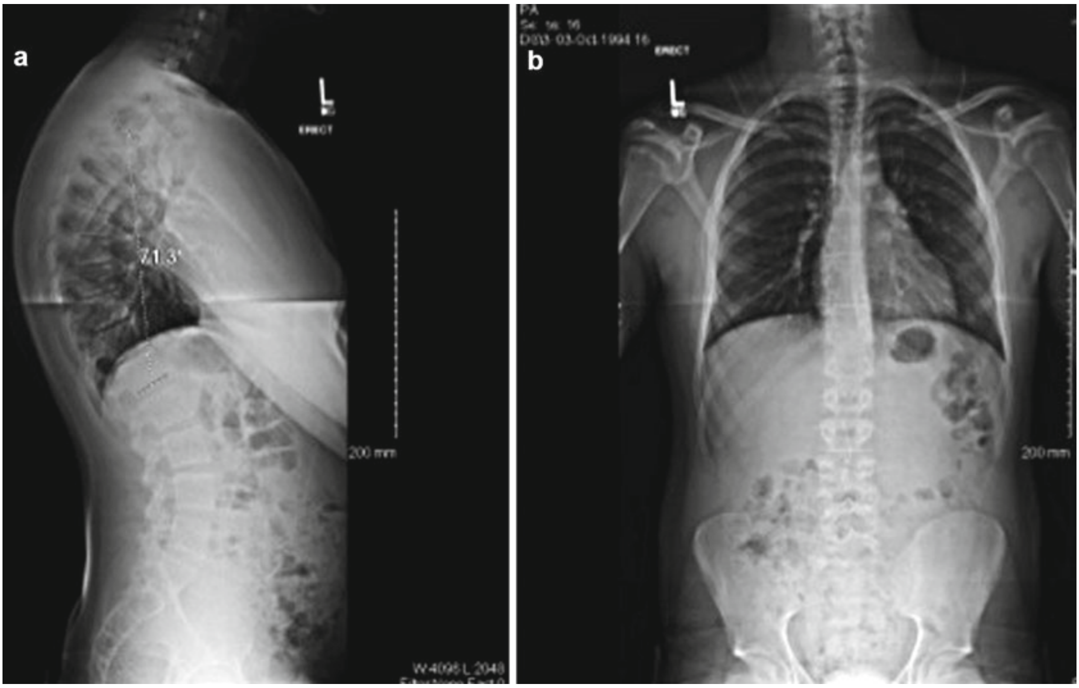


Fig. 22.1 (**a**) Standing lateral radiograph showing the rigid, fixed thoracic kyphosis centered at T9, measuring 71°. (**b**) Standing PA radiograph displaying no scoliosis in a 14-year-old boy

sagittal plane permits the head to be centered over the pelvis [7]. Spine curvature is inferred to be neutral when a plumb line from C7 intersects the posterior edge of the sacral end plate [11, 12]. Cervical alignment, thoracic kyphosis, lumbar lordosis, pelvic tilt, and sacral slope interact to maintain postural alignment.

Thoracic kyphosis ideally should be measured from the end plate of T2–T12. Frequently, however, radiographic obscuration from the shoulders requires that the thoracic kyphosis be measured from T5 to T12. Normal thoracic kyphosis in a young adult is generally 20–40° [4], but it can be greater because of a physiologic or pathologic process.

22.3 Case Example

A 14-year-old boy presents complaining of moderate back pain in the mid-thoracic region. The pain gradually worsens throughout the day and with increasing activity and improves with rest. Some mild relief is obtained with nonsteroidal anti-inflammatory drugs (NSAIDs) or acetaminophen. The parents state that he has always had bad posture but recently has lost some weight, and they have noted a prominent bump on his back. He is neurologically intact and has some mild tenderness to palpation in the mid-thoracic region. He also has tight hamstrings on examination and has a reduced popliteal angle. He stands with his shoulders slouched forward and is unable to straighten completely even with hyperextension. Plain PA and lateral radiographs reveal a 71° thoracic kyphosis and no scoliosis (Fig. 22.1).

22.4 Pathophysiology

The pathophysiology of Scheuermann kyphosis remains unclear. The proposed etiologies include osteonecrosis of the vertebral ring, weakening of the cartilaginous end plate, juvenile osteoporosis, abnormal cartilaginous matrix, abnormal growth hormone levels, and genetic predisposition. However, currently no definitive pathologic etiology has been identified.

22.5 History and Physical

Scheuermann kyphosis is the most common cause of hyperkyphosis in adolescents, in whom the reported prevalence ranges from 0.4 to 10 % [4, 31]. Boys age 10–15 years are most commonly affected, although some studies report an equal incidence among girls and boys [34]. Often the child's physical appearance is attributed to poor posture. As a result, an affected child is likely to develop postural concerns, a poor self-image, or back pain and may avoid participating in athletic activities.

Typically, there is a well-demarcated angular thoracic or thoracolumbar kyphosis, which is accentuated by forward bending, and a compensatory hyperlordosis of the lumbar spine. The apex is typically at T7–T9. The compensatory changes involving the cervical and lumbar lordosis contribute to a characteristic head and neck thrust associated with Scheuermann kyphosis (Fig. 22.2). The kyphosis is relatively rigid and typically cannot be corrected to the normal range with hyperextension. Some patients have associated scoliosis or contractures of the pectoralis, hip flexor, or hamstring muscles. Often, mild degree of scoliosis can be identified on Adam's forward bending using a scoliometer.

22.6 Differential Diagnosis

22.6.1 Postural Kyphosis

Postural kyphosis, or familial round-back deformity, is a flexible kyphosis that does not have the sharp, angulated, rigid pattern of deformity characteristic of Scheuermann kyphosis [1]. Postural kyphosis should be included in the differential diagnosis for a patient with thoracic kyphosis, although this condition does not have pathologic connotations. With forward bending, the patient has a gradual, gentle kyphosis in the sagittal plane that is easily corrected by standing erect or lying prone. Radiographs do not reveal anterior vertebral wedging or end plate irregularities. Although the patient may have mild back pain, the natural history of postural kyphosis is benign. Observation is recommended for an asymptomatic child. Nonsteroidal anti-inflammatory drugs and physical therapy with postural training exercises, hamstring stretching, and back extensor strengthening form the basis of treatment for back pain accompanying postural kyphosis.

22.6.2 Iatrogenic Kyphosis

Acquired kyphosis has been widely reported after laminectomy of the cervical or thoracic spine in children [9, 35]. Most studies of pediatric iatrogenic spine deformity pertain to the cervical spine, and the reported risk of cervical post-laminectomy kyphosis in the pediatric population ranges from 14 to 100 % [2, 9, 28, 35]. The risk of pediatric deformity has been linked to C2 involvement, relatively young patient age, preoperative malalignment, and irradiation. In surgery for Chiari malformations, the risk of postoperative kyphosis appears to increase when the cervical decompression extends below C2 [10].

Although most studies suggest that iatrogenic deformity more commonly develops in the cervical spine, one study found that the thoracic-thoracolumbar spine had a greater incidence of deformity (60 % versus 25 %, $P = 0.07$) [36]. There was a 16.6 % risk of kyphotic deformity after laminectomies at two or more levels in the thoracolumbar region. Patients younger than 18 years of age had a 50 % incidence of deformity, but those aged 18–30 years had only a 9 % risk [22]. The reported incidence of iatrogenic deformity in the pediatric lumbar spine varies considerably, but deformity appears to occur less frequently in the lumbar spine than in either the thoracic or cervical segments. The lordotic curvature of the lumbar spine may protect it against the development of kyphotic deformity. However, the current understanding of this pathology in children is limited.

Bracing appears to have a limited role in the long-term management of iatrogenic kyphosis and is inconclusive in preventing deformity. However, bracing can be useful to allow surgical intervention to be postponed until the patient

Fig. 22.2 (**a**) Lateral picture of a patient with Scheuermann kyphosis. Note the rib prominence and anterior displacement of the chin. (**b**) Corresponding lateral x-ray

reaches skeletal maturity or can better tolerate a surgical intervention.

During the initial surgical procedure, the surgeon should attempt to minimize the anatomic exposure and preserve as much of the facet capsule and facet joint as possible. The surgeon also should avoid involving C2, limit the exposure to as few levels as necessary, and consider performing laminoplasties [23]. The same techniques are used in the cervical and thoracic spine to limit the risk of iatrogenic kyphosis. A concurrent instrumented stabilization should be considered when junctional levels are to be bridged or the patient has a preexisting deformity, significant remaining growth potential (generally Risser 0 or 1), planned adjuvant radiation therapy, or neuromuscular compromise (Fig. 22.3). A variety of osteoplastic laminoplasty techniques have been designed to preserve the dorsal structures. However, little comparative research has been reported for the

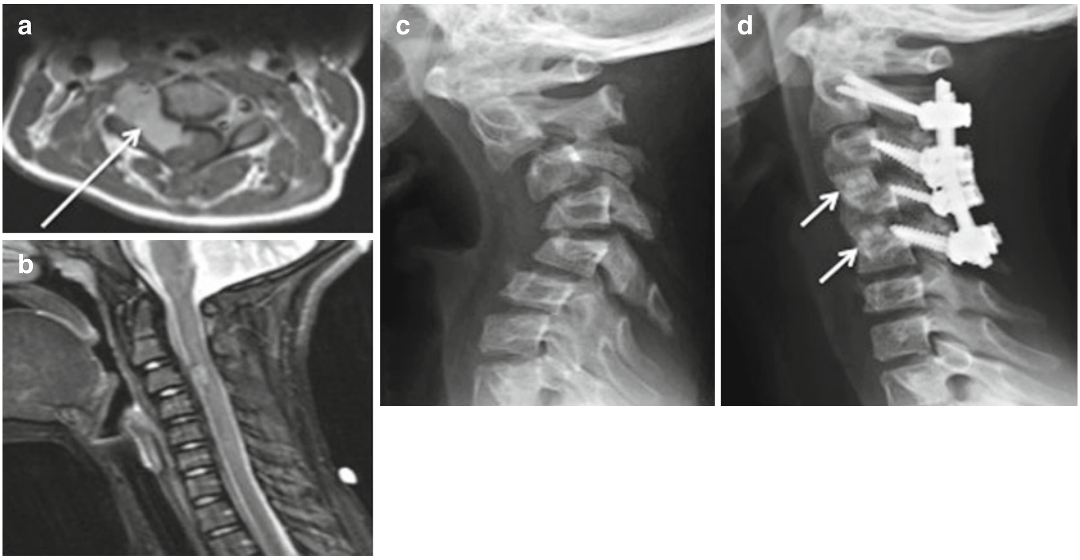


Fig. 22.3 Preoperative. (**a**) Axial MR image of an 11-year-old girl showing tumor (*arrow*) and (**b**) sagittal MR image showing the small degree of straightening of cervical lordosis. (**c**) Lateral radiograph showing iatrogenic cervical kyphosis that developed post surgery. (**d**) Lateral radiograph after anterior cervical discectomies and fusion with PEEK cages (*arrows*) and posterior instrumentation

pediatric population. In a retrospective comparison of patients treated with laminectomy or laminoplasty after intramedullary tumor resection, the rate of deformity was lower after laminoplasty [18]. After tumor resection, the rate of moderate to severe deformity development was significantly lower in patients who received instrumentation than in patients with laminectomies at more than four levels (10 % versus 56 %) [29]. Another benefit of upfront instrumentation in patients who have tumors that require post-resection radiation is the difficulty of bony fusion after radiation therapy [19]. As a result of failed bony fusion after radiation therapy, we are more proactive about instrumenting at the time of the original surgery or, if they present after radiation therapy and need instrumentation, we are using an autologous vascularized free fibular graft [14] (Fig. 22.4).

The deformity may develop immediately after the initial surgery or up to 74 months later. Early intervention and frequent follow-up may help limit the severity of deformity progression (Fig. 22.5). In patients with thin pedicles, pedicle screw placement can be very difficult (Fig. 22.6). For these patients, pedicle hooks, sublaminar wiring, or the "in-out-in" technique can be used (Fig. 22.7). The "in-out-in" technique involves starting a thoracic pedicle screw *in* the pedicle, allowing it the break *out* laterally, purchase the rib, and go back *in* the vertebral body. Not all patients require surgical intervention, but progressive deformity, neurologic symptoms, or intractable pain warrant surgical correction. A ventral approach is often preferred to correct a severe iatrogenic kyphotic deformity of the cervical spine and stabilize the spine. In some patients, a combined ventral and dorsal stabilization procedure may be required, but with the current expandable cages, even the most complex kyphoses can be managed with a vertebral column resection through a posterior costotransversectomy (Fig. 22.8). Other techniques to correct thoracolumbar deformities are Ponte and Smith-Pete osteotomies and pedicle subtraction osteotomies [21, 27].

22.6.3 Congenital Kyphosis

Congenital scoliosis is rare and of unclear etiology; it is estimated to occur in 0.1 % of births and is related to hemivertebra formation. Congenital

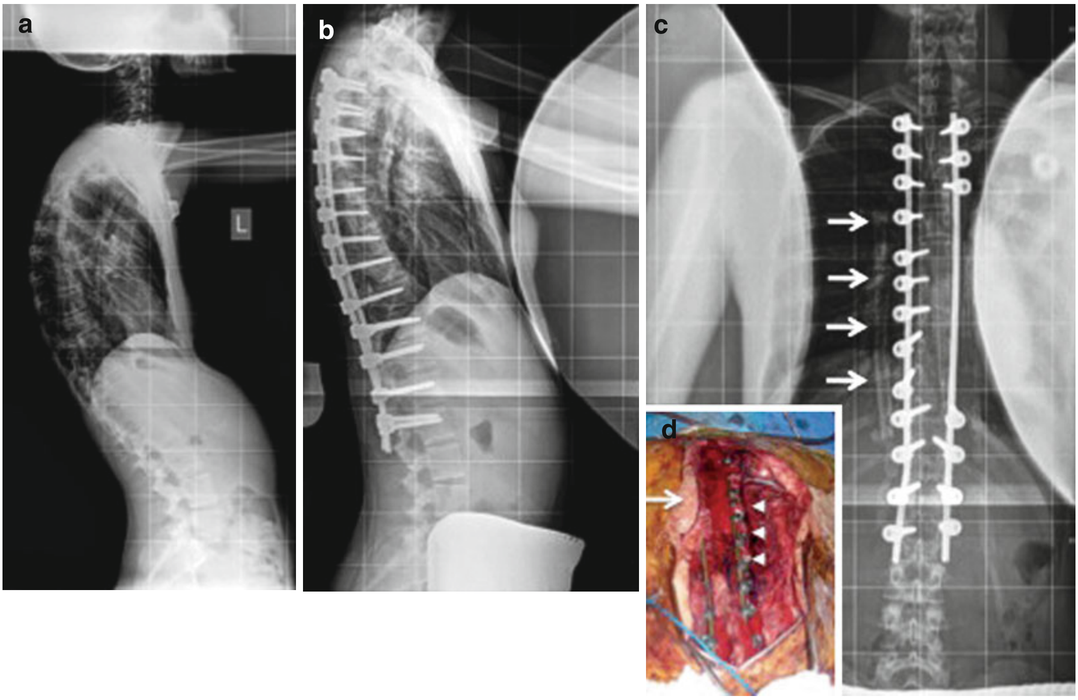


Fig. 22.4 (**a**) Standing scoliosis lateral radiograph showing patient's kyphosis. (**b**) Postoperative standing scoliosis lateral radiograph showing correction of kyphosis following monoaxial pedicle screw and rod fusion with vascularized fibular graft. (**c**) Postoperative AP radiograph with fibular graft visible (*arrows*). (**d**) Intraoperative photograph showing instrumented fusion, the latissimus dorsi free flap (*arrow*) and overlying skin, vascularized fibula (*arrowheads*) with a saphenous vein graft (*asterisk*) connecting to the fibular artery and vein, and the axillary artery and vein [19]

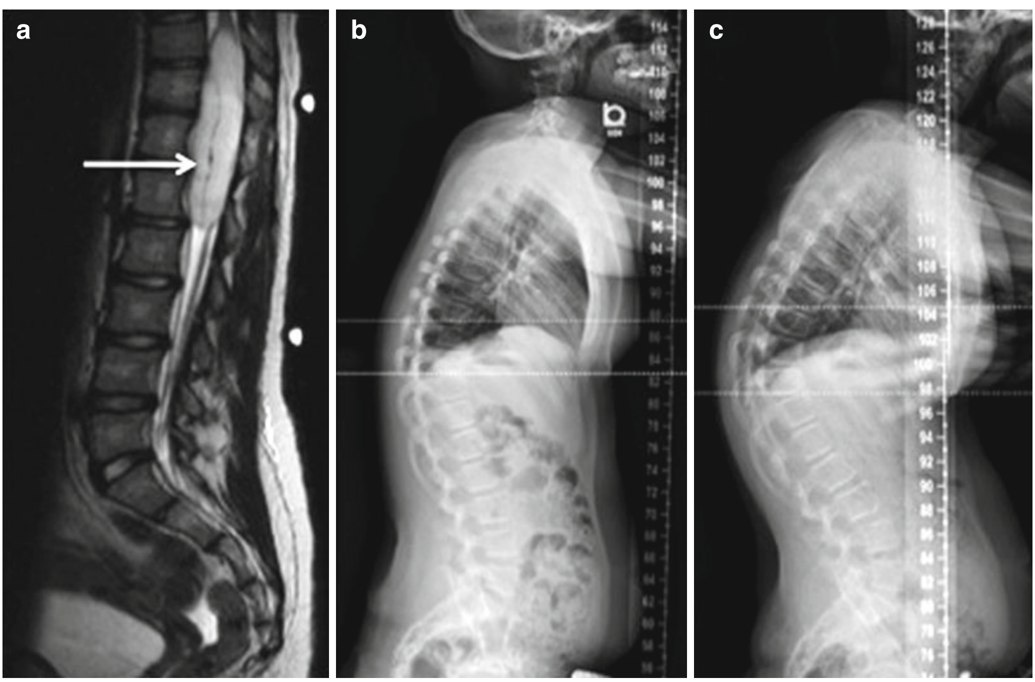


Fig. 22.5 (**a**) Preoperative sagittal MR image showing tumor (*arrow*). Postoperative lateral radiographs taken at (**b**) 2 weeks and (**c**) 6 months. Note the progressive iatrogenic kyphosis that has developed

kyphosis is even less common. Of 584 patients with a congenital spine abnormality, 112 had kyphoscoliosis and only 36 had pure kyphosis [20]. These anomalies can arise at any spine level but are most likely in the thoracolumbar junction between T10 and L1.

A type I anomaly (the most common) is caused by anterior failure of vertebral body formation. The subtypes are posterolateral quadrant vertebra, posterior hemivertebra, butterfly (sagittal cleft) vertebra, and anterior and anterolateral wedged vertebrae. A type II anomaly is derived from an anterior failure of vertebral body segmentation; its subtypes are distinguished by the presence of anterior or anterolateral unsegmented bars. A type III anomaly refers to mixed anomalies found in types I and II. A type IV anomaly is otherwise unclassifiable and typically has a larger curve than the other types. The most rapid curve progression is in type III (a mean of 5–8° per year, depending on patient age), followed by type I. Most neurologic deficits were found in patients with a sharp, angulated curve from a type I malformation [20].

Congenital kyphoscoliotic deformities can progress rapidly, especially during growth spurts,

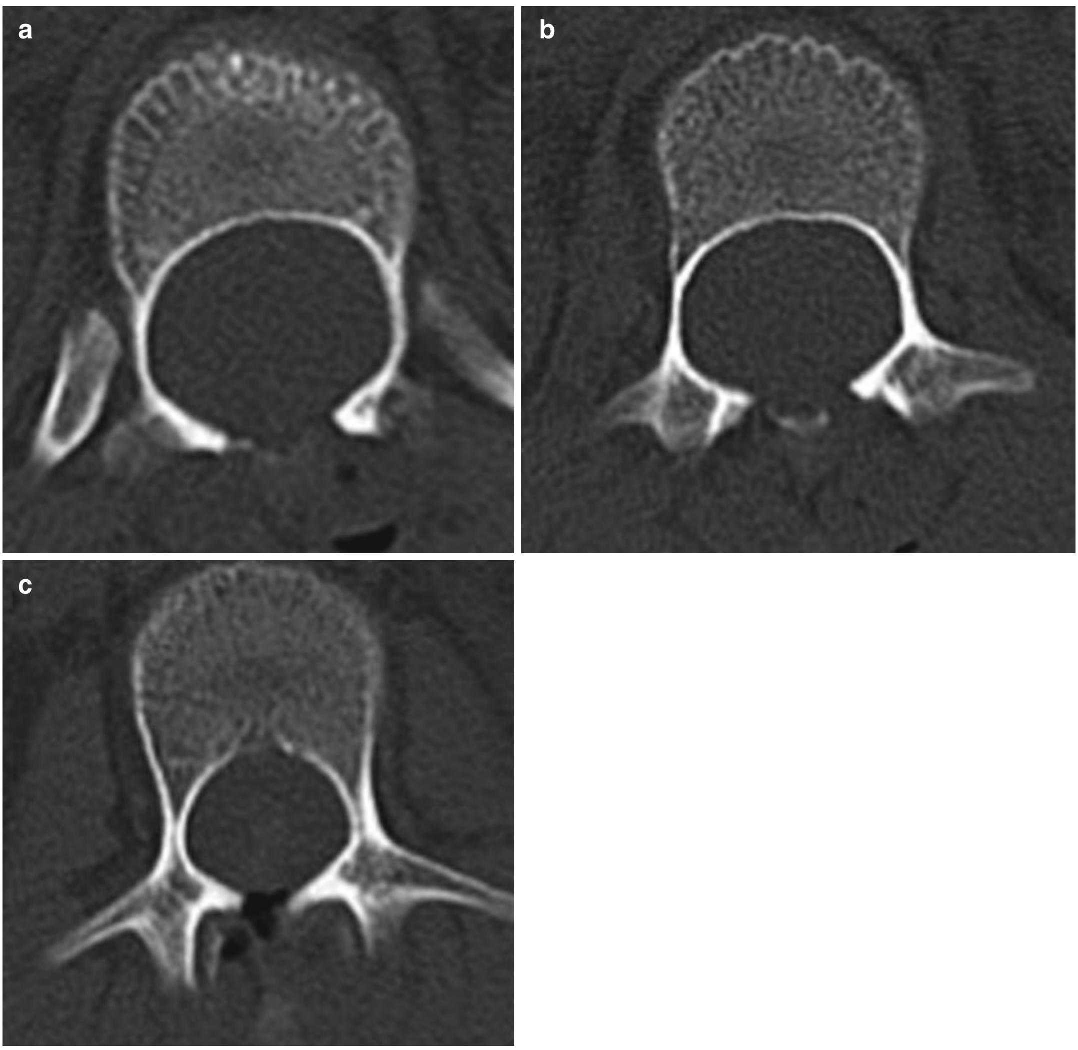


Fig. 22.6 This is the same patient as in Fig. 22.5. Note the very thin pedicles at (**a**) T12, (**b**) L1, and (**c**) L2 making pedicle screw placement very difficult. (**d**) Lateral radiograph of posterior instrumentation used to correct the iatrogenic kyphosis and stabilize the spine

Fig. 22.6 (continued)

and they should be monitored closely. Most of these deformities require surgical intervention; only 14 of 112 patients reached skeletal maturity without undergoing surgical intervention [20]. Surgical correction typically is recommended for a significantly progressive curve or after the development of a neurologic deficit, though a critical threshold for surgery has not yet been defined. Curve progression typically is defined as more than 10° of change, as radiographic landmarks are difficult to define in these patients. The surgical intervention typically entails short-segment instrumented fusion; a posterior asymmetric pedicle subtraction osteotomy or anteroposterior vertebral resection may be included. With a pedicle subtraction osteotomy, 10–24° of correction per level can be expected, depending on the level [21].

The preoperative planning should include MRI to detect any intraspinal abnormality that would require neurosurgical intervention before the deformity correction. CT and plain radiographs are used to visualize the complicated osseous anatomy. The general evaluation should include an echocardiogram and a renal ultrasonogram to detect any associated congenital abnormalities [6]. Although little has been published on the surgical risks in patients with congenital kyphosis, a congenital abnormality of the cervical spine probably entails the greatest risk because of the possibility of injuring the vertebral arteries. Surgery in the thoracic spine carries a risk of neurologic compromise whereas the lowest risk is associated with the lumbar spine.

The available surgical techniques for treating congenital kyphosis include pedicle subtraction osteotomies, vertebrectomies, costotransversectomies, anterior approaches, and combinations [13, 15, 25, 30]. A comparison of anteroposterior and posterolateral approaches for treating congenital scoliosis found a higher rate of minor complications in the patients treated through a posterolateral approach (40 % versus 8 %, $P=0.14$); however, the sample size was relatively small [13]. The clinical outcomes, quality of life, and radiographic measures of patients in the two groups were otherwise comparable [13]. The surgical priorities remain restoration of sagittal and coronal balance followed by correction of the curve magnitude. Some further correction can be expected in younger patients as a result of growth and surgical fusion.

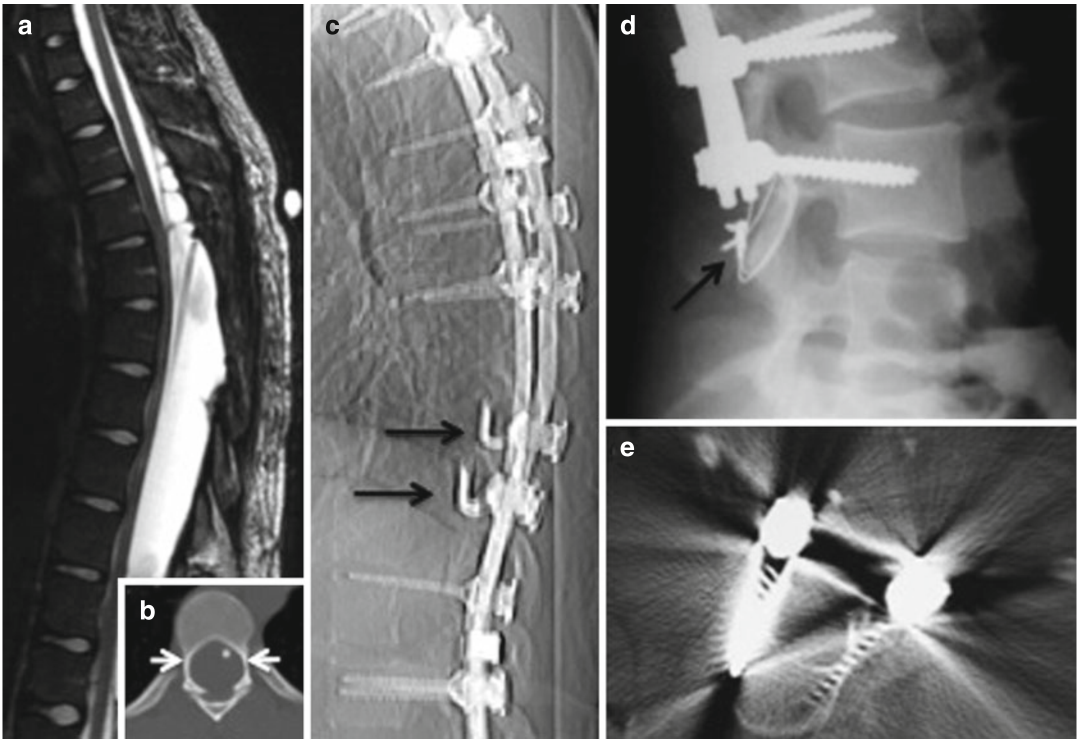


Fig. 22.7 Techniques for fixation in patients with thin pedicles. (**a**) Preoperative MR image showing large extradural thoracic CSF collection. (**b**) Postoperative axial CT image showing thinned out pedicles (*arrows*) and cystopleural shunt tubing (hyperdensity in the canal). (**c**) Postoperative lateral radiograph showing the use of pedicle hooks (*arrows*). (**d**) Postoperative lateral radiograph showing wiring to augment the pullout strength of the inferior pedicle screw in a T4–L3 fusion. (**e**) Axial CT illustrating "in-out-in" technique showing the screw entering at the lamina/transverse process junction, exiting bone lateral to the left pedicle, and reentering the vertebral body

22.6.4 Thoracolumbar Kyphosis from Hypoplasia

Thoracolumbar kyphosis secondary to lumbar hypoplasia is rare but should be differentiated from other etiologies. In seven normal, healthy patients, a mean kyphosis of 34.2° was normalized to −0.4° with no treatment. All patients had a wedge-shaped vertebra in the L1 or L2 region with an anterosuperior indentation (a beaked appearance) [5]. The diagnosis was based on radiographic appearance, lack of anomalies in the posterior elements, and improvement of the kyphosis with bipedal development. Such patients initially should be closely monitored to avoid an incorrect diagnosis, but they do not require bracing. Myelodysplasia, achondroplasia, endocrine abnormality, genetic metabolic disturbance, and other possible etiologies should be excluded from the differential diagnosis.

22.7 Investigations

Although some variation exists in the radiographic definition of Scheuermann kyphosis, commonly 5° of anterior wedging at each of three consecutive vertebrae, end plate irregularity, and Schmorl nodes are used as radiographic diagnostic criteria [31]. However, radiographic findings of irregular end plates, loss of disk height, anterior wedging of more than 10°, and overall hyperkyphosis also have been used as diagnostic criteria. Hyperextension radiographs with or without a bolster can be used to verify the rigidity of the curvature (Fig. 22.9). An MRI is a useful

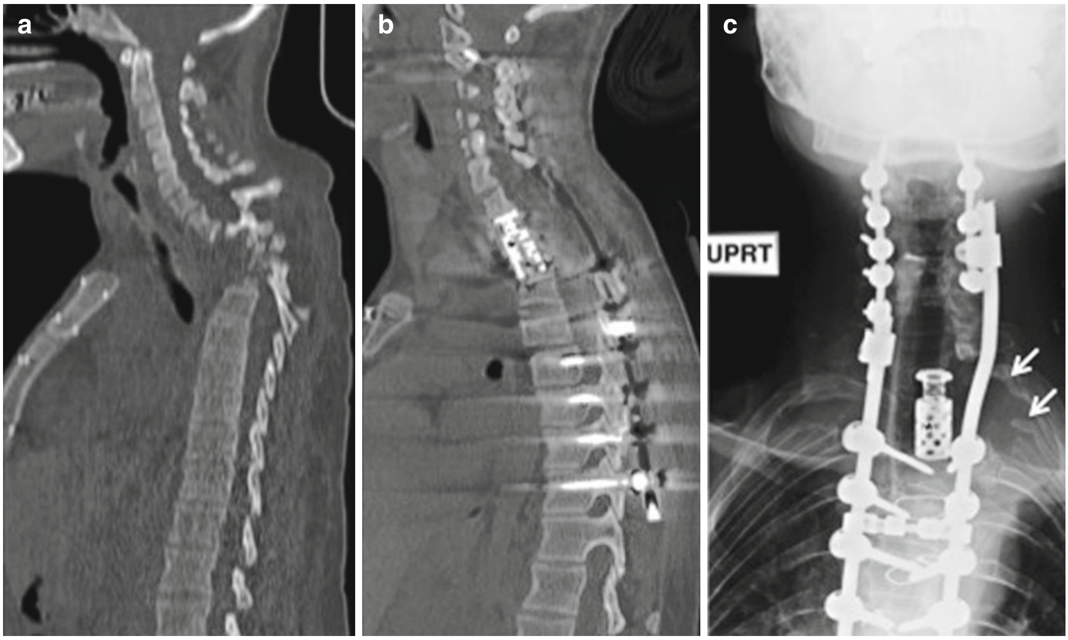


Fig. 22.8 (**a**) Preoperative midline sagittal CT of a 26-year-old female with neurofibromatosis type I showing a severe kyphotic deformity. (**b**) Postoperative midline sagittal CT after vertebral column resection and instrumentation with screws, rods, and an expandable cage (*asterisk*). (**c**) AP radiograph clearly illustrates the costotransversectomy (*arrows*) and the expandable cage (*asterisk*)

preoperative study to evaluate for disk herniations and early degenerative changes as well.

22.8 Nonsurgical Treatment

Bracing for Scheuermann kyphosis remains controversial. There is little evidence as to its effect on the natural history of kyphosis progression. Some studies have suggested that bracing leads to vertebral remodeling in immature patients [32, 33]. However, all available studies of bracing for kyphosis were small, retrospective, and limited to level IV evidence [17]. The criteria for bracing are a 50–75° curve and passive correction of 40 % or more. Patients with a curve greater than 75° may respond less favorably to bracing, and surgical intervention can be considered for these patients [24, 26]. With or without a bracing program, a rigorous schedule of exercise may be helpful, emphasizing thoracic extensor strengthening and endurance as well as hamstring stretching. Initial bracing achieves an almost 50 % reduction in the kyphosis in many patients, but some correction often is lost when the patient is weaned from brace wearing [26]. Bracing is routinely recommended for 16–23 h/day until apical wedging is corrected and then can be weaned gradually after skeletal maturity is attained.

22.9 Surgical Treatment

The current indications for surgical treatment are progressive kyphosis despite brace compliance, intractable pain, related neurologic deficit, or a persistent, significant deformity in a skeletally mature patient. The critical threshold of deformity for which surgery is recommended has not been defined. Current research recommendations on surgical indications and surgical approaches are limited to level IV evidence from retrospective cohort studies without control subjects [16, 17].

With earlier instrumentation constructs, surgical correction was followed over time by junctional kyphosis and loss of correction. These results led to interest in concomitant

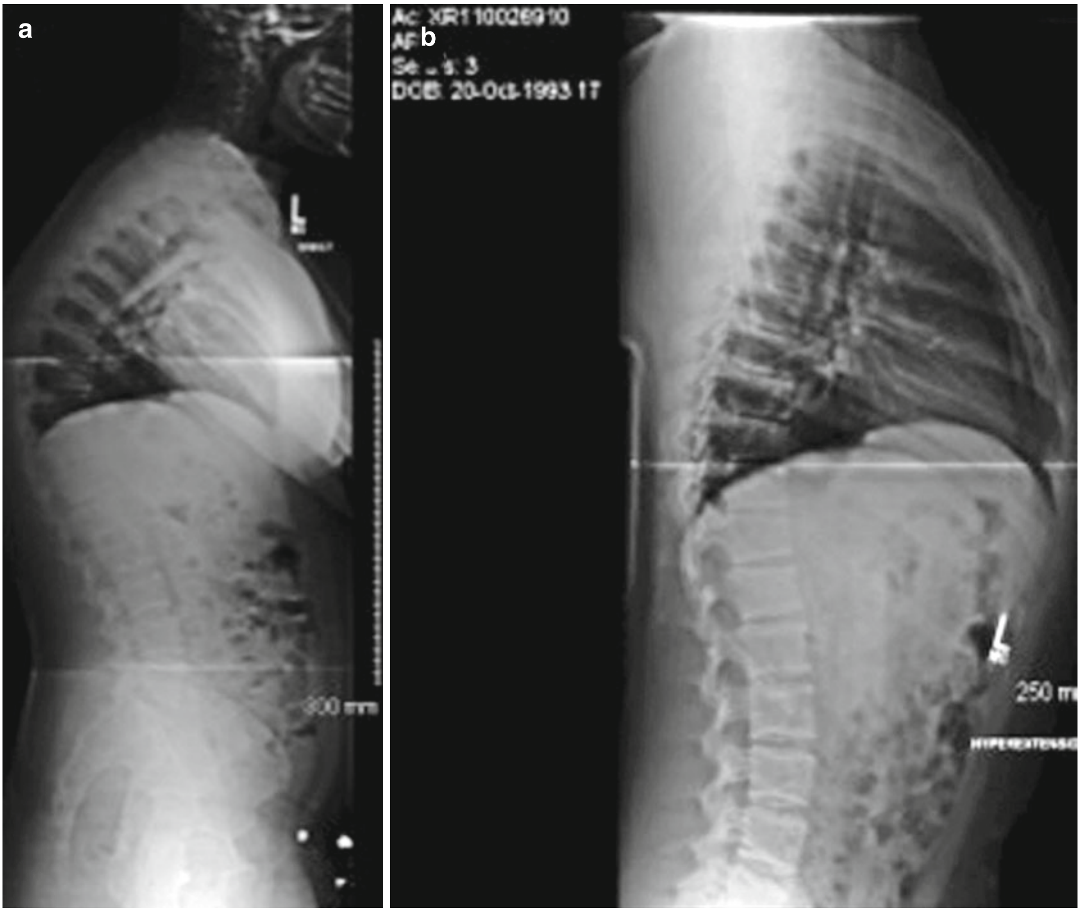


Fig. 22.9 (**a**) Lateral radiograph of a boy with Scheuermann kyphosis with a 75° kyphosis. (**b**) Lateral hyperextension radiograph over a bolster showing good correction of the kyphosis to 28°

anterior discectomies. However, combined anteroposterior and posterior-only constructs have had good radiographic results [16]. The anteroposterior approach led to slightly better maintenance of correction at follow-up, but the complication rate was higher than with the posterior-only approach. Proximal junctional kyphosis occurred in 32 % of patients and was related to arthrodesis short of the cephalad end vertebra, a higher magnitude of residual kyphosis, and a high pelvic incidence. Distal junctional kyphosis occurred in 5 % patients and always was associated with fusion selection rostral to the sagittal stable vertebra [16]. Posterior-only instrumentation and fusion have become popular, with complete facetectomies and partial excision of the inferior lamina (a so-called Ponte osteotomy). Anteroposterior and posterior-only procedures without extensive resection have not been compared in a research study. The selection of fusion levels is important and challenging, and overcorrection should be avoided (Fig. 22.10).

22.10 Postoperative Care

Given the magnitude of the surgery and potential blood loss, patients are monitored in the ICU overnight. Immediate postoperative care largely revolves around pain control and monitoring fluid shifts or blood loss. Patients undergo serial hematocrit checks and are transfused as needed. Patients also are placed on a patient-controlled anesthesia (PCA) and an aggressive bowel regiment. The following day patients are transferred

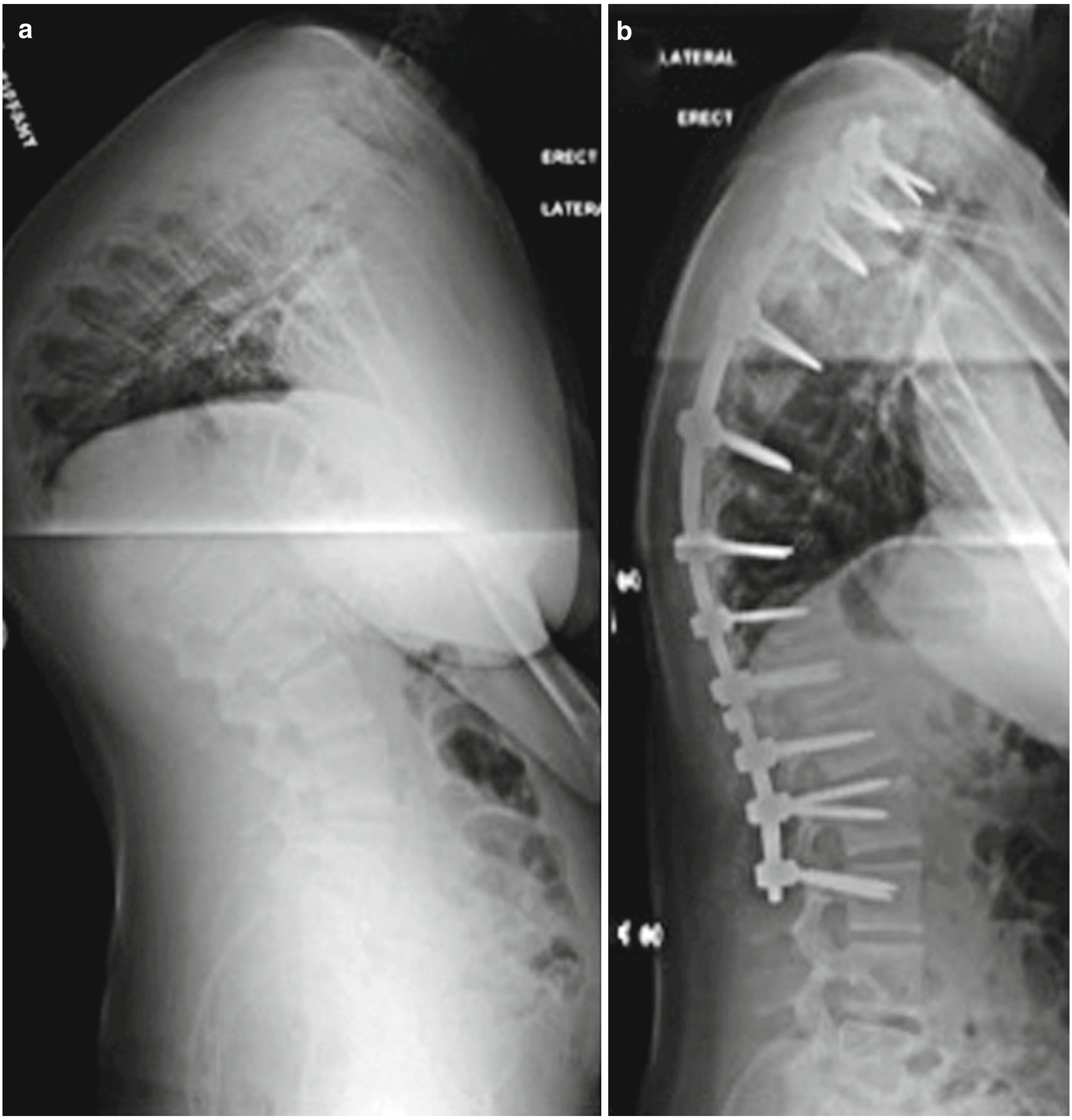


Fig. 22.10 (**a**) Lateral radiograph demonstrating severe kyphosis centered at T9. (**b**) Lateral postoperative radiograph illustrating the correction of previous kyphosis with a posterior spine fusion T2–L3

to the floor and ambulation is encouraged. Pediatric patients are at lower risk for developing deep venous thromboses, but early involvement of physiotherapy is important. Drains are typically removed around POD 3 when output has tapered significantly at which point antibiotics are discontinued. Patients are gradually transitioned from PCA to oral analgesics and muscle relaxants or ketorolac can be added for pain control. Patients can be discharged when they can ambulate, pain control is adequate, and they have had a regular bowel movement. Standing lateral and PA radiographs are typically obtained prior to discharge.

22.11 Complications

The Scoliosis Research Society Morbidity and Mortality Committee reported complications from 683 Scheuermann kyphosis surgeries [8].

Although they included adult and pediatric patients, 73 % of the cohort were less than 19 years of age. They noted a 14 % risk of developing complications in 683 procedures. The complication rate was only 12 % when limited to pediatric patients. Reported complications in the entire cohort included wound infections (3.8 %), unspecified (2.3 %), implant related (2.5 %), neurological compromise (1.9 %), pulmonary (1.3 %), hematologic (0.9 %), death (0.6 %), delayed neurological (0.3 %), durotomy (0.3 %), deep venous thrombosis (0.1 %), pulmonary embolus (0.1 %), sepsis (0.1 %), and cardiorespiratory (0.1 %). They noted a 14.8 % risk of complication from a posterior-only approach, 4.1 % from an anterior-only approach, and 16.9 % from a same-day anterior and posterior approach [8]. These findings are limited by the reporting nature of each institution and the vague categorization of each complication.

Lonner et al. compared outcomes from two groups of patients, those treated with an anterior-posterior approach and those treated posteriorly only. They noted a 23.8 % complication risk in those treated anteriorly alone as compared to 5.5 % in those treated with a posterior-only pedicle screw construct. Proximal and distal junctional kyphosis of greater than 10° occurred in 25 patients (32.1 %) and 4 patients (5.1 %), respectively [16].

Conclusion

Scheuermann kyphosis, postural kyphosis, iatrogenic kyphosis, and congenital kyphosis are the most common kyphotic deformities in children. However, many other pathologies can give rise to pediatric kyphoscoliosis. The possibility of a spinal cord injury, myelodysplasia, posttuberculous infection, achondroplasia, or inherited metabolic storage disease should be kept in mind during evaluation of a patient with a kyphotic deformity. The appropriate diagnosis is necessary for determining the natural history of the disease and deciding on the management options, based on the severity and progression of the deformity, its clinical presentation, the presence of a neurologic deficit, any cosmetic disfigurement, and the patient's skeletal maturity. The surgeon must carefully balance the expected course of the deformity against the risks and benefits of surgical intervention.

References

1. Ascani E, La Rosa G, Ascani C (2001) Scheuermann Kyphosis. In: The pediatric spine: principles and practice, 2nd edn. Lippincott Williams & Wilkins, Philadelphia
2. Bell DF, Walker JL, O'Connor G, Tibshirani R (1994) Spinal deformity after multiple-level cervical laminectomy in children. Spine (Phila Pa 1976) 19(4): 406–411
3. Bradford DS (1981) Vertebral osteochondrosis (Scheuermann's kyphosis). Clin Orthop Relat Res 158:83–90
4. Bradford DS, Moe JH, Montalvo FJ, Winter RB (1974) Scheuermann's kyphosis and roundback deformity. Results of Milwaukee brace treatment. J Bone Joint Surg Am 56(4):740–758
5. Campos MA, Fernandes P, Dolan LA, Weinstein SL (2008) Infantile thoracolumbar kyphosis secondary to lumbar hypoplasia. J Bone Joint Surg Am 90(8): 1726–1729
6. Chan G, Dormans JP (2009) Update on congenital spinal deformities: preoperative evaluation. Spine (Phila Pa 1976) 34(17):1766–1774
7. Cil A, Yazici M, Uzumcugil A, Kandemir U, Alanay A, Alanay Y, Acaroglu RE, Surat A (2005) The evolution of sagittal segmental alignment of the spine during childhood. Spine (Phila Pa 1976) 30(1): 93–100
8. Coe JD, Smith JS, Berven S, Arlet V, Donaldson W, Hanson D, Mudiyam R, Perra J, Owen J, Marks MC, Shaffrey CI (2010) Complications of spinal fusion for scheuermann kyphosis: a report of the scoliosis research society morbidity and mortality committee. Spine (Phila Pa 1976) 35(1):99–103
9. de Jonge T, Slullitel H, Dubousset J, Miladi L, Wicart P, Illes T (2005) Late-onset spinal deformities in children treated by laminectomy and radiation therapy for malignant tumours. Eur Spine J 14(8):765–771
10. Gangemi M, Renier D, Daussange J, Hirsch JF, Rigault P (1982) Children's cervical spine instability after posterior fossa surgery. Acta Neurol (Napoli) 4(1):39–43
11. Glassman SD, Bridwell K, Dimar JR, Horton W, Berven S, Schwab F (2005) The impact of positive sagittal balance in adult spinal deformity. Spine (Phila Pa 1976) 30(18):2024–2029
12. Jackson RP, McManus AC (1994) Radiographic analysis of sagittal plane alignment and balance in standing volunteers and patients with low back pain

matched for age, sex, and size. A prospective controlled clinical study. Spine (Phila Pa 1976) 19(14): 1611–1618
13. Jalanko T, Rintala R, Puisto V, Helenius I (2011) 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) 36(1):41–49
14. Jandali S, Diluna ML, Storm PB, Low DW (2011) Use of the vascularized free fibula graft with an arteriovenous loop for fusion of cervical and thoracic spinal defects in previously irradiated pediatric patients. Plast Reconstr Surg 127(5):1932–1938. doi:10.1097/PRS.0b013e31820cf4a6
15. Lenke LG, O'Leary PT, Bridwell KH, Sides BA, Koester LA, Blanke KM (2009) Posterior vertebral column resection for severe pediatric deformity: minimum two-year follow-up of thirty-five consecutive patients. Spine (Phila Pa 1976) 34(20):2213–2221
16. Lonner BS, Newton P, Betz R, Scharf C, O'Brien M, Sponseller P, Lenke L, Crawford A, Lowe T, Letko L, Harms J, Shufflebarger H (2007) Operative management of Scheuermann's kyphosis in 78 patients: radiographic outcomes, complications, and technique. Spine (Phila Pa 1976) 32(24):2644–2652
17. Lowe TG, Line BG (2007) Evidence based medicine: analysis of Scheuermann kyphosis. Spine (Phila Pa 1976) 32(19 Suppl):S115–S119
18. McGirt MJ, Chaichana KL, Atiba A, Bydon A, Witham TF, Yao KC, Jallo GI (2008) Incidence of spinal deformity after resection of intramedullary spinal cord tumors in children who underwent laminectomy compared with laminoplasty. J Neurosurg Pediatr 1(1):57–62
19. McLaughlin EJ, Heuer GG, Whitmore RG, Birknes JK, Belasco J, Sterman D, Low DW, Storm PB (2011) Treatment of a malignant peripheral nerve sheath tumor and its complications through a multidisciplinary approach. J Neurosurg Pediatr 7(5):543–548. doi:10.3171/2011.2.PEDS1175
20. McMaster MJ, Singh H (1999) Natural history of congenital kyphosis and kyphoscoliosis. A study of one hundred and twelve patients. J Bone Joint Surg Am 81(10):1367–1383
21. O'Shaughnessy BA, Kuklo TR, Hsieh PC, Yang BP, Koski TR, Ondra SL (2009) Thoracic pedicle subtraction osteotomy for fixed sagittal spinal deformity. Spine (Phila Pa 1976) 34(26):2893–2899
22. Papagelopoulos PJ, Peterson HA, Ebersold MJ, Emmanuel PR, Choudhury SN, Quast LM (1997) Spinal column deformity and instability after lumbar or thoracolumbar laminectomy for intraspinal tumors in children and young adults. Spine (Phila Pa 1976) 22(4):442–451
23. Ratliff JK, Cooper PR (2003) Cervical laminoplasty: a critical review. J Neurosurg 98(3 Suppl):230–238
24. Riddle EC, Bowen JR, Shah SA, Moran EF, Lawall H Jr (2003) The duPont kyphosis brace for the treatment of adolescent Scheuermann kyphosis. J South Orthop Assoc 12(3):135–140
25. Ruf M, Jensen R, Letko L, Harms J (2009) Hemivertebra resection and osteotomies in congenital spine deformity. Spine (Phila Pa 1976) 34(17): 1791–1799
26. Sachs B, Bradford D, Winter R, Lonstein J, Moe J, Willson S (1987) Scheuermann kyphosis. Follow-up of Milwaukee-brace treatment. J Bone Joint Surg Am 69(1):50–57
27. Sciubba DM, Gallia GL, McGirt MJ, Woodworth GF, Garonzik IM, Witham T, Gokaslan ZL, Wolinsky J-P (2007) Thoracic kyphotic deformity reduction with a distractible titanium cage via an entirely posterior approach. Neurosurgery 60(4 Suppl 2):223–230; discussion 230–231
28. Sim FH, Svien HJ, Bickel WH, Janes JM (1974) Swan-neck deformity following extensive cervical laminectomy. A review of twenty-one cases. J Bone Joint Surg Am 56(3):564–580
29. Simon SL, Auerbach JD, Garg S, Sutton LN, Telfeian AE, Dormans JP (2008) Efficacy of spinal instrumentation and fusion in the prevention of postlaminectomy spinal deformity in children with intramedullary spinal cord tumors. J Pediatr Orthop 28(2): 244–249
30. Smith JT, Gollogly S, Dunn HK (2005) Simultaneous anterior-posterior approach through a costotransversectomy for the treatment of congenital kyphosis and acquired kyphoscoliotic deformities. J Bone Joint Surg Am 87(10):2281–2289
31. Sorenson K (1964) Juvenile kyphosis: clinical appearances, radiography, aetiology and prognosis. Munksgaard, Copenhagen
32. Tribus CB (1998) Scheuermann's kyphosis in adolescents and adults: diagnosis and management. J Am Acad Orthop Surg 6(1):36–43
33. Wenger DR, Frick SL (1999) Scheuermann kyphosis. Spine (Phila Pa 1976) 24(24):2630–2639
34. Winter R (1991) The treatment of spinal kyphosis. Int Orthop 15(3):265–271
35. Yasuoka S, Peterson HA, Laws ER Jr, MacCarty CS (1981) Pathogenesis and prophylaxis of postlaminectomy deformity of the spine after multiple level laminectomy: difference between children and adults. Neurosurgery 9(2):145–152
36. Yeh JS, Sgouros S, Walsh AR, Hockley AD (2001) Spinal sagittal malalignment following surgery for primary intramedullary tumours in children. Pediatr Neurosurg 35(6):318–324

23 Congenital

William D. Long III and Jonathan N. Grauer

23.1 Overview

Spinal abnormalities encountered at birth encompass a large spectrum of conditions that may lead to a variety of spinal deformities. Environmental factors, genetics, vitamin deficiency, and drugs have been found to be associated in the development of vertebral anomalies [1]. Physiologic insult frequently occurs early during the embryologic period, and the resultant disruption in somitogenesis causes alterations that result in the failure of formation, failure of segmentation, or a combination of the two [2–5]. Mutations in the development of somites can produce any number of anomalies including hemivertebra, unilateral and segmental bars, vertebral agenesis, and congenital dislocated spine. These structural alterations can produce an imbalance in spinal growth, potentially leading to short stature, scoliosis, kyphosis, and kyphoscoliosis [2].

The approach to treating the patient with congenital spine deformity requires a multidisciplinary effort, as many children are syndromic with abnormalities in other organ systems [6]. Treatment for spinal congenital deformity seeks to optimize the child's overall function and growth potential. Surgical interventions have been developed to treat the subsequent curvature, as attempts to manage early-onset spinal deformities with external force through bracing or casting have rarely been effective [7]. Due to the complexity of anatomy in these patients and frequent lack of anchor points for implants, precise dissection and a thorough understanding of the deformity are necessary for successful outcomes.

The estimated prevalence of congenital spinal malformation ranges from 0.5 to 1 in 1,000 births [8]. The male to female ratio has been approximated at 1:1.4 [9]. Beals and colleagues [6] summarized three early seminal papers [4, 10, 11] that examined congenital scoliosis, noting that between 30 and 60 % of patients with vertebral deformities had other associated developmental anomalies. Cardiac, renal, and neurologic defects are commonly encountered in this population, as these systems develop around the same time during gestation (Table 23.1) [12]. As such, a multitude of syndromes have been identified that are associated with congenital spinal deformity (Table 23.2) [2]. Interestingly, however, a family history of congenital anomaly is rare [13]. The complex nature of this patient population warrants caution and diligence in order to ensure no critical finding is overlooked.

Spinal dysraphism describes a heterogeneous group of abnormalities resulting from defective closure of the neural tube, with subsequent anomalous development of the caudal cell mass [14]. This can result in open or closed neural tube defects. Spina bifida describes a group of disorders associated with the failure of the vertebral

W.D. Long III, M.D. • J.N. Grauer, M.D. (✉)
Department of Orthopaedics and Rehabilitation, Yale University School of Medicine, 800 Howard Ave, New Haven, CT 06511, USA
e-mail: jonathan.grauer@yale.edu

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_23, © Springer-Verlag Berlin Heidelberg 2014

Table 23.1 Associated anomalies with congenital spine deformity

Cardiac	Renal	Neurologic
Ventricular septal defects	Renal hypoplasia	Tethered cord
Atrial septal defects	Horseshoe kidney	Syrinx
Patent ductus arteriosus	Ectopic kidney	Low conus
Tetralogy of Fallot	Cloacal anomaly	Extra- or intradural mass
Transposition of great vessels	Exstrophy of the bladder	Chiari malformation
Sick sinus syndrome	Hydronephrosis	Dandy-Walker malformation
Pulmonary stenosis	Undescended testis	Diastematomyelia

Adapted with permission [2]

Table 23.2 Associated syndromes with congenital vertebral malformation

Aarskog syndrome (hypertelorism, brachydactyly, shawl scrotum)
Aase syndrome (triphalangeal thumb, congenital anemia)
Alagille syndrome (cholestasis, peripheral pulmonic stenosis, peculiar facies)
Albright hereditary osteodystrophy (short metacarpals, rounded facies, vicarious mineralization)
Apert syndrome (craniosynostosis, midfacial hypoplasia, syndactyly, broad distal phalanges)
Atelosteogenesis type I (giant cell chondrodysplasia)
Catel-Manzke syndrome (micrognathias, cleft palate, index finger hypertrophy)
Cervico-oculo-acoustic syndrome (Wildervanck and Klippel-Feil anomalies, abducens paralysis, sensorineural deafness)
CHARGE syndrome (coloboma, heart disease, atresia of the choanae, retarded growth, genital deformity, ear anomalies)
Deletion of 5p syndrome (cri du chat, microcephaly, downward palpebral fissures)
Distichiasis lymphedema syndrome (double row eyelashes, lymphedema)
Escobar syndrome (multiple pterygium syndrome)
Femoral hypoplasia-unusual facies syndrome (femoral hypoplasia, short nose, cleft palate)
Fetal alcohol syndrome (premature growth deficiency, microcephaly, short palpebral fissures)
Frontometaphyseal dysplasia (prominent supraorbital ridges, joint limitations, splayed metaphysic)
Gorlin syndrome (basal cell carcinoma, broad facies, rib anomalies)
Incontinentia pigmenti syndrome, Bloch-Sulzberger syndrome (patch alopecia, pigmented skin lesions)
Jarcho-Levin syndrome
Kabuki syndrome
Klippel-Feil syndrome (short neck, low hairline, early development of cervical vertebrae)
Larsen syndrome (multiple joint dislocations, flat facies, short fingernails)
Morquio syndrome (pectus carinatum, odontoid hypoplasia, genus valgus, corneal clouding)
Multiple synostosis syndrome (symphalangism syndrome)
MURCS (Mullerian duct anomaly, renal abnormalities, cervical vertebra defects)
Noonan syndrome (webbed neck, pectus excavatum, cryptorchidism, pulmonic stenosis)
Oculoauriculovertebral spectrum, Goldenhar syndrome (branchial arch defects, hemifacial macrosomia)
Pallister-Hall syndrome (hypothalamic, hypopituitarism, hamartoblastoma, imperforate anus, polydactyly)
Ritscher-Schinzel syndrome, 3C syndrome
Robinow syndrome (fetal face syndrome, flat facial profile, short forearms, hypoplastic genitalia)
Saethre-Chotzen syndrome (brachydactyly, maxillary hypoplasia, syndactyly, prominent ear crus)
Simpson-Golabi-Behmel syndrome
Spondylocarpotarsal synostosis syndrome (short stature, block vertebrae, carpal synostosis)
Smith-Magenis syndrome (broad midface, brachycephaly, brachydactyly, speech delay)
Trisomy 18 syndrome (clenched hand, short sternum, low-arch dermal ridge)
Turner syndrome, 45X syndrome, XO syndrome (broad chest, short female, lymphedema or its residua)
VATER association (vertebral defects, anal atresia, tracheoesophageal fistula, renal dysplasia)

Adapted with permission [2]

arch to close during development. This can lead to the presence of neural elements found outside of the vertebral canal. Spina bifida occulta is the condition where the posterior spinal elements fail to fuse, leaving the spinal canal covered only by skin and soft tissue, frequently identified by a hairy patch or dimple. Spina bifida cystica is a more severe manifestation of the disease, in which the meninges and nerves may be extruded.

Identification of any such defects as a meningocele, myelomeningoceles, lipomyelomeningocele, myeloceles, split cord malformations, or myelocystoceles merits neurosurgical evaluation prior to intervention [15].

Other congenital spine anomalies seen in consultation by spine surgeons include Chiari malformations, sacral agenesis, spondyloepiphyseal dysplasia, and congenital dislocated spine. Chiari malformations are deformities of the brain stem characterized by displacement of the cerebral tonsils through the foramen magnum, thereby blocking normal cerebrospinal flow [16]. They are commonly associated with congenital scoliosis and syrinx. Sacral agenesis, also known as caudal regression syndrome, occurs early in gestation, typically before the seventh week. It is frequently accompanied by other vertebral defects and lower extremity abnormalities, with the classic postnatal “sitting Buddha” posture. Neonates demonstrated a flexion-abduction contractures of hips with marked knee flexion and popliteal webbing and feet tucked in equinovarus [1]. It has been associated with maternal diabetes and the pathology thought to be related to insulin, although the exact mechanism has yet to be elucidated [17]. Spondyloepiphyseal dysplasia encompasses a rare group of disorders characterized by flattened vertebral bodies and abnormal epiphyses, with the congenital variant the most severe form of the disease [15, 18]. Congenital dislocated spine is a rare anomaly resulting from the developmental failure of the spine at a single level. A sudden sagittal translation leaves the alignment of the spine grossly displaced, frequently resulting in neurologic sequelae [19].

Dysraphism demonstrates a distinct morphology from closed vertebral centrum defects, likely due to the differences in the developmental influences during the formation of the bony anatomy versus the spinal cord and meninges. This independence confers a deviation in the initial development that displays unique patterning at birth. Closed anterior spine malformations demonstrate greater variation in the number of vertebrae involved and the overall pattern and resultant bony alignment [20]. These frequently result in coronal and sagittal plane deformities that benefit from operative stabilization. Congenital scoliosis and kyphosis are the two main deformities that fall under the purview of the orthopedic spine surgeon.

Early attempts to classify congenital spine malformations were based on the concept of embryologic maldevelopment. In 1968, Winter and colleagues developed a scheme to classify coronal congenital spine malformations based on an observation of 234 patients [4]. This classification system was based on posteroanterior and lateral radiographs, and it divided spinal pathology into abnormalities of formation, abnormalities of segmentation, or a mixed type that possessed features of both. It has since been used widely and adapted because of its utility in predicting progression of deformity. In 1982, McMaster and Ohtsuka added their experience with congenital scoliosis, demonstrating a predilection for certain malformations to progress over time [5]. Both Winter and McMaster applied a similar scheme for deformities in the sagittal plane resulting in kyphosis [21, 22]. Noting that this classification was based on x-rays and many times failed to account for the three-dimensional deformities encountered in this challenging population, the Spinal Deformity Study Group recently published a new classification based on three-dimensional computed tomography [20]. This scheme delineates four types of congenital anomalies: type I, solitary simple; type 2, multiple simple; type 3, complex; and type 4, segmentation failure. This system is based in an algorithm that seeks to provide an appropriate surgical treatment based on the deformity.

Failure of formation defects typically occur in the lateral or anterior spine during development, producing the classic scoliotic and kyphotic curves, respectively. This group encompasses such defects as hemivertebra, wedge vertebra, and butterfly vertebra. Hemivertebra can be further classified as segmented or free if a normal disc is present above and below the affected level or unsegmented if the vertebra lacks adjoining discs. Hemivertebra is a complete failure of formation, with only one pedicle, whereas wedge vertebrae are a partial failure of formation, possessing two pedicles with asymmetric laterality. Butterfly vertebrae are partial or complete failures of formation of the anterior and central

Table 23.3 Natural history of particular congenital spine deformities

Spinal deformity	Curve progression
Unilateral unsegmented bar with contralateral hemivertebrae	Rapid and relentless
Unilateral unsegmented bar	Rapid
Fully segmented hemivertebra	Steady
Partially segmented hemivertebra	Less rapid
Incarcerated hemivertebra	May slowly progress
Unsegmented hemivertebra	Little progression

portions of the body, resulting in two posterolateral fragments of bone attached to the neural arch, separated by a sagittal cleft. Failure of formation in the sagittal plane results in centrum hypoplasia of the anterior elements, producing a kyphotic curve. Centrum aplasia is the most severe form, in which the pedicle roots persist at the base but are not attached to the body, resulting in a significant angulation. *Failure of segmentation* results in block vertebra or segmental bar formation. Anterior segmentation failure, stemming from annulus fibrosus osseous metaplasia, leads to progressive kyphosis owing to the absence of anterior vertebral growth. Unilateral segmentation failure, characterized by fused lateral vertebral bodies, produces scoliotic curves as portions of the unsegmented spine are tethered during normal growth. Total segmentation failure leads to the formation of block vertebrae, which can result in significant shortening of the spine if many levels are involved. *Mixed-type defects*, such as a unilateral unsegmented bar with contralateral hemivertebra, are notorious for producing kyphoscoliotic deformities that progress rapidly without intervention. The utility of this classification based on the embryologic disruption in development is its association to predict which of these deformities and curves is likely to progress, thereby requiring surgical intervention (Table 23.3). Unsegmented or incarcerated hemivertebra demonstrates little progression, while block vertebra sometimes demonstrates no significant curvature at all. Fully segmented hemivertebra progresses steadily, while unilateral segmental bars curve rapidly. The unilateral unsegmented bar with contralateral hemivertebrae is the worst offender, characterized by rapid and unrelenting curvature in multiple planes. The natural history of congenital kyphotic deformities is associated with steady progression, with potential for neurologic compression. A thorough understanding of the anatomy, natural history, and risk of further deformity is necessary for the optimal care of the patient with congenital spine malformation [23].

23.2 Case Example

A 4-year-old boy is referred for evaluation of scoliosis. His gestational history is notable for a positive quadruple screen, and karyotyping revealed a Klinefelter variant with chromosomes 49-XXXXY. Intrauterine ultrasound was performed during the pregnancy, noting a right clubbed foot and enlarged cerebral ventricles. He was born at term with a weight of 5 lb and length of 19 in. During the newborn period, he failed his hearing screen although currently exhibits normal audiologic function. He is developmentally delayed, not crawling until the age of one, walking independently by 30 months, and has a vocabulary that is limited to approximately seven words. He underwent surgical intervention for his club foot at 1.5 years of age and was noted on recent ultrasound to have one kidney larger than the other. Physical examination reveals left thoracic prominence on forward bend test and an elevated left hemi-pelvis. Radiographs of the spine are shown in Fig. 23.1.

23.3 Pathology/Pathophysiology

A rudimentary understanding of vertebral development provides insight into understanding the pathogenesis of congenital spine malformation. The embryologic precursor of the vertebral body is termed the somite. Disruptions during somitogenesis, which begins to occur between 20 and 30 days of gestation, can affect the subsequent development of the spine [24]. Somite segmentation occurs at 6–8 weeks, prior to the

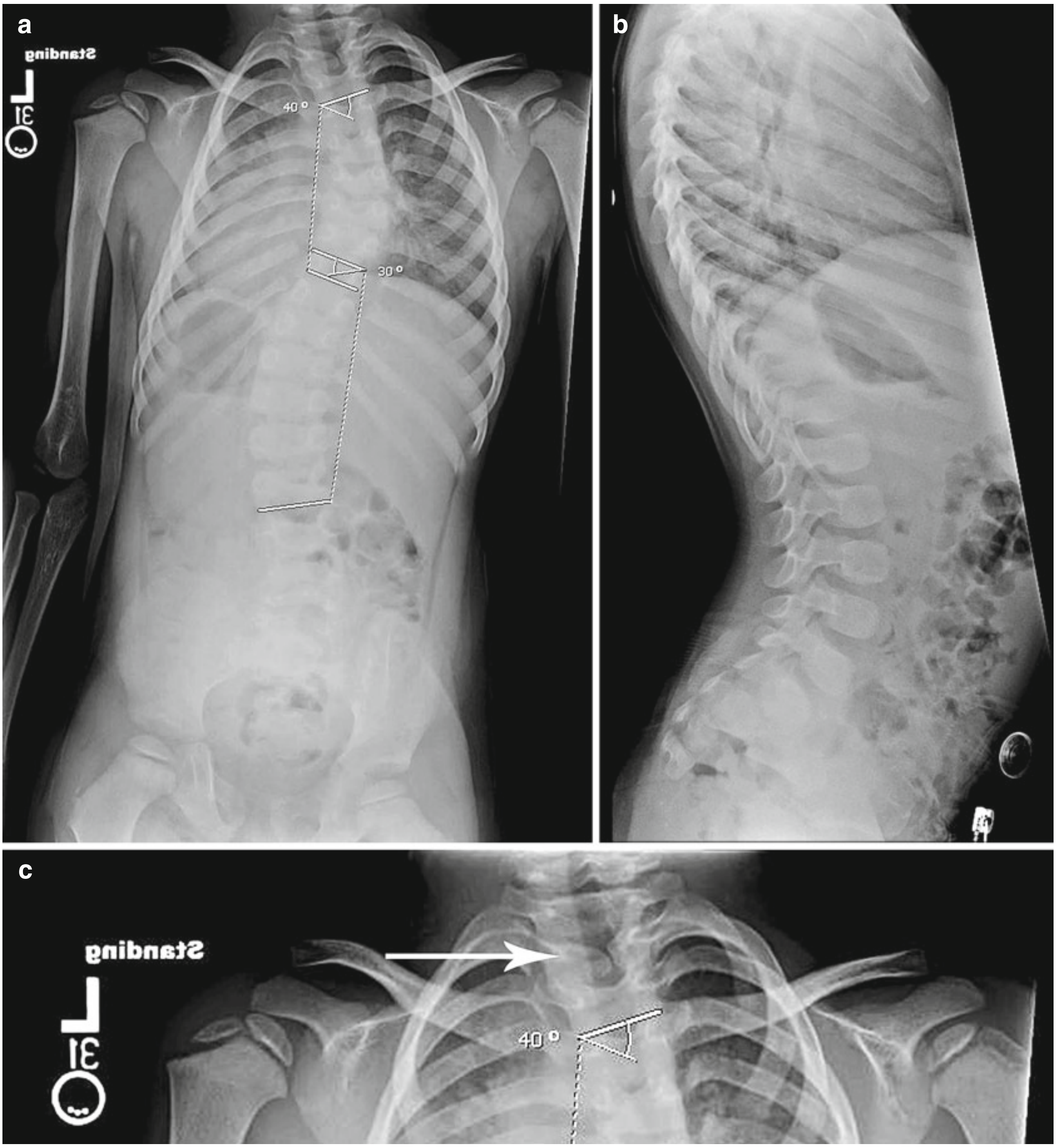


Fig. 23.1 X-rays of the case example. (**a**) Posteroanterior and (**b**) lateral radiographs showing the thoracic and thoracolumbar scoliotic curves with thoracic cage asymmetry. (**c**) A close-up of the cervicothoracic junction demonstrating a butterfly vertebra of T2

chondrification and ossification of the axial skeleton. Disruptions that affect the delivery of genetic instructions during these critical periods of formation and segmentation can result in the malformations detailed above [25]. The earliest stage at which detection of a vertebral anomaly can occur is in week 4–5, during the formation of the vertebral mesenchymal anlage [1]. This development is intimately related to the intersegmental arterial supply to the immature vertebral body, as cells closer to the artery develop more rapidly [26]. Disruptions in the formation of the cartilaginous vertebral body anlage occur early in development, prior to week 16 of gestation [3]. After birth, progressive curvatures that result from congenital malformation develop due to the asymmetric growth of the spine during infancy, childhood, and adolescence. Increased growth along the convexity of a curvature with a tethered or limited concave aspect produces the most progressive spinal deformities.

23.4 History and Physical Examination

The presentation of patients with congenital spinal deformity is variable, ranging from severely syndromic patients to those with a static asymptomatic spinal deformity. Many patients are referred for findings observed on routine screening during gestation and infancy, such as triple screen, chest x-ray, and prenatal ultrasound. Others present for evaluation after MRI for other conditions reveals an anomaly within the spine. Astute physical examination by the pediatrician may demonstrate hairy patches, sacral dimpling, cavus foot deformities, or hyperpigmentation, prompting spinal surgeon evaluation. There are numerous considerations in evaluating an infant or child with congenital spine malformation, and initial inspection should focus on age at presentation, the nature of the deformity, the presence of associated medical conditions, and any other associated congenital anomalies [2].

A thorough and careful review of the patient's gestational, family, and medical history is essential. Physical examination should include height and weight; evaluation of the spinal balance in the coronal and sagittal planes; evaluation of the orientation of the shoulder, head tilt, pelvic tilt, and leg lengths; and inspection of the skin to check for dimples, cysts, lesions, hyperpigmentation, or hair tufts. A complete neurologic examination is critical, although the absence of neurologic findings (motor or sensory deficits, bladder symptoms, asymmetric reflexes) does not preclude the presence of occult intraspinal anomaly [2].

Since pulmonary involvement secondary to chest wall deformity is commonly seen in patients with severe forms of congenital kyphoscoliosis, attention to the patient's respirations and thoracic cage anatomy is needed. Campbell et al. noted thoracic cage deformity and the development of thoracic insufficiency syndrome in children with congenital scoliosis and kyphosis [27]. They recommend the thumb excursion test to assess for asymmetric respiration and the use of accessory musculature for breathing [27]. Pulmonary function tests should be considered for any child undergoing surgery.

23.5 Diagnostic Imaging

An accurate and comprehensive radiologic evaluation is critical for patients suspected to have congenital spinal malformations. Prenatal imaging studies are most useful for identifying spinal dysraphism [14, 28]. Gestational ultrasound and fetal MRI have been useful for identification of intraspinal anomalies and extruded neural elements. Three-dimensional CT scans are being utilized in the third trimester to assess for skeletal dysplasia in high-risk patients [29, 30]. In the postnatal period, the initial modality of choice is x-ray. Standard upright posteroanterior (PA) and lateral views of the entire spine demonstrate the characteristics of spinal curvature under gravity, showing any signs of compensation and the sacro-pelvic relationship. Cobb angle measurements allow quantification of the degree of curvature [31]. Bending films (side bending, bolster bending, traction, push prone) are commonly obtained to document flexibility, although their utility in determining correction remains unstudied in this population.

Children with cervical spine involvement should have a cervical x-ray series to preclude atlantoaxial instability, with an atlantodens interval of less than 4 mm in children less than 8 years of age [32]. Three-dimensional CT scans are useful to observe the anterior and posterior spinal components, the relationships between adjacent vertebra, and the overall spinal morphology [20]. CT scan demonstrates finer bony detail compared to x-ray and can be useful to document thoracic cage and pulmonary volumes. MRI is the diagnostic technique of choice in this population for identifying intraspinal anomalies and should be performed in all children undergoing surgery

[33]. Dynamic MRI allows for the assessment of chest wall motion and respiration, while MR angiography may be useful for identifying vascular anomalies and the position of the vertebral artery prior to surgery [2].

23.6 Treatment

The primary goal of treatment of congenital vertebral malformation is to prevent the development of a severe deformity while optimizing the child's overall function and growth potential. Early diagnosis, anticipation, and prevention of deterioration are critical to achieve ideal results, as correction of a severely deformed spine is much more difficult than prevention. A thorough understanding of the type of malformation, knowledge of the natural history of the progression, frequent follow-up (4–6 months intervals), and understanding when to initiate surgical intervention are essential in providing optimal care.

Observation plays a role in patients with deformities in which the natural history is not known or in conditions that are known to progress slowly such as hemivertebra or block vertebra. Two periods of rapid growth merit closer follow-up, those being during the first 4 years of life and during the adolescent growth spurt. There is no role for observation in deformities that are known to uniformly progress, such as an unsegmented bar with contralateral hemivertebra or congenital kyphosis.

Nonoperative therapies that have been effective in idiopathic forms of scoliosis and kyphosis such as bracing have been ineffective in congenital spine anomalies [34]. Congenital scoliosis curves tend to be rigid as the defect is primarily in the bony anatomy, whereas idiopathic forms with long sweeping curves from soft tissue derangements are flexible and amenable to orthosis treatment. Alternative forms of therapy such as exercises, manipulation, and electrical stimulation have completely failed in the treatment of congenital spine deformity.

Surgical stabilization of the spine is considered the most effective treatment for severe or progressive congenital deformity. No single operative procedure can be applied to the numerous forms of deformity seen in congenital spine malformation. A thorough understanding of patient anatomy is required for hardware planning and postoperative care. A number of surgical principles are constant for patients undergoing surgical intervention of congenital spine deformity [33]. Neurologic monitoring is mandatory for anyone undergoing spinal surgery for congenital malformation, as these patients are at increased risk to develop postoperative paraplegia after instrumentation and fusion [35]. The anesthesia team should know how to perform a Stagnara wake-up test in case neuromonitoring shows a change in spinal function or becomes unreliable [36]. A number of spinal implants have been effective in patients of diminutive size, with 3.2-mm-diameter rod systems available in the surgeon's armamentarium; having two different systems available provides the surgeon with additional options in decision making intraoperatively [33]. The use of titanium implants is beneficial, as many patients with congenital spine deformity exhibit some other system anomaly, allowing postoperative MRI to be more distinct and useful for subsequent follow-up.

The type of surgery selected to correct congenital spine deformity depends on the age of the patient, the site and type of vertebral anomaly, and the size of the curvature. There are five main procedures for the surgical treatment of congenital spine deformity:

- Convex growth arrest (anterior and posterior hemiepiphysiodesis)
- Fusion with or without instrumentation, posterior and combined anterior/posterior approaches
- Hemivertebra excision
- Vertebral column resection
- Fusionless instrumentation systems

Convex growth arrest was first described by MacLennan [37]. It was designed to eliminate further convex growth while permitting concave growth to occur and lessen the curvature. This intervention is most applicable for young patients with short limited curves. The surgery is classically performed in two stages, with a preliminary anterior approach on the convexity to eliminate the anterior growth plates at the site of the anomaly and a subsequent procedure through a distinct, posterior exposure of the convexity at the site of the hemivertebra in order to perform a posterior convex fusion [38]. Postoperatively, the child is immobilized in a body cast and kept nonambulatory for 3–4 months. After the cast is removed, the child is placed in a spinal orthosis that is worn full time for an additional 12 months, allowing adequate time for fusion. This is considered a relatively safe procedure, although there is concern for often slow and uncertain correction of the deformity.

Spinal fusion with or without instrumentation through posterior alone or combined anterior/posterior approaches has become commonplace to stabilize relatively flexible curves secondary to congenital anomaly. Early fusion-alone surgeries demonstrated relative high pseudarthrosis rates and continued curve progression [39]. The use of spinal instrumentation to correct congenital scoliosis is first attributed to Hall and colleagues from their use of the Harrington rod in 1981 [40]. The evolution of instrumented segmental spinal systems has continued to allow improvement in surgical correction and fusion rates in this challenging population [41]. The use of posterior instrumentation at the time of the spinal fusion has several advantages, including a modest improvement in correction and a lower incidence of pseudarthrosis than a posterior spinal fusion in a Risser jacket alone [33, 40]. The use of posterior spinal implants can also negate the need for postoperative immobilization. There is a greater risk of neurologic complication, however, due to the distraction of the spinal cord while the child is under anesthesia [35]. As with any fusion surgery, attention is required to performing a meticulous arthrodesis bed, and the fusion levels must cover the entirety of the measured curve and extend to the central gravity line. Performing discectomies ensures an anterior growth arrest, which reduces or eliminates any bending of the fusion (crankshaft effect).

Hemivertebra excision was first reported by Royle in Australia in 1928 [42] and was later utilized as circumferential posterior/anterior operation [43]. It was refined by Harms and colleagues in 1991 to require a posterior-only approach [44, 45]. Most hemivertebrae have normal growth plates and will cause a resultant wedge-shaped deformity with maturation, with the exception of incarcerated and some nonsegmented forms. Due to the local deformity and asymmetric loads the adjacent vertebrae will experience, neighboring levels can demonstrate asymmetric growth as well [45]. Resection of a fully segmented hemivertebra in progressive scoliosis theoretically allows for near-complete correction of the spinal deformity and has been shown to be safe and effective [46]. Correction of these defects at a young age prevents progression and subsequent secondary structural curves, minimizing long fusion segments. Nordeen et al. describe a new technique for the treatment of congenital kyphosis, wherein the hemivertebra is *maintained*, while the adjacent intervertebral discs are excised and a rib graft strut or metal cage is placed anteriorly, followed by lateral vertebral body instrumentation [8].

Vertebral column resection entails the removal of two or more whole vertebral levels and is the most radical of all procedures for congenital vertebral malformation. Vertebrectomy creates some mobility of the spine allowing for correction yet inherently creating instability. Therefore, prior to resection, stable fixation must be achieved above and below the osteotomy sites with instrumentation. The anterior column requires reconstruction with structural graft, typically titanium mesh cages filled with autograft [33]. The procedure is fraught with risk to the neurologic structures but has been found to be a safe and effective operation in the well-trained surgeon [47].

Fusionless instrumentation surgery for congenital spinal anomaly has become an alternative intervention in treating curvature and thoracic dysplasia. Its rationale parallels traditional doctrine, that is, to achieve maximal spine length and mobility, to maintain thoracic and pulmonary function, and to minimize operations and surgical risk [7]. Expandable growing rods (GR) or vertical expandable prosthetic titanium rib devices (VEPTR) are the two main techniques utilized in the fusionless treatment of progressive spinal deformity. If a long section of spine is anomalous and deformed or if a long fusion is required to achieve control, then a fusionless, growth-oriented treatment may be preferable, particularly in the very young population. Early intervention at a period of mild deformity makes device implantation easier and probably facilitates symmetric chest growth. Late intervention, however, may have distinct advantages in some cases, as fewer lengthenings may be needed and implant anchor points and bone quality may be better with maturity. GR is considered internal bracing, working by controlling deformity through serial distractions while permitting continued spinal column growth. Some work has shown the benefit of GR in congenital malformations, with moderate curve corrections and improved space available for lung ratios [48]. VEPTR arose for the treatment of congenital scoliosis with fused rubs in children at risk for the development of thoracic insufficiency syndrome in an attempt to improve thoracic cage volumes for the developing lungs [49, 50]. The growing body of literature examining its utility has suggested benefit in the diminution of ventilator dependence, increased space available for lung, and increased lung volumes [7]. Appropriate patient selection and a thorough understanding of these instrumentation systems are required for their use.

23.7 Summary

Congenital spine abnormalities frequently result from physiologic insult during the embryologic period, with resultant disruption in somitogenesis. The resultant anatomic expression can be classified based on failures of formation, segmentation, or a combination of the two. Structural alterations of the spine at birth can further deform during growth, potentially resulting in to short stature, scoliosis, kyphosis, and kyphoscoliosis. A multidisciplinary approach should be engaged to ensure that this patient population receives comprehensive care, as multiple organ systems are frequently involved. A multitude of surgical options exist to stabilize and correct congenital deformity, and a thorough understanding of the natural history is critical for optimal outcomes.

References

1. Hensinger RN (2009) Congenital scoliosis: etiology and associations. Spine 34(17):1745–1750
2. Chan G, Dormans JP (2009) Update on congenital spine deformities: preoperative evaluation. Spine 34(17):1766–1774
3. Tanaka T, Uhthoff HK (1981) The pathogenesis of congenital vertebral malformations: a study based on observations made in 11 human embryos and fetuses. Acta Orthop Scand 52:413–425
4. Winter R, Moe J, Eilers V (1968) Congenital scoliosis: a study of 234 patients treated and untreated. J Bone Joint Surg Am 50-A:1–47
5. McMaster MJ, Ohtsuka K (1982) The natural history of congenital scoliosis: a study of two hundred and fifty-one patients. J Bone Joint Surg Am 64:1128–1147
6. Beals RK, Robbins JR, Rolfe B (1993) Anomalies associated with vertebral malformations. Spine 18(10):1329–1332
7. Yazici M, Emans J (2009) Fusionless instrumentation for congenital scoliosis: expandable spinal rods and vertical expandable prosthetic titanium rib in the management of congenital spine deformities in the growing child. Spine 34(17):1800–1807
8. Noordeen MHH, Garrido E, Tucker SK, Elsebaie HB (2009) The surgical treatment of congenital kyphosis. Spine 34(17):1808–1814
9. Bulent E, Tracy MR, Dormans JP et al (2004) Congenital scoliosis and vertebral malformations; characterization of segmental defects for genetic analysis. J Pediatr Orthop 24:674–682
10. Kuhns JG, Hormell RS (1952) Management of congenital scoliosis, review of one hundred and seventy cases. AMA Arch Surg 65:250–263
11. Bernard TN, Burke SW, Johnston CE, Roberts JM (1985) Congenital spine deformities: a review of 47 cases. Orthopedics 8:777–783
12. Purkiss SB, Driscoll B, Cole WG et al (2002) Idiopathic scoliosis in families of children with congenital scoliosis. Clin Orthop Relat Res 401:27–31
13. Winter RB, Moe JH, Lonstein JE (1983) A review of family histories in patients with congenital spinal deformities. Orthop Trans 7:32
14. Bulas D (2010) Fetal evaluation of spine dysraphism. Pediatr Radiol 40:1029–1037
15. Hervey-Jumper SL, Garton HJL, Wetjen NM, Maher CO (2011) Neurosurgical management of congenital malformations and inherited disease of the spine. Neuroimaging Clin N Am 21:719–731
16. Colombo LF, Motta F (2011) Consensus conference on Chiari: a malformation or an anomaly? Scoliosis and other orthopaedic deformities related to Chiari 1 malformation. Neurol Sci 32(Suppl 3):S341–S343, Electronic Publication Sept 14
17. Singh SK, Singh RD, Sharma A (2005) Caudal regression syndrome – case report and review of the literature. Pediatr Surg Int 21(7):578–581

18. Morita M, Miyamoto K, Nishimoto H et al (2005) Thoracolumbar kyphosing scoliosis associated with spondyloepiphyseal dyplasia congenital: a case report. Spine J 5(2):217–220
19. Viehweger E, Giacomelli MC, Glard Y et al (2009) Congenital dislocated spine: implications for orthopaedic management. J Pediatr Orthop 29(4):362–368
20. Kawakami N, Tsuji T, Imagama S et al (2009) Classification of congenital scoliosis and kyphosis: a new approach to the three-dimensional classification for progressive vertebral anomalies requiring operative treatment. Spine 34(17):1756–1765
21. Winter RB, Moe JH, Wang JF (1973) Congenital kyphosis – its natural history and treatment as observed in a study of one hundred thirty patients. J Bone Joint Surg Am 55:223–256
22. McMaster MJ, Singh H (1999) The natural history of congenital kyphosis and kyphoscoliosis. J Bone Joint Surg Am 81:1367–1383
23. Marks DS, Qaimkhani SA (2009) The natural history of congenital scoliosis and kyphosis. Spine 34(17): 1751–1755
24. Larsen W (1997) The fourth week. Differentiation of the somites and the nervous system; segmental development and integration. In: Larsen W (ed) Human embryology. Churchill Livingstone, New York, pp 73–104
25. Erol BK, Lou J, Dormans JP (2002) Etiology of congenital scoliosis. The University of Pennsylvania. Orthop J 15:37–42
26. Tanaka T, Uhthoff HK (1981) Significance of resegmentation in the pathogenesis of vertebral body malformation. Acta Orthop Scand 52:331–338
27. Campbell RM Jr, Smith MD, Mayes TC et al (2003) The characteristics of thoracic insufficiency syndrome associated with fused ribs and congenital scoliosis. J Bone Joint Surg Am 85-A:399–408
28. Rufener S, Ibrahim M, Parmar HA (2011) Imaging of congenital spine and spinal cord malformations. Neuroimaging Clin N Am 21:659–676
29. Ruanon R, Molho M, Roume J et al (2004) Prenatal diagnosis of fetal skeletal dysplasias by combining two-dimensional and three-dimensional ultrasound and intrauterine three-dimensional helical computed tomography. Ultrasound Obstet Gynecol 24:134–140
30. Cassart M, Massez A, Cos T et al (2007) Contribution of three-dimensional computed tomography in the assessment of fetal skeletal dysplasia. Ultrasound Obstet Gynecol 29:537–543
31. Cobb JR (1948) The American Academy of orthopedic surgeons instructional course lectures, vol 5. Edwards, Ann Arbor
32. Locke GR, Gardner JI, Van Epps EF (1966) Atlas-dens interval (ADI) in children: a survey based on 200 normal cervical spines. Am J Roentgenol Radium Ther Nucl Med 97:135–140
33. Hedequist DJ (2009) Instrumentation and fusion for congenital spine deformities. Spine 34(17): 1783–1790
34. Winter RB (1988) Congenital scoliosis. Orthop Clin North Am 19(2):395–408
35. MacEwen GD, Bunnell WP, Sriram K (1975) Acute neurological complications in the treatment of scoliosis. A report of the scoliosis research society. J Bone Joint Surg Am 57:404–408
36. Brustowicz RM, Hall JE (1988) In defense of the wake-up test. Anesth Analg 67:1019
37. MacLennan GD (1922) Scoliosis. BMJ 2:864
38. Andrew TA, Piggott H (1985) Growth arrest for progressive scoliosis. J Bone Joint Surg Br 67-B: 193–197
39. Winter RB, Moe JH (1982) The results of spinal arthrodesis for congenital spinal deformity in patients younger than five years old. J Bone Joint Surg Am 64:419–432
40. Hall JE, Herndon WA, Levine CR (1981) Surgical treatment of congenital scoliosis with or without Harrington instrumentation. J Bone Joint Surg Am 63:608–619
41. Hedequist DJ, Hall JE, Emans JB (2004) The safety and efficacy of spinal instrumentation in children with congenital spine deformities. Spine 29: 2081–2086
42. Royle ND (1928) The operative removal of an accessory vertebra. Med J Aust 1:467–468
43. Hedequist DJ, Hall JE, Emans JB (2005) Hemivertebra excision in children via simultaneous anterior and posterior approaches. J Pediatr Orthop 25: 60–63
44. Ruf M, Harms J (2003) Posterior hemivertebra resection with transpedicular instrumentation: early correction in children aged 1 to 6 years. Spine 28: 2132–2138
45. Ruf M, Jensen R, Letko L, Harms J (2009) Hemivertebra resection and osteotomies in congenital spine deformity. Spine 34(17):1791–1799
46. Bollini G, Docquier PL, Viehweger E et al (2006) Thoracolumbar hemivertebrae resection by double approach in a single procedure: long-term follow-up. Spine 31:1745–1757
47. Suk SI, Kim JH, Kim WJ et al (2002) Posterior vertebral column resection for severe spinal deformities. Spine 27:2374–2382
48. Elsebaie HB, Yazici M, Thompson GH et al (2007) Safety and efficacy of growing rod technique for pediatric congenital spinal deformities. In: 1st international congress of early onset scoliosis and growing spine, Madrid, 2–3 Nov 2007
49. Campbell RM, Smith MD, Hell-Vocke AK (2004) Expansion thoracoplasty: the surgical technique of opening-wedge thoracostomy. Surgical technique. J Bone Joint Surg Am 86-A:51–64
50. Emans JB, Caubet JF, Ordonez CL et al (2005) 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 30:558–568

Spondylolysis and Isthmic Spondylolisthesis

24

Sumeet Garg and Mark Erickson

24.1 Introduction

Back pain in the pediatric population is common, and increases during adolescence. The incidence of back pain has been reported as high as 50 % by the age of 15 years [1, 2]. While in most cases the pain is due to muscular pain or inflexibility, the most common structural cause of back pain in adolescents is spondylolysis with or without spondylolisthesis, the signs and symptoms of which are exaggerated upon lumbar spine hyperextension and rotation. A thorough history and physical exam is imperative when assessing for this condition in children with back pain.

Spondylolysis is a stress fracture of the pars interarticularis, usually at the L5 level. Five percent of the population has radiographic evidence of spondylolysis. The condition is believed to occur due to repetitive hyperextension of the lumbar spine and has increased prevalence in dancers, gymnasts, and football linemen. Prognosis is excellent with nonoperative treatment, and surgical management is rarely recommended.

Spondylolisthesis is a forward slippage of one vertebra on its adjacent vertebra. It occurs commonly in the degenerated lumbar spine. In children and adolescents, however, spondylolisthesis generally occurs due to displacement at the site of stress fracture of the pars interarticularis or due to dysplastic development of the L5/S1 posterior articulation. Surgery is not usually required to treat low-grade spondylolisthesis, but is almost universally required for the management of high-grade spondylolisthesis. Surgical management of high-grade spondylolisthesis is among the most debated and contentious topic of discussion among spine surgeons.

24.2 Case Example Spondylolisthesis

An 11-year-old female presented with a six-month history of back pain, stiffness, and worsening gait. She has been progressively walking on her toes. She has difficulty standing straight with her knees extended. She has lower back pain, but no numbness or paresthesias in her legs. She does have radicular pain in the anterior and lateral lower left leg. She denies bowel and bladder incontinence. She is an otherwise healthy female. Her neurologic exam demonstrated no focal deficits with 5/5 strength throughout the lower extremities and a normal sensory examination of the lower extremities. She has significant hamstring tightness, with popliteal angles of 60° on the left and 30° on the right. She has absent deep tendon reflex at the left Achilles tendon only. No neurologic symptoms with straight leg raise test bilaterally. Radiographs were taken (Fig. 24.1), demonstrating a grade 3 spondylolisthesis with dysplastic

S. Garg (✉) • M. Erickson
Department of Orthopaedic Surgery,
University of Colorado School of Medicine,
Children's Hospital Colorado,
13123 E. 16th Avenue, B060, Aurora, CO 80045, USA
e-mail: sumeet.garg@childrenscolorado.org;
mark. erickson@childrenscolorado.org

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_24, © Springer-Verlag Berlin Heidelberg 2014

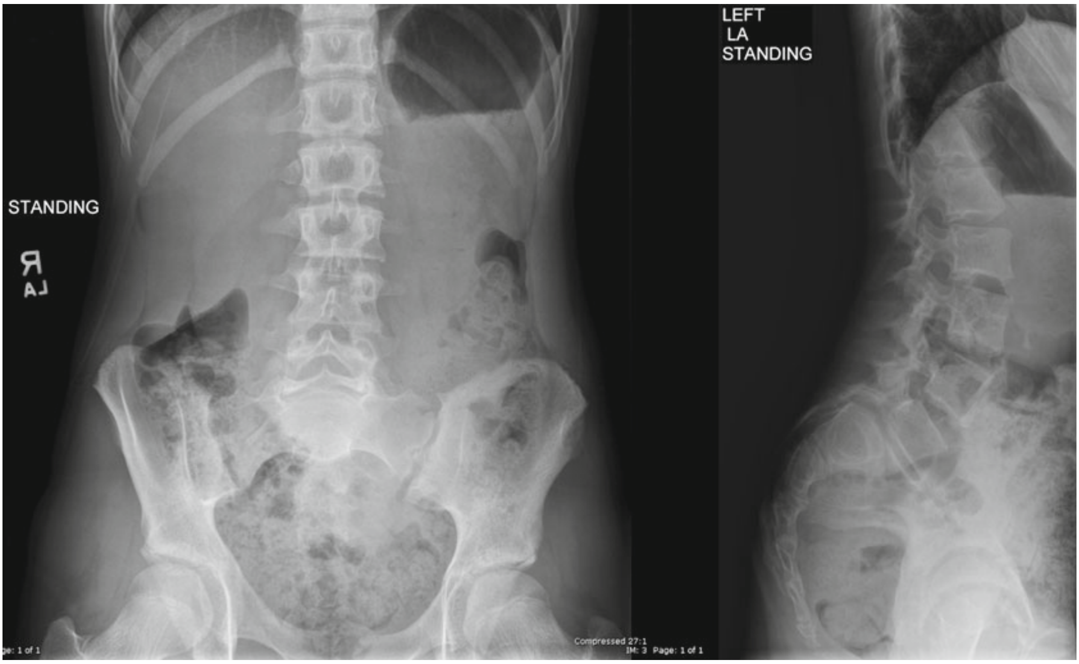


Fig. 24.1 AP and lateral lumbar spine radiographs demonstrating a grade 3 dysplastic spondylolisthesis. Note the elongated pars interarticularis of L5 without isthmic defect

features. She has been undergoing physical therapy for 6 months without any significant improvement of her symptoms.

24.3 Case Example: Spondylolysis Without Spondylolisthesis

A 12-year-old competitive gymnast presents with a 1–2-year history of vague back pain. Her pain has been increasing in frequency over the past 6 months and is now causing difficulty with completing her competitions and practice sessions. She describes the pain as dull, aching, and constant. The symptoms are at their worst in the hours after practice and competition. There are no radicular symptoms and the pain is fairly well localized to the lower lumbar region. She has normal bowel and bladder habits. Her examination is remarkable for a normal gait and neurological examination. However, her pain is readily reproduced with extension efforts in her lumbar spine (Fig. 24.2). Radiographs were obtained of her lumbar spine and are read as normal. She has had no previous treatment.

24.4 Pathology

In 90 % of patients with spondylolysis, a unilateral or bilateral defect of the pars interarticularis is observed at the L5–S1 junction [3]. Though the etiology is still unclear, spondylolysis is most likely due to the mechanical factors associated with the upright position, as there have been no reports in infants or non-ambulators, and it is rarely diagnosed before age 5; furthermore, the prevalence rate increases from 4 % at age 6 years to 6 % by age 18 years [4]. Spondylolysis is a consequence of mechanical factors such as repetitive microtrauma (stress fractures) and/or acute trauma.

The pars interarticularis is the weakest part of the posterior elements of the vertebra and is responsible for resisting shear stresses and therefore preventing anterior displacement of the vertebra (Fig. 24.3). During lumbar hyperextension, the load on the posterior bony arch in the normal spine increases dramatically across the lumbar vertebrae, with most of the force concentrating at L5. Therefore, spondylolysis is

Fig. 24.2 Provocative maneuvers, such as controlled lumbar extension, can be useful in eliciting tenderness/pain that mimics the patients' complaints. Such physical examination techniques help to elevate the clinicians' suspicion toward a diagnosis of spondylolysis

Fig. 24.4 Activities requiring extremely repetitive lumbar spine hyperextension, such as gymnastics, put athletes at risk for the development of spondylolysis

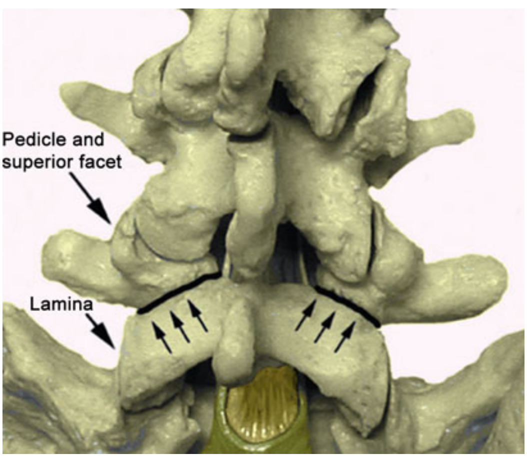


Fig. 24.3 Saw bones image depicting the anatomic location of spondylolysis, the pars interarticularis

particularly prominent among individuals participating in sports that involve repetitive hyperextension of the trunk, such as gymnastics, weight lifting, tennis, volleyball, baseball, cheerleading, swimming/diving, football, and soccer (Fig. 24.4) [5, 6]. Once a defect is created, these repetitive increased shear forces prevent healing, causing the center of gravity to move forward which further increases shear force at the lumbosacral junction. These forces are also increased as the sacrum becomes more vertical. Progression of deformity may result in secondary spondylolisthesis.

Spondylolisthesis occurs due to a variety of underlying pathology. The commonly used Wiltse classification separates spondylolisthesis into five types (Table 24.1) [7]. The isthmic and dysplastic types at the L5/S1 level are the most commonly seen in pediatrics. Isthmic type is an extension of a spondylolytic defect, whereas dysplastic type is a result of pathologic development

Table 24.1 Wiltse classification

Type 1: dysplastic
Type 2: isthmic
Type 3: degenerative
Type 4: posttraumatic
Type 5: pathologic

of the L5/S1 articulation. Dysplastic spondylolisthesis occurs either due to an elongated pars interarticularis at L5 and/or deficient facet formation at the L5/S1 level. These two anatomic features predispose to development of spondylolisthesis. Isthmic defects frequently occur in patients with preexisting dysplastic spondylolisthesis.

Another etiology-based classification was developed by Marchetti and Bartolozzi and groups isthmic and dysplastic together as developmental spondylolisthesis (type I) with subgroups being low and high grade with proposed prognostic implications on slip progression, neurologic symptoms, and surgical treatment. The second type in their classification was acquired spondylolisthesis (type II) encompassing degenerative, posttraumatic, and pathologic [8]. This chapter focuses on the management of isthmic and dysplastic (developmental) spondylolisthesis in the pediatric population.

Severity of spondylolisthesis is often described using the Meyerding classification. Each 25 % forward slippage of L5 on S1 advances the grade 1 level. Grade 5 occurs when there is 100 % displacement of L5 on S1 and the superior end plate of L5 is inferior to the sacral promontory. This condition is also known as spondyloptosis. In clinical practice, spondylolisthesis is classified as low grade (Meyerding 1 and 2) and high grade (Meyerding 3–5). Progression of deformity and need for surgery is infrequent in low-grade spondylolisthesis, especially the isthmic type. In contrast, surgery is almost always needed to relieve symptoms in high-grade spondylolisthesis [9].

Several anatomic and radiographic features have been used to describe spondylolisthesis and help evaluate risk of progression and assist with treatment decisions. These include the *Meyerding grade*, *slip angle*, and *pelvic incidence*. High-grade spondylolisthesis (Meyerding 3–5) have high rates of progression whereas low grades (Meyerding 1–2) have very low risks of progression. Slip angle is the angle between the superior end plate of L5 and the superior sacral end plate. Higher angles are more likely to have symptoms and progression. Pelvic incidence represents the combination of sacral slope and pelvic inclination and is uniform regardless of body position [10]. High pelvic incidence has been suggested to lead to increased risk of spondylolisthesis, but has not been shown to be prognostic for progression of spondylolisthesis [11]. As a fundamental descriptor of the lumbopelvic anatomy, it continues to be studied as a measure to assist with prognostic and treatment decisions.

Patients with dysplastic spondylolisthesis have a worse prognosis than those with isthmic spondylolisthesis. The dysplastic L5/S1 articulation allows for forward slippage without fracture of the pars interarticularis. The presence of intact posterior elements can lead to compression of the dural sac as L5 slips forward. Consequently, patients with dysplastic spondylolisthesis tend to be more symptomatic and present earlier than those with strictly isthmic spondylolisthesis. Neurologic symptoms are more common than in isthmic spondylolisthesis. Making the distinction between isthmic and dysplastic spondylolisthesis is critical since low-grade isthmic spondylolisthesis has a very low risk of progression versus near certain progression in dysplastic spondylolisthesis. Treatment will be more aggressive surgically in dysplastic spondylolisthesis and will be discussed in more detail in the treatment section.

24.5 History

Obtaining a thorough history is imperative when assessing a child or adolescent with back pain. Patients between the ages of 10 and 15 years old that participate in hyperextension sports are at high risk of spondylolysis. Common presentation includes dull aching pain that is localized in the lower back and exacerbated by activity. Patients may occasionally experience radicular-type pain. The nature of the pain, onset, character, location, and duration must all be taken into account. Pain associated with spondylolysis is often exacerbated with activities involving hyperextension of the lumbar spine. Gait changes may also be observed. Family history must also be taken into account, as there exists an inherited predisposition [4].

24.6 Physical Examination

Patients commonly present during late childhood or early adolescence with symptoms of localized midline low lumbar back pain and tenderness that is exacerbated by physical activity, prolonged standing, and/or lumbar hyperextension. In many cases, however, the patients are poorly able to localize their symptoms.

Effects on posture, gait, and transitional movements (spinal rhythm), as well as alignment and deformity issues, may also be observed. Sagittal and coronal alignment should be assessed, along with spinal mobility. Pain radiating to the buttock and posterior thighs upon walking or standing is a common finding. Hamstring tightness has also been noted in a majority (80 %) of patients [12].

Standard neurological exams are commonly normal among spondylolysis patients. Therefore, neurological assessment should also include sitting, standing, toe walking, heel walking, jumping, hopping, testing reflexes, and provocative maneuvers such as hyperextension of the lumbar spine.

24.7 Differential Diagnosis

Common differential diagnoses include muscle strain and overuse. Less commonly encountered are disc herniation, Scheuermann's disease, discitis, and apophyseal ring fracture. Tumors, JRA, ankylosing spondylitis, and intra-abdominal and intrathoracic causes may also be seen but are rare [1, 3, 12].

24.8 Diagnostic Imaging

24.8.1 X-Rays

Spondylolysis can be diagnosed by obtaining anteroposterior, lateral, and oblique radiographs of the lumbar spine. Bilateral pars defects can be identified on the lateral view, which should be obtained with the patient standing in order to identify any associated spondylolisthesis. The oblique view is optimal for identifying unilateral pars defects and offers a unique view of the pars often referred to as the "Scottie dog" (Fig. 24.5). A spondylolytic defect may appear as the collar or broken neck of the Scottie dog.

During the early phases, the spondylolytic defect may be easy to miss on plain radiographs and/or the pars may appear normal. If suspicious based on clinical findings, further investigation is warranted.

24.8.2 Bone Scan

A bone scan or SPECT scan is helpful in diagnosing spondylolysis in patients with normal x-rays (Fig. 24.6). Increased metabolic activity on the scan ("hot spots") are areas of increased osteoblastic activity indicating a stress reaction or subacute injury which may precede a fracture, while decreased metabolic activity ("cold spots") signify areas of nonunion [13].

24.8.3 CT Scan

A CT scan may offer superior visualization of the area in question. However, slices must be small (1.0–1.5 mm) or the lesion may be overlooked. In instances where a SPECT scan denotes an area of abnormal metabolic activity, a CT scan can then be targeted to this specific anatomic region. (Fig. 24.7)

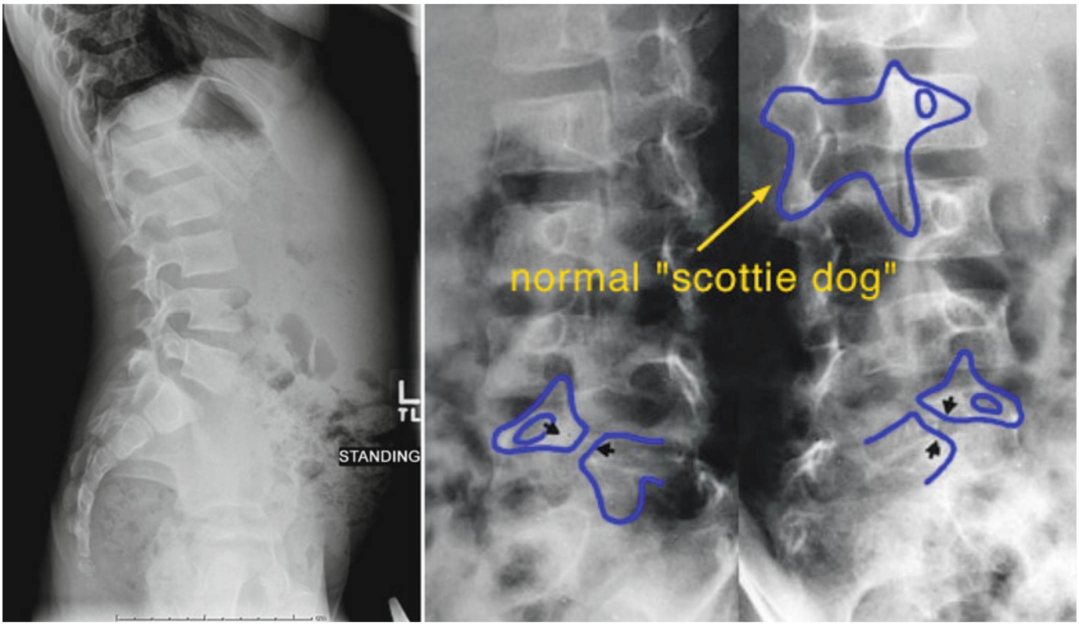


Fig. 24.5 Occasionally, bilateral spondylolyses can be visualized on lateral projection x-rays as seen in this figure. However, more commonly they are best seen on oblique views. Note that the "neck" of the "Scottie dog" is representative anatomically of the pars interarticularis

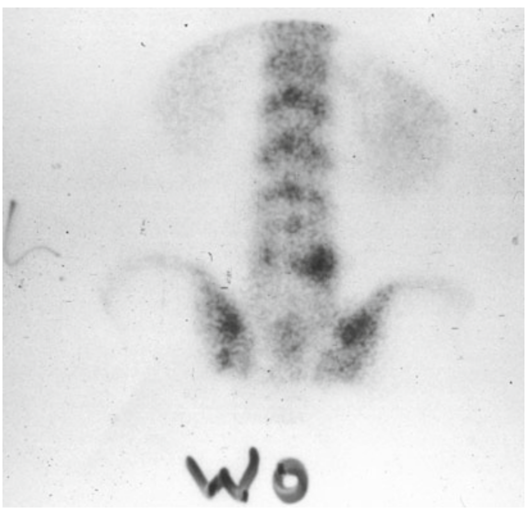


Fig. 24.6 This SPECT scan denotes a focal increase in metabolic activity unilaterally at the L5 region

Fig. 24.7 Fine cut CT scans are necessary in order to discern the spondylolytic defect, as seen in this image

24.8.4 MRI

An MRI scan can also be a useful diagnostic tool. If radicular symptoms are present, an MRI scan is indicated. MRI provides the opportunity to assess for associated degenerative disc disease and disc herniation. Viewing the pedicle, disc space, and parasagittal views at the level of the pars allows for assessment of the lesion as well as the surrounding soft tissue structures [14, 15].

24.9 Treatment: Spondylolysis

24.9.1 Nonoperative

Treatment for spondylolysis is largely nonoperative, unless prolonged conservative measures are unsuccessful. Management should largely focus on core strengthening, activity modification, and immobilization [16].

Core fitness programs often include physical therapy and Pilates or yoga-type activities. Focus should be placed on avoiding lumbar extension activities while strengthening core muscles and increasing the flexibility of the hamstrings.

Exercises should be complemented with activity modification. Patient recommendations should include avoiding high-risk hyperextension activities and sports until after pain resolves. Subsequently, patients may return gradually to normal activity. Patients who stop sports until pain resolution have been shown to have better outcomes [17]. Return to the same high-risk hyperextension activities, however, can lead to repetitive cycles of recurring back pain.

Immobilization, such as casting or bracing, may be used to reduce the shear stresses acting on the pars of the affected vertebrae. Bracing should include the application of a thoracolumbar spinal orthosis for 3–6 months. Bracing and casting have proved effective in successfully healing the majority of unilateral or bilateral spondylolytic defects if diagnosed early [3, 14, 18]. Close follow-up is imperative and patients whose pain does not resolve must be reevaluated.

24.9.2 Operative

Only after failure of prolonged attempts at nonoperative management should surgery be con-sidered for spondylolysis. In cases where it is unclear as to whether or not the spondylolysis is the source of the patient's pain, a localized in-jection into the pars interarticularis may prove beneficial.

The options available for surgical management are repair of the spondylolytic defect (pars repair) and single level fusion (Fig. 24.8). Indications for a single level fusion include failed attempts at pars repair, bilateral spondylolysis associated with mild spondylolisthesis, and bilateral L5 spondylolysis [12]. In some individuals, the pars interarticularis at L5 can be quite small and atrophic. The high likelihood of failure of pars repair in these patients makes L5–S1 fusion a more attractive option.

The optimal utility for pars repair would be a patient with mid-lumbar spondylolysis, no spondylolisthesis, no disc disease, and a positive local injection conforming pain relief. The principles surrounding a successful attempt at pars repair involve creating a biologic environment suitable to bone healing and achieving biomechanical stability. First, resecting fibrous tissue from the spondylolytic defect site and subsequently adding bone graft material establish the biologic environment. Stability can be achieved by a variety of implant choices including wires, intralaminar screws, and pedicle screws with wires and/or hooks.

24.9.3 Postoperative Management

Immobilization after surgery is not necessary and has not been shown to improve healing rates. Postoperative management consists of a 3–4-day hospital course, immediate ambulation, and expected return to full activities by 3–4 months.

24.9.4 Outcomes

With careful patient selection, including attention to detail in the nonoperative course of management, overall excellent results can be expected from operative management of spondylolysis. The vast majority of patients experience excellent relief of their pain and return to normal activities. Unfortunately, the biology surrounding spondylolytic defects is not always conducive to healing. Failed attempts at pars repair can typically be salvaged with a single level fusion with excellent outcomes being the standard.

24.9.5 Complications

Failure of healing in pars repair and pseudarthrosis after single level attempts at fusion are accepted risks in this population. The current literature is sparse relative to actual healing and

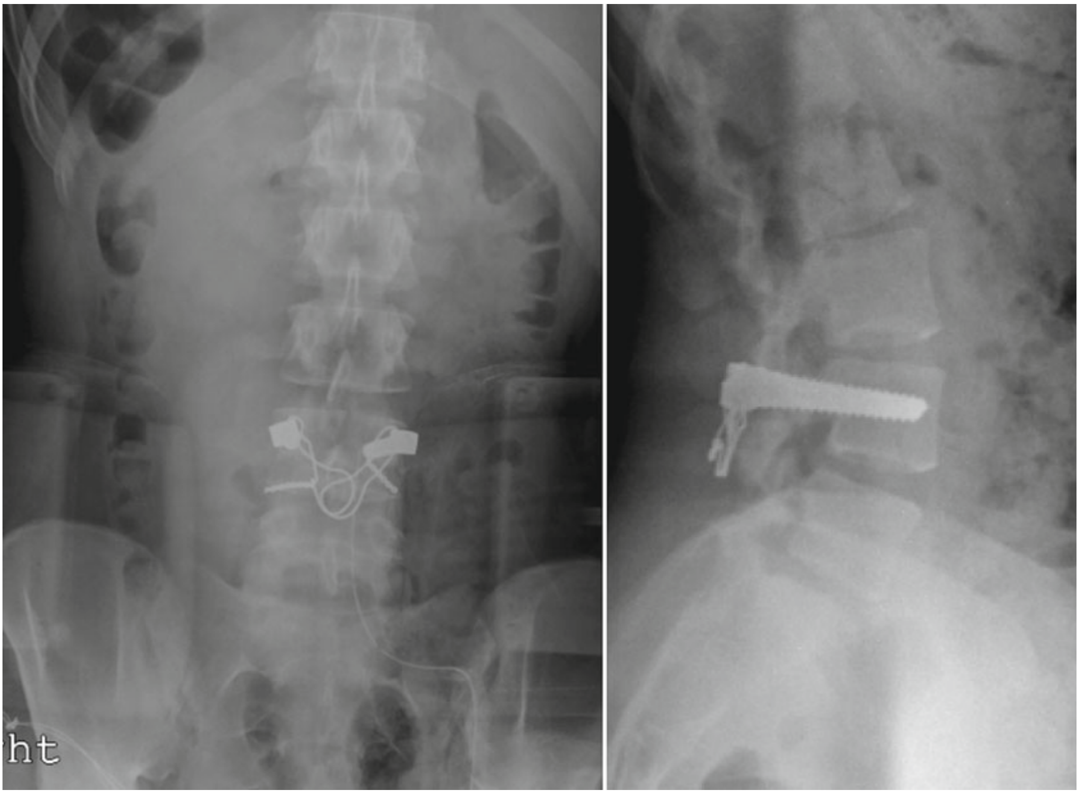


Fig. 24.8 AP and lateral radiographs demonstrating postoperative changes after surgical pars repair at L4. A bilateral pedicle screw/compression wiring technique was utilized to create stability and compression across the pars defect

fusion rates with high-quality data lacking. In general, rates of healing/fusion are reported to be in excess of 90 %. The risk of iatrogenic neurologic injury in this group of patients is low. Other complications that occur are consistent with all spine surgery such as infection, dural tear, implant prominence, implant failure, and medical complications.

24.10 Treatment: Spondylolisthesis

24.10.1 Nonoperative

Asymptomatic patients with low-grade isthmic spondylolisthesis do not require any routine treatment or follow-up. Progression is rare, and if it occurs, almost universally will cause symptoms and prompt return to the clinic for evaluation. Families should still be counseled to follow up for new back pain or neurologic symptoms. No activity restrictions are recommended. Bracing is not efficacious. Long-term studies have shown only rare cases of progression of pure isthmic spondylolisthesis. Symptomatic patients with low-grade isthmic spondylolisthesis benefit from a physical therapy program emphasizing spinal flexibility, core strengthening, aerobic conditioning, and hamstring stretching. Home program with daily exercises is recommended for best results. Advanced imaging with MRI is not indicated unless the nonoperative program fails to relieve symptoms. Long-term follow-up studies have demonstrated resolution of symptoms in more than 80 % of patients with symptomatic low-grade isthmic spondylolisthesis without surgery [16, 19, 20].

Patients with dysplastic low-grade spondylolisthesis should be followed up annually, regardless of symptoms, to monitor for progression. Symptomatic patients will benefit from a nonoperative therapy program. Surgery should be strongly considered for any patients with dysplastic spondylolisthesis showing progression regardless of symptoms. Once demonstrated, progression universally continues and will nearly always become symptomatic. Treatment prior to the deformity becoming high grade simplifies treatment decisions and carries less risk than surgical management of high-grade spondylolisthesis.

24.10.2 Operative

Operative treatment is indicated for patients with symptomatic low-grade isthmic spondylolisthesis that fails to respond to nonoperative management and for patients with dysplastic spondylolisthesis that is progressive. Presence of neurologic symptoms in particular is a generally accepted indication for surgical treatment. High-grade spondylolisthesis of any type also usually requires surgical treatment since symptoms are invariably present and nonoperative treatment is ineffective. For low-grade spondylolisthesis, uninstrumented posterolateral fusion from the transverse processes of L5 to the sacral ala is the gold standard. Surgical treatment of high-grade spondylolisthesis is more controversial. Treatment options proposed range from uninstrumented posterolateral fusion to full reduction of L5 on the sacrum via anterior and posterior approach. The decision whether to not reduce, partially reduce, or fully reduce the spondylolisthesis deformity is a topic of great contention among spine specialists.

24.10.3 Uninstrumented In Situ Fusion

Posterolateral fusion, in situ, without instrumentation is the preferred treatment for symptomatic low-grade spondylolisthesis without neurologic symptoms. This is done via a paramedian muscle splitting approach as originally described by Wiltse et al. [21]. This approach splits the paraspinal muscles and allows for direct exposure of the transverse process of L5 and sacral ala. Iliac crest autograft is placed between these structures, and often a flap of the superior sacral ala can be lifted off with an osteotome to bridge the gap to the transverse process of L5 and provide a base for the autograft. Immobilization after surgery is not necessary and has not been shown to improve fusion rates.

Results of uninstrumented in situ fusion for low-grade spondylolisthesis without neurologic symptoms are excellent [22, 23]. In general fusion, rates between 80 and 90 % are reported with generally excellent relief of symptoms. Some argue for this type of treatment even in patients *with* neurologic symptoms. Reports have demonstrated relief of radicular symptoms, hamstring tightness, and improvement of altered gait after in situ fusion. Persistent neurologic symptoms after arthrodesis can be addressed by second-stage decompression. Other complications include pseudarthrosis and consequent progression of spondylolisthesis. This occurs in 10–20 % of patients, even in some who have radiographically solid fusion [22]. Surprisingly, despite a high rate of pseudarthrosis, often symptom relief at long-term follow-up is equivalent despite quality of the fusion mass after in situ uninstrumented fusion [24]. Although no dural exposure or reduction is attempted, cauda equina dysfunction has still been reported after in situ uninstrumented fusion and is best addressed by immediate decompression [25].

The presence of motor defects or bowel or bladder dysfunction, however, should compel surgeons to neurologic decompression as part of the surgical treatment. Traditionally, a Gill laminectomy is done with complete removal of the posterior elements of L5. In isthmic cases, or dysplastic cases with isthmic defect, there is retained superior articular facet of L5 that also must be removed. The decompression should allow for full relief of the L5 and S1 nerve roots along with the dural sac. Frequently, in high-grade spondylolisthesis, sacral dome osteotomy is also required to relieve tension on the dural sac. Return of pulsations of the dural sac generally heralds appropriate decompression. Although in situ posterolateral fusion can also be utilized following thorough neurologic decompression, instrumentation is usually placed because of the instability created by the bony resection.

24.10.4 Instrumented Fusion (Includes Reduction Discussion, Transsacral Implants, Fibular Graft)

Addition of instrumentation to fusion for lumbar spondylolisthesis has been established to improve fusion rates in adult degenerative spondylolisthesis. If reduction of the deformity in isthmic or dysplastic spondylolisthesis is to be attempted, then instrumentation is mandatory. The decision to reduce, and how much to reduce, a spondylolisthesis deformity in children and adolescents is controversial. Proponents believe correction of the deformity, especially the slip angle, will improve long-term functional outcomes by correction of the abnormal sagittal plane caused by the spondylolisthesis [26]. Furthermore, they believe fusion rates are higher with instrumented versus uninstrumented in situ fusion. High-quality data is lacking for both of these assumptions. Those against reduction point to the lack of evidence supporting reduction along with presence of data showing increased rates of neurologic deficits following attempts at reduction.

Increasingly, the push from experts in the field has been to a middle ground in high-grade spondylolisthesis with decompression of the neural elements, partial reduction with a primary goal of slip angle improvement as a way to restore sagittal plane alignment without the neurologic risks of a full reduction [27]. If reduction is to be attempted, decompression of the dural sac and visualization of the nerve roots are mandatory to avoid iatrogenic compression with reduction. Posterior sacral dome osteotomy is often needed to decompress the dural sac. Strong segmental pedicle screw fixation of the vertebrae and sacrum is important to achieve and hold reduction. Fixation of L5–S1 can be either via segmental screws or screws directed supero-posterior from the S1 pedicle into the L5 vertebral body [28]. High-grade spondylolisthesis usually benefits from additional fixation to L4 and into the pelvis via iliac bolts or S2 alar iliac screws. Reduction is done by patient positioning with the hips extended on the table preoperatively and by distraction and posterior reduction of L5 (and L4 if instrumented). Extended tab (reduction) pedicle screws are useful in L4 and L5 when attempting reduction to allow for posterior force on these levels during attempted reduction.

In high-grade cases, including spondyloptosis, spinal fixation can also be supplemented with a fibular auto- or allograft from the sacrum into the body of L5 between the S1 and L5 screws. This can be done regardless of whether reduction is being attempted. This technique was popularized by Bohlman and affords excellent fusion rates in high-grade spondylolisthesis with relief of symptoms [29–31]. Bohlman did not have any neurologic deficits postoperatively in his published series, but other authors have described neurologic deficit postoperatively with this technique [31]. Use of an ACL reamer through the sacrum and L5 is a helpful technique for bony tunnel preparation for the fibular allograft. The graft can be placed either from the posterior approach as described by Bohlman or anteriorly as described by Sasso et al. [32]. If reduction is attempted, anterior support in the L5–S1 disc is beneficial for supporting the corrected sagittal plane and for improving fusion rates. This can be done either via anterior interbody fusion or posterior or transforaminal lumbar interbody fusion based on the surgeon's preference.

Results of instrumented fusion with reduction have been published recently demonstrating high fusion rates, improved clinical status, and low rates of neurologic injury [27, 33–36]. No direct trials comparing uninstrumented and instrumented fusion with reduction have been done to compare the two techniques. Pseudarthrosis is a complication of instrumented fusion with reduction; however, the rates seem to be less than with uninstrumented fusion in the small series reported. The most feared complication, neurologic deficit, which occurs in up to 25 % of cases, however, is most often transient with a permanent deficit incidence less than 5 % [37, 38]. A recent large review of the SRS morbidity and mortality database for pediatric spondylolisthesis surgery reported a 5 % rate of neurologic injury, of which 94 % of patients had improvement (half of who had full resolution) [37]. Other complications that occur less frequently are similar to those for all spine surgery such as infection, dural tear, implant prominence, implant failure, and miscellaneous postoperative medical complications.

24.10.5 L5 Corpectomy and Fusion

Gaines has described a comprehensive surgical treatment of high-grade spondylolisthesis and spondyloptosis that involves complete vertebrectomy of L5 [39]. The body of L5 is removed initially via an anterior approach, followed by posterior decompression, instrumentation of L4 and the sacrum, and reduction of L4 onto the sacrum with posterolateral fusion. There is a high rate of neurologic injury described, with 23 of 30 patients having transient motor and/or sensory L5 injury, of which two have permanent motor deficit. Other surgeons have not published results of this surgical treatment. The neurologic complication rate described by Gaines is higher than other described reduction techniques preserving L5 from a posterior-only approach. Although some centers continue to offer this treatment for high-grade spondylolisthesis, most utilize some form of instrumented posterior fusion with or without reduction.

24.10.6 Postoperative Management

After surgery, it is recommended to initially keep the hips and knees flexed with pillows under the knees. This reduces neural tension. The pillows are slowly removed over the postoperative course as the patient tolerates. Mobilization is encouraged as soon as postoperative day #1. Neuropathic pain is common, in particular when there has been dural retraction as part of the surgical procedure. Medications such as gabapentin and tricyclic antidepressants can be useful in the management of acute neuropathic pain postoperatively. These can usually be weaned off within 1–2 months following surgery.

In the past, patients with uninstrumented fusion in situ were often immobilized in a pantaloon cast after surgery. Currently, most surgeons do not use any immobilization following surgical treatment of spondylolisthesis regardless of use of instrumentation. Activity restrictions postoperatively should include limitation of bending at the waist for a minimum of 6 months. Patients can generally resume light activities such as swimming, running, and bicycling after 1–2 months when the soft tissue is healed. Contact sports are discouraged until at least 6 months, ideally until radiographic evidence of arthrodesis is appreciated.

24.10.7 Case Example: Spondylolisthesis Outcome

The presence of dysplastic features and high-grade spondylolisthesis prompted the recommendation for surgical treatment. The patient underwent posterior spinal fusion with instrumentation from L5 to S1 with transforaminal lumbar interbody fusion at L5–S1. This was done through a left-sided hemilaminectomy which allowed for decompression of the dural sac and in particular the left L5 and S1 nerve roots. There was no attempt at reduction, only postural correction of the slip angle to neutral via positioning with hips extended and knees flexed on the operating table. She had relief of her radicular symptoms immediately, and her gait improved steadily after surgery. By 6 months after surgery, she had complete relief of her back and leg pain and restoration of normal gait. Radiographs demonstrated stable fixation (Fig. 24.9).

24.10.8 Case Example: Spondylolysis Without Spondylolisthesis Outcome

SPECT scan imaging revealed a focal area of increased uptake unilaterally at the right L5 pars interarticularis. Subsequently, she stopped her gymnastics activities. Physical therapy focused on achieving and maintaining core fitness, while eliminating lumbar spine hyperextension, was introduced successfully. Over the ensuing 4 months, she experienced complete resolution

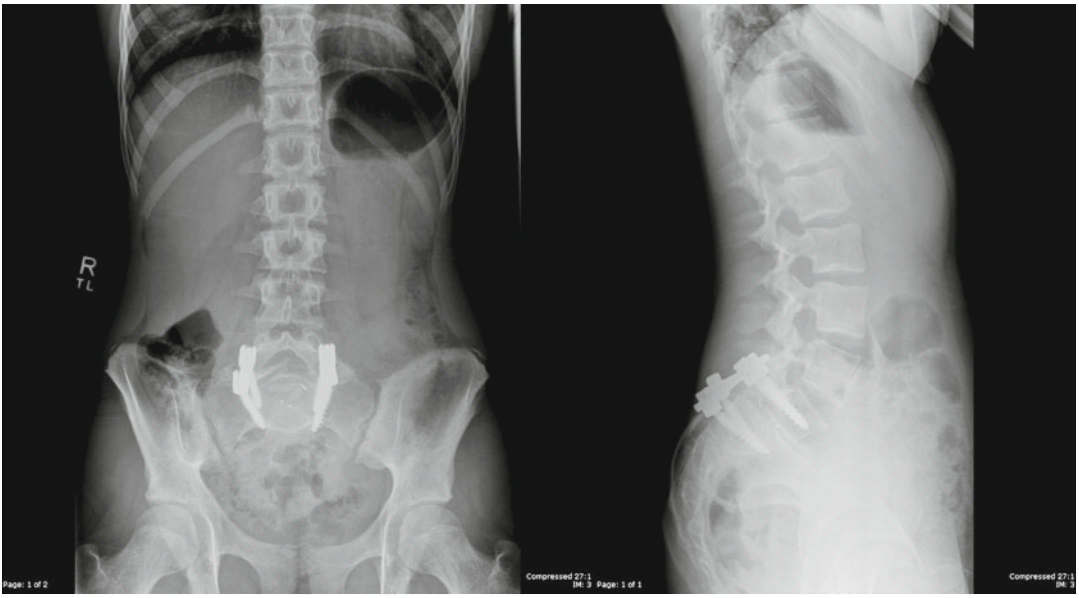


Fig. 24.9 AP and lateral lumbar spine radiographs demonstrating postsurgical changes of L5–S1 posterior fusion with TLIF at L5–S1. Radiographic markers identify cage in the L5–S1 interspace

of her back pain. Her gymnastics activity was gradually reintroduced over an additional 2 months without recurrence of her symptoms.

Questions

1. Which types of spondylolisthesis are most common in the pediatric population?
   (a) Pathologic and isthmic
   (b) Pathologic and degenerative
   (c) Isthmic and dysplastic
   (d) Isthmic and posttraumatic
   Preferred response (c): Isthmic and dysplastic are the most common types of spondylolisthesis in the pediatric population and can occur in tandem. Degenerative spondylolisthesis is common in the adult population, most frequently at L4–L5. Pathologic spondylolisthesis is rare in all ages.
2. Patients with high pelvic incidence have:
   (a) Increased prevalence of spondylolisthesis
   (b) Increased rates of progression of deformity
   (c) Low fusion rates following surgery
   (d) Elevated sacral slope
   Preferred response (a): While some argue that increased pelvic incidence results in increased rates of progression, only an increased prevalence of spondylolisthesis has been demonstrated in population studies. No relationship to fusion rates following surgery has been established. Pelvic incidence is the summation of sacral slope and pelvic tilt.
3. A patient with spondylolysis is most likely to have long-term problems including?
   (a) Progressive spondylolisthesis
   (b) Incontinence
   (c) Chronic back pain
   (d) No long-term spinal problems
   Preferred answer (d): Spondylolysis generally is a self-limiting problem that responds to improving back strength and flexibility. Progression to spondylolisthesis is infrequent, and incontinence has not been described. Chronic back pain can occur with spondylolysis, but is not the most likely outcome.
4. Surgical intervention is most likely to be required in which clinical scenario?
   (a) 16-year-old gymnast with symptomatic spondylolysis
   (b) 8-year-old with dysplastic spondylolisthesis, Meyerding grade 2
   (c) 18-year-old with isthmic spondylolisthesis, Meyerding grade 1

(d) 12-year-old with isthmic spondylolisthesis, Meyerding grade 2

Preferred answer (b): A young child with dysplastic spondylolisthesis has a high risk of progression of deformity and development of neurologic symptoms. Any progression should prompt consideration of surgical treatment with spinal fusion. Patients with low-grade isthmic spondylolisthesis have an extremely low rate of progression and should be treated symptomatically with strengthening and flexibility.

References

1. Burton AK, Clarke RD, McClune TD, Tillotson KM (1996) The natural history of low back pain in adolescents. Spine (Phila Pa 1976) 21(20):2323–2328
2. Balague F, Skovron ML, Nordin M, Dutoit G, Pol LR, Waldburger M (1995) Low back pain in schoolchildren. A study of familial and psychological factors. Spine (Phila Pa 1976) 20(11):1265–1270
3. Morita T, Ikata T, Katoh S, Miyake R (1995) Lumbar spondylolysis in children and adolescents. J Bone Joint Surg Br 77(4):620–625
4. Tsirikos AI, Garrido EG (2010) Spondylolysis and spondylolisthesis in children and adolescents. J Bone Joint Surg Br 92(6):751–759
5. Kim HJ, Green DW (2011) Spondylolysis in the adolescent athlete. Curr Opin Pediatr 23(1):68–72
6. Soler T, Calderon C (2000) The prevalence of spondylolysis in the Spanish elite athlete. Am J Sports Med 28(1):57–62
7. Wiltse LL, Newman PH, Macnab I (1976) Classification of spondylolisis and spondylolisthesis. Clin Orthop Relat Res 117:23–29
8. Marchetti P, Bartolozzi P (1997) Classification of sponydlolisthesis as a guideline for treatment. In: Bridwell K (ed) The textbook of spinal surgery. Lippincott-Raven, Philadelphia, p 1211
9. Meyerding H (1932) Spondylolisthesis. Surg Gynecol Obstet 54:371
10. Labelle H, Roussouly P, Berthonnaud E, Dimnet J, O'Brien M (2005) The importance of spino-pelvic balance in L5-s1 developmental spondylolisthesis: a review of pertinent radiologic measurements. Spine (Phila Pa 1976) 30:S27–S34
11. Huang RP, Bohlman HH, Thompson GH, Poe-Kochert C (2003) Predictive value of pelvic incidence in progression of spondylolisthesis. Spine (Phila Pa 1976) 28:2381–2385; discussion 5
12. Hensinger RN (1989) Spondylolysis and spondylolisthesis in children and adolescents. J Bone Joint Surg Am 71(7):1098–1107
13. van den Oever M, Merrick MV, Scott JH (1987) Bone scintigraphy in symptomatic spondylolysis. J Bone Joint Surg Br 69(3):453–456
14. Sairyo K, Katoh S, Takata Y et al (2006) MRI signal changes of the pedicle as an indicator for early diagnosis of spondylolysis in children and adolescents: a clinical and biomechanical study. Spine (Phila Pa 1976) 31(2):206–211
15. Yamane T, Yoshida T, Mimatsu K (1993) Early diagnosis of lumbar spondylolysis by MRI. J Bone Joint Surg Br 75(5):764–768
16. Pizzutillo PD, Hummer CD 3rd (1989) Nonoperative treatment for painful adolescent spondylolysis or spondylolisthesis. J Pediatr Orthop 9:538–540
17. El Rassi G, Takemitsu M, Woratanarat P, Shah SA (2005) Lumbar spondylolysis in pediatric and adolescent soccer players. Am J Sports Med 33(11):1688–1693
18. Sys J, Michielsen J, Bracke P, Martens M, Verstreken J (2001) Nonoperative treatment of active spondylolysis in elite athletes with normal X-ray findings: literature review and results of conservative treatment. Eur Spine J 10(6):498–504
19. Frennered AK, Danielson BI, Nachemson AL (1991) Natural history of symptomatic isthmic low-grade spondylolisthesis in children and adolescents: a seven-year follow-up study. J Pediatr Orthop 11:209–213
20. Beutler WJ, Fredrickson BE, Murtland A, Sweeney CA, Grant WD, Baker D (2003) The natural history of spondylolysis and spondylolisthesis: 45-year follow-up evaluation. Spine (Phila Pa 1976) 28:1027–1035; discussion 35
21. Wiltse LL, Bateman JG, Hutchinson RH, Nelson WE (1968) The paraspinal sacrospinalis-splitting approach to the lumbar spine. J Bone Joint Surg Am 50:919–926
22. Seitsalo S, Osterman K, Hyvarinen H, Schlenzka D, Poussa M (1990) Severe spondylolisthesis in children and adolescents. A long-term review of fusion in situ. J Bone Joint Surg Br 72:259–265
23. Lenke LG, Bridwell KH, Bullis D, Betz RR, Baldus C, Schoenecker PL (1992) Results of in situ fusion for isthmic spondylolisthesis. J Spinal Disord 5:433–442
24. Harris IE, Weinstein SL (1987) Long-term follow-up of patients with grade-III and IV spondylolisthesis. Treatment with and without posterior fusion. J Bone Joint Surg Am 69:960–969
25. Schoenecker PL, Cole HO, Herring JA, Capelli AM, Bradford DS (1990) Cauda equina syndrome after in situ arthrodesis for severe spondylolisthesis at the lumbosacral junction. J Bone Joint Surg Am 72:369–377
26. Labelle H, Roussouly P, Chopin D, Berthonnaud E, Hresko T, O'Brien M (2008) Spino-pelvic alignment after surgical correction for developmental spondylolisthesis. Eur Spine J 17:1170–1176
27. Boachie-Adjei O, Do T, Rawlins BA (2002) Partial lumbosacral kyphosis reduction, decompression, and posterior lumbosacral transfixation in high-grade isthmic spondylolisthesis: clinical and radiographic results in six patients. Spine (Phila Pa 1976) 27:E161–E168

28. Abdu WA, Wilber RG, Emery SE (1994) Pedicular transvertebral screw fixation of the lumbosacral spine in spondylolisthesis. A new technique for stabilization. Spine (Phila Pa 1976) 19:710–715
29. Bohlman HH, Cook SS (1982) One-stage decompression and posterolateral and interbody fusion for lumbosacral spondyloptosis through a posterior approach. Report of two cases. J Bone Joint Surg Am 64:415–418
30. Smith MD, Bohlman HH (1990) Spondylolisthesis treated by a single-stage operation combining decompression with in situ posterolateral and anterior fusion. An analysis of eleven patients who had long-term follow-up. J Bone Joint Surg Am 72:415–421
31. Roca J, Ubierna MT, Caceres E, Iborra M (1999) One-stage decompression and posterolateral and interbody fusion for severe spondylolisthesis. Spine (Phila Pa 1976) 24:709–714
32. Sasso RC, Shively KD, Reilly TM (2008) Transvertebral transsacral strut grafting for high-grade isthmic spondylolisthesis L5-S1 with fibular allograft. J Spinal Disord Tech 21:328–333
33. Goyal N, Wimberley DW, Hyatt A et al (2009) Radiographic and clinical outcomes after instrumented reduction and transforaminal lumbar interbody fusion of mid and high-grade isthmic spondylolisthesis. J Spinal Disord Tech 22:321–327
34. Laursen M, Thomsen K, Eiskjaer SP, Hansen ES, Bunger CE (1999) Functional outcome after partial reduction and 360 degree fusion in grade III-V spondylolisthesis in adolescent and adult patients. J Spinal Disord 12:300–306
35. Hu SS, Bradford DS, Transfeldt EE, Cohen M (1996) Reduction of high-grade spondylolisthesis using Edwards instrumentation. Spine (Phila Pa 1976) 21:367–371
36. Ruf M, Koch H, Melcher RP, Harms J (2006) Anatomic reduction and monosegmental fusion in high-grade developmental spondylolisthesis. Spine (Phila Pa 1976) 31:269–274
37. Fu KM, Smith JS, Polly DW Jr et al (2011) Morbidity and mortality in the surgical treatment of six hundred five pediatric patients with isthmic or dysplastic spondylolisthesis. Spine (Phila Pa 1976) 36:308–312
38. Sansur CA, Reames DL, Smith JS et al (2010) Morbidity and mortality in the surgical treatment of 10,242 adults with spondylolisthesis. J Neurosurg Spine 13:589–593
39. Gaines RW (2005) L5 vertebrectomy for the surgical treatment of spondyloptosis: thirty cases in 25 years. Spine (Phila Pa 1976) 30:S66–S70

Spine Trauma: Occipital and Upper Cervical Spine

25

Xuan Lo, Raymond W. Hwang, Harvey E. Smith, David Gendelberg, and Alexander R. Vaccaro

25.1 Case

A 72-year-old healthy female presents with neck pain after a fall from standing. A trauma evaluation was performed per acute trauma life support protocol. She is found to have stable vital sign and is alert and oriented. No other injuries are identified. Her neurologic examination is without focal deficits throughout her upper and lower extremities. A CT scan of her neck was obtained (Fig. 25.1).

The patient was found to have a type IIC odontoid fracture with displacement. The patient was taken to the operating room for a definitive fixation utilizing a screw-rod construct with C1 lateral mass screws and C2 par screws (Fig. 25.2). The patient progressed to a solid fusion.

X. Lo
Tufts University School of Medicine,
Boston, MA, USA

R.W. Hwang, M.D.
Department of Orthopaedic Surgery,
New England Baptist Hospital,
Boston, MA, USA

H.E. Smith, M.D.
Department of Orthopaedic Surgery,
Tufts University School of Medicine,
New England Baptist Hospital,
Boston, MA, USA

D. Gendelberg
Department of Orthopaedic Surgery,
The Rothman Institute,
Philadelphia, PA, USA

A.R. Vaccaro, M.D., Ph.D. (✉)
Department of Orthopaedic Surgery,
Thomas Jefferson University and The Rothman Institute,
925 Chestnut Street, Fifth Floor,
Philadelphia, PA 191017, USA
e-mail: alexvaccaro3@aol.com

25.2 Anatomy

The skull base consists of the clivus, the occipital condyles, and the foramen magnum. The cranium articulates with the spinal column through two uniquely shaped vertebrae, C1 and C2. These bones stabilize the neck and protect the spinal cord while allowing for a substantial amount of flexion, extension, and rotation of the head. The base of the skull articulates with C1 through two convex projections, the occipital condyles. These protrude inferiorly from the anterolateral edges of the foramen magnum.

The atlas directly supports the weight of the skull; it is a ring-shaped structure comprised of two neural arches that connect a pair of lateral masses. The occipital condyles rest directly on the concave superior facets of the C1 lateral masses. This articulation provides the most range of motion of any spinal level: flexion and extension of 25° with rotation and lateral bending of 5° each [1]. The stability of the occipitocervical junction arises from the ligaments of the facet capsules (occiput–C1 and C1–C2), the anterior longitudinal ligament, the tectorial membrane (a continuation of the posterior longitudinal ligament), the apical and alar ligaments, and the transverse ligament of the atlas; the transverse ligament coupled with the superior and inferior

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_25, © Springer-Verlag Berlin Heidelberg 2014

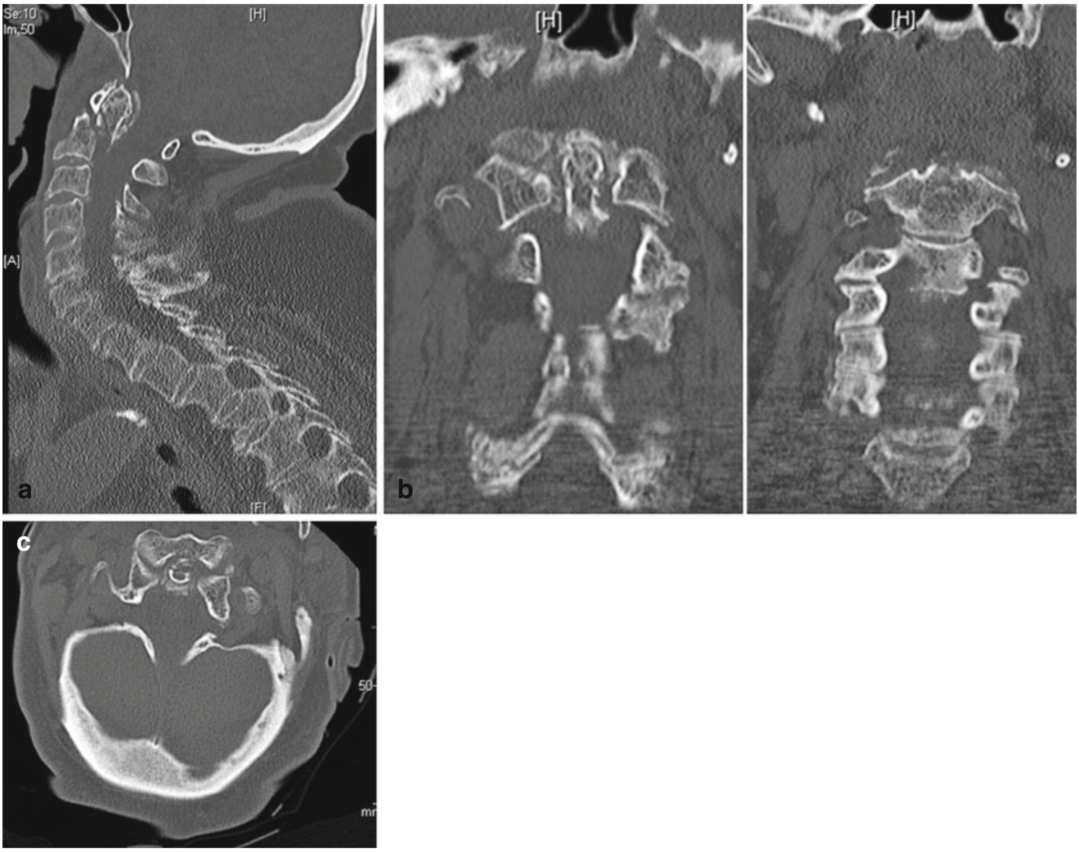


Fig. 25.1 CT scan of the cervical spine demonstrating a Type IIC odontoid fracture

longitudinal fascicules forms the cruciate ligament complex. Furthermore, the atlanto-occipital membrane, interspinous ligament, ligamentum nuchae, rectus posterior major/minor, and obliquus capitis superior/inferior provide additional stability to this joint. The transverse process of the atlas contains the foramen transversarium and vertebral artery. After ascending through this foramen, the vertebral artery courses posteriorly and medially around the lateral mass and over the posterior arch before entering the dura mater, coalescing with the contralateral vertebral artery to form the basilar artery.

The atlas in turn articulates with a second uniquely shaped vertebra (C2) or the axis. The axis is named for the odontoid process or dens that extends cranially from the body of the axis into the ring of the atlas, providing an axis for rotation. The lateral masses extend posterolaterally from the body and include the superior and inferior facets. The foramen transversarium, through which the vertebral artery travels, extends laterally from the lateral masses. Representing the embryological vertebral body of C1 [2], the odontoid articulates with the anterior arch of C1 via a true synovial joint and is stabilized against this arch by the transverse ligament. The transverse ligament arises from the medial tubercles of the lateral masses of the C1 and resists anterior displacement of C1 on C2 and extremes of flexion. In addition to the articulation of the odontoid process with the anterior arch, the superior facets of C2 articulate with the inferior facets of C1 via a biconcave synovial joint [2, 3]. Given the natural axial arrangement of the atlantoaxial articulation, this joint provides rotation of 40°, flexion and extension of 20°, and lateral bending of 5° [1]. Unlike other areas of the spine, there is no intervertebral disk between C1 and C2. The atlas is sometimes described as a “bony meniscus” between the skull

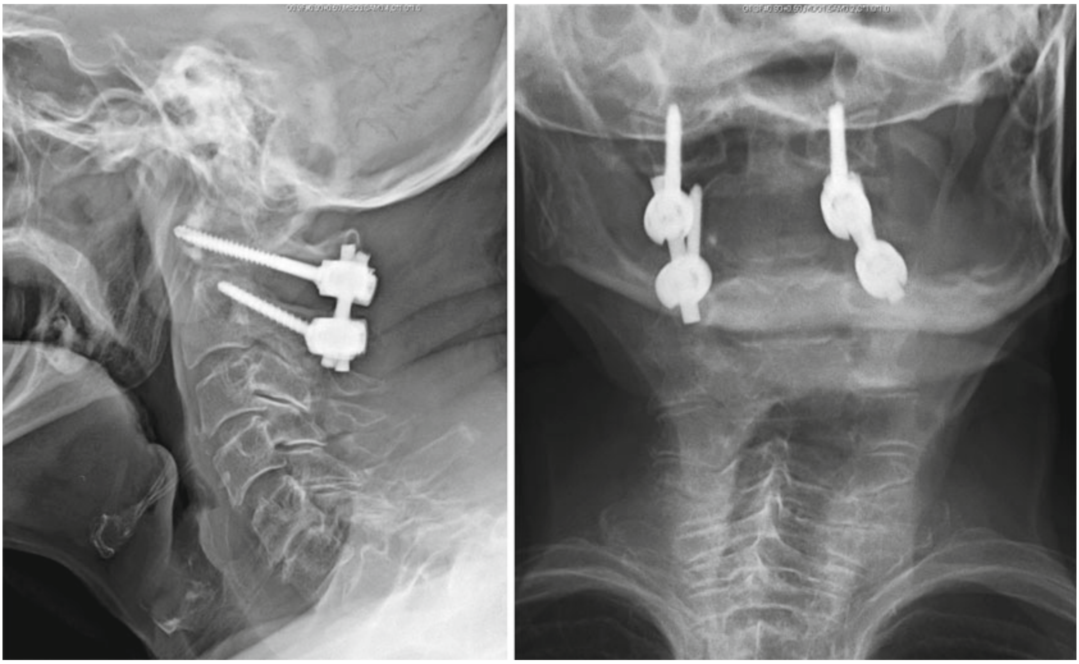


Fig. 25.2 Anterior-posterior and lateral x-rays of the cervical spine status-post C1 lateral mass and C2 pars screw instrumentation

and the axis [3]. Fractures of C2 represent about 20 % of cervical fractures [4]. Three distinct injury patterns that affect C2 include odontoid fractures, hangman fractures (traumatic spondylolisthesis), and fractures of the body of C2.

The upper cervical articulations are stabilized by ligaments both internal (intrinsic) and external (extrinsic) to the spinal canal [5]. The intrinsic ligaments confer the most stability [6, 7]. Three layers of ligaments exist anterior to the dura from ventral to dorsal: the odontoid ligaments, the cruciate ligament, and the tectorial membrane. The odontoid ligaments include the apical and alar ligaments. The paired alar ligaments that run from the odontoid process to the occipital condyles of the skull are the principal restraints for axial rotation and to a lesser extent lateral bending [3]. Incompetence of the alar ligaments results in up to a 30 % increase in axial rotation across Occiput–C1 and C1–2 [8]. The apical ligament runs vertically between the tip of the odontoid and the foramen magnum. The cruciate ligament lies dorsal to the odontoid ligaments. The most important component of this ligament is the transverse ligament. The vertical bands of the cruciate ligament connect the foramen magnum to the posterior body of axis. Overlying these ligaments is the tectorial membrane that runs from the anterior rim of the foramen magnum down along the posterior aspect of the vertebral bodies, continuing as the posterior longitudinal ligament [8, 9]. The tectorial membrane resists hyperextension [6]. The extrinsic ligaments include the ligamentum nuchae, which extends from the occipital protuberance to the posterior arch of C1 and the cervical spinous processes. Other extrinsic ligaments include the facet joint capsules and the anterior and posterior atlanto-occipital membrane [9, 10].

25.3 Injury Incidence and Mechanism

25.3.1 Occipital Condyle Fracture

Occipital condyle fractures are fractures of the skull base near the articulation with C1. The condyles support the weight of the skull on top of the articulation with the spine. They also serve as the superior attachments for the alar ligaments that run down to the odontoid, acting as the limits to rotation and lateral bending of the skull [11]. Occipital condyle fractures are usually caused by a compression injury, most commonly motor vehicle accident (60–66 %), fall (11–20 %), motorcycle accident (9–11 %), and bicycle accident (4–7 %) [11, 12]. Occipital condyle fractures were originally thought to be a very rare injury. Anderson and Montesano's classifications were made from six original cases and 20 found in the literature [13]. In the past, most of these cases were usually identified postmortem. This is a reflection of how difficult the condition is to recognize on plain film and the usual good outcomes. With the widespread use of CT, incidental findings of occipital condyle fractures have increased substantially, ranging as high as 4–19 % in upper cervical trauma [14, 15].

Occipital condyle fractures are divided into three types as outlined by Anderson and Montesano [13]. Type I injury is a comminuted fracture from impaction of the condyle from an axial loading injury. These tend to be relatively stable as the tectorial membrane and contralateral alar ligament stabilize the atlanto-occipital articulation [13]. Originally believed to be rare, type I is the most commonly recognized type on high-resolution CT scan [12, 16]. Type II is an extension of a basilar skull fracture, often from a direct blow to the skull. These fractures are typically stable. The exception occurs when a piece of the condyle is completely severed and migrates into a position that compresses the brainstem, precipitated by sudden head rotation [17]. Type III is an avulsion fracture involving the alar ligaments that run from the condyles to the odontoid process and portends the worst outcome as ligament interruption leads to instability [12]. The mechanism is either rotation or lateral bending as the alar ligaments limit the extremes of those motions. In Anderson and Montesano's original description of the type III fracture, one of their four patients subsequently died from a pontine hemorrhage [13]. A second classification system described by Tuli is based on displacement and stability and designed to guide treatment. Type 1 is nondisplaced and stable. Type 2A is displaced but stable, and type 2B is displaced and unstable [10]. Tuli proposed that type 2B requires surgical intervention, while the remainder can be managed with immobilization.

25.3.2 Occipitocervical Dissociation and Instability

Occipitocervical dissociation (atlanto-occipital dislocation) involves total separation of the articulation between the skull and upper cervical spine due to complete ligamentous disruption. They are among the most devastating injuries due to their high mortality and permanent neurologic injury rates, occurring in 5–8 % of fatal motor vehicle crashes and 19–35 % of cervical spine fatalities. Occipitocervical dissociation accounts for 8–18 % of immediate deaths after a blunt trauma [18]. The most common mechanism is a pedestrian being struck by a car [19, 20]. Mechanism of death is usually acute neurogenic shock and loss of respiratory function [3]. Of survivors, 40 % are quadriplegic or quadriparetic, 40 % hemiplegic or hemiparetic, and 20 % are neurologically intact [3]. A report exists of one patient with both occipitocervical and atlantoaxial disassociation while remaining neurologically intact [21]. Children are particularly at risk due to their shallower occipitocervical joints, looser ligaments, and larger head to vertebrae ratio [9]. In milder cases where ligamentous disruption is not complete, occipitocervical instability may result. Such injuries are classified by the direction of instability and displacement, with anterior displacement as type I, vertical displacement as type II, and posterior displacement as type III. Vertical displacement is further divided into type IIA involving displacement between the occiput and C1, and type IIB involving C1 and C2 [22].

25.3.3 Fractures of the Atlas

Fractures of atlas (C1) are relatively common, comprising 2–13 % of all cervical fractures with most studies reporting around 10 % [23] and for approximately 25 % of craniocervical injuries [24–27], and there is a 50 % incidence of associated cervical fractures [25]. Most common mechanisms include motor vehicle accidents and falls that combine to account for 80–85 % of atlas fractures [2]. Outcomes from atlas fractures are often good, as fractured components of the ring tend to expand, creating more room for the spinal cord. A variety of mechanisms of injury create several possible fractures of the atlas. A symmetrical force placed along the axis of the spine can result in multiple fractures of the ring of the atlas, termed a burst fracture (also known as a Jefferson fracture). These axial compression forces often are associated with flexion, extension, and lateral bending forces [2]. An asymmetrical force placed along the axis of the spine may result in fracture of a lateral mass. Fracture of just the posterior ring may result from a hyperextension injury, as the posterior ring of the atlas provides a bony limitation to extension [9]. In burst fractures, the distance between the lateral masses is increased as the ring is fractured and expands. This may result in rupture of the transverse ligament, either in the midsubstance or as an avulsion from the medial tubercles of the lateral masses. In contrast to transverse ligament rupture from hyperflexion injuries, the alar ligaments are spared, as the distance from the odontoid and occipital condyles is unchanged. The Jefferson classification, modified by Gehweiler, is comprised of five types [28]. Type I involves the posterior arch only. Type II involves the anterior arch only. Type III involves bilateral posterior arch fractures with a unilateral/bilateral anterior arch fracture. Type IV is a lateral mass fracture. Type V is a transverse fracture of the anterior arch. The classic Jefferson fracture is a type III fracture with lateral displacement of the lateral masses. The most common atlas fracture is type I followed by type III. Atlas fractures frequently occur with other cervical spine fractures. The most common combination is atlas and axis fractures (odontoid) [29].

25.3.4 Rupture of the Transverse Ligament

The transverse ligament is critical in atlantoaxial stability. C1 fracture stability is often defined by the integrity of the transverse ligament. Disruption of the transverse ligament can result from C1 burst fractures (as described above) as well as odontoid process separation from the posterior surface of the anterior arch of C1. The primary mechanism by which the latter occurs is a flexion injury that often also results in rupture of the alar ligaments that span the odontoid to the occipital condyles. Because the normal function of the transverse ligament is to hold the odontoid tight against the posterior wall of the anterior arch of the atlas, disruption of this ligament widens the atlantodens interval (ADI). Ligament rupture is classified as midsubstance (type I) ruptures and avulsion (type II) ruptures from the insertion on the lateral mass [30]. Rupture of the alar ligaments alone can result from an extreme rotational stress, because the alar ligaments resist extreme rotational motion. This allows for greater than normal rotation of the C1 across the C2 to the point where the lateral mass of the C1 rotates past the ipsilateral mass of the C2 below it. At this point, the lateral mass of C1 may lock behind the lateral mass of the C2, causing patients to present with a fixed head rotation and pain. This is primarily seen in children and especially in patients with atlantoaxial laxity such as Down's syndrome or Marfan's syndrome [31].

25.3.5 Odontoid Fractures

The odontoid is prone to fracture, as it is a structure that protrudes from the body of C2 and bears translational stresses of the head upon the spine. The mechanism is often a hyperextension- or hyperflexion-type injury [32]. Odontoid fractures are the most common C2 fracture and comprise about 8–18 % of all cervical fractures, with neurological deficits in 10–20 % of cases [33]. They comprise up to 75 % of childhood cervical spine fractures due to the large head to body ratio amplifying stresses on the odontoid [26].It is also the most common cervical spine fracture in the elderly. Odontoid fractures are classified by location of fracture as proposed by Anderson and D'Alonzo. Type I is a fracture of the superior tip, above the transverse ligament, and may be due to alar ligament avulsion from occipital distraction

[34]. Type I fractures are the least common odontoid fracture and are generally stable [35]. Type II odontoid fractures occur at the base of the odontoid and are the most common. Type II fractures have unpredictable healing and a high risk of nonunion especially when there is greater than 6 mm of translation, failed reduction, age over 50, or angulation greater than 10°. A mobile odontoid nonunion can lead to late-onset myelopathy due to the resulting upper cervical instability [36, 37]. Type II fractures have been further divided into A,B, and C subcategories as proposed by Grauer et al. [32]. Type IIA is a fracture with less than 1 mm of displacement. Type IIB is displaced transversely more than 1 mm or fractured anterior-superior to posterior-inferior. Type IIC is comminuted or fractured anterior-inferior to posterior-superior [32]. Type III fractures are within the body of the axis. These are more stable than type II fractures with better rates of union.

25.3.6 Traumatic Spondylolisthesis of the Axis

Traumatic spondylolisthesis of C2 involves fractures through the pars interarticularis. Often termed the "hangman's fracture," the pattern of fracture is similar to that of a judicial hanging but the mechanism and outcomes are different. The mechanism for a judicial hanging involves sudden hyperextension and distraction. The mechanism for traumatic spondylolisthesis is generally hyperextension and hyperflexion forces typically associated with high-speed motor vehicle accidents or falls from a height; they comprise up to 15 % of cervical fractures [34]. As the mechanism is usually different from that of judicial hanging, spinal cord injury is rare (approximately 6.5 %). The classification system described by Effendi et al. in 1981 and later elaborated by Levine and Edwards is based on fracture morphology and the mechanism of injury [38]. Type I fractures occur through the isthmus and has a fracture line that is vertical with no angulation and minimal (<3 mm) translation. The injury mechanism of type I fractures is hyperextension and axial loading. The articulation between C2 and C3 is intact and the ligaments are intact. A type IA, or atypical hangman's, occurs when the fracture lines of each isthmus are not parallel; the mechanism is hyperextension and lateral bending. In type IA fractures, the fracture line may run obliquely into the vertebral body and may involve the foramen of the vertebral artery. Type 2 fractures involve significant angulation and translation of greater than 3 mm. As with type I fractures, the fracture lines of type II fractures are predominately vertical. The injury mechanism of a type II fracture is a combination of hyperextension and axial loading forces coupled with a flexion force. Type 2A fractures involve minimal translation but significant angulation. The injury mechanism of a type II fracture is a flexion-distraction force; the fracture line is oblique. Type 2A fractures are more unstable than type 2 and have a propensity to displace into greater angulation with traction. A type III fracture represents a type I fracture combined with bilateral dislocation of the C2–C3 facet joints or a unilateral facet dislocation coupled with a contralateral isthmus fracture.

25.4 Clinical Manifestations

Patients who sustain upper cervical spine fractures have a high incidence of concomitant head trauma, and altered mental status may complicate the history and physical examination. Consequently, all patients with head injury should be assumed to have a cervical spine injury and undergo appropriate radiographic examination. Advanced trauma life support procedures should be performed first to maintain airway, breathing, and circulation. Careful examination of the spine with inspection, palpation, and neurological testing should then be performed while the head, neck, and spine are immobilized in neutral alignment. Neurologic examination should include cranial nerve, peripheral motor, sensory, and reflex testing. Patients with upper cervical spine fractures frequently present with neck pain, spasms, and limited neck motion. Difficulty swallowing may result from retropharyngeal swelling. Cranial nerves VI, VII, IX, XI, and XII in particular may be affected; however, some patients may have no neurological defects on initial exam [9]. Vertebral artery injuries may manifest as transient loss of consciousness, diplopia, and/or posterior fossa ischemia. Many of the described fractures and injuries share similar clinical findings of neck pain, loss of range of motion, and vascular or neural compromise [9].

25.4.1 Occipital Condyle Fractures

There is a wide range of severity and injury patterns with occipital condyle fractures, with a corresponding range of clinical presentations. Some patients can present with only pain and tenderness, whereas others may demonstrate significant neurologic compromise. As with many upper cervical spine injuries, a concomitant head injury may be present. Traumatic brain injury is detected in up to half of patients found to have occipital condyle fractures, and other cervical spine injuries are found in 40 % [16]. The occipital condyles are in close proximity to the brain stem, IX, X, XI, XII cranial nerves, and jugular vein. Damage to the lower cranial nerves, especially CN XII (hypoglossal), can occur in up to 30 % of patients with occipital condyle fractures [39], and the hypoglossal nerve is the most frequently involved lower cranial nerve due to its proximity to the occipital condyle [17]. Unilateral paralysis of CN IX–XII is termed Collet-Sicard syndrome. Condyle fractures may compromise the posterior-inferior cerebellar artery leading to ischemia of the lateral medulla and Wallenberg syndrome [40]. The clinician must maintain a high index of suspicion for this injury, as the majority of patients with occipital condyle fractures have nonspecific findings [12].

25.4.2 Occipitocervical Dissociation and Instability

Patients with occipitocervical dissociation have a high mortality rate. Prompt recognition of dissociation and/or instability and immobilization is essential. Patients may present with neurological abnormalities, including lower cranial nerve paresis (particularly CN VI, X, and XII), hemiparesis, quadriparesis, respiratory dysfunction (including apnea), and complete high cervical cord motor deficits.

25.4.3 Atlas and Axis Fractures

Often the presenting symptoms are neck pain, spasms of cervical neck muscles, and limited neck motion. Compression of the C2 nerve is possible, as are compression of CN IX-XII in the Collet-Sicard pattern. Collet-Sicard syndrome presents as paralysis of the tongue on the ipsilateral side, ipsilateral vocal cord and gag reflex dysfunction, and ipsilateral weakness of the sternocleidomastoid and trapezius muscle; the clinical manifestation is hoarseness, difficulty swallowing, and impaired speech. The vertebral artery may be at risk as well leading to headache, visual abnormalities, and nausea. Retropharyngeal swelling can lead to dysphagia [2]. Neurologic deficits rarely accompany isolated C1 or C2 fractures, given the capacious nature of the spinal canal at this level.

25.5 Diagnostic Imaging

The standard cervical spine radiographic evaluation includes the lateral, anterior-posterior, and open-mouth odontoid views. The open-mouth odontoid view allows visualization of C1, its lateral masses and the odontoid. While up to 85 % of cervical fractures can be visualized on the lateral view, anterior-posterior and open-mouth views should routinely be obtained [9, 41].

On lateral X-ray, the clivus should point to the tip of the odontoid with the tip of the clivus (basion) within 5 mm of the odontoid. Anterior soft tissue swelling greater than 5 mm in the adult (>7 mm in child) at the C3 level raises suspicion of a C1 anterior arch fracture [9, 25]. A distance of more than 2 mm between the occiput and C1 raises suspicion of trauma [9]. A retropharyngeal hematoma should raise suspicion for an upper cervical injury. Many fractures are best visualized and characterized by CT. MRI allows visualization of ligamentous disruption and is useful in identifying injury to the transverse ligament. Flexion-extension lateral radiographs may allow detection of instability in the upper cervical spine, but the role of flexion-extension radiographs in the acutely injured patient is controversial due to the inherent splinting that is present with muscle spasm and the risk of a neurologic catastrophe in the setting of acute ligamentous instability.

25.5.1 Occipital Condyle Fracture

Occipital condyle fractures are poorly diagnosed by radiographs. As CT is the imaging modality of choice. All patients with a suspected occipital condyle fracture should undergo this examination [15, 42, 43]. In a study of over 50 patients with documented occipital condyle fracture by axial CT with 2D reconstruction, none of these patients were initially recognized by lateral cervical plain film [42]. The authors also recommended the use of very thin 1.2 mm sections to achieve the resolution needed to identify very small type I and type II fractures. Possible disruption of the alar ligaments, and thus fracture instability, can be determined by MRI.

25.5.2 Occipitocervical Dislocation and Instability

A number of parameters have been described on plain film to detect occipitocervical dissociation, including Wackenheim's clival line, Power's ratio, X-line, and Wholey method (dens-basion line) [44]. However, the sensitivity of these imaging parameters is generally in the 60–70 % range, making them of limited utility in clinical practice for ruling out an injury [45]. A more accurate plain film indicator of injury utilizes the basion-axial interval and basion-dens interval as described by Harris et al. [46]. This interval is obtained by measuring from the tip of the clivus (basion) to the tip of the odontoid (dens) and also from the basion to the extended line from the posterior cortex of the axis. It is also nicknamed the rule of 12 as both the basion-axial and basion-dens intervals should be less than 12 mm. Reported sensitivity and specificity are 96 and 98 % [45]. Neurologic abnormalities in the setting of normal radiographs should prompt additional imaging with CT or MRI. The presence of prevertebral soft tissue swelling on plain film and/or subarachnoid hemorrhage at the craniovertebral junction on CT/MRI should prompt consideration of the diagnosis of occipitocervical dissociation or instability.

25.5.3 Atlas Fractures and Rupture of the Transverse Ligament

On plain film, lateral, open-mouth odontoid views should be obtained. Any displacement of the C1 lateral mass over the C2 lateral mass suggests burst fracture. The transverse ligament runs between the two lateral masses and maintains the odontoid process against the anterior arch of C1. Normally, the atlantodens interval (ADI) is less than 3 mm in an adult and less than 5 mm in a child. An ADI greater than 3 mm in an adult implies an incompetent transverse ligament [47]. An ADI greater than 7 mm implies disruption of the alar ligaments and tectorial membrane. Normal C1 lateral mass overhang on C2 on open-mouth odontoid view is <6.9 mm. Total lateral overhang of the C1 lateral masses on C2 greater than 7.0 mm after a burst fracture implies transverse ligament disruption (rule of Spence) [48]. Advanced imaging studies (CT and MRI) should be considered to further define fracture patterns and to better characterize ligamentous injury.

25.6 Nonsurgical Treatment

25.6.1 Occipital Condyle Fractures

Unilateral occipital condyle fractures can often be treated nonsurgically. Some authors have described no immobilization for stable type I fractures while others suggest a collar for 6–8 weeks [10, 49]. Stable type II fractures can be treated in a hard collar; stability is preserved due to the absence of injury to the alar ligaments or tectorial membrane. Displaced type II fractures should be placed in a halo vest for 8–12 weeks [9]. Type III occipital condyle fractures represent an avulsion fracture that involves the alar ligaments. With avulsion of the occipital condyle, the contralateral alar ligament is stressed, making the injury potentially unstable. Type III occipital condyle fractures necessitate careful consideration of the upper cervical spine ligaments; if the contralateral alar ligament and tectorial membrane are uninjured, the fracture may be amenable to management with a hard collar or halo-vest immobilization. Union rates overall for all types are as high as 88 % [16, 50]. Occipital condyle fractures resulting in an unstable craniocervical junction likely necessitate occipital–C1–C2 posterior arthrodesis. In general, significantly displaced fracture fragments and/or rupture of the ligaments are believed to portend instability due to the presumed low union rate of a significantly displaced fracture. Note that prior to the advent of advanced imaging studies, many fractures were likely undiagnosed, and there is a consequent paucity of higher-level clinical evidence regarding the outcomes of management of this fracture.

25.6.2 Occipitocervical Dissociation and Instability

In type I occipitocervical instability, a reduction can be accomplished by placing a bolster behind the thorax to bring the head more posterior. In type III posterior displacement, the bolster can be placed behind the occiput to bring the head more anterior. Following reduction, a halo vest should be applied. In type II vertical displacement, care must be made to avoid traction due to the risk of further disassociation and neurologic catastrophe. Reduction can be achieved with gentle downward force and halo placement. These injuries require subsequent arthrodesis for stabilization [9].

25.6.3 Atlas Fractures

Most isolated C1 fractures can be treated nonoperatively. In posterior arch fractures of the C1, the injury is usually stable and can be managed with a cervical orthosis. Anterior arch fractures can also be treated with a rigid cervical collar. In Jefferson burst fractures, the amount of displacement dictates treatment. Displacement less than 5 mm can be treated with halo vest for 3 months with good results [9]. Displacement greater than 5 mm may benefit from skeletal traction followed by halo placement. In most patients with isolated C1 fractures, the majority heal well without nonunion, instability, or neurological deficits [2]. Late instability must be ruled out by dynamic imaging (e.g., flexion-extension X-rays). Instability requires fusion to prevent neurological injury.

25.6.4 Rupture of the Transverse Ligament

If the atlantodens interval is less than 5 mm and the patient is neurologically intact, a nonoperative treatment can be attempted with a cervical collar or halo vest; this may be effective in type II avulsion ruptures, which may be managed with external immobilization for 3 months and follow-up imaging studies. Up to 74 % of type II injuries heal spontaneously with immobilization [30]. Type I injuries heal poorly by comparison and often require surgery.

25.6.5 Odontoid Fractures

For most type I and III fractures, management is generally nonoperative. Nondisplaced type I fractures can be managed with a cervical collar for 6–8 weeks, but in the setting of a type I fracture, upper cervical instability and occipitocervical dislocation must be excluded. The management of type II odontoid fractures is controversial – particularly in the elderly, there is a high rate of nonunion, irrespective of treatment type. Nonoperative treatment can be attempted with minimally displaced type II (less than 5 mm of displacement and/or 10° of angulation). However, even with minimal displacement, nonoperative management of type II odontoid fractures is associated with a relatively high nonunion rate, particularly in the elderly patient [51, 52]. Nonunion of a type II fracture is a significant clinical concern due to the risk of late-onset myelopathy [53]. Type II fractures with displacement greater than 5 mm or 10° of angulation are generally managed surgically due to the high rate of nonunion [54]. Type III fractures occur through the body of C2 which has a good blood supply and overall good potential for healing. Minimally displaced type III fractures generally heal, but type III fractures with anterior displacement may be unstable and are at risk of displacement with closed management.

25.6.6 Traumatic Spondylolisthesis of the Axis

For type I fractures through both pars and less than 3 mm displacement and no angulation, immobilization with collar or halo vest is sufficient. For type II fractures with more than 3 mm displacement, angulation, and C3 anterior compression, reduction with gentle traction may be attempted by placing a bolster behind the shoulders. Rigid immobilization with a halo vest or cervical orthosis should be performed for 8–10 weeks. For type IIA fractures, reduction with slight extension may be attempted with subsequent halo-vest immobilization. Type IIA fractures should not be placed in traction due to the risk of axial displacement and neurologic catastrophe. Type III injuries are often not amenable to closed reduction and necessitate surgical treatment. In studies of nonsurgical treatment, 90 % of type I fractures achieved stable union compared to 70 % of type II fractures [55].

25.7 Surgical Treatment

The options for surgical treatment include direct fixation of fractures or arthrodesis of the involved levels. Occiput–C1 fusion will reduce neck flexion-extension by approximately 50 %, while C1–C2 fusion will reduce cervical rotation by approximately 50 %. Traditionally, C1–C2 fusion was performed with posterior wiring techniques such as the Gallie or Brooks constructs, utilizing bone graft placed between the posterior arches of C1 and C2 and compression with sublaminar wires. Contemporary constructs utilize occipital plates, C1 lateral mass, and C2 pars, pedicle, or translaminar screw constructs. The Magerl screw is a C1–C2 transarticular screw that is placed through the posterior aspect of the inferior facet of C2, through the C1–C2 articulation into the lateral mass of C1, thereby achieving a C1–C2 fusion. In part due to the increased risk to the vertebral artery with placement of transarticular C1–C2 screws, C1 lateral mass-C2 pars instrumentation was popularized by Harms and Melcher [56]. A C1 lateral mass screw is placed posteriorly through the lateral mass of C1, generally utilizing fluoroscopic or surgical navigation image guidance. C2 screws can be placed through the pars, pedicle, or lamina of C2.

25.7.1 Occipital Condyle Fracture

Surgery is generally not required for most occipital condyle fractures, as the majority will heal with either collar or halo immobilization. However, cases of potential craniocervical instability or significant fracture displacement as identified by CT scan require occipitocervical fusion to C2; these cases usually correspond to Tuli type IIB injuries [57].

25.7.2 Occipitocervical Dissociation and Instability

Occipitocervical dislocation often requires posterior occipitocervical fusion to C2 [58]. Severe cases involving both occipitocervical and atlantoaxial dissociation can be fixed and fused using an occipital plate with C1 lateral mass, C2 pars interarticularis, and C3 lateral mass screws with bone graft [21].

25.7.3 Atlas Fractures

Burst fractures with displacement over 6.9 mm are associated with disrupted transverse ligament.

Midsubstance ruptures of the transverse ligament are particularly difficult to heal and require C1–C2 fusion after reduction [59]. In cases of progressive displacement, C1 nonunion, or instability after nonsurgical immobilization, C1–C2 or occiput–C2 arthrodesis can be performed [2, 9]. In cases of combined C1–C2 fractures, the C2 fracture and integrity of the transverse ligament generally dictates treatment. External immobilization is generally recommended unless instability is demonstrated on upright and supine radiographs in an orthosis. Integrity of the ligamentous complex at the craniovertebral junction is a determining factor in considering if an occipitocervical fusion is necessary.

25.7.4 Rupture of the Transverse Ligament

Type I ruptures through the midsubstance have poor healing rates [30]. They can be managed with a C1–C2 arthrodesis construct such as C1–C2 transarticular screws or C1 lateral mass with C2 pars/pedicle screws. Type II injuries can be treated with rigid immobilization and close follow-up.

25.7.5 Odontoid Fractures

Surgical management of type II fractures is controversial. Patient-specific considerations include the degree of fracture displacement, angulation, patient age, and medical comorbidities. With direct anterior odontoid screw fixation across the fracture, healing rates have been reported as high as 86–100 %. Purported benefits of screw fixation include preservation of rotation of the atlantoaxial articulation. However, osteoporosis is a relative contraindication to anterior screw fixation [60] due to concerns about screw pullout, and significant comminution precludes direct osteosynthesis via an anterior approach. Additionally, one must first achieve an appropriate fracture reduction prior to anterior screw fixation. Rupture of the transverse ligament is a contraindication to anterior screw fixation. Outcomes with anterior screw fixation in chronic fractures are worse, and therefore, the technique is not recommended in the setting of a chronic fracture. Posterior approaches to odontoid fractures involve arthrodesis of C1–C2, via either transarticular C1–C2 fixation or C1 lateral mass-C2 pars/pedicle/laminar fixation [61].

25.7.6 Traumatic Spondylolisthesis of the Axis

A fracture with more than 11 % angulation at the C2–C3 interspace is considered by some to be a possible indication for surgical fixation [29]. The vast majority of these angulated fractures however are treated adequately nonoperatively. Type II fractures that fail closed reduction can be managed with cervical collar or halo-vest immobilization. Open reduction internal fixation and subsequent collar or halo-vest immobilization may also be a consideration but is rarely indicated. Type III fractures warrant surgical treatment to perform an open reduction of the facet dislocation at C2–C3. Fixation of the hangman fracture component can be achieved using a lag screw fixation technique; however, many treat the hangman component nonoperatively [62, 63]. Late instability may be treated with C2–C3 anterior diskectomy and fusion or a C1–C3 posterior fusion.

25.8 Postoperative Complications

In addition to general surgical complications such as infection, common problems include postoperative residual neck pain, limitation of range of motion, and persistent neurological compromise [2]. Patients may feel (C2) greater occipital nerve irritation. Cranial nerves VI, IX, X, XI, and XII dysfunction may be persistent or only recover slightly after reduction [64]. Injury to the vertebral artery can result in a perfusion abnormality of the posterior-inferior cerebellar artery and resultant lateral medullary (Wallenberg) syndrome. Clinical manifestations of Wallenberg's syndrome include CN V, IX, X, and XI dysfunction, i.e., Horner's syndrome, contralateral pain/temp loss, and cerebellar ataxia. The use of C1–C2 transarticular screws has a risk of vertebral artery injury in 4.1 % of patients, stroke in 0.2 %, and overall mortality in 0.1 % [65]. This construct is contraindicated in aberrant coursing vertebral artery or fixed C1–C2 subluxation [66, 67]. Elderly patients have marked complications with halo-vest immobilization, with a 66 % complication rate and 42 % mortality rate [68]. Relatively good results have been reported by some institutions with halo-vest immobilization, although this was in a cohort of patients with a mean age of 41 years [69]. The management of type II odontoid fractures in the elderly is particularly problematic, with significant perioperative medical morbidity irrespective of management type [70].

References

1. White AA, Panjabi MM (1990) Kinematics of the spine. Clinical biomechanics of the spine, 2nd edn. JB Lippincott, Philadelphia, pp 85–125
2. Kakarla UK, Chang SW, Theodore N, Sonntag VK (2010) Atlas fractures. Neurosurgery 66:60–67
3. Fisher CG, Sun JC, Dvorak M (2001) Recognition and management of atlanto-occipital dislocation: improving survival from an often fatal condition. Can J Surg 44:412–420
4. Pryputniewicz DM, Hadley MN (2010) Axis fractures. Neurosurgery 66:68–82
5. Standring S (2005) Gray's anatomy: the anatomical basis of clinical practice, 39th edn. Elsevier Churchill Livingstone, London
6. Werne S (1957) Studies in spontaneous atlas dislocation. Acta Orthop Scand 23:1–150
7. White AA, Panjabi MM (1978) The clinical biomechanics of the occipitoatlantoaxial complex. Orthop Clin North Am 9:867–878
8. Dvorak J, Schneider E, Saldinger P, Rahn B (1988) Biomechanics of the craniocervical region: the alar and transverse ligaments. J Orthop Res 6:452–461
9. Jackson RS, Banit DM, Rhyne AL, Darden BV (2002) Upper cervical spine injuries. J Am Acad Orthop Surg 10:271–280
10. Tuli S, Tator CH, Fehlings MG, Mackay M (1997) Occipital condyle fractures. Neurosurgery 41: 368–376
11. Aulino JM, Tutt LK, Kaye JJ, Smith PW, Morris JA (2005) Occipital condyle fractures: clinical presentation and imaging findings in 76 patients. Emerg Radiol 11:342–347
12. Bloom AI, Neeman Z, Slasky BS, Floman Y, Milgrom M, Rivkind A, Bar-Ziv J (1997) Fracture of the occipital condyles and associated craniocervical ligament injury: incidence, CT imaging and implications. Clin Radiol 52:198–202
13. Anderson PA, Montesano PX (1988) Morphology and treatment of occipital condyle fractures. Spine (Phila Pa 1976) 13:731–736
14. Leventhal MR, Boydston WR, Sebes JI, Pinstein ML, Watridge CB, Lowrey R (1992) The diagnosis and treatment of fractures of the occipital condyle. Orthopedics 15:944–947
15. Link TM, Schuierer G, Hufendiek A, Horch C, Peters PE (1995) Substantial head trauma: value of routine CT examination of the cervicocranium. Radiology 196:741–745
16. Malham GM, Ackland HM, Jones R, Williamson OD, Varma DK (2009) Occipital condyle fractures: incidence and clinical follow-up at a level 1 trauma centre. Emerg Radiol 16:291–297
17. Caroli E, Rocchi G, Orlando ER, Delfini R (2005) Occipital condyle fractures: report of five cases and literature review. Eur Spine J 14:487–492
18. Lador R, Ben-Galim PJ, Weiner BK, Hipp JA (2010) The association of occipitocervical dissociation and death as a result of blunt trauma. Spine J 10:1128–1132
19. Bucholz RW, Burkhead WZ (1979) The pathological anatomy of fatal atlanto-occipital dislocations. J Bone Joint Surg Am 61:248–250
20. Davis D, Bohlman H, Walker AE, Fisher R, Robinson R (1971) The pathological findings in fatal craniospinal injuries. J Neurosurg 34:603–613
21. Bisson E, Schiffern A, Daubs MD, Brodke DS, Patel AA (2010) Combined occipital-cervical and atlantoaxial disassociation without neurologic injury. Spine (Phila Pa 1976) 35:E316–E321
22. Traynelis VC, Marano GD, Dunker RO, Kaufman HH (1986) Traumatic atlanto-occipital dislocation. Case report. J Neurosurg 65:863–870
23. Levine AM, Edwards CC (1986) Treatment of injuries in the C1-C2 complex. Orthop Clin North Am 17: 31–44
24. Hadley MN, Dickman CA, Browner CM, Sonntag VK (1988) Acute traumatic atlas fractures: management and long term outcome. Neurosurgery 23: 31–35
25. Levine AM, Edwards CC (1991) Fractures of the atlas. J Bone Joint Surg Am 73:680–691
26. Sherk HH (1978) Fractures of the atlas and odontoid process. Orthop Clin North Am 9:973–984
27. Landells CD, Van Peteghem PK (1988) Fractures of the atlas: classification, treatment and morbidity. Spine (Phila Pa 1976) 13:450–452
28. Gehweiler J, Duff D, Salutario M, Miller M, Clark M (1976) Fractures of the atlas vertebra. Skeletal Radiol 23:97–102
29. Hadley MN (2002) Isolated fractures of the atlas in adults. Neurosurgery 50:S120–S124
30. Dickman CA, Greene KA, Sonntag VK (1996) Injuries involving the transverse atlantal ligament: classification and treatment guidelines based upon experience with 39 injuries. Neurosurgery 38: 44–50

31. Crook TB, Eynon CA (2005) Traumatic atlantoaxial rotatory subluxation. Emerg Med J 22: 671–672
32. Grauer JN, Shafi B, Hilibrand AS, Harrop JS, Kwon BK, Beiner JM, Albert TJ, Fehlings MG, Vaccaro AR (2005) Proposal of a modified, treatment-oriented classification of odontoid fractures. Spine J 5: 123–129
33. Clark CR, White AA (1985) Fractures of the dens. A multicenter study. J Bone Joint Surg Am 67:1340–1348
34. Hadley MN, Dickman CA, Browner CM, Sonntag VK (1989) Acute axis fractures: a review of 229 cases. J Neurosurg 71:642–647
35. Anderson LD, D'Alonzo RT (1974) Fractures of the odontoid process of the axis. J Bone Joint Surg Am 56:1663–1674
36. Hart R, Saterbak A, Rapp T, Clark C (2000) Nonoperative management of dens fracture nonunion in elderly patients without myelopathy. Spine (Phila Pa 1976) 25:1339–1343
37. Kirankumar MV, Behari S, Salunke P, Banerji D, Chhabra DK, Jain VK (2005) Surgical management of remote, isolated type II odontoid fractures with atlantoaxial dislocation causing cervical compressive myelopathy. Neurosurgery 56:1004–1012
38. Levine AM, Edwards CC (1981) Fractures of the atlas. J Bone Joint Surg Am 73:680–691
39. Erol FS, Topsakal C, Kaplan M, Yildirim H, Ozveren MF (2007) Collet-Sicard syndrome associated with occipital condyle fracture and epidural hematoma. Yonsei Med J 48:120–123
40. Miyazaki C, Katsume M, Yamazaki T, Aoki K, Kuroki T, Takasu N (2000) Unusual occipital condyle fracture with multiple nerve palsies and Wallenberg syndrome. Clin Neurol Neurosurg 102:255–258
41. MacDonald RL, Schwartz ML, Mirich D, Sharkey PW, Nelson WR (1990) Diagnosis of cervical spine injury in motor vehicle crash victims: how many X-rays are enough? J Trauma 30:392–397
42. Bloom AI, Neeman Z, Floman Y, Gomori J, Bar-Ziv J (1996) Occipital condyle fracture and ligament injury: imaging by CT. Pediatr Radiol 26:786–790
43. Noble ER, Smoker WR (1996) The forgotten condyle: the appearance, morphology, and classification of occipital condyle fractures. AJNR Am J Neuroradiol 17:507–513
44. Bono CM, Vaccaro AR, Fehlings M, Fisher C, Dvorak M, Ludwig S, Harrop J (2007) Spine trauma study group. Measurement techniques for upper cervical spine injuries: consensus statement of the Spine Trauma Study Group. Spine (Phila Pa 1976) 32:593–600
45. Dziurzynski K, Anderson PA, Bean DB, Choi J, Leverson GE, Marin RL, Resnick DK (2005) A blinded assessment of radiographic criteria for atlanto-occipital dislocation. Spine (Phila Pa 1976) 30:1427–1432
46. Harris JH, Carson GC, Wagner LK, Kerr N (1994) Radiologic diagnosis of traumatic occipitovertebral dissociation: 2. comparison of three methods of detecting occipitovertebral relationships on lateral radiographs of supine subjects. AJR Am J Roentgenol 162:887–892
47. Oda T, Panjabi MM, Crisco JJ, Oxland TR, Katz L, Nolte LP (1991) Experimental study of atlas injuries. II. Relevance to clinical diagnosis and treatment. Spine (Phila Pa 1976) 16:S466–S473
48. Spence KF, Decker S, Sell KW (1970) Bursting atlantal fracture associated with rupture of the transverse ligament. J Bone Joint Surg Am 52: 543–549
49. Karam YR, Traynelis VC (2010) Occipital condyle fractures. Neurosurgery 66:56–59
50. Anderson PA (1998) The injuries to the occipital cervical articulation. In: Anderson PA, Clark CR, Dvorak J et al (eds) Cervical spine, 3rd edn. Lippincott-Raven, Philadelphia, pp 387–399
51. Polin RS, Szabo T, Bogaev CV, Replogle RE, Jane JA (1996) Nonoperative management of types II and III odontoid fractures: the Philadelphia collar versus the halo vest. Neurosurgery 38:450–457
52. Wang GJ, Mabie KN, Whitehill R, Stamp WG (1984) The nonsurgical management of odontoid fractures in adults. Spine (Phila Pa 1976) 9:229–230
53. Crockard HA, Heilman AE, Stevens JM (1993) Progressive myelopathy secondary to odontoid fractures: clinical, radiological, and surgical features. J Neurosurg 78:579–586
54. Seybold EA, Bayley JC (1998) Functional outcome of surgically and conservatively managed dens fractures. Spine (Phila Pa 1976) 23:1837–1845
55. Levine AM, Edwards CC (1985) The management of traumatic spondylolisthesis of the axis. J Bone Joint Surg Am 67:217–226
56. Harms J, Melcher RP (2001) Posterior C1-C2 fusion with polyaxial screw and rod fixation. Spine 26:2467–2471
57. Maserati MB, Stephens B, Zohny Z, Lee JY, Kanter AS, Spiro RM, Okonkwo DO (2009) Occipital condyle fractures: clinical decision rule and surgical management. J Neurosurg Spine 11:388–395
58. Bambakidis NC, Feiz-Erfan I, Horn EM, Gonzalez LF, Baek S, Yüksel KZ, Brantley AG, Sonntag VK, Crawford NR (2008) Biomechanical comparison of occipitoatlantal screw fixation techniques. J Neurosurg Spine 8:143–152
59. McGuire RA, Harkey HL (1995) Unstable Jefferson's fracture treated with transarticular screws. Orthopedics 18:207–209
60. Harrop JS, Przybylski GJ, Vaccaro AR et al (2000) Efficacy of anterior odontoid screw fixation in elderly patients with type II odontoid fractures. Neurosurg Focus 8:e6
61. Coyne TJ, Fehlings MG, Wallace MC, Bernstein M, Tator CH (1995) C1-C2 posterior cervical fusion: long-term evaluation of results and efficacy. Neurosurgery 37:688–692
62. Levine AM (1998) Traumatic spondylolisthesis of the axis (Hangman's fracture). In: Levine AM, Eismont FJ, Garfin SR, Zigler JE (eds) Spine trauma. WB Saunders, Philadelphia, pp 278–299

63. Verheggen R, Jansen J (1998) Hangman's fracture: arguments in favor of surgical therapy for type II and III according to Edwards and Levine. Surg Neurol 48:23–61
64. Connolly B, Turner C, DeVine J, Gerlinger T (2000) Jefferson fracture resulting in Collet-Sicard syndrome. Spine (Phila Pa 1976) 25:395–398
65. Wright NM, Lauryssen C (1998) Vertebral artery injury in C1-2 transarticular screw fixation: results of a survey of the AANS/CNS section on disorders of the spine and peripheral nerves. J Neurosurg 88:634–640
66. Abou Madawi A, Solanki G, Casey AT, Crockard HA (1997) Variation of the groove in the axis vertebra for the vertebral artery. Implications for instrumentation. J Bone Joint Surg Br 79:820–823
67. Paramore CG, Dickman CA, Sonntag VK (1996) The anatomical suitability of the C1-2 complex for transarticular screw fixation. J Neurosurg 85:221–224
68. Tashjian RZ, Majercik S, Biffl WL, Palumbo MA, Cioffi WG (2006) Halo-vest immobilization increases early morbidity and mortality in elderly odontoid fractures. J Trauma 60:199–203
69. Bransford RJ, Stevens DW, Uyeji S et al (2009) Halo vest treatment of cervical spine injuries: a success and survivorship analysis. Spine (Phila Pa 1976) 34:1561–1566
70. Smith HE, Kerr SM, Maltenfort M et al (2008) Early complications of surgical versus conservative treatment of isolated type II odontoid fractures in octogenarians: a retrospective cohort study. J Spinal Disord Tech 21:535–539

Spine Trauma: Subaxial Cervical Spine (C3–C7)

26

Kelli L. Crabtree, Paul M. Arnold, and Karen K. Anderson

26.1 Introduction

The cervical spine is the most mobile portion of the spinal column, which also makes it the most common location of traumatic spinal injury. The extent of injury can range from a minor strain of a ligament without any neurologic deficit to a fracture dislocation with ligamentous disruption causing complete cord injury. The patient's neurologic outcome is influenced by multiple factors, including the mechanism, force, and level of injury, as well as the patient's age and medical comorbidities.

26.2 Case Examples

A 38-year-old female fell down a short flight of stairs. The patient had immediate onset of lower extremity weakness. Lateral X-ray showed C6–C7 injury (Fig. 26.1a), and CT scan showed C6–C7 subluxation and spinal cord compression with kyphosis (Fig. 26.1b). MRI showed signal change in the spinal cord (Fig. 26.1c). After initial screening, trauma evaluation revealed no other injuries. The patient was taken to the operating room for open reduction and stabilization of the injury.

A 29-year-old man suffered a fall resulting in an incomplete spinal cord injury. Diagnostic work-up revealed multiple fractures: C5 burst fracture, C4 facet fracture, and C5 lamina fracture. The patient underwent C5 vertebrectomy and C4–C6 anterior fusion (Figs. 26.2a–d and 26.3a–f).

26.3 Pathophysiology

The subaxial cervical spine encompasses the region immediately inferior to the axis (C2) through the C7 vertebral body and is responsible for providing the majority of forward and lateral flexion of the neck as well as almost 50 % of cervical rotation. Thus, it is not surprising that over 75 % of all cervical spine injuries in adults occur in the subaxial region, given its inherent mobility [4, 39, 48, 63]. This percentage is slightly less in children due to their ligamentous laxity, incompletely ossified vertebral bodies, and underdeveloped neck muscles, all of which predispose them to fractures between the occiput and C2 [34].

Possible injuries to the subaxial cervical spine include fractures, subluxations, dislocations, and ligamentous tears; these injuries can be isolated

K.L. Crabtree, M.D. • K.K. Anderson, B.S.
Department of Neurosurgery, University of Kansas Medical Center, Kansas City, KS, USA
e-mail: kcrabtree@kumc.edu; kanderson3@kumc.edu

P.M. Arnold, M.D. (✉)
Department of Neurosurgery, University of Kansas Medical Center, 3901 Rainbow Blvd, Mail Stop 3021, Kansas City, KS 66160, USA
e-mail: parnold@kumc.edu

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_26, © Springer-Verlag Berlin Heidelberg 2014

or can occur in conjunction with one another. The cervical spinal cord can be damaged directly by traction forces or transection or by compression forces from bone, intervertebral disc, ligament, and/or hematoma. The cord can also sustain an indirect, yet severe, insult if its vascular supply is disrupted.

With any traumatic injury, it is critical to assess spinal stability. In the subaxial cervical spine, this is commonly done using the Holdsworth two-column theory. The anterior column includes the vertebral bodies and intervertebral discs aligned by the anterior longitudinal ligament (ALL) and the posterior longitudinal ligament (PLL). The posterior column is comprised of the spinal canal surrounded by the vertebral arch and posterior ligament complex. If only one column is disrupted, the risk of spinal cord injury is low because the other column can maintain the structural integrity of the spine. If both columns are injured, the cervical spine can move as two independent units with the potential to cause severe cord compromise.

The treatment of subaxial cervical trauma is based on multiple variables including the mechanism of injury, neurologic deficit, spinal column alignment, type of bony injury, and expected long-term stability. Allen and Ferguson published the first mechanistic classification, in which six categories of injury were described as follows: vertical compression, compressive flexion, distractive flexion, compressive extension, lateral flexion, and distractive extension. This classification was published in 1982, the pre-CT era, and is mostly descriptive [1, 10] (Fig. 26.4).

To address concerns and limitations of previous classification systems, a subcommittee of the Spine Trauma Study Group recently proposed the subaxial cervical spine injury classification (SLIC) system, classifying injuries according to three primary considerations: injury morphology, the patient's neurologic status, and the

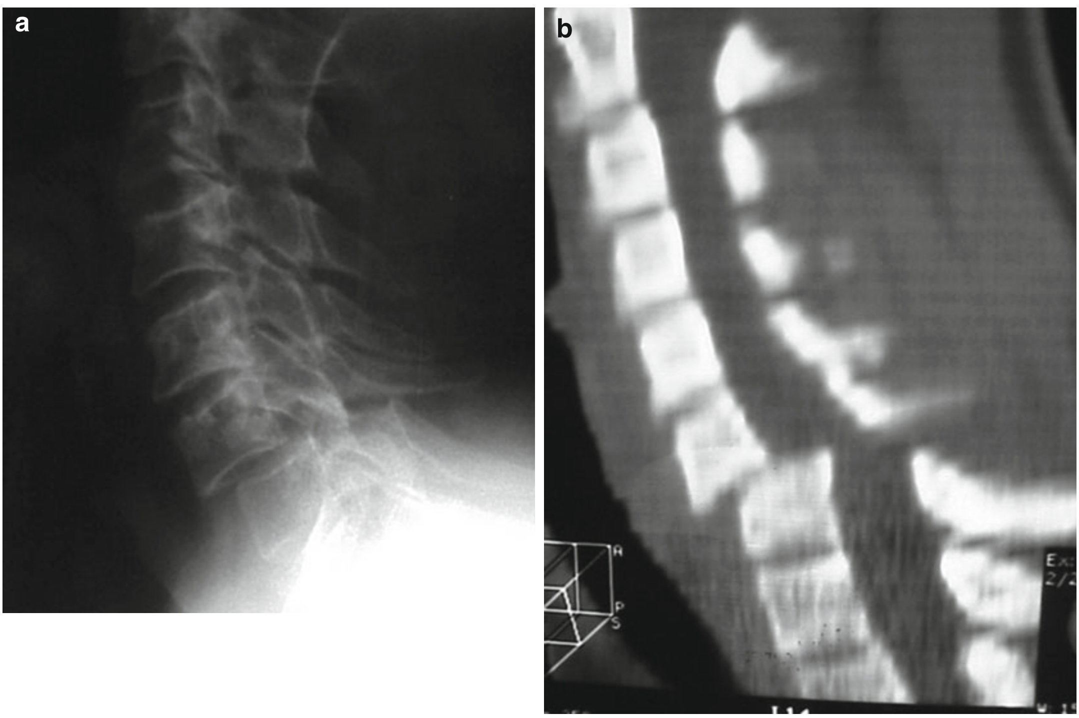


Fig. 26.1 A 38-year-old female fell down a short flight of stairs. The patient had immediate onset of lower extremity weakness. Lateral X-ray showed C6–C7 injury (**a**). CT scan showed C6–C7 subluxation and spinal cord compression with kyphosis (**b**). MRI showed signal change in the spinal cord. After initial screening, trauma evaluation revealed no other injuries. The patient was taken to the OR for open reduction and stabilization of the injury (**c**)

Fig. 26.1 (continued)

integrity of the discoligamentous complex (DLC), which includes the ALL, PLL, ligamentum flavum, facet capsule, and interspinous and supraspinous ligaments [23, 63]. This system is helpful in assessing the pattern and severity of the injury and in guiding management decisions regarding treatment and prognosis. The score obtained on the SLIC scale influences the clinical decision for surgical versus nonsurgical management [47].

26.4 Mechanisms of Injury

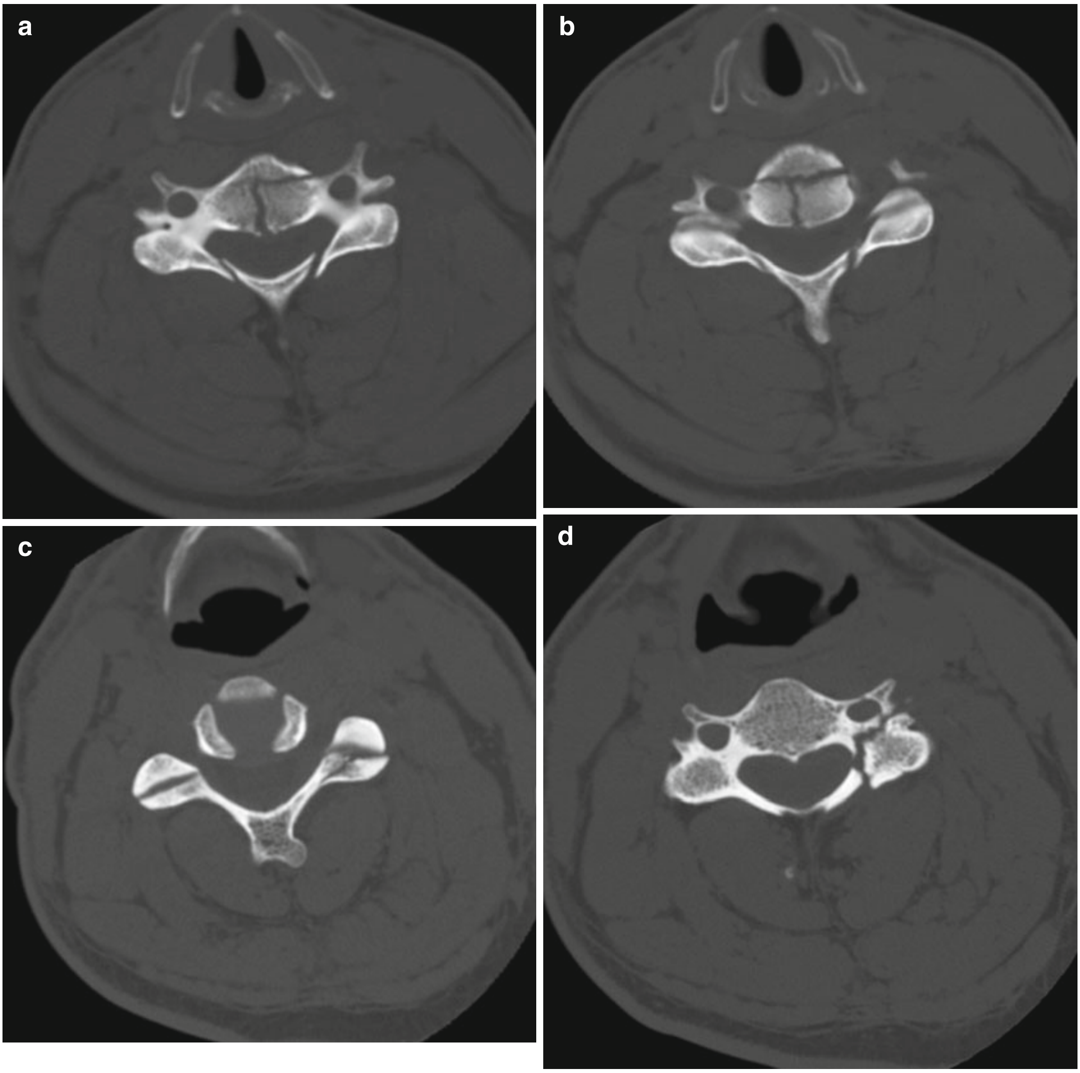
Hyperflexion is the most common type of injury to the cervical spine and is typically seen following trauma such as motor vehicle collisions, falls, or diving into shallow water. Axial loading and hyperextension are other mechanisms of injury seen in subaxial cervical spine trauma (Figs. 26.5a–c and 26.6a–d). Because it is the fulcrum of cervical movement and maximum flexion, C5–C6 is the most frequently injured level [5, 35] (Fig. 26.7).

Hyperflexion can cause compression of the anterior vertebral body, creating a wedge fracture which is generally stable. However, wedge fractures with more than 50 % loss of vertebral body height, or compression fractures with a significant retropulsed bony fragment, should be considered unstable due to associated ligamentous injury [41, 65] (Fig. 26.8a, b). Spinous process fracture, also called clay-shoveler's fracture, is a stable injury that occurs when hyperflexion causes avulsion of the spinous process, most commonly at the level of C7. While typically the result of neck flexion, these fractures can also occur by a direct blow to the spinous process or by whiplash injury [8].

Teardrop fractures occur when extreme flexion or axial loading forces cause one vertebral body to collide with the one below it, displacing a small "teardrop"-shaped bony fragment anteriorly while the inferior margin of the fractured vertebral body is forced posteriorly into the spinal canal [55] (Fig. 26.9). These fractures can result in complete disruption of the ligaments, the facet joints, and the intervertebral disc and thus are usually considered unstable. Many patients with a teardrop fracture will present with significant neurologic compromise on initial examination [54] (Fig. 26.10a, b).

Distraction flexion injuries result in damage to the posterior column. The degree of injury can range from mild stable sprain to moderate subluxation to severe facet dislocation. Sprains never involve injury to the bony elements, while subluxation injuries can occur with or without bony fractures or dislocations. As one might expect, there is a significantly higher incidence of spinal cord injury in cases of subluxation with associated fractures [31, 42] (Fig. 26.11a, b).

Bilateral facet dislocations occur when severe anterior flexion causes disruption of the intervertebral disc, ALL, facet joint capsules, and posterior ligamentous complex, all of which allow the inferior articulating facets of the upper vertebrae to move over the superior facets of the lower vertebrae. This unstable process results in anterior displacement of the spine and neurological injury in nearly 100 % of



Figs. 26.2 (**a**–**d**) A 29-year-old man suffered a fall resulting in an incomplete spinal cord injury. Axial CT shows vertical fracture through the vertebral body as well as laminar and facet fracture

patients [9, 64] (Fig. 26.12). Unilateral facet dislocations involve a rotational component to the hyperflexion injury and are considered more stable than bilateral dislocations, although neurologic deficit still occurs in approximately 75 % of cases [22].

While trauma is generally considered to occur in the young, hyperextension injuries to the cervical spine are most likely to occur in older age groups due to underlying cervical spondylosis [24, 40] (Fig. 26.13a–c). Most extension injuries in these patients damage the ALL, which allows the cord to be compressed briefly between the degenerative body and disc anteriorly and the hypertrophic ligamentum flavum posteriorly. When extension forces cause impaction of the posterior arches and facet joints, the result can be fractures of the lamina, pedicles, or spinous processes. Although cord injury can occur, most hyperextension injuries are considered stable [59].

Central cord syndrome, a hyperextension injury originally described by Schneider et al. [56], is the most common incomplete spinal cord injury. In elderly patients, it can occur after low-impact trauma, such as a fall. Recent studies have shown that central cord syndrome is associated with axonal injury in the lateral columns, with relative preservation of the gray matter [14, 32, 52, 62]. Patients typically present with weakness in all four extremities, with the upper extremities worse than the lower extremities. Hand function is usually the weakest and the slowest to recover. Gait abnormalities and urinary dysfunction are also evident. Classical surgical management of these patients stressed delayed operative intervention to allow the spinal cord edema and swelling to resolve. However, more recent literature has found that early surgery can be performed with acceptable risks [29, 67]. Surgery involves decompression at the level of injury and fixation and fusion where appropriate.

26.5 History and Physical Examination

Obtaining the history in a trauma patient can be difficult. If the patient is unable to provide his or her own history, then details from EMS, the trauma team, and the patient's family and/or witnesses should be obtained whenever possible. Key features of the trauma history include the mechanism of injury, time since injury, any loss of consciousness, and neurologic complaints, as well as neurologic status at the scene and any changes in status since that time. While trauma patients are generally young and healthy, it is still important to identify any medical comorbidities that may predispose the patient to certain types of injuries. These include ankylosing spondylitis, diffuse idiopathic skeletal hyperostosis, previous

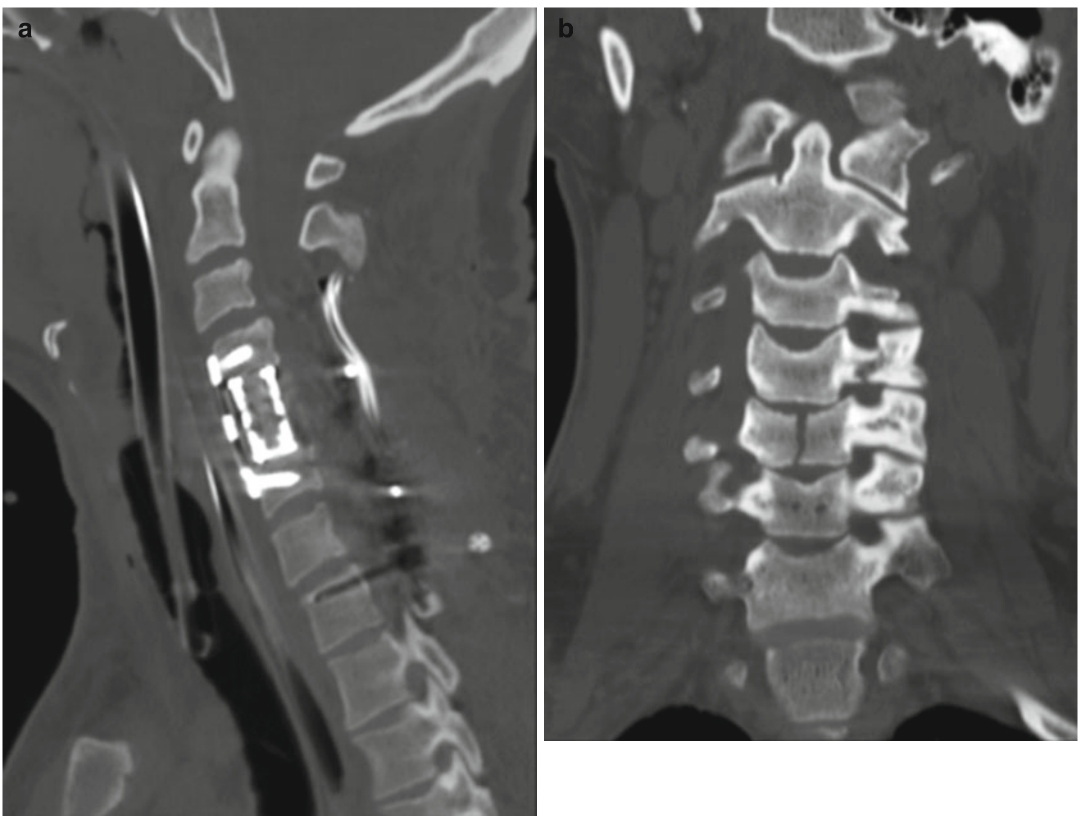


Fig. 26.3 (**a**–**f**) A 29-year-old man suffered a fall resulting in an incomplete spinal cord injury. Diagnostic work-up revealed multiple fractures: C5 burst fracture, C4 facet fracture, and C5 lamina fracture. The patient underwent C5 vertebrectomy and C4–C6 anterior fusion

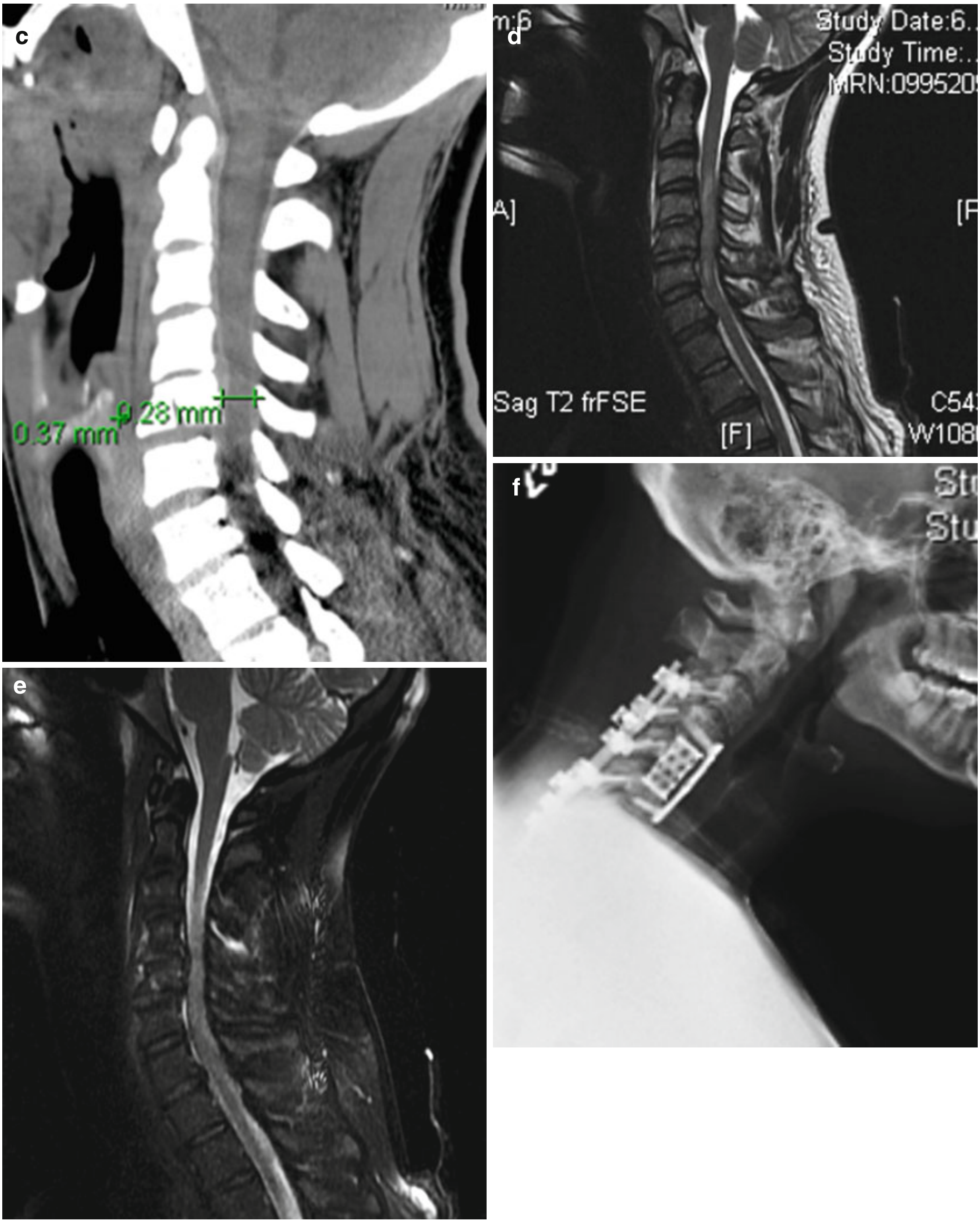


Fig. 26.3 (continued)

cervical spine fusion, and osteoporosis, as well as connective tissue disorders leading to ligamentous laxity. It is also necessary to know what medications the patient takes, whether prescribed or illicit, as well as the medications that have recently been administered by medical staff, because many agents can affect the accuracy of the physical exam.

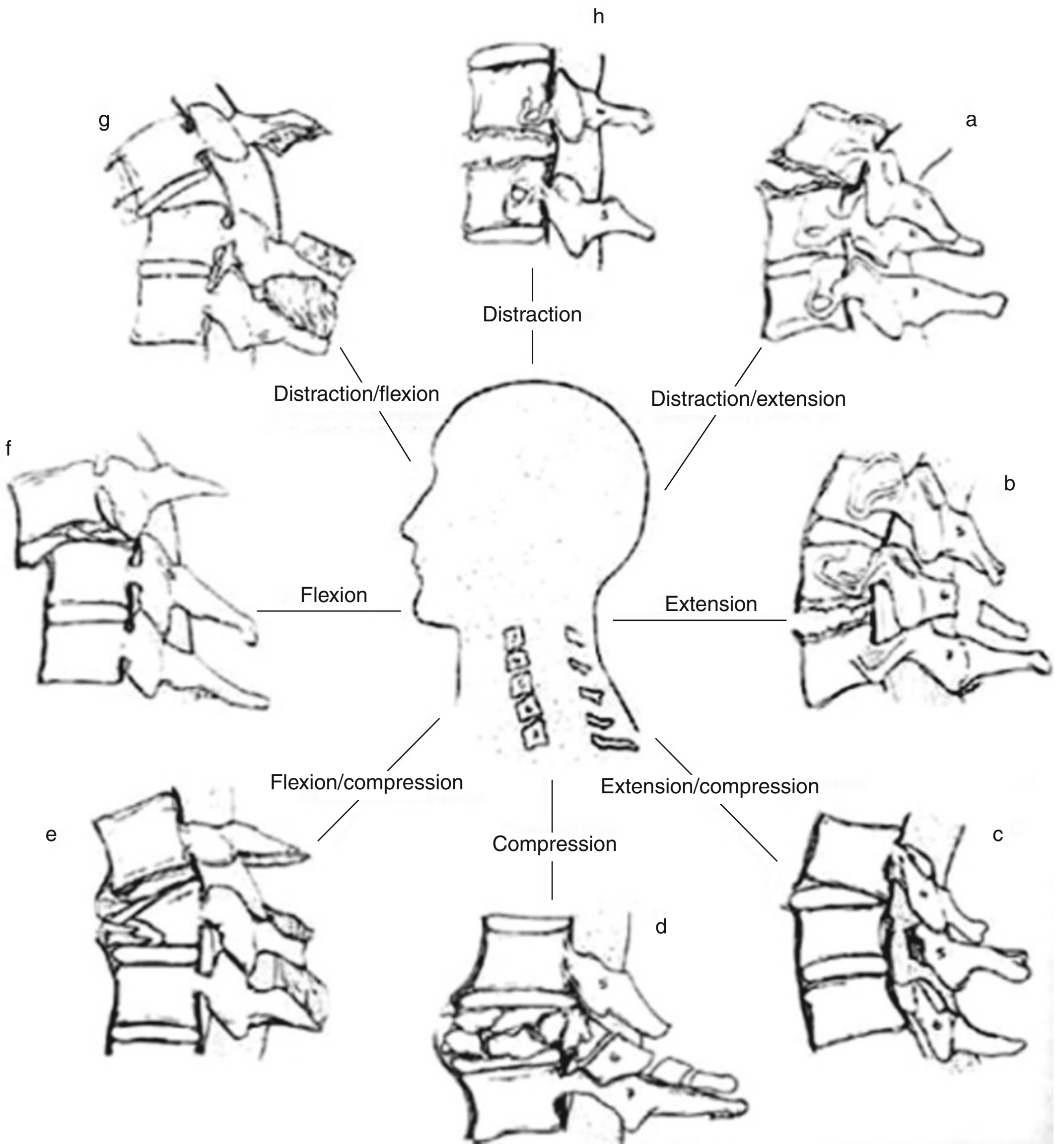


Fig. 26.4 (**a–h**) The Allen & Ferguson subaxial cervical spine injury classification system according to mechanism of injury

A thorough neurologic exam is absolutely essential in any patient with questionable spine injury, since the treatment plan is often based on the degree of neurologic compromise. In addition to a detailed neurologic exam (see Chap. 2), the physical exam must include palpation of the cervical spine assessing for bony tenderness, crepitus, and the presence of a "step-off," or widened interspinous space. Properly assessing neck pain is crucial because 84 % of patients with a cervical spine fracture will complain of midline neck pain on exam [58]. Removal of the cervical collar without radiographic imaging is allowed only if the patient is awake, alert, neurologically intact, not intoxicated or drugged, has no neck pain or tenderness, and has no distracting injuries [15–17].

The most common and widely used tool to assess neurologic status is the ASIA (American Spinal Injury Association) score (Fig. 26.14). This system can be easily and rapidly applied. It

Fig. 26.5 (**a**–**c**) Plain X-ray and CT scan of C7 compression fracture

Fig. 26.6 (**a**–**d**) Hyperflexion injury with anterior and posterior ligamentous injury. The patient was densely paraparetic on admission. Following surgery, she regained the ability to walk

involves testing motor function on a 0–5 scale in five muscle groups in each of the four limbs for a total possible score of 100. This score can be used at any time point in the patient's course of treatment to assess recovery. Perianal sensation and sphincter function should also be assessed [2].

26.6 Differential Diagnosis

Congenital variants of the cervical spine commonly mimic traumatic lesions and need to be correctly identified in order to prevent any unnecessary surgical intervention. For example, the congenital

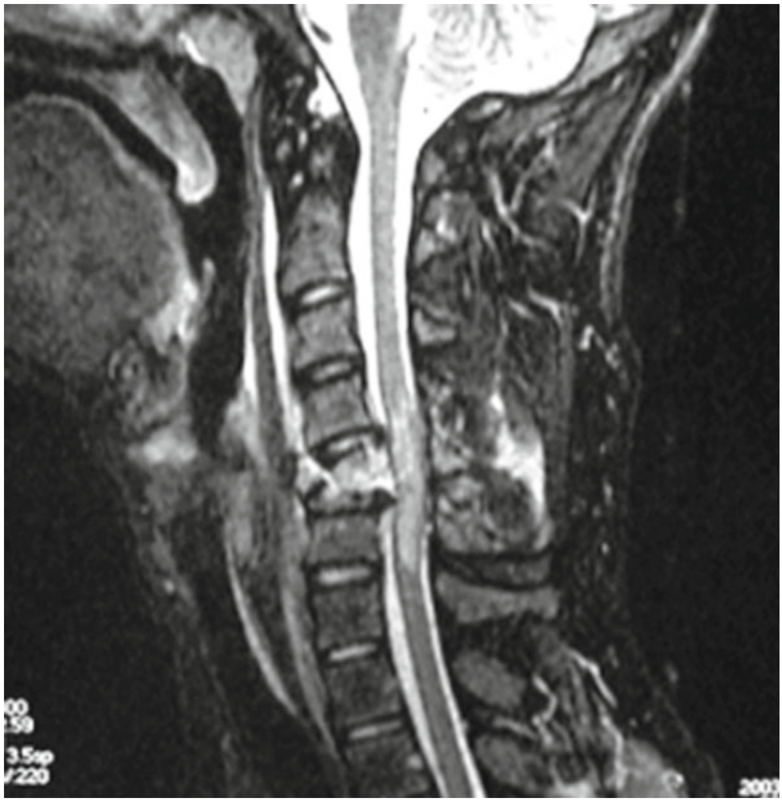


Fig. 26.7 Hyperflexion injury with C5 compression fracture. This sagittal T2 MRI also shows posterior ligamentous injury

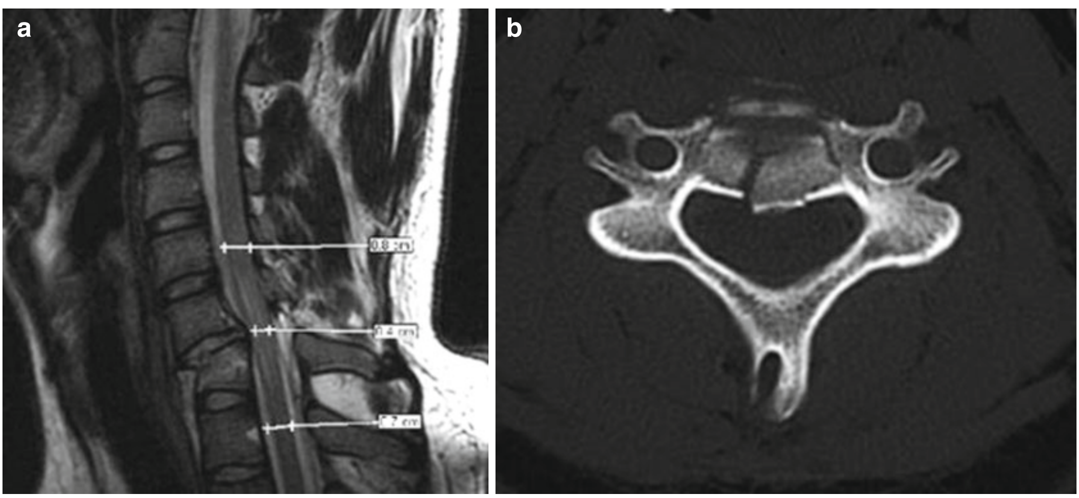


Fig. 26.8 (**a**, **b**) Hyperflexion injury with compression fracture of C7 and ligamentous injury

absence of a cervical pedicle is a rare developmental anomaly that is commonly mistaken for a unilateral facet dislocation following acute trauma [45, 46, 57]. These patients do not typically require surgery, although several have undergone an operation in the past when the congenital lesion was not correctly identified during the initial presentation.

Spine surgeons must also differentiate between acute and chronic traumatic injuries to avoid overtreatment in stable patients. Chronic fractures are

Fig. 26.9 Plain X-ray shows C6 teardrop fracture

Fig. 26.10 (**a**, **b**) A 37-year-old man was involved in an MVA. He had a teardrop-type fracture of C6. He suffered an incomplete spinal cord injury and underwent combined anterior-posterior stabilization for this very unstable injury. The black arrow indicates widening of the C4-5 disk space. The white arrow indicates the C6 fracture

commonly identified during the work-up following an acute traumatic event. They typically appear sclerotic without any surrounding edema or tissue injury on imaging. While the patient may not be aware of the old fracture, they can usually point to a previous incident that corresponds with the injury.

26.7 Investigations/Diagnostic Imaging

The Eastern Association for the Surgery of Trauma (EAST) recommends obtaining a thin-cut axial CT from the occiput to T1 with sagittal and coronal reconstructions as the initial screening modality in suspected cervical spine trauma [15, 18–20] (Figs. 26.15a, b and 26.16). When CT is not available or not indicated, a cross table lateral radiograph of the cervical spine should be obtained, along with anteroposterior and open-mouth odontoid views [51]. Radiographic signs suggestive of traumatic cervical spine injury

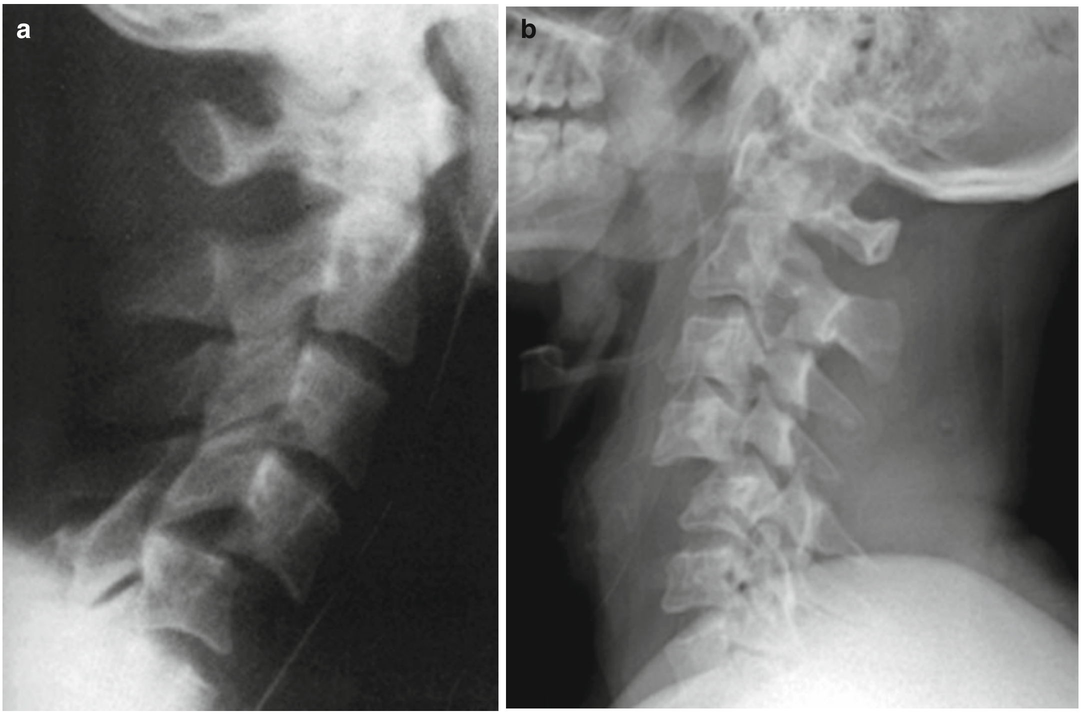


Fig. 26.11 (**a**, **b**) Plain X-ray showing C4–C5 bilateral locked facets

Fig. 26.12 MRI of patient with bilateral facet injury. There is signal change in the spinal cord

include loss of lordosis, kyphotic angulation, torticollis, widened interspinous or intervertebral disc space, vertebral rotation, discontinuity of contour lines, enlargement of the retropharyngeal space, and tracheal deviation [13, 66] (Fig. 26.17a, b). Once a cervical spine fracture is identified, a complete radiographic examination of the entire spine is necessary, because noncontiguous spinal column injuries have been reported in 10–15 % of patients with cervical injury [3, 12, 43].

If CT and three-view X-ray series are negative, dynamic flexion-extension films can be performed in an alert, neurologically intact patient with persistent neck pain to assess for instability secondary to ligamentous damage. A negative study does not rule out injury, so if the patient continues to have pain, the cervical spine should remain immobilized and the films repeated in 2–3 weeks [28, 30, 33, 60].

MRI is an extremely useful imaging modality in cervical spine trauma patients, as it can identify cervical cord changes, disc herniations, hematomas, soft tissue injuries, and ligamentous pathol-

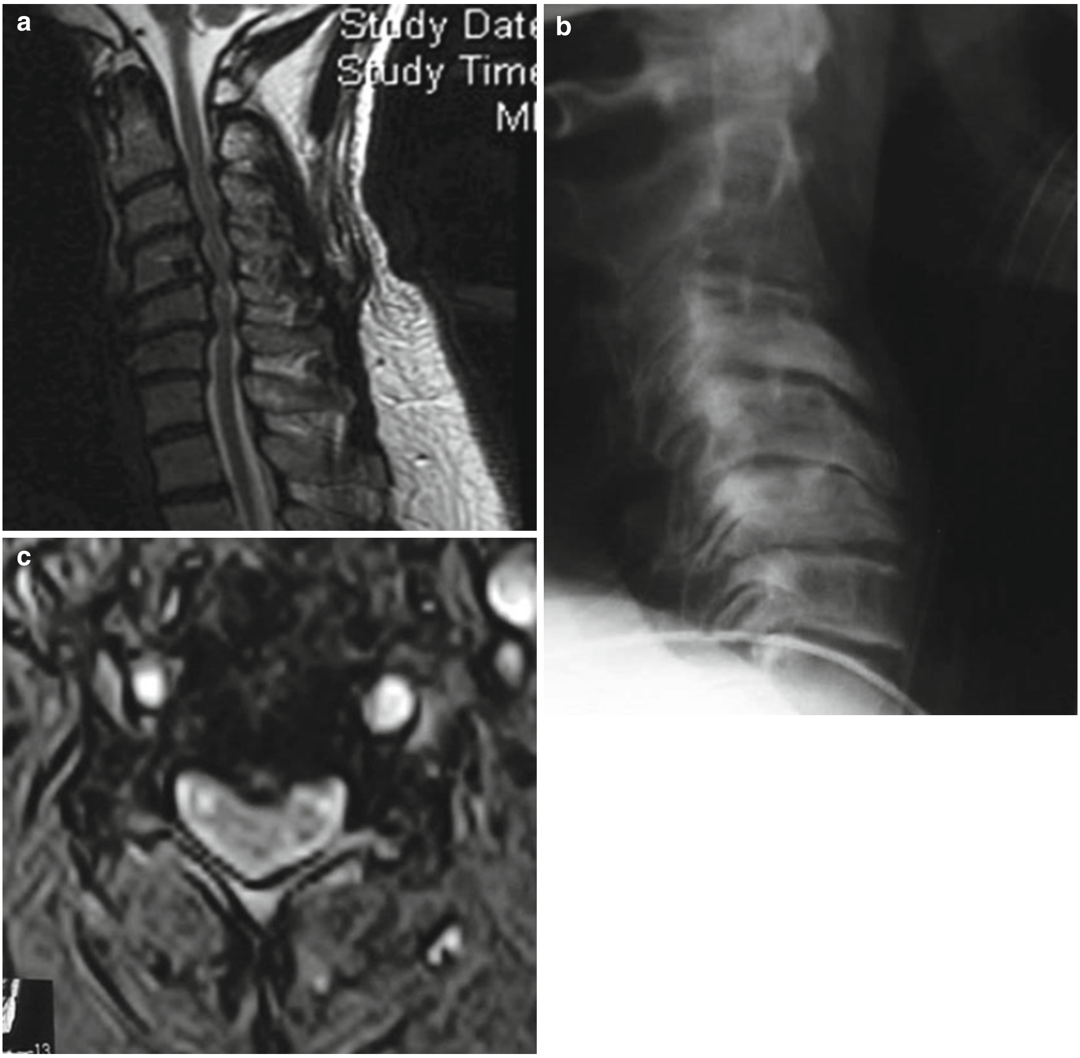


Fig. 26.13 (a–c) MRI shows severe multilevel cervical stenosis. This patient fell from a chair and suffered a central cord syndrome

ogy not easily seen on CT [26, 44] (Figs. 26.18 and 26.19). In patients who cannot undergo an MRI, CT myelogram may be used to accurately assess the relationship of bony spinal elements to the cervical cord and nerve roots [27, 53].

26.8 Treatment

The initial management of any trauma patient requires that the physician recognize the possibility of cervical injury and then properly immobilize the head and neck throughout the entire resuscitation, triage, and radiographic process until cervical fracture or instability has been definitively excluded [37, 50]. Missing a cervical injury on initial evaluation can result in delayed treatment, instability, and permanent neurologic deficit [39, 49].

A unique complication of cervical spinal cord injury is spinal shock. This usually occurs in the first 1–2 days after injury and consists of hypotension with euvolemia, as well as a loss of all motor, sensory, and reflex function below the injured level. This is due to loss of sympathetic tone and

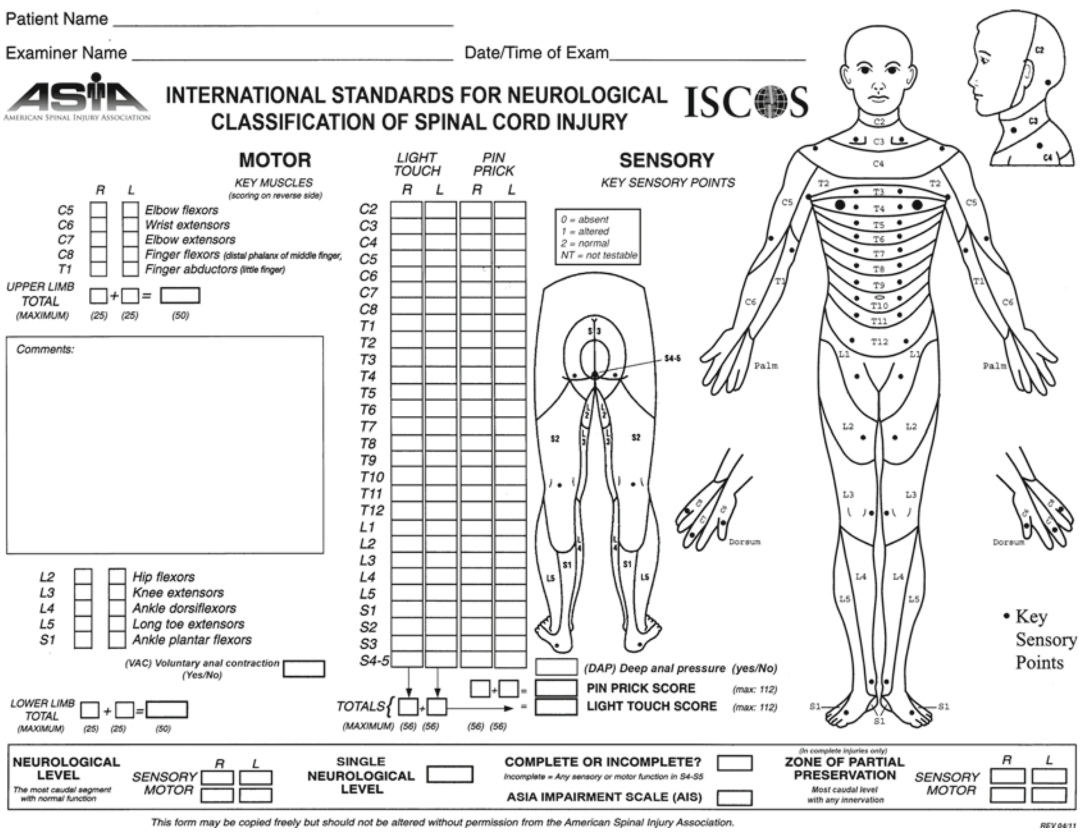


Fig. 26.14 Diagrammatic representation of the ASIA classification of spinal cord injury

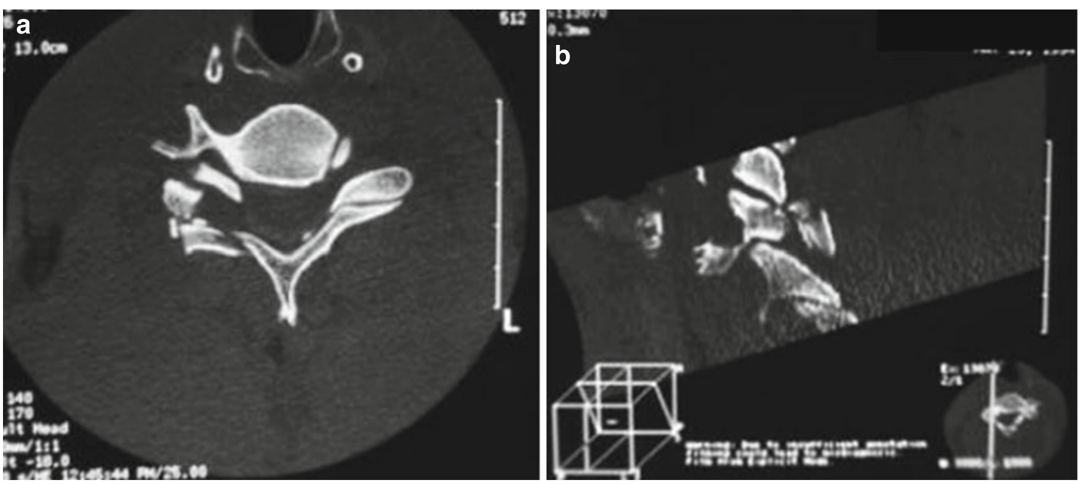


Fig. 26.15 (**a**, **b**) Thin-cut CT shows unilateral facet injury

leaves the parasympathetics unopposed, causing bradycardia. This condition must be differentiated from hypovolemic shock, which is usually due to blood loss and is treated with appropriate fluid replacement. Spinal shock is managed with sympathetic drugs, including dopamine for hypotension and atropine for bradycardia.

The general management of subaxial cervical fractures, subluxations, and dislocations involves early cervical immobilization and closed reduction of the injury whenever possible. This is then followed by stabilization, which can be operative or nonoperative depending on the type of injury, neurologic status, and overall stability of the patient.

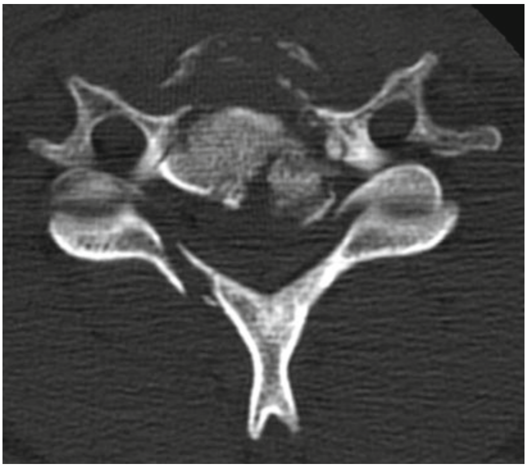


Fig. 26.16 CT scan shows injury to anterior and posterior elements

26.8.1 Nonoperative Treatment

Patients with non-displaced stable vertebral body fractures, or isolated posterior element fractures without posterior ligamentous disruption or significant kyphosis, heal well with external immobilization. In contrast, only 25 % of facet dislocations that have been successfully reduced will heal with 3 months of nonoperative immobilization [22]. It has been suggested that all facet dislocations undergo surgical stabilization following reduction, given this low success rate, the need for frequent X-rays throughout this time period, and the potential for re-dislocation [36].

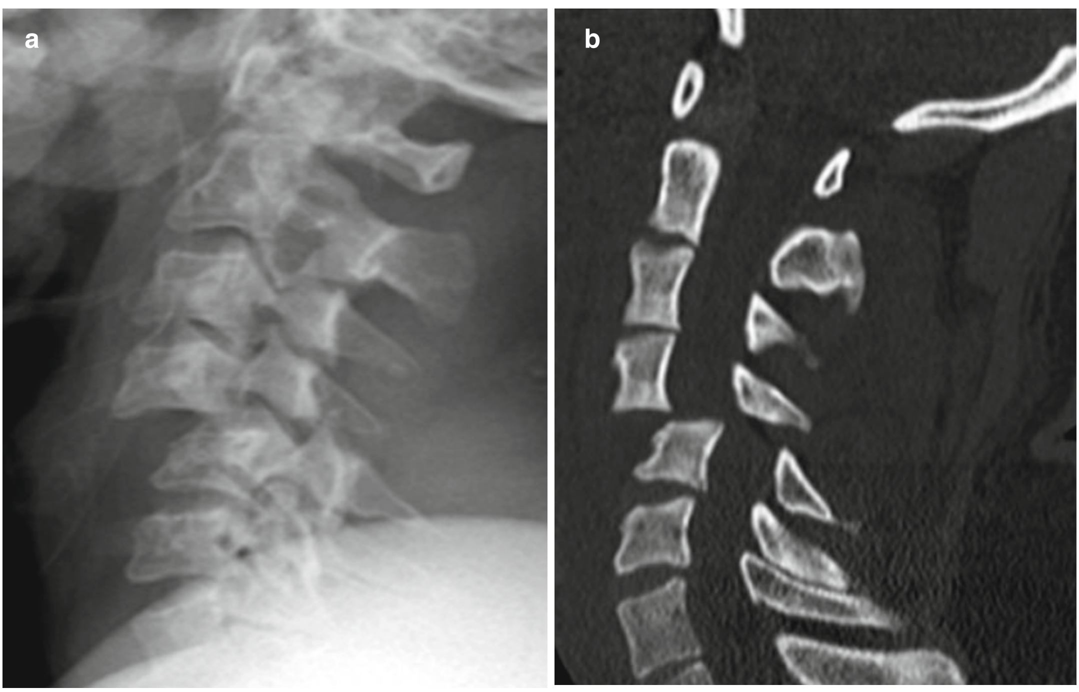


Fig. 26.17 (**a**, **b**) Plain X-ray and CT show C4–C5 bilateral locked facets

Fig. 26.18 MRI of patient with stenosis and central cord syndrome

26.8.2 Operative Treatment

Acute surgical decompression is generally warranted in patients with an incomplete spinal cord injury and radiographic evidence of spinal canal compromise, as surgery may facilitate some return of spinal cord function in these patients [11, 25]. In contrast, patients with complete spinal cord lesions should not undergo an urgent decompression, as they generally do not recover any neurologic function and would be at risk for sustaining further neurologic injury at a higher spinal level [11, 25] (Figs. 26.2a–d and 26.3a–f from case examples).

Patients with unstable subaxial cervical injuries and those who fail closed reduction typically require internal immobilization with surgical fusion. The decision to go from an anterior, posterior, or combined approach depends on the mechanism of injury and site of instability.

Indications for an anterior approach include most extension injuries, severe fractures of the posterior elements that preclude posterior stabilization, as well as herniated disc or fractured vertebral body with bone retropulsed into the spinal canal as seen in unstable burst or teardrop fractures. The anterior operation generally involves a corpectomy for decompression of neural elements and removal of damaged bone in addition to strut graft fusion with compression plates. Supplemental posterior fixation may be necessary in osteoporotic patients or cases of extensive cervical injury and instability, including teardrop fractures and burst fractures [38].

Posterior immobilization and fusion is the procedure of choice for most flexion injuries,

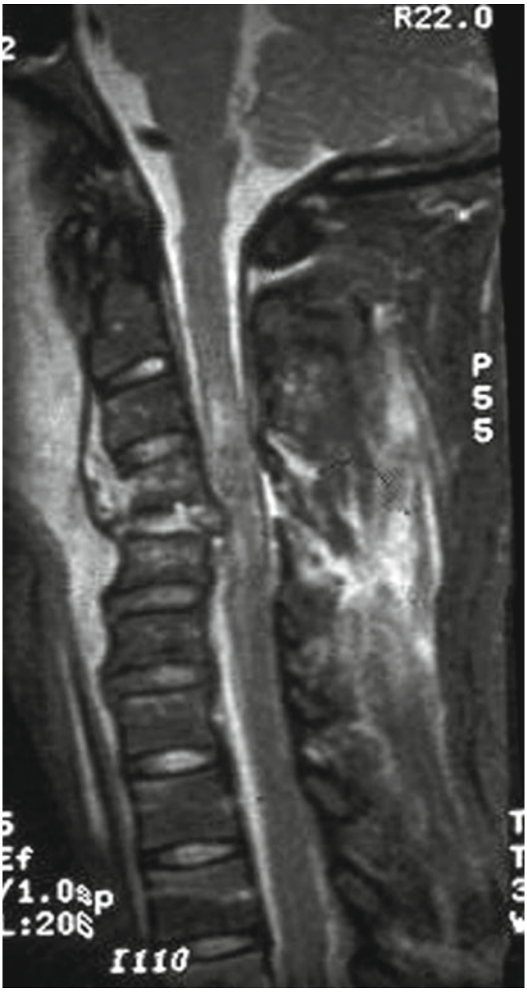


Fig. 26.19 MRI of patient with C4 compression fracture and kyphosis. There is posterior ligamentous damage as well as spinal cord edema

including posterior ligamentous injury, traumatic subluxation, unilateral or bilateral locked facets, lateral mass fractures, and simple wedge-compression fractures [7].

26.9 Postoperative Care

Antibiotics should be continued for 24 h postoperatively, and the patient's diet should be advanced slowly as tolerated, especially in patients with an anterior approach as there may be associated neck swelling. The patient should be placed on range of motion, lifting, and work restrictions until these restrictions can be cleared in clinic. External immobilization with a rigid cervical collar or cervicothoracic orthosis should be continued for several weeks following internal immobilization and fusion or for up to 15 weeks if cervical fusion was performed without internal immobilization. Patients should be instructed on proper wound care and follow-up in 7–14 days postoperatively for wound check and removal of any sutures or staples. Patients may be reevaluated in clinic with AP and lateral cervical spine films at 6 weeks, 3 months, 6 months, and up to 1 year. If the patient is doing well, he or she is typically released from follow-up after 6 months or 1 year.

26.10 Potential Complications

Patients can suffer from hardware problems due to posterior wire failure if an improper wire gauge was used for the type of fracture treated or if there was inadequate postoperative immobilization due to patient compliance issues or improper brace selection. Other hardware issues can occur following anterior plating if the plate fractures or if the screws pull out, loosen, or break. In a patient presenting with new neurologic deficits postoperatively, one must also consider nerve root, spinal cord, or vertebral artery injury due to incorrect screw placement or movement [21, 61]. Other complications seen following an anterior cervical approach include dysphagia, which increases the risk of aspiration, and potential hematoma formation, which causes airway compromise [6, 21]. These complications should be treated emergently if they arise. As with any surgery, there is always a risk of postoperative wound infection, especially in posterior cervical cases. The patient should be given proper wound care instructions upon discharge and instructed to return with any signs of wound infection.

Questions

1. A 27-year-old male was involved in an MVA. After appropriate resuscitation, neurologic exam revealed 3/5 strength in the triceps and hand muscles bilaterally, as well as lower

extremity weakness. Which diagnostic tests are helpful in evaluating this patient?
(a) Plain X-ray of the cervical spine
(b) Thin-cut CT scan of the cervical spine
(c) MRI of the cervical spine
(d) All of the above
Answer (d): All these diagnostic tests may offer unique information regarding the extent of this patient's injury.

2. Which of the following is *not* associated with central cord syndrome?
(a) Hyperflexion injury
(b) Normal hand strength
(c) Weakness in all four extremities
(d) Cervical stenosis
Answer (b): Bilateral hand weakness, not normal hand strength, is a characteristic of central cord syndrome.

3. Thin-cut CT is least helpful in diagnosing which of the following conditions?
(a) Facet fracture
(b) Laminar fracture
(c) Spinal cord hematoma
(d) Compression fracture
(e) Bilateral locked facets
Answer (c): MRI would be a better test to evaluate spinal cord hematoma.

References

1. Allen BL Jr, Ferguson RL, Lehmann TR, O'Brien RP (1982) A mechanistic classification of closed, indirect fractures and dislocations of the lower cervical spine. Spine 7(1):1–27
2. American Spinal Injury Association (2011) International standards for neurological classification of spinal cord injury. American Spinal Injury Association, Chicago
3. Barrett TW, Mower WR, Zucker MI, Hoffman JR (2006) Injuries missed by limited computed tomographic imaging of patients with cervical spine injuries. Ann Emerg Med 47(2):129–133
4. Bono CM, Schoenfeld A, Rampersaud R, Levi A, Grauer J, Arnold P, Fehlings M, Dvorak M, Vaccaro AR (2011) Reproducibility of radiographic measurements for subaxial cervical spine trauma. Spine 36(17):1374–1379
5. Boran S, Lenehan B, Street J, McCormack D, Poynton A (2011) A 10-year review of sports-related spinal injuries. Ir J Med Sci 180(4):859–863
6. Brislin BT, Hilibrand AS (2002) Avoidance of complications in anterior cervical spine revision surgery. Contemp Spine Surg 3(3):19–25
7. Brodke DS, Anderson PA, Newell DW, Grady MS, Chapman JR (2003) Comparison of anterior and posterior approaches in cervical spinal cord injuries. J Spinal Disord Tech 16(3):229–235
8. Brummett R, Rushton S (2003) Imaging in spine trauma. In: Vaccaro AR (ed) Fractures of the cervical, thoracic, and lumbar spine. Marcel Dekker, New York
9. Carrino JA, Manton GL, Morrison WB, Vaccaro AR, Schweitzer ME, Flanders AE (2006) Posterior longitudinal ligament status in cervical spine bilateral facet dislocations. Skeletal Radiol 35(7):510–514
10. Chapman JR, Anderson PA (1997) Cervical spine trauma. In: Frymoyer JW (ed) The adult spine: principles and practice, vol 1, 2nd edn. Lippincott-Raven, Philadelphia, p 1270
11. Chen TY, Dickman CA, Eleraky M, Sonntag VK (1998) The role of decompression for acute incomplete cervical spinal cord injury in cervical spondylosis. Spine 23(22):2398–2403
12. Choi SJ, Shin MJ, Kim SM, Bae SJ (2004) Non-contiguous spinal injury in cervical spinal trauma: evaluation with cervical spine MRI. Korean J Radiol 5(4):219–224
13. Clark WM, Gehweiler JA, Laib R (1979) Twelve significant signs of cervical spine trauma. Skeletal Radiol. doi:10.1007/BF00360936
14. Collignon F, Martin D, Lénelle J, Stevenaert A (2002) Acute traumatic central cord syndrome: magnetic resonance imaging and clinical observations. J Neurosurg 96(1 Suppl):29–33
15. Como JJ, Diaz JJ, Dunham CM, Chiu WC, Duane TM, Capella JM, Holevar MR, Khwaja KA, Mayglothling JA, Shapiro MB, Winston ES (2009) 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 67(3):651–659
16. Congress of Neurological Surgeons (2002) Radiographic assessment of the cervical spine in asymptomatic trauma patients. Neurosurgery 50(3 Suppl):S30–S35
17. Congress of Neurological Surgeons (2002) Radiographic assessment of the cervical spine in symptomatic trauma patients. Neurosurgery 50(3 Suppl):S36–S43
18. Daffner RH (2011) Imaging of vertebral trauma II: radiography, computed tomography, and myelography. In: Daffner RH (ed) Imaging of vertebral trauma, 3rd edn. Cambridge University Press, New York
19. Daffner RH, Hackney DB (2007) ACR appropriateness criteria on suspected spine trauma. J Am Coll Radiol 4(11):762–775
20. Daffner RH, Sciulli RL, Rodriguez A, Protetch J (2006) Imaging for evaluation of suspected cervical

spine trauma: a 2-year analysis. Injury 37(7): 652–658
21. Daniels AH, Riew KD, Yoo JU, Ching A, Birchard KR, Kranenburg AJ, Hart RA (2008) Adverse events associated with anterior cervical spine surgery. J Am Acad Orthop Surg 16(12):729–738
22. Dvorak MF, Fisher CG, Aarabi B, Harris MB, Hurbert RJ, Rampersaud YR, Vaccaro A, Harrop JS, Nockels RP, Madrazo IN, Schwartz D, Kwon BK, Zhao Y, Fehlings MG (2007) Clinical outcomes of 90 isolated unilateral facet fractures, subluxations, and dislocations treated surgically and nonoperatively. Spine 32(26):3007–3013
23. Dvorak MF, Fisher CG, Fehlings MG, Rampersaud YR, Oner FC, Aarabi B, Vaccaro AR (2007) The surgical approach to subaxial cervical spine injuries: an evidence-based algorithm based on the SLIC classification system. Spine 32(23):2620–2629
24. Ehara S, Shimamura T (2001) Cervical spine injury in the elderly: imaging features. Skeletal Radiol 30(1):1–7
25. Fehlings MG, Sekhon LH, Tator C (2001) The role and timing of decompression in acute spinal cord injury: what do we know? what should we do? Spine 26(24 Suppl):S101–S110
26. Fraser JF, Diwan AD, Peterson M, O'Brien MF, Mintz DN, Khan SN, Sandhu HS (2002) Preoperative magnetic resonance imaging screening for a surgical decision regarding the approach for anterior spine fusion at the cervicothoracic junction. Spine 27(7):675–681
27. Furman MB, Giovanniello MT, O'Brien EM (2003) Incidence of intravascular penetration in transforaminal cervical epidural steroid injections. Spine 28(1):21–25
28. Goodnight TJ, Helmer SD, Dort JM, Nold RJ, Smith RS (2008) A comparison of flexion and extension radiographs with computed tomography of the cervical spine in blunt trauma. Am Surg 74(9): 855–857
29. Guest J, Eleraky MA, Apostolides PJ, Dickman CA, Sonntag VK (2002) Traumatic central cord syndrome: results of surgical management. J Neurosurg 97 (1 Suppl):25–32
30. Halpern CH, Milby AH, Guo W, Schuster JM, Gracias VH, Stein SC (2010) Clearance of the cervical spine in clinically unevaluable trauma patients. Spine 35(18):1721–1728
31. Harrop JS, Jeyamohan S, Sharan A, Ratliff J, Flanders A, Maltenfort M, Falowski S, Vaccaro A (2008) Acute cervical fracture or congenital spinal deformity? J Spinal Cord Med 31(1):83–87
32. Harrop JS, Sharan A, Ratliff J (2006) Central cord injury: pathophysiology, management, and outcomes. Spine J 6(6 Suppl):198S–206S
33. Khan SN, Erickson G, Sena MJ, Gupta MC (2011) Use of flexion and extension radiographs of the cervical spine to rule out acute instability in patients with negative computed tomography scans. J Orthop Trauma 25(1):51–56
34. Khanna G, El-Khoury GY (2007) Imaging of cervical spine injuries of childhood. Skeletal Radiol 36(6): 477–494
35. Khanna G, El-Khoury GY (2011) Vertebral injuries in children. In: Daffner RH (ed) Imaging of vertebral trauma, 3rd edn. Cambridge University Press, New York
36. Koivikko MP, Myllynen P, Santavirta S (2004) Fracture dislocations of the cervical spine: a review of 106 conservatively and operatively treated patients. Eur Spine J 13(7):610–616
37. Krantz BE, Ali J, Aprahamin C (eds) (2004) Advanced trauma life support for doctors (ATLS): student course manual, 7th edn. American College of Surgeons Committee on Trauma, Chicago
38. Kwon BK, Anderson PA (2008) Injuries of the lower cervical spine. In: Browner BD, Jupiter JB, Levine AM, Trafton PG, Krettek C (eds) Skeletal trauma: basic science, management and reconstruction, 4th edn. WB Saunders, Philadelphia
39. Kwon BK, Vaccaro AR, Grauer JN, Fisher CG, Dvorak MF (2006) Subaxial cervical spine trauma. J Am Acad Orthop Surg 14(2):78–89
40. Lomoschitz FM, Blackmore CC, Mirza SK, Mann FA (2002) Cervical spine injuries in patients 65 years old and older: epidemiologic analysis regarding the effects of age and injury mechanism on distribution, type, and stability of injuries. AJR Am J Roentgenol 178(3):573–577
41. Looby S, Flanders A (2011) Spine trauma. Radiol Clin North Am 49(1):129–163
42. Macias MY, Wolfla CE (2008) Biomechanical considerations for early operative intervention in vertebral column fractures and dislocations. In: Loftus CM (ed) Neurosurgical emergencies. Thieme Medical, New York
43. Miller CP, Brubacher JW, Biswas D, Lawrence BD, Whang PG, Grauer JN (2011) The incidence of noncontiguous spinal fractures and other traumatic injuries associated with cervical spine fractures: a 10-year experience at an academic medical center. Spine 36(19):1532–1540
44. Miyanji F, Furlan JC, Aarabi B, Arnold PM, Fehlings MG (2007) Acute cervical traumatic spinal cord injury: MR imaging findings correlated with neurologic outcome–prospective study with 100 consecutive patients. Radiology 243(3):820–827
45. Moon SJ, Lee JK, Seo BR, Kim SH (2008) Traumatic subluxation associated with absent cervical pedicle: case report and review of the literature. Spine 33(18):E663–E666
46. Oh YM, Eun JP, Koh EJ, Choi HY (2009) Posterior arch defects of the cervical spine: a comparison between absent pedicle syndrome and spondylolysis. Spine J 9(7):e1–e5
47. Okonkwo DO, Gasco J, Shaffrey CI, Smith HE, Vaccaro AR (2010) Cervical spine kinematics and the biomechanics of the injured cervical spine: subaxial cervical junction (C3-C7). In: Vaccaro AR, Anderson P

(eds) Cervical spine trauma. Wolters Kluwer, Philadelphia
48. Patel AA, Hurlbert RJ, Bono CM, Bessey JT, Yang N, Vaccaro AR (2010) Classification and surgical decision making in acute subaxial cervical spine trauma. Spine 35(21 Suppl):S228–S234
49. Platzer P, Hauswirth N, Jaindl M, Chatwani S, Vecsei V, Gaebler C (2006) Delayed or missed diagnosis of cervical spine injuries. J Trauma 61(1): 150–155
50. Poonnoose PM, Ravichandran G, McClelland MR (2002) Missed and mismanaged injuries of the spinal cord. J Trauma 53(2):314–320
51. Quann JT, Sidwell RA (2011) Imaging of the cervical spine in injured patients. Surg Clin North Am 91(1):209–216
52. Quencer RM, Bunge RP, Egnor M, Green BA, Puckett W, Naidich TP, Post MJ, Norenberg M (1992) Acute traumatic central cord syndrome: MRI-pathological correlations. Neuroradiology 34(2): 85–94
53. Ragab A, Deshazo RD (2008) Management of back pain in patients with previous back surgery. Am J Med 121(4):272–278
54. Rao SK, Wasyliw C, Nunez DB Jr (2005) Spectrum of imaging findings in hyperextension injuries of the neck. Radiographics 25(5):1239–1254
55. Savas PE (2003) Biomechanics of injuries to the cervical spine. In: Vaccaro AR (ed) Fractures of the cervical, thoracic, and lumbar spine. Marcel Dekker, New York
56. Schneider RC, Cherry G, Pantek H (1954) The syndrome of acute central cervical spinal cord injury; with special reference to the mechanisms involved in hyperextension injuries of cervical spine. J Neurosurg 11(6):546–577
57. Sheehan J, Kaptain G, Sheehan J, Jane J Sr (2000) Congenital absence of a cervical pedicle: report of two cases and review of the literature. Neurosurgery 47(6):1439–1442
58. Stiell IG, Clement CM, McKnight RD, Brison R, Schull MJ, Rowe BH, Worthington JR, Eisenhauer MA, Cass D, Greenberg G, MacPhail I, Dreyer J, Lee JS, Bandiera G, Reardon M, Holroyd B, Lesiuk H, Wells GA (2003) The Canadian C-spine rule versus the NEXUS low-risk criteria in patients with trauma. N Engl J Med 349(26):2510–2518
59. Sundgren PC, Flanders AE (2008) Acute spinal trauma and injuries. In: Hodler J, von Schulthess GK, Zollikofer CL (eds) Diseases of the brain, head and neck, spine: diagnostic imaging and interventional techniques. Springer, Milan
60. Tomycz ND, Chew BG, Chang YF, Darby JM, Gunn SR, Nicholas DH, Ochoa JB, Peitzman AB, Schwartz E, Pape HC, Spiro RM, Okonkwo DO (2008) MRI is unnecessary to clear the cervical spine in obtunded/comatose trauma patients: the four-year experience of a level I trauma center. J Trauma 64(5): 1258–1263
61. Traynelis VC, Eichholz KM, Cusick JF, Haid RW Jr (2005) Dorsal subaxial cervical instrumentation techniques. In: Benzel EC (ed) Spine surgery: techniques, complication avoidance, and management, vol 2, 2nd edn. Elsevier, Philadelphia
62. Trombly R, Guest JD (2007) Acute central cord syndrome arising from a cervical epidural abscess: case report. Neurosurgery 61(2):E424–E425; discussion E425
63. 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 (2007) 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 32(21): 2365–2374
64. Vaccaro AR, Madigan L, Schweitzer ME, Flanders AE, Hilibrand AS, Albert TJ (2001) Magnetic resonance imaging analysis of soft tissue disruption after flexion-distraction injuries of the subaxial cervical spine. Spine 26(17):1866–1872
65. Van Goethem JW, Maes M, Ozsarlak O, van den Hauwe L, Parizel PM (2005) Imaging in spinal trauma. Eur Radiol 15(3):582–590
66. White AA, Panjabi MM (1990) The problem of clinical instability in the human spine: a systematic approach. In: White AA, Panjabi MM (eds) Clinical biomechanics of the spine, 2nd edn. JB Lippincott, Philadelphia
67. Yamazaki T, Yanaka K, Fujita K, Kamezaki T, Uemura K, Nose T (2005) Traumatic central cord syndrome: analysis of factors affecting the outcome. Surg Neurol 63(2):95–99

27 Thoracolumbar Spine (T1–L2)

Yasutsugu Yukawa

27.1 Introduction

The thoracolumbar junction is a common site for spinal injuries. The thoracic spine is distinguished from the cervical and lumbar spine by the presence of the ribs and their articulations. The thoracic spine shows physiological kyphosis, and the lumbar spine shows physiological lordosis. The thoracolumbar region is a transitional zone of spinal alignment. The rib cage restricts motion of the thoracic spine and contributes to stiffness and stability. The rigid thoracic spine and the mobile lumbar spine create a junction exposed to a concentration of stresses, leading to a higher likelihood of injuries between T10 and L2 compared with other spinal regions. These injuries include compression fractures, burst fractures, flexion distraction injuries (seat belt-type injuries), and fracture dislocations. Among these, unstable injuries result in the loss of trunk support and neural injury to the spinal cord, conus medullaris, and/or cauda equina.

Y. Yukawa
Department of Orthopaedic Surgery,
Chubu Rosai Hospital,
1-10-6 Komei, Minato-ku, Nagoya 455-8530, Japan

University of Utah Neurosciences Center,
590 Wakara Way, Salt Lake City, UT 84108, USA
e-mail: yukawa.ort@chubuh.rofuku.go.jp

27.2 Case Study

A 19-year-old male fell from a height of 25 ft while painting at work, landing on his buttocks. He immediately experienced severe back pain and left buttock pain, and he was brought to the emergency room of the nearest hospital for evaluation. He was then transferred to a spine center for the treatment of L2 vertebral and left iliac fractures (Fig. 27.1). He complained of severe low back pain, left hip pain, and left lower limb numbness, but no weakness or loss of bowel/bladder control.

Anteroposterior (AP) and lateral radiographs and computed tomography (CT) imaging of the thoracolumbar spine showed a burst fracture of the L2 vertebral body with 60 % canal compromise (Fig. 27.2). Magnetic resonance imaging (MRI) revealed encroachment of the spinal canal and severe compression of the cauda equina (Fig. 27.3). The patient underwent L1–L3 posterior instrumentation with pedicle screws and hooks, followed by anterior decompression and right iliac bone grafting (Fig. 27.4). Left iliac fracture was treated conservatively. One year later, radiography and CT demonstrated secure bony union with good alignment and complete canal clearance. He returned to his previous work without pain or neurologic deficit (Figs. 27.5 and 27.6).

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_27, © Springer-Verlag Berlin Heidelberg 2014

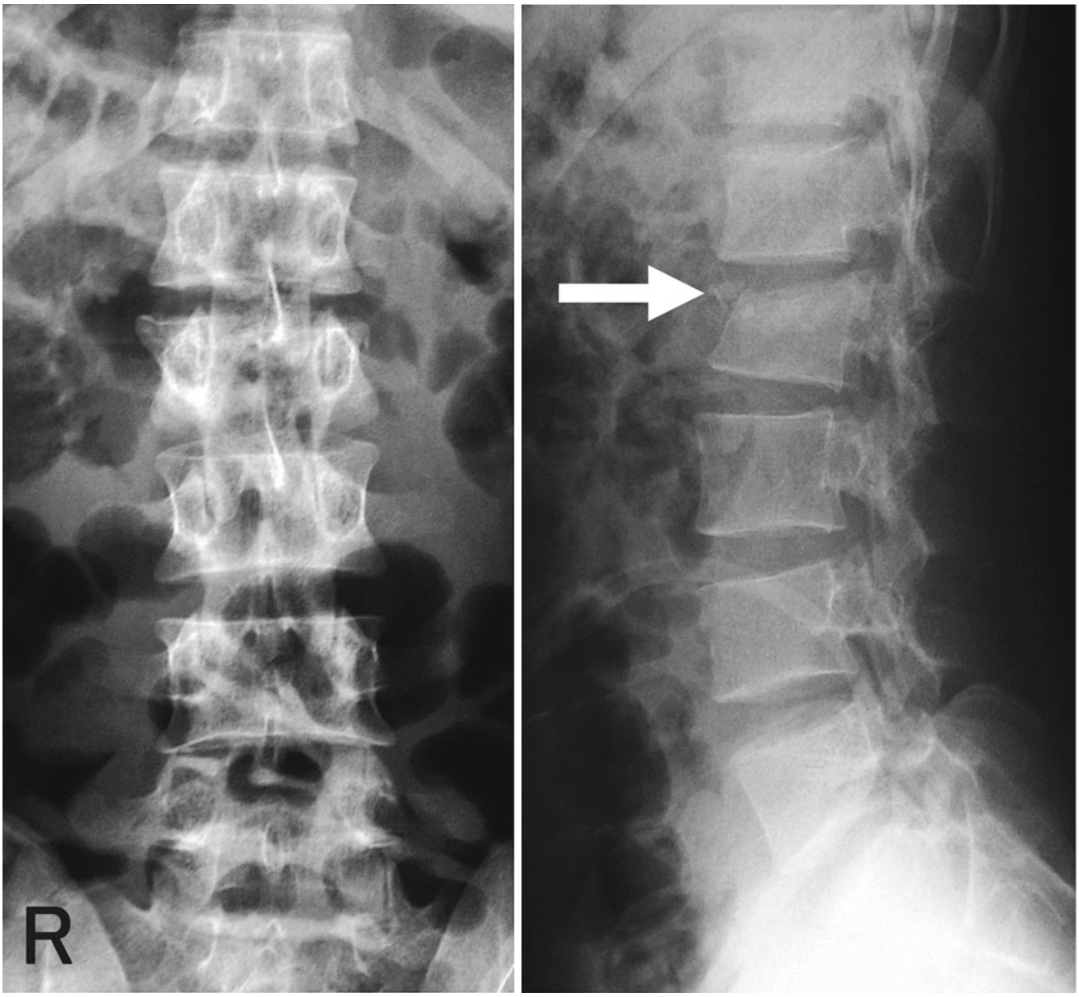


Fig. 27.1 Anteroposterior and lateral X-ray

27.3 Pathology (Basics of Pathology and Pathophysiology)

The thoracic spine is more resistant to flexion/extension and axial rotation forces than the cervical or lumbar spine because of the presence of the rib cage. Two to three times the amount of compressive load can be tolerated relative to other spinal segments before instability develops. Because of these differences, very high mechanical forces are required to cause thoracic vertebral injuries, making concomitant injuries to the other organs and/or extremities more likely. Moreover, spinal injuries to the upper and mid-thoracic spine (T1–T10) are very rare.

Another important difference between the thoracic and lumbar spine is the spinal cord. The spinal canal is relatively narrow in the thoracic spine, making the space available for the cord significantly smaller. The blood supply to the thoracic spinal cord is also relatively sparse, increasing the susceptibility of this level to ischemia with lesser degrees of compression [23].

Thus, injury to the upper and mid-thoracic spine usually induces critical neural damage. Damage to intercostal nerve roots in the thoracic spine does not have as great of a functional consequence as similar injuries to nerve roots at the lumbar or sacral levels because of the relative lack of motor function associated with the thoracic nerve roots.

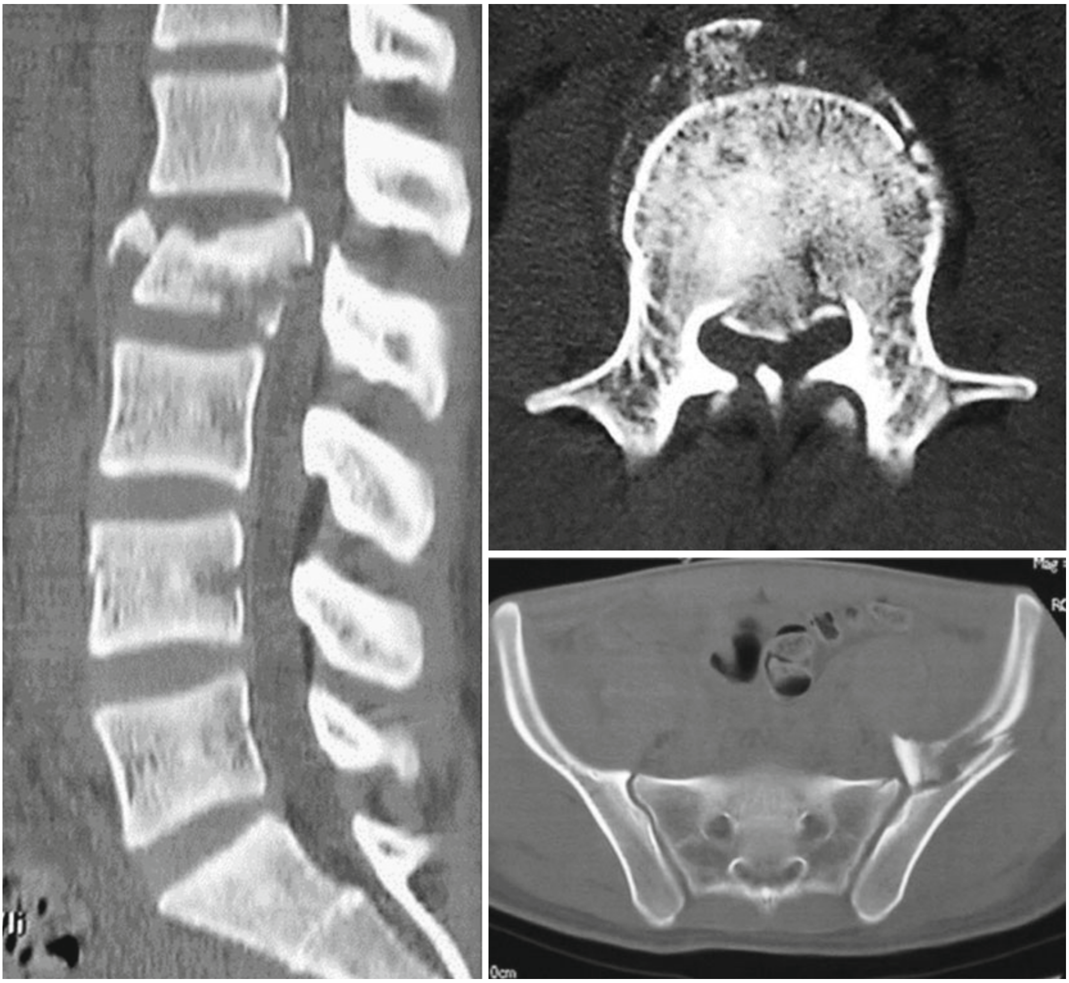


Fig. 27.2 Sagittal and axial CT scans of L2 and pelvic CT scan

The pattern of spinal injury is characteristic to the level. Fracture–dislocation is often seen at the upper or mid-thoracic spine (T1–T11). Burst fractures frequently occur at the thoracolumbar spine (T12–L2) [15].

27.4 History and Physical Exam Findings

The mechanism of injury and medical history suggest the level or degree of spine injury and offer valuable information to surgeons looking for occult injury. Likewise, checking for bruises in the obtunded patient can also be very helpful as signs of visceral injuries.

The initial evaluation must consist of the standard trauma primary survey, as prescribed by the advanced trauma life support protocol. This primary survey focuses on immediately identifying life-threatening injuries and beginning resuscitation if appropriate. Airway, breathing, circulation, disability, and environment are the five components of the primary survey. Thoracolumbar trauma is often associated with thoracic and abdominal injuries. These include hemothorax, pneumothorax, liver lacerations, aortic injuries, and bowel injuries. A thorough neurologic examination is also very important in every trauma patient.

Once the patient is in a stable condition, the secondary survey should be conducted. The spine must be examined by inspection and palpation.

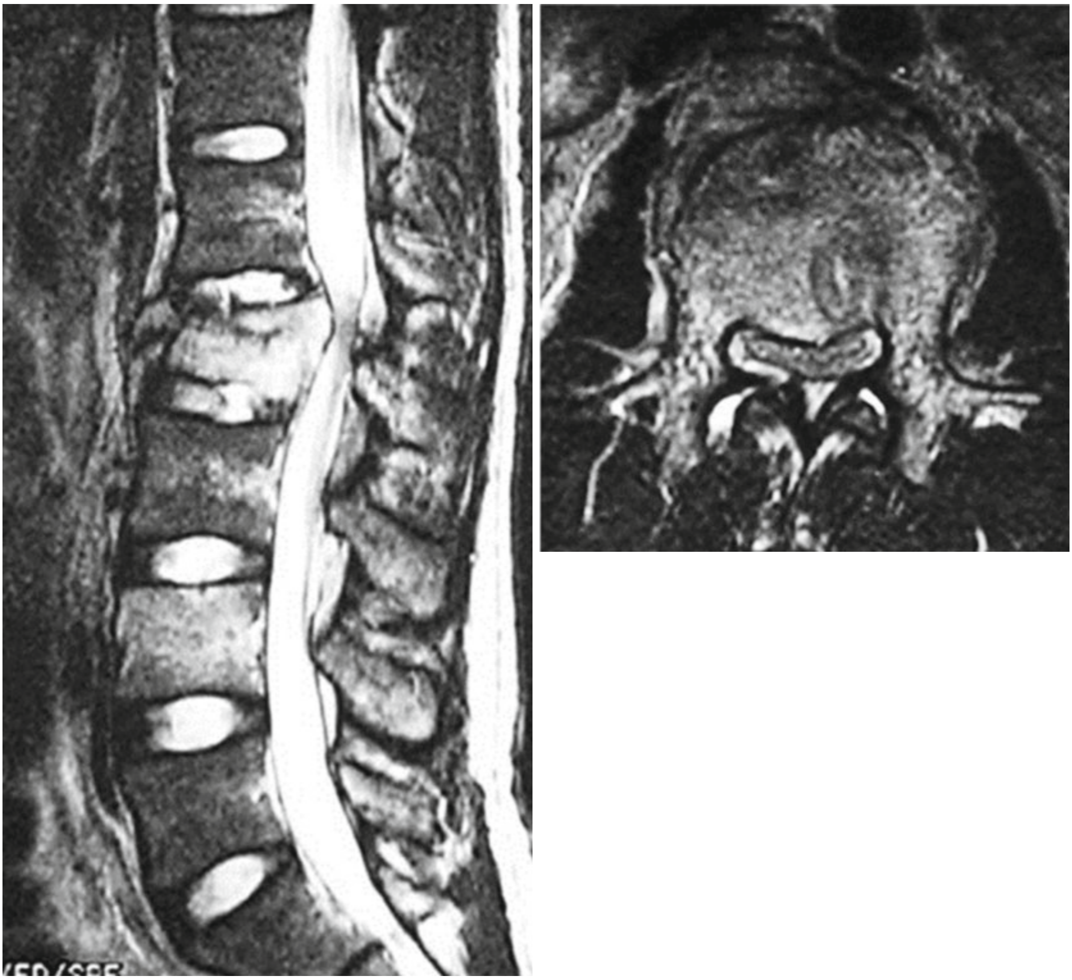


Fig. 27.3 Sagittal and axial MR imagings of L2

All spinous processes are palpated to assess for tenderness, step-offs, and widening, any of which suggest injury to the posterior elements. During the secondary survey, a detailed neurologic evaluation can be performed to identify deficits and, if necessary, determine the level of spinal cord injury (SCI). The impairment scale created by the American Spinal Injury Association is a validated method to reliably classify the severity and level of SCI. Serial examinations are necessary to detect deterioration during management. Once a final assessment of SCIs is established, the patient should be removed from the rigid board to prevent pressure sores (particularly SCI patients with impaired sensory and motor functions).

27.5 Differential Diagnosis

Thoracolumbar injuries (with the exception of compression fractures) are usually induced by high-energy accidents. Pathological fractures should be considered if these injuries occur in young or middle-aged subjects after minor injury (e.g., falls). Pathological fractures are caused by osteopenia, spinal tumors, metastatic tumors, and infection. The pedicle sign in the AP view of

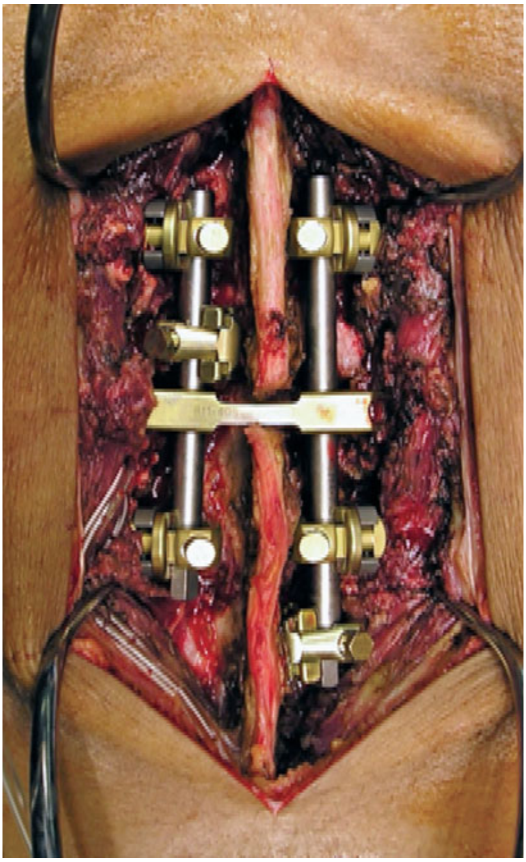


Fig. 27.4 Intraoperative picture after posterior instrumentation with pedicle screws and hooks

plain radiography, MRI, CT, and bone scintigraphy is helpful in the diagnosis.

27.6 Investigations/Diagnostic Imaging

Typically, plain radiographs in the AP and lateral planes can be used as a screening test, particularly if the mechanism of injury involves the possibility of compromise of the spinal column. Radiographs should be inspected for disruptions in spinal alignment or severe changes in angulation associated with translational motion. Changes in vertebral body height with retropulsion of fragments should alert the physician to the potential of compression of the spinal cord. If bony injuries are not evident on radiographs, ligamentous injuries can be assessed by investigating changes in the interspinous process distance and facet joint space.

Further evaluation with CT is necessary if plain radiography suggests bony or ligamentous injury at any level. CT offers the best resolution of bony anatomy, especially at the cervicothoracic and thoracolumbar junctions (which are difficult to assess by plain radiography). Not only axial images but also sagittal and coronal reconstruction images can improve diagnostic accuracy. Occasionally 3D reconstructions may help in evaluation of complex fractures.

MRI is used to correlate the level of fracture or neurologic impairment with SCI. Ligamentous injuries and disruptions that may have otherwise been missed by CT or plain radiography are detected in MRI. This imaging method is now a vital tool to evaluate soft tissues (including the spinal cord). MRI can also provide information about pathological fractures caused by tumors, osteoporosis, or infection.

Complete sets of imaging studies are needed in each patient for decision-making and surgical planning. These include preoperative plain radiography (AP and lateral views), CT, and MRI. Occasionally, flexion and extension radiographs in the presence of a physician are necessary to assess the integrity of the posterior ligament complex. The latter should be examined in the obtained images to assess if the fracture is a three-column injury.

27.7 Classification of Thoracolumbar Injury

Despite numerous attempts at classifying thoracolumbar spinal injuries, there remains no consensus on a single unifying algorithm of management. The ideal system should provide diagnostic and prognostic information, exhibit adequate reliability and validity, and be easily applicable to clinical practice. A few representative classifications are outlined below.

The modified Denis classification system is based on a three-column theory of spinal instability [6]. In this system, the spine is divided into

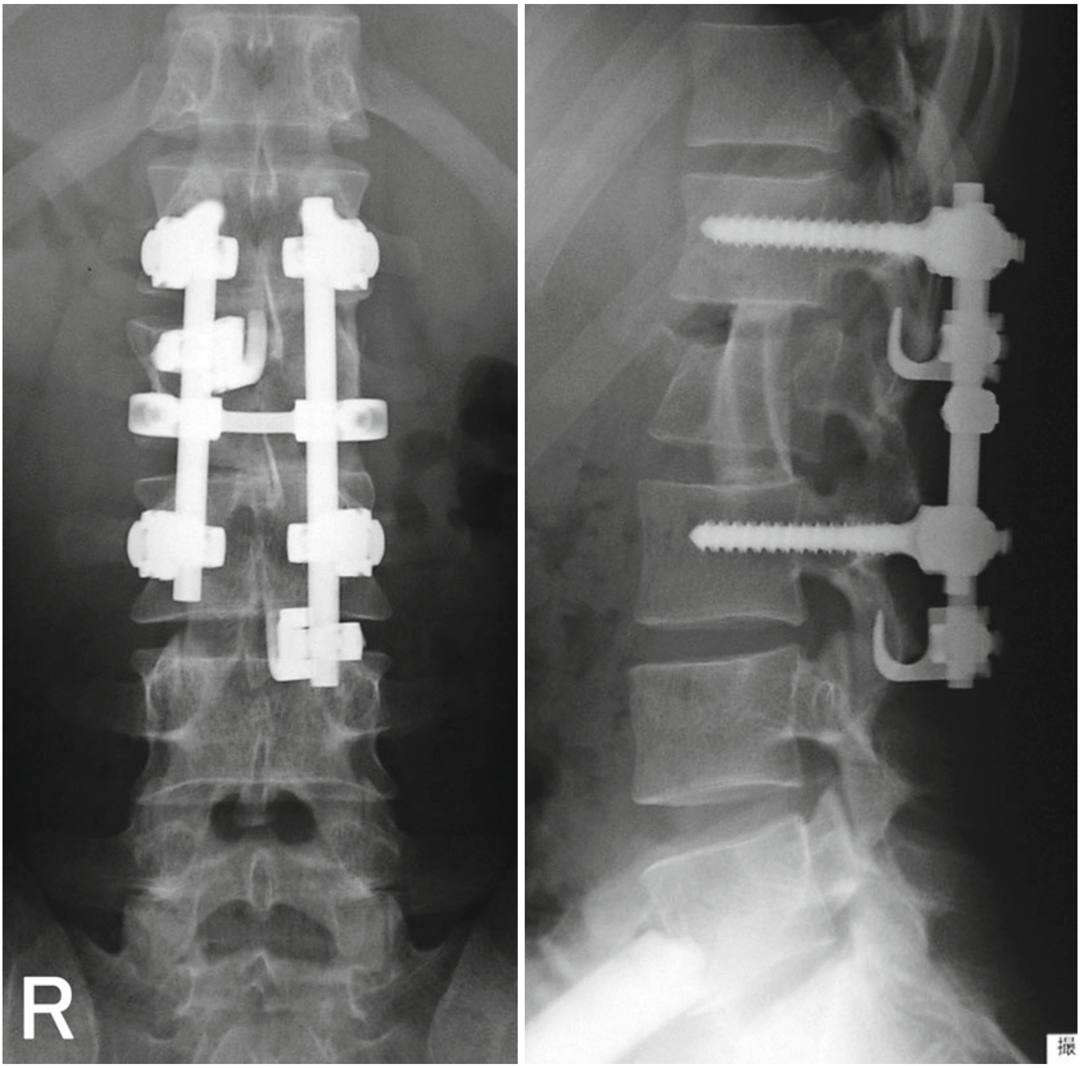


Fig. 27.5 Postoperative AP and lateral X-rays

three columns: anterior, middle, and posterior. The middle column (composed of the posterior longitudinal ligament, posterior annulus, and posterior aspect of the vertebral body) was hypothesized to be the most critical segment. The anterior column is composed of the anterior longitudinal ligament and anterior half of the annulus and vertebral body. The posterior column includes the laminae, facet joints, yellow ligaments, and posterior ligamentous complex. Failure of two or three columns is considered an unstable injury.

A more comprehensive classification (the AO classification) was proposed by Magerl et al. based on progressive pathomorphology and the mechanism of injury [16]. There are three main types of injury: A (compression), B (distraction), and C (rotation). These types are subdivided into three groups that are defined by the fracture pattern and column involved. This classification takes into account the soft tissue injury and helps to differentiate unstable fracture patterns along with predicting the likelihood of an associated neurologic deficit.

A new classification system for thoracolumbar spine injuries (including assessment of injury severity) was proposed by Vaccaro and the Spine Trauma Study Group [22]. This classification

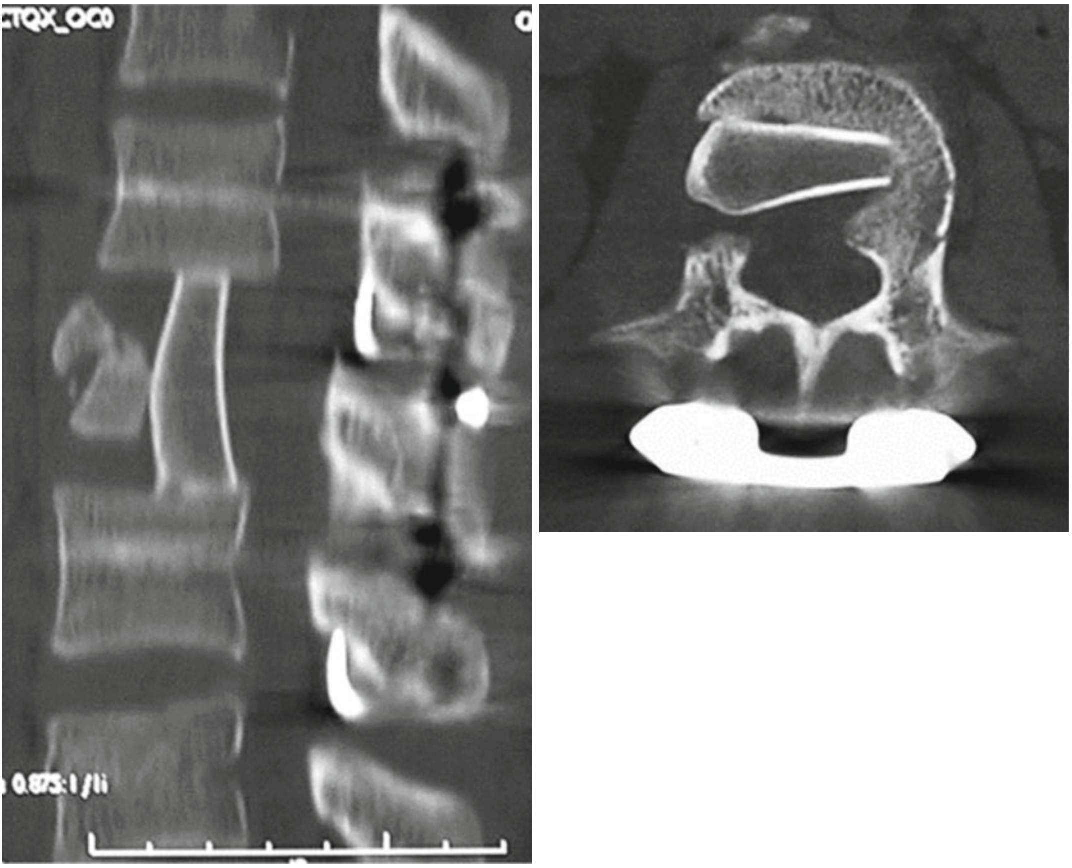


Fig. 27.6 Postoperative sagittal and axial CT scans 1 year after surgery

system–the Thoracolumbar Injury Classification and Severity Score (TLICS)–was devised based on three injury characteristics: (i) morphology of injury determined by radiographic appearance, (ii) integrity of the posterior ligamentous complex, and (iii) neurologic status of the patient. This scoring system is intended for easy application and facilitates decision-making in terms of the need for surgical versus nonsurgical care as well as the approach of surgical treatment in unstable injury patterns.

27.8 Nonsurgical Treatments

The goals of spinal trauma management involve maximizing neurologic recovery, reducing and stabilizing the spinal column, and enabling early mobilization and rehabilitation. If SCI is identified, early institution of corticosteroid therapy should be considered to improve outcome. Methylprednisolone is reported to be the only effective drug for SCIs [1–3]. However, its effectiveness has been controversial, inducing various complications at a considerably high prevalence. Currently, many spinal surgeons do not use methylprednisolone for patients with SCI, or they use it selectively in patients who have a low risk-factor profile for corticosteroid complications [11].

Once the patient has been stabilized and diagnosed with appropriate imaging, the indications for conservative or surgical treatments must be decided. Stable injuries that consist of compression fractures and stable burst fractures can be treated nonsurgically.

Conservative treatment of thoracolumbar fractures through postural reduction and immobilization has historically been as effective as surgery for most fractures [24]. However, several authors noted a significant occurrence of late neurologic deterioration in conservatively treated patients [6, 17]. Advances in spinal instrumentation and a decrease in surgical morbidity and mortality have made surgery more feasible. Nonetheless, the treatment of thoracic fractures remains controversial, especially in complete SCI and neurologically intact patients.

If there is no evidence of gross instability or symptoms, the patient is placed in an external orthosis and followed closely with radiography in the outpatient setting. Patients with stable thoracolumbar injury wear a thoracolumbosacral orthosis (TLSO) brace for 8–12 weeks. In this period, very close follow-up is needed to monitor for the potential progression of deformity. If patients experience neurologic deterioration or development of pain, diagnostic imaging should be obtained and the possibility of surgical correction entertained. After orthotic treatment, patients undergo a period of back-strengthening and hyperextension exercises.

27.9 Surgical Treatments (Options of Surgery with Discussion of Positive/Negative Features of Each Procedure and Basic Description of Procedures)

27.9.1 Indications and Advantages of Procedures

Stability of the vertebral column in the thoracolumbar region is dependent upon the integrity of the osseous and ligamentous components. Once these structures are disrupted, the stability of the vertebral column is compromised, resulting in an unstable spine.

The indication for surgery is a fracture between T1 and L2 with at least one of the following criteria:

(a) Incomplete neurologic deficit
(b) Canal compromise by retropulsed fragments with stenotic ratios: T11,12 = 35 %; L1 = 45 %; L2 = 55 % [10]
(c) Severe vertebral damage with kyphotic deformity, segmental kyphotic deformity >20°, and anterior body height <50 %
(d) Fracture with disruptions of the posterior ligamentous complex

Once a decision to proceed to surgery has been made, the choice of surgical approach is guided by the location and characteristic features of the fracture. The two main goals of surgeries for thoracolumbar injuries are (i) adequate decompression of the spinal canal to maximize neurologic recovery and (ii) creation of spinal stability to prevent painful deformity and potential future neurologic deterioration.

27.10 Timing of Surgery

The indication for emergency surgical intervention in thoracic and lumbar trauma is progressive neurologic deficit in an unstable fracture pattern with significant compression of the spinal cord. However, studies have shown that even delayed decompression of persistent compression of the thoracolumbar spinal cord can be beneficial in terms of improved neurologic status [13].

Multiple variations exist based on three methods to decompress the thecal sac: anterior, posterior, and posterolateral. There are two reconstructive techniques: anterior instrumentation and posterior instrumentation. Three representative procedures are often used in reconstructive surgery: posterior surgery, anterior surgery, and anterior–posterior combined surgery.

27.10.1 Posterior Surgery

The posterior approach is used to indirectly decompress the anterior thecal sac through distraction, fracture reduction, and realignment via ligamentotaxis at the spinal cord level with segmental spinal instrumentation. Ligamentotaxis requires a partially intact posterior longitudinal ligament and posterior annulus fibrosus (Sharpey's fibers) to help push the fracture fragments anterior as it is stretched. Distraction instrumentation has been shown to be effective in reducing the number of retropulsed bone fragments ventrally and out of the spinal canal (especially if performed within 2 days of injury). However, laminectomy alone after thoracic and thoracolumbar injuries has been shown to be associated with a significant prevalence of kyphotic deformity and neurologic deterioration. Therefore, this procedure should not be used as an isolated treatment strategy.

Posterior pedicle screw implants potentially allow for shorter posterior fixation in non-osteoporotic patients. The addition of supplemental laminar hooks or cables increases the construct stability and as such reduces the likelihood of implant failure [5]. Despite the increased rigidity of pedicle screw systems, posterior short-segment fixation for unstable thoracolumbar fractures has resulted in high failure rates. With an injured and deficient anterior column, a posterior spinal construct bears most of the axially applied loads. This leads to the potential for arthrodesis and early instrumentation failure. McCormack et al. developed a load-sharing classification system of spinal fractures based on plain radiography and CT to evaluate the success of fracture treatment with posterior short-segment pedicle screw fixation [18]. According to their classification, short-segment hardware failure is more likely to occur if there is >30 % vertebral body comminution in sagittal images of CT, more apposition of fragments, and kyphotic correction >10°. Ebelke et al. reported that all failures of short-segment pedicle screw fixation occurred in patients who did not undergo anterior bone grafting and that no patient who underwent such grafting experienced instrumentation failure [7]. Even long-segment posterior fixation with multiple anchors is a risk for failure if a significant gap or bony deficiency exists at the level of the injured vertebral body. For fracture–dislocation at a lower thoracic level, transforaminal thoracic interbody fusion was reported as a new technique to carry out the anterior column support and posterior instrumentation from the posterior approach alone [14].

The upper and mid-thoracic regions are surrounded by the rigid rib cage. The range of motion at this level is less than at other levels. Anatomically, it is difficult to reconstruct the anterior column support via an anterior approach at the upper and mid-thoracic level. Therefore, long-segment fixation between T1 and T9 is considered acceptable.

The obvious advantages of the posterior approach are its familiarity to all spinal surgeons and the relative ease of placing pedicle screws. The approach avoids potential injury to intra-abdominal or retroperitoneal structures that are at risk during anterior procedures.

27.10.2 Anterior Surgery

Anterior decompression by vertebrectomy is useful for compromise of the spinal canal due to retropulsion of vertebral bone and disc fragments. At the level of the spinal cord, the anterior approach is the most direct and reliable means of visualizing and decompressing neural tissue [4]. Anterior surgery also allows for direct restoration of the load-bearing function of the anterior spine. Eighty percent of the axial load of the intact spine is transmitted through the anterior column.

An extrapleural approach for the thoracic spine (T10–T12) with removal of the rib two levels superior to the injured level or a retroperitoneal approach for the lumbar spine (L1 and L2) just below the 12th rib is utilized to expose the injured vertebra. The left-side approach is favored below T10 for several anatomical reasons. On the left side, the aorta can be mobilized and manipulated more easily than the thin-walled vena cava. In addition, retroperitoneal dissection around the liver on the right side can be avoided.

During surgery, once the vertebral body is identified, the desired level should be confirmed by inserting a marker needle into the disc space followed by radiographic or fluoroscopic imaging. The intervertebral discs and the cartilaginous end plate cephalad and caudad to the injured level are almost fully excised after taking care not to damage the adjacent intact bony end plates.

The vertebral body and displaced fragments in the spinal canal are completely removed until the base of the contralateral pedicle is clearly visualized. A tricortical iliac crest bone graft is commonly used as the interbody spacer. Recently, allograft bone has been used as a substitute for iliac crest bone to avoid donor-site problems. However, the rate of union is less than that of autograft bone. Expandable cages packed with bone chips or autograft is used in place of strut bone grafts in older patients with osteoporosis.

Various anterior instrumentation systems are available that may be applied to the anterior spinal elements after an adequate decompression and fusion procedure [12]. Anterior spinal fusion constructs are the best choice for bony healing if the posterior ligament complex is intact. In the absence of significant posterior osteoligamentous disruption, an anterior-alone procedure utilizing a dual rod or plating fixation device may confer adequate spinal stability, avoiding the need for posterior instrumentation [9]. If significant posterior instability exists or if more than one vertebral body has been resected, a second-stage posterior stabilization and fusion procedure is usually recommended.

Anterior instrumentation usually involves ligation of segmental vessels and exposure of vertebral bodies at ≥3 levels. Taking down the diaphragm is necessary to access injuries at the thoracolumbar junction and is an invasive procedure for patients with insufficient pulmonary function or for elderly patients. A significant prevalence of severe complications is associated with anterior surgeries. Anterior thoracolumbar decompression followed by reconstruction is technically demanding and may have a steep learning curve [19].

27.10.3 Anterior–Posterior Combined Surgery

Anterior–posterior combined surgery is applied for severe destructive and unstable injury at the thoracolumbar junction. This includes fracture–dislocation and a burst fracture with disruption of the posterior ligament complex.

If the spine is in the dislocated or subluxation position, a reduction procedure should be performed first. A posterior approach provides access to injured facets and access for inserting pedicle screws and laminar hooks. In the next step, anterior decompression provides a full view of the anterior spinal canal if fracture dislocations are associated with a burst fracture or significant compromise of the spinal canal. The vertebral column is reconstructed with an autograft/allograft or a cage/lift.

The anterior–posterior combined procedure has several advantages: 360° observation of fractures, direct canal decompression, rigid fixation with posterior instrumentation, and anterior strut reconstruction [8, 15, 21, 26]. Decompression can be performed safely under a stable condition after posterior fixation with instrumentation. This surgery has few disadvantages: the need for two skin incisions and the relatively high level of invasiveness. This procedure bypasses the need for circular incision of the diaphragm. Hence, the invasiveness is comparable with that of sole anterior instrumentation surgery [12, 15].

The direct decompression potentiates good neural recovery. Rigid fixation promotes a high prevalence of union and a lower prevalence of instrumentation failure. Combined surgery with posterior instrumentation, followed by anterior decompression and strut grafting, yields good fracture reduction as well as a high prevalence of fusion and neurologic recovery in the treatment of thoracolumbar burst fractures [15, 25, 26].

27.11 Postoperative Care (Basics of In-Hospital Care and Early Home Care)

- Bracing
- A hard corset (TLSO) is usually applied for approximately 12 weeks.
- Activity
- Ambulation and rehabilitation protocols are initiated on the day of drain removal (usually 2–3 days after surgery).
- Follow-up
- Patients with paralysis are transferred to a rehabilitation unit several days after surgery.

For these patients, prevention of secondary diseases, optimization of function, and reintegration into the community are paramount. Patients are instructed to visit the clinic and undergo follow-up radiography 1, 3, 6, 12, and 24 months after surgery.

27.12 Potential Complications (Focus on Signs/Symptoms for Early Recognition of Intraoperative and Postoperative Complications)

27.12.1 Potential Intraoperative Complications

Intraoperative complications include problems related to the particular approaches to the spine, patient positioning on the surgical table, and neurologic deterioration. Iatrogenic neurologic deficit and instrument migration may occur in any surgical approach. Pseudomeningocele, major vessel injury, chylothorax, atelectasis, and pleural effusion are possible complications in anterior surgery.

Traumatic or iatrogenic dural tears should be repaired through laminectomy if encountered in posterior surgery. This can be technically difficult in anterolateral decompression and may require a fascial patch and/or lumbar cerebrospinal fluid drain.

If neurologic deterioration is detected postoperatively without a known source, nothing can be done other than to administer high-dose corticosteroids. Prophylaxis must be applied continuously throughout the procedure. Intraoperative neurophysiological monitoring is one example of such prophylaxis methods [20].

27.12.2 Postoperative Complications

Postoperative complications fall into four broad categories: general medical complications, problems associated with specific surgical approaches and positioning, postoperative infection, and instrumentation failure after pseudarthrosis. Early mobilization of patients with paralysis or multisystem trauma is important to prevent pulmonary complications, decubitus, deep vein thrombosis, and pulmonary embolism. The diagnosis of postoperative infections requires a high index of suspicion. Infections after posterior surgical approaches are generally more common than those after anterior approaches. Wound drainage and unexplained fever are usually the earliest signs of infection. Enhanced CT imaging may demonstrate an abscess at the surgical site. Early irrigation, debridement, and appropriate use of antibiotics are the hallmarks of successful treatment of this complication.

Once loss of fixation is noted on follow-up radiographs, the surgeon should take immediate steps to prevent further displacement or malalignment of the implant. This may include reoperation at the same site by changing the instrumentation and/or stabilizing the spine via alternative surgical approaches.

Questions (Typical In-Training Examination-Type Questions)

1. A 32-year-old woman complains of severe low back pain after falling from a horse. Examination reveals no neurologic disorder. Radiography shows L1 burst fractures with 15° of local kyphosis between T12 and L2. Which examinations are needed?
   (a) Nothing
   (b) CT
   (c) MRI
   (d) CT and MRI
   Preferred response (d): Canal compromise due to displaced fragments and soundness of the posterior ligament complex are key factors in deciding the need for surgery. Therefore, CT and MRI are strongly recommended.
2. CT demonstrates 30 % of canal compromise by displaced fragments. MRI shows an intact posterior ligament complex and no compression of neural elements. How do you treat this patient?
   (a) Hard bracing or body cast
   (b) Decompression (laminectomy)
   (c) Posterior decompression and fixation with instrumentation

(d) Anterior decompression and fixation with instrumentation
(e) Anterior–posterior combined surgery with instrumentation

Preferred response (a): She has no neurologic deficits, and her L1 spine is not severely injured. Hard bracing or a body cast followed by a hard brace is suitable.

3. A female patient was unfortunately lost from serial follow-up. 6 months later, she returned to the clinic with progressed kyphotic deformity and severe back pain. Radiography demonstrated angular deformity of the L1 vertebral body and 25° of local kyphosis between T12 and L2. The collapse of the T12–L1 disc and the wide gap between T12 and L1 spinal processes were seen on MRI. How do you treat this patient?
(a) Hard bracing
(b) Posterior surgery with instrumentation
(c) Anterior surgery with instrumentation
(d) Anterior–posterior combined surgery with instrumentation

Preferred response (d): A wide anterior gap between T12 and L1 could occur after posterior reduction and fixation in the neutral position. Posterior surgery with short-segment pedicle screw fixation and anterior column reconstruction with bone graft are recommended in patients with late-onset deformities.

References

1. Bracken MB, Shepard MJ, Collins WF et al (1990) A randomized controlled trial of methylprednisolone or naloxone in the treatment of acute spinal cord injury: results of the second national acute spinal cord injury study. N Engl J Med 322:1405–1411
2. Bracken MB, Holford TR (1993) Effects of timing of methylprednisolone or naloxone administration on recovery of segmental and long-tract neurological function in NASCIS 2. J Neurosurg 79:500–507
3. Bracken MB, Shepard MJ, Holford TR et al (1998) Methylprednisolone or tirilazad mesylate administration after acute spinal cord injury: 1-year follow-up: results of the third national acute spinal cord injury randomized controlled trial. J Neurosurg 89:699–706
4. Bradford DS, McBride GG (1987) Surgical management of thoracolumbar spine fractures with incomplete neurologic deficits. Clin Orthop Relat Res 218: 201–216
5. Chiba M, McLain RF, Yerby SA, Moseley TA, Smith TS, Benson DR (1996) Short-segment pedicle instrumentation. Biomechanical analysis of supplemental hook fixation. Spine 21(3):288–294
6. Denis F (1983) The three column spine and its significance in the classification of acute thoracolumbar spinal injuries. Spine 8(8):817–831
7. Ebelke DK, Asher MA, Neff JR, Kraker DP (1991) Survivorship analysis of VSP spine instrumentation in the treatment of thoracolumbar and lumbar burst fractures. Spine 16(8 Suppl):S428–S432
8. Gertzbein SD, Court-Brown CM, Jacobs RR, Marks P, Martin C, Stoll J, Fazl M, Schwartz M, Rowed D (1988) Decompression and circumferential stabilization of unstable spinal fractures. Spine 13(8): 892–895
9. Ghanayem AJ, Zdeblick TA (1997) Anterior instrumentation in the management of thoracolumbar burst fractures. Clin Orthop Relat Res (335):89–100. Review
10. Hashimoto T, Kaneda K, Abumi K (1988) Relationship between traumatic spinal canal stenosis and neurologic deficits in thoracolumbar burst fractures. Spine 13(11):1268–1272
11. Ito Y, Sugimoto Y, Tomioka M, Kai N, Tanaka M (2009) Does high dose methylprednisolone sodium succinate really improve neurological status in patient with acute cervical cord injury?: a prospective study about neurological recovery and early complications. Spine (Phila Pa 1976) 34(20):2121–2124
12. Kaneda K, Taneichi H et al (1997) Anterior decompression and stabilization with the Kaneda device for thoracolumbar burst fractures associated with neurological deficits. J Bone Joint Surg Am 79(1):69–83
13. Kostuik JP (1988) Anterior fixation for burst fractures of the thoracic and lumbar spine with or without neurological involvement. Spine 13(3):286–293
14. Machino M, Yukawa Y, Ito K, Nakashima H, Kato F (2010) A new thoracic reconstruction technique "transforaminal thoracic interbody fusion (TTIF)": a preliminary report of clinical outcomes. Spine 35(19):E1000–E1005
15. Machino M, Yukawa Y, Ito K, Nakashima H, Kato F (2011) Posterior/anterior combined surgery for thoracolumbar burst fractures. Posterior instrumentation with pedicle screws and laminar hooks, anterior decompression and strut-grafting. Spinal Cord 49(4):573–579
16. Magerl F, Aebi M, Gertzbein SD et al (1994) A comprehensive classification of thoracic and lumbar injuries. Eur Spine J 3(4):184–201
17. McAfee PC, Bohlman HH, Yuan HA (1985) Anterior decompression of traumatic thoracolumbar fractures with incomplete neurological deficit using a retroperitoneal approach. J Bone Joint Surg Am 67(1):89–104
18. McCormack T, Karaikovic E, Gaines RW (1994) The load sharing classification of spine fractures. Spine (Phila Pa 1976) 19(15):1741–1744
19. Oskouian RJ Jr, Johnson JP (2002) Vascular complications in anterior thoracolumbar spinal reconstruction. J Neurosurg 96(1 Suppl):1–5

20. Schwartz DM (2003) Intraoperative neurophysiological monitoring during post-traumatic spine surgery. In: Vaccaro AR (ed) Fractures of the cervical, thoracic and lumbar spine. Marcel Dekker, New York, pp 373–383
21. Shiba K et al (1994) Transpedicular fixation with Zielke instrumentation in the treatment of thoracolumbar and lumbar injuries. Spine 19(17):1940–1949
22. Vaccaro AR, Lehman RA Jr, Hurlbert RJ et al (2005) 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) 30(20):2325–2333
23. White AA 3rd, Panjabi MM, Posner I et al (1981) Spinal stability: evaluation and treatment. Instr Course Lect 30:457–483
24. Wood K, Buttermann G et al (2003) Operative compared with nonoperative treatment of a thoracolumbar burst fracture without neurological deficit. A prospective, randomized study. J Bone Joint Surg Am 85-A(5):773–781
25. Yukawa Y, Kato F, Ito K et al (2006) Anteroposterior combined surgery for thoracolumbar burst fractures - anterior decompression, strut-grafting and posterior instrumentation with pedicle screws and laminar hook. Paper presented at ASIA-ISCoS combined meeting, Boston, 25–28 June 2006
26. Yukawa Y (2010) Anterior and posterior surgery and fixation for burst fractures. In: Patel VV, Burger E, Brown CW (eds) Spine trauma, surgical techniques, 1st edn. Springer, Berlin /Heidelberg, pp 299–309

Spine Trauma: Low Lumbar Spine (L3–5)

28

Vu H. Le and Nitin Bhatia

28.1 Introduction

The tenets in treating spinal fractures include restoration of anatomic alignment, stabilization of fracture, decompression of neural elements, preservation of motion segments, promotion of early mobilization, and reduction of complications. Although these treatment principles stay relatively consistent as one goes from the cervical to the lumbar spine, the low lumbar spine has unique characteristics inherent to its anatomy and biomechanical behavior such as its extremely mobile lordotic alignment, its increased canal to neural element ratio, and its attachment to the sacrum that make it interesting, and occasionally quite challenging, when diagnosing and treating low lumbar fractures.

V.H. Le, M.D.
Department of Orthopaedic Surgery,
University of California, Irvine,
101 The City Drive, Pavilion 3,
Orange, CA 92868, USA
e-mail: vle3@uci.edu;

N. Bhatia, M.D. (✉)
Spine Service, Department of Orthopaedic Surgery,
UC Irvine Medical Center,
University of California, Irvine,
101 The City Drive, Pavilion 3,
2nd Floor, Orange, CA 92868, USA
e-mail: nitinbhatia@yahoo.com

28.2 Case Example

A 25-year-old female presents with acute back pain after being involved in an automobile accident in which she was a restrained passenger. At the scene, the car was found to be upside down, and the passengers needed to be extricated by emergency personnel. Upon her arrival in the emergency department, she has deformities in multiple extremities, in addition to low back pain. On examination, she has gross movement of her fingers and toes. Sensation to light touch is intact throughout, as is her rectal tone. Hoffman's and Babinski's signs are negative for any neurological deficits. Her spine exam shows midline tenderness to palpation at the level of the iliac crest, but no step-off or wound is appreciated. A trauma workup including computed tomography (CT) of the lumbar spine shows a burst fracture of L4 with about 75–80 % canal compromise, 60–70 % vertebral height loss, disruption of the facet complexes, and loss of normal lumbar lordosis (Fig. 28.1a, b). Magnetic resonance imaging (MRI) portrays disrupted anterior and posterior ligamentous complexes (Fig. 28.2). Due to the instability caused by the aforementioned characteristics, the patient was taken to surgery for L4 corpectomy with an expandable structural cage and L2–S1 posterior fusion and instrumentation with L3–L4 posterior decompression (Fig. 28.3a, b).

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_28, © Springer-Verlag Berlin Heidelberg 2014

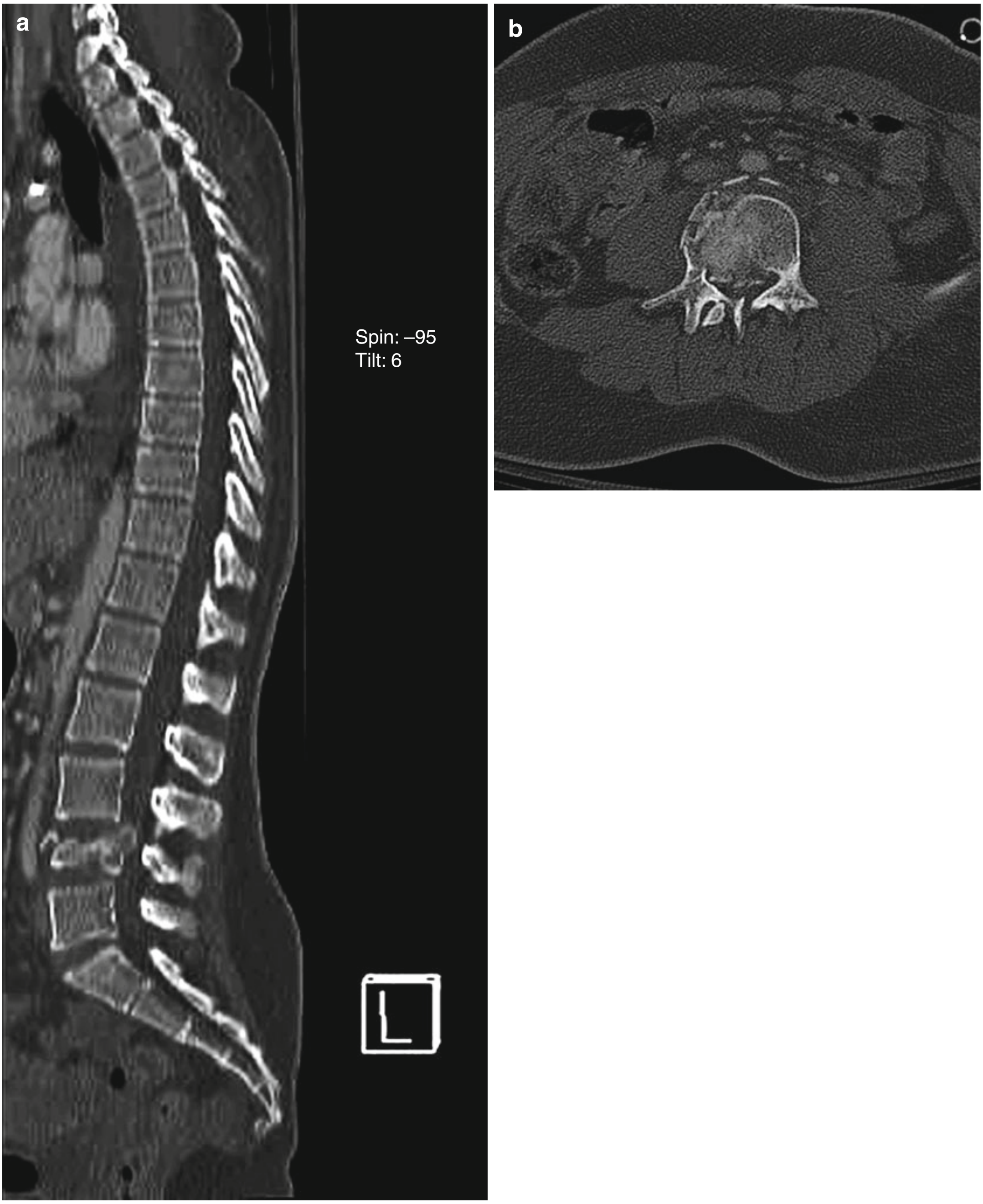


Fig. 28.1 (**a**) Midsagittal and (**b**) axial CT views showing L4 burst with more than 50 % vertebral body weight loss, 70–80 % canal compromise, and loss of normal lumbar lordosis

28.3 Anatomy

Although more attention has been given to injuries of the thoracolumbar spine due to its being the most common site of injury in thoracic and lumbar spine fractures, the low lumbar spine has distinctive aspects that must be kept in mind when approaching the diagnosis and treatment of L3–5 spinal fractures. Normal

Fig. 28.2 Sagittal T2-weighted fat-suppressed sequence demonstrating disruption of anterior longitudinal ligament, retropulsed fragments of L4 vertebral body causing root impingement, and posterior ligamentous complex injury as evident by disruption of ligamentum flavum and interspinous ligaments

lordosis of the lumbar spine ranges from 30° to 60° [1, 2]. This is largely due to the downward slope of the sacral ala, which along with the S1 body forms the lumbosacral junction with L5. This interchanging between the flexible lordotic lumbar curve and the fixed kyphotic sacral curve serves as a transitional area of high shear stress, exposing it to possible postoperative or post-traumatic nonunion. Since the lumbar facet joints are oriented in the sagittal plane, the primary motion of this segment is flexion and extension. The morphology of the pedicles has also been described to assist in the placement of pedicle screws. In general, from L1 to L5 there is an increase in size of the transverse diameter of the pedicles as well as the degree of the transverse pedicle axis [3, 4].

The spinal cord tapers into the conus medullaris, which usually ends at the L1 level. Therefore, the rest of the caudad spinal canal only houses nerve roots that are collectively known as cauda equina. This leads to a favorable canal to neural element ratio that allows the low lumbar to have significant canal stenosis before neurological impingement is encountered.

In 1983, Denis introduced the popular three-column system of describing the thoracolumbar vertebra [5]. The anterior column consists of the anterior halves of the vertebral body and intervertebral disc, along with the anterior longitudinal ligament. The middle column comprises of the posterior halves of the vertebral body and intervertebral disc, along with the posterior longitudinal ligament. Finally, the posterior column is made up of the pedicles, laminae, spinous process, facet joints, and posterior ligamentous complex (PLC), which contains the interspinous and supraspinous ligaments, ligamentum flavum, and facet capsules. The PLC is an integral structure in stabilizing the spine by serving as a tension band.

28.4 Mechanism of Injury

Like other regions of the spine, various force vectors can cause low lumbar spinal fractures. These forces include flexion, axial compression, lateral compression, flexion rotation, flexion distraction, and extension. Flexion results from a forward-bending moment involving anterior compression or wedging of the vertebral body and placing the posterior column in tension. Axial compression involves placing a downward force along the axis of the spine. This is akin to hammering a nail down along its long axis instead of striking it obliquely. Examples of axial compression injuries include compression fracture and burst fractures (Fig. 28.4). Compression fractures involve only the anterior column, where as burst fractures involves at least both the anterior and middle

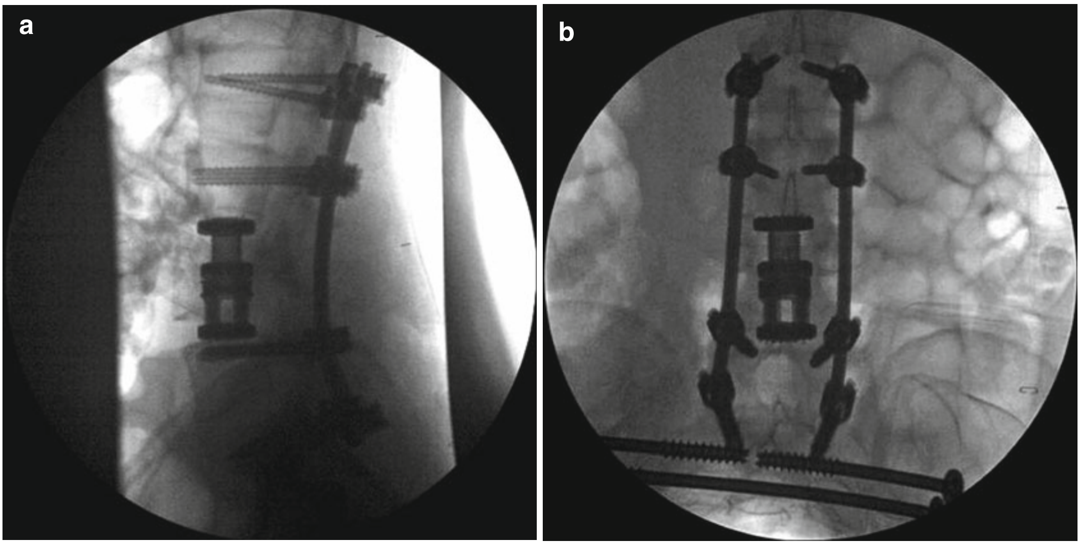


Fig. 28.3 (**a**) Lateral and (**b**) PA intraoperative fluoroscopy of lumbar spine after L4 corpectomy with placement of an expandable cage and posterior instrumentation of L2–S1

Fig. 28.4 Lateral conventional radiograph of lumbar spine showing compression fracture of L3 involving the anterior column with minimal vertebral height loss, intact posterior vertebral body wall, and no evidence of loss of lordosis

columns and may also include the posterior column. Lateral compression injuries involve a side bending moment resulting with resulting compression of half of the vertebral body and tension on the other half. This can lead to focal scoliosis in the coronal plane due to asymmetric vertebral body collapse and contralateral distraction.

The next three mechanisms can affect all three columns of the vertebra. Although rare, extension injuries place the anterior column in tension and compress the posterior column. In the ankylosing spondylitis (AS) and diffuse idiopathic skeletal hyperostosis (DISH) patient, significant displacement or distraction can occur with extension injuries due to the immobile nature of the ankylosed spine which makes it less able to absorb energy before failing. In contrast, flexion-distraction injuries involve a forward-bending force applied to the vertebral body. This is classically seen in patients involved in front impact motor vehicle accidents using lap-only seat belts. This injury pattern is known as a bony Chance fracture if the force results in bony disruption of all three columns or a ligamentous Chance variant if it disrupts the discoligamentous structures of the columns instead. The bony Chance fractures can frequently be treated nonsurgically, whereas ligamentous Chance injuries may require surgical intervention due to the inability of the discoligamentous structures to heal. If the axis of rotation in flexion-distraction injuries is anterior to the spinal column, as in lap-only seat belt injuries, distraction will be seen in all three columns. If, however, the axis of rotation is within the vertebral body, distraction in the posterior and middle column with compression of the anterior column may occur. Lastly, flexion-rotation injuries typically result in facet dislocation due to the failure of the middle and posterior columns in shear and anterior column in flexion. These injuries tend to be highly unstable.

Unique to the low lumbar spine, a retrospective study (Dai 2002) looking at 54 patients with low lumbar fractures showed that 25 of the patients sustained compression fractures, 21 with burst fractures, 5 with fracture dislocation, and 3 with flexion-distraction injuries [6]. In that group, only 3 had complete neurologic deficits, 17 had incomplete neurological injuries, and 34 were neurologically intact. This study illustrates that most low lumbar injuries involve axial loading as demonstrated by the high number of patients sustaining compression and burst fractures, as opposed to flexion distraction or flexion rotation. It also shows that with the favorable canal to neural element ratio, neurologic injury is infrequent with low lumbar fractures.

Denis also described minor injuries involving the transverse proves, spinous process, lamina, pars interarticularis, or facets [5]. Transverse and spinous process fractures can occur from direct trauma or avulsion injury from the paraspinous musculature. Pars fracture can result from low-energy hyperextension injury and is usually seen in young adults and teens participating in sports such as gymnastics.

28.5 Fracture Classification

Spinal stability refers to the ability of the spinal column to withstand stresses without progressive deformity or neurologic compromise [7]. However, judging if a fracture is stable or not is still a debate among surgeons, especially when it comes to burst fractures. Numerous fracture classifications have attempted to describe the morphology of the fracture, to predict its stability, and to give prognostic factors. To date, however, there is no consensus as to how to approach every type of fracture, and it is still a matter of surgeon's preference.

In the 1960s, Holdsworth presented one of the early spinal column models in describing the two-column model that consists of an anterior column and a posterior column [8]. He further described compression and burst fractures as stable fractures due to the intact posterior ligamentous complex, and any type of dislocation as unstable. The shortcoming of this system is that it does not incorporate any neurological status and does not provide any prognostic factors or treatment recommendations.

As described above, Denis introduced the three-column theory by adding the middle column, which plays an important stabilizing role as demonstrated in a cadaveric study (Panjabi 1995) [9]. He defined mechanical instability as disruption of at least two of the three columns. Neurologic instability is any injury that involves neurologic deficit. By association, any injury causing neurologic instability is also mechanically unstable, therefore needing surgery [10]. He also subclassifies each major type of injury such as bony versus ligamentous injuries in flexion distraction and flexion rotation, thereby, providing treatment guidance.

Subsequent classification systems, such as the Magerl system, attempt to be more detailed in terms of predicting mechanical and neurological instabilities, but they have also been accompanied with complexity and impracticality leading to poor inter- and intra-observer reliability [11, 12]. Nonetheless, the most recent classification, the thoracolumbar injury classification and severity score (TLICSS), shows promising signs despite the fact that it has not been validated by a prospective randomized study. This system looks at three components: fracture morphology, neurologic involvement, and posterior ligamentous complex status and assigns appropriate points based on the severity of each component [13, 14]. Treatment is then based on the sum of the components.

28.6 History and Physical Examination

Assessment of any trauma patient begins with a systematic approach involving basic life support and advanced trauma life support. After airway and breathing are taken care of, circulation is

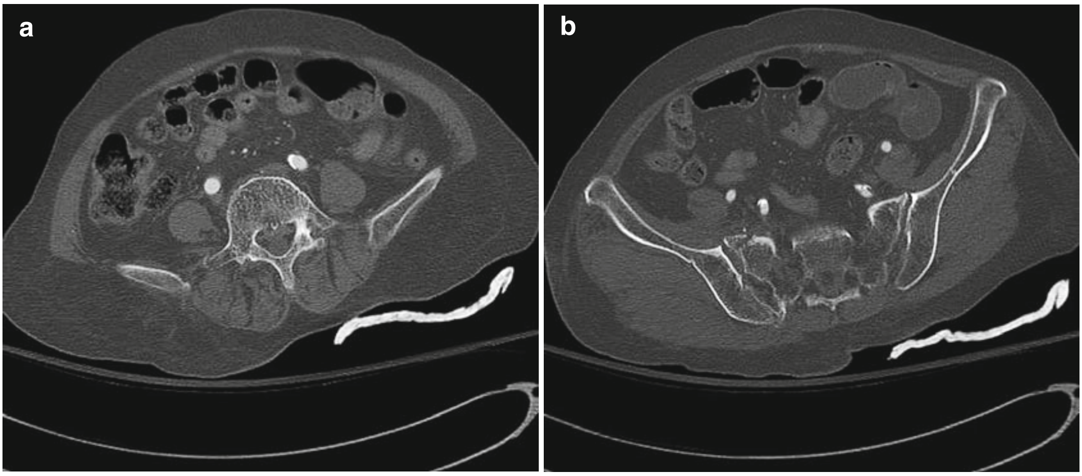


Fig. 28.5 (**a**) and (**b**) Axial CT sequences transverse fracture at the tip showing right L5 transverse process fracture with associated right sacral ala fracture

addressed. Hypotension in light of brachycardia suggests neurogenic shock, which should be treated with intravenous fluids, vasopressors, and/or inotropic agents. Rarely does a low lumbar fracture cause spinal cord injury since the cord ends at the L1 level, but studies have shown that there is a 5–15 % chance of noncontiguous spinal injury so patients with low lumbar injuries should be evaluated for other levels of spinal injury as well [15, 16]. Once the patient is hemodynamically stable, a primary examination includes log rolling the patient while the cervical spine is stabilized, and the spine is assessed for wounds, tenderness, and step-off of the spinous processes.

The neurologic examination involves assessment of motor, sensation, reflex, and rectal tone statuses. Usage of the American Spinal Injury Association (ASIA) form to record the exam helps to keep track of the progress of the patient. Upper motor lesions associated with cervical or thoracic injuries can manifest as abnormal Babinski, Hoffman, or clonus tests and show diffuse significant neurologic compromise including frank paralysis. Spinal shock, which usually takes 24–48 h to resolve, may mask the true deficits, therefore, the bulbocavernosus reflex must be checked because it is the first to return due to it being the lowest cord mediated reflex. The bulbocavernosus reflex can be affected in a lower motor neuron injury involving the sacral nerve roots so one might confuse this injury for spinal shock [17].

Three neurological injuries pertaining to low lumbar fractures are cauda equina syndrome, conus medullaris syndrome, and most commonly nerve root injuries. Generally, cauda equina syndrome portrays lower motor neuron deficits whereas conus medullaris syndrome presents with combined upper and lower motor neuron deficits. A useful distinction between the two conditions is that cauda equina syndrome may have asymmetric involvement, whereas conus medullaris syndrome tends to have a bilateral presentation. Nerve root injuries result in functional deficits involving that respective myotome and dermatome. This can be caused by direct compression of the nerve by the fracture fragments or entrapment of the nerve with the laminar fracture. Lamina fractures, especially in burst fractures, can lead to nerve root entrapment or dural tears.

Certain fracture types can also be related with injuries in other anatomic areas. Flexion-distraction injuries can be associated with abdominal organ injury. Fractures to the transverse processes, especially L5, may be caused by cephalad displacement of an unstable pelvic fracture (Fig. 28.5a, b).

Along the line of initial evaluation, the National Acute Spinal Cord Injury Studies (NASCIS) have recommended the usage of high-dose methylprednisolone in patients sustaining spinal cord injury from blunt trauma, as long as not more than 8 h have passed since the time of injury [18–21]. The rationale is to lessen the damage from inflammatory processes and oxidation. The current recommendations include administration of 30 mg/kg intravenous bolus followed by 5.4 mg/kg for 23 h if presenting within 3 h of the injury or for 48 h if presenting between 3 and 8 h from time of injury. Although the studies were randomized controlled trials, many have questioned their methodologies and clinical effectiveness, not to mention the reported complications of wound infection and pulmonary impairment in the steroid group. There are studies refuting the effectiveness of high-dose steroids in spinal cord injuries [22, 23]. Another limitation of high-dose steroid is its ineffectiveness in lower motor neuron deficits, which is likely to occur in low lumbar fractures. Since injuries to the low lumbar spine do not result in spinal cord injury, use of high-dose steroids for these injuries is not indicated by these studies, and surgeon discretion should be used.

28.7 Imaging

In the trauma setting, CT has overtaken conventional radiography as the modality of choice when it comes to spinal imaging. In fact, studies have shown that CT scans are more sensitive, accurate, faster, and cost effective when assessing spinal trauma [24, 25]. The sagittal cuts display the integrity of the vertebral body and facet alignments. On the midsagittal view, the corresponding aspects of the vertebrae should line up well with each other to make nice arcing lines such as the anterior and posterior walls of the vertebral bodies, the spinolaminar junctions, and the posterior borders of the spinous processes. Moreover, the facets should line up on top of each other, resembling shingles. If the facets do not "shingle" over one another, that facet joint is either subluxated, perched, or dislocated. The axial views would show an "empty" or "naked" facet sign due to the incongruity of the facet joint. The axial views also show a more accurate estimation of canal compromise from a burst fracture.

When looking at an anteroposterior (AP) plain radiograph of the low lumbar spine, the surrounding soft tissue must be looked at for hematoma or soft tissue edema. The interspinous process and interpedicular distances should be similar, respectively. Increased interspinous distance suggests a flexion injury that might have affected the posterior ligamentous complex. Misalignment of the spinous processes on the AP view denotes a rotational injury. Increased interpedicular space signifies an axial compression injury such as a burst fracture.

The limitations with the aforementioned imaging techniques are their inabilities to display soft tissues and neural elements well. Hence, MRI scans can be used for further injury clarification. The T2-weighted fat-suppressed MRI series are very helpful in determining injuries to the soft tissues, neural elements, and bony structures, especially in occult cases by demonstrating bright signals. This series can distinguish between acute and chronic injuries such as in compression fractures. An acute fracture would show bright bony edema within the vertebral body, whereas a healed injury would not. In the past, a fat-suppressed T2-weighted sagittal sequence was shown to be highly sensitive and specific for assessing the posterior ligamentous complex injury [26]. However, recent studies have shown that the sensitivity and specificity for detecting PLC injury are lower than reported due to the difference in interpreting which structure is injured or not between a radiologist and intraoperative findings [27]. Hence, although the MRI scan can provide further information, care must be taken to not over-interpret the findings as being suggestive of more instability than actually exists.

28.8 Treatment

28.8.1 Nonsurgical Versus Surgical

Nonsurgical and surgical treatments are based on neurological and mechanical instabilities. Neurologic deficits secondary to fractures with ongoing neural compression suggest mechanical instability, and surgery is frequently recommended

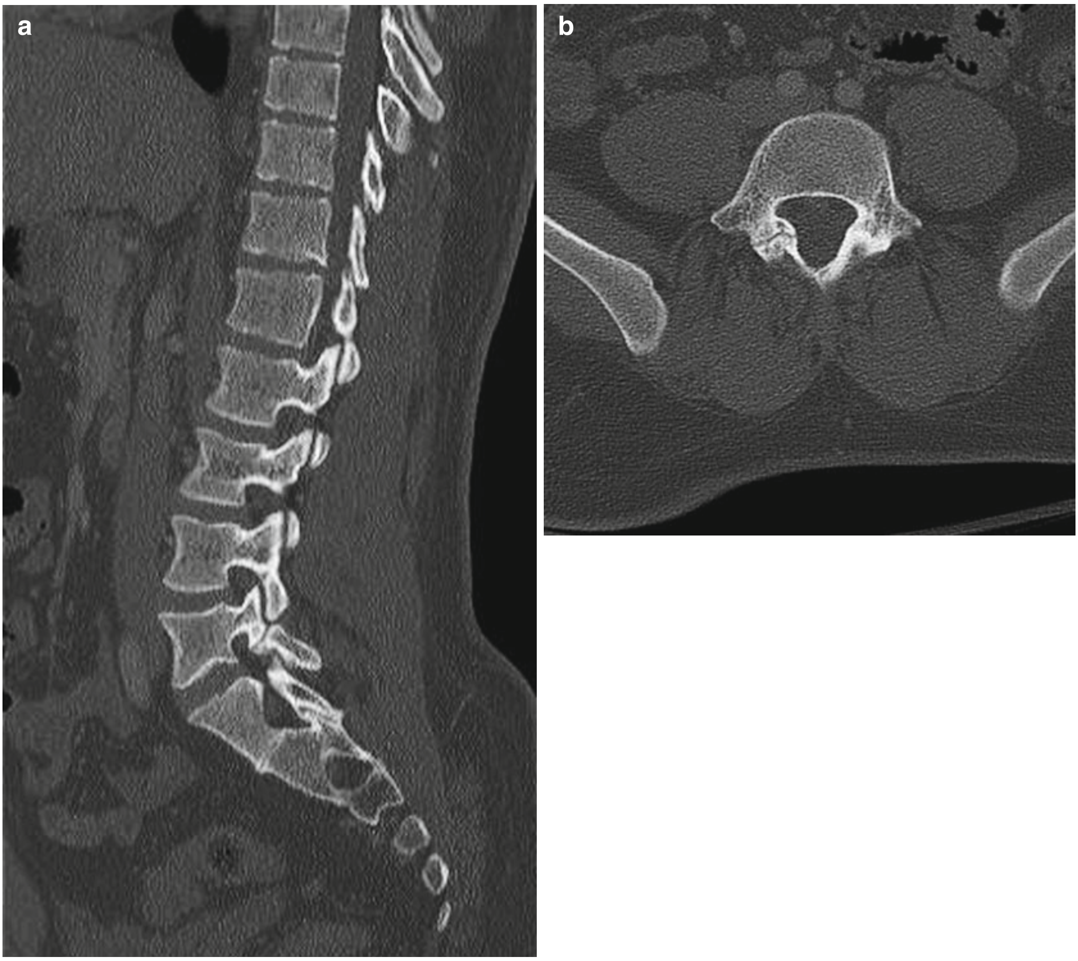


Fig. 28.6 (**a**) Sagittal and (**b**) axial CT views displaying right pars interarticularis fracture of L5

for decompression and stabilization. In the absence of neurologic deficits, however, the need for surgery is still debated because there is no widely accepted algorithm to predict mechanical instability. Although subject to interpretation, mechanical instability refers to injuries which have caused or will lead to sagittal or coronal deformity or any significant ligamentous disruption due to the inability of these ligaments to heal adequately.

28.8.2 Nonsurgical

In general, stable fractures involve isolated minor fractures such as transverse process fractures, lamina fractures, and pars fractures. These can be treated with pain control, activity modification, early mobilization, and bracing in the case of spondylolysis (Fig. 28.6a, b). A recent study, however, showed that bracing did not influence functional outcome after spondylolysis [28]. The exception is a lamina fracture with a traumatic dural tear that does not resolve with short-term bed rest. In this case, dural repair is indicated. Major fractures that are considered stable are certain compression fractures, burst fractures, and bony flexion-distraction fractures.

Traumatic compression fractures are generally stable due to the involvement of the anterior column only. It is hard to predict which will become unstable, but the commonly used criteria are more than 50 % vertebral body weight loss and more than 30° focal kyphosis [29, 30]. This is suggestive of significant PLC injury, which can be confirmed with an MRI and warrants surgery for stabilization if indicated. The need for surgical intervention for traumatic compression fractures is quite uncommon. Osteoporotic compression fractures can lead to similar findings in patients without significant trauma. Vertebral augmentation procedures, such as kyphoplasty or vertebroplasty, may be useful in both traumatic and osteoporotic compression fractures that are not healing appropriately or are causing debilitating pain. Nonoperative treatment of compression fractures includes pain control, activity modification, and early mobilization. A molded thoracolumbosacral orthosis (TLSO) with a leg extender for fractures caudal to L3 for 3 months can also be used, although the leg extender can be quite problematic in both fit and function. Serial radiographs are used to ensure that there is no further displacement or malalignment during the 3 months healing period.

The determination of the stability of burst fractures can be challenging. According to Denis' definition, all burst fractures should be unstable because, by definition, they involve at least two of the three columns. Studies, however, have shown that many burst fractures can be treated successfully nonoperatively as long as there are no progressive neurological deficits and the PLC is intact [31, 32]. As with compression fractures, the commonly stated criteria for instability in the absence of neurologic compromise and PLC disruption are vertebral height loss of more than 50 %, focal kyphosis more than 30°, and canal encroachment of more than 50 %. Despite these numbers, some studies have shown successful nonoperative treatment with bracing and early mobilization even with excessive canal compromise because of eventual bony resorption [31, 32]. A retrospective study (Seybold 1999) comparing operative versus nonoperative treatment of L3–L5 burst fractures showed that functional outcome between the two groups was similar, with a large percentage of the surgical group needing further surgery [33]. In that study, both groups had significant initial presentations in terms of mean canal compromise (34 % for nonoperative group versus 53 % for operative group), kyphosis, (−5.5° vs. +1.8°), and vertebral height loss (27.5–27.9 %). Because the surgical group did not present with any parameter far exceeding the aforementioned radiographical criteria for instability, it is uncertain whether they needed surgery to begin with. A limitation with the study is that it did not comment on the ligamentous integrity, which would have played a role in distinguishing stable and unstable fractures. Hence, the interpretation of stable and unstable burst fractures varies widely among surgeons. The most important criteria to establish the stability of these injuries and resultant need for surgery include the competency of the PLC, especially the facet joint complexes, and the sagittal alignment of the lumbosacral spine.

Flexion-distraction bony injuries can be treated nonsurgically as long as spinal alignment is appropriate and the patient is neurologically intact. Similar to the nonsurgical treatment of compression and burst fractures, nonoperative treatment of flexion-distraction injuries includes a rigid TLSO brace, pain control, and early mobilization. Standing radiographs in the brace should be obtained to ensure that there is no displacement when the patient is upright. Because these injuries are caused by flexion forces and are reduced with extension forces, supine imaging, including cross table x-rays, CT scans, and MRI scans, may appear misleadingly well aligned in unstable injuries. Serial radiographs are taken upright in the brace, and dynamic flexion and extension views may be obtained at the 3 months mark to confirm stability. Ligamentous flexion-distraction injuries require surgical intervention more aggressively than similar bony injuries, and care should be taken to evaluate the spinal stability and healing potential in these variants.

28.8.3 Surgical

Low lumbar fracture patterns that lead to mechanical instability, worsening neurologic injury, or unacceptable spinal alignment may require surgical intervention. Injuries that lead to dislocation are obviously unstable regardless if there is neurologic compromise or not. As stated, compression and burst fractures that result in neurologic compromise, PLC disruption, and excessive kyphotic deformity warrant surgery, as do ligamentous Chance variant injuries and displaced extension injuries in AS or DISH patients.

Surgical goals include reduction of malalignment, mechanical stabilization, and neurologic decompression. Stabilization with instrumentation can be performed through an anterior, posterior, or combined approach. According to a cadaveric biomechanical study, circumferential fixation provides the strongest fixation, followed by posterior fixation with anterior strut grafting, then posterior fixation alone, and lastly anterior fixation with strut grafting [34]. Despite this finding, the aim is to avoid morbidity and mortality as much as possible and yet still obtain optimal results. Unless anterior reconstruction and/or decompression is necessary, operative risks are still relatively high even when the anterior approach is done with minimally invasive approaches [35].

28.8.3.1 Posterior Approach

Posterior surgery, including decompression and instrumented fusion, is useful in low lumbar fractures especially those requiring stabilization without significant neurologic deficit. The posterior approach to the low lumbar spine is commonly performed, and in general surgeons and operating room staff are more comfortable with this approach versus the anterior or lateral approaches. In the absence of excessive anterior column failure or anterior neurologic compression, posterior stabilization with instrumentation is the preferred approach. The introduction of rigid fixation with pedicle screws and rods theoretically allows for shorter fixation, therefore obviating the need to include other mobile levels and preserving motion. Short-segment constructs, which include one level above and below the injured vertebra, should be approached with caution as they can have high failure rates if the right indications are not met [36, 37]. Even with the addition of cross-links in hopes to minimize the length of posterior fixation in an unstable burst fracture model, construct stiffness was not restored to preinjury level, except for lateral bending [38]. For that reason, long posterior constructs alone or short-segment posterior instrumentation with anterior reconstruction, to minimize the stress on the posterior short-segment fixation, should be used for highly unstable injury patterns. With fractures of L4 or L5, fixation to the sacrum is important in reestablishing lordosis. Sacral fixation is generally easily done posteriorly as opposed to anteriorly due to the overlying iliac vessels. Fixation involving the lumbosacral junction must be rigid to avoid pseudoarthrosis and loss of sagittal alignment, and extension to the S2 segment or the ilium may be useful to further backup and protect the sacral fixation.

28.8.3.2 Anterior Approach

Anterior surgery for low lumbar injuries is useful for decompression of a highly compromised spinal canal and to provide further stabilization via anterior column reconstruction in highly unstable injuries. Numerous studies have shown excellent results in terms of fusion rate and neurologic improvement with anterior decompression, reconstruction, and instrumented stabilization alone with certain burst fractures [39, 40]. A comminuted vertebral body with more than 50 % vertebral height loss may need a corpectomy and replacement with structural bone graft or structural cages with bone graft in order to reconstruct the failed anterior column. It has also been shown that without instrumented stabilization, anterior reconstruction with structural graft alone has poor outcomes with restoration of sagittal alignment [41]. Thus, if the anterior approach is the primary treatment, consider augmenting the structural graft with anterior instrumentation. If not, then posterior instrumentation should be performed to stabilize the anterior graft.

28.8.3.3 Combined Approach

Although there is increased morbidity with the combined anterior and posterior approach, this approach may be required in highly unstable injuries involving all three columns. Examples include burst fractures with more than 50 % loss of vertebral height and PLC disruption with significant canal narrowing.

28.8.3.4 Decompression

The need for surgical decompression after low lumbar trauma is based on the amount of neurologic deficit and ongoing canal compromise. The widely accepted indication to perform decompression is neurological deficit from neural impingement. The method of decompression, however, is as controversial as when to decompress. Direct decompression involves removing the offending structure, whereas indirect decompression is via ligamentotaxis to restore anatomic alignment, which ultimately puts the neural elements back to their original places. Commonly, direct decompression is performed in light of neurological deficits from neural impingement. In the low lumbar spine where the nerve roots are more forgiving than the spinal cord in terms of neural manipulation, decompression can be done anteriorly or posteriorly. This is based on the approach warranted by the fracture pattern and surgeon preference. In patients with significant canal compromise without neurologic deficit, patients may not require prophylactic decompression due to eventual remodeling of the involved vertebrae.

28.8.3.5 Postoperative Care

Postoperative management is based upon the kind of surgery performed, surgeon preference, and the patient's concomitant injuries. Physical therapy is initiated as early as feasible. Based on the stability of the fixation determined during surgery, postoperative bracing may or may not be required. Serial standing radiographs are obtained, and fracture healing generally occurs 3 months post injury.

28.8.3.6 Surgical Complications

Postoperative complications include neurologic decline, nonunion, hardware failure leading to loss of fixation, dural tears, and infection. Changes in neurologic status can result from migration of an unstable fracture in an asleep patient or could be due to malpositioned hardware such as inappropriately placed pedicle screws causing root impairment. Dural tears can be controlled with meticulous surgical techniques. Hardware failure can either be due to inadequate preoperative planning in terms of length and location of the construct or from poor bone quality. Likewise, nonunion and infection can be attributed to either the surgeon's techniques or patient's predisposition. In order to minimize these potential mishaps, a surgeon has to develop a well-thought-out plan preoperatively and execute it appropriately. Complication rates are higher in post-trauma patients versus patients undergoing elective surgery due to the inability to obtain preoperative medical optimization, associated injuries, and a higher catabolic state due to the traumatic injuries.

28.9 Summary

Although its incidence is less common than thoracolumbar trauma [42], low lumbar trauma shares similar concepts in terms of diagnosis and treatment. There are, however, unique characteristics relating to the lumbar spine's lordotic alignment, increased canal to neural element ratio, and lumbosacral junction that predispose it to different fracture patterns, treatments, and outcomes as compared to its counterpart. There is a lack of studies evaluating low lumbar trauma, and further exploration in this area may be useful.

Questions

1. The posterior ligamentous complex is thought to act as a tension band. Which of the following is not considered part of the complex?
   (a) Interspinous ligament
   (b) Ligamentum flavum
   (c) Supraspinous ligament
   (d) Posterior longitudinal ligament
   Preferred answer: (d) The complex consists of the interspinous and supraspinous ligaments, ligamentum flavum, facet capsules.
2. According to a biomechanical study comparing fixation techniques, which of the following demonstrates the least amount of rigidity?
   (a) Circumferential fixation
   (b) Anterior fixation with strut grafting
   (c) Posterior fixation alone
   (d) Posterior fixation with anterior strut grafting

Preferred answer: (b) Circumferential fixation provides the strongest fixation, followed by posterior fixation with anterior strut grafting, then posterior fixation alone, and lastly anterior fixation with strut grafting.

3. In low lumbar fractures, which of the following neurological deficits is most likely to occur due to its anatomy?
   (a) Central cord syndrome
   (b) Brown-Sequard syndrome
   (c) Conus medullaris syndrome
   (d) Nerve root impingement

   Preferred answer: (d) The spinal cord ends at the L1 level, and nerve roots make up the content of the spinal canal distal to that. Due to this anatomy, low lumbar fractures tend to affect the nerve roots more so than the spinal cord.

4. Which imaging modality is best used to detect subtle posterior ligamentous complex injury?
   (a) Conventional radiographs
   (b) CT
   (c) MRI
   (d) Bone scan

   Preferred answer: (c) Up to date, MRI is still the preferred modality used to assess PLC injury in light of normal radiographic and CT findings.

References

1. Ponnappan RK, Lee JY (2008) Thoracolumbar trauma. In: Fischgrund J (ed) Orthopaedic knowledge update 9. American Academy of Orthopaedic Surgeons, Rosemont
2. Browner B, Jupiter J, Levine A, Trafton P, Krettek C (2009) Low lumbar fractures. In: Skeletal trauma, 4th ed. Saunders, Philadelphia
3. Zindrick MR, Wiltse LL, Doornik A et al (1987) Analysis of the morphometric characteristics of the thoracic and lumbar pedicles. Spine 12:160–166
4. Krag MH, Weaver DL, Beynnon BD et al (1988) Morphometry of the thoracic and lumbar spine related to transpedicular screw placement for surgical spinal fixation. Spine 13:27–32
5. Denis F (1983) The three-column spine and its significance in the classification of acute thoracolumbar spinal injuries. Spine 8:817–831
6. Dai LD (2002) Low lumbar spinal fractures: management options. Injury 33:579–582
7. Panjabi MM, White AA (1980) Basic biomechanics of the spine. Neurosurgery 7:76–93
8. Holdsworth FW (1963) Fractures, dislocations and fracture – dislocations of the spine. J Bone Joint Surg Br 45:6–20
9. Panjabi MM, Oxland TR, Kifune M et al (1995) Validity of the three-column theory of thoracolumbar fractures. A biomechanic investigation. Spine 20:1122–1127
10. Denis F (1984) Spinal instability as defined by the three-column spine concept in acute spinal trauma. Clin Orthop Relat Res 189:65–76
11. Mirza SK, Mirza AJ, Chapman JR et al (2002) Classifications of thoracic and lumbar fractures: rationale and supporting data. J Am Acad Orthop Surg 10:364–377
12. Wood KB, Khanna G, Vaccaro AR et al (2005) Assessment of two thoracolumbar fracture classification systems as used by multiple surgeons. J Bone Joint Surg Am 87:1423–1429
13. Vaccaro AR, Lehman RA, Hurlbert RJ et al (2005) A new classification of thoracolumbar injuries: the importance of injury morphology, the integrity of the posterior ligamentous complex, and neurologic status. Spine 30:2325–2333
14. Vaccaro AR, Lim MR, Hurlbert RJ et al (2006) Surgical decision making for unstable thoracolumbar spine injuries: results of a consensus panel review by the Spine Trauma Study Group. J Spinal Disord Tech 19:1–10
15. Henderson RL, Reid DC, Saboe LA (1991) Multiple noncontiguous spine fractures. Spine 16:128–131
16. Calenoff L, Chessare JW, Rogers LF et al (1978) Multiple level spinal injuries: importance of early recognition. AJR Am J Roentgenol 130:665–669
17. Herkowitz H, Garfin S, Eismont F, Bell G, Balderston R (2011) Thoracic and lumbar spinal injuries. In: Rothman-Simeone the spine, 6th ed. Saunders, Philadelphia
18. Bracken MB, Shepard MJ, Collins WF et al (1990) A randomized, controlled trial of methylprednisolone or naloxone in the treatment of acute spinal-cord injury: results of the Second National Acute Spinal Cord Injury Study. N Eng J Med 322:1405–1411
19. Bracken MB, Shepard MJ, Collins WF et al (1992) Methylprednisolone or naloxone treatment after acute spinal cord injury: 1-year follow-up data: results of the second National Acute Spinal Cord Injury Study. J Neurosurg 76:23–31
20. Bracken MB, Shepard MJ, Holford TR et al (1997) 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. National Acute Spinal Cord Injury Study. JAMA 277:1597–1604
21. Bracken MB, Shepard MJ, Holford TR et al (1998) Methylprednisolone or tirilazad mesylate administration after acute spinal cord injury: 1-year follow up. Results of the third National Acute Spinal Cord Injury randomized controlled trial. J Neurosurg 89:699–706

22. George ER, Scholten DJ, Buechler CM (1995) Failure of methylprednisolone to improve the outcome of spinal cord injuries. Am Surg 61:659–664
23. Prendergast MR, Saxe JM, Ledgerwood AM et al (1994) Massive steroids do not reduce the zone of injury after penetrating spinal cord injury. J Trauma 37:576–579
24. Berry GE, Adams S, Harris MB et al (2005) Are plain radiographs of the spine necessary during evaluation after blunt trauma? Accuracy of screening torso computed tomography in thoracic/lumbar spine fracture diagnosis. J Trauma 59:1410–1413
25. Inaba K, Munera F, McKenney M (2006) Visceral torso computed tomography for clearance of the thoracolumbar spine in trauma: a review of the literature. J Trauma 60:915–920
26. Lee HM, Kim HS, Kim DJ et al (2000) Reliability of magnetic resonance imaging in detecting posterior ligament complex injury in thoracolumbar spinal fractures. Spine 25:2079–2084
27. Vaccaro AR, Rihn JA, Saravanja D et al (2009) Injury of the posterior ligamentous complex of the thoracolumbar spine: a prospective evaluation of the diagnostic accuracy of magnetic resonance imaging. Spine 34:E841–E847
28. Klein G, Mehlman CT, McCarty M (2009) Nonoperative treatment of spondylolysis and grade 1 spondylolisthesis in children and young adults: a meta-analysis of observational studies. J Pediatr Orthop 29:146–156
29. Day B, Kokan P (1977) Compression fractures of the thoracic and lumbar spine from compensable injuries. Clin Orthop Relat Res 124:173–176
30. Tonbul M, Yilmaz MR, Ozbaydar MU et al (2008) Long term results of conservative treatment for thoracolumbar compression fractures. Acta Orthop Traumatol Turc 42:80–83
31. Cantor JB, Lebwohl NH, Garvey T et al (1993) Nonoperative management of stable thoracolumbar burst fractures with early ambulation and bracing. Spine 18:971–976
32. Wood K, Buttermann G, Mehbod A et al (2003) Operative compared with nonoperative treatment of a thoracolumbar burst fracture without neurological deficit. A prospective, randomized study. J Bone Joint Surg Am 85:773–781
33. Seybold EA, Sweeney CA, Fredrickson BE et al (1999) Functional outcome of low lumbar burst fractures: a multicenter review of operative and nonoperative treatment of L3-L5. Spine 24:2154
34. Kallemeier PM, Beaubien BP, Buttermann GR et al (2008) In vitro analysis of anterior and posterior fixation in an experimental unstable burst fracture model. J Spinal Disord Tech 21:216–224
35. Olinger A, Hildebrandt U, Mutschler W et al (1999) First clinical experience with an endoscopic retroperitoneal approach for anterior fusion of the lumbar spine fractures from T12 to L5. Surg Endosc 13:1215–1219
36. Alanay A, Acaroglu E, Yazici M et al (2001) The effect of transpedicular intracorporeal grafting in the treatment of thoracolumbar burst fractures on canal remodeling. Eur Spine J 10:512–516
37. Tezeren G, Kuru I (2005) Posterior fixation of thoracolumbar burst fracture: short segment pedicle fixation versus long segment instrumentation. J Spinal Disord Tech 18:485–488
38. Wahba GM, Bhatia N, Bui CN et al (2010) Biomechanical evaluation of short-segment posterior instrumentation with and without crosslinks in a human cadaveric unstable thoracolumbar burst fracture model. Spine 35:278–285
39. Kaneda K, Taneichi H, Abumi K et al (1997) Anterior decompression and stabilization with the Kaneda device for thoracolumbar burst fractures associated with neurological deficits. J Bone Joint Surg Am 79:69–83
40. Sasso RC, Best NM, Reilly TM et al (2005) Anterior only stabilization of three column thoracolumbar injuries. J Spinal Disord Tech 18:S7–S14
41. McAfee PC, Bohlman HH, Yuan HA (1985) Anterior decompression of traumatic thoracolumbar fractures with incomplete neurological deficit using a retroperitoneal approach. J Bone Joint Surg Am 67:89–104
42. Gertzbein SD (1992) Scoliosis Research Society. Multicenter spine fracture study. Spine 17:528–540

Sacral Spine, Pelvis, and Pelvic Ring 29

Megan Brady, Stephen Tolhurst, and Timothy Moore

29.1 Anatomy

The sacrum is confined and supported by both bony and ligamentous restraints. It consists of five fused, nonarticulated vertebral segments (Fig. 29.1). The sacrum also has three articulations: one with the L5 vertebral body through the L5–S1 disk complex and two involving a true synovial joint with the innominate bones bilaterally (sacroiliac joints). The coccyx is the vestigial "tail" off the sacrum's inferior 5th segment. The sacrum assumes a kyphotic posture of 45–60° to the perpendicular axis of the body. The ligamentous restraints attached to the sacrum are considered the strongest ligaments in the body. The bilateral sacrospinous ligaments anchor each side of the sacrum to the ischial spine of the pelvis, while the sacrotuberous ligaments anchor the sacrum to the ischial tuberosity. The long and short posterior sacroiliac ligaments prevent rotational forces of the hemipelvis in lateral compression injuries that will be discussed later. Because of these stout ligamentous restraints, the sacrum usually incurs bony injury before pure ligamentous or sacroiliac joint dislocations occur.

Posteriorly, the sacrum is covered by the multifidus musculature, lumbosacral fascia, and relatively thin layer of subcutaneous tissue and skin. The sacrum acts as a roof to important neurovascular structures that lie on its anterior border. The L5 nerve root runs just lateral to the L5–S1 disk and over the top of the sacral ala and can be injured with any force directed to this area. The lumbosacral plexus controls urinary continence and micturition, fecal continence and defecation, and sexual function. The internal iliac artery and vein course just anterior to the sacrum bilaterally, while sigmoid colon and rectum lie in the midline and can be damaged in high-energy mechanisms.

29.2 History and Physical Examination

The evaluation of a patient with a sacral injury begins with the mechanism of injury. The sacrum can be injured by direct blow or through indirect forces on the pelvic ring. Strict adherence to ATLS protocols is imperative. The physical examination and inspection of the patient may provide clues to a pelvic ring and/or sacral injury. This injury can present with a wide spectrum of neurologic deficits. A thorough neurologic evaluation must be performed including rectal examination for sphincter testing and perianal sensation. This includes individual root testing for L5 with ankle dorsiflexion and toe extension, lateral thigh, lower leg, and dorsum of foot sensation. An isolated L5 deficit can indicate a fracture involving

M. Brady, M.D. • T. Moore, M.D. (✉)
Department of Orthopaedic Surgery, MetroHealth Medical Center H6, 2500 MetroHealth Drive, Cleveland, OH 44109, USA
e-mail: tmoore@metrohealth.org

S. Tolhurst, M.D.
Texas Back Institute, 6020 W. Parker Rd, Suite 200, Plano, TX 75093, USA
e-mail: stolhurst@texasback.com

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_29, © Springer-Verlag Berlin Heidelberg 2014

Fig. 29.1 Dorsal and ventral views of sacrum (Photograph courtesy of Hamman-Todd Museum, Cleveland, OH)

the sacral ala. Testing of the S1 nerve root involves ankle plantarflexion, lateral thigh, posterior lower leg, and plantar foot sensation. Complete below the knee paralysis may indicate severe injury to the lumbosacral plexus from a displaced sacral fracture. Cauda equina syndrome may indicate a fracture dislocation through S1 or S2. Instability with rocking the pelvis by applying pressure on bilateral anterior superior iliac spines may indicate an unstable injury pattern with or without neurological injury. Inspection of the skin overlying the sacrum and perineum may reveal bogginess, step-offs, crepitus, ecchymosis, or penetrating wounds.

29.3 Investigations

Initially, a standard trauma AP view of the pelvis should be obtained. Signs on plain radiographs that should alert the physician to a possible sacral

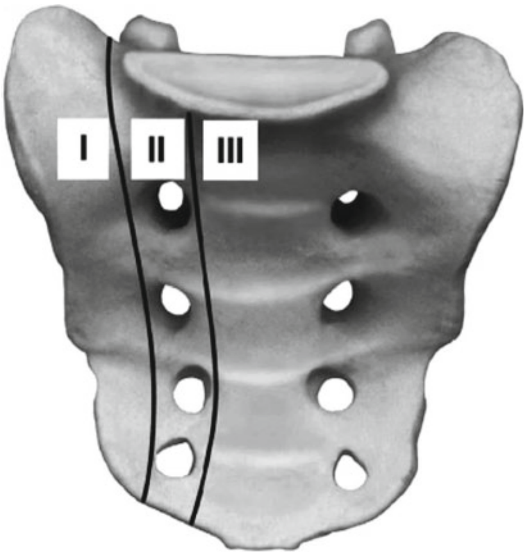


Fig. 29.2 Denis [2] classification of sacral fractures. *Zone I* fractures occur lateral to the sacral foramina, *zone II* fractures involve the sacral foramina, *zone III* fractures occur medial to the sacral foramina (Mehta et al. [3]. © 2006 AAOS)

fracture include transverse process fractures at L4 or L5 and pelvic ring disruptions. If there is significant suspicion for a sacral fracture, plain films should be promptly followed with advanced imaging (typically computed tomography [CT]), as only 30 % of sacral fractures are demonstrated on plain films [1]. In the author's experience, 1–2 mm cuts as well as coronal and sagittal reconstruction views are helpful in identifying fracture type and planning surgical treatment. Magnetic resonance imaging (MRI) is useful particularly when neural injury is suspected as it more accurately demonstrates neural compression and injury than CT.

29.4 Classification Systems

Multiple classification systems have been developed for sacral fractures and generally fall into one of three areas: those associated with pelvic ring injuries, those intrinsic to the sacrum, and those involving the lumbosacral junction. The Denis classification [2] is commonly applied to describe the morphometry of sacral fractures in the first two categories and can be used in conjunction with pelvic ring fracture classification systems (Letournel and Tile systems). The Denis classification describes three zones of injury, with higher grades corresponding to an increased incidence of neurologic injury (Fig. 29.2). Zone I injuries are in the sacral ala lateral to the foramina and are the most common type, with a 6 % rate of neurologic injury typically affecting the L4 and L5 nerve roots. Zone II fractures are vertical with the fracture line entering the sacral foramina. This is the second most common type with a 28 % incidence of neurologic injury, most commonly involving the L5, S1, or S2 nerve roots. Vertical shear in a type II fracture implies instability and may require operative treatment as malunions in this region lead to poor functional outcomes. Zone III fractures lie medial to the foramina in the spinal canal. This is the least common fracture type but is associated with a 57 % rate of neurologic injury. Zone III injuries with a transverse component have also been further subclassified by Roy-Camille [4] and Strange-Vongsen and Lebech [5] (Fig. 29.3) according to the degree of kyphosis and translation of the fracture. Type 1 demonstrates only kyphosis, type 2 demonstrates kyphosis with incomplete anterior sacral translation, and type 3 fractures have complete sacral displacement. Type 4 fractures comprise a broad category with any of these types in conjunction with comminution of the S1 body. Complex type III fracture patterns can also be graded according to the letter of the alphabet they resemble (i.e., U, H, Lambda, and T) (Fig. 29.4).

Injury at the lumbosacral junction is typically associated with very high-energy trauma since the lumbosacral ligaments are very strong. These can be viewed as unilateral or bilateral facet dislocations. These fractures are classified by the Isler system [5]. Type I fractures are vertical fractures lateral to the L5–S1 facet and are unlikely to affect lumbosacral stability. Type II fractures traverse through the L5–S1 facet and are further subclassified as either extra-articular fractures of the lumbosacral junction or facet dislocations with variable displacement. Type III fractures cross into the neural foramen medial to the L5–S1 facet joint, are inherently unstable, and often require operative stabilization.

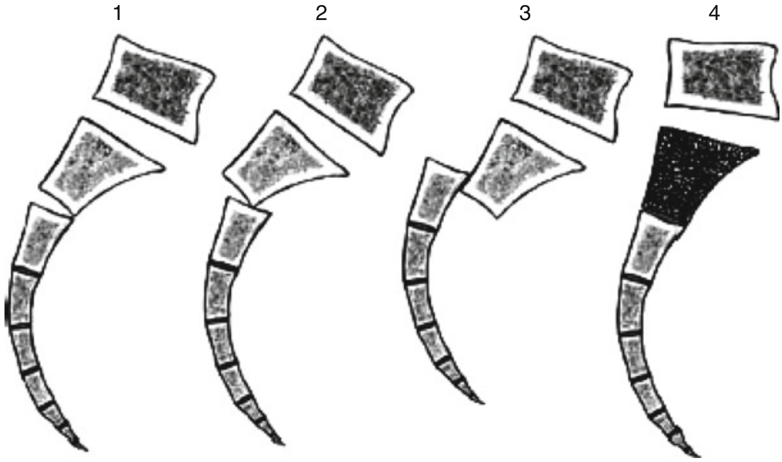


Fig. 29.3 Modification of Denis [2] zone III fractures by Roy-Camille [4] and Strange-Vongsen [5]. *Type 1* fractures have minimal kyphosis, *type 2* fractures are kyphotic with translation, *type 3* fractures involve fractures with dislocations, *type 4* fractures involve severe comminution (Mehta et al. [3]. © 2006 AAOS)

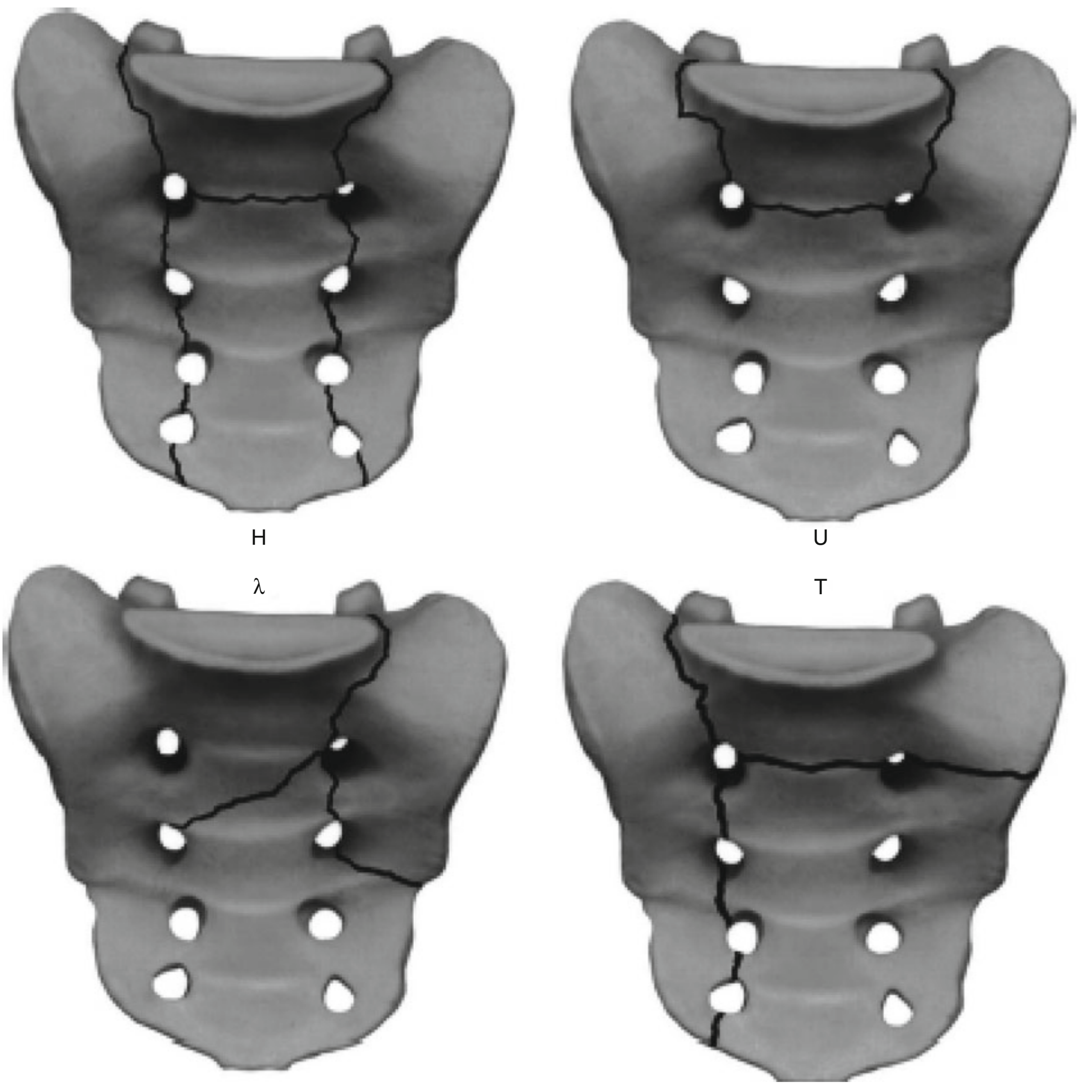


Fig. 29.4 Modification of Denis [2] zone III fractures by Roy-Camille [4] and Strange-Vongsen [5]. *H*, *U*, *λ*, and *T* fractures

29.5 Treatment Options

Initial evaluation of a patient with a traumatic sacral fracture should follow an appropriate ATLS workup and consideration of other injuries as the mechanism of injury is frequently high energy. Mehta et al [3]. recommended that sacral fractures should be classified into one of four basic clinical scenarios upon presentation: (1) fractures with associated pelvic ring injuries, (2) fractures with associated lumbosacral facet injury, (3) fractures with associated lumbosacral dislocation, and (4) fractures with neurologic injury and persistent cauda equina or spinal cord compression. Indications for nonoperative treatment are poorly delineated in the literature. In general, authors advocate operative management for fractures with significant soft tissue compromise, incomplete neurologic deficits with evidence of compression, and significant lumbosacral ligament disruption causing significant pelvic ring instability [6].

In the case of sacral fractures with associated pelvic ring injuries, initial assessment centers on evaluation of whether the pelvic ring is stable or unstable and the overall constellation of injuries with which the patient presents. When the pelvis is significantly unstable, temporary stability may be achieved with use of a pelvic binder or wrapping a sheet about the pelvis. If these techniques are inadequate, external fixation or pelvic clamp placement are appropriate temporizing operative options. If hemodynamic instability without other obvious cause persists, acute angiographic embolization of bleeding vessels should follow promptly thereafter to minimize blood loss. Some authors also advocate open packing of the retroperitoneal space to control bleeding. Evaluation of pelvic ring injuries as well as surgical techniques for anterior stabilization is beyond the scope of this chapter and discussed elsewhere; however, it should be noted that the anterior component of pelvic ring injuries should be treated before addressing the posterior pelvic ring and associated sacral fracture as this confers improved posterior stability, particularly when the patient is prone [3, 6]. Although multiple techniques for posterior stabilization of sacral fractures have been described, sacroiliac screws are most commonly performed as both vertical sacral fractures and sacroiliac joint disruptions are amenable to this technique and it can be safely performed percutaneously [7]. Horizontal or more complex fracture types are not amenable to this technique and require a more complex construct such as iliolumbar fixation.

In the case of sacral fractures with neurologic injury or persistent compression, debate exists as to the overall benefit of decompression as neurologic improvement rates of up to 80 % have been reported with both operative and nonoperative management. This confusion is largely due to the poor quality of the literature in this area. Incomplete neurologic injury or intact neurologic function more frequently necessitates operative management than those with complete neurologic injury. Decompression of sacral roots is generally performed in conjunction with fracture stabilization as decompression alone frequently worsens instability [6]. Although the role of decompressive surgery is debated, it is indicated when neurologic deficits are noted with objective evidence of neural compression [3]. Additionally, decompression should ideally be performed at the time of stabilization as late decompression is more difficult due to epineural fibrosis and increased scarring in the central canal [8]. Although timing of decompression and stabilization is frequently dictated by the severity of associated injuries, prompt surgical treatment allows for early mobilization and has been linked to improved outcomes. Routt reported that in Denis zone I–II fractures, surgical delays >5 days post injury were associated with less accurate closed reduction [7]. Additionally, Denis [4] reported worse outcomes in patients with neurologic injury treated >2 weeks post injury.

Sacral fractures associated with lumbosacral junction injuries frequently require operative treatment. Signs of instability such as rotational malalignment, anterolisthesis at the lumbosacral junction, or facet dislocation are typically treated with surgical stabilization. Multiple methods have been described for stabilization of these injuries, but iliolumbar fixation with lower lumbar pedicle screw and iliac screw fixation is biomechanically most stable [9]. Additionally, the surgical exposure allows easy access for neurologic decompression if indicated. Frank lumbosacral dislocation with bilaterally dislocated L5–S1 facets is a rare but severe injury and can occur in conjunction with multiple sacral fracture patterns. U-shaped sacral fractures are a similar injury but represent a dislocation of the sacrum from the pelvis. Although the necessity of surgery in these fracture patterns is clear, multiple treatments have been proposed for this rare injury.

29.6 Summary

Traumatic sacral fractures can present as a solitary injury, but are frequently observed in association with a pelvic ring injury or lumbosacral facet injury in a multiply injured patient. Plain films are inadequate to assess most sacral fractures, and concern for this injury should prompt advanced imaging (CT or MRI). Neurologic injury commonly occurs, particularly with more medial fractures (Denis zone II and III). When clinically feasible, early surgical decompression and fracture stabilization is appropriate for patients who have objective evidence of neural compression and a neurologic deficit as epinural fibrosis may diminish the benefit of late decompression. Multiple forms of skeletal fixation exist. Sacroiliac screws are appropriate for most vertical sacral fractures without significant comminution. For comminuted sacral fractures or lumbosacral facet injuries/dislocations, lumbopelvic fixation affords the greatest biomechanical stability, and the surgical exposure allows for direct neurologic decompression.

29.7 Pelvis and Pelvic Ring

Injuries to the pelvic ring typically result from high-energy trauma and are associated with bony and soft tissue injuries of the pelvis. These injuries may be associated with visceral and urogenital trauma, as these structures are all located within the pelvis. Neurovascular injuries to the lower extremities can also occur with pelvic ring disruptions. The incidence of neurologic injury following pelvic ring injury is approximately 21 %, seen primarily in unstable injuries. The overall mortality rate associated with unstable pelvic ring injury ranges between 8 and 11 % [10–13].

The initial management of patients with acute pelvic ring disruption revolves around stabilization of the pelvic ring. Patients that survive the initial trauma may have morbidity associated with neurovascular injuries, intra-abdominal trauma, or urogenital trauma. Pelvic ring deformity may be a cause of chronic pain. The treatment goal with high-energy pelvic ring disruptions is to treat life-threatening injuries and stabilize the pelvis in an anatomic position to avoid complications associated with pelvic malalignment or nonunion.

29.8 Anatomy of the Pelvis

The bony pelvis consists of two innominate bones and the sacrum (Fig. 29.5). Each innominate bone consists of three fused bones; the pubic, ilium, and ischium. The ilium comprises the superior portion of the innominate, with the ischium making up the inferior portion. The pubis extends medially from each acetabulum, creating the anterior aspect of the pelvis.

The integrity of the pelvic ring depends upon the surrounding soft tissues, as the bones offer only minor intrinsic stability. The ligaments of the pelvis provide stability of the ring and include the anterior and posterior sacroiliac ligamentous complexes and the ligaments of the pelvic floor (sacrospinous and sacrotuberous ligaments). The pubic symphysis is the fibrocartilaginous joint in the anterior portion of the pelvis, joining the two pubic bones of either hemipelvis. This joint is supported by the pubic ligaments.

The anterior and posterior sacroiliac ligaments play a major role in pelvic stability. The posterior sacroiliac ligaments exert a tension-band effect on the pelvic ring. The iliolumbar ligaments attach the transverse processes of the fifth lumbar body to the iliac crests, enhancing the tension-band effect. The posterior sacroiliac ligaments are among the strongest ligamentous structures in the human body.

The sacrospinous and sacrotuberous ligaments (located in the pelvic floor) also provide stability. The sacrospinous ligament originates from the lateral aspect of the sacrum and attaches to the ischial spine. This ligament acts to resist external rotation of the hemipelvis. The sacrotuberous ligament extends from the posterior sacroiliac

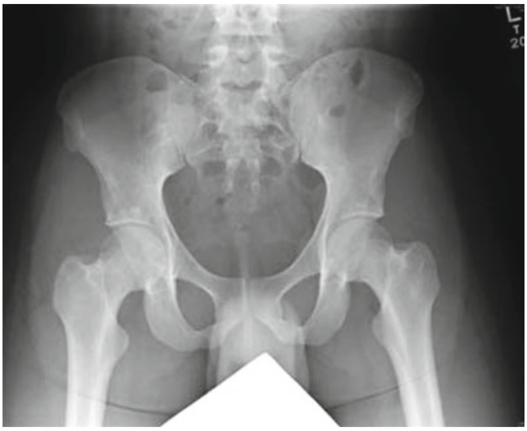


Fig. 29.5 AP radiography of normal pelvis

ligaments and inserts onto the ischial tuberosity, acting to counteract shearing forces in the vertical direction as well as resist external rotation.

It is important to note that major blood vessels and neural structures are located within the pelvis. The iliac, obturator, and femoral arteries all lie within close proximity to the bony pelvis. The lumbosacral plexus also lies along the sacrum. The femoral, sciatic, and obturator nerves are all at risk for injury following pelvic ring disruption. Additionally, the bladder and urethra also reside near the anterior aspect of the pelvic ring. Long-term sequelae of pelvic ring injuries include paresthesias, muscle weakness, and sexual dysfunction resulting from injury to nearby anatomic structures.

29.9 Radiographic Evaluation

The standard anteroposterior (AP) (Fig. 29.6) radiograph of the pelvis provides limited information regarding the integrity of the pelvic ring. With the patient supine, the plane of the pelvic brim is oblique to the x-ray beam while obtaining the AP pelvic radiography. Other projections are required to ascertain anterior and posterior displacement of the pelvic ring as well as caudad or cephalad displacement of the hemipelvis. These projections were developed and described by Pennal and Sutherland in 1961 [14].

The inlet view (Fig. 29.7) of the pelvis is obtained with the patient supine. The x-ray beam is directed approximately 40°caudad. The inlet view better shows anterior or posterior displacement of the pelvic ring. The inlet view also reveals rotation of the anterior aspect of the pelvis [14].

Fig. 29.6 AP pelvis with intact symphysis, with healing right iliac wing fracture

Fig. 29.7 Inlet view, demonstrating healing right iliac wing fracture

The outlet view (Fig. 29.8) is obtained with the x-ray beamed directed approximately 40° cephalad. This view demonstrates vertical displacement of either hemipelvis. In addition, the sacral foramina are best evaluated on the outlet view.

Computed tomography (CT) (Fig. 29.9) provides additional information regarding injury to

Fig. 29.8 Outlet view, again demonstrating right iliac wing fracture

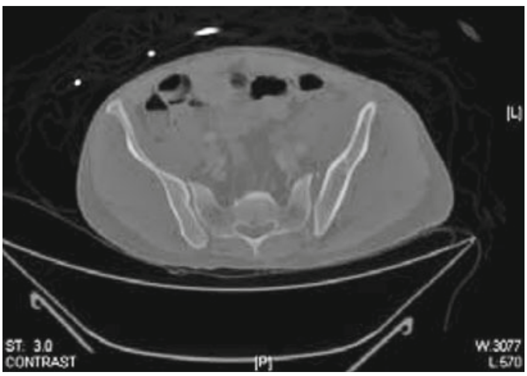


Fig. 29.9 CT scan demonstrating left sacroiliac joint widening

the pelvic ring. CT is especially useful to evaluate injury to the posterior pelvic ring, as it readily demonstrates sacral fractures and sacroiliac joint widening are damaged. This renders the pelvis completely unstable.

29.10 Description and Classification of Pelvic Ring Injury

It is essential to realize that the pelvis is a ring. If the ring is disrupted in one area, it must be disrupted in another region. Simply stated, when an anterior lesion is noted to disrupt the pelvic ring, a posterior lesion must also exist.

Anterior disruptions include symphyseal diastasis, pubic rami fractures, or both rami fractures and symphyseal disruptions.

Posterior injuries include fractures, either through the ilium or sacrum. Posterior disruptions also include sacroiliac joint dislocations (pure) or fracture dislocations.

Pelvic fractures may be associated with total stability, partial stability, or complete instability of the ring. Stability is defined as "the ability of the pelvis to withstand physiologic force without deformation" [15] and depends upon the integrity of the posterior aspect of the ring. Stability relies on the strong ligamentous structures of the posterior pelvis, including the anterior and posterior sacroiliac, sacrotuberous, and sacrospinous ligaments. These key structures prevent displacement of the pelvic ring with normal weight-bearing forces. Tile described a classification system based on stability of the pelvic ring [16]. Type A fractures are stable and minimally displaced. Type B fractures show partial stability of the ring, with partial disruption of the posterior pelvic ring. Posterior or vertical displacement does not occur in these types of injuries. Type C injuries are completely unstable and result from high-energy mechanisms. Type A and B injuries comprise 70–80 % of all pelvic injuries [15].

Young and Burgess described a classification of pelvic fractures based on the mechanism of injury in 1990 [10], which is the most frequently used classification system. The system takes into account the direction of deforming force as well as the amount of bony displacement. This classification system has traditionally been used to predict mortality, transfusion requirements, and associated injuries in patients with pelvic ring disruption. The classification according to direction of the force causing the ring disruption falls into one of three categories: anteroposterior compression, lateral compression, and vertical shear.

Anteroposterior compression (APC) injuries result from a force applied against the front or back of the pelvis (Fig. 29.10). These injuries are defined by pubic symphyseal diastasis and

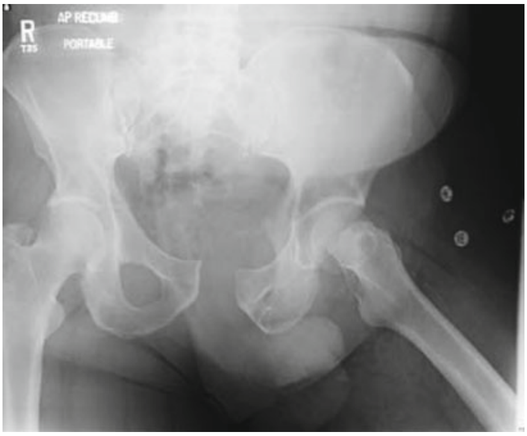


Fig. 29.10 AP pelvis from trauma bay demonstrating symphyseal diastasis, consistent with APC type of injury

Fig. 29.11 LC-1 injury, with fractures of the left superior and inferior pubic rami. Also noted to have impacted fracture of the left sacral ala, which is difficult to appreciate

sacroiliac joint widening [10]. The pelvis undergoes progressively increased amounts of external rotation with higher degrees of injury. Initially, the pubic symphysis is disrupted. Symphyseal disruption is followed by injury to the anterior sacroiliac ligaments. If the energy continues to produce further external rotation, the pelvic floor ligaments are thought to rupture next. The posterior sacroiliac ligaments are the last stabilizing structure to fail. Once the posterior sacroiliac ligaments fail, the pelvis is rendered completely rotational unstable. The external rotation deformity leads to tensile forces, which act to stretch or tear neurovascular structures within the pelvis. The AP radiograph reveals symphyseal diastasis or a vertical rami fracture, which is associated with sacroiliac joint widening. The anteroposterior injuries are subdivided into three groups: APC I– III injuries.

APC I injuries describe slight symphyseal widening (traditionally thought to be <2.5 cm). The anterior sacroiliac, posterior sacroiliac, sacrotuberous, and sacrospinous ligaments are intact, resulting in a stable injury. APC II injuries are associated with anterior sacroiliac joint widening. Anatomically, the anterior sacroiliac ligaments and the pelvic floor ligaments (sacrotuberous and sacrospinous) have been disrupted. The posterior sacroiliac ligaments remain intact. These injuries are described as "open book" in nature, due to the external rotation noted on x-ray. The intact posterior sacroiliac ligaments act as a hinge, allowing the anterior pelvis to "book open."

APC III injuries are the most severe variety of anteroposterior compression. These injuries result from high-energy mechanisms, such as crush injuries. The hemipelvis is completely separated from pelvic ring. The posterior sacroiliac ligaments have failed, along with the anterior sacroiliac, sacrotuberous, and sacrospinous ligaments. This severe injury is associated with major vascular, neurological, or visceral pathology. APC injuries are associated with higher risk of bleeding, as the vasculature is subjected to tensile forces that may tear the large vessels.

Lateral compression (LC) ring injuries result from a laterally directed force (Fig. 29.11). These injuries are associated with "T-bone"-type motor vehicle mechanisms. The laterally directed force imparts an internal rotation injury to the affected hemipelvis. The internal rotation deformity leads to shortening of ligamentous and neurovascular structures. Unlike APC injuries, the pelvic ring acts to produce a "tamponade" phenomenon. The lateral compression fractures also further classified into three subdivisions: LCI–LCIII. LCI ring injuries reveal fractures of the pubic rami with an associated sacral compression fracture. The sacral compression fracture occurs on the same side as the impaction force. The sacral fracture may not be obvious on the AP views, but is better visualized on the inlet and outlet projections. CT allows for improved visualization of sacral involvement.

LCII injuries involve rami fractures as well as an iliac wing (crescent) fracture. The iliac crescent fracture fragment consists of a piece of the posterior ilium, which is attached to the sacrum by the intact posterior sacroiliac ligaments. The injury involves medial rotation of the iliac wing near the anterior aspect of the affected sacroiliac joint [10].

LCIII pelvic ring disruptions are associated with a more severe energy imparted to the pelvis via a lateral blow. The greater amount of energy results in external rotation of the contralateral pelvis. These injuries are associated with rollover mechanisms, occurring after a struck pedestrian is subsequently rolled over by a motor vehicle [10]. The impacted hemipelvis is internally rotated, while the contralateral hemipelvis is externally rotated (windswept deformity).

Finally, vertical shear injuries are associated with cephalad displacement of one or both hemipelvis (Fig. 29.12). Vertical shear injuries result from high-energy axial loads to the hemipelvis. Patients may have a history of a fall from a height or ejection from a motorcycle. The vertical displacement may be evident on the initial AP view, but is best seen on the outlet view (Fig. 29.13).

Fig. 29.12 AP pelvis demonstrating pubic symphyseal disruption and superior translation of the left hemipelvis

Fig. 29.13 Outlet view of previous patient, demonstrating vertical translation of the left hemipelvis

29.11 Clinical Evaluation

The evaluation of any patient with a suspected pelvic ring injury begins with a thorough history and physical examination. The patient most often reports a high-energy mechanism of injury, such as motor vehicle collision, crush injury, or fall from a height. Occasionally, elderly patients may suffer a pelvic ring injury resulting from a fall from standing. Injuries to the pelvic ring resulting from low-energy trauma are vastly different than high-energy injuries. Based on the Young-Burgess classification, the mechanism of injury allows prediction of the fracture pattern. Crush injuries are associated with APC-type injuries. Falls from a height may result in vertical shear disruptions. Patients involved in a “T-bone” collision will present with lateral compression injuries. The history alerts the clinician to be suspicious for vascular disruption and other associated injuries.

Physical examination may reveal pelvic malrotation. This may be appreciated by palpation of the anterior iliac spines and iliac crests. Additionally, the patient may present with a shortened extremity. If there is no obvious fracture of the lower extremity, pelvic ring disruption must be suspected. Finally, compression of the iliac crests may elucidate gross instability and motion of the pelvic ring.

Clinical evaluation of any patient with a pelvic ring injury must follow standard ATLS protocol, beginning with stabilization of airway and breathing. Fluid resuscitation requires the placement of two large-bore intravenous catheters to allow administration of 2 L crystalloid solution. This is followed by administration of blood products if the patient does not respond to crystalloid resuscitation. It is essential to closely monitor the patient's hemodynamic status, utilizing a urinary catheter and arterial line. The patient should be examined for associated urogenital trauma prior to insertion of urinary catheter. 4 % of patients with a pelvic fracture sustain a bladder injury, while 2 % of pelvic ring injuries are associated with a urethral injury [17]. Blood at the urethral meatus, scrotal hematoma, hematuria, and a high-riding prostate detected upon rectal examination are clinical signs of urogenital trauma. Any resistance with attempted urinary catheterization is also indicative of urogenital injury and needs further evaluation. Retrograde urethrogram is indicated with any suspicion of urogenital injury.

Open pelvic fractures are associated with lacerations of the perineal region. Open pelvic fractures may be associated with injury to the vagina, urethra, anus, or rectum. These are highly contaminated injuries and require urgent surgical debridement, as these injuries may result in sepsis and multiorgan failure. A multidisciplinary approach is paramount, as diverting colostomy is indicated when there is bowel injury to prevent development of sepsis [17].

Bleeding associated with pelvic ring injuries is usually venous in nature and will respond to limiting pelvic volume via binding. Life-threatening bleeding is most often attributable to injury to branches of the internal iliac artery, most often the internal pudendal artery. Patients with pelvic ring injuries often have associated injuries to other body systems. Therefore, the source of bleeding must be identified emergently in order to stabilize the patient. Pelvic binding using a sheet is an inexpensive, noninvasive, and expedient method to reduce pelvic volume, thereby controlling pelvic bleeding. Appropriate sheet placement is essential to provide effective control of bleeding. The sheet should be placed inferior to the anterior superior iliac spines and centered over the bilateral greater trochanters [17]. Angiography with embolization is indicated in patients who do not respond to pelvic binding in the face of adequate fluid resuscitation. Pelvic sources of bleeding are initially diagnosed on pelvic CT images, showing contrast extravasation. Angiography requires patient transport to the angiography suite, which can be hazardous for an unstable patient. The patient must be closely monitored while in the angiography suite, as rapid resuscitation may become necessary should the patient become hemodynamically unstable. Complications associated with angiographic embolization of intrapelvic sources of bleeding include gluteal muscle necrosis, which adversely affect surgical treatment of pelvic fractures. The risks and benefits of angiographic embolization must be carefully weighed and tailored to each individual clinical scenario. Thus some authors advocate retroperitoneal packing instead of embolization as a first treatment after pelvic binding.

29.12 Treatment of Pelvic Ring Injuries

The treatment of pelvic ring injuries depends upon the severity of the injury. Stable injuries have a minimal risk of displacement and respond to nonoperative treatment. The patient may bear weight as tolerated immediately after the injury. LCI and APC I injuries are examples of stable injuries, which are amenable to early weight bearing and mobilization. The type of treatment depends upon the type of injury (stable versus unstable). Unstable pelvic fractures require surgical intervention to achieve stability of the ring.

APC-type injuries require open reduction internal fixation of the pubic diastasis (traditionally greater than 25 cm) through anterior symphyseal plating. An alternative treatment for APC-type injuries involves placement of an anterior pelvic external fixator.

Unstable posterior ring injuries also require some operative treatment. Injuries to the sacroiliac joint or fractures of the sacrum are often amenable to closed reduction and percutaneous treatment using an iliosacral screw. For more severely displaced injuries to the sacroiliac joint, treatment options include open reduction internal fixation or sacral bars. Finally, more severe injuries require anterior and posterior fixation for stability. Early care allows improved patient mobilization as well as early control of pelvic bleeding. Fixation of the ring provides stability to the injury, which affords better patient comfort and pulmonary function through improved patient mobilization.

29.13 Complications Associated with Pelvic Ring Injuries

Acute complications include hemorrhage and nerve injury. These typically present at the time of injury. Later complications include pain, which may be secondary to pelvic deformity resulting from the fracture. It is essential to recognize that patients with pelvic ring injuries are most often poly-trauma patients. Mortality in the acute phase of these injuries often results from hemorrhage, head injury, or multisystem organ failure.

In summary, patients with pelvic ring injuries have an overall mortality rate of approximately 10 %. These injuries are often associated with other systemic and bony injuries. Patients with unstable pelvic ring injuries require early surgical fixation to provide stability of the pelvic ring and to avoid long-term complications associated with pelvic ring malunion.

References

1. Routt ML Jr, Simonian PT, Swiontkowski MF (1997) Stabilization of pelvic ring disruptions. Orthop Clin North Am 28:369–398
2. Denis F, Davis S, Comfort T (1988) Sacral fractures: an important problem. Retrospective analysis of 236 cases. Clin Orthop 227:67–81
3. Mehta S, Auerbach JD, Born CT, Chin KR (2006) Sacral fractures. J Am Acad Orthop Surg 14: 656–665
4. Roy-Camille R, Saillant G, Gagna G et al (1985) Transverse fracture of the upper sacrum. Suicidal jumper's fracture. Spine 10:838–845
5. Strange-Vongsen HH, Lebech A (1991) An unusual type of fracture in the upper sacrum. J Orthop Trauma 5:200–203
6. Vaccaro AR, Kim DH, Brodke DS et al (2004) Diagnosis and management of sacral spine fractures. J Bone Joint Surg Am 86-A:166–175
7. Routt ML Jr, Nork SE, Mills WJ (2000) Percutaneous fixation of pelvic ring disruptions. Clin Orthop Relat Res 375:15–29
8. Zelle BA, Gruen GS, Hunt T, Speth SR (2004) Sacral fractures with neurological injury: is early decompression beneficial? Int Orthop 28:244–251
9. Schildhauer TA, Ledoux WR, Chapman WR, Henley MB, Tencer AF, Routt ML Jr (2003) Triangular osteosynthesis and iliosacral screw fixation for unstable sacral fractures. A cadaveric and biomechanical evaluation under cyclic loads. J Orthop Trauma 17:22–31
10. Burgess A et al (1990) Pelvic ring disruptions: effective classification system and treatment protocols. J Trauma 30(7):848–856
11. Manson T et al (2010) Young-Burgess classification of pelvic ring fractures: does It predict mortality, transfusion requirements, and Non-orthopaedic injuries? J Orthop Trauma 24(10):603–609
12. Starr A et al (2002) Pelvic ring disruptions: prediction of associated injuries, transfusion requirement, pelvic arteriography, complications, and mortality. J Orthop Trauma 16(8):553–561
13. Reilly MC et al (1996) Neurologic injuries in pelvic fractures. Clin Orthop Relat Res 329:28–36
14. Pennal G et al (1980) Pelvic disruption: assessment and classification. Clin Orthop Relat Res 151:12–21
15. Tile M (1996) Acute pelvic fractures: I. Causation and classification. J Am Acad Orthop Surg 4(3):143–150
16. Tile M (1988) Pelvic ring fractures: should they be fixed? J Bone Joint Surg Br 70-B(1):1–12
17. Flint L, Cryer HG (2009) Pelvic fracture: the last 50 years. J Orthop Trauma 69(3):483–488

Acute Traumatic Spinal Cord Injury: Epidemiology, Evaluation, and Management

30

Jefferson R. Wilson, Newton Cho, and Michael G. Fehlings

30.1 Epidemiology

The annual incidence of traumatic spinal cord injury (SCI) varies depending on the region considered, with international estimates ranging from 10 to 85 per million population [1–5]. In the United States, SCI is diagnosed in approximately 1 in 40 patients presenting to a major trauma center, with up to 15% of patients dying prior to arrival at hospital [4–6]. At present there are over 1 million people living with SCI in the United States, with a documented prevalence in the developed world ranging from 681 to 750 per million [7–10].

In the majority of published epidemiological studies, the age distribution of SCI appears bimodal with the first peak occurring in adolescence/young adulthood between the ages of 15 and 30 (related to an increase in injuries secondary to violence, sports accidents, and motor vehicle accidents) and the second peak occurring in those greater than 70 years old (related to an increase in fall-related SCI in the elderly) [11, 12]. Approximately 60–75 % of all injuries are cervical in location, whereas thoracic and lumbosacral injuries are substantially less common, accounting for 15 and 10 % of all injuries, respectively [7, 13]. As regards etiology, motor vehicle accidents and falls are generally the most common causes of injury; however, the lack of consistency in the classification of etiology across the literature precludes a more in-depth analysis on this topic [14, 15].

30.2 Pathophysiology

The final degree of neurological tissue destruction that occurs after traumatic SCI is a product of an orderly sequence of pathological processes [16, 17]. Initiating this sequence is the primary injury sustained during the initial traumatic event. At a macroscopic level, this injury component often results from a compressive force applied to the spinal cord arising from (1) bone, disc, or ligamentous material extruded into the spinal canal; (2) extradural hematoma formation; or (3) dynamic instability of the spinal column [18, 19]. In light of the limited elasticity of the spinal cord, primary injury can also result from applied distractive forces, often associated with vascular shearing, petechial hemorrhage, and axonal disruption [20]. Within seconds of their occurrence,

J.R. Wilson • N. Cho
Division of Neurosurgery and Spinal Program, Department of Surgery, University of Toronto, 399 Bathurst Street, Toronto Western Hospital, Fourth Floor West Wing, Toronto, ON M5T 2S8, Canada

M.G. Fehlings, M.D., Ph.D., FRCSC, FACS (✉)
Division of Neurosurgery and Spinal Program, Department of Surgery, University of Toronto, 399 Bathurst Street, Toronto Western Hospital, Fourth Floor West Wing, Toronto, ON M5T 2S8, Canada

UHN & Department of Surgery, University of Toronto, Head Spinal Program, Krembil Neuroscience Center, 399 Bathurst St. Toronto Western Hospital, Suite 4WW-446, Toronto, ON M5T 2S8, Canada
e-mail: michael.fehlings@uhn.on.ca

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_30,

these primary injury mechanisms initiate a cascade of deleterious pathobiological processes, collectively referred to as secondary injury mechanisms. Initial vascular disruption and petechial hemorrhage formation are followed in close temporal sequence by the appearance of edema, an increase in intramedullary interstitial pressure, and release of vasoactive proteins, which leads to vasospasm of spinal cord microvasculature and reduced tissue perfusion [21–24]. The resulting ischemic changes incite a combination of processes that exacerbate the degree of tissue destruction and include interstitial and cellular ionic imbalance, free radical formation, glutaminergic excitotoxicity, lipid peroxidation, and generation of arachidonic acid metabolites [25–27]. The end result is the incremental expansion of the primary traumatic lesion in a rostral and caudal direction, beginning immediately after the original event and culminating several weeks later [19]. Although little can be done from a therapeutic standpoint to correct damage sustained during the primary injury, by mitigating the evolution of secondary injury events, there is opportunity to preserve remnant viable neurological tissue and hence optimize outcomes.

30.3 Prehospital Period

Concerning the prehospital care of an individual with a suspected SCI, three overriding principles apply. First, all spinal cord injured patients should be transferred expeditiously to a regional healthcare center equipped with the resources and expertise to offer definitive medical and surgical care [28, 29]. Several quasi-experimental studies from both Canada and Europe have demonstrated the potential impact of prompt transport of patients to a specialty center on improving clinical outcomes. Tator et al. compared two time periods of SCI care within the Canadian province of Ontario, before and after the creation of a dedicated SCI unit and the development of improved medical transport systems [30]. These changes translated into fewer deaths, reduced complication rates, and shorter hospital stays for patients treated in the more modern era [31]. The decision of transport modality (ground ambulance vs. helicopter vs. fixed wing aircraft) should reflect a balance between geographical considerations and the patient's clinical status, with the overall aim of delivering the injured patient at the specialty center as rapidly as possible. Second, for all patients where spinal injury is a possibility, transfer should proceed to definitive care with complete immobilization of the craniospinal axis [32]. This is accomplished through placement of a rigid cervical collar while maintaining in-line stabilization of the neck and by transferring a patient, through use of a log-roll maneuver, to a rigid spinal board. Third, given the high incidence of pulmonary and hemodynamic complications among patients with acute SCI, patients' cardiorespiratory status must be continuously monitored throughout the transport process. Furthermore, the transporting team should be equipped with the resources and expertise necessary to provide advanced airway, respiratory, or cardiovascular support at any point through the transfer process, should a deterioration in clinical status occur [33].

30.4 Neurological Evaluation

At hospital arrival, determination of injury severity by performing a neurological assessment is important for purposes of communicating prognosis to the patient and their family, establishing a frame of reference to which future neurological exams can be compared and for formulating a definitive management strategy [34]. At present, all examinations should proceed according to the fourth edition of the International Standards for Neurological Classification of Spinal Cord Injury proposed by the American Spinal Injury Association(ASIA) [35]. Using these criteria, three separate indices are recorded. ASIA motor score (AMS) records muscle power in 10 myotomes bilaterally (each with a maximum power of 5) resulting in a cumulative score with a minimum value of 0 and a maximum value of 100. For ASIA sensory score (ASS), pinprick and light touch sensation are assessed in 28 dermatomes bilaterally (C2–S5), with each dermatome receiving a score of 0, 1, or 2 depending on whether the sensation is absent, abnormal, or normal, respectively. This results in cumulative scores for both pinprick and

Table 30.1 ASIA Impairment Severity (AIS) grade classification

AIS grade A	No motor or sensory function is preserved in the sacral segments
AIS grade B	Sensory but no motor function is preserved below the neurological level and includes the sacral segments
AIS grade C	Motor function is preserved below the neurological level, and more than half of key muscles below this level have a muscle grade less than 3
AIS grade D	Motor function is preserved below the neurological level, and more than half of key muscles below this level have a muscle grade of 3 or more
AIS grade E	No neurological deficit

light touch with minimum values of 0 and maximum values of 112. Finally ASIA Impairment Severity grade is determined according to the criteria outlined in Table 30.1. According to these criteria, a patient with a complete injury has no sensory or motor function in the distal most sacral segment, whereas a patient with an incomplete injury does have evidence of sensory or motor preservation within this segment ("sacral sparing"). Also important to delineate is the neurological level of injury, which is defined as the most caudal segment of the spinal cord with normal sensory and motor function on both sides of the body. Finally, the zone of partial preservation applies only to complete or AIS grade A injuries and refers to those myotomes and dermatomes caudal to the neurological level of injury that remain partially innervated. The presence of a zone of partial preservation has shown to be a positive predictor of neurological recovery among patients with complete lesions [36–39].

Although there is individual variation depending on the series examined, AIS grade A injuries are generally the most common followed by AIS grade D injuries [13]. Among incomplete injuries, central cord syndrome (CCS) is the most commonly observed. CCS is frequently seen among elderly patients with preexistent cervical spondylosis and spinal canal stenosis who undergo a cervical hyperextension injury, typically due to a fall [40]. Patients with CCS classically present with motor deficits greater in the upper extremities than the lower extremities and greater in the proximal muscle groups than distal. Also, they can have a variable pattern of sensory disturbance below the level of the lesion and, less frequently, sphincter dysfunction. This pattern of neurological deficit is directly related to the somatotopic organization of motor fibers in the cervical spinal cord, with fibers serving the upper extremities located more medially than fibers serving the lower extremities. Given that the center of the spinal cord has a watershed vascular supply, the upper extremity motor fibers are the most susceptible to hypoperfusion in the setting of cord swelling after SCI.

Anterior cord syndrome presents with bilateral paresis and loss of temperature/pain sensation, with preservation of light touch sensation. Brown-Sequard (hemicord) syndrome presents with unilateral motor and light touch sensory loss and contralateral loss of pain and temperature sensation below the lesion. These are less frequently encountered incomplete SCI syndromes [41].

30.5 Radiographic Evaluation

Characterization of specific anatomical injury characteristics obtained through diagnostic imaging allows for the enactment of a patient specific treatment plan. Initial screening investigations for trauma patients suspected of harboring a spinal injury may include spinal X-rays and or CT scan [42, 43]. At present, however, in light of the finding that X-rays alone may miss up to two-thirds of cervical fractures, CT is currently the preferred modality for diagnosing spinal fractures [44, 45]. Moreover, the three-dimensional CT reconstructions provide an accurate representation of the bony anatomy helpful for purposes of surgical planning. While X-ray and CT are best suited to diagnose spinal fractures and dislocations, they are less effective at identifying injuries to the neural elements or to soft tissues such as the posterior ligamentous complex and the disco-ligamentous complex. For these purposes, spinal MRI is the preferred diagnostic imaging modality.

Specific MRI findings have shown importance in aiding in treatment decision making in SCI. Because the need for urgent decompressive surgery is dependent upon the presence or absence of spinal cord compression, having access to an objective standardized method for diagnosing compression is imperative. The Fehlings group has provided a method to determine maximal spinal cord compression by comparing the anteroposterior(AP) cord diameter at the level of injury with the AP cord diameter at the nearest normal levels above and below, using the midsagittal T2-weighted MRI [46]. The advantage to this method, in contrast to other measures such as cord area, circumference, and circularity, is that it can be rapidly applied by any clinician with minimal training required. In a similar fashion, maximal spinal canal compromise is calculated by comparing the AP canal diameter at the level of injury with the AP canal diameter at nearest normal levels above and below, using the midsagittal T1-weigthed MRI. Spinal canal compromise on CT was found to be a poor surrogate for detecting spinal cord compression, shown by the fact that many of the patients with less than 25 % canal compromise on CT demonstrated evidence of spinal cord compression on T2-weighted MRI. This finding highlights the utility of MRI as an aid in surgical decision making in the post-SCI setting. In follow-up studies assessing the psychometric properties of these measurements, reliability and responsiveness were found to be consistently strong [47, 48].

A variety of MRI parameters have also proven useful as markers of long-term neurological prognosis. Consistently MRI signal change compatible with intramedullary hemorrhage, as well as spinal cord swelling, has been associated with a more severe pattern of neurological deficit at presentation in addition to a diminished potential for recovery at long-term follow-up [49–52]. The degree of spinal cord compression has also shown to be an important predictor of future neurological status, with more compression portending a worse outcome [53]. In contrast, signal change consistent with intramedullary edema, in the absence of the previously mentioned characteristics, has been associated with a favorable prognosis for recovery at follow-up [54]. The ability to prognosticate outcome based on initial imaging characteristics facilitates rehabilitation planning and aids in communication with patients and their families (Table 30.2).

Table 30.2 Primary and secondary injury mechanisms after traumatic SCI

Primary injury mechanisms	Secondary injury mechanisms
Initial force applied to spinal cord	Edema
Spinal cord contusion/ laceration	Increased interstitial pressures
Petechial hemorrhage formation	Release of vasoactive proteins
Axonal shearing	Vasoconstriction and vascular thrombosis
Vascular disruption	Ischemia
	Cellular ionic imbalance
	Free radical formation
	Cellular membrane lipid peroxidation
	Excitotoxic glutamine release

30.6 Medical Management

30.6.1 Intensive Care Unit Admission

At present, the medical management of SCI is centered around the treatment of cardiovascular and respiratory dysfunction. The preeminent goal of these supportive measures is to ensure adequate perfusion to and oxygenation of the injured spinal cord, by avoiding systemic hypoxia and hypotension. The first 2 weeks are particularly precarious for patients with SCI as it is during this period that they are most prone to the development of acute complications such as hypotension, cardiac dysrhythmias, hypoxemia, and pulmonary dysfunction [55]. Not unsurprisingly, patients with cervical injuries, as well as those with more severe injuries (AIS grade A), are the most susceptible to experiencing these complications. In a study of 50 patients with acute SCI, those with motor complete injuries were 5.5 times more likely to develop hypotension refractory to volume expansion, as compared to those with incomplete lesions [56]. In a larger European

Table 30.3 Guidelines for the medical management of acute spinal cord injury

Care setting	Patients with severe cervical level SCI should be managed in an intensive care unit setting with continuous hemodynamic, cardiac, and respiratory monitoring for the first 7–14 days post injury
Hemodynamics	Hypotension should be avoided with MAP maintained between 85 and 90 mmHg for the first 7 days post injury
Pharmacologic therapy	Administration of methylprednisolone for either 24 or 48 h is an option that should only be undertaken with the knowledge of the potential for an increased occurrence of complications
Thromboembolism prophylaxis	Thromboembolism prophylaxis is recommended including the use of low molecular weight heparin, pneumatic compression boots, and rotating beds

Modified from Hadley et al. [91]; March Supplement

series, 70 % of patients with complete SCI experienced respiratory insufficiency during the acute hospital stay, compared to just 27 % among patients with incomplete SCI [29]. For these reasons, SCI patients, particularly those with severe cervical lesions, should be monitored in an intensive care unit (ICU) setting for the first 7–14 days after injury [57]. The continuous physiological monitoring and aggressive medical management performed in the ICU setting have been associated in many studies with lower rates of morbidity and mortality and improved rates of neurological recovery at long-term follow-up [56, 58, 59] (Table 30.3).

30.6.2 Blood Pressure Management

The deleterious effects of hypotension after any traumatic injury to the central nervous system are now well established. A large body of preclinical literature offers compelling biological rationale to justify the optimization of spinal cord perfusion by avoiding hypotension in the acute post-SCI setting [24]. A variety of factors predispose SCI patients to the development of hypotension, with the major causes including interruption of sympathetic output leading to the loss of vascular tone and bradycardia (neurogenic shock), hypovolemia due to blood loss from associated traumatic injuries, and venous blood pooling due to skeletal muscle paralysis. The latter two causes of hypotension will manifest with classic clinical stigmata of hypovolemia such as tachycardia and cool clammy skin due to superficial vasoconstriction. The treatment of these conditions begins with aggressive fluid resuscitation using isotonic crystalloid administered intravenously and may also include blood transfusion and/or surgical control measures in the case of an associated hemorrhage. In contrast, neurogenic shock is characterized by hypotension with a low heart rate and warm hyperemic skin. This condition is primarily managed through administration of vasopressor medications, with a preference for those such as dopamine and norepinephrine, which cause peripheral vasoconstriction.

While performing a high-quality clinical study identifying optimal blood pressure cutoff points remains challenging in light of ethical concerns, there is consistency in the finding across several smaller studies that the institution of aggressive blood pressure targets leads to improved clinical outcomes [56, 60]. Based on the results of these studies (largely class III evidence), the American Association of Neurological Surgeons, in their 2002 guidelines for the management of acute SCI, have suggested that the mean arterial pressure of all SCI patients be maintained at 85–90 mmHg for the first 7 days after injury [61].

30.6.3 Administrations of Corticosteroids

Although there have been over a dozen clinical trials published to date evaluating the efficacy of a variety of pharmaceutical agents in the treatment of SCI, no safe and universally effective drug therapy has emerged [62]. Of all the drugs that have been evaluated, corticosteroids have been the most intensively investigated and were the subject of the first large randomized trials in traumatic SCI [63].

The first National Acute Spinal Cord Injury Study (NASCIS I) compared a 10-day regimen of high-dose methylprednisolone sodium succinate (MPSS) to low-dose MPSS and found no difference between the treatment groups in motor and sensory recovery at 6-week or 6-month follow-up [64]. Overall mortality and complication rates were the same between the groups, with the exception of wound infection rates, which were significantly higher among patients who received the high-dose regimen. On the heels of laboratory evidence indicating that higher doses of steroid would be required to appreciate a clinical effect, the second National Acute Spinal Cord Injury Study (NASCIS II) was undertaken [65, 66]. This study compared higher-dose MPSS administration, commenced within 24 h and continued for 24 h, to naloxone and placebo. In the primary analysis, there was superior sensory recovery reported among the steroid treated patients, with no treatment effect observed with respect to motor recovery. However, in a sub-analysis of patients who received treatment within 8 h from injury, greater motor recovery was observed among those treated with MPSS, a finding confirmed by a single small Japanese study which restricted enrollment to those receiving treatment within 8 h [67]. The third and final NASCIS study had three treatment arms comparing a 24- and a 48-h MPSS infusion (NASCIS II dose) to a third treatment group that received tirilazad mesylate [68]. At 6-week and 6-month follow-up, no significant differences were found between groups with respect to neurological recovery. However, in a predefined sub-analysis, patients treated between 3 and 8 h post injury had greater motor and functional recovery if treated with the 48-h MPSS regimen as compared to the 24-h regimen. However, the 48-h dose was associated with a significantly higher rate of pneumonia and severe sepsis when compared to the 24-h group.

In the wake of the NASCIS trials, clinicians remain divided on the routine use of MPSS administration in the context of acute SCI [69]. From a methodological standpoint, one of the largest issues remains that only in sub-analysis comparisons, not powered to answer the primary research question, were positive steroid treatment effects observed. However, perhaps the most concerning point, in light of the fact that the 48-h MPSS regimen given in NASCIS III represents the largest dose of steroid ever examined for clinical use, is the increased potential for infectious complications. Given these issues, the Canadian Neurosurgical Society, the Canadian Spine Society, and the Canadian Association of Emergency Physicians have adopted the recommendation that "a high-dose, 24-h infusion of methylprednisolone, started within 8 h after an acute closed spinal cord injury is not a standard treatment nor a guideline for treatment but, rather, a treatment option, for which there is level II and III evidence" [70].

30.7 Surgery for SCI

In the post-SCI setting, surgery is performed to achieve two specific goals: (1) to decompress the spinal cord and (2) to restore stability to the spinal column. A large body of preclinical literature supports the observation that persistent compression of the spinal cord represents a potentially reversible form of secondary injury, which, if uncorrected, can lead to a greater degree of neurological tissue destruction and worsened clinical outcomes [71–76]. Hence, decompression is performed with a rationale of mitigating secondary injury events. As regards the second goal, the presence of ongoing spinal instability, whether secondary to ligamentous or bony injury, can lead to repetitive spinal cord trauma, therefore exacerbating the degree of primary neurological injury. With advances in surgical technique and increased use of spinal instrumentation, instability can be corrected, preventing additional injury, obviating the need for prolonged immobilization, and expediting the commencement of rehabilitation protocols.

30.7.1 Surgical Timing

Many animal studies have documented the deleterious effects of persistent spinal cord compression on outcomes after SCI, as well as the potential beneficial effects of decompression surgery in this setting [77–80]. In a recent systematic review of the preclinical literature, of the 19 studies evaluating decompression in several different animals models, 11 reported a time-dependent effect favoring early surgery [81]. Illustrating this point, in one of the individual studies included in the review, rats were subjected to various durations of persistent spinal cord compression after weight drop induced SCI [74]. After a 6-week recovery period, functional status was noted to be progressively superior among rats with shorter duration of spinal cord compression. These results have led to the development of the clinical hypothesis that patients with SCI and evidence of persistent spinal cord compression treated with early surgery will experience superior clinical outcomes as compared to those patients who undergo surgery in a delayed fashion or not at all.

Although there is compelling biological rationale to support surgical spinal cord decompression in human patients with SCI, several practical issues have limited the translation of this therapy to the clinic. Traditionally, one dominating concern among clinicians has been the risk of worsening the degree of neurological injury by exposing patients to the potential risks of perioperative hypotension and or hypoxia. Considering the surgery itself, until the dissemination of instrumented fusion techniques in the last 15–20 years, decompression performed alone acted to further destabilize the spinal column, exposing the patient to the risk of further injury. Additionally, transport delays, as well as the time required to perform diagnostic investigations and medical stabilization of an injured patients, often preclude the possibility of expedient surgical management [82, 83]. In spite of the practical challenges, many clinical studies have been performed to evaluate the role of decompressive surgery in SCI, but more specifically, to define a therapeutic window that affords maximal neuroprotection balanced with the practical time constraints encountered in real-world medicine.

To identify an optimal surgical therapeutic window, a variety of time points have been used to distinguish early versus late surgical decompression. For the two most intensively investigated cutoff points, 24 and 72 h post injury, the results of efficacy studies are divergent, with several reporting clear-cut benefit with early intervention, while others have failed to find such a treatment effect. According to a recent systematic review of studies using the 24-h cutoff point, one surgical series found that surgery performed before 24 h was associated with improved neurological recovery, while several other papers associated early surgical decompression with shorter length of hospitalization, shorter length of intensive care unit stay, and reduced intraoperative blood loss [81]. In contrast, additional studies incorporating the 24-h cutoff have failed to find an effect for early surgical decompression on neurological recovery and operative blood loss. A similar dichotomy in outcomes, neurological and otherwise, exists among studies evaluating the 72-h post-injury time cutoff [81]. In spite of the lack of consensus among studies regarding the relative efficacy of early surgical decompression, there is consistency in the finding that early surgery, regardless of the time cutoff considered, is safe and not associated with a worse neurological status at follow-up. It is important to note however that the majority of studies on this topic are of low methodological quality, with results from a large systematic comparative analysis absent to date.

In order to help provide a more definitive answer regarding surgical timing after SCI, the Surgical Trial in Acute Spinal Cord Injury Study (STASCIS) was undertaken. This large prospective study involving five North American centers completed patient enrollment in 2009 with results anticipated in late 2011. The primary goal of this study is to evaluate the relative effectiveness of early (<24 h post injury) vs. late (>24 h post injury) surgical decompression on neurological recovery at 6-month follow-up. Preliminary data seem to support the authors' hypothesis that surgical decompression prior to 24 h post injury results in a greater degree of neurological recovery at follow-up [84].

30.8 Mortality and Complications

Mortality after SCI is heavily front-loaded with 15–20 % of patients dying prior during the prehospital period [4]. After hospital admission, the case fatality rate for SCI drops with 5–15 % patients dying during the acute hospital admission [4, 13].

Risk factors for inhospital mortality include age over 60, male sex, and cervical neurological level of injury [4, 7, 13]. In the long term, mortality rates for SCI patients decline; however, rates continue to be elevated relative to age matched noninjured individuals [85, 86]. According to data from the Model Systems SCI database, mortality rates were 3.8 % during the first year post injury, 1.6 % during the second year post injury, and 1.2 % per year over the next 10 years [87].

Given the physiological disruptions that occur as a result of injury, SCI patients are susceptible to a myriad of medical complications. At present it appears that respiratory complications, particularly pneumonia, followed closely by urosepsis are the most common causes of death among individuals with SCI [85, 88, 89]. Other commonly encountered secondary complications include decubitus ulcer development, autonomic dysreflexia, depression, thrombotic complications, and heart disease. Given the fact that complications develop in 20–35 % of patients after SCI, a high degree of vigilance is required on the part of medical personnel to recognize their occurrence and promptly institute appropriate treatment [7, 90].

References

1. Albert T, Ravaud J (2005) Rehabilitation of spinal cord injury in France: a nationwide multicentre study of incidence and regional disparities. Spinal Cord 43:357–365
2. Surkin J, Gilbert B, Harkey H, Sniezek J, Currier M (2000) Spinal cord injury in Mississippi. Findings and evaluation, 1992–1994. Spine 25:716–721
3. Chen Y, Chen S, Chiu W et al (1997) A nationwide epidemiological study of spinal cord injury in geriatric patients in Taiwan. Neuroepidemiology 18:241–247
4. Dryden D, Saunders L, Rowe B et al (2003) The epidemiology of traumatic spinal cord injury in Alberta, Canada. Can J Neurol Sci 30:113–121
5. Karacan I (2000) Traumatic spinal cord injuries in Turkey: a nation-wide epidemiological study. Spinal Cord 38:697–701
6. Burney R, Maio R, Maynard F (1993) Incidence, characteristics, and outcome of spinal cord injury at trauma centers in North America. Arch Surg 128:596–599
7. Ackery A, Tator C, Krassioukov A (2004) A global perspective on spinal cord injury epidemiology. J Neurotrauma 21:1355–1370
8. O'Connor P (2005) Prevalence of spinal cord injury in Australia. Spinal Cord 43:42–46
9. Paralysis facts and figures (2011) http://www.christopherreeve.org/site/c.mtKZKgMWKwG/b.5184189/k.5587/Paralysis_Facts__Figures.htm. Accessed 12 July 2011
10. Wyndaele M, Wyndaele J (2006) Incidence, prevalence, and epidemiology of spinal cord injury: what learns a worldwide literature survey? Spinal Cord 44:523–529
11. Pickett G, Campos-Benitez M, Keller J, Duggal N (2006) Epidemiology of traumatic spinal cord injury in Canada. Spine 31:799–805
12. Couris C, Guilcher S, Munce S et al (2010) Characteristics of adults with incident traumatic spinal cord injury in Ontario Canada. Spinal Cord 48:40–44
13. Sekhon L, Fehlings M (2001) Epidemiology, demographics, and pathophysiology of acute spinal cord injury. Spine 26:S1–S12
14. Pickett W, Simpson K, Walker J, Brison R (2003) Traumatic spinal cord injury in Ontario, Canada. J Trauma 55:1070–1076
15. DeVivo M (1997) Causes and costs of spinal cord injury in the United States. Spinal Cord 35:809–813
16. Fehlings M, Sekhon L (eds) (2000) Cellular, ionic and biomolecular mechanisms of the injury process. American Association of Neurologic Surgeons, Chicago
17. Tator C, Fehlings M (1991) Review of the secondary injury theory of acute spinal cord trauma with emphasis on vascular mechanisms. J Neurosurg 75:15–26
18. Amar A, Levy M (1999) Pathogenesis and pharmacological strategies for mitigating secondary damage in acute spinal cord injury. Neurosurgery 44:1027–1039
19. Schwartz G, Fehlings M (2002) Secondary injury mechanisms of spinal cord trauma: a novel therapeutic approach for the management of secondary pathophysiology with the sodium channel blocker riluzole. Prog Brain Res 137:177–190
20. Dolan E, Transfeldt E, Tator C (1980) The effect of spinal distraction on regional spinal cord blood flow in cats. J Neurosurg 53:756–764
21. Sandler A, Tator C (1976) Review of the effect of spinal cord trauma on the vessels and blood flow in the spinal cord. J Neurosurg 45:638–646
22. Tator C (1984) Vascular effects and blood flow in acute spinal cord injuries. J Neurosurg Sci 28:115–119
23. Sandler A, Tator C (1976) Effects of acute spinal cord compression injury on regional spinal cord blood flow in primates. J Neurosurg 45:660–676
24. Wallace M, Tator C (1987) Successful improvement of blood pressure, cardiac output, and spinal cord blood flow after experimental spinal cord injury. Neurosurgery 20:710–714

25. Rothman S, Olney J (1986) Glutamate and the pathophysiology of hypoxic-ischemic brain damage. Ann Neurol 19:105–111
26. Jorgensen M, Diemer N (1982) Selective neuron loss after cerebral ischemia in the rat: possible role of transmitter glutamate. Acta Neurol Scand 66:536–546
27. Braughler J, Duncan L, Chase R (1985) Interaction of lipid peroxidation and calcium in the pathogenesis of neuronal injury. Cent Nerv Syst Trauma 2: 269–283
28. Hachen H (1974) Emergency transportation in the event of acute spinal cord lesion. Paraplegia 12: 33–37
29. Hachen H (1977) Idealized care of the acute injured spinal cord in Switzerland. J Trauma 17: 931–936
30. Tator C, Rowed D, Schwartz M et al (1984) Management of acute spinal cord injuries. Can J Surg 27:289–294
31. Tator C, Duncan E, Edmonds V, Lapczak L, Andrews D (1993) Changes in epidemiology of acute spinal cord injury from 1947 to 1981. Surg Neurol 40: 207–215
32. Bernhard M, Gries A, Kremer P, Bottiger B (2005) Spinal cord injur–prehospital management. Resuscitation 66:127–139
33. Hadley M, Walters B, Grabb P et al (2002) Transportation of patients with acute traumatic spine injuries. Neurosurgery 50:S18–S20
34. Burns A, Ditunno J (2001) Establishing prognosis and maximizing functional outcomes after spinal cord injury. Spine 26:S137–S145
35. AmericanSpinalInjuryAssociation (2000) International standards for neurological classification of spinal cord injury. AmericanSpinalInjuryAssociation, Chicago
36. Dittuno J, Sipski M, Posuniak E, Chen Y, Staas W, Herbison G (1987) Wrist extensor recovery in traumatic quadriplegia. Arch Phys Med Rehabil 68:287–290
37. Dittuno J, Stover S, Freed M, Ahn J (1992) Motor recovery of the upper extremities in traumatic quadriplegia: a multicenter study. Arch Phys Med Rehabil 73:431–436
38. Katoh S, Masry WE (1995) Motor recovery of patients presenting with motor paralysis and sensory sparing following cervical spinal cord injuries. Paraplegia 33:506–509
39. Wu L, Marino R, Herbison G, Dittuno J (1992) Recovery of zero-grade muscles in the zone of partial preservation in motor complete quadriplegia. Arch Phys Med Rehabil 73:40–43
40. Chen L, Yang H, Yang T, Xu Y, Bao Z, Tang T (1997) Effectiveness of surgical treatment for traumatic central cord syndrome. J Neurosurg Spine 10:3–8
41. Roth E, Park T, Pantg T (1991) Traumatic cervical Brown-Sequard and Brown-Sequard plus syndromes: the spectrum of presentations an outcomes. Paraplegia 29:582–589
42. ACPS (1993) Advanced trauma life support system program for physicians. American College of Physicians and Surgeons, Chicago
43. Mirvis S, Diaconis J, Chirico P (1989) Protocol-driven radiologic evaluation of suspected cervical spine injury: efficacy study. Radiology 170: 831–834
44. Acheson M, Livingston R, Richardson M, Stimac G (1987) High-resolution CT scanning in the evaluation of cervical spine fractures: comparison with plain film examinations. AJR Am J Roentgenol 148: 1179–1185
45. Woodring J, Lee C (1993) Limitations of cervical radiography in the evaluation of acute cervical trauma. J Trauma 34:32–39
46. Fehlings M, Rao S, Tator C et al (1999) The optimal radiologic method for assessing spinal canal compromise and cord compression in patients with cervical spinal cord injury Part 2: results of a multicenter study. Spine 24:605–613
47. Furlan J, Kailaya-Vasan A, Aarabi B, Fehlings M (2011) A novel approach to quantitatively assess post-traumatic cervical spinal canal compromise and spinal cord compression: a multicenter responsiveness study. Spine (Phila Pa 1976) 36:784–793
48. Furlan J, Fehlings M, Massicotte E (2007) A quantitative and reproducible method to assess cord compression and canal stenosis after cervical spine trauma: a study of interrater and intrarater reliability. Spine 32:2083–2091
49. Miyanji F, Furlan J, Aarabi B, Arnold P, Fehlings M (2007) Acute cervical traumatic spinal cord injury: MR imaging findings correlated with neurologic outcome–prospective study with 100 consecutive patients. Radiology 243:820–827
50. Liao C, Lui T, Chen L, Chuang C, Huang Y (2005) Spinal cord injury without radiological abnormality in preschool-aged children: correlation of magnetic resonance imaging findings with neurological outcomes. J Neurosurg 103:S17–S23
51. Tewari M, Gifti D, Singh P (2005) Diagnosis and prognostication of adult spinal cord injury without radiographic abnormality using magnetic resonance imaging: analysis of 40 patients. Surg Neurol 63:204–209
52. Shimada K, Tokioka T (1999) Sequential MR studies of cervical cord injury: correlation with neurological damage and clinical outcome. Spinal Cord 37: 410–415
53. Yamashita Y, Takahashi M, Matsuno Y et al (1991) Acute spinal cord injury: magnetic resonance imaging correlated with myelopathy. Br J Radiol 64:201–209
54. Silberstein M, Tress B, Hennessy O (1992) Prediction of neurologic outcome in acute spinal cord injury: the role of CT and MR. AJNR Am J Neuroradiol 13:1597–1608
55. Lehman K, Lane J, Piepmeier J, Batsford W (1987) Cardiovascular abnormalities accompanying acute spinal cord injury in human: Incidence, time course and severity. J Am Coll Cardiol 10:46–52

56. Levi L, Wolf A, Belzber H (1993) Hemodynamic parameters in patients with acute cerical cord trauma: description, intervention, and prediction of outcome. Neurosurgery 33:1007–1017
57. Hadley M, Walters B, Grabb P (2002) Management of acute spinal cord injuries in an intestive care unit or other monitored setting. Neurosurgery 50: S51–S57
58. Reines H, Harris R (1987) Pulmonary complications of acute spinal cord injuries. Neurosurgery 21: 193–196
59. Gschaedler R, Dollfus P, Mole J, Mole L, Loeb J (1979) Reflections on the intensive care of acute cervical spinal cord injuries in a general traumatology centre. Paraplegia 17:58–61
60. Wolf A, Levi L, Mirvis S et al (1991) Operative management of bilateral facet disclocation. J Neurosurg 75:883–890
61. Hadley M, Walters B, Grabb P et al (2002) Blood pressure management after acute spinal cord injury. Neurosurgery 50:S58–S62
62. Rowland J, Hawryluk G, Kwon B, Fehlings M (2008) Current status of acute spinal cord injury pathophysiology and emerging therapies: promise on the horizon. Neurosurg Focus 25:E2
63. Hawryluk G, Roland J, Kwon B, Fehlings M (2008) Protection and repair of the injured spinal cord: a review of completed, ongoing, and planned clinical trials for acute spinal cord injury. Neurosurg Focus 25:E14
64. Bracken M, Collins W, Freeman D et al (1984) Efficacy of methylprednisolone in acute spinal cord injury. JAMA 251:45–52
65. Braughler J, Hall E (1984) Effects of multi-dose methylprednisolone sodium succinate administration on injured cat spinal cord neurofilament degradation and energy metabolism. J Neurosurg 61:290–295
66. Bracken M, Shepard M, Collins W et al (1990) A randomized, controlled trial of methylprednisolone or naloxone in the treatment of acute spinal-cord injury. Results of the Second National Acute Spinal Cord Injury Study. N Engl J Med 322:1405–1411
67. Otani K, Abe H, Kadoya S (1994) Beneficial effects of methylprednisolone sodium succinate in the treatment of acute spinal cord injury. Sekitsui Sekuzui J 7:633–647
68. Bracken M, Shepard M, Holford T et al (1997) 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. National Acute Spinal Cord Injury Study. JAMA 277:1597–1604
69. Hurlbert R (2000) Methylprednisolone for acute spinal cord injury: an inappropriate standard of care. J Neurosurg 93:1–7
70. Hugenholtz H, Cass D, Dvorak M (2002) High dose methylprednisilone for acute closed spinal cord injury—only a treatment option. Can J Neurol Sci 29:227–235
71. Brodkey J, Richards D, Blasingame J, Nulsen F (1972) Reversible spinal cord trauma in cats. Additive effects of direct pressure and ischemia. J Neurosurg 37:591–593
72. Carlson G, Minato Y, Okada A et al (1997) Early time-dependent decompression for spinal cord injury: vascular mechanisms of recovery. J Neurotrauma 14:951–962
73. Delamarter R, Sherman J, Carr J (1995) Pathophysiology of spinal cord injury. Recovery after immediate and delayed decompression. J Bone Joint Surg Am 77:1042–1049
74. Dimar J, Glassman S, Raque G, Zhang Y, Shields C (1999) The influence of spinal canal narrowing and timing of decompression on neurologic recovery after spinal cord contusion in a rat model. Spine 24:1623–1633
75. Dolan E, Tator C, Endrenyi L (1980) The value of decompression for acute experimental spinal cord compression injury. J Neurosurg 53:749–755
76. Guha A, Tator C, Endrenyi L (1987) Decompression of the spinal cord improves recovery after acute experimental spinal cord compression injury. Paraplegia 25:324–339
77. Carlson G, Gorden C, Oliff H, Pillai J, LaManna J (2003) Sustained spinal cord compression: Part I: time-dependent effect on long-term pathophysiology. J Bone Joint Surg Am 85:86–94
78. Kobrine A, Evans D, Rizzoli H (1979) Experimental acute balloon compression of the spinal cord. Factors affecting disappearance and return of the spinal evoked response. J Neurosurg 51:841–845
79. Nystrom B, Berglund J (1988) Spinal cord restitution following compression injuries in rats. Acta Neurol Scand 78:467–472
80. Tarlov I (1954) Spinal cord compression studies. III. Time limits for recovery after gradual compression in dogs. AMA Arch Neurol Psychiatry 71:588–597
81. Furlan J, Noonan V, Cadotte D, Fehlings M (2009) Timing of decompressive surgery of spinal cord after traumatic spinal cord injury: an evidence-based examination of Pre-clinical and clinical studies. J Neurotrauma 27:1–29
82. Tator C, Fehlings M, Thorpe K, Taylor W (1999) Current use and timing of spinal surgery for management of acute spinal surgery for management of acute spinal cord injury in North America: results of a retrospective multicenter study. J Neurosurg 91:12–18
83. Botel U, Glaser E, Niedeggen A (1997) The surgical treatment of acute spinal paralysed patients. Spinal Cord 35:420–428
84. Wilson J, Fehlings M (2011) Emerging approaches to the surgical management of acute traumatic spinal cord injury. Neurotherapeutics 8:187–194
85. Krause J, Carter R, Pickelsimer E, Wilson D (2008) A prospective study of health and risk of mortality after

spinal cord injury. Arch Phys Med Rehabil 89:1482–1491
86. Krause J, Sternberb M, Lottes S, Maides J (1997) Mortality after spinal cord injury: an 11-year prospective study. Arch Phys Med Rehabil 78:815–821
87. DeVivo M, Stover S (1995) Long term survival and causes of death. In: Stover SL, DeLisa JA, Whiteneck GG (eds) Spinal cord injury. Aspen Publishing, Gaithersburg
88. DeVivo M, Kartus P, Stover S, Rutt R, Fine P (1987) Seven year survival following spinal cord injury. Arch Neurol 44:872–875
89. Kiwerski J, Weiss M, Chrostowska T (1981) Analysis of patients after cervical spine trauma. Paraplegia 19:347–351
90. Krassioukov A, Furlan J, Fehlings M (2003) Medical co-morbidities, secondary complications, and mortality in elderly with acute spinal cord injury. J Neurotrauma 20:391–399
91. Hadley MN, Walters BC, Grabb PA et al (2002) Guidelines for the management of acute cervical spine and spinal cord injuries. Clin Neurosurg 49: 407–498

Part IV

Tumor, Infection, Inflammatory and Metabolic Conditions

31 Primary Spine Tumors

Marco Ferrone and Joseph Schwab

31.1 Case Example

A twenty-two-year-old female presented to her primary care physician's office complaining of mid-thoracic pain. The pain started suddenly 2 months prior to her visit when she bent over to pick up her 1-year-old son. She is otherwise healthy with no known medical problems. She is neurologically intact and has point tenderness to her mid-thoracic spine.

31.2 History and Physical Examination

The patient's history helps begin the formation of a differential diagnosis. In general, patients under 30 years old have benign conditions with exception of Ewing's sarcoma and osteosarcomas, while patients older than 30 years of age are more likely to have malignant diagnoses like myeloma or metastatic disease with the exception of common benign conditions like bone islands and hemangiomas [1]. Aggravating and alleviating factors can help establish mechanical instability or be pathomnemonic, as in relief from NSAIDs with osteoid osteoma. Furthermore, medical comorbidities and past treatments can help guide the differential diagnosis; eliciting a history of radiation therapy exposure could be very helpful in establishing a diagnosis of radiation-associated osteosarcoma. A detailed neurologic exam and serial exams for evolving conditions are always important and can be a red flag for progression of disease. In this case, the patient is 22, and she has the sudden onset of pain without antecedent trauma. Sudden onset of pain is typically muscular in origin; however, it is also possible that the patient has developed a fracture. She has point tenderness to one of her spinous processes in the mid-thoracic spine, which should make one suspicious for a bony abnormality. Since she has not had any trauma, one must consider a pathologic fracture and this should be sought out with further imaging.

31.3 Differential Diagnosis

The lesion in our case is located within the vertebral body. The location of the lesion is helpful in forming a differential diagnosis as some tumors have a predilection for the vertebral body or posterior elements. Again, the location of the lesion should be taken in context with the patient's age and history. We mentioned that this patient was 22 and that most lesions occurring in this age group (<30) are benign. However, there are malignant tumors that occur in the vertebral body in this age group including osteosarcoma. Other

M. Ferrone (✉)
Department of Ortho, Harvard Medical School,
Brigham and Women's Hospital,
75 Francis Street, Boston, MA 02115, USA
e-mail: mferrone@partners.org

J. Schwab
Department of Orthopaedic Surgery,
Harvard Medical School, Massachusetts General Hospital,
37 Chestnut Street, Boston, MA 02108, USA
e-mail: jhschwab@partners.org

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_31, © Springer-Verlag Berlin Heidelberg 2014

malignant bone tumors that occur in this location include chordomas, lymphoma, and plasmacytoma; however, they tend to occur in patients over 30 years old. Benign tumors must also be on the differential diagnosis including hemangioma, giant cell tumor (GCT), and eosinophilic granuloma (EG). If this lesion were located in the posterior elements in a similarly aged patient, then benign diagnoses would dominate the differential diagnosis including aneurysmal bone cyst (ABC), osteoid osteoma, osteoblastoma, and osteochondroma. However, one must not forget Ewing's sarcoma which can occur in this age group. Chondrosarcoma can occur anywhere in the vertebrae, but it usually occurs in older patients.

31.4 Investigations

In certain instances, laboratory data can be helpful. Mankin has advocated ordering complete blood count (CBC), erythrocyte sedimentation rate (ESR), calcium, phosphorous, alkaline phosphatase, blood urea nitrogen (BUN), creatinine, glucose, serum, and urine immunoelectrophoresis (SPEP and UPEP). Currently C-reactive protein (CRP) can be added to that list as should tumor markers such as AFP (alpha-fetoprotein), carcinoembryonic antigen (CEA), and prostate-specific antigen (PSA) if metastatic disease is strongly suspected [2].

When evaluating a patient for a tumor, the first imaging one should obtain is a plain radiography. A plain radiograph can help narrow the differential diagnosis based on the location of the tumor, its radiographic appearance, the impact the lesion has on the normal bone, and the impact that the normal bone has on the tumor. For instance, a bony lesion in the posterior elements associated with a soft tissue mass containing "popcorn"-like calcification is indicative of a cartilage-based lesion such as an osteochondroma or chondrosarcoma. A well-circumscribed, bone-forming lesion in the posterior elements with no soft tissue mass is consistent with an osteoid osteoma. A small area of dense bone with no surrounding bony changes is consistent with an enostosis or bone island. They represent compact bone within the spongiosa. On the other extreme, if a bone-forming lesion is noted infiltrating the adjacent bone with a soft tissue mass, then one should consider malignant tumors such as an osteosarcoma.

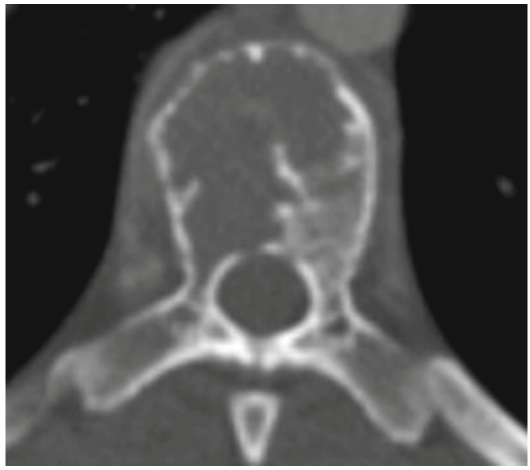


Fig. 31.1 Axial view of CT scan showing a lytic lesion with no bony matrix centered in the vertebral body. Additional images show endplate fracture

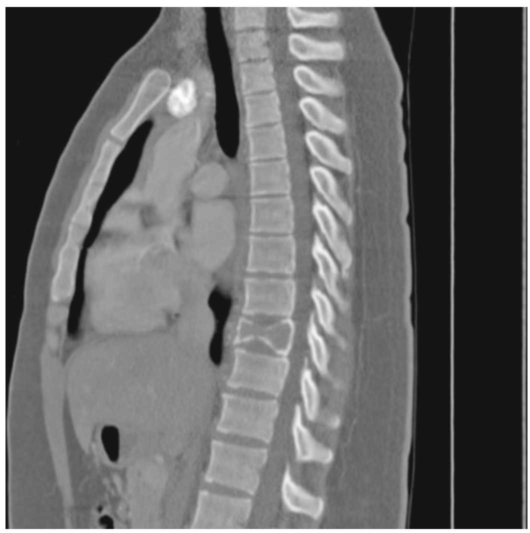


Fig. 31.2 Sagittal view of CT scan showing a lytic lesion with no bony matrix centered in the vertebral body and endplate fracture

Computed tomography is often very helpful in describing the nature of a bone tumor. In our case examples, a CT scan revealed a lytic lesion with no bony matrix centered in the vertebral body (Figs. 31.1 and 31.2). Furthermore, it demonstrated a fracture in the vertebral body. This fracture corresponds to the patient's history in which

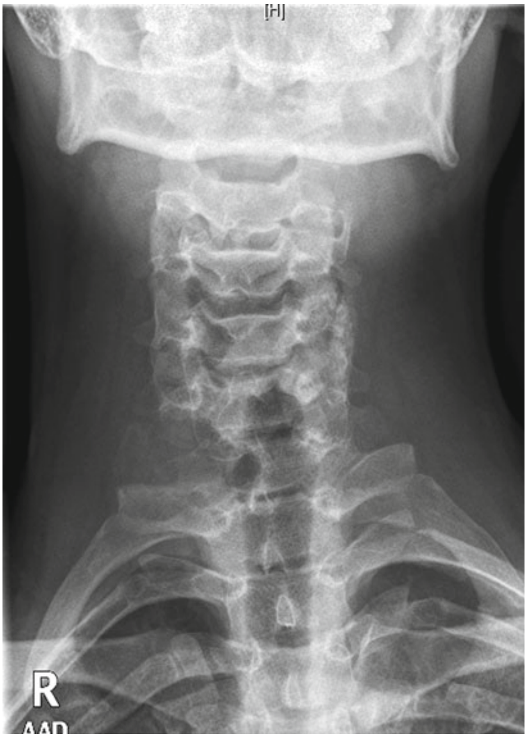


Fig. 31.3 Aneurysmal bone cyst showing characteristic balooning out of cortex

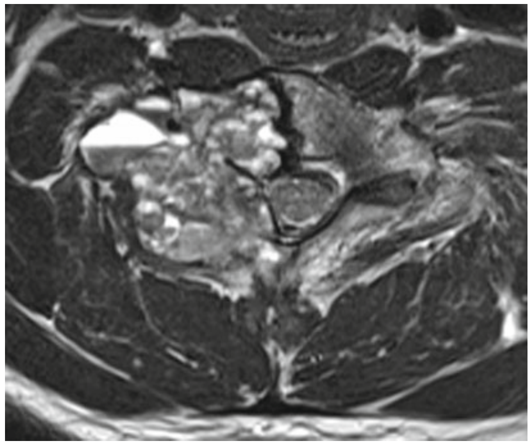


Fig. 31.4 MRI of same patient of Fig. 31.3, aneurysmal bone cyst showing typical fluid-fluid levels

she developed sudden pain. The absence of bony matrix helps narrow down the differential diagnosis on this patient. Bone-forming tumors such as osteoid osteomas, osteoblastomas, and osteosarcomas typically have a bony matrix evident on plain radiographs and CT. Similarly, cartilaginous tumors typically have stippled calcification evident on plain images or CT. In rapidly growing malignant bone tumors such as osteosarcoma, one might see periosteal elevation with associated calcification, so-called Codman's triangle.

Aneurysmal bone cysts (ABC) can occur in the same age group as the patient in our case; however, they have a characteristic appearance on plain radiograph (Fig. 31.3) and CT in which the cortex appears to balloon out from its normal location as if the bone were inflated with air like a balloon. Again, ABCs generally occur in the posterior elements and expand the bone which is not what we are seeing in the case presented here. Further, they often have fluid/fluid lines, which are better seen on MRI (Fig. 31.4) but can also be seen on CT. Eosinophilic granulomas (EG) can be associated with lytic lesions in the vertebral body. In fact, EG is associated with profound lytic disease in which the vertebrae completely collapse causing so-called vertebra plana. EG is a benign condition that occurs often in the vertebral body, but it usually occurs in children under age 10. Our patient is 22 and so the diagnosis of EG is less likely.

Magnetic resonance imaging (MRI) is warranted when working up a lesion particularly if the diagnosis is in question or if surgical treatment is considered. One of the reasons for ordering an MRI is to evaluate the extent of soft tissue involvement outside of the vertebrae. Clearly, if the patient has neurologic signs or symptoms, then an MRI is warranted to evaluate for nerve or spinal cord compression. One of the most common lesions found incidentally in the spine is that of hemangioma. Hemangiomas are hamartomas which means they are composed of normal cells occurring in abnormal locations. They have a characteristic appearance on plain radiograph and CT in which vertically oriented coarsened trabeculae are noted. Axial CT scans often reveal a polka-dot appearance which corresponds to imaging the trabeculae in cross section. Furthermore, they have a characteristic MRI appearance in which areas of the tumor demonstrate increased signal on T1-weighted images and other areas demonstrate increased signal on T2-weighted images. The increased T1 signal corresponds to the presence of fat in the tumor, and the increased

T2 signal demonstrates the presence of a vascular supply. Hemangiomas are generally asymptomatic and typically found incidentally.

A bone scan is often indicated in the workup of bone tumors. Not only does it provide information as to the metabolic impact the tumor is having on the bone, it also provides important information with regard to the extent of the tumor. When one is working up a potentially metastatic lesion, a bone scan is imperative. It provides information regarding staging, and it may reveal a more accessible lesion for biopsy. Bone scans demonstrate the relative activity of osteoblasts by measuring the degree of radiolabeled phosphate derivatives placed in the matrix by local osteoblasts. Both normal osteoblasts and osteoblasts associated with tumors take up radiolabeled phosphate from the blood.

Positron emission tomography (PET) scans measure the relative degree of glucose uptake by cells. Malignant cells rely heavily on glucose for metabolism, and so malignant cells take up more glucose than most tissues outside of the brain. PET is gaining popularity in the staging of cancer; however, most bone tumors can be identified based on the image modalities mentioned above; thus the role of PET has not been fully elucidated in the workup of primary bone tumors.

After obtaining a history, physical examination, and imaging when the diagnosis is in question, a biopsy should be considered. However, a biopsy is usually a confirmatory test. The differential diagnosis should be relatively short by the time a biopsy is considered. Most bone pathologists are keen to know the history as well as the outcome of routine imaging before they make a diagnosis based on histologic examination. The results of a biopsy should not be a surprise in that the histologic diagnosis should have been under consideration prior to the biopsy. When the results of a biopsy are a complete surprise, then one must relook at all aspects of the workup to learn where clues were missed. The answer is usually present in the history or plain radiographs.

Once the decision has been made to proceed with a biopsy, then the method of biopsy must be chosen. A CT-guided biopsy is often adequate; however, when a CT biopsy fails to provide diagnostic tissue, then an open biopsy can be performed. In the thoracic or lumbar spine, a transpedicular approach is often used to reach a vertebral body lesion, and a direct approach to the posterior elements is most commonly used for posteriorly based lesions. Lesions in the cervical spine pose greater problems due to the relative density of sensitive anatomic structures including the trachea, esophagus, spinal cord, and carotid and vertebral arteries. Image guidance is helpful and an experienced musculoskeletal radiologist is a must. In our case a transpedicular biopsy was performed which obtained sufficient tissue for diagnosis.

The histologic diagnosis was consistent with a giant cell tumor of the bone. Giant cell tumor tends to present after closure of the physes, in the 20–40-year-old, with a slight female predilection. It is also more common in Asians [3]. Spinal occurrences make up 5–10 % of GCTs, most often appearing in the vertebral bodies, and extraosseous extension is not uncommon [4]. The sacrum is where 90 % present, followed by lumbar, thoracic, and cervical regions. GCT tends to appear in the upper sacrum and tends to lateralize to an ala [5]. Pregnant patients may experience worsening of symptoms or tumor growth secondary to a hormonal response. Histologically, giant cell tumors made up of three cell populations the giant cell tumor stromal cells (GCTSC), mononuclear histiocytic cells (MNHC), and the characteristic multinucleated giant cells (MNGC). Presenting symptoms are most commonly pain and neurologic compromise [6]. Negative bone scans have been reported, but the majority are likely to show uptake at the lesion [7]. Plain radiographs will show lytic areas without matrix that may have an expansile appearance. CT imaging will have similar features to the radiographs. In addition, they can demonstrate the extraosseous soft tissue component and cystic areas of hemorrhage or associated ABC. MR imaging can be heterogeneous on both T1 and T2 sequences and can have areas of hemorrhage which are bright on both T1 and T2 sequences. The lesion is likely to enhance with gadolinium in all but the cystic areas.

31.5 Nonsurgical Treatments

It is important to note that not all bone tumors require surgical treatment. In fact many benign tumors can be followed with history and physical examination as well as radiographs if they are asymptomatic. Systemic treatment in the form of NSAIDs has been advocated by some for osteoid osteomas. For malignant bone tumors such as Ewing's sarcoma or osteosarcoma, systemic chemotherapy combined with surgery is the standard of care. Radiation therapy has been advocated by some for the management of malignant tumors about the spine. It is important to note that the field of radiation oncology is evolving and the use of high-precision radiation therapy is gaining momentum. High-precision radiation therapy allows the delivery of higher doses of radiation from multiple directions to concentrate at the tumor site, thus sparing normal tissues and thereby decreasing the toxicities associated with radiation. This has traditionally been the rate-limiting step of radiation therapy. This is mentioned here because what have historically been considered radiation-resistant tumors may be treated with radiation in the future at higher doses than used previously. In the example we presented here, the use of systemic agents targeting the RANK/RANKL has been investigated because of the long association between giant cell tumors and osteoclasts. In fact, giant cell tumors were at one time known as osteoclastomas. More recently denosumab (Amgen), a RANK-ligand inhibitor, has shown promise in some early studies of giant cell tumor [8]. Other drugs within this class are entering the clinical trial realm, and they will likely be available in the coming years.

31.6 Surgical Treatments

There are two main reasons why one should consider using preoperative angiography. The first is to help decrease blood loss in surgery by embolizing the tumor. This is particularly true when an intralesional resection is planned and the tumor is known to be vascular such as a giant cell tumor [9]. Tomita et al. describe embolizing the level of the tumor and also the level above and below as this decreases the blood supply to the involved vertebrae by nearly 80 % [10].

The second reason to consider embolization is to identify the dominant blood vessel to the spinal cord. Historically, ligation of this vessel was thought to increase the risk of inducing paralysis. However, Tomita has shown in an animal model and subsequently in a small series of patients that ligation of the so-called artery of Adamkiewicz by itself does not increase the risk of paralysis. Tomita reports that ligating multiple segmentals including the dominant artery can lead to paralysis but not the ligation of the dominant artery in isolation [11].

The treatment of primary spinal tumors is dictated by the nature of the tumor (benign vs. malignant) as well as the anatomic location and extent of the tumor. The WBB (Weinstein, Boriani, Biagini) classification system (Fig. 31.5) is meant to guide the surgeon when an en bloc excision is considered [12]. En bloc excision is usually reserved for malignant tumors; however, benign but aggressive tumors may also warrant en bloc resection. En bloc resection means removing the tumor in one piece. It is not synonymous with a negative margin. Margins are determined by the pathologist via histologic examination of the specimen. A negative margin indicates that the tumor was not exposed during surgery and that normal tissue surrounds the tumor. A positive margin indicates that the tumor was cut through or otherwise exposed during surgery. This has classically been considered a suboptimal margin for malignant bone tumors. When a tumor is removed in multiple pieces, it is known as an intralesional resection. Margins do not play into the histologic evaluation of intralesionally resected tumors as a negative margin was not the goal of the surgery. Instead, the extent of resection should be commented upon by the surgeon such as gross total resection or subtotal resection.

The WBB classification is meant to assist the surgeon in making decisions as to whether an oncologically sound margin can be obtained. The WBB system divides the spine into 12 radially based segments like the face of a clock on an

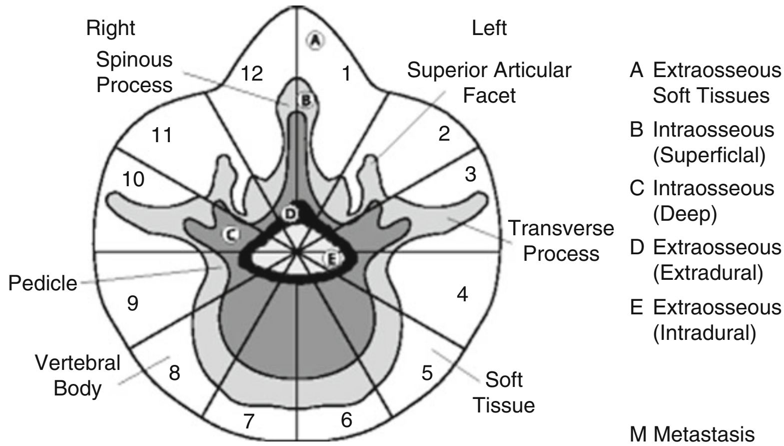


Fig. 31.5 The Weinstein, Boriani, Biagini Classification for spine tumor for use when considering en bloc excision

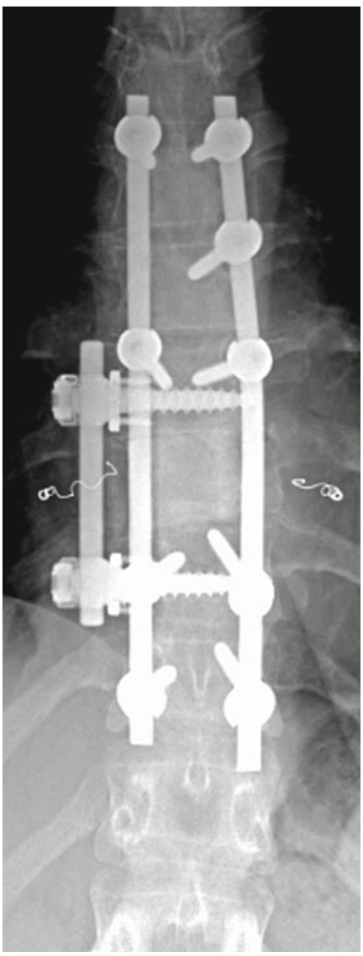


Fig. 31.6 Postoperative AP view of our case example patient after tumor resection, strut grafting and fixation

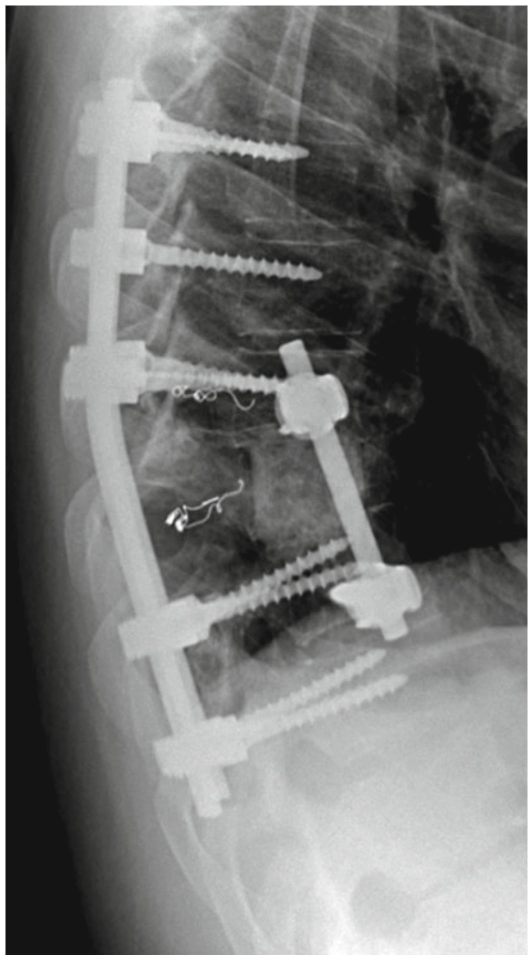


Fig. 31.7 Postoperative lateral view of our case example patient after tumor resection, strut grafting and fixation

axial cut through the spine. Further, the spine is divided into five zones indicating the depth of spinal involvement. Zone "a" indicates the mass extends outside the bony spine, zone "b" involves the superficial bone of the spine, and zone "c" involves the deep bone of the spine. Zone "d" indicates the tumor extends to the epidural space, and zone "e" is an intradural tumor. According to the WBB classification, at least one pedicle must be free of tumor for an oncologically sound margin to be obtainable. Further, it is less favorable to have zone "d" involvement when a negative margin is required.

In our case the tumor is benign. However, giant cell tumors can be locally aggressive, and those with a soft tissue mass (Enneking stage III) [13] have high rates of local recurrence after simple curettage. A recent study by Boriani et al. revealed that Enneking stage II tumors like the one presented here are adequately treated with intralesional curettage and they do not require en bloc removal [14]. In this case the spine was stabilized with pedicle screws above and below. In addition, the posterior elements of the involved vertebrae including the pedicles were removed. This was followed by a thoracotomy and complete intralesional resection of the tumor. An allograft femoral strut was used to reconstruct the anterior column (Figs. 31.6 and 31.7). This was a gross total intralesional resection.

Surgical staging system for musculoskeletal tumors (Enneking and MSTS)				
Benign				
1. Latent	G 0	T 0	M 0	
2. Active	G 0	T 0	M 0	
3. Aggressive	G 0	T 1–2	M 0–1	
Malignant: Stage determined by three different sub-categories				
– **Grade**: Histology with aid of radiographic findings and clinical correlation				
G 1: Low grade, uniform cell type without atypia, few mitoses				
G 2: High grade, atypical nuclei, mitoses pronounced				
– **Site**				
T 1: Intracompartmental (confined within limits of periosteum)				
T 2: Extracompartmental (breach in an adjacent joint cartilage, bone cortex (or periosteum) fascia lata, quadriceps, and joint capsule)				
– **Metastasis**				
M 0: No identifiable skip lesions or distant metastases				
M 1: Any skip lesions, regional lymph nodes, or distant metastases				
Enneking's staging system of malignant bone tumors, CORR 1980				
Ia	Low grade, intracompartmental	G 1	T 1	M 0
Ib	Low grade, extracompartmental	G 1	T 2	M 0
IIa	High grade, intracompartmental	G 2	T 1	M 0
IIb	High grade, extracompartmental	G 2	T 2	M 0
IIIa	Low or High grade, intracompart. w/metastases	G 1–2	T 1	M 1
IIIb	Low or High grade, extracompart. w/metastases	G 1–2	T 2	M 1

31.7 Postoperative Care

The postoperative management of patients with spinal tumors is unique in two important ways. The first is related to the potential hypercoagulable state found in malignant disease. These patients may be at higher risk for deep venous thrombosis/pulmonary embolism. One must consider more robust anticoagulation in this population. This of course must be balanced with the risk of bleeding adjacent to the spinal cord.

The second important difference is in long-term follow-up of these patients. Local recurrence is a potential problem in any bone tumor that warrants resection in the first place. These patients must be followed periodically with clinical examination and/or appropriate imaging studies.

31.8 Potential Complications

Exsanguination is a potential complication of removing even benign bone tumors. Giant cell tumors and ABCs carry well-known bleeding risks. Further, en bloc resection carries with it a high rate of complication from massive bleeding to paralysis to death. The rate and severity of complications correlates with the complexity of the resection and the inherent vascularity of the tumor being treated. Furthermore, the use of systemic therapy such as with the treatment of osteosarcoma can predispose patients to infection particularly if their white blood cell count has not been given appropriate time to recover. The use of radiation is expanding its use in primary tumors and is likely to increase over time. This will increase the well-known rate of wound complications found after radiation.

Questions

1. Which of the following best describes the meaning of en bloc resection?
   (a) Removing a tumor with negative margins.
   (b) It is a pathology term that does not have a surgical meaning.
   (c) It implies the piecemeal removal of a tumor.
   (d) It is a descriptive term used by surgeons when they remove a tumor in one piece.
   Answer (d)

   Explanation: En bloc resection is a surgical term. It means the tumor was removed in one piece. It does not imply a negative margin as the margin status is interpreted by the pathologist. One can remove a tumor in an en bloc fashion and still have a positive margin.
2. Which of the following presentations most closely fits that which one would expect for a patient with osteoblastoma?
   (a) 65-year-old smoker with new onset severe back pain and a pathologic vertebral fracture
   (b) 3-year-old patient with multiple skin lesions and organ dysfunction with a completely collapsed vertebrae
   (c) 21-year-old female with a large, infiltrating lytic lesion of the vertebral body with associated soft tissue mass
   (d) 18-year-old male with 8 months of thoracic back pain and a bone-forming lesion in the posterior elements
   Answer (d)

   Explanation: Osteoblastoma occurs most commonly in the second decade of life. It is a bone-forming lesion, and it is most commonly seen in the posterior elements when it occurs in the spine.
3. All of the following are most likely lytic except?
   (a) Giant cell tumor
   (b) Plasmacytoma
   (c) EG
   (d) Chondrosarcoma
   Answer (d)

   Explanation: Chondrosarcoma often has stippled calcification. This is sometimes described as "popcorn" calcification. It is not usually lytic unless it has dedifferentiated.

References

1. Greenspan A (2004) Orthopedic imaging: a practical approach, 4th edn. Lippincott Williams & Wilkins, Philadelphia, p xvii, 982 p
2. Holland JF (2000) Cancer medicine, 5th edn. B.C. Decker, Hamilton/New York, 2546 p
3. Thomas DM, Skubitz KM (2009) Giant cell tumour of bone. Curr Opin Oncol 21(4):338–344

4. Hart RA, Boriani S, Biagini R, Currier B, Weinstein JN (1997) A system for surgical staging and management of spine tumors. A clinical outcome study of giant cell tumors of the spine. Spine (Phila Pa 1976) 22(15):1773–1782; discussion 83
5. Laredo JD, el Quessar A, Bossard P, Vuillemin-Bodaghi V (2001) Vertebral tumors and pseudotumors. Radiol Clin North Am 39(1):137–163, vi
6. Ozaki T, Liljenqvist U, Halm H, Hillmann A, Gosheger G, Winkelmann W (2002) Giant cell tumor of the spine. Clin Orthop Relat Res 2002–401:194–201
7. Botsford DJ, Esses SI (1991) Normal radionuclide scan in a giant cell tumor of the spine. Orthopedics 14(7):790–792
8. Thomas D, Henshaw R, Skubitz K, Chawla S, Staddon A, Blay JY, Roudier M, Smith J, Ye Z, Sohn W, Dansey R, Jun S (2010) Denosumab in patients with giant-cell tumour of bone: an open-label, phase 2 study. Lancet Oncol 11(3):275–280
9. Mendel E, Bourekas E, Gerszten P, Golan JD (2009) Percutaneous techniques in the treatment of spine tumors: what are the diagnostic and therapeutic indications and outcomes? Spine (Phila Pa 1976) 34(22 Suppl):S93–S100
10. Kato S, Kawahara N, Tomita K, Murakami H, Demura S, Fujimaki Y (2008) Effects on spinal cord blood flow and neurologic function secondary to interruption of bilateral segmental arteries which supply the artery of Adamkiewicz: an experimental study using a dog model. Spine (Phila Pa 1976) 33(14):1533–1541
11. Murakami H, Kawahara N, Tomita K, Demura S, Kato S, Yoshioka K (2010) Does interruption of the artery of Adamkiewicz during total en bloc spondylectomy affect neurologic function? Spine (Phila Pa 1976) 35(22):E1187–E1192
12. Boriani S, Weinstein JN, Biagini R (1997) Primary bone tumors of the spine. Terminology and surgical staging. Spine (Phila Pa 1976) 22(9):1036–1044
13. Enneking W, Dunham W, Gebhardt M, Malawar M, Pritchard D (1990) A system for the classification of skeletal resections. Chir Organi Mov 75(1 Suppl):217–240
14. Boriani S, Bandiera S, Casadei R, Boriani L, Donthineni R, Gasbarrini A, Pignotti E, Biagini R, Schwab JH (2012) Giant cell tumor of the mobile spine: a review of 49 cases. Spine (Phila Pa 1976) 37(1):E37–E45

Metastatic Spine Tumors

32

Byung C. Yoon, Camilo Molina, and Daniel M. Sciubba

32.1 Introduction

Metastatic spine tumors are a frequent problem in patients afflicted with cancer and are often associated with significant morbidity and mortality. The skeletal system is the third most common site of metastasis [62]. Among the skeletal system, the spine is most frequently involved, with autopsy studies showing spinal metastasis in up to 70 % of patients who die of cancer [62]. Thoracic spine lesions comprise the majority of these cases (approximately 70 %) followed by lumbosacral and cervical lesions [71]. Breast cancer is the most common primary tumor to metastasize to the spine, with 16–37 % of breast cancer patients developing spine metastasis. Other common primary tumors include prostate, lung, kidney, and thyroid tumors [71].

Spinal metastases can be intradural or extradural, with the majority being extradural [62]. Tumor cells can spread via various routes including: hematogenous spread, direction extension, and CSF seeding [65]. The hematogenous spread is believed to be most common, either through segmental arteries to the vertebral marrow (e.g., lung cancer) or through the valveless extradural Batson's venous plexus (e.g., breast cancer) [65, 71]. Alternatively, tumors in the thorax, abdomen, and pelvis including lung, prostate, bladder and colorectal cancers can extend or invade directly into the vertebral column [65]. Seeding of tumor cells in the CSF occurs most frequently from surgical manipulation of primary CNS tumors and can result in intradural and intramedullary metastases [65].

B.C. Yoon, M.D., Ph.D. • C. Molina, B.A., M.D.
D.M. Sciubba, M.D. (✉)
Department of Neurosurgery,
The Johns Hopkins Hospital, Johns Hopkins University,
600 N Wolfe Street, Meyer Building 5-185a,
Baltimore, MD 21287, USA
e-mail: dsciubb1@me.com

32.2 Clinical Presentation and Diagnosis

Up to 14 % of patients with spinal metastases, or more than 20,000 patients in the United States annually, develop symptoms from the disease [62]. The most common symptom is pain, present in up to 85–96 % of cases [26, 71]. Pain can be caused from periosteal stretching and inflammation from local tumor growth, which results in constant, dull, nocturnal pain (nocturnal pain is often a "red flag" for tumor or infection) [65]. Spinal metastases can also result in spine instability, which can lead to mechanical pain that worsens with movement or axial loading. Alternatively, they can compress nerve roots and cause radicular pain, that is typically sharp or shooting in character [65]. Other clinical presentations include weakness (in up to 85 % of patients), pathologic fractures (Fig. 32.1), motor and/or sensory deficits, and/or bladder and sphincter dysfunction [2, 65]. In addition, patients with spinal metastasis can present with more

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_32, © Springer-Verlag Berlin Heidelberg 2014

Fig. 32.1 60 year old male with a history of esophageal cancer. Sagittal T1 MRI demonstrating multi-level metastatic spine disease, with the L4 vertebral segment most severely affected by a pathologic fracture

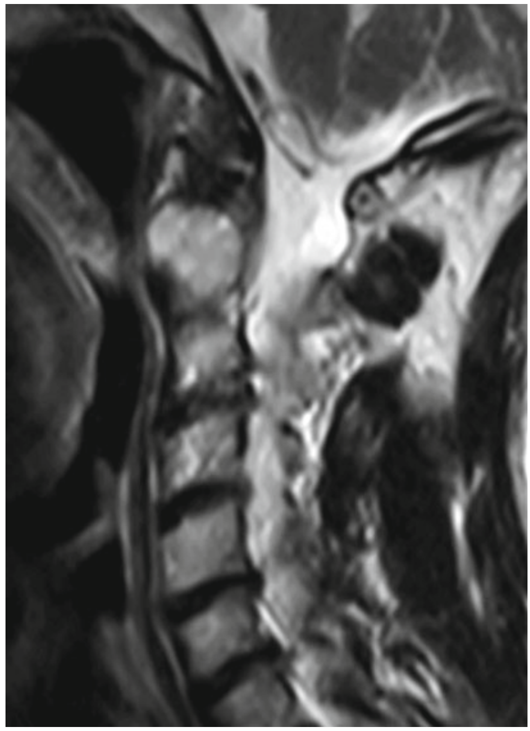


Fig. 32.2 56 year old male with history of metastatic renal cell carcinoma. Sagittal T2 MRI demonstrating a large mass within the C2 vertebral body without significant extension into the spinal canal or cord compression

constitutional symptoms such as weight loss, anorexia, and/or organ dysfunction.

Imaging plays an essential role in diagnosing and evaluating metastatic tumors of the spine. Magnetic resonance imaging (MRI) allows high-resolution imaging of the tumor and surrounding soft tissue (Fig. 32.2). Furthermore, different types of tumors can have characteristic appearances on different MR sequences (i.e., T1 and T2), with and without gadolinium contrast [2, 3, 28, 37, 65, 72]. Computed tomography (CT) is particularly useful in defining the bony anatomy of the spine, assessing the extent of bony involvement and bony destruction [65] (Fig. 32.3). Positron emission tomography (PET) scan can also be used to identify cystic or necrotic tumors [65, 71].

A definitive diagnosis can be made by tissue biopsy. Percutaneous biopsy is frequently used using a transpedicular or transforaminal approach under fluoroscopy or CT guidance [1, 4, 11, 22, 36, 67]. The rates of tissue diagnosis have been reported to be 71–93 % [6, 11, 25, 39, 61]. Percutaneous biopsy is also associated with low complication rates; in one study on CT-guided needle biopsy, only 9 out of 430 biopsies were associated with complications, mostly limited to transient paresis and hematoma [45, 61].

32.3 Management Considerations

Management of metastatic tumor depends on various factors including patient age, comorbidities, tumor type/size, and functional status.

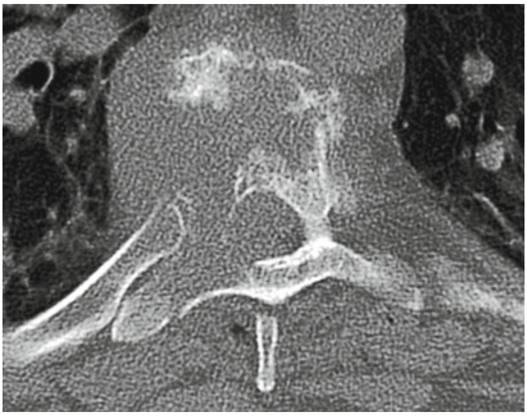


Fig. 32.3 Axial CT reconstruction demonstrating soft tissue infiltration of the vertebral body and pedicle

Several treatment modalities are available including radiation, surgical, and medical therapy. Devising a management plan is therefore a highly individualized process that typically involves a multidisciplinary approach with specialists from oncology, hematology, surgery, pain, and radiology. The extent of treatment with associated morbidities must be balance with the patient's pain, function, and life expectancy.

32.4 Radiation Therapy

External beam radiation therapy (EBRT) is a mainstay of treatment for metastatic spine tumors, especially for pain relief and disease control [20, 65]. The therapy is typically given in daily fractions over 2–4 weeks to minimize damage to the spinal cord [20, 59]. However, the exact radiation regimen is typically individualized to each patient based on a number of factors including their survival prognosis as well as primary tumor pathology and size [58]. EBRT is an effective intervention to reduce morbidities associated with spinal metastasis. In one study, improvement of motor deficit was seen in 25 % of patients with no further progression of compression in 59 % [60]. However, damage to the surrounding tissues from radiation is a concern, particularly in treating tumors that have invaded into the spinal cord [49, 58, 70].

Stereotactic radiosurgery (SRS) allows more precise targeting of the tumor by using multiple radiation beams that are focused on a specific target [33]. This minimizes exposure to adjacent tissues and allows higher doses of radiation to be given per treatment session [33, 49, 65]. Commonly used SR systems include Novalis (BrainLAB), Synergy S (Elekta), and the Cyberknife system (Accuray Inc., California). These systems are capable of irradiating the target region with accuracy of near 1 mm [33]. SRS has been associated with favorable outcomes with studies showing pain reduction and tumor control in 80 % and 90–100 % of cases, respectively [12, 24]. For instance, Gerzten et al. performed a prospective study examining the outcome of SRS treatment for 500 metastatic spinal lesions [24]. The study found pain reduction in 86 % of cases during the follow-up period of 3–53 months and control of radiographically determined tumor progression in 88 % of cases [24]. Adverse effects of SRS include dysphagia, diarrhea, and nocturia but are typically infrequent [12]. Recurrence of tumor near the irradiated site, possibly due to insufficient dose and/or missed lesion, has been reported as well [33].

32.5 Surgical Treatment

The goals of surgery include cord decompression, tumor excision, and spinal stabilization. With advancements in techniques, surgery has become an increasingly important modality in managing patients with metastatic spinal tumors, either as a stand-alone treatment or in combination with other treatment modalities. For instance, a randomized, prospective study by Patchell et al. [54] reported a better outcome in patients who received a combination of EBRT and direct decompressive resection compared to those treated with EBRT alone. Patients who received combination of surgery and EBRT were associated with a higher rate of ambulation (84 % vs. 57 %), greater maintenance of ambulation after treatment (62 % vs. 19 %), longer median survival time (126 days vs. 100 days), and improved functional ability and muscle strength [54].

In general, indications for surgery include spinal instability, rapidly progressing neurologic deficit, and/or radioresistant tumor [2, 71]. However, the decision for surgery often involves careful assessment of surgical risks versus benefit. A number of risk stratification schemes are available for risk assessment in surgical candidates: WBB surgical staging system [5] as well as classification systems developed by DeWald et al. [14] and Enneking et al. [18] [2, 5, 14, 17]. One of the most commonly used scoring systems was developed by Tokuhashi et al. [68] (Table 32.1). The Tokuhashi scoring system assigns up to two points in each of six different categories covering general condition, extraspinal body metastases, spinal metastases, organ metastases, primary site of cancer, and myelopathy [2, 13, 68, 69]. Lesions with scores ≤8 are generally considered nonsurgical [65]. In addition to these factors, life expectancy of >3 months is typically indicated to be a surgical candidate [65, 71].

Several surgical options are available and can be divided largely into open versus minimally invasive approaches. With open surgery, tumors can be approached either anteriorly or posteriorly depending on the location and type of the tumor. Anterior approaches can involve anterolateral cervical approach, sternotomy, and/or thoracotomy and are favorable for approaching metastatic tumors involving the vertebral bodies, which are most frequently involved [65]. However, posterior decompression is preferred in cases where the risk of organ damage is high, particularly for tumors in the upper thoracic and lower lumbosacral regions [57]. Alternatively, a posterolateral approach (e.g., posterolateral costotransversectomy) can be used for anterior decompression in cases where an anterior approach is difficult [71].

Spinal stabilization is required in some cases due to circumferential decompression and/or pathologic fracture (Fig. 32.4). The extent of spinal destabilization that warrants surgical intervention is still debated. However, several schemes are available to help guide decision for surgery. An example is a vertebral strength index developed by Dimar et al., which allows determination of spinal stability based on remaining intact vertebral body and bone density [16]. In general, >50 % loss of vertebral height or increasing angular deformity are considered to be concerning [71].

The outcomes of surgical intervention have been increasingly more favorable, particularly with the use of anterior decompression (Table 32.2). For instance, Kostuik et al. [41] reported neurological improvement in 71 % of cases treated with

Table 32.1 Revised evaluation system for the prognosis of metastatic spine tumors

Characteristic	Score
General condition (performance status)	
Poor (PS 10–40 %)	0
Moderate (PS 50–70 %)	1
Good (PS 80–100 %)	2
No. of extraspinal bone metastases foci	
≥3	0
1–2	1
0	2
No. of metastases in the vertebral body	
≥3	0
1–2	1
0	2
Metastases to the major internal organs	
Unremovable	0
Removable	1
No metastases	2
Primary site of the cancer	
Lung, osteosarcoma, stomach, bladder, esophagus, pancreas	0
Liver, gallbladder, unidentified	1
Others	2
Kidney, uterus	3
Rectum	4
Thyroid, breast, prostate, carcinoid tumor	5
Palsy	
Complete (Frankel A, B)	0
Incomplete (Frankel C, D)	1
None (Frankel E)	2

Adapted from Tokuhashi et al. [68]
Criteria of predicted prognosis: total score (TS) 0–8 < 6 months; TS 9–11 ≥ 6 months; TS 12–15 ≥ 1

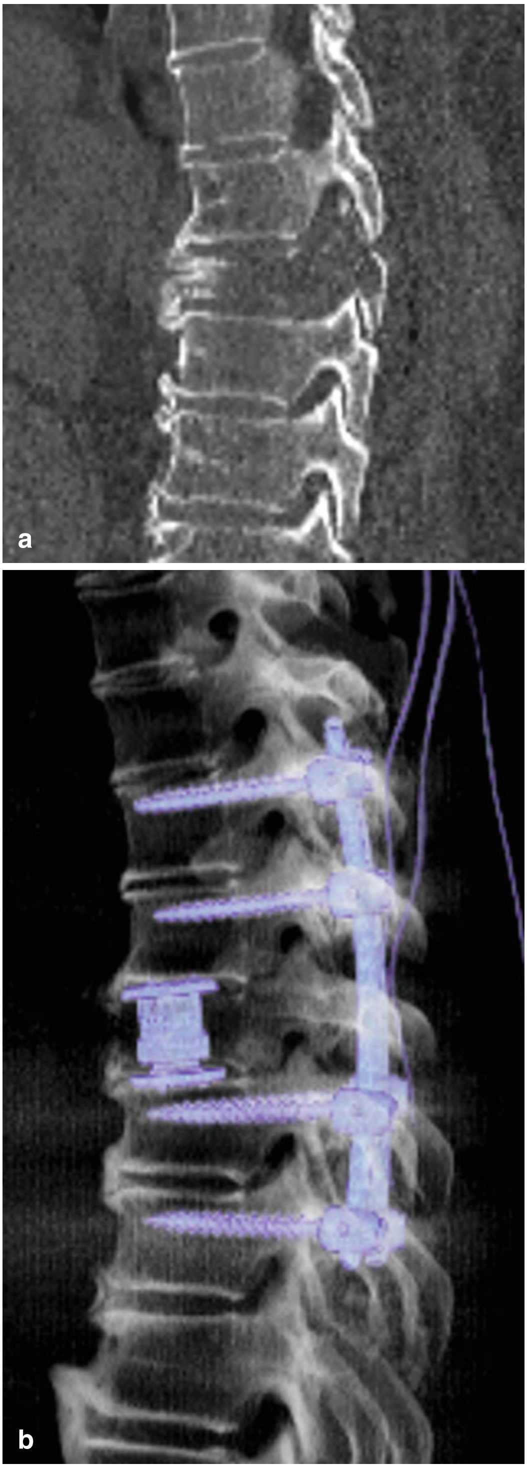


Fig. 32.4 43 year old male with history of multiple myeloma A. Pre-operative sagittal CT reconstruction of the thoracic spine demonstrating a T8 vertebral fracture. B. CT scan status post vertebral corpectomy and pedicle screw and cage reconstruction

anterior decompressions, whereas posterior decompressions were associated with improvements in 40 % of cases. In another study by Overby et al. [53], neurological improvement was observed in 75 % of patients who underwent anterolateral decompression for radiation-refractory metastatic epidural spinal cord compression.

32.5.1 Minimally Invasive Treatment

Minimally invasive approaches include surgical excision of the tumor using endoscopic visualization or minimal access as well as percutaneous vertebral augmentation. The advantages of minimally invasive approaches include decreased surgery-associated trauma and blood loss and shorter length of hospitalization [8, 15, 35, 38].

Endoscopic approaches include video-assisted thoracoscopic surgery (VATS), laparoscopic retroperitoneal approach, and endoscopy-assisted posterolateral decompression. As with open surgery, the type of endoscopic approach is dictated in large part by the location of the tumor [52]. Thoracic vertebrectomy, discectomy, corpectomy, spinal reconstructions, stabilization, and biopsies can be performed using these approaches [30, 46, 48, 52, 63, 71]. These approaches also have been associated with favorable outcomes, particularly for pain reduction. Infrequent complications include transient intercostals neuralgia, atelectasis, trocar injury, and temporary paraparesis. Disadvantages include a steep learning curve, prolonged operative time, and high equipment cost [52].

Minimal access surgeries are modifications of conventional open approaches, where the incision is kept at minimum, and exposure is facilitated by the use of specialized retractor systems (e.g., SynFrame)[22, 52]. Mini-open thoracotomy involves a 5- to 10-cm incision and is used for thoracolumbar tumors [22, 40]. Retroperitoneal mini-approach is preferred for lesions in the lumbar region. Outcomes of minimal access approaches have been promising. A study by Huang et al. [34] showed comparable rates of blood loss, complication rates, and survival rates between minimal access and open thoracotomy

Table 32.2 Outcomes of surgical treatment for metastatic spinal tumors

Study	Surgery type	Patient characteristics	Outcome	Complications
Overby and Rothman [53]	Anterolateral decompression	No—12	Neurological deficit improvement[a]—75 %	DVT—8.3 %
		Age—51.9 (mean)	Improved ambulation—58.3 %	Urinary tract sepsis—8.3 %
		Breast, prostate, lung mets	Restored bladder continence—83.3 %	
Fidler [19]	Anterior decompression and stabilization	No—17	Pain improvement—94.1 %	Local recurrence in 1 case
		Age—53 (mean)	Neuro improvement[b]—93.3 %	
		Pts with pathologic fx	Improved ambulation—82.3 %	
			Restored bladder continence—2/2	
Kostuik et al. [41]	Decompression and stabilization (anterior or posterior)	No—100	Pain improvement—85 %	Instrumentation failure—2 %
		(71 patients with metastatic spinal lesions)	Neurological deficit improvement—71 % (anterior decompression), 40 % (posterior decompression)	Infection—4 %
			Restored bowel/bladder continence—47 %	Pseudoarthrosis—3 %
Gokaslan et al. [27]	Transthoracic vertebrectomy	No—72	Pain improvement—92.3 %	Epidural hematoma (4.2 %), PE (1.4 %), Atelectasis, ileius, AFib, wound infection
		Age—56 (median)	Neuro improvement[b]—76.1	
		Renal, breast, melanoma, sarcoma, lung mets	Restored ambulation—76.9 %	

DVT deep vein thrombosis, *Fx* fractures, *PE* pulmonary embolism, *AFib* atrial fibrillation
[a]Neurological grading based on Brice and McKissock [7]
[b]Neurological function assessed by the Frankel score [23]

for treatment of spine metastasis in T3–T12. Minimal access approach, however, was associated with significantly shorter hospital stay, with only 6.9 % requiring a 2-day ICU stay compared to 88 % in patients treated with standard thoracotomy [34].

32.6 Percutaneous Vertebral Augmentation

Percutaneous vertebroplasty and kyphoplasty can be an effective treatment option for patients with pathologic compression fractures with minimal deformity [71]. Both approaches involve injection of a surgical cement (e.g., polymethylmethacrylate [PMMA]) into the vertebral body [29]. In kyphoplasty, however, a balloon is introduced and inflated within the vertebral body to create a cavity that can be filled with PMMA. Percutaneous vertebral augmentation has shown to result in significant pain reduction in 80–100 % of patients with spinal metastasis [52, 56, 66]. The most frequent complication is cement extravasation (2–73 % of cases)[10, 21, 29, 32, 42, 44, 55]. They are mostly asymptomatic but can cause more adverse effects such as severe paraplegia [43, 45]. Contraindications to percutaneous vertebral augmentation include patients with active infection such as epidural abscess, sepsis, osteomyelitis, and/or uncorrectable coagulopathy [29].

32.7 Medical Treatment

Options for medical therapy in management of spinal metastasis include hormone therapy, chemotherapy, bisphosphonates, corticosteroids, and analgesic agents. With an exception of a few hormonal treatments or chemotherapies, most medical therapy is not curative but is limited to the role of an adjuvant therapy. Hormone therapy can be used in treatment of specific types of tumors such as breast cancer and prostate cancer. For instance, tamoxifen and aromatase inhibitors are used for breast cancer and gonadotropin-releasing hormone agonists for prostate cancer [9, 50]. Chemotherapy can be used postoperatively as or preoperatively as adjuvant or neoadjuvant therapy, respectively. [62, 64, 65]. Bisphosphonates inhibit bone resorption and can help reduce pathologic fractures as well as progression of osteolytic tumors and maintain bone mineral density for patients on hormonal therapy (e.g., prostate cancer patients pharmacologically castrated) [2, 31, 51]. Other medical treatments include corticosteroids for reduction of spinal cord edema and nonsteroidal anti-inflammatory drugs (NSAIDs) and opioids for pain management [13, 47, 65].

32.8 Concluding Remarks

Metastatic spine tumors cause significant morbidity and mortality. Surgical interventions have been playing an increasingly critical role in managing patients with metastatic tumors. Modification of traditional surgical approaches as well as endoscopic techniques has helped with efficient management of symptoms and disease control with reduced procedure-associated morbidity. Further improvements in surgical techniques and other treatment modalities also promise better management of metastatic spine tumors in the future.

References

1. Adapon BD, Legada BD Jr, Lim EV, Silao JV Jr, Dalmacio-Cruz A (1981) CT-guided closed biopsy of the spine. J Comput Assist Tomogr 5(1):73–78
2. Aebi M (2003) Spinal metastasis in the elderly. Eur Spine J 12(Suppl 2):S202–S213. doi:10.1007/s00586-003-0609-9
3. Baur A, Stabler A, Arbogast S, Duerr HR, Bartl R, Reiser M (2002) Acute osteoporotic and neoplastic vertebral compression fractures: fluid sign at MR imaging. Radiology 225:730–735
4. Bender CE, Berquist TH, Wold LE (1986) Imaging-assisted percutaneous biopsy of the thoracic spine. Mayo Clin Proc 61(12):942–950
5. Boriani S, Weinstein JN, Biagini R (1997) Primary bone tumors of the spine. Terminology and surgical staging. Spine (Phila Pa 1976) 22(9):1036–1044
6. Brenac F, Huet H (2001) Diagnostic accuracy of the percutaneous spinal biopsy. Optimization of the technique. J Neuroradiol 28(1):7–16, MDOI-JNR-03-2001-28-1-0150-9861-101019-ART11 [pii]
7. Brice J, McKissock W (1965) Surgical treatment of malignant extradural spinal tumours. Br Med J 1(5446):1341–1344
8. Connelly CS, Manges PA (1998) Video-assisted thoracoscopic discectomy and fusion. AORN J 67(5):940–945, 947–948, 950 passim quiz 953–946
9. Conti PD, Atallah AN, Arruda H, Soares BG, El Dib RP, Wilt TJ (2007) Intermittent versus continuous androgen suppression for prostatic cancer. Cochrane Database Syst Rev (4):CD005009. doi:10.1002/14651858.CD005009.pub2
10. Cotten A, Dewatre F, Cortet B, Assaker R, Leblond D, Duquesnoy B, Chastanet P, Clarisse J (1996) Percutaneous vertebroplasty for osteolytic metastases and myeloma: effects of the percentage of lesion filling and the leakage of methyl methacrylate at clinical follow-up. Radiology 200(2):525–530
11. Dave BR, Nanda A, Anandjiwala JV (2009) Transpedicular percutaneous biopsy of vertebral body lesions: a series of 71 cases. Spinal Cord 47(5):384–389. doi:10.1038/sc.2008.108, sc2008108 [pii]
12. Degen JW, Gagnon GJ, Voyadzis JM, McRae DA, Lunsden M, Dieterich S, Molzahn I, Henderson FC (2005) CyberKnife stereotactic radiosurgical treatment of spinal tumors for pain control and quality of life. J Neurosurg Spine 2(5):540–549. doi:10.3171/spi.2005.2.5.0540
13. Delank KS, Wendtner C, Eich HT, Eysel P (2011) The treatment of spinal metastases. Dtsch Arztebl Int 108(5):71–80. doi:10.3238/arztebl.2011.0071
14. DeWald RL, Bridwell KH, Prodromas C, Rodts MF (1985) Reconstructive spinal surgery as palliation for metastatic malignancies of the spine. Spine (Phila Pa 1976) 10(1):21–26
15. Dickman CA, Rosenthal D, Karahalios DG, Paramore CG, Mican CA, Apostolides PJ, Lorenz R, Sonntag VK (1996) Thoracic vertebrectomy and reconstruction using a microsurgical thoracoscopic approach. Neurosurgery 38(2):279–293
16. Dimar JR 2nd, Voor MJ, Zhang YM, Glassman SD (1998) A human cadaver model for determination of pathologic fracture threshold resulting from tumorous destruction of the vertebral body. Spine (Phila Pa 1976) 23(11):1209–1214

17. Enneking WF, Spanier SS, Goodman MA (1980) A system for the surgical staging of musculoskeletal sarcoma. Clin Orthop Relat Res 153:106–120
18. Enneking WF, Spanier SS, Goodman MA (2003) A system for the surgical staging of musculoskeletal sarcoma. 1980. Clin Orthop Relat Res (415):4–18. doi:10.1097/01.blo.0000093891.12372.0f
19. Fidler MW (1986) Anterior decompression and stabilisation of metastatic spinal fractures. J Bone Joint Surg Br 68(1):83–90
20. Finn MA, Vrionis FD, Schmidt MH (2007) Spinal radiosurgery for metastatic disease of the spine. Cancer Control 14(4):405–411
21. Fourney DR, Schomer DF, Nader R, Chlan-Fourney J, Suki D, Ahrar K, Rhines LD, Gokaslan ZL (2003) Percutaneous vertebroplasty and kyphoplasty for painful vertebral body fractures in cancer patients. J Neurosurg 98(1 Suppl):21–30
22. Frangou E, Fourney DR (2009) Minimally invasive treatment of spinal tumors. Sem Spine Surg 21(2): 112–120
23. Frankel HL, Hancock DO, Hyslop G, Melzak J, Michaelis LS, Ungar GH, Vernon JD, Walsh JJ (1969) The value of postural reduction in the initial management of closed injuries of the spine with paraplegia and tetraplegia I. Paraplegia 7(3): 179–192
24. Gerszten PC, Burton SA, Ozhasoglu C, Welch WC (2007) Radiosurgery for spinal metastases: clinical experience in 500 cases from a single institution. Spine (Phila Pa 1976) 32(2):193–199. doi:10.1097/01.brs.0000251863.76595.a2, 00007632-200701150-00009 [pii]
25. Ghelman B, Lospinuso MF, Levine DB, O'Leary PF, Burke SW (1991) Percutaneous computed-tomography-guided biopsy of the thoracic and lumbar spine. Spine (Phila Pa 1976) 16(7):736–739
26. Gilbert RW, Kim JH, Posner JB (1978) Epidural spinal cord compression from metastatic tumor: diagnosis and treatment. Ann Neurol 3(1):40–51. doi:10.1002/ana.410030107
27. Gokaslan ZL, York JE, Walsh GL, McCutcheon IE, Lang FF, Putnam JB Jr, Wildrick DM, Swisher SG, Abi-Said D, Sawaya R (1998) Transthoracic vertebrectomy for metastatic spinal tumors. J Neurosurg 89(4):599–609. doi:10.3171/jns.1998.89.4.0599
28. Grossman RI, Yousem DM (1994) Neuroradiology. The requisites. Mosby Year Book, St Louis, pp 491–495
29. Halpin RJ, Bendok BR, Liu JC (2004) Minimally invasive treatments for spinal metastases: vertebroplasty, kyphoplasty, and radiofrequency ablation. J Support Oncol 2(4):339–351; discussion 352–335
30. Han PP, Kenny K, Dickman CA (2002) Thoracoscopic approaches to the thoracic spine: experience with 241 surgical procedures. Neurosurgery 51(5 Suppl): S88–S95
31. Hillner BE, Ingle JN, Berenson JR, Janjan NA, Albain KS, Lipton A, Yee G, Biermann JS, Chlebowski RT, Pfister DG (2000) American Society of Clinical Oncology guideline on the role of bisphosphonates in breast cancer. American Society of Clinical Oncology Bisphosphonates Expert Panel. J Clin Oncol 18(6):1378–1391
32. Hodler J, Peck D, Gilula LA (2003) Midterm outcome after vertebroplasty: predictive value of technical and patient-related factors. Radiology 227(3):662–668. doi:10.1148/radiol.2273011930, 2273011930 [pii]
33. Hsu W, Nguyen T, Kleinberg L, Ford EC, Rigamonti D, Gokaslan ZL, Lim M (2010) Stereotactic radiosurgery for spine tumors: review of current literature. Stereotact Funct Neurosurg 88(5):315–321. doi:10.1159/000319959, 000319959 [pii]
34. Huang TJ, Hsu RW, Li YY, Cheng CC (2006) Minimal access spinal surgery (MASS) in treating thoracic spine metastasis. Spine (Phila Pa 1976) 31(16):1860–1863. doi:10.1097/01.brs.0000225995.56028.46, 00007632-200607150-00020 [pii]
35. Huang TJ, Hsu RW, Liu HP, Hsu KY, Liao YS, Shih HN, Chen YJ (1997) Video-assisted thoracoscopic treatment of spinal lesions in the thoracolumbar junction. Surg Endosc 11(12):1189–1193
36. Jelinek JS, Kransdorf MJ, Gray R, Aboulafia AJ, Malawer MM (1996) Percutaneous transpedicular biopsy of vertebral body lesions. Spine (Phila Pa 1976) 21(17):2035–2040
37. Jung HS, Jee WH, McCauley TR, Ha KY, Choi KH (2003) Discrimination of metastatic from acute osteoporotic compression spinal fractures with MR imaging. Radiographics 23(1):179–187
38. Kim SJ, Sohn MJ, Ryoo JY, Kim YS, Whang CJ (2007) Clinical Analysis of Video-assisted Thoracoscopic Spinal Surgery in the Thoracic or Thoracolumbar Spinal Pathologies. J Korean Neurosurg Soc 42(4):293–299. doi:10.3340/jkns.2007.42.4.293
39. Kornblum MB, Wesolowski DP, Fischgrund JS, Herkowitz HN (1998) Computed tomography-guided biopsy of the spine. A review of 103 patients. Spine (Phila Pa 1976) 23(1):81–85
40. Kossmann T, Jacobi D, Trentz O (2001) The use of a retractor system (SynFrame) for open, minimal invasive reconstruction of the anterior column of the thoracic and lumbar spine. Eur Spine J 10(5):396–402
41. Kostuik JP, Errico TJ, Gleason TF, Errico CC (1988) Spinal stabilization of vertebral column tumors. Spine (Phila Pa 1976) 13(3):250–256
42. Ledlie JT, Renfro M (2003) Balloon kyphoplasty: one-year outcomes in vertebral body height restoration, chronic pain, and activity levels. J Neurosurg 98(1 Suppl):36–42
43. Lee BJ, Lee SR, Yoo TY (2002) Paraplegia as a complication of percutaneous vertebroplasty with polymethylmethacrylate: a case report. Spine (Phila Pa 1976) 27(19):E419–E422. doi:10.1097/01.BRS.0000025697.64547.BE
44. Lieberman IH, Dudeney S, Reinhardt MK, Bell G (2001) Initial outcome and efficacy of "kyphoplasty" in the treatment of painful osteoporotic vertebral compression fractures. Spine (Phila Pa 1976) 26(14):1631–1638

45. Lopes NM, Lopes VK (2004) Paraplegia complicating percutaneous vertebroplasty for osteoporotic vertebral fracture: case report. Arq Neuropsiquiatr 62(3B):879–881, S0004-282X2004000500027 [pii]/S0004-282X2004000500027
46. Mack MJ, Regan JJ, Bobechko WP, Acuff TE (1993) Application of thoracoscopy for diseases of the spine. Ann Thorac Surg 56(3):736–738
47. Maxwell T (2000) Cancer pain management in the elderly. Geriatr Nurs 21(3):158–163. doi:10.1067/mgn.2000.108267, S0197-4572(00)37320-7 [pii]
48. McAfee PC, Regan JR, Fedder IL (1995) Anterior thoracic corpectomy for spinal cord compression performed endoscopically. Surg Laparosc Endosc 5(5):339–344
49. Mell LK, Mundt AJ (2005) Radiation therapy in the elderly. Cancer J 11(6):495–505
50. Morandi P, Rouzier R, Altundag K, Buzdar AU, Theriault RL, Hortobagyi G (2004) The role of aromatase inhibitors in the adjuvant treatment of breast carcinoma: the M. D. Anderson Cancer Center evidence-based approach. Cancer 101(7):1482–1489. doi:10.1002/cncr.20522
51. Neville-Webbe HL, Coleman RE (2010) Bisphosphonates and RANK ligand inhibitors for the treatment and prevention of metastatic bone disease. Eur J Cancer 46(7):1211–1222. doi:10.1016/j.ejca.2010.02.041, S0959-8049(10)00168-1 [pii]
52. Ofluoglu O (2009) Minimally invasive management of spinal metastases. Orthop Clin North Am 40(1):155–168. doi:10.1016/j.ocl.2008.09.006, S0030-5898(08)00071-0 [pii]
53. Overby MC, Rothman AS (1985) Anterolateral decompression for metastatic epidural spinal cord tumors. Results of a modified costotransversectomy approach. J Neurosurg 62(3):344–348. doi:10.3171/jns.1985.62.3.0344
54. Patchell RA, Tibbs PA, Regine WF, Payne R, Saris S, Kryscio RJ, Mohiuddin M, Young B (2005) Direct decompressive surgical resection in the treatment of spinal cord compression caused by metastatic cancer: a randomised trial. Lancet 366(9486):643–648. doi:10.1016/S0140-6736(05)66954-1, S0140-6736(05)66954-1 [pii]
55. Peters KR, Guiot BH, Martin PA, Fessler RG (2002) Vertebroplasty for osteoporotic compression fractures: current practice and evolving techniques. Neurosurgery 51(5 Suppl):S96–S103
56. Pflugmacher R, Beth P, Schroeder RJ, Schaser KD, Melcher I (2007) Balloon kyphoplasty for the treatment of pathological fractures in the thoracic and lumbar spine caused by metastasis: one-year follow-up. Acta Radiol 48(1):89–95. doi:10.1080/02841850601026427, 771214513 [pii]
57. Quraishi NA, Gokaslan ZL, Boriani S (2010) The surgical management of metastatic epidural compression of the spinal cord. J Bone Joint Surg Br 92(8):1054–1060. doi:10.1302/0301-620X.92B8.22296, 92-B/8/1054 [pii]
58. Rades D, Abrahm JL (2010) The role of radiotherapy for metastatic epidural spinal cord compression. Nat Rev Clin Oncol 7(10):590–598. doi:10.1038/nrclinonc.2010.137, nrclinonc.2010.137 [pii]
59. Rades D, Heidenreich F, Karstens JH (2002) Final results of a prospective study of the prognostic value of the time to develop motor deficits before irradiation in metastatic spinal cord compression. Int J Radiat Oncol Biol Phys 53(4):975–979, S0360301602028195 [pii]
60. Rades D, Hoskin PJ, Karstens JH, Rudat V, Veninga T, Stalpers LJ, Schild SE, Dunst J (2007) Radiotherapy of metastatic spinal cord compression in very elderly patients. Int J Radiat Oncol Biol Phys 67(1):256–263. doi:10.1016/j.ijrobp.2006.08.011, S0360-3016(06)02742-8 [pii]
61. Rimondi E, Staals EL, Errani C, Bianchi G, Casadei R, Alberghini M, Malaguti MC, Rossi G, Durante S, Mercuri M (2008) Percutaneous CT-guided biopsy of the spine: results of 430 biopsies. Eur Spine J 17(7):975–981. doi:10.1007/s00586-008-0678-x
62. Rose PS, Buchowski JM (2011) Metastatic disease in the thoracic and lumbar spine: evaluation and management. J Am Acad Orthop Surg 19(1):37–48. doi:19/1/37[pii]
63. Rosenthal D, Marquardt G, Lorenz R, Nichtweiss M (1996) Anterior decompression and stabilization using a microsurgical endoscopic technique for metastatic tumors of the thoracic spine. J Neurosurg 84(4):565–572. doi:10.3171/jns.1996.84.4.0565
64. Rusch VW, Giroux DJ, Kraut MJ, Crowley J, Hazuka M, Johnson D, Goldberg M, Detterbeck F, Shepherd F, Burkes R, Winton T, Deschamps C, Livingston R, Gandara D (2001) Induction chemoradiation and surgical resection for non-small cell lung carcinomas of the superior sulcus: initial results of Southwest Oncology Group Trial 9416 (Intergroup Trial 0160). J Thorac Cardiovasc Surg 121(3):472–483. doi:10.1067/mtc.2001.112465, S0022-5223(01)61659-3 [pii]
65. Sciubba DM, Petteys RJ, Dekutoski MB, Fisher CG, Fehlings MG, Ondra SL, Rhines LD, Gokaslan ZL (2010) Diagnosis and management of metastatic spine disease. J Neurosurg Spine 13(1):94–108. doi:10.3171/2010.3.SPINE09202
66. Shimony JS, Gilula LA, Zeller AJ, Brown DB (2004) Percutaneous vertebroplasty for malignant compression fractures with epidural involvement. Radiology 232(3):846–853. doi:10.1148/radiol.2323030353, 2323030353 [pii]
67. Sucu HK, Bezircioglu H, Cicek C, Ersahin Y (2003) Computerized tomography-guided percutaneous transforaminodiscal biopsy sampling of vertebral body lesions. J Neurosurg 99(1 Suppl):51–55
68. Tokuhashi Y, Matsuzaki H, Oda H, Oshima M, Ryu J (2005) A revised scoring system for preoperative evaluation of metastatic spine tumor prognosis. Spine (Phila Pa 1976) 30(19):2186–2191, 00007632-200510010-00011[pii]
69. Tokuhashi Y, Matsuzaki H, Toriyama S, Kawano H, Ohsaka S (1990) Scoring system for the preoperative evaluation of metastatic spine tumor prognosis. Spine (Phila Pa 1976) 15(11):1110–1113

70. Wasil T, Lichtman SM, Gupta V, Rush S (2000) Radiation therapy in cancer patients 80 years of age and older. Am J Clin Oncol 23(5):526–530
71. White AP, Kwon BK, Lindskog DM, Friedlaender GE, Grauer JN (2006) Metastatic disease of the spine. J Am Acad Orthop Surg 14(11):587–598. doi:14/11/587[pii]
72. Yuh WT, Zachar CK, Barloon TJ, Sato Y, Sickels WJ, Hawes DR (1989) Vertebral compression fractures: distinction between benign and malignant causes with MR imaging. Radiology 172(1):215–218

33 Primary Spinal Infections

David B. Bumpass and Jacob M. Buchowski

33.1 Case Example

A 48-year-old male presented with 2 months of increasing thoracic spine and bilateral shoulder pain. He had known osteoarthritis of the right shoulder and had received two intra-articular steroid injections in the past, the most recent 7 months prior to presentation. He denied any radiation of pain into the upper or lower extremities and had not experienced weakness, sensory loss, balance problems, or bowel/bladder incontinence. His spinal pain was made worse with coughing, deep breathing, and bending forward. Review of systems revealed that the patient has experienced subjective fevers and intermittent night sweats.

On physical exam, the patient had tenderness to palpation in the upper thoracic spine. He did not display any motor weakness in the upper or lower extremities. Sensation was intact throughout. Reflexes were 1+ symmetrically. He displayed no clonus, negative Hoffmann and Babinski signs, and a negative Romberg test.

D.B. Bumpass, M.D.
Department of Orthopaedic Surgery, Washington University in St. Louis,
St. Louis, MO, USA

J.M. Buchowski, M.D., MS (✉)
Center for Spinal Tumors,
Department of Orthopaedic Surgery,
Washington University in St. Louis,
660 S. Euclid Ave., Campus Box 8233,
St. Louis, MO 63110, USA
e-mail: buchowskij@wustl.edu

Plain film radiographs of the cervical and thoracic spine demonstrated slight disk space narrowing at T2–T3 (Fig. 33.1). Cervical and thoracic magnetic resonance imaging (MRI) studies with and without gadolinium revealed a lesion at T2–T3 involving both the vertebral bodies and the intervertebral disk. This lesion extended anteriorly into the mediastinum adjacent to the esophagus and lung pleura. The lesion also extended posteriorly into the epidural space, creating stenosis of the spinal canal.

Laboratory studies demonstrated a white blood cell (WBC) count of 10.9, an erythrocyte sedimentation rate (ESR) of 51.0, and a C-reactive protein level of 71.9.

A computed tomography (CT)-guided biopsy of the lesion was performed, and the specimen sent for histological and microbiological analysis. Cultures were positive for methicillin-sensitive *Staphylococcal aureus* (MSSA). No malignant tissue was seen on histology.

The patient was diagnosed with vertebral osteomyelitis and diskitis at T2–T3, with a small epidural abscess extending from the vertebral infection. Intravenous antibiotic therapy was initiated with ceftriaxone and maintained for 6 weeks. After 6 weeks, the patient's pain was much improved, he was not experiencing fevers, and he felt able to return to work. On exam, he had no neurologic deficits. A repeat thoracic spine MRI showed progressive destruction of the vertebral end plates but improvement in the extent of the prevertebral and epidural infection.

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_33, © Springer-Verlag Berlin Heidelberg 2014

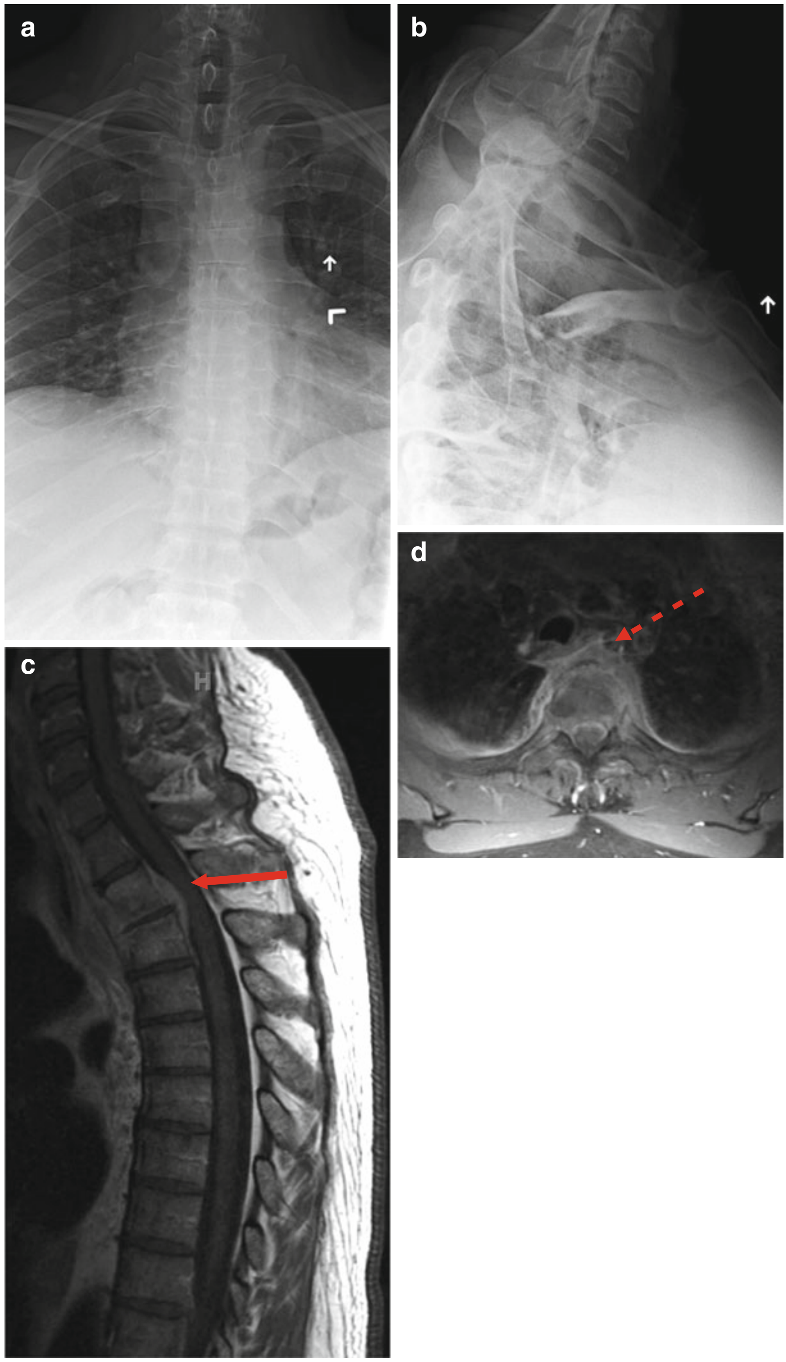


Fig. 33.1 Images of a 48-year-old male with T2–T3 vertebral osteomyelitis with epidural abscess formation, as well as T2–T3 diskitis. He presented with 2 months of increasing upper thoracic spine pain, made worse by coughing, deep breathing, and bending forward. He also had subjective fevers and night sweats. No neurologic deficits were present on physical exam, but he did have tenderness to palpation of the upper thoracic spine. Plain film radiographs (**a**, **b**) demonstrate only slight disk space narrowing at T2–T3. T1 (**c**) and T2 (**d**) MRI images demonstrated abnormal signal within the T2 and T3 vertebral bodies, narrowing of the disk space, and an abscess extension into the epidural space (*solid arrow*) as well as the posterior mediastinum (*dashed arrow*). A technetium-99 bone scan (**e**) showed increased tracer uptake in the upper thoracic spine. CT-guided biopsy yielded *Staphylococcus aureus*. He was treated with intravenous antibiotics

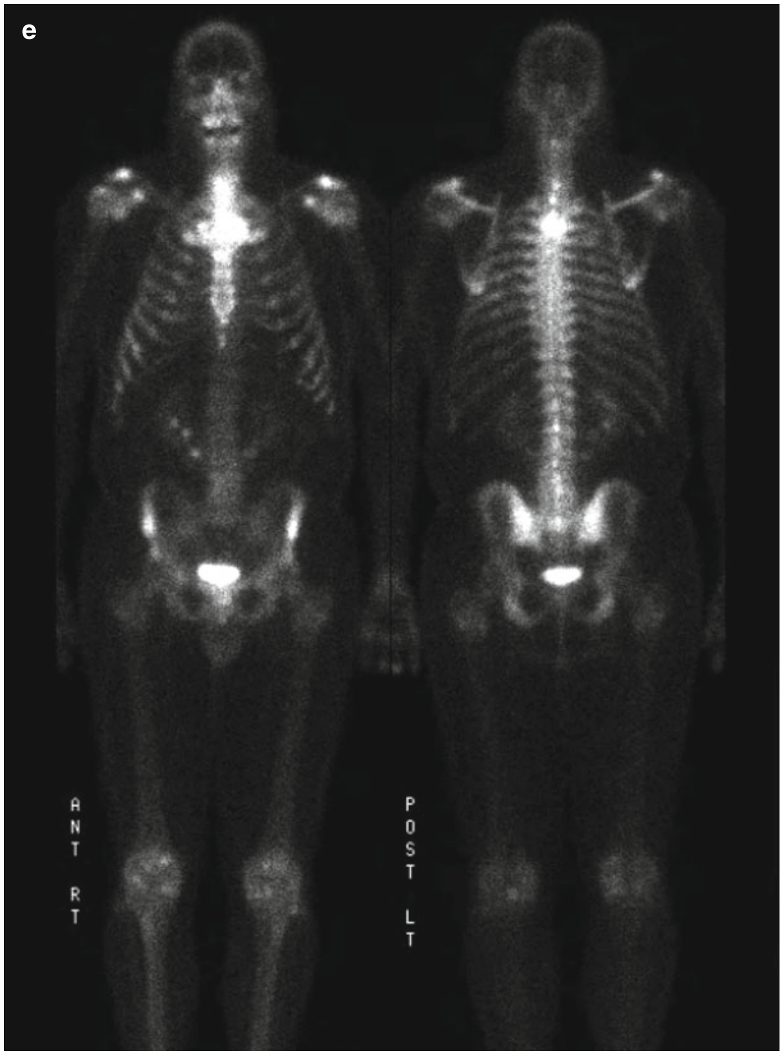


Fig. 33.1 (continued)

33.2 Differential Diagnosis for Spinal Infections

Vertebral osteomyelitis vs. diskitis vs. epidural abscess
Pott's disease (tuberculous infection of the spine)
Disk herniation
Degenerative disk disease
Primary spinal tumor
Metastatic tumor
Epidural hematoma
Transverse myelitis
Syringomyelia
Spinal cord infarction

33.3 Vertebral Osteomyelitis

33.3.1 Epidemiology and Pathophysiology

Between 2 and 4 % of all pyogenic osteomyelitis cases occur in the vertebrae [69]. Prior to the advent of antibiotic medications, mortality from vertebral osteomyelitis was nearly one in every four patients [45]. This condition is more common in men and in the elderly, with peak incidence during the seventh decade [7, 16, 44]. Incidence of vertebral osteomyelitis is thought to be increasing, due in part to intravenous drug abuse and the use of long-term intravenous access devices in elderly patients [73, 74]. Carragee found that 40 % of patients in his series had immune suppression resulting from diabetes mellitus, corticosteroid use, chemotherapy for cancer or rheumatologic conditions, hepatic or renal failure, myelodysplasia, or malnutrition. He also found that 10 % of the patients in his series had prior radiation therapy to the spine for metastatic cancer [7]. Infective endocarditis is also a risk factor for development of vertebral osteomyelitis; spinal infection occurs in approximately 10 % of endocarditis cases and is sometimes the presenting sign of a cardiac valve infection [52].

While most common in adults, vertebral osteomyelitis can occur in neonates and children as well and must be included in the differential diagnosis for occult infection and back pain in these patients [19, 23, 36]. Significant neurologic deficits have been reported in neonates with osteomyelitis [26].

The lumbar spine is the most common site of adult pyogenic osteomyelitis [7, 16, 25, 44, 74]. In a series by Krogsgaard et al. [44], 59 % of cases were in the lumbar spine, 33 % were thoracic, and 8 % were cervical [44]. Most patients have involvement of only a single motion segment (two vertebrae and the interposed disk), but up to 13 % of cases can involve multiple motion segments [7, 16].

Vertebral osteomyelitis typically begins with hematogenous seeding to the vertebral body. There are two possible routes. One is arterial, with bacteria entering the vertebral bodies through the nutrient arterioles [80]. The other is venous, via the valveless perivertebral venous plexus [2]. This venous plexus is in communication with the pelvic venous plexus, explaining how urinary and pelvic infections can indirectly seed the vertebral column via retrograde venous flow [2, 80]. Similarly, there is a prevertebral venous plexus in the cervical spine that may function as a hematogenous route for peripharyngeal infections to travel [56]. The posterior spinal elements are rarely involved in vertebral osteomyelitis [25].

Once a vertebra is infected with osteomyelitis, direct local spread can occur [73]. The avascular disk can be destroyed by the infection, which can then extend into the adjacent vertebra. Cervical infections can breach the prevertebral fascia and spread into the mediastinum or supraclavicular fossa. Lumbar infections can track along the psoas muscle, even reaching the piriformis fossa or perianal spaces. If the infection penetrates into the subarachnoid space, then meningitis results.

33.3.2 History and Physical Exam

Clinical presentation varies depending on the location and extent of the infection, but back pain is seen in more than 85 % of patients with vertebral osteomyelitis [7, 74]. There is often paraspinal muscle spasm as well. Pain is not necessarily dependent on activity level and often wakes the patient from sleep at night. A significant number of patients are afebrile at presentation, with case series ranging from 15 to 66 % of patients [7, 25, 74]. Adjacent soft tissue infections may also cause symptoms, such as a psoas abscess causing pain with hip extension or a paravertebral cervical abscess causing dysphagia or torticollis [73].

Neurologic deficits of varying severity are common. Patients are more likely to suffer neurologic injury from vertebral osteomyelitis if they have diabetes mellitus or rheumatoid arthritis, are elderly, are using systemic steroids, or have a more cephalad level of infection [20]. Torda et al. [74] found that 11 of 20 (55 %) patients had limb weakness at presentation. Of these 11 patients, 4 also had dysesthesias, 3 had objective sensory loss, and 3 had urinary retention [74]. Carragee 1997 found that 9 of 111 (8 %) of patients developed complete spinal cord injury at some point during their disease course. An additional 21 patients (19 %) had incomplete neurologic deficits; 11 were cauda equina lesions, 2 were conus lesions, and 8 were cord lesions [7].

Previous studies have found that between 27 and 46 % of patients had an identifiable primary source of infection [7, 44]. Of these, the genitourinary tract was the source in up to 43 % of cases; Krogsgaard et al. [44] found that 18 of 137 (13 %) patients had a genitourinary procedure in the 3 months prior to the diagnosis of vertebral osteomyelitis [7, 16, 20, 44]. Torda et al. [74] found that 8 of 20 patients had vertebral osteomyelitis from intravenous cannula-related sepsis [74].

33.3.3 Laboratory Studies and Imaging

The most commonly isolated bacterium in vertebral osteomyelitis is *Staphylococcus aureus*. Torda et al. [74] identified *Staph aureus* as the causative organism in 68 % of cases, almost half of which were methicillin-resistant *Staph aureus* (MRSA) [74]. Genitourinary source infections are often caused by *Escherichia coli* and *Proteus mirabilis*. Patients who abuse intravenous drugs can present with *Pseudomonas aeruginosa* osteomyelitis [79]. Coagulase-negative *Staphylococcus* species, *Propionibacterium acnes*, and *Streptococcus viridans* can cause more indolent presentations of vertebral osteomyelitis and should not be dismissed as contaminants; *Strep viridans* is the most common organism in infections seeded from endocarditis [7, 52]. Children with sickle cell anemia frequently have *Salmonella* osteomyelitis [73].

In patients with normal immune systems, Carragee reported elevation of WBC count in 44 % of patients and elevation of ESR in 100 %. However, in patients with suppressed immune systems, only 30 % had elevated WBC counts and 89 % had elevated ESR values [7]. Blood cultures are positive in approximately 30–60 % of patients [7, 16, 74]. Cultures from bone biopsies are positive in up to 73 % of cases [74]. Core biopsy is preferable to fine needle aspiration, as a better specimen is obtained. Open biopsy should be performed if the locus of infection is not accessible to radiographically guided biopsy, when significant bone damage with neurologic injury is present, or when closed biopsy has not yielded a causative organism. During the diagnostic workup, cultures of blood, urine, and bone should be obtained prior to initiation of antibiotic therapy unless the patient is septic [73]. Cultures should be kept for at least 10 days to detect less-virulent bacteria. All tissue cultures should also be sent to pathology for analysis to rule out malignant or metabolic bone diseases and to stain for acid-fast bacilli and fungi. An echocardiogram should be done to rule out endocarditis as a source of infection, particularly in patients with known valvular defects [52].

Imaging is crucial in establishing the diagnosis of vertebral osteomyelitis (Fig. 33.2). Torda et al. [74] found plain radiographic abnormalities in all 20 (100 %) of their patients, most commonly loss of disk height [74]. However, only eight (40 %) patients demonstrated bone or disk destruction. In infants, radiographs may initially be normal, but the infection can rapidly progress to demonstrating complete or near-complete obliteration of the affected vertebral bodies [19]. Children with vertebral osteomyelitis have normal initial radiographs in 46 % of cases [23].

MRI has been reported to have a sensitivity for vertebral osteomyelitis of 96 % and a specificity of 92 %, making this the imaging modality of choice [51]. Use of MRI has been shown to be diagnostic even within the first 2 weeks of symptoms, significantly shortening the time to diagnosis [6]. Usually, vertebral osteomyelitis also causes signal changes of the adjacent intervertebral disk on MRI; if no disk involvement is seen, then neoplasm, tuberculosis, and fungal infection should be ruled out with further laboratory tests, as these processes usually do not involve the disk [1, 40]. Technetium-99 bone scans have been shown to be positive in almost 90 % of patients, but are not highly specific as they cannot differentiate infection from fracture or malignancy [51, 74].

33.3.4 Nonsurgical Treatment

Intravenous antibiotics are the standard initial treatment for vertebral osteomyelitis, once appropriate cultures have been obtained. Generally, at least 6–8 weeks of antibiotic therapy is needed [75]. In a series of 20 patients, Torda et al. [74] cited a mean duration of intravenous antibiotic therapy of 34 days. Subsequently, oral antibiotic therapy was maintained for between 21 and 240 days. Three of their 20 patients (15 %) developed relapse of infection, were treated with repeat courses of intravenous and oral antibiotics, and did not have further recurrences [74]. MRSA osteomyelitis may respond better to a combination treatment of intravenous vancomycin and oral rifampin, which animal studies have shown to have better penetration into bone [17, 30]. Predictors for successful nonoperative treatment are age less than 60 years, a healthy immune status, and a downtrending ESR [7]. Malnutrition is often seen in patients with vertebral osteomyelitis and should be treated to maximize the patient's chances of eliminating infection. A serum albumin of <3 g/dL, absolute lymphocyte count of <800/mL, and serum transferrin of <150 µg/dL all are indicative of malnutrition [73]. If after 4 weeks of antibiotic therapy, the patient has not clinically improved, has persistently elevated inflammatory markers, or has an MRI showing no improvement in the extent of infection and

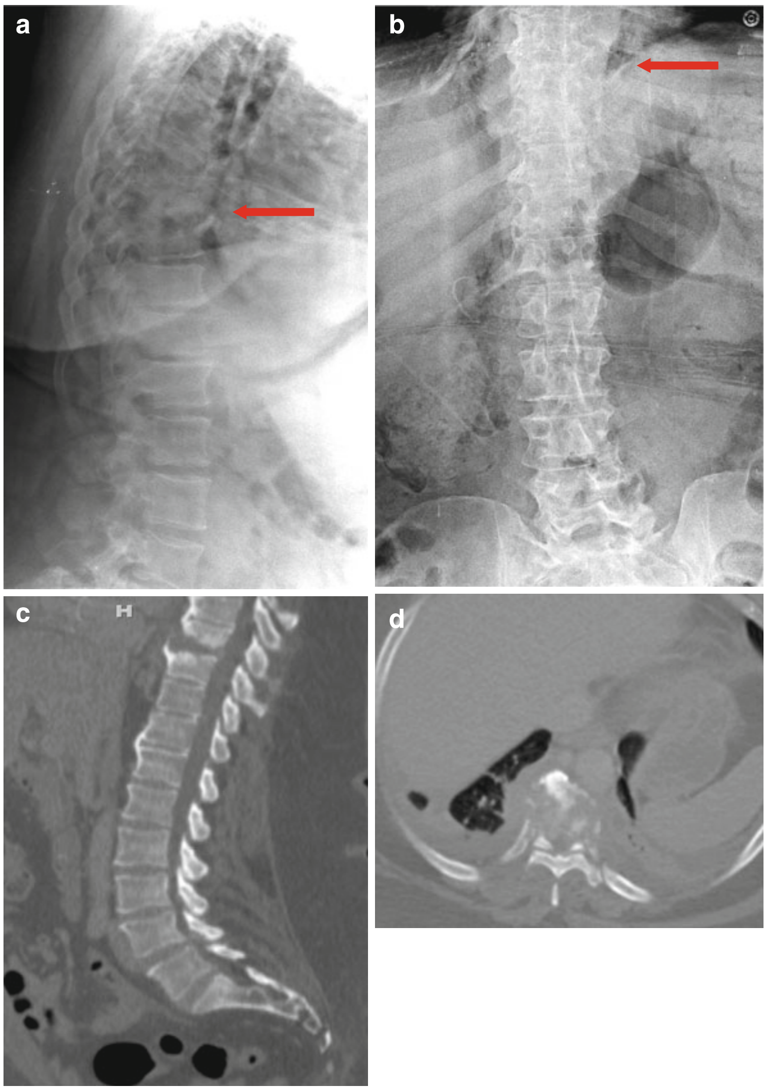
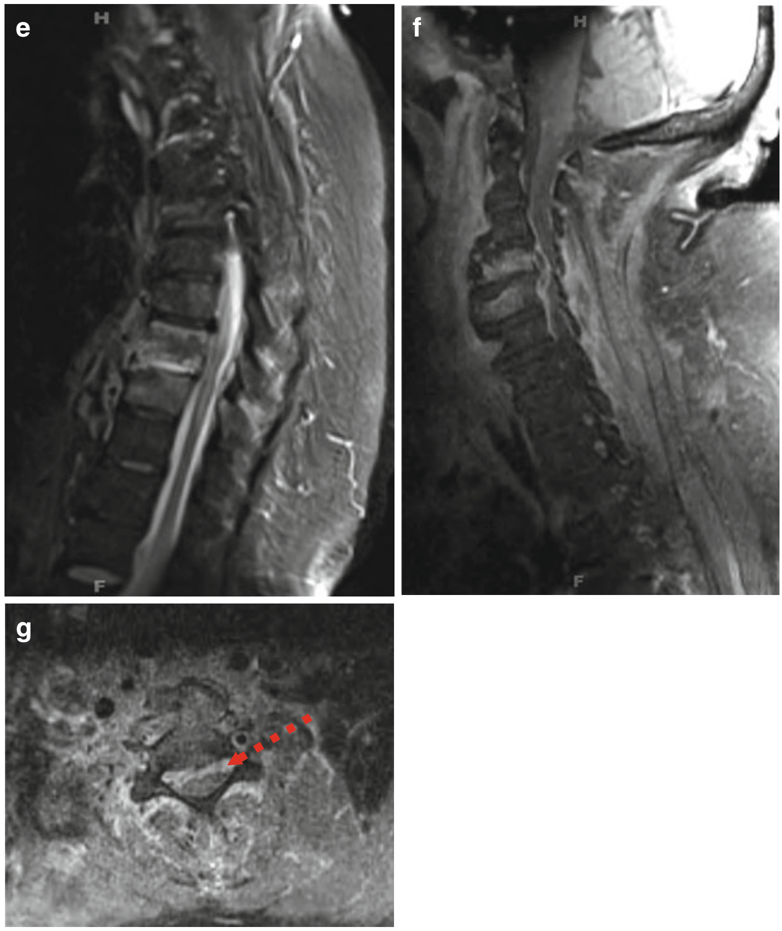


Fig. 33.2 Images of a 51-year-old female who initially presented with progressive bilateral lower extremity paralysis over several months. Radiographs (**a**,**b**) and a CT scan (**c**, **d**) revealed T8–T9 vertebral osteomyelitis and diskitis (*solid arrows*). An MRI STIR sequence (**e**) demonstrated increased signal intensity of the vertebral bodies and disk, as well as mild cord signal change. A bone biopsy yielded MRSA, and the patient was treated with intravenous antibiotics as her neurologic lesion was complete and she had multiple medical comorbidities. Four months later, the patient presented with bilateral upper extremity paralysis. T1 MRI with gadolinium (**f**, **g**) demonstrated osteomyelitis at C4–C5 with an anterior epidural abscess (*dashed arrow*). An anterior C5 corpectomy was initially performed to decompress the spine, followed by a posterior C3–C6 fusion

Fig. 33.2 (continued)



no progression of vertebral fusion, then operative treatment should be considered [75].

In infants and children, aggressive antibiotic therapy should be maintained for several weeks. Often the infection results in destruction of part or all of a vertebral body, leaving the child with a hemivertebra or segmentation failure. Bracing should be used to prevent development of severe short-segment kyphosis or scoliosis [19].

33.3.5 Surgical Treatment

Surgical treatment for vertebral osteomyelitis is often necessary either acutely or after failure of medical management, particularly when neurologic deficits or spinal instability is present [75]. Torda et al. [74] reported that 5 of 20 (25 %) patients required surgical debridement [74]. Because the infection is generally within the vertebral body, an anterior surgical approach is typically utilized. Emery et al. [22] reported excellent results in 21 patients who underwent anterior debridement with autogenous bone grafting. Six of these patients had preoperative neurologic deficits (Five were Frankel grade D, and one was Frankel grade C); all six recovered motor and sensory function. No patients had recurrence of infection, and only one patient developed a pseudarthrosis. No significant postoperative kyphotic deformities occurred [22]. Eismont et al. [20] found better results in osteomyelitis patients presenting with paralysis who underwent anterior debridement rather than laminectomy, both in the cervical and lumbar spine [20]. Recently, Gonzalvo et al. [27] challenged the superiority of anterior debridement in a report of nine patients with vertebral osteomyelitis and only minor neurologic deficits (Frankel grades D and E), all of whom were treated with simultaneous posterior debridement and single-level fusion. No patients required subsequent surgeries, and all patients had successful fusions after a mean 5-year follow-up period [27]. Infections in the thoracic spine can be treated either with costotransversectomy or thoracotomy [20].

While there is concern over placing metal implants in an infected space, there are case series demonstrating low risk of persistent infection with instrumentation in place. Carragee treated 42 of 111 (38 %) patients with surgical debridement and arthrodesis; instrumentation was placed in 14 of these patients, and all had successful outcomes despite presence of infection at time of implantation [7]. Korovessis et al. [43] placed titanium cages at the site of vertebral debridement in 17 patients with osteomyelitis via combined anterior/posterior approaches. At a mean follow-up of 4 years, no patients had recurrent vertebral infections, loss of sagittal correction, or cage migration [43].

33.3.6 Prognosis

Mortality has improved with use of antibiotics, and in recent series, it is often difficult to determine if those patients who died succumbed because of the vertebral infection or from other comorbidities. In the first several decades after the advent of antibiotics, series of vertebral osteomyelitis patients reported mortality rates between 0 and 5 % [25, 70, 80]. However, more recently Carragee reported that 18 of 111 (16 %) patients died, 10 patients within 6 months of diagnosis and 8 of those within 1 month. Most had serious comorbidities. Perhaps this reflects the increasing danger of antibiotic-resistant forms of bacteria, particularly MRSA.

Morbidity from vertebral osteomyelitis can be significant. Torda et al. reported that out of 20 patients, two (10 %) had residual urinary retention and lower extremity weakness. Other noted complications included bacterial endocarditis, hepatic abscess, and ischiorectal abscess [74]. In Carragee's series, of the nine (8 %) patients who experienced a complete spinal cord injury, four fully recovered, one had Frankel grade D function, two had grade C function, and two died without recovering spinal function. Of the 21 (19 %) incomplete neurologic lesions, nine completely resolved, and twelve had permanent partial injury. Approximately 10 % of patients have chronic severe back pain at 2-year follow-up [7].

33.4 Epidural Abscesses

33.4.1 Epidemiology and Pathophysiology

Epidural abscesses are potentially life-threatening infections with high risk for neurologic impairment, and so prompt diagnosis is critical. Incidence of epidural abscesses is highest in the sixth and seventh decades of life and is more common in men by a ratio of approximately two to one [3, 10, 15]. Diabetes mellitus, intravenous drug abuse, spine trauma, paraspinal abscess, skin ulcers, and cellulitis have all been identified as predisposing factors [10, 13, 39, 42, 58]. Endocarditis is another well-documented source of infection for epidural abscesses [10, 21]. Also, epidural catheters for anesthesia or analgesia are a risk factor for development of epidural abscesses, and any back pain or new neurologic deficits in patients with these catheters should be thoroughly evaluated for possible epidural infection [5, 41, 64].

The epidural space is not an uninterrupted space between the spinal ligaments and dura but rather an organized and septated arrangement of spaces along the spinal cord. Anterior to the spinal cord, the dura is directly adjacent to the posterior longitudinal ligament, resulting in a potential rather than an actual space. Posterior and lateral to the cord, there are segmental actual epidural spaces [32]. The clinical importance of this anatomy is that epidural abscesses typically do not form circumferentially and they usually only span a few vertebral segments. Series have reported that abscesses span a mean of 3.5 vertebral body levels, with a range of 1–6 levels; at least 65–70 % of abscesses are ventral to the spinal cord, while 17–23 % are dorsal and only 12–14 % are circumferential [10, 39, 46]. Epidural abscesses can form at any level along the spine, with different series reporting varying distributions of infections [10, 15, 39, 66].

33.4.2 History and Physical Examination

As with osteomyelitis, back pain is the most common presenting symptom, affecting more than 70 % of patients. The pain is typically severe and unrelenting [3, 46, 58]. Approximately 60 % of patients are febrile at presentation [58]. Neurologic deficits at time of presentation occur in approximately 57–75 % of patients [10, 39]. These include radiculopathy, bowel or bladder dysfunction, paresis, and plegia. In a large meta-analysis, [58] found that 31 % of patients presented with paraparesis or paraplegia [58].

33.4.3 Laboratory Studies and Imaging

Elevated WBC counts are present in more than 50 % of cases, and the ESR is nearly always elevated [54, 58, 60]. Blood cultures are positive in 35–69 % of cases [10, 13, 46], and abscess cultures from aspiration or biopsy are positive in approximately 85 % of cases [10, 13]. Cerebrospinal fluid (CSF) was drawn in 30 of 35 patients in a series by Danner and Hartman [13], and only two of these patients grew bacteria from the CSF. One of these displayed clinical signs of meningitis [13]. Thus, lumbar puncture should only be performed in patients with symptoms consistent with meningitis, as penetrating the dura near an epidural abscess may introduce bacteria into the spinal fluid [58].

Staphylococcus aureus is the causative organism in 45–65 % of cases [10, 13–15, 39]. However, Gram-negative rods and anaerobic bacteria may be increasingly common causative agents of epidural abscesses [14, 15].

MRI is the most useful imaging modality, as it has a high diagnostic accuracy for epidural abscess [54, 60] (Fig. 33.3). The abscess is hypointense or isointense on T1-weighted images and hyperintense on T2-weighted images [60]. Use of gadolinium contrast further improves the capability of MRI to differentiate abscesses from surrounding tissues [63]. MRI can delineate between abscess and tumor, disk herniation, spinal cord infarction, or other spinal pathologies and does not require a lumbar puncture as with CT myelography [58]. If MRI is contraindicated, then CT myelogram is also an excellent study, although there is risk of introducing bacteria into the intradural space if needle placement is near a purulent collection.

In one report, 73 % of patients with epidural abscesses also had MRI signal abnormalities consistent with adjacent diskitis and/or vertebral osteomyelitis [10]; an anterior abscess location has independently been shown to be a risk factor for development of these other loci of spinal infection [66]. Identification of the extent of infection is crucial in establishing a proper treatment plan.

33.4.4 Treatment

Emergent surgical debridement followed by antibiotic therapy remains the standard of care [54]. Surgical approach should be dictated by the location of the abscess. In series of operative treatment for epidural abscesses, most cases were performed via posterior laminectomy. However, anterior debridement with corpectomy sometimes is required if there is a large anterior abscess, particularly in the cervical spine (Fig. 33.2) [58, 60]. Del Curling et al. [15] reported that only 2 of 29 patients initially treated operatively required repeat debridements; in 84 % of their cases, the surgical wound was primarily closed.

Duration of antibiotic therapy is typically at least 2–6 weeks of intravenous agents, sometimes followed by a course of oral antibiotics [15, 60, 66]. Choice of antibiotic is dictated by bacterial cultures. Duration of treatment should

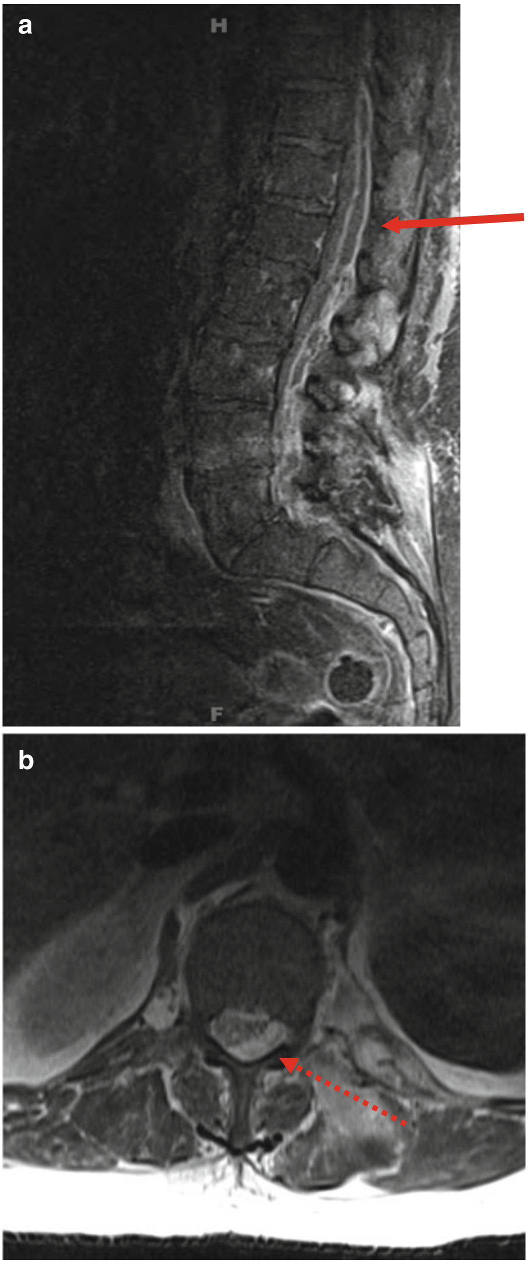


Fig. 33.3 Images of a 64-year-old male with a thoracolumbar epidural abscess. He presented with 3 weeks of increasing pain and progressive left leg weakness. Initially he received a left hip intra-articular injection, as this was thought to be the origin of his pain. He subsequently became febrile and also developed mental status changes and constipation. Sagittal T1 MRI with contrast (**a**) demonstrated a ring-enhancing posterior epidural lesion extending from T11 to L5 that compressed the spinal sac (*solid arrow*). Axial T2 MRI without contrast (**b**) further elucidated the degree of spinal sac compression (*dashed arrow*). After operative debridement via an L1-L4 hemilaminectomy, cultures grew *Staphylococcus aureus*. The patient recovered full neurological function

be guided by the patient's symptoms, serial WBC and ESR values, and follow-up imaging [60, 66]. Adjunctive administration of corticosteroids to patients with epidural abscesses has been shown to negatively impact neurologic recovery [13].

Successful percutaneous drainage has also been described for posterior thoracolumbar and lumbar epidural abscesses [12, 34, 48, 72]. In some of these cases, CT-guided needle aspiration was sufficient to decompress the abscess [48]. Other patients were treated by placement of an epidural catheter using fluoroscopic guidance, and in certain cases, irrigation was pumped through these catheters to facilitate clearance of the infection [34, 72]. Intravenous antibiotics were initiated as adjunctive treatment in all cases.

Some authors have advocated nonoperative treatment for epidural abscesses [39, 49, 53, 78]. In these small series, patients had no neurologic deficits and typically had an identified organism from blood or abscess cultures. The patients were treated with 4–8 weeks of parenteral antibiotics. Most had complete recoveries, but several still required surgical debridement. However, other studies have shown that patients treated with antibiotic therapy alone have significantly greater chance for unfavorable outcome, even if these patients selected for nonoperative treatment did not initially have sepsis or neurologic deficits [10, 31]. Harrington et al. [28] stated that nonoperative treatment should only be attempted in patients with no neurologic deficits, a clearly identified causative organism, a clearly decreasing WBC count and ESR with antibiotic therapy, percutaneous drainage of any identified abscess collections, and capability to closely observe for and operatively treat the patient if sudden neurologic changes arise [28].

33.4.5 Prognosis

Neurologic improvement is more likely with shorter duration of symptoms, prompt initiation of treatment, and less severe initial neurologic deficits [13, 31, 58]. Predictors of poor outcome from epidural abscesses include advanced age, severity of thecal sac compression, and sepsis [39]. Curry et al. [10] reported neurologic improvement in 38 % of patients, deterioration in 29 %, and no change in 33 % [10]. Mortality ranged from 7 to 20 % in various recent series [13, 15, 39, 58].

33.5 Diskitis

33.5.1 Pediatric Diskitis

33.5.1.1 Epidemiology and Pathophysiology

Pediatric diskitis is generally considered a separate pathological entity from vertebral osteomyelitis, although arguments have been made that both are manifestations of what should be called pyogenic spondylitis [61]. Typically, pediatric diskitis occurs in children younger than 5 years. The lumbar spine is most commonly affected, and only rarely is more than one disk involved [4, 50, 61, 77]. The disk is seeded hematogenously, as vascular channels exist in the cartilaginous end plates of the pediatric vertebrae that allow bacteria to invade the annulus fibrosus from the adjacent vertebral physeal blood supply. These vascular channels close once the physes ossify, explaining why diskitis is more common in young children than adults [29, 62, 68]. Also, in children, there is collateral flow among the intraosseous arteries within the vertebral bodies that persists until adolescence. Infarction and bacterial deposition are therefore much less likely within the mid-body than at the vertebral end plates, resulting in vertebral osteomyelitis occurring less frequently in young children than adults [57].

Differential diagnosis should include vertebral osteomyelitis, leukemia, sacroiliac joint septic arthritis, Scheuermann's kyphosis, eosinophilic granuloma (characterized by vertebra plana on plain films), and osteoid osteoma or osteoblastoma (generally found in the posterior elements) [18]. Children with vertebral osteomyelitis are often older (mean age 7.5 years for osteomyelitis vs. 2.8 years for diskitis), more likely to be febrile, and in many cases appear systemically ill [23].

33.5.1.2 History and Physical Examination

Clinically, a history of a recent illness or infection is common [50, 77]. Approximately 25 % of patients present with fever >38 °C [61]. The child can present with neck, back, thigh, or abdominal pain. Unwillingness to bear weight is often observed, and so diskitis must be considered in any young child who refuses to stand or walk. Paravertebral muscle spasm, local tenderness to palpation, and loss of lumbar lordosis may be present as well [4, 18, 50, 61, 77].

33.5.1.3 Laboratory Studies and Imaging

In pediatric diskitis, WBC count is normal in 90 % of patients, but ESR is nearly always elevated [11, 61]. Blood cultures are negative in nearly 90 % of patients as well [23]. Positive culture rates of disk biopsies range from 37 to 60 % of patients. The most common causative organism is *Staph aureus* [18, 77]. However, [26] found *Kingella kingae* in 17 % of children who underwent biopsy, and this Gram-negative species may be an increasingly common cause of pediatric musculoskeletal infections [26, 47]. Since disk culture is often not conclusive and involves a procedure, and because MRI provides a powerful diagnostic tool, biopsy is not frequently performed for pediatric diskitis unless patients fail to respond clinically to empiric antibiotic treatment [18, 61].

Approximately 75 % of children with diskitis will display changes on plain radiographs [9, 23]. Several stages seen on plain radiographs have been described, distinct from osteomyelitis: initial disk space narrowing, then new subperiosteal bone formation at the end plates, followed by end-plate erosion and sclerosis. Sometimes the disk space then balloons, and there is notable vertebral body erosion [11, 38]. Technetium-99 bone scans are positive in more than 92 % of patients [33]. MRI has been shown to have a sensitivity of 93 % and specificity of 97 % for diskitis and detects disk changes at least several days before plain films and bone scans [71]. T1-weighted sequences show decreased signal at an end plate-disk-end plate unit, while T2-weighted sequences show increased signal [61]. Also, MRI best demonstrates epidural or paraspinal abscesses; up to 75 % of children with diskitis will also have an inflammatory paravertebral mass [4].

33.5.1.4 Treatment

Currently accepted treatment for pediatric diskitis is intravenous antibiotics. Rest and bracing are often used in conjunction with antibiotics for symptomatic relief, but the preponderance of data suggests that intravenous antibiotics shorten duration of symptoms and reduce recurrence [61]. In a review of case series published since 1990, total length of treatment should be at least 4–6 weeks, with 1–2 weeks of parenteral therapy followed by 4–6 weeks of oral antibiotics [4, 23, 26, 61, 68]. Choice in antibiotic is generally empirical and should cover *Staph aureus*; good success has been documented with cefazolin or nafcillin intravenously and cephalexin or dicloxacillin orally [61]. Clinical improvement and serial CRP measurements should be used to evaluate progress, as CRP normalizes more rapidly than ESR with successful antibiotic treatment [18]. A thoracolumbosacral orthosis can provide significant relief; 4 weeks is a typical duration for wear [11, 18, 68]. Surgical debridement or drainage is performed only when the patient fails to improve on appropriate antibiotic therapy, becomes septic, or has a progressing neurologic deficit [18]. Even paravertebral abscesses do not necessarily require drainage and can improve with antibiotic therapy alone [61]. Once treatment has been completed, the patient should be followed periodically for 12–18 months to insure that symptoms do not recur.

33.5.1.5 Prognosis

Outcomes for children with diskitis are excellent. Brown et al. [4] cited a series of patients, all who were initially treated with intravenous antibiotics for 2 weeks, followed by oral antibiotics for a minimum of another 2 weeks. Half of the patients also wore a brace. More than 50 % of the patients had improvement in their symptoms in less than 4 days, and an additional 45 % experienced relief in less than 3 weeks. At a mean of 21 months, all children who followed up were symptom-free [4]. Crawford et al. [9] presented a series of 36 children with diskitis, all who eventually were asymptomatic. Of note, 74 % of these children still had vertebral changes on plain radiographs even once they were entirely asymptomatic, suggesting that radiographs alone should not be used to dictate the duration of treatment [9].

33.5.2 Adult Diskitis

33.5.2.1 Epidemiology and Pathophysiology

Adult diskitis most commonly occurs after an invasive spinal procedure but can occur spontaneously as well [33, 38]. However, spontaneous diskitis is much less common than in children. The vascular channels through the vertebral end plates have closed, thus reducing the available routes for bacteria to reach the disk spaces from the vertebral body blood supply [29]. Diskitis can develop easily from contiguous vertebral osteomyelitis or an epidural abscess. Mean age in multiple series is in the 50 and 60s [33, 38]. The lumbar spine is most commonly affected [33].

33.5.2.2 History and Physical Examination

Adult patients typically complain primarily of back pain, and radicular symptoms are common [38]. The pain often is subacute, having been present for weeks or even months prior to diagnosis [38]. More than 25 % of patients have a history of prior bacteremia [33]. Neurologic deficits in absence of a concurrent epidural abscess or extensive vertebral osteomyelitis are rare.

33.5.2.3 Laboratory Studies and Imaging

Laboratory tests follow trends similar to pediatric diskitis. ESR is elevated in nearly all cases. Most cases of adult diskitis are caused by *Staph aureus* or Gram-negative rods [33]. In a series of 16 patients, [33] reported that 12 of these patients underwent disk aspiration and 10 of these patients (83 %) had positive aspirate cultures. Ten patients (63 %) had negative blood cultures at time of diagnosis [33].

MRI is the best choice of imaging to assist with diagnosis. As with children, technetium-99 bone scans are also highly sensitive and can be used in patients who cannot undergo MRI scanning [33]. Plain radiographs should be obtained to rule out any spinal deformity that has resulted from the infection, but often only a loss of disk height is seen.

33.5.2.4 Treatment

Nearly all patients can be treated nonoperatively with intravenous antibiotics. A typical course of treatment lasts 6–8 weeks. Choice of antibiotic should be determined by culture sensitivities [33]. Surgical treatment is typically indicated only when neurologic deficit is present, and in these cases, an epidural abscess or other cause of spinal cord compression is likely present.

33.5.2.5 Prognosis

Generally, appropriate antibiotic treatment alone is curative in adults with diskitis. Honan et al. [33] reported a 94 % success rate, with the one exception being a patient who died of unrelated causes. However, Kemp et al. [38] described three patients who presented with complete paralysis from diskitis, and only one recovered. The authors noted that significant granulation tissue from the disk space had invaded the spinal canal in the patients who presented with complete paralysis. Thus, close monitoring of these patients is necessary during treatment to insure that they are not worsening.

33.6 Atypical Spinal Infections

33.6.1 Tuberculosis

Tuberculosis (TB) has become an increasingly common infection in the United States over the past two decades, due in part to the rise of HIV, development of drug-resistant mycobacteria, and an influx of immigrants from countries with high rates of TB [59]. The spine is the most common site of extrapulmonary disease in TB patients. Spinal TB infection was first described by British surgeon Percivall Pott in 1779 and is known today as Pott's disease [35].

Unlike pyogenic infections, back pain caused by TB is more insidious and is often accompanied by fevers, malaise, weight loss, and night sweats. A flank mass is commonly palpable as well. Complicating diagnosis is the fact that 80 % of spinal TB patients do not have active pulmonary disease [59, 65]. However, the diagnosis of Pott's disease should be considered in any patient presenting with back pain and fevers who has lived in countries with high rates of TB or those with immunodeficiencies such as HIV.

The bacteria usually reach the spine via hematogenous spread from a focus of infection in the lungs [65]. There are two major patterns of spinal TB infection. One is destruction of contiguous vertebrae, including the intervertebral disks; often there is a paraspinal abscess as well (Fig. 33.4). The other form of infection spares the intervertebral disk, instead tracking along the anterior longitudinal ligament to involve the next adjacent vertebral body [65]. The thoracic spine is most commonly involved [76]. Infection often results in kyphotic deformity and neurologic deficits. Psoas abscess formation is common in spinal TB, occurring in more than 40 % of patients [76]. Gadolinium-enhanced MRI is useful to differentiate abscesses from granulation tissue (ring enhancing vs. diffuse enhancement, respectively) [65].

Up to 60 % of spinal TB patients have neurologic compromise, and this is caused by abscess extension into the epidural space, retropulsion of necrotic bone, or from the progression of deformity [73, 76]. Lab tests should include WBC count and ESR, as well as a purified protein derivative (PPD) skin test. WBC count is often not elevated, and ESR is normal in about 25 % of cases, in contrast to pyogenic spinal infections [73]. Cultures can take 6 weeks to grow, often resulting in slow diagnosis. PCR is highly sensitive and specific for detection of *M. tuberculosis* in formalin-fixed tissue samples. Ziehl-Neelsen acid-fast staining of histology specimens is useful, as is the identification of caseating granulomas and giant cell formation [65].

Initial treatment for spinal TB is multidrug antibiotic therapy consisting of isoniazid, rifampin, ethambutol, and pyrazinamide, typically

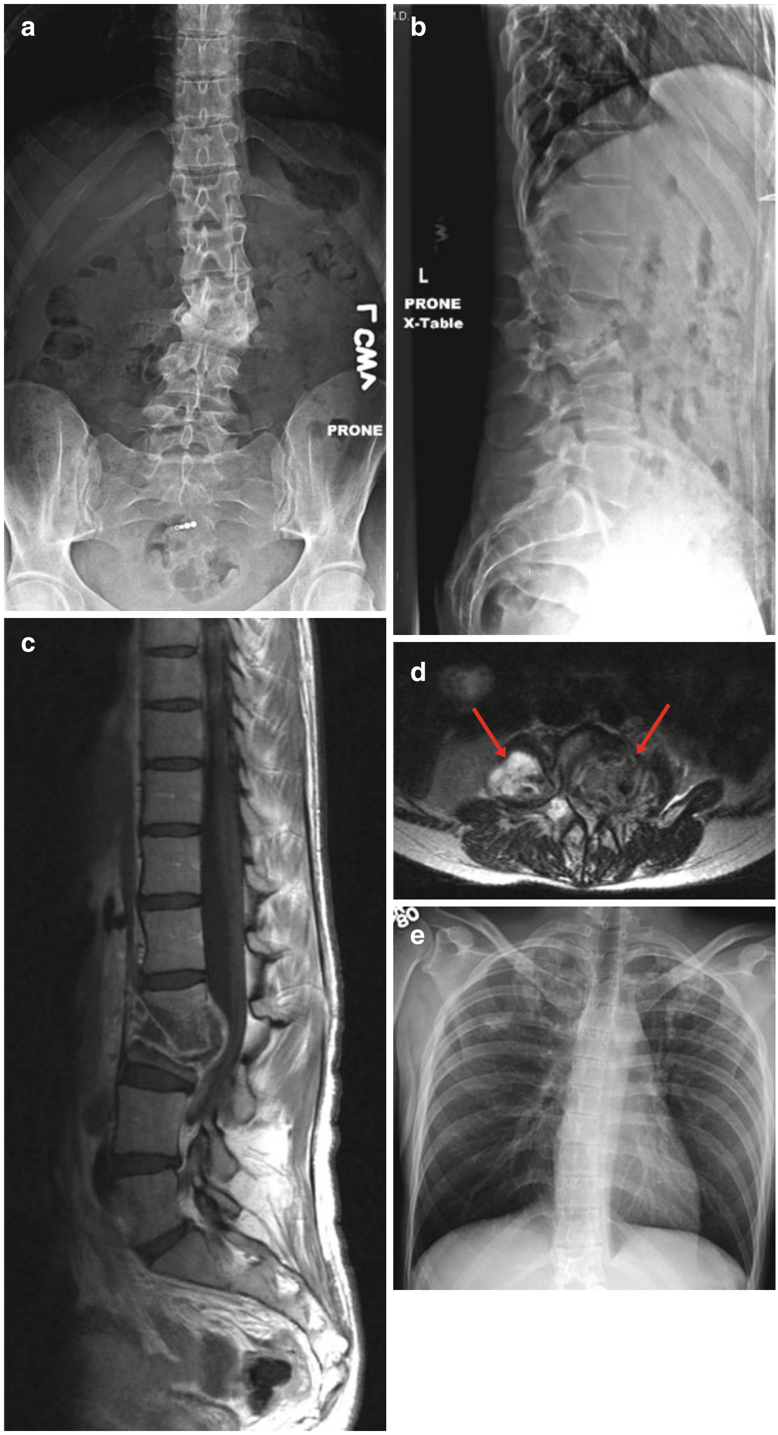


Fig. 33.4 Images of a 23-year-old female with spinal tuberculosis, or Pott's disease. She presented with several months of low back pain, night sweats, a productive cough, and unintentional weight loss of 20 lbs. She was incarcerated several years ago. Tuberculin skin testing was positive. Plain radiographs (**a**, **b**) demonstrated collapse of the L2 and L3 vertebral bodies, with resulting loss of lumbar lordosis. T1 (**c**) and T2 (**d**) MRI scans revealed retropulsion of the vertebral fragments into the spinal canal, as well as development of large bilateral psoas abscesses (*arrows*). A chest radiograph (**e**) and chest CT (**f**) demonstrated calcifications in both lung apices, consistent with granuloma formation

Fig. 33.4 (continued)

for 6–9 months. Pyridoxine is given adjunctively to prevent vitamin B6 deficiency. Infectious disease consultation is imperative, as patterns of mycobacterial drug resistance are regional [65]. Patients with severe or progressive deformity, neurologic deficits, unresponsiveness to medication, or non-diagnostic biopsy are candidates for surgical treatment [59]. The Hong Kong procedure is the classic surgical treatment for spinal TB and consists of anterior debridement, spinal canal decompression, kyphotic deformity correction, and fusion using autograft (iliac crest or fibula) for strut support of the affected segment(s) [8]. Adjunctive posterior instrumentation is often placed, but posterior laminectomy alone will compound the deformity by weakening the posterior elements. Successful treatment with placement of anterior instrumentation in addition to strut grafts has also been described, without recurrent or persistent infection from colonized implants [35, 81].

33.6.2 Brucellosis

Brucellosis, caused by *Brucella* bacterial species transmitted by cattle, goats, and hogs, is the most prevalent zoonotic infection worldwide. Generally human infection results from contact with infected meat or unpasteurized milk products. While uncommon in the United States, Mexico has a high incidence of the disease; other geographic regions with high infection rates include the Middle East and Central Asia [55]. Brucellosis can cause significant musculoskeletal complaints such as sacroiliitis and back pain, as well as fever, weight loss, and malaise [67]. Spinal infection commonly affects the anterior inferior vertebral end plate, particularly in the lower lumbar spine. Generally only a single spinal level is affected. Blood cultures are frequently positive, aiding diagnosis [65]. Also, serological tests such as the standard agglutination test and Coombs test are nearly always positive [67]. Microbiological staining demonstrates Gram-negative coccobacilli and negative acid-fast staining, and histology demonstrates non-caseating granulomas [65]. Treatment for brucellosis is appropriate antibiotic therapy; commonly this consists of doxycycline and intramuscular gentamicin or streptomycin for 2–3 weeks, then doxycycline combined with rifampin or trimethoprim/sulfamethoxazole for up to 6 months [65]. Immobilization with bracing also is helpful for pain relief. Frequent follow-up is necessary, as recurrence is common. Surgical intervention does not play a major role in brucellosis spondylitis but is indicated in rare cases [37].

33.6.3 Fungal Infections

Fungal infections can also occur in the spine. Patients who are immunocompromised have long-term central venous catheters, or those receiving parenteral nutrition are at increased risk [24, 73]. Causative species include *Candida, Blastomyces, Coccidioides, Cryptococcus,* and *Aspergillus.* The spine is the most common location for osseous dissemination in blastomycosis and cryptococcal infections [40]. Diagnosis can be difficult; [24] cited a mean time from symptom onset to treatment of more than 3 months [24]. Patients are often afebrile, and the WBC count is frequently within normal ranges [24]. Fungal culture and potassium hydroxide (KOH) staining from blood and tissue biopsy specimens may yield a causative organism but often are negative. Rather, antigen detection assays are much more sensitive and specific tests. Polymerase chain reaction (PCR) is proving to be increasingly helpful with fungal infection diagnosis as well. Histology can demonstrate yeast or hyphae from biopsy tissue or aspirate [40, 65]. MRI is very useful for diagnosing the extent of infection, and typically the intervertebral disks are spared in fungal osteomyelitis [40]. Kyphosis is uncommon, as these infections are indolent in nature; however, compression fractures can eventually occur [65]. While nonoperative management can be attempted with intravenous antifungal drugs, successful treatment often requires operative debridement as well. Generally, the anterior spine is the site of involvement, so debridement via a posterior approach requires costotransversectomy or extracavitary approach. An anterior approach provides direct visualization of and access to the infected vertebral bodies [40].

Frazier et al. presented a series of 11 patients with fungal spine infections; eight experienced some degree of paralysis as a result of infection. Causative organisms included *Candida, Aspergillus,* and *Coccidioides*. Ten patients were treated with surgery; anterior debridement proved to be more successful than posterior laminectomy, as none of those in the former group required subsequent revision surgery. All patients were also treated with intravenous antifungal drugs, typically amphotericin B. Two patients in the series died during hospitalization, indicating the potential severity of these infections [24].

Questions

1. What is the most informative imaging modality for most types of spinal infections?
   (a) Plain radiographs
   (b) Computed tomography
   (c) Gallium-67 nuclear medicine scan
   (d) Magnetic resonance imaging
2. Which of the following tests would not be part of a standard workup for vertebral osteomyelitis?
   (a) Complete blood cell count
   (b) Lumbar puncture
   (c) Blood cultures
   (d) Echocardiogram
3. Which of the following is FALSE?
   (a) Epidural abscesses can rapidly lead to neurologic injury if diagnosis or treatment is delayed.
   (b) Vertebral osteomyelitis is often seen in patients with urosepsis.
   (c) Brucellosis often causes sacroiliitis.
   (d) Pediatric diskitis often requires surgical intervention.

Answers: 1—(d), 2—(b), 3—(d)

References

1. An HS, Seldomridge JA (2006) Spinal infections: diagnostic tests and imaging studies. Clin Orthop Relat Res 444:27–33
2. Batson OV (1940) The function of the vertebral veins and their role in the spread of metastases. Ann Surg 112(1):138–149
3. Bluman EM, Palumbo MA, Lucas PR (2004) Spinal epidural abscess in adults. J Am Acad Orthop Surg 12(3):155–163
4. Brown R, Hussain M, McHugh K, Novelli V, Jones D (2001) Discitis in young children. J Bone Joint Surg Br 83(1):106–111
5. Byers K, Axelrod P, Michael S, Rosen S (1995) Infections complicating tunneled intraspinal catheter systems used to treat chronic pain. Clin Infect Dis 21(2):403–408
6. Carragee EJ (1997) The clinical use of magnetic resonance imaging in pyogenic vertebral osteomyelitis. Spine (Phila Pa 1976) 22(7):780–785
7. Carragee EJ (1997) Pyogenic vertebral osteomyelitis. J Bone Joint Surg Am 79(6):874–880
8. Medical Research Council Working Party on Tuberculosis of the Spine. A controlled trial of anterior spinal fusion and debridement in the surgical management of tuberculosis of the spine in patients on standard chemotherapy: a study in Hong Kong (1974). Br J Surg 61 (11):853–866
9. Crawford AH, Kucharzyk DW, Ruda R, Smitherman HC Jr (1991) Diskitis in children. Clin Orthop Relat Res 266:70–79
10. Curry WT Jr, Hoh BL, Amin-Hanjani S, Eskandar EN (2005) Spinal epidural abscess: clinical presentation, management, and outcome. Surg Neurol 63(4):364–371; discussion
11. Cushing AH (1993) Diskitis in children. Clin Infect Dis 17(1):1–6
12. Cwikiel W (1991) Percutaneous drainage of abscess in psoas compartment and epidural space. Case report and review of the literature. Acta Radiol 32(2):159–161
13. Danner RL, Hartman BJ (1987) Update on spinal epidural abscess: 35 cases and review of the literature. Rev Infect Dis 9(2):265–274
14. Darouiche RO, Hamill RJ, Greenberg SB, Weathers SW, Musher DM (1992) Bacterial spinal epidural abscess. Review of 43 cases and literature survey. Medicine (Baltimore) 71(6):369–385
15. Del Curling O, Gower DJ Jr, McWhorter JM (1990) Changing concepts in spinal epidural abscess: a report of 29 cases. Neurosurgery 27(2):185–192

16. Digby JM, Kersley JB (1979) Pyogenic non-tuberculous spinal infection: an analysis of thirty cases. J Bone Joint Surg Br 61(1):47–55
17. Dworkin R, Modin G, Kunz S, Rich R, Zak O, Sande M (1990) Comparative efficacies of ciprofloxacin, pefloxacin, and vancomycin in combination with rifampin in a rat model of methicillin-resistant Staphylococcus aureus chronic osteomyelitis. Antimicrob Agents Chemother 34(6):1014–1016
18. Early SD, Kay RM, Tolo VT (2003) Childhood diskitis. J Am Acad Orthop Surg 11(6):413–420
19. Eismont FJ, Bohlman HH, Soni PL, Goldberg VM, Freehafer AA (1982) Vertebral osteomyelitis in infants. J Bone Joint Surg Br 64(1):32–35
20. Eismont FJ, Bohlman HH, Soni PL, Goldberg VM, Freehafer AA (1983) Pyogenic and fungal vertebral osteomyelitis with paralysis. J Bone Joint Surg Am 65(1):19–29
21. Elian D, Hassin D, Tomer A, Bank H, Eisenstein Z (1984) Spinal epidural abscess: an unusual complication of bacterial endocarditis. Infection 12(4):258–259
22. Emery SE, Chan DP, Woodward HR (1989) Treatment of hematogenous pyogenic vertebral osteomyelitis with anterior debridement and primary bone grafting. Spine (Phila Pa 1976) 14(3):284–291
23. Fernandez M, Carrol CL, Baker CJ (2000) Discitis and vertebral osteomyelitis in children: an 18-year review. Pediatrics 105(6):1299–1304
24. Frazier DD, Campbell DR, Garvey TA, Wiesel S, Bohlman HH, Eismont FJ (2001) Fungal infections of the spine. Report of eleven patients with long-term follow-up. J Bone Joint Surg Am 83-A(4):560–565
25. Garcia A Jr, Grantham SA (1960) Hematogenous pyogenic vertebral osteomyelitis. J Bone Joint Surg Am 42-A:429–436
26. Garron E, Viehweger E, Launay F, Guillaume JM, Jouve JL, Bollini G (2002) Nontuberculous spondylodiscitis in children. J Pediatr Orthop 22(3): 321–328
27. Gonzalvo A, Abdulla I, Riazi A, De La Harpe D (2011) Single-level/single-stage debridement and posterior instrumented fusion in the treatment of spontaneous pyogenic osteomyelitis/discitis: long-term functional outcome and health-related quality of life. J Spinal Disord Tech 24(2):110–115
28. Harrington P, Millner PA, Veale D (2001) Inappropriate medical management of spinal epidural abscess. Ann Rheum Dis 60(3):218–222
29. Hassler O (1969) The human intervertebral disc. A micro-angiographical study on its vascular supply at various ages. Acta Orthop Scand 40(6):765–772
30. Henry NK, Rouse MS, Whitesell AL, McConnell ME, Wilson WR (1987) Treatment of methicillin-resistant Staphylococcus aureus experimental osteomyelitis with ciprofloxacin or vancomycin alone or in combination with rifampin. Am J Med 82(4A):73–75
31. Hlavin ML, Kaminski HJ, Ross JS, Ganz E (1990) Spinal epidural abscess: a ten-year perspective. Neurosurgery 27(2):177–184
32. Hogan QH (1991) Lumbar epidural anatomy. A new look by cryomicrotome section. Anesthesiology 75(5):767–775
33. Honan M, White GW, Eisenberg GM (1996) Spontaneous infectious discitis in adults. Am J Med 100(1):85–89
34. Hori K, Kano T, Fukushige T, Sano T (1997) Successful treatment of epidural abscess with a percutaneously introduced 4-French catheter for drainage. Anesth Analg 84(6):1384–1386
35. Jin D, Qu D, Chen J, Zhang H (2004) One-stage anterior interbody autografting and instrumentation in primary surgical management of thoracolumbar spinal tuberculosis. Eur Spine J 13(2):114–121
36. Jones NS, Anderson DJ, Stiles PJ (1987) Osteomyelitis in a general hospital. A five-year study showing an increase in subacute osteomyelitis. J Bone Joint Surg Br 69(5):779–783
37. Katonis P, Tzermiadianos M, Gikas A, Papagelopoulos P, Hadjipavlou A (2006) Surgical treatment of spinal brucellosis. Clin Orthop Relat Res 444: 66–72
38. Kemp HB, Jackson JW, Jeremiah JD, Hall AJ (1973) Pyogenic infections occurring primarily in intervertebral discs. J Bone Joint Surg Br 55(4):698–714
39. Khanna RK, Malik GM, Rock JP, Rosenblum ML (1996) Spinal epidural abscess: evaluation of factors influencing outcome. Neurosurgery 39(5):958–964
40. Kim CW, Perry A, Currier B, Yaszemski M, Garfin SR (2006) Fungal infections of the spine. Clin Orthop Relat Res 444:92–99
41. Kindler CH, Seeberger MD, Staender SE (1998) Epidural abscess complicating epidural anesthesia and analgesia. An analysis of the literature. Acta Anaesthesiol Scand 42(6):614–620
42. Koppel BS, Tuchman AJ, Mangiardi JR, Daras M, Weitzner I (1988) Epidural spinal infection in intravenous drug abusers. Arch Neurol 45(12):1331–1337
43. Korovessis P, Petsinis G, Koureas G, Iliopoulos P, Zacharatos S (2006) Anterior surgery with insertion of titanium mesh cage and posterior instrumented fusion performed sequentially on the same day under one anesthesia for septic spondylitis of thoracolumbar spine: is the use of titanium mesh cages safe? Spine (Phila Pa 1976) 31(9):1014–1019
44. Krogsgaard MR, Wagn P, Bengtsson J (1998) Epidemiology of acute vertebral osteomyelitis in Denmark: 137 cases in Denmark 1978-1982, compared to cases reported to the National Patient Register 1991-1993. Acta Orthop Scand 69(5):513–517
45. Kulowski J (1936) Pyogenic osteomyelitis of the spine: an analysis and discussion of 102 cases. J Bone Joint Surg Am 18:343–364
46. Lu CH, Chang WN, Lui CC, Lee PY, Chang HW (2002) Adult spinal epidural abscess: clinical features and prognostic factors. Clin Neurol Neurosurg 104(4):306–310
47. Lundy DW, Kehl DK (1998) Increasing prevalence of Kingella kingae in osteoarticular infections in young children. J Pediatr Orthop 18(2):262–267

48. Lyu RK, Chen CJ, Tang LM, Chen ST (2002) Spinal epidural abscess successfully treated with percutaneous, computed tomography-guided, needle aspiration and parenteral antibiotic therapy: case report and review of the literature. Neurosurgery 51(2):509–512; discussion 512
49. Mampalam TJ, Rosegay H, Andrews BT, Rosenblum ML, Pitts LH (1989) Nonoperative treatment of spinal epidural infections. J Neurosurg 71(2):208–210
50. Menelaus MB (1964) Discitis. An inflammation affecting the intervertebral discs in children. J Bone Joint Surg Br 46:16–23
51. Modic MT, Feiglin DH, Piraino DW, Boumphrey F, Weinstein MA, Duchesneau PM, Rehm S (1985) Vertebral osteomyelitis: assessment using MR. Radiology 157(1):157–166
52. Morelli S, Carmenini E, Caporossi AP, Aguglia G, Bernardo ML, Gurgo AM (2001) Spondylodiscitis and infective endocarditis: case studies and review of the literature. Spine (Phila Pa 1976) 26(5):499–500
53. Nordberg G, Mark H (1998) Epidural abscess after epidural analgesia treated successfully with antibiotics. Acta Anaesthesiol Scand 42(6):727–731
54. Nussbaum ES, Rigamonti D, Standiford H, Numaguchi Y, Wolf AL, Robinson WL (1992) Spinal epidural abscess: a report of 40 cases and review. Surg Neurol 38(3):225–231
55. Pappas G, Papadimitriou P, Akritidis N, Christou L, Tsianos EV (2006) The new global map of human brucellosis. Lancet Infect Dis 6(2):91–99
56. Parke WW, Rothman RH, Brown MD (1984) The pharyngovertebral veins: an anatomical rationale for Grisel's syndrome. J Bone Joint Surg Am 66(4):568–574
57. Ratcliffe JF (1985) Anatomic basis for the pathogenesis and radiologic features of vertebral osteomyelitis and its differentiation from childhood discitis. A microarteriographic investigation. Acta Radiol Diagn (Stockh) 26(2):137–143
58. Reihsaus E, Waldbaur H, Seeling W (2000) Spinal epidural abscess: a meta-analysis of 915 patients. Neurosurg Rev 23(4):175–204; discussion 205
59. Rezai AR, Lee M, Cooper PR, Errico TJ, Koslow M (1995) Modern management of spinal tuberculosis. Neurosurgery 36(1):87–97; discussion 97–88
60. Rigamonti D, Liem L, Sampath P, Knoller N, Namaguchi Y, Schreibman DL, Sloan MA, Wolf A, Zeidman S (1999) Spinal epidural abscess: contemporary trends in etiology, evaluation, and management. Surg Neurol 52(2):189–196; discussion 197
61. Ring D, Johnston CE 2nd, Wenger DR (1995) Pyogenic infectious spondylitis in children: the convergence of discitis and vertebral osteomyelitis. J Pediatr Orthop 15(5):652–660
62. Rudert M, Tillmann B (1993) Lymph and blood supply of the human intervertebral disc. Cadaver study of correlations to discitis. Acta Orthop Scand 64(1):37–40
63. Sadato N, Numaguchi Y, Rigamonti D, Kodama T, Nussbaum E, Sato S, Rothman M (1994) Spinal epidural abscess with gadolinium-enhanced MRI: serial follow-up studies and clinical correlations. Neuroradiology 36(1):44–48
64. Sillevis Smitt P, Tsafka A, van den Bent M, de Bruin H, Hendriks W, Vecht C, Teng-van de Zande F (1999) Spinal epidural abscess complicating chronic epidural analgesia in 11 cancer patients: clinical findings and magnetic resonance imaging. J Neurol 246(9):815–820
65. Skaf GS, Kanafani ZA, Araj GF, Kanj SS (2010) Non-pyogenic infections of the spine. Int J Antimicrob Agents 36(2):99–105
66. Soehle M, Wallenfang T (2002) Spinal epidural abscesses: clinical manifestations, prognostic factors, and outcomes. Neurosurgery 51(1):79–85; discussion 86–77
67. Solera J, Lozano E, Martinez-Alfaro E, Espinosa A, Castillejos ML, Abad L (1999) Brucellar spondylitis: review of 35 cases and literature survey. Clin Infect Dis 29(6):1440–1449
68. Song KS, Ogden JA, Ganey T, Guidera KJ (1997) Contiguous discitis and osteomyelitis in children. J Pediatr Orthop 17(4):470–477
69. Stauffer RN (1975) Pyogenic vertebral osteomyelitis. Orthop Clin North Am 6(4):1015–1027
70. Stone DB, Bonfiglio M (1963) Pyogenic vertebral osteomyelitis. A diagnostic pitfall for the internist. Arch Intern Med 112:491–500
71. Szypryt EP, Hardy JG, Hinton CE, Worthington BS, Mulholland RC (1988) A comparison between magnetic resonance imaging and scintigraphic bone imaging in the diagnosis of disc space infection in an animal model. Spine (Phila Pa 1976) 13(9):1042–1048
72. Tabo E, Ohkuma Y, Kimura S, Nagaro T, Arai T (1994) Successful percutaneous drainage of epidural abscess with epidural needle and catheter. Anesthesiology 80(6):1393–1395
73. Tay BK, Deckey J, Hu SS (2002) Spinal infections. J Am Acad Orthop Surg 10(3):188–197
74. Torda AJ, Gottlieb T, Bradbury R (1995) Pyogenic vertebral osteomyelitis: analysis of 20 cases and review. Clin Infect Dis 20(2):320–328
75. Tsiodras S, Falagas ME (2006) Clinical assessment and medical treatment of spine infections. Clin Orthop Relat Res 444:38–50
76. Turunc T, Demiroglu YZ, Uncu H, Colakoglu S, Arslan H (2007) A comparative analysis of tuberculous, brucellar and pyogenic spontaneous spondylodiscitis patients. J Infect 55(2):158–163
77. Wenger DR, Bobechko WP, Gilday DL (1978) The spectrum of intervertebral disc-space infection in children. J Bone Joint Surg Am 60(1):100–108
78. Wheeler D, Keiser P, Rigamonti D, Keay S (1992) Medical management of spinal epidural abscesses: case report and review. Clin Infect Dis 15(1):22–27
79. Wiesseman GJ, Wood VE, Kroll LL, Linda L (1973) Pseudomonas vertebral osteomyelitis in heroin addicts. Report of five cases. J Bone Joint Surg Am 55(7):1416–1424

80. Wiley AM, Trueta J (1959) The vascular anatomy of the spine and its relationship to pyogenic vertebral osteomyelitis. J Bone Joint Surg Br 41-B: 796–809

81. Yilmaz C, Selek HY, Gurkan I, Erdemli B, Korkusuz Z (1999) Anterior instrumentation for the treatment of spinal tuberculosis. J Bone Joint Surg Am 81(9):1261–1267

Intradural Spinal Cord Tumors

34

Ricky R. Kalra and Andrew T. Dailey

34.1 Introduction

Spinal cord tumors are classified in three different categories to guide diagnosis and treatment: extradural, intradural extramedullary, and intramedullary spinal tumors. In this chapter, we will focus primarily on intradural tumors since their primary treatment is surgical resection. Extradural tumors, which are frequently metastatic and include a wide range of neoplasms with varying treatment options, are discussed elsewhere in this book. Intramedullary spinal cord tumors, which arise from cells within the spinal cord, account for just 5 % of all central nervous system tumors and 15 % of all spinal tumors. A small minority of these tumors are associated with heritable diseases, including Von Hippel–Lindau (VHL) disease, which causes hemangioblastomas, and neurofibromatosis types 1 and 2 (NF-1 and NF-2), which cause astrocytomas and ependymomas, respectively [2, 49]. Approximately 20–30 % of patients with VHL disease or NF-2 have intramedullary tumors. Patients with intramedullary tumors typically present initially with pain and sensory changes, followed by motor weakness and, then at later stages, bowel and bladder deficits. In this chapter, we will discuss an extensive list of intramedullary and some pertinent intradural extramedullary tumors, emphasizing their epidemiology, symptoms, radiological features, histopathology, and treatment modalities.

34.2 Ependymomas

The ependymal lining of the ventricles and central canal is associated with the development of ependymomas. Their occurrence within the central nervous system is usually sporadic but can also be associated with NF-2. Epidemiological studies estimate that 30 % of all ependymomas are spinal [58]. They are the most common spinal cord tumor in adults and the second most common in children after astrocytomas [28, 58].

Ependymomas are located within the central canal of the spinal cord and cause symptoms related to expansion of the neural elements. Pediatric patients may also present with hydrocephalus. Because of the slow-growing nature of the lesions, the average patient presents 2–4 years after the onset of symptoms [28]. Mean age of presentation in adults is 40 years [28, 58].

Ependymomas are more frequent in the cervical and thoracic regions than in the lumbosacral region. On imaging, they often show an expansion of the cord. CT findings are spinal canal widening and thinned pedicles. Contrast-enhanced CT scans show a well-circumscribed solid lesion. T1-weighted MR sequences of ependymomas are isointense to the spinal cord,

R.R. Kalra, M.D. • A.T. Dailey, M.D. (✉)
Department of Neurosurgery, University of Utah, 175 N. Medical Drive East, Salt Lake City 84132, UT, USA
e-mail: neuropub@hsc.utah.edu

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_34, © Springer-Verlag Berlin Heidelberg 2014

and the administration of contrast agent enhances the lesion. T2-weighted sequences of ependymomas are hyperintense and may show a syrinx related to the central canal obstruction as well as cord edema surrounding the lesion (Figs. 34.1, 34.2, and 34.3) [24, 60].

Spinal ependymomas are subdivided into four grades by the World Health Organization (WHO), including subependymomas (grade 1), myxopapillary ependymomas (grade 1), classic ependymomas (grade 2), and anaplastic ependymomas (grade 3). Slow-growing subependymomas are considered benign ventricular lesions and are rarely encountered in the spinal cord. They are composed of nests of ependymal cells in a dense glial fibrillary matrix and are often associated with small cysts. Myxopapillary ependymomas occur almost exclusively in the conus–cauda equina–filum terminale region. They are composed of cuboidal tumor cells arranged in a circular pattern around vascular cores. Classic ependymomas are further subdivided into four categories: cellular, papillary, clear-cell, and tanycytic. The cellular variety is the most common and microscopically presents with perivascular pseudorosettes, moderate cellularity, and rare nuclear atypia. The papillary subtype resembles choroid plexus papilloma, with well-formed papillae with blood vessels covered by a smooth layer of tumor cells. The clear-cell variety resembles oligodendrogliomas but contains ependymal and perivascular rosettes. The tanycytic subtype is extremely rare and similar to astrocytoma. It is characterized by streams of piloid, or hair-like, cells having "ependymal" nuclei, which resemble tanycytes. Tanycytes are bipolar special cells related to hypothalamic/endocrine function. Anaplastic ependymomas differ from the other varieties by displaying increased mitotic activity, increased cellularity, microvascular proliferation, infiltrative patterns, and necrosis. Macroscopically, low-grade intramedullary ependymomas are well-circumscribed, slow-growing, centrally located soft red lesions. They are well circumscribed and may show cystic changes [27, 30, 38, 47].

Treatment guidelines recommend immediate gross total resection of the lesion to remove tumor burden and mass effect and to reduce the risk of recurrence. In cases in which a gross total resection cannot be achieved, studies support adjuvant treatment with high-dose radiation. Survival studies estimate an 85 % 5-year survival

Fig. 34.1 T1-weighted MRI with contrast enhancement showing an ependymoma

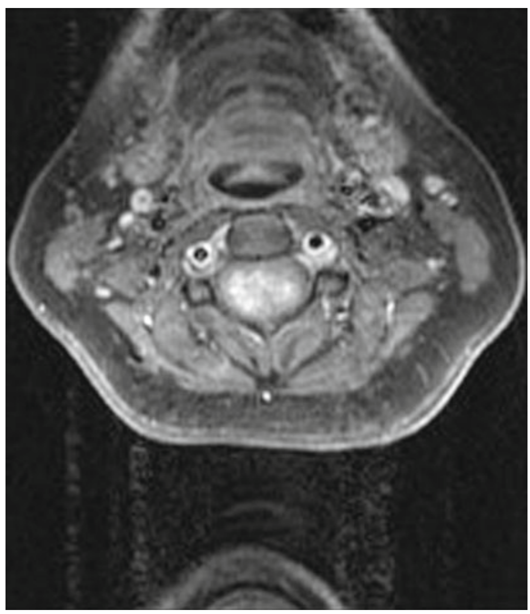


Fig. 34.2 T1-weighted MRI with contrast enhancement depicting an ependymoma

Fig. 34.3 T2-weighted MRI showing an ependymoma. Vertebral levels are indicated (C2, C4, C6, T1, T3, T5)

rate, including patients with subtotal resection and adjuvant radiation [28, 58].

34.3 Astrocytomas

Astrocytomas are the most common type of intramedullary tumor in children and second most common in adults after ependymomas. Several types of astrocytoma have been recognized; the most common is the pilocytic astrocytoma. High-grade astrocytomas are infrequent in the spinal cord. Spinal pilocytic astrocytomas are usually found before age 30, with many found during childhood. They often are associated with syndromes in the juvenile population, including Turcot syndrome, tuberous sclerosis complex, Li-Fraumeni syndrome, and NF-1 [2, 49].

Patients with spinal astrocytomas have symptoms of pain and myelopathy. Because these lesions are slow growing, bony remodeling with associated scoliosis may also occur [31].

The cervical spine is the most common location of spinal astrocytomas, followed by the thoracic and lumbar regions. The lesions are generally 1–3 cm in size and cause oblong fusiform expansion of the spinal cord. CT findings without contrast enhancement show cord enlargement, while contrast-enhanced images show mild enhancement. MR findings on T1 sequence also suggest cord enlargement, and approximately 40 % of tumors have intratumoral cysts or syringohydromyelia. Contrast-enhanced T1-weighted MR sequences show heterogenous contrast enhancement [42].

Histopathological grading of spinal astrocytomas is based on the highest degree of anaplasia observed. Grade 1 astrocytomas [juvenile pilocytic astrocytomas (JPAs)] are the most common subtype in the pediatric population. Grade 2 astrocytomas (fibrillary) are often found to be diffusely infiltrating the parenchyma. Grade 3 lesions are anaplastic in nature, and grade 4 tumors are classified as glioblastoma multiforme. Microscopic histopathology of JPAs shows elongated cells with intracellular Rosenthal fibers and granular eosinophilic bodies. Grade 2 lesions show hypercellularity, nuclear pleomorphism, and diffuse infiltration into the spinal cord. Tumors of grades 3 and 4 show vascular proliferation, necrosis, and active mitotic features. Gross pathological analysis of spinal cord astrocytomas shows an expanded spinal cord [27, 47].

Gross total resection is the goal of treatment, but the infiltrative nature of astrocytomas makes this goal difficult to achieve. The secondary goal of treatment is reducing tumor burden by surgical resection. Survival statistics reveal an 80 % 5-year survival rate for patients with low-grade tumors and a 30 % 5-year survival rate for those with high-grade lesions [51]. The use of adjuvant postoperative radiation therapy remains a controversial topic among researchers. Most studies show no additional survival with radiation therapy unless tumors are grade 3 or 4 [51, 52].

34.4 Hemangioblastomas

Hemangioblastomas represent approximately 12 % of all intramedullary tumors and 4 % of spinal tumors [53]. They are commonly associated with VHL disease but can also occur sporadically.

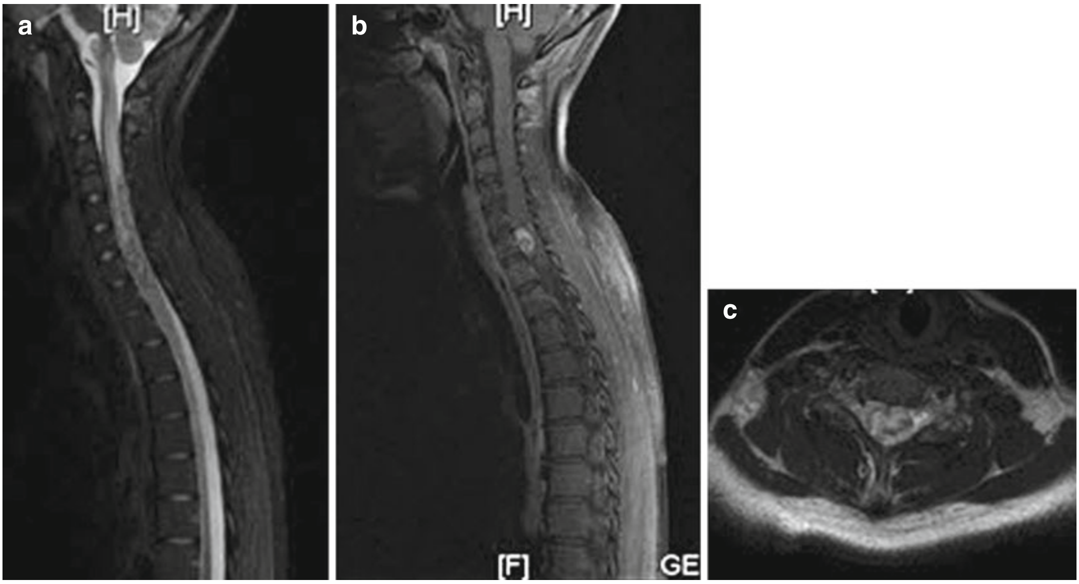


Fig. 34.4 (**a**) T2-weighted MRI, (**b**, **c**) T1-weighted MRI with contrast enhancement showing a hemangioblastoma

According to studies, approximately 25 % of VHL patients have spinal hemangioblastomas [34, 53], so genetic testing for VHL disease is recommended for patients diagnosed with such tumors [43, 46]. The majority of patients come to medical attention during the fifth decade of life. A rare clinical presentation of acute hemorrhage has been reported in the literature, but the majority of patients have a slow onset of symptoms [61]. These tumors tend to occur predominantly in the posterior aspect of the spinal cord. In one series, most of the tumors were located in the thoracic spine [46].

Contrast-enhanced MR imaging is the ideal imaging modality for these tumors. They appear as enhancing intramedullary masses with possible cysts associated with them. Because of their posterior location, many of these tumors show extensive cord edema surrounding the lesion (Fig. 34.4) [12].

Hemangioblastomas are low-grade, well-circumscribed, capillary-rich neoplasms. Microscopic findings reveal vacuolated stromal cells with extensive capillary networks. They are classified by the WHO as grade 1 [27, 47].

Gross total surgical resection is the treatment of choice of patients with symptomatic lesions, and most tumors can be safely removed completely without sequelae [46]. Asymptomatic tumors are generally observed with conservative management and serial imaging. Preoperative embolization, if possible, has been shown in studies to reduce intraoperative bleeding; however, no studies have shown superior outcome with preoperative embolization [46].

34.5 Cavernous Malformations

Cavernous malformations of the spinal cord account for only 3–5 % of all intramedullary lesions [21]. They occur most frequently between the 3rd and 6th decades of life, with a peak around the 4th decade. Cavernomas show a female predominance, with a female-to-male incidence ratio of 2:1 [21]. The annual risk of hemorrhage associated with cavernous malformations is thought to be about 2–4 % per year [21].

Because these lesions may result in repeated hemorrhages, multiple clinical patterns of the disease have been documented. Patients may present with stepwise neurological deterioration with intermittent recovery, slow progressive neurological decline, sudden onset of symptoms with rapid decline because of a large acute hemorrhage, or

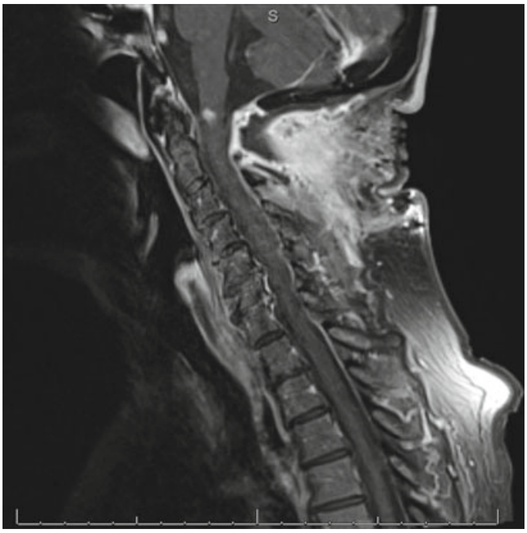


Fig. 34.5 T1-weighted MRI with contrast enhancement showing a cavernous malformation

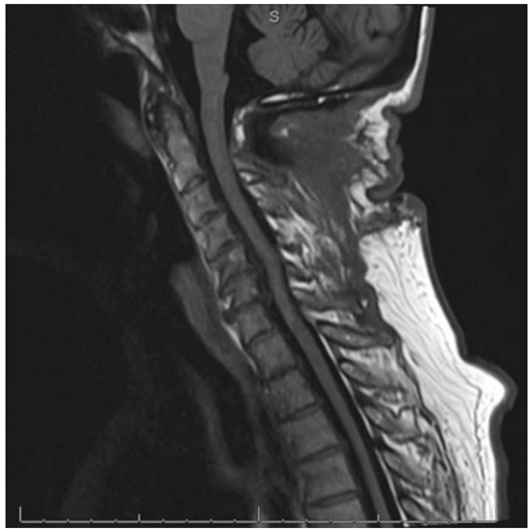


Fig. 34.6 T1-weighted MRI depicting a cavernous malformation

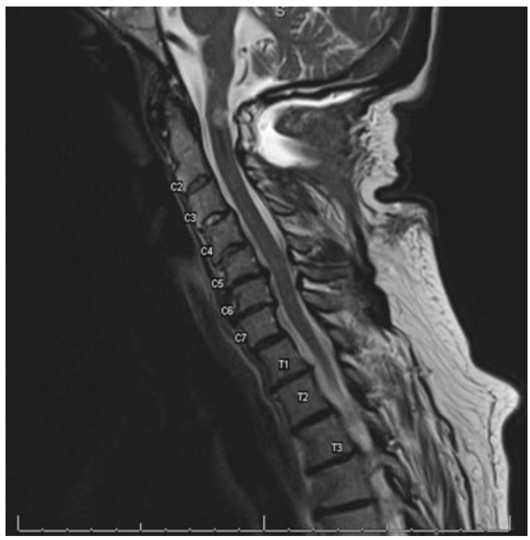


Fig. 34.7 T2-weighted MRI showing a cavernous malformation. Vertebral levels C2–T3 are indicated

sudden onset of symptoms with slow decline after a smaller acute hemorrhage. The patterns of deterioration highlight the heterogenous nature of these lesions [21, 44, 57].

T1-weighted MR imaging is heterogenous because of varying ages of blood products, and the mixed heterogeneity gives the lesions a lobular or "popcorn"-type shape. Gadolinium administration causes minimal enhancement of the lesions. T2-weighted images show hypointense rims (hemosiderin) with heterogenous mass in the cord. Cavernous malformations are angiographically occult. Gradient echo sequences (GRE) confirm the hemorrhagic nature of these lesions (Figs. 34.5, 34.6, and 34.7) [5, 8].

Fifty percent of spinal cavernomas are located in the thoracic spine, 40 % in the cervical spine, and 10 % in the conus [8]. Twenty percent of lesions are associated with familial cavernous malformation syndrome. The familial variety results from mutations on chromosome 3 that predispose patients to multiple lesions in an infratentorial location and an earlier age of presentation because of increased rates of hemorrhage. Microscopic histopathological evaluation of cavernous malformations shows vascular spaces with a single layer of endothelial cells. These vascular spaces are separated by poorly constructed collagen walls with no support from elastin, smooth muscle, or other structural proteins. Cavernomas do not contain neural tissue between vascular spaces and lack histological features of capillaries. Macroscopically, they appear as discrete lobulated brownish "popcorn" nodules with hemosiderin-stained capsules [27, 47].

These vascular malformations can remain clinically occult for decades before an acute hemorrhage commences the chronic and progressive stage of these lesions. Symptomatic lesions as well as asymptomatic lesions that are safely accessible should be treated to avoid the devastating consequences associated with a large bleed. Surgical gross total resection remains the treatment of choice. The annual risk of hemorrhage and associated neurological morbidity compared with the postoperative outcomes favors surgical intervention. Most lesions can be resected completely, and a majority of patients with prior hemorrhage improve clinically [21, 59].

34.6 Dermoid Cysts

Dermoid cysts are unique intramedullary lesions that represent just 1 % of all spinal cord tumors but 10 % of all spinal cord tumors in patients under the age of 15 years [20]. They are benign germ cell tumors that are the consequence of embryological errors during neural tube closure. All dermoid cysts are congenital. Most are discovered within the second decade of life. Studies show that males are more often affected than females [20, 48].

These slow-growing congenital lesions contain cells that gradually produce cholesterol-laden products, resulting in continuous growth. Thus, most patients present with mass effect symptoms; however, case studies also show patients presenting with acute chemical meningitis secondary to rupture and dissemination of inflammatory cholesterol products into the cerebral spinal fluid [48].

Dermoid cysts can be located extramedullary (60 %) as well as intramedullary (40 %) and most commonly occur in the lumbosacral spine. T1-weighted MR imaging with contrast agent shows mild ring enhancement, while imaging without contrast enhancement shows variable findings from hypo- to hyperdense. T2-weighted sequences show high signal intensity from the mass [3].

Gross surgical specimens appear as smooth, well-demarcated masses containing thick yellowish material. Microscopic pathological evaluation reveals an outer connective tissue capsule composed of stratified squamous epithelium with the inner content composed of epithelial keratin and cholesterol crystals [27].

Dermoid cysts are treated by surgical resection. Their adherent nature makes removal difficult without damage to neural structures and often requires lengthy dissection. Additionally, care must be taken to avoid spillage of the cyst material into the cerebrospinal fluid (CSF), which may cause chemical meningitis. Complete gross total resection is considered curative. Subtotal resections may result in recurrence [48].

34.7 Gangliogliomas

Gangliogliomas are composed of ganglion cells and glial elements. They usually occur in the temporal lobes and cerebellum in children and young adults. Spinal occurrence is speculated to be less than 1 % of all spinal neoplasms [32, 36]. MR findings on T1-weighted sequences show mixed signal intensity, whereas T2-weighted images show a heterogenous appearance. Gadolinium-enhanced studies show patchy enhancement of the lesion [55].

Case studies report that spinal gangliogliomas have low malignant potential. The majority of the cases reported were located in the cervical cord. Microscopically, gangliogliomas appear astrocytic in nature, with large cells displaying dysplastic features, perivascular infiltration, Rosenthal fibers, and eosinophilic granular bodies. Macroscopically, they appear as solid mass with cystic changes [27].

Surgical gross total resection is the treatment of choice. In case studies, subtotal resection resulted in a high frequency of recurrence, both radiologically and clinically [36, 54].

34.8 Lipomas

Spinal lipomas arise from premature separation of ectoderm from neuroectoderm during the neurulation process. They may occur intradurally or at the distal cord/filum area. They represent less than 1 % of intradural spinal lesions [22]. Peak ages for presentation are younger than 5 years and during the 3rd and 4th decades. There is a female predisposition for intradural lipomas [22, 45].

Lipomas are composed of normal fat cells, change continuously depending on body fat content, and may regress with fat-deficient diets and grow during steroid therapy and other fat-retaining metabolic conditions such as pregnancy [22, 45]. Microscopically, they appear as masses of fat globules. Macroscopically, they appear as partially capsulated tumors completely contained within the dura [27].

The most common location for intradural lipomas is the thoracic spine, followed by the cervical and then lumbosacral regions. T1-weighted MR imaging shows hyperintense rounded mass attached to the cord; lipomas do not enhance on contrast-enhanced studies. T2 sequences also show a hyperintense lesion within the cord [1, 7].

Surgical decompression of the affected spinal segments is the procedure of choice; however, lipomas do not have a good dissection plane with respect to the cord and seem “infiltrative” in nature. Thus, decompression and removal of non-adherent portions is the appropriate treatment. Weight loss is a nonsurgical option reserved for obese patients that may result in decrease in the size of the lesion [22, 23].

34.9 Melanocytomas

Melanocytes originate in the neural crest and are found throughout the leptomeninges, with higher concentrations around the brain stem and upper cervical spinal cord. These cells give rise to melanocytomas, which are rare pigmented primary neoplasm of the central nervous system involving both the brain and the spinal cord. They differ from melanoma both histologically and clinically. Men and women are affected in equal numbers, and the lesions have no peak age at which they present [4, 25].

T1-weighted MR imaging shows hyperintense intradural lesions that heterogeneously enhance with gadolinium administration. T2-weighted sequences show a lesion that is hypointense to the cord [19].

Some authors have speculated that melanocytic lesions constitute a spectrum from benign melanocytomas to malignant melanoma. On gross pathological analysis, these lesions appear black. Microscopically, they feature spindle cells, with minimal nuclear activity and variable amounts of melanin [25, 27]. Surgical resection is the treatment of choice, both for pathological evaluation and to remove the mass effect associated with the lesions [6].

34.10 Schwannomas

Schwannomas are the most common intradural extramedullary tumors. They are cystic, well-encapsulated tumors that arise from the dorsal sensory root. Some tumors involve both the intra- and extradural space and present as “dumbbell”-shaped lesions [15].

On T1-weighted MR images, schwannomas are usually isointense to the cord but enhance with gadolinium administration. On T2-weighted sequences, these tumors are hyperintense [14].

On pathological examination, schwannomas arise from a single nerve fascicle, typically off of dorsal spinal nerve roots, displacing the other fascicles. On microscopic analysis, these tumors have a three-layered capsule with fibrous tissue, nerve tissue, and a transitional layer. The cell of origin is the Schwann cell, and the tumors classically have a biphasic pattern with Antoni A and B regions [29].

Complete surgical resection is the only treatment modality that is curative, and frequently, the involved nerve root must be sacrificed [15].

34.11 Meningiomas

Meningiomas are dural-based, benign slow-growing intradural lesions. The majority of meningiomas are found in the thoracic region, with an increased prevalence in female patients [50].

On T1-weighted MR imaging sequences, meningiomas are isointense to the spinal cord;

Fig. 34.8 T1-weighted MRI with contrast enhancement depicting a meningioma

with gadolinium administration, these tumors enhance avidly. T2-weighted sequences also show that the majority is isointense to the cord. They usually displace rather than invade the cord (Fig. 34.8) [17].

On pathological analysis, most meningiomas are solitary, firm, and well demarcated and have a dural attachment. On microscopic analysis, most meningiomas are psammomatous with calcium concentrations, meningothelial, fibrous, or transitional. Rarely, patients present with more aggressive atypical or anaplastic features including increased mitosis/cellularity [37].

Surgical resection is the treatment modality of choice, and residual tumor burden directly correlates with recurrence and progression-free survival. In patients with aggressive disease in whom subtotal resection is achieved, radiation may be considered for tumor control [13].

34.12 Metastasis

Intramedullary metastases of cancers are rare and occur in less than 1 % of patients with spinal metastasis [56].The most common tumor parent types are breast and lung carcinomas. Most patients who present with metastatic lesions to the spinal cord have brain metastases as well and are in poor general health. Metastatic lesions to the brain may also spread into the spinal cord via "drop metastases." These CSF-disseminated cells often preferentially are located in the lumbosacral region [39].

The differential diagnosis for patients with cancers presenting with myelopathy may include paraneoplastic myelopathies, radiation-induced myelopathies, and chemotherapy-associated symptoms. MR imaging of the spinal axis is the only way to confirm metastasis [16, 39].

Because the majority of the patients presenting with spinal metastases have severe tumor burden, surgical intervention is rarely undertaken. Unless symptomatic relief is the purpose of the surgery, the majority of patients are started or continued on chemoradiation protocols [39].

34.13 Treatment Modalities

34.13.1 Surgery

Generally, surgery is the treatment of choice for intramedullary or extramedullary intradural lesions. Multiple studies confirm excellent clinical results with gross total resections of tumors [31, 32, 35]. Surgery may be less effective for diffuse and infiltrative lesions, for which the goal of the treatment shifts from gross total resection to tissue diagnosis.

Surgical resection of the tumor is achieved by exposing the lesion while preserving the surrounding neural elements. Most of these tumors are resected via a posterior approach. The patient should be monitored with somatosensory-evoked potentials and motor-evoked potentials throughout surgery. The use of multimodality neuromonitoring is critical for allowing more thorough resection of ependymoma and astrocytoma in a safe manner [41]. The laminectomy must be planned such that it is adequate to expose the entire lesion. Intraoperative ultrasound guidance can improve the localization of the lesion and reduce the size of the laminectomy, durotomy, and myelotomy. Myelotomy is usually done in the midline, at the posterior median septum, unless the tumor is located laterally, in which case the dorsal root entry zone may be used. Minimal coagulation of posterior surface vessels should be done to avoid damage to the sensory tracts. The myelotomy should be performed by opening the pia with microscissors and then using blunt dissection to open the rest of the cord. The spinal cord opening should be extensive enough to expose the entire tumor [40]. Ideally, a plane between the tumor and the cord should be present. Lack of a clear plane may indicate an infiltrative process and shift the purpose of the surgery to tissue sampling [31, 32, 35]. Tumor planes may be found by gently lifting spinal tissue of the tumor and evaluating the neoplasm for a tumor capsule. Additionally, the presence of the syrinx will intrinsically create a tumor boundary and allow for plane development. Also, hemosiderin rings and recent hemorrhage may define the margin of the tumor in the case of vascular lesions. Once the tumor margins have been delineated, gross total resection, whether en bloc or piecemeal, is possible. In the process of debulking, care must be taken to preserve normal vasculature while coagulating tumor-feeding vessels. Once the tumor is resected, care must be taken to close the wound in a systematic fashion to avoid CSF leaks and CSF fistulas [40].

34.13.2 Adjuvant Treatments

34.13.2.1 Radiation Therapy

Intramedullary spinal tumors exert their influence primarily through a mass effect; thus, surgical debulking has been the primary treatment modality. Adjuvant radiotherapy has played a very limited role, principally to supplement treatment in patients with incompletely resected neoplasms. Radiation in young children is associated with substantial adverse effects; thus, radiation oncologists have delayed or avoided radiotherapy completely in children. In the adult population, radiotherapy is more prevalent, but only to a limited extent. In low-grade astrocytomas and ependymomas, however, radiotherapy has been shown to improve outcome in patients with subtotal resections [28, 32, 33].

34.13.2.2 Chemotherapy

Chemotherapy is not a standard treatment modality in patients with intramedullary tumors. After surgery or radiation, some authors [9, 26] have used chemotherapy agents for recurrent ependymomas and low-grade astrocytomas. These include platinum-based agents and nitrosourea-based protocols. The overall effect on survival and outcomes of chemotherapy is still unclear [9–11, 26].

34.13.3 Outcomes

In a large study, overall survival of patients with intramedullary tumors after surgery was 90 % at 1 year, 75 % at 5 years, and 73 % at 10 years [40]. The survival outcome of patients has been directly linked to the histology of the tumor/grade, tumor recurrence, preoperative Karnofsky performance scale score, and length of symptoms prior to surgery [18]. For patients with grade I lesions, survival outcomes of 84 % after 10 years were noted [40]. Survival of patients with grade II lesions was similar. Patients with grade III and grade IV lesions showed very poor survival; the majority of patients were dead within 1 year, and very few survived past 5 years (Table 34.1).

Table 34.1 Survival rates for patients with intramedullary tumors [40]

	Survival rate (%)		
Grade	1-year survival	5-year survival	10-year survival
I	93	84	84
II	100	92	92
III	89	42	21
IV	20	0	0
Total	87	76	74

Conclusion

Spinal intramedullary tumors are relatively rare but do cause significant morbidity and mortality to patients. Their prevalence in the younger population results in long-term implications for patients, extensive costs to the health-care systems, and severe burden to families. Recent improvements in radiological imaging, especially MR imaging, have allowed earlier characterization of many of these lesions. Surgical gross total resection when possible is the mainstay of treatment. Radiation and chemotherapy treatments play a limited role overall and are used only in specific tumor types. Survival outcome of patients is typically good for patients with grades I and II tumors; survival is directly linked to the histology of the tumor, tumor recurrence, preoperative Karnofsky performance scale score, and length of symptoms.

References

1. Aoki N (1990) Rapid growth of intraspinal lipoma demonstrated by magnetic resonance imaging. Surg Neurol 34:107–110
2. Baleriaux DL (1999) Spinal cord tumors. Eur Radiol 9:1252–1258
3. Barkovich A (2000) Pediatric neuroimaging. Lippincott-Raven, Philadelphia
4. Barth A, Pizzolato GP, Berney J (1993) Intramedullary meningeal melanocytoma. Neurochirurgie 39:188–194
5. Bourgouin PM, Tampieri D, Johnston W, Steward J, Melancon D, Ethier R (1992) Multiple occult vascular malformations of the brain and spinal cord: MRI diagnosis. Neuroradiology 34:110–111
6. Brat DJ, Giannini C, Scheithauer BW, Burger PC (1999) Primary melanocytic neoplasms of the central nervous systems. Am J Surg Pathol 23:745–754
7. Brunberg JA, Latchaw RE, Kanal E, Burk DL Jr, Albright L (1988) Magnetic resonance imaging of spinal dysraphism. Radiol Clin North Am 26:181–205
8. Chabert E, Morandi X, Carney MP, Riffaud L, Louail C, Carsin-Nicol B (1999) Intramedullary cavernous malformations. J Neuroradiol 26:262–268
9. Chamberlain MC (2001) Recurrent intracranial ependymoma in children: salvage therapy with oral etoposide. Pediatr Neurol 24:117–121
10. Chamberlain MC (2002) Etoposide for recurrent spinal cord ependymoma. Neurology 58:1310–1311
11. Chamberlain MC (2002) Salvage chemotherapy for recurrent spinal cord ependymoma. Cancer 95: 997–1002
12. Chu BC, Terae S, Hida K, Furukawa M, Abe S, Miyasaka K (2001) MR findings in spinal hemangioblastoma: correlation with symptoms and with angiographic and surgical findings. AJNR Am J Neuroradiol 22:206–217
13. Cohen-Gadol AA, Zikel OM, Koch CA, Scheithauer BW, Krauss WE (2003) Spinal meningiomas in patients younger than 50 years of age: a 21-year experience. J Neurosurg 98:258–263
14. Colosimo C, Cerase A, Denaro L, Maira G, Greco R (2003) Magnetic resonance imaging of intramedullary spinal cord schwannomas. Report of two cases and review of the literature. J Neurosurg 99:114–117
15. Conti P, Pansini G, Mouchaty H, Capuano C, Conti R (2004) Spinal neurinomas: retrospective analysis and long-term outcome of 179 consecutively operated cases and review of the literature. Surg Neurol 61:34–43; discussion 44
16. Costigan DA, Winkelman MD (1985) Intramedullary spinal cord metastasis. A clinicopathological study of 13 cases. J Neurosurg 62:227–233
17. Covert S, Gandhi D, Goyal M, Woulfe J, Belanger E, Miller W, Modha A (2003) Magnetic resonance imaging of intramedullary meningioma of the spinal cord: case report and review of the literature. Can Assoc Radiol J 54:177–180
18. Cristante L, Herrmann HD (1994) Surgical management of intramedullary spinal cord tumors: functional outcome and sources of morbidity. Neurosurgery 35:69–74; discussion 74–76
19. Czarnecki EJ, Silbergleit R, Gutierrez JA (1997) MR of spinal meningeal melanocytoma. AJNR Am J Neuroradiol 18:180–182
20. de Baecque C, Snyder DH, Suzuki K (1977) Congenital intramedullary spinal dermoid cyst associated with an Arnold-Chiari malformation. Acta Neuropathol 38:239–242
21. Deutsch H, Jallo GI, Faktorovich A, Epstein F (2000) Spinal intramedullary cavernoma: clinical presentation and surgical outcome. J Neurosurg 93:65–70
22. Dyck P (1992) Intramedullary lipoma. Diagnosis and treatment. Spine (Phila Pa 1976) 17:979–981
23. Endoh M, Iwasaki Y, Koyanagi I, Hida K, Abe H (1998) Spontaneous shrinkage of lumbosacral lipoma in conjunction with a general decrease in body fat: case report. Neurosurgery 43:150–151; discussion 151–152
24. Fine MJ, Kricheff II, Freed D, Epstein FJ (1995) Spinal cord ependymomas: MR imaging features. Radiology 197:655–658
25. Glick R, Baker C, Husain S, Hays A, Hibshoosh H (1997) Primary melanocytomas of the spinal cord: a report of seven cases. Clin Neuropathol 16:127–132
26. Gornet MK, Buckner JC, Marks RS, Scheithauer BW, Erickson BJ (1999) Chemotherapy for advanced CNS ependymoma. J Neurooncol 45:61–67
27. Graham D, Lantos P (2002) Greenfield's neuropathology. Arnold, London
28. Hanbali F, Fourney DR, Marmor E, Suki D, Rhines LD, Weinberg JS, McCutcheon IE, Suk I, Gokaslan

ZL (2002) Spinal cord ependymoma: radical surgical resection and outcome. Neurosurgery 51:1162–1172; discussion 1172–1174
29. Hasegawa M, Fujisawa H, Hayashi Y, Tachibana O, Kida S, Yamashita J (2001) Surgical pathology of spinal schwannomas: a light and electron microscopic analysis of tumor capsules. Neurosurgery 49:1388–1392; discussion 1392–1393
30. Hirose Y, Aldape K, Bollen A, James CD, Brat D, Lamborn K, Berger M, Feuerstein BG (2001) Chromosomal abnormalities subdivide ependymal tumors into clinically relevant groups. Am J Pathol 158:1137–1143
31. Houten JK, Cooper PR (2000) Spinal cord astrocytomas: presentation, management and outcome. J Neurooncol 47:219–224
32. Houten JK, Weiner HL (2000) Pediatric intramedullary spinal cord tumors: special considerations. J Neurooncol 47:225–230
33. Isaacson SR (2000) Radiation therapy and the management of intramedullary spinal cord tumors. J Neurooncol 47:231–238
34. Isu T, Abe H, Iwasaki Y, Akino M, Koyanagi I, Hida K, Miyasaka K, Saito H (1991) Diagnosis and surgical treatment of spinal hemangioblastoma. No Shinkei Geka 19:149–155
35. Iwasaki Y, Hida K, Sawamura Y, Abe H (2000) Spinal intramedullary ependymomas: surgical results and immunohistochemical analysis of tumour proliferation activity. Br J Neurosurg 14:331–336
36. Jallo GI, Freed D, Epstein FJ (2004) Spinal cord gangliogliomas: a review of 56 patients. J Neurooncol 68:71–77
37. Kabus D, Sidhu GS, Wieczorek RL, Choi HS (1993) Metastatic meningioma. Hemangiopericytoma or angioblastic meningioma? Am J Surg Pathol 17: 1144–1150
38. Kahan H, Sklar EM, Post MJ, Bruce JH (1996) MR characteristics of histopathologic subtypes of spinal ependymoma. AJNR Am J Neuroradiol 17:143–150
39. Kalayci M, Cagavi F, Gul S, Yenidunya S, Acikgoz B (2004) Intramedullary spinal cord metastases: diagnosis and treatment – an illustrated review. Acta Neurochir (Wien) 146:1347–1354; discussion 1354
40. Klekamp J, Samii M (2007) Surgery of spinal tumors. Springer, New York
41. Kothbauer KF, Deletis V, Epstein FJ (1998) Motor-evoked potential monitoring for intramedullary spinal cord tumor surgery: correlation of clinical and neurophysiological data in a series of 100 consecutive procedures. Neurosurg Focus 4(5):e1
42. Kulkarni AV, Armstrong DC, Drake JM (1999) MR characteristics of malignant spinal cord astrocytomas in children. Can J Neurol Sci 26:290–293
43. Lee DK, Choe WJ, Chung CK, Kim HJ (2003) Spinal cord hemangioblastoma: surgical strategy and clinical outcome. J Neurooncol 61:27–34
44. Lee KS, Spetzler RF (1990) Spinal cord cavernous malformation in a patient with familial intracranial cavernous malformations. Neurosurgery 26:877–880
45. Lee M, Rezai AR, Abbott R, Coelho DH, Epstein FJ (1995) Intramedullary spinal cord lipomas. J Neurosurg 82:394–400
46. Lodrini S, Lasio G, Cimino C, Pluchino F (1991) Hemangioblastomas: clinical characteristics, surgical results and immunohistochemical studies. J Neurosurg Sci 35:179–185
47. Louis D, Ohgaki H, Wiestler O, Cavenee W (eds) (2007) WHO classification of tumors of the central nervous system. International Agency for Research on Cancer, Lyon
48. Lunardi P, Missori P, Gagliardi FM, Fortuna A (1989) Long-term results of the surgical treatment of spinal dermoid and epidermoid tumors. Neurosurgery 25:860–864
49. Mellon CD, Carter JE, Owen DB (1988) Ollier's disease and Maffucci's syndrome: distinct entities or a continuum. Case report: enchondromatosis complicated by an intracranial glioma. J Neurol 235:376–378
50. Naderi S (2000) Spinal meningiomas. Surg Neurol 54:95
51. Ng A, Pizer B, May P (2000) Congenital spinal astrocytoma: how favourable is the long-term outcome? Br J Neurosurg 14:366–370
52. Nishio S, Morioka T, Fujii K, Inamura T, Fukui M (2000) Spinal cord gliomas: management and outcome with reference to adjuvant therapy. J Clin Neurosci 7:20–23
53. O'Keefe T, Ramirez H Jr, Huggins MJ, Bennett WF, Blumenkopf B (1985) Computed tomography detection of cervical spinal cord hemangioblastoma: a case report. J Comput Tomogr 9:249–252
54. Park CK, Chung CK, Choe GY, Wang KC, Cho BK, Kim HJ (2000) Intramedullary spinal cord ganglioglioma: a report of five cases. Acta Neurochir (Wien) 142:547–552
55. Patel U, Pinto RS, Miller DC, Handler MS, Rorke LB, Epstein FJ, Kricheff II (1998) MR of spinal cord ganglioglioma. AJNR Am J Neuroradiol 19:879–887
56. Reddy P, Sathyanarayana S, Acharya R, Nanda A (2003) Intramedullary spinal cord metastases: case report and review of literature. J La State Med Soc 155:44–45
57. Sandalcioglu IE, Wiedemayer H, Gasser T, Asgari S, Engelhorn T, Stolke D (2003) Intramedullary spinal cord cavernous malformations: clinical features and risk of hemorrhage. Neurosurg Rev 26:253–256
58. Schwartz TH, McCormick PC (2000) Intramedullary ependymomas: clinical presentation, surgical treatment strategies and prognosis. J Neurooncol 47:211–218
59. Spetzger U, Gilsbach JM, Bertalanffy H (1995) Cavernous angiomas of the spinal cord clinical presentation, surgical strategy, and postoperative results. Acta Neurochir (Wien) 134:200–206
60. Sun B, Wang C, Wang J, Liu A (2003) MRI features of intramedullary spinal cord ependymomas. J Neuroimaging 13:346–351
61. Yu JS, Short MP, Schumacher J, Chapman PH, Harsh GR 4th (1994) Intramedullary hemorrhage in spinal cord hemangioblastoma. Report of two cases. J Neurosurg 81:937–940

Rheumatoid Arthritis

35

Scott D. Daffner and Colleen M. Watkins

35.1 Case Example

A 54-year-old woman with a long-standing history of rheumatoid arthritis returns to clinic complaining of worsening suboccipital neck pain. She has known atlantoaxial instability which has been followed regularly for many years. She denies changes in gait or balance, difficulty with fine motor skills, or radicular symptoms. On examination, she has mild tenderness to palpation posteriorly in the proximal cervical spine. Flexion slightly exacerbates her neck pain, and extension of the neck produces a clunking sensation. Motor strength is 5/5, sensation is intact, and deep tendon reflexes are normal in both upper and lower extremities. She has no long tract signs. Radiographic examination (Fig. 35.1) demonstrates progression of her C1–C2 instability with significant motion on flexion and extension imaging and a posterior atlanto-dens interval measuring approximately 10 mm on flexion. Because of her progressive instability and concern over the decreasing space available for the cord, she underwent posterior C1–C2 fusion with C1 lateral mass and C1–C2 transarticular fixation.

35.2 Pathophysiology and Pathoanatomy

The disease process within the cervical spine is the same as elsewhere in the body; rheumatoid arthritis targets synovial joints, and the multiple synovial joints of the cervical spine are no exception [1]. The underlying inflammatory process consists of T lymphocytes, macrophages, and plasma cells in a hypervascular synovial pannus [29, 37]. As inflammation and pannus formation progress, erosion of joint, capsular, and bony structures can occur, leading to progressive instability. The cervical spine typically exhibits three patterns of instability: atlantoaxial (C1–C2) subluxation (AAS), subaxial subluxation, or cranial settling (basilar invagination or atlantoaxial impaction). These patterns may occur separately or in combination with one another.

35.2.1 Atlantoaxial Instability

The most common instability pattern seen in the rheumatoid cervical spine is AAS, which is the result of progressive pannus formation around the odontoid and inflammatory destruction of the upper cervical spine [1, 31, 54]. The periodontoid pannus may compress the spinal cord and can deform or erode the alar, apical, and transverse ligaments; the dens; and the C1–C2 lateral articular masses [11]. This results in instability (either static or dynamic) of 50–70 % of patients [41, 42, 52]. Anterior subluxation is most

S.D. Daffner, M.D. (✉) • C.M. Watkins, M.D.
Department of Orthopaedics, West Virginia University, 3400 Health Sciences Center – 9196, Morgantown 26506-9196, WV, USA
e-mail: sdaffner@hsc.wvu.edu

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_35, © Springer-Verlag Berlin Heidelberg 2014

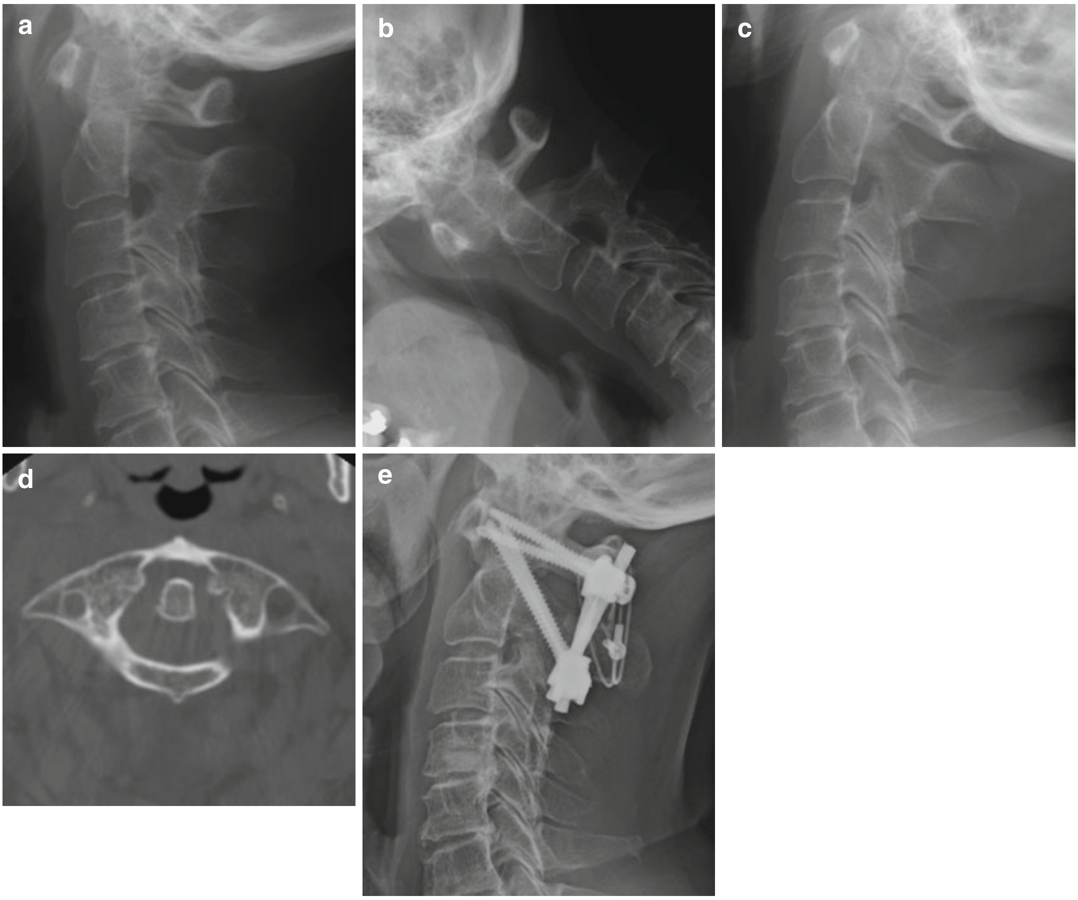


Fig. 35.1 A 54-year-old woman presents with worsening atlantoaxial subluxation (see text for details). Lateral neutral (**a**), flexion (**b**), and extension (**c**) radiographs demonstrate C1–C2 instability with subluxation and decreased posterior atlanto-dens interval (<10 mm) on flexion and reduction of the subluxation on extension. An axial CT scan through the C1 ring (**d**) demonstrates increased anterior atlanto-dens interval. Postoperative lateral radiograph (**e**) following a posterior C1–C2 fusion utilizing C1 lateral mass screws and C2–C1 transarticular screw fixation

common; posterior, lateral, and rotational instabilities are also possible [7, 40]. Subluxation anteriorly of up to 3 mm is normal in adults. Subluxation of 3–6 mm suggests disruption of the transverse ligament, and more than 9 mm may be representative of disruption of all periodontoid capsuloligamentous structures and is an indication for surgery [38, 41, 42, 57]. When coupled with instability, the buildup of periodontoid pannus may cause spinal cord impingement and myelopathy. Paralysis may be the result of both neural compression and/or impairment of the vascular supply to the neural structures [17]. While instability may promote worsening pannus formation, surgical stabilization may lead to a decrease in the size of the pannus [31].

35.2.2 Subaxial Subluxation

The second most common instability pattern, subaxial subluxation, occurs due to destruction of the facet joints, interspinous ligaments, and degenerative changes of the intervertebral disc [30, 52]. The decrease in disc height, coupled with bony erosion of the facet joints and ligamentous laxity, creates a “step ladder” appearance of multilevel anterior subluxation

Fig. 35.2 Lateral radiograph depicting multilevel subaxial subluxation in a patient with rheumatoid arthritis. Note the step ladder appearance of the vertebrae

(Fig. 35.2). This instability may be static or dynamic. Soft tissue hypertrophy in conjunction with altered sagittal alignment may lead to neural compression.

35.2.3 Cranial Settling

Cranial settling is the least common, but most concerning instability pattern. It is due mostly to destruction of the cartilage of the atlantoaxial and occipitoatlantal joints rather than ligamentous laxity. As the cranium settles more caudally, the odontoid becomes positioned relatively more cranially, which places patients at increased risk for sudden death secondary to stenosis of the foramen magnum and brainstem. This may occur either statically or dynamically. Direct compression can block normal flow of cerebrospinal fluid and lead to obstructive hydrocephalus or syringomyelia.

35.3 History and Physical Exam Findings

The most common presenting symptom (to spine surgeons) is neck pain; however with instability, nerve impingement may occur. In the upper cervical spine, this may manifest as occipital pain or headaches due to compression of the greater and lesser occipital nerves. Such findings likely suggest atlantoaxial instability or cranial settling, whereas mid to lower cervical pain may be more likely with subaxial subluxation. As instability increases in the subaxial spine, painful sagittal deformity may occur, which can eventually become a fixed deformity. In patients with AAS, spontaneous reduction of subluxation with neck extension may produce a clunking sensation; this is referred to as the Sharp-Purser test [49].

As instability progresses, the cross-sectional area of the spinal canal decreases, leading to compression on the spinal cord or brainstem. Mechanical compression and ischemic changes secondary to compression of the spinal cord may cause myelopathy and vertebrobasilar dysfunction [17]. Patients may develop a so-called cruciate paralysis which can mimic a mild central cord syndrome [3]. Instability may also lead to compression of the vertebral artery. Disruption of vertebrobasilar blood flow can lead to Wallenberg's syndrome or lateral medullary infarction. This manifests as ipsilateral palsies of cranial nerves V, IX, X, and XI; cerebellar ataxia; Horner's syndrome; facial pain; and contralateral loss of pain and temperature sensation [22]. Quadriplegia, locked-in syndrome, and sudden death have also been reported in rheumatoid patients [37, 41, 42, 53].

Patients must be evaluated within the context of their disease process. For example, upper extremity deformities may mask typical myelopathic symptoms such as difficulty with fine motor skills; worsening ambulation may initially be attributed to hip or knee involvement. The clinician, therefore, must maintain a high

Table 35.1 The Ranawat classification for pain and neurologic deficit in patients with rheumatoid arthritis

Pain	
Grade 0	No pain
Grade 1	Mild, intermittent
Grade 2	Moderate
Grade 3	Severe
Neurologic function	
Class I	No deficit
Class II	Subjective weakness, hyperreflexia, dysesthesias
Class IIIA	Objective paresis and long tract signs, ambulatory
Class IIIB	Quadriparesis, nonambulatory, or unable to feed oneself

Adapted from Ranawat et al. [43]

index of suspicion so as not to attribute early myelopathic signs to progression of peripheral disease. Ranawat developed a grading system which is predictive of outcome (Table 35.1) [43]. Casey et al. examined patients who were Ranawat Class IIIB (quadriparetic, nonambulatory) and found that only 25 % of patients undergoing surgery had a good outcome, with a 30-day mortality rate of 13 % which increased to 60 % at 4 years postoperatively [10]. Thus, it is important to identify and treat patients early in the disease process.

35.4 Differential Diagnosis

The findings associated with rheumatoid arthritis of the cervical spine may be seen in other disease processes; therefore, it is critical to realize that just because one or more of these findings is noted on radiographic examination, it does not mean the patient has rheumatoid arthritis. Calcium pyrophosphate deposition disease, or pseudogout, may cause the development of a periodontoid pannus similar in appearance to that of rheumatoid arthritis [60]. Similarly, psoriatic arthritis may mimic the upper cervical findings characteristic of the rheumatoid spine [33]. Infection, in particular tuberculosis, can cause bony erosions and soft tissue mass accumulation around the odontoid [50]. Clinically, myelopathy may also occur from degenerative spondylosis or disc herniation. Furthermore, subaxial subluxation may result from progressive degenerative changes of the discs and facet joint complexes.

35.5 Diagnostic Imaging

Initial cervical screening should be performed with plain radiography. A standard series of anteroposterior, open-mouth odontoid, and lateral views with [controlled] flexion and extension is appropriate and should provide basic information about the presence of any of the common instability patterns. Several radiographic measurements may be made which can assess the degree of instability.

35.5.1 Atlantoaxial Instability

For AAS, the most important measurement is the atlanto-dens interval. Classically, the anterior atlanto-dens interval (AADI), which is defined as the distance between the posterior margin of the anterior C1 ring and the anterior surface of the dens, has been used. However, the posterior atlanto-dens interval (PADI; defined as the distance between the posterior surface of the dens and the anterior margin of the posterior C1 ring) gives a more accurate assessment of the true space available for the spinal cord, and thus, the PADI may be a more reliable predictor of neurologic outcome [6]. An AADI of up to 3 mm is normal in adults, whereas 3–6 mm suggests instability (disruption of the transverse ligament) and greater than 9 mm indicates definite instability (disruption of all periodontoid capsuloligamentous structures) [57]. Boden et al. reported that patients with a PADI greater than or equal to 14 mm were more likely to have neurologic recovery following surgical treatment and those with a PADI of less than 10 mm had no recovery [6]. Open-mouth odontoid views can detect lateral subluxation (greater than 2 mm of displacement). Posterior subluxation can occur in patients with a fracture of the odontoid or an absent dens (secondary to bony destruction) and can be assessed on the lateral views.

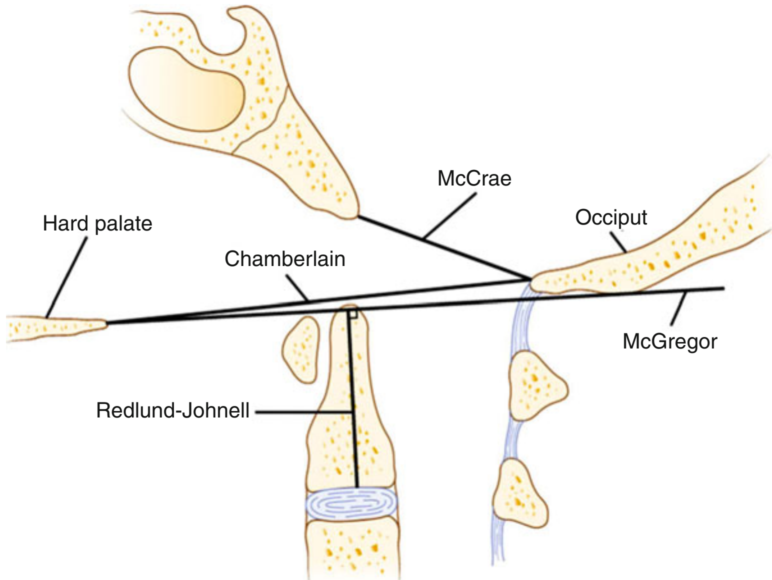


Fig. 35.3 Schematic depicting lines used for radiographic measurement of cranial settling. See text for details (From Daffner and Emery [16]. Used without permission. Copyright © 2011, 2006, 1999, 1992, 1982, 1975 by Saunders, an imprint of Elsevier Inc.)

35.5.2 Cranial Settling

Several measurement techniques have been described for assessing cranial settling and basilar invagination (Fig. 35.3). McGregor's line is drawn from the posterior edge of the bony palate to the base of the occiput, and if the dens is more than 4.5 mm above this line, cranial settling has occurred. A measurement described by Redlund-Johnell and Peterson places a vertical line from the midpoint of the caudal margin of C2 running to McGregor's line; they defied cranial settling to have occurred when the line is less than 34 mm in men and 29 mm in women [44]. Ranawat defined cranial settling as a distance less than 15 mm in men (13 mm in women) along a line drawn from the C2 pedicle to the transverse axis of the C1 ring [43]. Clark et al. described the "station of the atlas" by noting the position of the atlas relative to the odontoid process, which is divided in thirds [13]. The C1 ring normally resides adjacent to the upper third, with mild settling defined as migration to the middle third and severe cranial settling if the ring is adjacent to the caudal third of the dens. Although anatomic variations (such as erosion of the dens) can make any of these measurements individually less sensitive or specific, Riew et al. found that using a combination of the Clark station, the Redlund-Johnell line, and the Ranawat criteria provided sensitivity of 94 % and negative predictive value of 91 % [45].

35.5.3 Subaxial Subluxation

Lateral flexion-extension radiographs provide an assessment of subaxial settling. "Instability" has long been defined as greater than 3.5 mm of translation or greater than 11° of angular change between adjacent motion segments [58]. More importantly, however, is the space available for the spinal cord. One study reported that a canal sagittal diameter less than 14 mm should be considered "critical stenosis" for patients with rheumatoid arthritis [6].

35.5.4 Advanced Imaging

Computed tomography scans (CT) provide excellent bony detail. When combined with intrathecal contrast (CT myelogram), the degree of cord compression can be assessed. Magnetic resonance imaging (MRI) provides the best definition of soft tissue structures and best delineates the presence of periodontoid pannus, the degree of spinal cord compression, nerve impingement, or the extent of brainstem compression [4, 9, 18]. The cervicomedullary angle may be measured on midsagittal MR images as the intersection of a line drawn along the anterior surface of the brainstem and the anterior aspect of the spinal cord; a measurement of less than 135° correlates with myelopathic findings [9].

35.6 Treatment

35.6.1 Nonsurgical Treatment

Advances in medical management of rheumatoid arthritis have delayed the natural progression of the disease in many patients. Fewer patients are presenting for surgical intervention. Patients with mild intermittent pain without radiographic evidence of instability or findings of myelopathy may be managed with a soft cervical collar or physical therapy. Such patients should be followed closely at regular intervals for progression of radiographic instability or neurologic symptoms.

35.6.2 Surgical Treatment

35.6.2.1 Indications and Preoperative Management

The goals of surgery are spinal stabilization, decompression of neural elements, restoration of sagittal alignment, and pain relief. The presence of instability with or without pain, myelopathy, or other neurologic deficit in a patient with rheumatoid arthritis is a relative indication for early surgery. An AADI of greater than 9 mm, PADI of less than 14 mm, subaxial subluxation greater than 3.5 mm, spinal cord compression (or space available for the cord less than 14 mm), or cranial settling are all radiographic indications for surgery. The decision for surgery, however, must be made in the context of the patient's entire clinical picture.

Early surgery is generally more beneficial to patients. Schmitt-Sody et al. found better neurologic recovery when treating patients who were Ranawat Class II (seven of ten patients improved) as opposed to those who were Class III (one of eleven improved; two patients deteriorated) [48]. Agarwal et al. found that when surgery was performed before cranial settling occurred, there was less likelihood of recurrent (subaxial) cervical instability [1].

Preoperative skeletal traction should be considered for patients with severe basilar invagination. Gentle, midline traction of 7–12 lb, avoiding hyperextension or hyperflexion, for 3–7 days can improve alignment, neurologic symptoms, and pain [32, 39, 55]. Patients should be monitored with frequent neurologic examinations, and plain radiographs must be obtained to avoid overdistraction.

Perioperative management of rheumatoid medications is also important. Many of the commonly used drugs function as immune modulators or anti-inflammatories, which can place patients at risk for perioperative complications such as wound infections, delayed wound healing, or delayed fusion. Some medications can have systemic effects if abruptly discontinued. Patients on chronic corticosteroids may require stress dosing perioperatively. There are currently no studies in the literature specifically addressing use of these medications in patients undergoing spinal surgery. The surgeon should work closely with the patient's rheumatologist to develop a plan for perioperative dosing of rheumatoid medications.

35.6.2.2 Operative Procedures

The surgical approach should be determined by the location of the underlying pathology and the surgical goals. In many ways, this is similar to the means of deciding on approach for treating patients with cervical spondylotic myelopathy [12, 19]. Anterior, posterior, or combined approaches may be used.

Atlantoaxial Instability

Posterior fusion is the procedure of choice for treatment of C1–C2 instability. Brooks or Gallie sublaminar wiring techniques require an intact posterior arch of C1 [8, 20]. With the advent of screw fixation, wiring techniques are now used only as an adjuvant, for example, to hold a bone graft in place. Transarticular screw fixation, as described by Magerl, provides multidirectional stability [34]. More recently, posterior C1–C2 screw-rod constructs have become more popular. As described by Harms and Melcher, this technique entails placement of screws into the lateral mass of C1 and the pars interarticularis or pedicle of C2 bilaterally, followed by attachment of vertical rods and bone grafting [23]. This procedure has become the technique of choice. In patients with abnormal C2 anatomy (small pedicles or a large vertebral artery), screws may be placed into the C2 lamina and connected to the C1 screws [59].

Occipitocervical Fusion

Occipitocervical fusion is the technique of choice for treatment of cranial settling or for patients with fixed atlantoaxial subluxation causing cord impingement (from the posterior C1 ring) [32, 36, 39]. This technique allows a C1 laminectomy to be performed while still providing adequate surface for bone grafting. Wiring techniques are largely of historical significance and may only be used to secure a graft. Rigid fixation with a variety of occipital plate-screw-rod constructs is now the preferred method [21, 26, 47, 51].

Resection of the Odontoid

In most patients, the periodontoid pannus will resorb with posterior stabilization, and a C1 laminectomy and occipitocervical fusion may be all that is necessary [31]. An anterior decompression and resection of the odontoid may be required in cases of irreducible anterior extradural compression by the pannus or a severely migrated odontoid [15, 28]. The transoral approach is technically straightforward; however, it is associated with a high risk of infection. The high retropharyngeal anterior approach is an alternative [35]. Supplemental posterior fusion is required and may be performed before or after the anterior procedure.

Subaxial Fusion

Fixed subaxial subluxation may best be treated with an anterior cervical decompression and fusion. There is a relative risk of anterior graft resorption due to the rheumatoid disease process and a risk of settling of the graft into osteoporotic bone; therefore, one should consider supplemental posterior fusion. Mobile subaxial subluxation is best treated with traction (usually intraoperative positioning rather than preoperative traction is all that is required) and posterior instrumented fusion. Lateral mass screw-rod constructs are the preferred technique; they can be placed safely and provide excellent fixation [2, 24].

35.7 Postoperative Care

After surgery, all patients should be monitored very closely for neurologic changes and airway compromise. All patients receive intravenous antibiotics for 24 h postoperatively. Prophylaxis for deep vein thrombosis is generally mobilization and use of sequential compression devices and compression stockings. Patients are usually placed in a rigid cervical collar for 6–12 weeks postoperatively. With the advent of rigid internal fixation, postoperative halo immobilization is rarely required. Patients are mobilized early during their hospitalization. After the collar is removed, outpatient physical therapy may begin for strengthening and gentle range of motion. Early and regular clinical follow-up is recommended to monitor wound healing in this population which is at risk for infection. Frequent radiographic imaging is required to monitor for healing of the fusion as well as any potential instrumentation-related complications (e.g., loosening or breakage). The optimal timing for resumption of rheumatoid medications is not clear, and this should be discussed and a plan developed preoperatively with the patient and his or her rheumatologist.

35.8 Potential Complications

Complications of cervical spine surgery in patients with rheumatoid arthritis include but are not limited to bleeding, infection, wound dehiscence, damage to neurovascular structures, quadriplegia, nonunion or delayed fusion, hardware failure, and subaxial subluxation adjacent to a fused segment. Anterior fusion procedures add the risks of anterior approach (such as dysphagia, dysphonia, and damage to anterior cervical soft tissue structures) and are also at risk for graft resorption and anterior column collapse or instability due to the progression of the disease or vertebral body osteoporosis. Long-term clinical and radiographic follow-up is required to monitor for the development of progressive symptoms at previously unaffected (or asymptomatic) levels. Clarke et al. found that 39 % of patients who underwent C1–C2 fusion developed subaxial subluxation at an average 8.3 year follow-up [14]. Ronkainen et al. found that age, presence of atlantoaxial instability, and perioperative complications were independent predictors of long-term mortality after cervical surgery in patients with rheumatoid arthritis [46].

Questions

1. A 56-year-old woman with a history of rheumatoid arthritis presents to clinic complaining of worsening neck pain, subjective upper and lower extremity weakness, and difficulty with fine motor skills. On examination, motor strength is slightly diminished, gait is normal, and she is hyperreflexic. The most important preoperative imaging finding which correlates with neurologic recovery following surgical treatment is:
   (a) Anterior atlanto-dens interval of 5 mm
   (b) Posterior atlanto-dens interval of >14 mm
   (c) Basilar invagination of 7 mm
   (d) Subaxial subluxation of <3 mm
   (e) Rotatory subluxation of <8°
   Preferred response: (b)
   Patients with rheumatoid arthritis and neurologic deterioration due to C1–C2 instability are more likely to achieve neurologic recovery if the posterior atlanto-dens interval (PADI) is greater than 10 mm on preoperative radiographic examination, and at least one study suggests that patients with neurologic symptoms and a PADI greater than 14mm will achieve full recovery of motor function.
2. A 62-year-old woman with rheumatoid arthritis presents complaining of suboccipital neck pain, motor weakness, and difficulty with fine motor skills and gait/balance. Examination demonstrates hyperreflexia, unsteady gait, and mild objective weakness. Plain radiographs demonstrate the tip of the dens to be approximately 6 mm above a line drawn from the bony palate to the occiput, and MRI demonstrates a large pannus behind the dens, compressing the spinal cord with associated hyperintense signal on T2-weighted imaging. The most appropriate surgical management of this patient would be:
   (a) Posterior C1–C2 fusion with C1 lateral mass and C2 pedicle screws
   (b) Posterior C1–C2 fusion with C1–C2 transarticular screws
   (c) C1 decompressive laminectomy
   (d) Posterior occiput to C3 fusion
   (e) C1 laminectomy and posterior occipitocervical fusion
   Preferred response: (e)
   This patient has cranial settling and neurologic symptoms stemming from the large rheumatoid pannus placing pressure on the spinal cord behind the dens at the level of the C1 ring. A C1 laminectomy will allow decompression of the spinal cord, but because of the cranial settling, an occipitocervical fusion must be performed. C1 laminectomy alone would not halt the progression of the basilar invagination and may lead to worsening neurologic symptoms if the brainstem becomes involved. Occipitocervical fusion alone does not address the need to decompress the spinal cord at the C1 level. Posterior C1–C2 fusion does not address either pathologic process.
3. C1–C2 instability with an anterior atlanto-dens interval of 5mm suggests disruption of what structure(s)?
   (a) Alar ligaments
   (b) Apical ligament
   (c) Transverse ligament
   (d) Apical, alar, and transverse ligaments
   (e) None of the above as this is a normal anatomic variant
   Preferred response: (c)
   An anterior atlanto-dens interval (AADI) of up to 3 mm is normal in adults. Subluxation of between 3 and 6 mm suggests disruption of the transverse ligament. Subluxation greater than

9 mm suggests disruption of apical, alar, and transverse ligaments and is an indication for surgery.

4. After surgical stabilization, periodontoid pannus has been found to:
   (a) Continue to proliferate at an increased rate
   (b) Continue to proliferate at a decreased rate
   (c) Migrate cranially
   (d) Migrate caudally
   (e) Decrease in size

   Preferred response: (e)

   Following surgical stabilization, periodontoid pannus has been found to resorb and decrease in size. On rare occasions, it may persist following posterior fusion, necessitating an anterior resection of the odontoid and the pannus via either transoral or high retropharyngeal approach.

References

1. Agarwal AK, Peppelman WC, Kraus DR et al (1992) Recurrence of cervical spine instability in rheumatoid arthritis following previous fusion: can disease progression be prevented by early surgery? J Rheumatol 19:1364–1370
2. An HS, Gordin R, Renner K (1991) Anatomic considerations for plate-screw fixation of the cervical spine. Spine 16(10 suppl):S548–S551
3. Bell HS (1970) Paralysis of both arms from injury of the upper portion of the pyramidal decussation: "cruciate paralysis.". J Neurosurg 33:376–380
4. Bell GR, Stearns KL (1991) Flexion-extension MRI of the upper rheumatoid cervical spine. Orthopedics 14:969–974
5. Bland JH (1974) Rheumatoid arthritis of the cervical spine. J Rheumat 3:319–341
6. Boden SD, Dodge LD, Bohlman HH et al (1993) Rheumatoid arthritis of the cervical spine: a long term analysis with predictors of paralysis and recovery. J Bone Joint Surg 75A:1282–1297
7. Bogduk N, Major GA, Carter J (1984) Lateral subluxation of the atlas in rheumatoid arthritis: a case report and postmortem study. Ann Rheum Dis 43:341–346
8. Brooks AL, Jenkins EB (1978) Atlanto-axial arthrodesis by the wedge compression method. J Bone Joint Surg 60A:279–284
9. Bundschuh C, Modic MT, Kearney F et al (1988) Rheumatoid arthritis of the cervical spine: surface-coil MR imaging. AJR Am J Roentgenol 151:181–187
10. Casey AT, Crockard HA, Bland JM et al (1996) Predictors of outcome in the quadriparetic nonambulatory myelopathic patient with rheumatoid arthritis: a prospective study of 55 surgically treated Ranawat class IIIb patients. J Neurosurg 85:574–581
11. Chen TY, Lin KL, Ho HH (2004) Morphologic characteristics of atlantoaxial complex in rheumatoid arthritis and surgical consideration among Chinese. Spine 29:1000–1004
12. Chin KR, Ozuna R (2000) Options in the surgical treatment of cervical spondylotic myelopathy. Curr Opin Orthop 11:151–157
13. Clark CR, Goetz DD, Menezes AH (1989) Arthrodesis of the cervical spine in rheumatoid arthritis. J Bone Joint Surg 71A:381–392
14. Clarke MJ, Cohen-Gadol AA, Ebersold MJ et al (2006) Long term incidence of subaxial cervical spine instability following cervical arthrodesis surgery in patients with rheumatoid arthritis. Surg Neurol 66:136–140
15. Crockard HA, Calder I, Ransford AO (1990) One-stage transoral decompression and posterior fixation in rheumatoid atlanto-axial subluxation. J Bone Joint Surg 72B:682–685
16. Daffner SD, Emery SE (2011) Surgical management of rheumatoid arthritis. In: Herkowitz HN, Garfin SR, Eismont FJ, Bell GR, Balderston RA (eds) Rothman-simeone: the spine, 6th edn. Elsevier, Philadelphia, pp 651–669
17. Delamarter RB, Bohlman HH (1994) Postmortem osseous and neuropathologic analysis of the rheumatoid cervical spine. Spine 19:2267–2274
18. Dvorak J, Grob D, Baumgartner H et al (1989) Functional evaluation of the spinal cord by magnetic resonance imaging in patients with rheumatoid arthritis and instability of upper cervical spine. Spine 14:1057–1064
19. Emery SE, Bohlman HH, Bolesta MJ et al (1998) Anterior cervical decompression and arthrodesis for the treatment of cervical spondylotic myelopathy. Two to seventeen-year follow-up. J Bone Joint Surg 80A:941–951
20. Gallie WE (1939) Fractures and dislocations of the cervical spine. Am J Surg 46:495–499
21. Grob D, Dvorak J, Panjabi M et al (1994) The role of plate and screw fixation in occipitocervical fusion in rheumatoid arthritis. Spine 19:2545–2551
22. Gurley JP, Bell GR (1997) The surgical management of patients with rheumatoid cervical spine disease. Rheum Dis Clin North Am 23:317–332
23. Harms J, Melcher R (2001) Posterior C1-C2 fusion with polyaxial screw and rod fixation. Spine 26:2467–2471
24. Heller JG, Carlson GD, Abitbol JJ et al (1991) Anatomic comparison of the Roy-Camille and Magerl techniques for screw placement in the lower cervical spine. Spine 16(10 suppl):S552–S557
25. Heywood AWB, Meyers OL (1986) Rheumatoid arthritis of the thoracic and lumbar spine. J Bone Joint Surg 68B:362–368
26. Huckell CB, Buchowski JM, Richardson WJ et al (1999) Functional outcome of plate fusions for disorders of the occipitocervical junction. Clin Orthop 359:136–145
27. Kawaguchi Y, Matsuno H, Kanamori M et al (2003) Radiologic findings of the lumbar spine in patients

with rheumatoid arthritis, and a review of pathologic mechanisms. J Spinal Disord Tech 16:38–43
28. Kerschbaumer F, Kandziora F, Klein C et al (2000) Transoral decompression, anterior plate fixation, and posterior wire fusion for irreducible atlantoaxial kyphosis in rheumatoid arthritis. Spine 25:2708–2715
29. Kontinnen Y, Santavirta S, Bergroth V et al (1986) Inflammatory involvement of cervical spine ligaments in rheumatoid arthritis. Acta Orthop Scand 57:587
30. Kudo H, Iwano K (1991) Surgical treatment of subaxial cervical myelopathy in rheumatoid arthritis. J Bone Joint Surg 73B:474–480
31. Larsson E-M, Holtås S, Zygmunt S (1989) Pre- and postoperative MR imaging of the craniocervical junction in rheumatoid arthritis. Am J Roentgenol 152:561–566
32. Lipson SJ (1985) Cervical myelopathy and posterior atlanto-axial subluxation in patients with rheumatoid arthritis. J Bone Joint Surg 67A:593–597
33. Lu K, Lee TC (1999) Spontaneous regression of periodontoid pannus mass in psoriatic atlantoaxial subluxation. Case report. Spine 24:578–81
34. Magerl F, Seeman PS (1986) Stable posterior fusion of the atlas and axis by transarticular screw fixation. In: Kehr P, Weidner A (eds) Cervical spine. Springer, Berlin, pp 322–327
35. McAfee PC, Bohlman HH (1989) One-stage anterior cervical decompression and posterior stabilization with circumferential arthrodesis: a study of twenty-four patients who had a traumatic or a neoplastic lesion. J Bone Joint Surg 71A:78–88
36. McAfee PC, Cassidy JR, Davis RF et al (1991) Fusion of the occiput to the upper cervical spine: a review of 37 cases. Spine 16(Suppl 10):S490–S494
37. Mikulowski P, Wollheim FA, Rotmil P et al (1975) Sudden death in rheumatoid arthritis with atlanto-axial dislocation. Acta Med Scand 198:445–451
38. Papadopoulos SM, Dickman CA, Sonntag VK (1991) Atlantoaxial stabilization in rheumatoid arthritis. J Neurosurg 74:1–7
39. Peppelman WC, Krauss DR, Donaldson WF III et al (1993) Cervical spine surgery in rheumatoid arthritis: improvement of neurologic deficit after cervical spine fusion. Spine 18:2375–2379
40. Rana NA (1989) Natural history of atlanto-axial subluxation in rheumatoid arthritis. Spine 14:1054–1056
41. Rana NA, Hancock DO, Taylor AR et al (1973) Atlanto-axial subluxation in rheumatoid arthritis. J Bone Joint Surg 55B:458–470
42. Rana NA, Hancock DO, Taylor AR et al (1973) Upward translocation of the dens in rheumatoid arthritis. J Bone Joint Surg 55B:471–477
43. Ranawat CS, O'Leary P, Pellicci P et al (1979) Cervical spine fusion in rheumatoid arthritis. J Bone Joint Surg 61A:1003–1010
44. Redlund-Johnell I, Pettersson H (1984) Radiographic measurements of the craniovertebral region. Designed for evaluation of abnormalities in rheumatoid arthritis. Acta Radiol Diagn 25:23–28
45. Riew KD, Hilibrand AS, Palumbo MA et al (2001) Diagnosing basilar invagination in the rheumatoid patient: the reliability of radiographic criteria. J Bone Joint Surg 83A:194–200
46. Ronkainen A, Niskanen M, Auvinen A, Aalto J, Luosujarvi R (2006) Cervical spine surgery in patients with rheumatoid arthritis: long term mortality and its determinants. J Rheumatol 33:517–522
47. Sasso RC, Jeanneret B, Fischer K et al (1994) Occipitocervical fusion with posterior plate and screw instrumentation. A long-term follow-up study. Spine 19:2364–2368
48. Schmitt-Sody M, Kirchhoff C, Buhmann S et al (2008) Timing of cervical spine stabilisation and outcome in patients with rheumatoid arthritis. Int Orthop 32:511–516
49. Sharp J, Purser DW (1961) Spontaneous atlantoaxial dislocation in ankylosing spondylitis and rheumatoid arthritis. Ann Rheum Dis 20:47–50
50. Shukla D, Mongia S, Devi BI et al (2005) Management of craniovertebral junction tuberculosis. Surg Neurol 63:101–6
51. Smith MD, Anderson P, Grady MS (1993) Occipitocervical arthrodesis using contoured plate fixation: an early report on a versatile fixation technique. Spine 18:1984–1990
52. Smith PH, Benn RT, Sharp J (1972) Natural history of rheumatoid cervical luxations. Ann Rheum Dis 31:431–439
53. Sunahara N, Matsunaga S, Mori T et al (1997) Clinical course of conservatively managed rheumatoid arthritis patients with myelopathy. Spine 22:2603–2608
54. Toolanen G, Larsson SE, Fagerlund M (1986) Medullary compression in rheumatoid atlanto-axial subluxation evaluated by computerized tomography. Spine 11:191–194
55. van Asselt KM, Lems WF, Bongartz EB et al (2001) Outcome of cervical spine surgery in patients with rheumatoid arthritis. Ann Rheum Dis 60:448–452
56. Ward MW (2004) Decreases in rates of hospitalization for manifestations of severe rheumatoid arthritis, 1983–2001. Arthritis Rheum 50:1122–1131
57. Weissman BN, Aliabadi P, Weinfeld MS et al (1982) Prognostic features of atlantoaxial subluxation in rheumatoid arthritis patients. Radiology 144:745–751
58. White AA, Panjabi MM (1990) Clinical biomechanics of the spine, 2nd edn. JB Lippincott, Philadelphia
59. Wright NM (2004) Posterior C2 fixation using bilateral, crossing C2 laminar screws: case series and technical note. J Spinal Disord Tech 17:158–162
60. Zünkeler B, Schelper R, Menezes AH (1996) Periodontoid calcium pyrophosphate dihydrate deposition disease: "pseudogout" mass lesions of the craniocervical junction. J Neurosurg 85:803–809

Ankylosing Spondylitis and Diffuse Idiopathic Skeletal Hyperostosis

36

Xuan Luo, Harvey E. Smith, Raymond Hwang, and Scott D. Daffner

36.1 Introduction

Ankylosing spondylitis (AS) and diffuse idiopathic skeletal hyperostosis (DISH) are two distinct musculoskeletal diseases with similar clinical and radiological characteristics. Both commonly manifest with back pain, believed to result from reduced range of motion in the spine and peripheral joints due to abnormal bone formation. Recognition of these conditions out of the more benign causes of back pain is crucial, as their chronic course can result in debilitating kyphosis, loss of flexibility, and a predisposition to spinal cord threatening fractures with minimal trauma [1, 2].

Ankylosing spondylitis, in brief, is a systemic *immune-mediated* attack on the entheses of the spine and other joints, especially the sacroiliac joints, resulting in bone destruction and subsequent abnormal bone reformation; its exact etiology is unknown. It is associated with many medical comorbidities such as anterior uveitis [1]. Diffuse idiopathic skeletal hyperostosis, on the other hand, is a *nonimmune-mediated* process of increased bone deposition in spinal ligaments and entheses. The underlying etiology is also unknown but appears related to advancing age, obesity, and diabetes [2].

Case

Patient is a 55-year-old male with polytrauma from a head-on motor vehicle collision with bilateral open femur fractures and an open tibia-fibula fracture. AP pelvis (Fig. 36.1) demonstrated fused SI joints. Sagittal CT scan demonstrated a

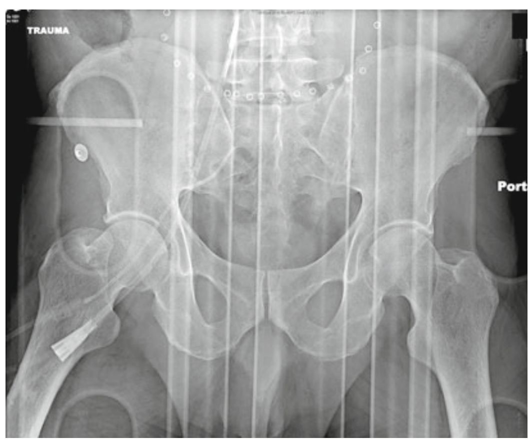


Fig. 36.1 AP X-ray of the pelvis, demonstrating fused SI joints

X. Luo
Medical Student,
Tufts University School of Medicine, Boston, MA, USA

H.E. Smith, M.D. (✉)
New England Orthopaedic and Spine Surgery,
830 Boylston Street, Suite 211 Chestnut Hill,
Newton, MA 02467, USA
e-mail: harveysmith27@gmail.com

R. Hwang, M.D.
Spine Surgery Fellow,
New England Baptist Hospital,
Boston, MA, USA

S.D. Daffner, M.D.
Department of Orthopaedic Surgery,
West Virginia University, Morgantown, WV, USA

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_36, © Springer-Verlag Berlin Heidelberg 2014

Fig. 36.2 Sagittal CT scan: note the anterior column extension distraction injury. (**a**) Coronal view. (**b**) Sagittal reconstruction

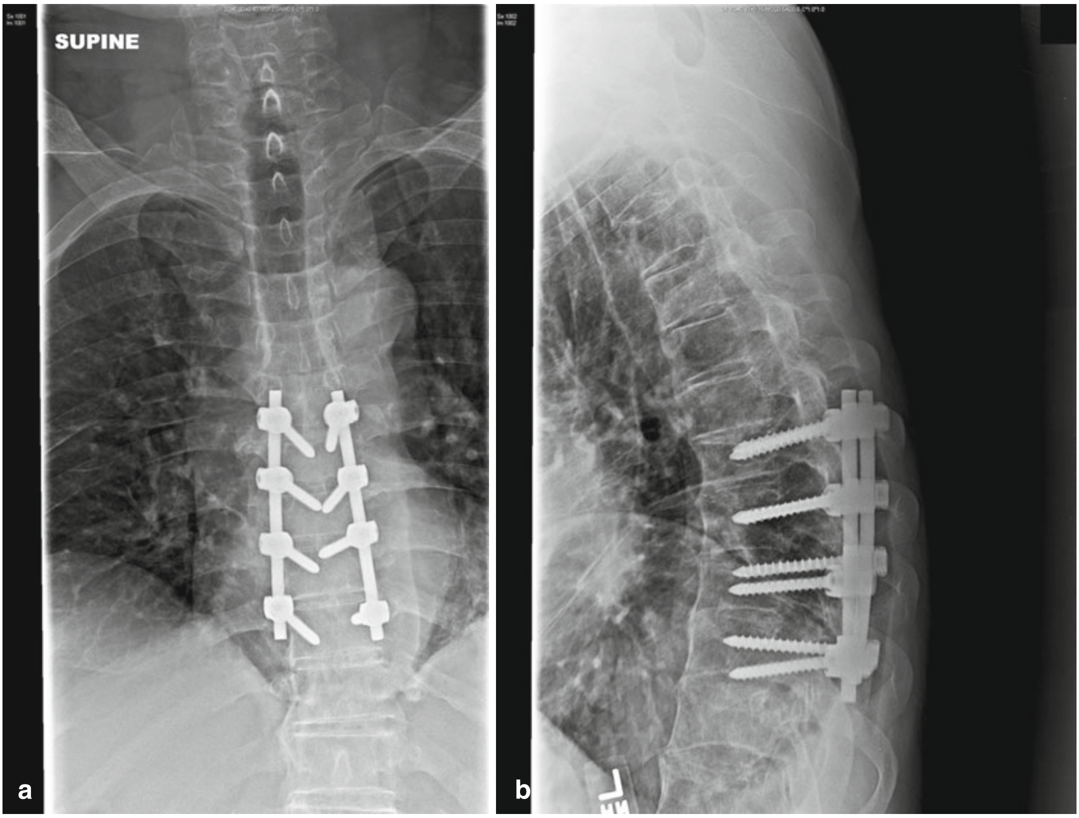


Fig. 36.3 AP (**a**) and Lateral (**b**) postoperative images demonstrate short-segment percutaneous instrumentation for stabilization of the fracture

discontinuity in the anterior osteophytes consistent with extension-distraction injury of T8–T9 (Fig. 36.2). Due to the nature of his multiple injuries and a desire to mobilize the patient quickly to chair after fixation of the extremities, it was elected to proceed with percutaneous stabilization of the T8–T9 fracture (Fig. 36.3).

36.1.1 History

Ankylosing spondylitis is an aptly named disease, deriving from the Greek word "ankylos" which carries the double meaning of "fusion" and "bent," both describing late manifestations of the disease. The first recognized descriptions came during the late 1,800 s, with a trio of papers published by Bechterew in Russia, Strumpell in Germany, and Marie in France. Bechterew originally described three patients ages 39, 52, and 56 who shared spinal stiffness, back pain, kyphosis, diffuse peripheral weakness, and mild peripheral sensory loss. Strumpell and Marie's later descriptions differed slightly in that spinal involvement was mostly thoracic and marked neurological deficit was a requirement for inclusion, though they found no peripheral joint involvement [3]. As a result of these pioneering papers, ankylosing spondylitis is also referred to as Bechterew's disease or Strumpell Marie's disease.

The earliest reports of diffuse idiopathic skeletal hyperostosis (DISH) in the mid-nineteeth century were based on examination of cadaveric spines of elderly patients, which revealed consistent patterns of bony change in the thoracic spine, often on the right side. It was not until the mid-twentieth century that clinicians began to extensively investigate these radiological patterns in living individuals. Forestier gave an apt description of the "flowing" bony formations found on the anterolateral aspects of the spine and bridging ossification across intervertebral discs that are now the hallmarks of the disease [4]. As a result of that detailed description, DISH is often referred to as Forestier's disease. The name DISH did not come about until a few years later when Resnick recognized that the classically described spinal disease occurs in the setting of extraspinal enthesis in such locations such as the pelvis and calcaneus, thus earning the disease's *diffuse* prefix [5].

36.1.2 Epidemiology

In the family of inflammatory diseases of the spine, the spondylarthopathies, ankylosing spondylitis is the most common, comprising between 45 and 61 % of all reported spondylarthopathies [6, 7]. Prevalence of ankylosing spondylitis differs depending on the population and study performed. Studies in Scandinavia have found the prevalence of ankylosing spondylitis to be between 1.9 and 2.2 % in males and .3–.6 % in females. In individuals expressing the HLA-B27 complex, the prevalence increases to 6.7 %. Most notably, in patients with HLA-B27 and back pain, the prevalence jumps to 22.5 % [8]. Studies in the United States have found similar rates. Early American studies in the 1970s found a prevalence of 1.29 % in the Caucasian population, with a markedly reduced prevalence in the African American population [9, 10]. More recent American data in 1998 confirms that the prevalence has remained relatively stable at 1.97 % for men, .73 % for women, and .48 % for African Americans [11]. In Asian countries, the reported prevalence is much lower at .23 %, though this may be due to under-recognition of the disease in those countries [12]. In addition, there appears to be a male predominance of ankylosing spondylitis. In Marie's early description of the disease, he wondered if it was only coincidence that all the affected patients were men. Initial studies in the 1940s supported a highly skewed ratio for men to women of 9–10:1 [13]. However, as the disease became better characterized in women, the ratio of men to women shifted closer to 2 or 3:1 [8, 14]. The most recent data collected through self-reported survey found a 2.5: 1 ratio for men compared to women [15].

As with ankylosing spondylitis (AS), the estimation of the prevalence of diffuse idiopathic skeletal hyperostosis (DISH) depends greatly on the population and the study performed. DISH may be related to age, obesity, diabetes, and glucose intolerance, all conditions rising in epidemic proportions in well-developed societies. Early large studies performed in Scandinavia found a relatively modest prevalence of DISH, ranging from .7 to 3.8 % in men and .4 to 2.6 % in women. Investigators found sharp increases with age, obesity, and weaker associations with diabetes and glucose intolerance [16, 17]. However, as DISH is often asymptomatic in patients until there is significant disease progression, true prevalence may be far higher as there may be a significant population with asymptomatic and unrecognized disease. On autopsy in patients over age 65 years, 28 % of spines showed changes consistent with DISH [18]. Later studies in smaller populations have reported as high as 27.3 % in men and 12.8 % in women over 50 and up to 36.6 % in men over 75 [19]. In the most recent study in older men, the overall prevalence was 42 % and as high as 56 % in men over the age of 80 [20]. There is an unusually high reported prevalence in Pima Indians, as high as 48 % overall in men over 55, well correlated with the population's high incidence of obesity, diabetes, and insulin resistance [21].

36.2 Pathogenesis

36.2.1 Ankylosing Spondylitis

The exact pathogenesis of ankylosing spondylitis is an area of active and ongoing research. Ankylosing spondylitis at its core is an immune-mediated inflammatory process most heavily involving tendon and ligament insertion sites of the sacroiliac joints and spine [22]. A proposed model of the ankylosing mechanism in AS starts with an initial inflammation mediated by monocytes and osteoclasts that cause erosive bone and cartilage destruction. The destroyed bone is then replaced by fibrous tissue which eventually becomes re-ossified by osteoblasts. The re-ossification no longer follows the structure of the original bone but rather that of the fibrous replacement, forming bony bridges between vertebrae called syndesmophytes. In late disease, the extent of the syndesmophytes results in a spine that is affectively auto-fused, the "bamboo" spine of late AS [23].

There are a number of theories concerning the etiology of the initial inflammatory process. At the core of the leading theories are the concepts of *susceptibility* and *exposure*. Ankylosing spondylitis manifests in individuals with the preexisting immunological circumstances that portend susceptibility to a precipitating event. The existence of susceptible individuals was recognized in the 1970s with the discovery that ankylosing spondylitis was tightly associated with the MHC-I molecule type HLA-B27 [24]. In fact, 90–95 % of persons affected with ankylosing spondylitis carry the HLA-B27 type. In subsequent research, this was further refined to certain subtypes, notably B2705, 2703, 2704, and 2707 being associated with AS [1]. The HLA-B27 molecule is responsible for presenting foreign antigens of bacteria or viruses from within infected cells to the surface for signaling to CD8 cytotoxic T cells. Faulty signaling may cause CD8 cytotoxic T cells to attack HLA-B27 cells that are not infected. Transgenic mice expressing human HLA-B27 develop a systemic inflammatory process that involves many organ systems, including the axial and peripheral joints [25].

Although over 90 % of ankylosing spondylitis patients express HLA-B27, only 5 % of HLA-B27 patients develop ankylosing spondylitis – this supports the hypothesis that additional factors are necessary to produce disease in the susceptible population. It has been hypothesized that certain pathogens produce antigens that activate the CD8 T cells against HLA-B27-expressing cells. Yersinia and Chlamydia species have been suspected as the exposure pathogens, although that research has been inconclusive [1]. In a transgenic mouse model for systemic inflammation from human HLA-B27, the inflammatory cascade was prevented if the mice were raised in a sterile environment [26].

In addition to HLA-B27, other proteins are likely involved. Linkage studies in twins demonstrate that HLA-B27 accounts for at most half of the genetic component of the disease [27]. Other hypothesized proteins include IL-1, IL-23, IL-17, and interferon gamma [22].

36.2.2 Diffuse Idiopathic Skeletal Hyperostosis

The etiology and pathogenesis of diffuse idiopathic skeletal hyperostosis is even less well understood than ankylosing spondylitis. It is distinct from ankylosing spondylitis in that it is a nonimmune-mediated process that results in excessive bone formation. There have been observed associations with metabolic diseases and obesity. The prevalence of DISH has been reported to be as high as 40 % in obese patients, 30 % in diabetes Type II patients, and 22 % in glucose intolerance patients [28]. The association of DISH with metabolic disease is controversial and study dependent [29, 30]. There have been investigations into involvement of insulin-like growth factors and vitamin A and K [31–33]. Other investigations of associated diseases involve dysregulation of parathyroid hormone, multiple myeloma, and rheumatoid arthritis. Additional studies have found hypervascularity of the ossified ligaments in DISH, suggesting vascular etiologies [34]. Curiously, in the thorax, DISH predominantly affects only the right side. The pattern is reversed in situs inversus, possibly due to location of the vasculature and protective effects of a pulsatile abdominal aorta.

36.3 Clinical Manifestations

Clinical judgment is paramount to the correct diagnosis and management of both AS and DISH. It is important to recognize that the primary symptom of many of ankylosing spondylitis and DISH patients is back pain, one of the most common and broadest differentials in medicine. Consequently, the diagnosis of AS or DISH is frequently incidental. A high index of suspicion in the setting of an ankylosed spinal segment is necessary, as unfortunately, these patients are at risk of devastating neurological compromise in the setting of relatively minor fractures. They have a significant risk of fracture displacement due to the long lever-arm forces of the ankylosed motion segments [35].

36.3.1 Ankylosing Spondylitis

Ankylosing spondylitis has a varied clinical presentation. The hallmarks of the condition are spinal *and* sacroiliac joint involvement. The primary symptom is back pain that is inflammatory in nature, often reported as most severe in intensity on waking, improved by exercise but not rest, worse at night, and better after rising. In addition to back pain, sacroiliac joint inflammation may cause buttock pain [36]. With disease progression, patients have progressive loss of spinal motion, with an end result of a rigid kyphosis that may limit chest expansion and prevent visualization of the horizon. The kyphosis is particularly debilitating, limiting both ambulation, balance, and feeding. Beyond the axial skeleton, peripheral involvement includes enthesitis, at the achilles, hip, shoulder, and jaw [36]. Extra-skeletal manifestations are also a feature of ankylosing spondylitis, the most common being anterior uveitis, ranging from 14 to 40 % in patients. Conversely, about 11 % of patients presenting with anterior uveitis were found to have ankylosing spondylitis based on a 7-year cohort of 514 patients [37]. There also may be gender differences in clinical presentation. Studies have noted a higher prevalence of cervical pain and greater change in the sacroiliac joints and cervical spine on radiological study in women [38]. However, radiological differences between the genders is controversial [39]. In addition, women may have more peripheral joint involvement and uveitis than men [40].

The eventual fusion of the spine causes a number of additional problems in late stage AS. One is the fixed kyphosis of the spine, eventually resulting in the "chin on chest" deformity where the patient can no longer look to see the horizon. The other is the predisposition to fractures with only minor trauma [35]. As syndesmophytes bridge the vertebral joints, mobility in each individual joint is lost. The spine begins to move and transmit force as a single rigid lever arm, which can exert enormous torque on adjacent vertebral bodies [35]. Most common sites of fracture are the cervical regions especially C5–C6, due to the heavy weight of the head supported on the relatively small cervical vertebral bodies and the mid thoracic spine due to the long lever arm. Rarely, severe complications can arise, causing neurological deficits due to atlanto-axial subluxation [41] or cauda equina syndrome [42]. In any case, there should be a high index of suspicion for fractures in patients with even minor trauma and pain. Consideration should also be given to treat these fractures more aggressively due to the risk of displacement and neural injury.

36.3.2 Diffuse Idiopathic Skeletal Hyperostosis

Diffuse idiopathic skeletal hyperostosis is often asymptomatic in patients and discovered incidentally on radiograph. In fact, in a comparison of elderly patients with DISH to normal controls, the DISH patients overall reported less back pain [20]. The most common reported symptoms are intermittent, non-radiating thoracic pain, associated with limited range of motion and stiffness [43]. Later disease also involves cervical spine, as growing osteophytes interfere with swallowing and motion, and also the lumbar spine, with more patients presenting with low back pain [2]. Cervical osteophytes most commonly manifest as dysphagia, up to 28 % of the time; however, more life threatening symptoms have been reported [5, 44]. The quality of the dysphagia is described as fluctuant, insidiously progressive, worse with swallowing solids, or with neck extension. Other cervical manifestations include chronic neck pain, hoarseness, foreign body sensation, and very rarely medullar compression from ossification of the posterior longitudinal ligament [45]. Additionally, cervical osteophytes may cause impingement on the airway, causing stridor, laryngeal edema, chronic obstructive pneumonia, and difficult or complicated intubation due to clinically hidden tracheal deviation

[46–48]. Long cervical osteophytes may even cause thoracic outlet syndrome and may cause acute respiratory distress in severe cases, requiring emergent tracheotomy [49, 50]. Extraspinal manifestations include tendinitis and enthesophytes (osseous outgrowths at the tendon attachment sites) in the pelvis and other peripheral joints [5, 29]. Ossification of the posterior ligament of the cervical spine may result in spinal cord compression and neurological compromise [2].

Similar to ankylosing spondylitis, fusion of the spine results in significant lever-arm forces on adjacent sections, increasing mechanical risks for fracture and displacement [35]. However, most recent studies have found that the risk for mortality for DISH after fracture may be exaggerated in prior studies and no different from controls [51].

36.4 Differential Diagnosis

AS and DISH are in a family of diseases that involve arthritic changes in the spine and peripheral joints. These include reactive arthritis, psoriatic arthritis, spondyloarthritis related to inflammatory bowel disease, and rheumatoid arthritis. Thus, it is important to establish any history of prior GI or GU infections, prior skin involvement, past history and family history of inflammatory bowel diseases and associated findings of inflammatory bowel disease, and small peripheral joint involvement, with rheumatoid factor screen [7].

36.5 Investigations/Diagnostic Imaging

36.5.1 Ankylosing Spondylitis

The diagnosis of AS is based on a constellation of clinical symptoms and radiologic findings. Diagnosis can be made when one *clinical criteria*, either (1) inflammatory back pain of at least 3 months duration, (2) limitation of lumbar spine motion in sagittal and frontal planes, or (3) decreased chest expansion relative to normal values for sex and age, is combined with the *radiological criteria* of either (1) bilateral sacroiliitis grade 2 or higher or (2) unilateral sacroiliitis grade 3 or higher [36]. To assess forward lumbar flexion, physicians often perform the Schober's test, which involves marking the back to document the amount of lengthening of the skin as a patient bends. Lateral lumbar flexion and decreased chest expansion are also useful in assessing back mobility. Occasionally, palpation and stress of the sacroiliac joint provides a diagnosis of sacroiliitis. The most sensitive test is imaging for sacroiliac joint widening, erosion, and sclerosis, especially with bilateral involvement; inflammation of the SI joints will eventually result in fusion. Sacroiliitis can be investigated with plain film, CT, or MRI; however, MRI has been shown to be the most sensitive. Classically described ossification and bridging of the spine are often late manifestations. In these severe cases, marginal vertebral body erosions, bony bridges called syndesmophytes spanning neighboring vertebral bodies, ossified spinal ligaments, and squared vertebral bodies can be seen on imaging. The total fusion of the spinal column in extremely advanced disease gives rise to the bamboo spine appearance that is classically described [36]. Ophthalmic exam for anterior uveitis may also be contributory. HLA-B27 testing may be useful keeping in mind that the vast majority of HLA-B27 patients do not have AS. In patients with back pain and HLA-B27, prevalence increases to 22 % [8]. Patients with suspected AS and back pain should be carefully investigated for fractures through both the vertebral disc and body, even with a history of minor or no trauma [52]. For fracture screening, multidetector CT is more sensitive than plain film or MRI [53]. If diagnosis continues to be equivocal, a trial of NSAIDs may demonstrate the inflammatory nature of the back pain and aid in AS diagnosis [54] (Table 36.1).

36.5.2 Diffuse Idiopathic Skeletal Hyperostosis

In diffuse idiopathic skeletal hyperostosis, the clinical findings are often more subtle and nonspecific, and much of the investigation is heavily reliant on radiological evidence. The

Table 36.1 Chest expansion values and Schober's test values

Chest expansion test	Schober's test
Chest circumference if measured at full inspiration and full expiration. The difference between the two measurements should normally be at least 1 in.	Place a mark on the patients back at approximately L5 and another mark 15 cm above the first mark, while the patient is upright. With the patient forward flexing fully (reaching for their toes) the marks should increase to at least 20 cm apart in normals

Fig. 36.4 AP (**a**), Lateral (**b**) x-ray and sagittal CT (**c**) scan of a patient with cervical DISH. Note the large syndesmophytes, which in this patient caused significant dysphagia

classic description is of large syndesmophytes and flowing laminated ossification across 3–4 vertebral joints especially on the right thoracic spine [2]. The appearance is similar to wax melting off a candle. Signs of degenerative joint/bone disease are uncommon. Involvement of the cervical spine is less common; when involved large osteophytes are present in this region (Fig. 36.4). As with AS, patients presenting with axial spine pain after relatively minor mechanical injury should be presumed to have a fracture until proven otherwise. An unrecognized fracture in this patient population can have devastating neurologic compromise if there is subsequent fracture displacement. Involved peripheral joints may show radiologic bone proliferation in the pattern of "whiskering" calcifications at the entheses. The sacroiliac joints are not involved, in contrast to AS [2]. The lack of disc height degeneration and absence of sacroiliac changes are used to distinguish DISH from degenerative joint diseases or inflammatory diseases such as ankylosing spondylitis [55].

36.6 Nonsurgical Treatment

36.6.1 Ankylosing Spondylitis

Treatment of ankylosing spondylitis includes physical therapy, education, pain control, anti-inflammatory drugs, disease-modifying antirheumatic drugs (DMARDs), and biologics [36, 54].

Physical therapy has been shown to improve function in the short term in limited studies. However, the long-term benefits are not well established at this time. Cognitive behavioral therapy has been shown to provide mild pain relief and management of anxiety. There is the added benefit of educating the patients on their susceptibility to fractures with trauma [54]. NSAIDs are the first line of management of AS. Beyond pain symptom relief, there is evidence of their efficacy in slowing disease progression as monitored by radiologic evaluation [56, 57]. Corticosteroids have limited support for use in AS beyond pain relief afforded by local injections into inflamed SI joints [36]. Simple analgesics are less effective than NSAIDs in controlling symptoms and are only indicated in patients where pain cannot be controlled by NSAIDs alone.

The antirheumatoid agents sulfasalazine and methotrexate have shown little to modest benefit. Sulfasalazine yields improvement in reported spinal stiffness but little improvement in patient function or objective evaluation of disease. Methotrexate was found to be effective in small studies, but overall, the evidence for its use is weak [36, 54]. For patients who fail to respond to two NSAIDs in 3 months with moderate disease, infliximab, etanercept, and adalimumab are the most powerful agents in use in ankylosing spondylitis. Unlike methotrexate or sulfasalazine which are presumed to slow the immune system by slowing production of nucleic acids and thus DNA, infliximab, etanercept, and adalimumab work by directly blocking the signaling proteins of inflammation. These drugs have shown improvement in clinical symptoms, imaging progression, and quality of life. In fact, they may work better in ankylosing spondylitis than they do in rheumatoid arthritis. Biologic treatment is often long term, as stopping treatment often brings rapid return of symptoms. These agents also predispose patients to infections, especially reactivation of latent TB, and patients should be evaluated for TB prior to their use [58, 59].

36.6.2 Diffuse Idiopathic Skeletal Hyperostosis

Treatment options for diffuse idiopathic skeletal hyperostosis are primarily for symptom relief. Studies for specific therapies are scarce, as the majority of patients are asymptomatic for long periods of time and often follow a benign course. Physical therapy can relieve pain and improve function based on anecdotal experiences from providers. Pain is often treated with analgesics and NSAIDs [2]. Local heat application is often used for additional pain relief. There are small studies involving alternative therapies such as chiropractic manipulations and acupuncture that report modest benefit. Other therapies reported include local steroid injections, lidocaine, massage, and therapeutic ultrasound. Additionally, there are reports of the use of low-dose radiation for the prevention of heterotopic bone formation after hip arthroplasty. There has been no therapy that has been reported to slow the natural history of the disease. One study investigating 24 weeks of exercise therapy found modest improvement in subjective pain and mobility as measured by the Schober bending test [60].

36.7 Surgical Treatment

Surgery for both conditions is a rare indication. For both ankylosing spondylitis and diffuse idiopathic skeletal hyperostosis, fractures may necessitate surgical management [35, 61, 62]. Additionally, severely kyphotic ankylosing spondylitis patients may benefit from surgical correction of "chin on chest deformity" [63, 64]. In patients with diffuse idiopathic skeletal hyperostosis, dysphagia caused by large cervical osteophytes are an additional indication for surgery [65].

36.7.1 Fractures

The surgical indications for spinal fracture in the setting of AS or DISH are controversial, and there is little consensus in the literature. Clinical decision-making should be based on the presence of active compression of the neural elements in the presence of a neurologic deficit, the presence of an epidural hematoma, or the need to achieve biomechanical stability.

In one study of spinal fractures in patients with ankylosing spondylitis and diffuse idiopathic skeletal hyperostosis, spinal cord injury was found in over half of the cases. Diagnosis was delayed in 19 % of the cases, leading to further neurological decline in 81 % of those patients [61]. Late neurologic decline is frequently the consequence of fracture displacement or epidural hematoma. Patients with ankylosing spondylitis are particularly at risk for spinal epidural hematomas that may lead to rapid compressive neurologic compromise. Early studies found up to a 50 % risk for spinal epidural hematoma with fracture in ankylosing spondylitis patients compared to nearly none in controls [66, 67]. The mechanism of this association is unclear as epidural hematomas also occur more often in ankylosing spondylitis patients undergoing epidural anesthesia [68, 69]. Surgical indications are controversial, but generally, surgery is utilized for fracture stabilization if closed immobilization fails or is not appropriate or to decompress neural elements in the setting of neurologic deficit. Studies have yet to demonstrate an effect of surgery on outcomes in the setting of neurologic deficit, and patients treated conservatively had shorter hospital stays after fracture. Overall, a well-supported consensus on when and how to operate remains to be established. When surgery is performed, posterior long-segment instrumentation is the most commonly performed procedure [35].

36.7.2 Kyphosis Correction

In extreme kyphosis, ankylosing spondylitis patients have trouble maintaining their sight lines on the horizon for ambulation. In addition, the compression of the abdominal cavity may cause reduced vital lung capacity and increased abdominal pressures [63]. There are three described surgical approaches in the literature: opening wedge osteotomy, polysegmental wedge osteotomy, and closing wedge osteotomy.

One of the first operations described for correcting kyphosis is the opening wedge osteotomy, traditionally performed on the lumbar spine. This process involves cutting away portions of the laminae and spinous processes and facets, creating an osteotomy of the posterior column. This posterior gap is then closed, with a concomitant lengthening of the anterior column (disc space). The anterior length of the spinal column is increased; the posterior length is slightly decreased. Because this process involves forcibly lengthening the anterior spine, it may cause vascular damage, the most devastating being tearing of the abdominal aorta that overlies the anterior spine. The outcome of these procedures is often mixed, with success in half of patients, poor outcomes in 6 %, and mixed outcomes in between [70]. Early reports have documented mortalities as high as 12 %, transient neurologic damage at 30 %, and permanent neurologic damage at 3 %. Other side effects reported include nerve root irritation, retrograde ejaculation, and deep vein thrombosis.

The next procedure to be described is polysegmental wedge osteotomy, a procedure that resembles opening wedge osteotomy spanning multiple vertebrae [71]. Small cuts are made from the posterior aspects of 4–6 adjacent vertebrae, and the posterior column is shortened and then fixed with instrumentation. In a report of 173 patents, this process resulted in fewer complications than monosegmental methods, and it was hypothesized that the strain on each individual vertebrae is reduced. However, other studies have found significant complications with the additional implants involved in polysegmental procedures. Overall, the success rate for polysegmental wedge osteotomy ranges from being similar to monosegmental opening wedge to worse [72].

In 1963, Scudese and Calabro introduced another method of closing wedge osteotomy or pedicle subtraction osteotomy. This process involves cutting a portion from the posterior vertebrae body itself. This gap is closed without lengthening the anterior column [73]. The benefit of this process over the opening wedge is that the posterior length of the spinal column is shortened while the anterior length is unchanged, decreasing the risk of vascular compromise. A potential risk is the increased bone resection around the spinal canal, posing the possible increased risk of damage to the neural elements. Success rates reported are varied but overall similar to those of opening wedge and polysegmental approaches [74].

To correct a severe chin on chest deformity, a C7–T1 osteotomy is performed to allow for large correction angles. A complete laminectomy is performed at C6 and C7 levels. Complete foraminotomies are carried out at the C7–T1 joints. The patient's neck is extended slowly and the osteotomy site is internally fixed, before placement of morselized bone graft from the osteotomy. Patients are generally placed in a halo vest with plaster jacket for 3 months [75].

Despite numerous small studies measuring post-op satisfaction, there is little evidence-based literature assessing the relative merits of various osteotomies [64]. However, because open wedge is generally thought to carry a higher risk of vascular damage than closed wedge osteotomy, the closing wedge technique has largely replaced the open wedge approach [63]. A consideration is that the open wedge (e.g., Smith-Peterson) osteotomies necessitate a mobile disc space anteriorly to facilitate the anterior column lengthening; in the absence of an open anterior disc space, a closing wedge (e.g., pedicle subtraction) osteotomy is necessary to restore lordosis, as the anterior column cannot be readily lengthened through opening of the disc space.

36.7.3 Dysphagia

Long osteophytes in advanced cervical diffuse idiopathic skeletal hyperostosis can lead to dysphagia, odynophagia, hoarseness, and airway compromise. Often, symptoms can be managed conservatively with steroids, analgesics, muscle relaxants, and diet changes [76]. Surgical cervical osteophyte resection is reserved for severe progressive cases. Prior to surgery, history, and clinical exam are crucial to determining whether the patient is a good candidate for surgery. In a study of patients who underwent cervical osteophyte removal, the mean symptom duration was 7.7 months and included dysphagia, rigidity, and sensation of foreign body. Lateral cervical X-ray and CT of the neck allow for assessment of the location and extent of osteophytes. Contrast swallow X-rays are used to assess the esophagus for compression and dysmotility and exclude for other causes of dysphagia. Fiberoptic laryngoscopic exams are used to assess for retropharyngeal bulging or hypo-motility of the vocal cords from compression by osteophytes [55].

There are three main methods described in the literature for removal of cervical osteophytes, namely, transoral, retrovascular, and prevascular transcervical approaches. The transoral approach was first described by Uppal and Wheatley and involves going through the posterior pharyngeal wall to access the vertebrae. Due to the high location of this approach, it is best for osteophytes located in C1 to C3. However, this operation is a clean but contaminated operation due to incision through the oral cavity and may carry a higher risk of infection [77].

The transcervical approaches are divided into retrovascular (posterolateral) and prevascular (anterolateral). With retrovascular (posterolateral) approach, the carotid sheath is retracted medially and the osteophytes are accessed posterior to the sheath. The thyroid vessels and omohyoid are retracted with the carotid sheath and thus are at relatively lesser risk for injury than with an anterior approach. The retrovascular approach allows for greater exposure of the prevertebral space; however, this is at the cost of greater retraction forces on the carotid sheath, resulting in more reports of carotid and sympathetic chain injuries.

The most commonly used approach today is the prevascular (anterolateral) approach; this method allows better access of lower vertebrae, especially C2–C7. The prevascular approach is further subdivided into left and right approaches. The left-sided approach is often favored because of less chance of damaging the laryngeal nerve. Ultimately, however, the location of the osteophytes themselves plays the biggest role in deciding left or right approaches. Most patients have rapid resolution of dysphagia within 2 weeks [65].

36.8 Postoperative Care

Surgical procedures in ankylosing spinal disease patients often carry high risks and long recovery times. In addition to routine post-op care, monitor patients closely for complications, discussed in the next section. Patients often necessitate postoperative intensive care unit observation and postoperative bracing for 3–6 months. For patients that underwent C7–T1 osteotomy for chin on chest deformity, halo ring fixation was placed for 3 months and cervicothoracic orthosis placed for another 3 months. Patients are followed by CT scans every 3 months to assess for fusion status [75]

36.9 Potential Complications

Complications include infection, bleeding, and neurological compromise when working near the spine. Reported complications during kyphosis correction include neurological compromise resulting in lower extremity weakness, loss of bladder control, or nerve root pain. As previously mentioned, with opening wedge osteotomy, there are rare reports of vascular compromise or aortic rupture [70]. With polysegmental osteotomy, implant complications often arise [78]. Closing wedge osteotomy carries a relatively increased risk of damage to the neural elements due to the large amounts of vertebral body bone resection near the spinal canal [74]. There are reports of pneumothorax and hematoma and a chance for secondary loss of correction [63, 64]. Additionally, there are reported cases of abdominal compartment syndrome, when increasing abdominal pressures exceed the capillary pressure to maintain perfusion, requiring emergency exploratory laparotomy [79]. In patients undergoing C7–T1 osteotomy for chin on chest deformity, the most common complication was postoperative neck pain. A majority of patients had difficulty swallowing immediately post-op, though nearly all patients improve with time. The most serious complications include quadriplegia or death usually immediately or soon after the operation [75].

In DISH patients, cervical osteophyte resection carries the risk of infection, bleeding, and parasethesias. The most common reported complication is vocal fold paralysis. Less common complications include vertebral disc prolapse, fistulas, transient aspiration, and Horner's syndrome [80]. There are case reports of asphyxiation due to neck hematoma following anterior approach neck surgery [81]. Recurrence of osteophytes causing recurrent dysphagia occurs in approximately 10–20 % of patients in 10 years [82].

References

1. Dakwar E, Reddy J, Vale FL et al (2008) A review of the pathogenesis of ankylosing spondylitis. Neurosurg Focus 24:E2
2. Belanger TA, Rowe DE (2001) Diffuse idiopathic skeletal hyperostosis: musculoskeletal manifestations. J Am Acad Orthop Surg 9:258–267
3. Leden I (1994) Did Bechterew describe the disease which is named after him? A question raised due to the centennial of his primary report. Scand J Rheumatol 23:42–45
4. Forestier J, Lagier R (1971) Ankylosing hyperostosis of the spine. Clin Orthop Relat Res 74:65–83
5. Resnick D, Shaul SR, Robins JM (1975) Diffuse idiopathic skeletal hyperostosis (DISH): Forestier's disease with extraspinal manifestations. Radiology 115: 513–524
6. Collantes E, Zarco P, Munoz E et al (2007) Disease pattern of spondyloarthropathies in Spain: description

of the first national registry (REGISPONSER) extended report. Rheumatology (Oxford) 46:1309–1315
7. Braun J, Bollow M, Remlinger G et al (1998) Prevalence of spondylarthropathies in HLA-B27 positive and negative blood donors. Arthritis Rheum 41: 58–67
8. Gran JT, Husby G, Hordvik M (1985) Prevalence of ankylosing spondylitis in males and females in a young middle-aged population of Tromso, northern Norway. Ann Rheum Dis 44:359–367
9. Carter ET, McKenna CH, Brian DD et al (1979) Epidemiology of Ankylosing spondylitis in Rochester, Minnesota, 1935–1973. Arthritis Rheum 22:365–370
10. Baum J, Ziff M (1971) The rarity of ankylosing spondylitis in the black race. Arthritis Rheum 14:12–18
11. Lawrence RC, Helmick CG, Arnett FC et al (1998) Estimates of the prevalence of arthritis and selected musculoskeletal disorders in the United States. Arthritis Rheum 41:778–799
12. Ng SC, Liao Z, Yu DT et al (2007) Epidemiology of spondyloarthritis in the People's Republic of China: review of the literature and commentary. Semin Arthritis Rheum 37:39–47
13. West HF (1949) Aetiology of Ankylosing Spondylitis. Ann Rheum Dis 8:143–148
14. Hill HF, Hill AG, Bodmer JG (1976) Clinical diagnosis of ankylosing spondylitis in women and relation to presence of HLA-B27. Ann Rheum Dis 35: 267–270
15. Helmick CG, Felson DT, Lawrence RC et al (2008) Estimates of the prevalence of arthritis and other rheumatic conditions in the United States. Part I. Arthritis Rheum 58:15–25
16. Julkunen H, Heinonen OP, Knekt P et al (1975) The epidemiology of hyperostosis of the spine together with its symptoms and related mortality in a general population. Scand J Rheumatol 4:23–27
17. Julkunen H, Knekt P, Aromaa A (1981) Spondylosis deformans and diffuse idiopathic skeletal hyperostosis (DISH) in Finland. Scand J Rheumatol 10: 193–203
18. Boachie-Adjei O, Bullough PG (1987) Incidence of ankylosing hyperostosis of the spine (Forestier's disease) at autopsy. Spine (Phila Pa 1976) 12:739–743
19. Kiss C, O'Neill TW, Mituszova M et al (2002) The prevalence of diffuse idiopathic skeletal hyperostosis in a population-based study in Hungary. Scand J Rheumatol 31:226–229
20. Holton KF, Denard PJ, Yoo JU et al (2011) Diffuse idiopathic skeletal hyperostosis and its relation to back pain among older men: the MrOS Study. Semin Arthritis Rheum 41(2):131–138
21. Spagnola AM, Bennett PH, Terasaki PI (1978) Vertebral ankylosing hyperostosis (Forestier's disease) and HLA antigens in Pima Indians. Arthritis Rheum 21:467–472
22. Pham T (2008) Pathophysiology of ankylosing spondylitis: what's new? Joint Bone Spine 75:656–660
23. Sieper J, Appel H, Braun J et al (2008) Critical appraisal of assessment of structural damage in ankylosing spondylitis: implications for treatment outcomes. Arthritis Rheum 58:649–656
24. Brewerton DA, Hart FD, Nicholls A et al (1973) Ankylosing spondylitis and HL-A 27. Lancet 1: 904–907
25. Taurog JD, Maika SD, Simmons WA et al (1993) Susceptibility to inflammatory disease in HLA-B27 transgenic rat lines correlates with the level of B27 expression. J Immunol 150:4168–4178
26. Taurog JD, Richardson JA, Croft JT et al (1994) The germfree state prevents development of gut and joint inflammatory disease in HLA-B27 transgenic rats. J Exp Med 180:2359–2364
27. Brown MA, Kennedy LG, MacGregor AJ et al (1997) Susceptibility to ankylosing spondylitis in twins: the role of genes, HLA, and the environment. Arthritis Rheum 40:1823–1828
28. Coaccioli S, Fatati G, Di Cato L et al (2000) Diffuse idiopathic skeletal hyperostosis in diabetes mellitus, impaired glucose tolerance and obesity. Panminerva Med 42:247–251
29. Mader R, Sarzi-Puttini P, Atzeni F et al (2009) Extraspinal manifestations of diffuse idiopathic skeletal hyperostosis. Rheumatology (Oxford) 48:1478–1481
30. Daragon A, Mejjad O, Czernichow P et al (1995) Vertebral hyperostosis and diabetes mellitus: a case–control study. Ann Rheum Dis 54:375–378
31. Pavelkova A, Pavelka K (2006) Diffuse idiopathic skeletal hyperostosis and its relation to metabolic parameters. Vnitr Lek 52:477–480
32. Eckertova M, Krskova K, Penesova A et al (2009) Impaired insulin secretion and uptake in patients with diffuse idiopathic skeletal hyperostosis. Endocr Regul 43:149–155
33. Halverstam CP, Zeichner J, Lebwohl M (2006) Lack of significant skeletal changes after long-term, low-dose retinoid therapy: case report and review of the literature. J Cutan Med Surg 10:291–299
34. el Miedany YM, Wassif G, el Baddini M (2000) Diffuse idiopathic skeletal hyperostosis (DISH): is it of vascular aetiology? Clin Exp Rheumatol 18: 193–200
35. Westerveld LA, Verlaan JJ, Oner FC (2009) Spinal fractures in patients with ankylosing spinal disorders: a systematic review of the literature on treatment, neurological status and complications. Eur Spine J 18:145–156
36. McVeigh CM, Cairns AP (2006) Diagnosis and management of ankylosing spondylitis. BMJ 333:581–585
37. Martin TM, Smith JR, Rosenbaum JT (2002) Anterior uveitis: current concepts of pathogenesis and interactions with the spondyloarthropathies. Curr Opin Rheumatol 14:337–341
38. Resnick D, Dwosh IL, Goergen TG et al (1976) Clinical and radiographic abnormalities in ankylosing spondylitis: a comparison of men and women. Radiology 119:293–297
39. Braunstein EM, Martel W, Moidel R (1982) Ankylosing spondylitis in men and women: a clinical and radiographic comparison. Radiology 144:91–94

40. Marks SH, Barnett M, Calin A (1983) Ankylosing spondylitis in women and men: a case–control study. J Rheumatol 10:624–628
41. Ramos-Remus C, Gomez-Vargas A, Guzman-Guzman JL et al (1995) Frequency of atlantoaxial subluxation and neurologic involvement in patients with ankylosing spondylitis. J Rheumatol 22:2120–2125
42. Bartleson JD, Cohen MD, Harrington TM et al (1983) Cauda equina syndrome secondary to long-standing ankylosing spondylitis. Ann Neurol 14:662–669
43. Mata S, Fortin PR, Fitzcharles MA et al (1997) A controlled study of diffuse idiopathic skeletal hyperostosis. Clinical features and functional status. Medicine (Baltimore) 76:104–117
44. Resnick D (1978) Diffuse idiopathic skeletal hyperostosis. AJR Am J Roentgenol 130:588–589
45. Mulleman D, Mammou S, Griffoul I et al (2005) Ossification of the posterior longitudinal ligament of the cervical spine and SAPHO syndrome. J Rheumatol 32:1361–1364
46. Papakostas K, Thakar A, Nandapalan V et al (1999) An unusual case of stridor due to osteophytes of the cervical spine: (Forestier's disease). J Laryngol Otol 113:65–67
47. Broadway JW (1994) Forestier's disease (ankylosing hyperostosis): a cause for difficult intubation. Anaesthesia 49:919–920
48. Ozkalkanli MY, Katircioglu K, Ozkalkanli DT et al (2006) Airway management of a patient with Forestier's disease. J Anesth 20:304–306
49. Kritzer RO, Rose JE (1988) Diffuse idiopathic skeletal hyperostosis presenting with thoracic outlet syndrome and dysphagia. Neurosurgery 22: 1071–1074
50. Gokce A, Beyzadeoglu T, Hanci L et al (2007) Diffuse idiopathic skeletal hyperostosis as a cause of acute respiratory distress in early postoperative period of total knee arthroplasty. Arch Orthop Trauma Surg 127:553–555
51. Schoenfeld AJ, Harris MB, McGuire KJ et al (2011) Mortality in elderly patients with hyperostotic disease of the cervical spine after fracture: an age- and sex-matched study. Spine J 11:257–264
52. Wang YF, Teng MM, Chang CY et al (2005) Imaging manifestations of spinal fractures in ankylosing spondylitis. AJNR Am J Neuroradiol 26:2067–2076
53. Koivikko MP, Kiuru MJ, Koskinen SK (2004) Multidetector computed tomography of cervical spine fractures in ankylosing spondylitis. Acta Radiol 45:751–759
54. Sangala JR, Dakwar E, Uribe J et al (2008) Nonsurgical management of ankylosing spondylitis. Neurosurg Focus 24:E5
55. Presutti L, Alicandri-Ciufelli M, Piccinini A et al (2010) Forestier disease: single-center surgical experience and brief literature review. Ann Otol Rhinol Laryngol 119:602–608
56. Sidiropoulos PI, Hatemi G, Song IH et al (2008) Evidence-based recommendations for the management of ankylosing spondylitis: systematic literature search of the 3E Initiative in Rheumatology involving a broad panel of experts and practising rheumatologists. Rheumatology (Oxford) 47:355–361
57. Song IH, Poddubnyy DA, Rudwaleit M et al (2008) Benefits and risks of ankylosing spondylitis treatment with nonsteroidal antiinflammatory drugs. Arthritis Rheum 58:929–938
58. Braun J, Sieper J (2004) Biological therapies in the spondyloarthritides–the current state. Rheumatology (Oxford) 43:1072–1084
59. Elewaut D, Matucci-Cerinic M (2009) Treatment of ankylosing spondylitis and extra-articular manifestations in everyday rheumatology practice. Rheumatology (Oxford) 48:1029–1035
60. Al-Herz A, Snip JP, Clark B et al (2008) Exercise therapy for patients with diffuse idiopathic skeletal hyperostosis. Clin Rheumatol 27:207–210
61. Caron T, Bransford R, Nguyen Q et al (2010) Spine fractures in patients with ankylosing spinal disorders. Spine (Phila Pa 1976) 35:E458–E464
62. Whang PG, Goldberg G, Lawrence JP et al (2009) The management of spinal injuries in patients with ankylosing spondylitis or diffuse idiopathic skeletal hyperostosis: a comparison of treatment methods and clinical outcomes. J Spinal Disord Tech 22:77–85
63. Kiaer T, Gehrchen M (2010) Transpedicular closed wedge osteotomy in ankylosing spondylitis: results of surgical treatment and prospective outcome analysis. Eur Spine J 19:57–64
64. Brox JI, Helle A, Sorensen R et al (2009) Functional outcome after lumbar closing wedge osteotomy in ankylosing spondylitis. Int Orthop 33:1049–1053
65. Urrutia J, Bono CM (2009) Long-term results of surgical treatment of dysphagia secondary to cervical diffuse idiopathic skeletal hyperostosis. Spine J 9:e13–e17
66. Bohlman HH (1979) Acute fractures and dislocations of the cervical spine. An analysis of three hundred hospitalized patients and review of the literature. J Bone Joint Surg Am 61:1119–1142
67. Wu CT, Lee ST (1998) Spinal epidural hematoma and ankylosing spondylitis: case report and review of the literature. J Trauma 44:558–561
68. Perez-Lopez C, Isla A, Gomez Sierra A et al (2004) Cervical epidural hematoma without fracture in a patient with ankylosing spondylitis. A case report. J Neurosurg Sci 48:91–94; discussion 4
69. Robins K, Saravanan S, Watkins EJ (2005) Ankylosing spondylitis and epidural haematoma. Anaesthesia 60: 624–625
70. Weale AE, Marsh CH, Yeoman PM (1995) Secure fixation of lumbar osteotomy. Surgical experience with 50 patients. Clin Orthop Relat Res (321):216–22
71. Wilson MJ, Turkell JH (1949) Multiple spinal wedge osteotomy; its use in a case of Marie-Strumpell spondylitis. Am J Surg 77(6):777–782
72. Hehne HJ, Zielke K, Bohm H (1990) Polysegmental lumbar osteotomies and transpedicled fixation for correction of long-curved kyphotic deformities in ankylosing spondylitis. Report on 177 cases. Clin Orthop Relat Res Sep;(258):49–55

73. Scudese VA, Calabro JJ (1963) Vertebral wedge osteotomy. Correction of rheumatoid (ankylosing) spondylitis. JAMA 186:627–631
74. Lazennec JY, Saillant G, Saidi K et al (1997) Surgery of the deformities in ankylosing spondylitis: our experience of lumbar osteotomies in 31 patients. Eur Spine J 6:222–232
75. Belanger TA, Milam RA, Roh JS et al (2005) Cervicothoracic extension osteotomy for chin-on-chest deformity in ankylosing spondylitis. J Bone Joint Surg Am 87:1732–1738
76. Umerah BC, Mukherjee BK, Ibekwe O (1981) Cervical spondylosis and dysphagia. J Laryngol Otol 95:1179–1183
77. Uppal S, Wheatley AH (1999) Transpharyngeal approach for the treatment of dysphagia due to Forestier's disease. J Laryngol Otol 113:366–368
78. van Royen BJ, de Kleuver M, Slot GH (1998) Polysegmental lumbar posterior wedge osteotomies for correction of kyphosis in ankylosing spondylitis. Eur Spine J 7:104–110
79. Sugrue PA, O'Shaughnessy BA, Nasr F et al (2009) Abdominal complications following kyphosis correction in ankylosing spondylitis. J Neurosurg Spine 10: 154–159
80. Kibel SM, Johnson PM (1987) Surgery for osteophyte-induced dysphagia. J Laryngol Otol 101:1291–1296
81. Miake G, Arizono T, Yamaguchi T et al (2009) Case of critical complication after surgery for ankylosing spinal hyperostosis. Fukuoka Igaku Zasshi 100:104–107
82. Miyamoto K, Sugiyama S, Hosoe H et al (2009) Postsurgical recurrence of osteophytes causing dysphagia in patients with diffuse idiopathic skeletal hyperostosis. Eur Spine J 18:1652–1658

37 Osteoporosis and the Aging Spine

Jacques Hacquebord and Michael J. Lee

Osteoporotic spine fractures can follow multiple different patterns, but vertebral compression fractures (VCF) are the most common to occur. VCF are the leading cause of disability and morbidity in the elderly [3, 4]. Pain can be severe, and progressive vertebral collapse with resultant kyphotic deformity can occur. Osteoporotic VCF have been shown to negatively affect quality of life, physical function, mental health, and survival, with the effects related to spinal deformity severity [5–7]. Acute pain can also be severe with the potential for chronic pain to develop. This is believed to result from (1) structural instability leading to progressive collapse, (2) spinal deformity causing altered spinal biomechanics, and (3) pseudoarthrosis of involved vertebrae. Spinal deformity, often a kyphotic deformity, may also predispose the patient to additional fractures of the spine. It has been postulated that the kyphotic deformity shifts the center of gravity anteriorly, thus increasing flexion-bending moments at the apex of the kyphosis [8]. Prevention of kyphotic deformity or correction of an existing deformity may therefore be important in preventing further fractures and resulting sequelae. Patients with a vertebral fragility fracture have a tenfold increase for a subsequent vertebral fracture [9]. The expectations for spine surgeons in addressing osteoporosis are to (1) treat direct sequelae of osteoporosis, (2) treat the resultant deformity, and (3) consider osteoporosis as it relates to spinal reconstructive surgery.

J. Hacquebord, M.D. • M.J. Lee, M.D. (✉)
Department of Sports Medicine and Orthopaedic Surgery, University of Washington Medical Center, 1959 Pacific Ave NE, 356500, Seattle, WA 98195, USA
e-mail: jihoon2000@hotmail.com

37.1 Case Example

An 85-year-old woman presents with sudden onset of back pain. She does not recall an antecedent event associated with the onset of her symptoms. She has been treated for a hip fracture in the past 2 years. On examination, she is neurologically intact without any focal deficits, but does have increased pain in the upright position. Imaging demonstrates multiple compression fractures at T12, L1, and L2 (Fig. 37.1).

37.2 Pathology/Pathophysiology

Bone is a dynamic and well-organized tissue composed of mineral, proteins, water, and cells, with exact composition dependent on patient age, health, diet, and anatomic location. At the microscopic level, bone consists of two forms: woven (primitive) and lamellar bone. Lamellar bone is highly organized with stress-oriented collagen giving it isotropic properties. In the mature skeleton, lamellar bone can be further categorized into trabecular (spongy or cancellous) or cortical (dense or compact) bone. The three main cell types in bone include osteoclasts, osteoblasts, and osteocytes. The exact pathophysiology of

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_37, © Springer-Verlag Berlin Heidelberg 2014

Fig. 37.1 Sagittal CT scan and sagittal STIR imaging demonstrating edematous changes within the T12, L1, and L4 vertebral bodies, suggestive of the acuity of these fractures

osteoporosis is a rapidly advancing field but can be divided into two different categories: osteoclast mediated and osteoblast mediated. Osteoclast-mediated osteoporosis is characterized by rapid bone loss. This is more commonly seen in postmenopausal women with women affected six times more commonly than men and affecting trabecular bone (vertebral body and distal radius). Osteoblast-mediated osteoporosis is more related to aging and an overall decrease in bone production. Women are affected twice as commonly as men. The ultimate result is a decreased rate of bone formation relative to bone destruction, contrary to osteomalacia which represents a dysregulation of bone mineralization with normal osteoid production.

37.3 Risk Factors

In the typical adult, bone mass is lost at roughly 0.5 % per year after peak bone mass is achieved in the early 30 s. The major determinants for whether or not osteoporosis occurs are dependent on peak bone mass and the rate of bone loss. Risk factors for osteoporosis can be divided

Table 37.1 Risk factors for osteoporosis

Non-modifiable
Advancing age
Female sex
Asian or Caucasian ethnicity
Menopause
Personal/family history of fracture
Disease states
Endocrine (e.g., hypercortisolism, hyperthyroidism, hypogonadism)
Chronic disease
Rheumatoid arthritis
Tumors
Modifiable
Diet – deficient calcium and vitamin D
Alcohol (>80 g/day)
Body mass index <22 kg/m^2
Smoking
Medications (e.g., glucocorticoids, immunosuppressives)
Inactivity

into non-modifiable and modifiable risk factors. Non-modifiable include advancing age, female sex, Caucasian or Asian ethnicity, rheumatoid arthritis, endocrine disorders, chronic disease, and personal history or family history in first-degree relative of fracture. Modifiable risk factors include low body weight, hormonal deficiencies, long-term use of medications affecting skeletal homeostasis (e.g., glucocorticoids, immunosuppressive medications), smoking, heavy alcohol use, inactivity, and diet deficient in calcium and vitamin D (Table 37.1).

37.4 Diagnosis

Osteoporosis is an advancing systemic disease that is often clinically silent until low energy fragility fractures occur. Fractures of the spine, along with the hips and wrists, are the most common sites with patients complaining of localized pain in the effected areas. Before becoming symptomatic, screening studies can be used to identify osteoporosis. Dual-energy x-ray absorptiometry (DEXA) is the screening study of choice and is used to calculate a DEXA score from the

Table 37.2 Indications for DEXA scan

Postmenopausal women >65 years old
Women >65 with 1 or more risk factors
Patient with fragility fracture (s)
Women on prolonged hormone replacement therapy
Osteopenia on x-rays

patient's bone mineral density. Indications for DEXA scan are listed in Table 37.2. A t-score is used to compare the patient's calculated bone mineral density to healthy same-gender young adults. For each 1 standard deviation below the mean, fracture risk increases 1.5–3-fold. A t-score of −1 to −2.5 defines osteopenia with a score of −2.5 or less positive for osteoporosis. A z-score compares bone mineral density with age-matched controls. It is important to note that DEXA values can be falsely elevated if assessed in areas of previous fractures, scoliosis, sclerosis, osteophytosis, vascular disease, or other disease processes that increase calcification.

In patients that have sustained a suspected osteoporotic spine fracture, it is important that a thorough history be obtained. These fractures occur in the setting of minimal to no trauma and are symptomatic with a deep, focal, midline spine pain. Symptoms should be primarily mechanical. It is important to know the course of the patient's symptoms and also any related symptoms or previous fractures. Past medical history, particularly systemic infection, malignancy, and tuberculosis, should be known. Patients with bladder or bowel incontinence, unexplained weight loss, night sweats, or other constitutional symptoms require more extensive investigation. Physical examination should begin with evaluation of patient's overall status and sagittal spinal balance, and should also include a thorough neurologic exam. Areas of point tenderness should also be identified, particularly over the spinous processes, as this is a hallmark of acute VCF and burst fractures. In contrast, sacral insufficiency fractures can present with pain over the sacral body, in the sacroiliac joint, or in a bandlike distribution across the low back. Laboratory studies can also be obtained. These can include sedimentation rates, C-reactive protein, protein electrophoresis, and differential blood counts to evaluate for malignancy or infection. To distinguish between osteoporosis and osteomalacia, serum calcium, serum phosphorous, alkaline phosphatase, and urinary calcium can be obtained. These lab values are typically normal for osteoporosis and abnormal in osteomalacia.

37.5 Imaging of Patients with Suspected Osteoporotic Spine Fractures Serve to Evaluate for

- Vertebral collapse
- Location and extent of lytic lesions
- Pedicular involvement
- Cortical destruction
- Epidural or foraminal stenosis
- Acuity of fracture

Imaging should begin with plain radiographs of the spine, obtained upright if possible to reveal overall sagittal and coronal spinal balance. Upright radiographs can be followed serially or compared to previous films to reveal a progressive or new vertebral collapse, endplate erosion, or changes in overall spinal balance. Magnetic resonance imaging (MRI) is useful in determining foraminal or epidural involvement. Furthermore, MRI is able to detect edema that is indicative of an acute fracture. This is well seen on sagittal MRI with short tau inversion recovery (STIR) sequences that highlight marrow edema seen in acute fractures. Indications for MRI in this setting are to determine whether or not patient's acute pain correlates with an acute fracture. Finally, MRI is also useful to evaluate for and rule out malignancy and infection as a cause of a patient's symptoms [10]. For patients that are unable to undergo MRI, computed axial tomography (CT) should be obtained.

37.6 Treatment

Early diagnosis and treatment of osteoporosis before a fragility fracture occurs is the optimal situation. This includes weight-bearing exercises and calcium and vitamin D supplementation. This does not increase total bone mass, but rather decreases bone resorption and mineralizes osteoid. Intermittent parathyroid hormone is anabolic and leads to early, dramatic increases in bone mass. Bisphosphanates, a family of medications that strongly inhibits bone resorption, have been beneficial in treating osteoporosis and decreasing rates of vertebral fractures (40–50 %) [11]. The spine surgeon, however, more commonly becomes involved in the care of the patient once the fragility vertebral fracture has already occurred. In the management of osteoporotic fractures, the goal for treatment is:

- Decreasing pain
- Early mobilization
- Preservation of spinal stability
- Prevention of neurologic compromise

Nonoperative management includes bed rest, narcotic medications for pain management, and spinal orthosis. However, these options are suboptimal. Bed rest is associated with additional bone mineral density loss. Extended bed rest also has significant other morbidities associated with it, including aspiration, pneumonia, and pressure sores. Narcotic pain medications, once again in an elderly population, has significant adverse effects that may be as functionally debilitating as the fragility fracture. The use of a spinal orthosis also has many compliance difficulties with patients. The American Academy of Orthopaedic Surgeons Clinical Practice Guideline summary for the treatment of symptomatic osteoporotic spinal compression fractures was unable to recommend these nonoperative treatment options [12].

Vertebroplasty and kyphoplasty are two minimally invasive procedures used to address VCF that were developed to address the pain and deformity. Vertebroplasty involves the percutaneous fluoroscopically guided injection of polymethylmethacrylate (PMMA) directly into a fractured vertebral body to stabilize osteoporotic VCF. Kyphoplasty differs in that it involves the percutaneous insertion of an inflatable bone tamp into a fractured vertebral body under fluoroscopic guidance. Inflation of the bone tamp elevates the endplates, restores the vertebral body height, and creates a cavity to be filled with bone void filler, most commonly PMMA. In addition to decreasing pain from acute fracture as in vertebroplasty, kyphoplasty is more likely to reduce spinal kyphosis and improve vertebral body height. Indications for both of these procedures are pain and more specifically:

- Stabilization of a painful or progressive osteoporotic and osteolytic VCF
- Painful vertebra due to metastases or multiple myeloma
- Kummell's disease
- Painful vertebral hemangioma

Contraindications, however, include fractures that neurologic compromise, burst-like fracture patterns, involvement of the posterior vertebral body wall, or morphology restricting access to the vertebral body.

37.7 Vertebroplasty

With patients placed prone and under fluoroscopic guidance, an 11–13-gauge needle is placed toward the center of the vertebral body using a transpedicular or extrapedicular approach [13]. Biopsy needles can be used to obtain samples before cement injection. PMMA is most commonly used and is mixed with barium to be viewed fluoroscopically. Once the appropriate viscosity has been obtained, between 2 and 10 mL of cement is injected. The mechanism of pain relief in vertebroplasty is unclear with hypotheses including mechanical immobilization of fracture [14] and deafferentation of fractured vertebra from the exothermic reaction of PMMA polymerization. Studies have shown 60–100 % of patients with pain reduction maintained for months up to 10 years [15, 16]. Vertebroplasty has a low-published complication rate, with most complications resulting from extravertebral cement leakage. While the large majority of cement leakage is asymptomatic, there is potential for spinal cord injury, nerve root compression, or pulmonary embolism, and these have been case reported in the literature. Extravertebral cement leakage is an inherent weakness of vertebroplasty due to necessity to inject low-viscosity cement at high pressure. Furthermore, vertebroplasty is not designed to correct spinal deformity.

37.8 Kyphoplasty

Kyphoplasty differs from vertebroplasty in that it involves the percutaneous placement and expansion of an inflatable bone tamp in the effected vertebral body. When expanded, the tamp lifts the endplate(s) thereby restoring vertebral body height and creates a void in which PMMA is injected. Because of this difference, kyphoplasty has the potential to correct spinal deformity. Inflation of the bone tamp is continued until (1) fracture is reduced, (2) maximal balloon pressure or volume is reached, and (3) cortical wall contact occurs. Unlike in vertebroplasty, the cement used in kyphoplasty is thicker and injected at a lower pressure. A multicenter trial that involved treating 2,194 fractures with kyphoplasty showed 90 % of patients reporting pain relief within the first 2 weeks of the procedure and a complication rate of 0.2 % per fracture [17]. Lieberman and associates observed significant improvements in physical function, vitality, mental health, and social function scores of the SF-36 questionnaire after kyphoplasty [18]. It is important to note that there remains significant disagreement regarding the benefits of vertebroplasty and kyphoplasty. Recently published AAOS guidelines regarding the treatment of symptomatic osteoporotic VCF were unable to recommend vertebroplasty, however did recommend kyphoplasty as an acceptable treatment modality [12] (Figs. 37.2 and 37.3).

With the aging population, reconstructive spine surgeries are ever more commonly performed in an older population. In these instances, the spine surgeon is increasingly faced with the fixation of spinal instrumentation in osteoporotic bone. It is essential to determine whether or not an osteoporotic spine can support spinal implants. A study by Soshi et al. determined that pedicle screws should be avoided in patients with a bone mineral density less than 0.3 g/cm^2 [19]. In

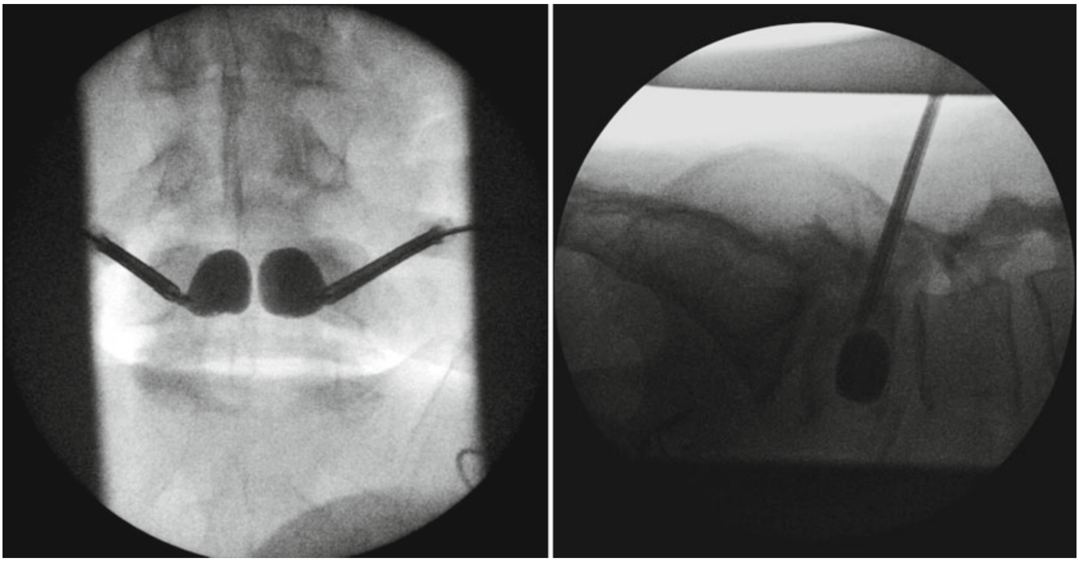


Fig. 37.2 Anterior-posterior and lateral intraoperative images of balloon inflation of L5 body during L5 kyphoplasty

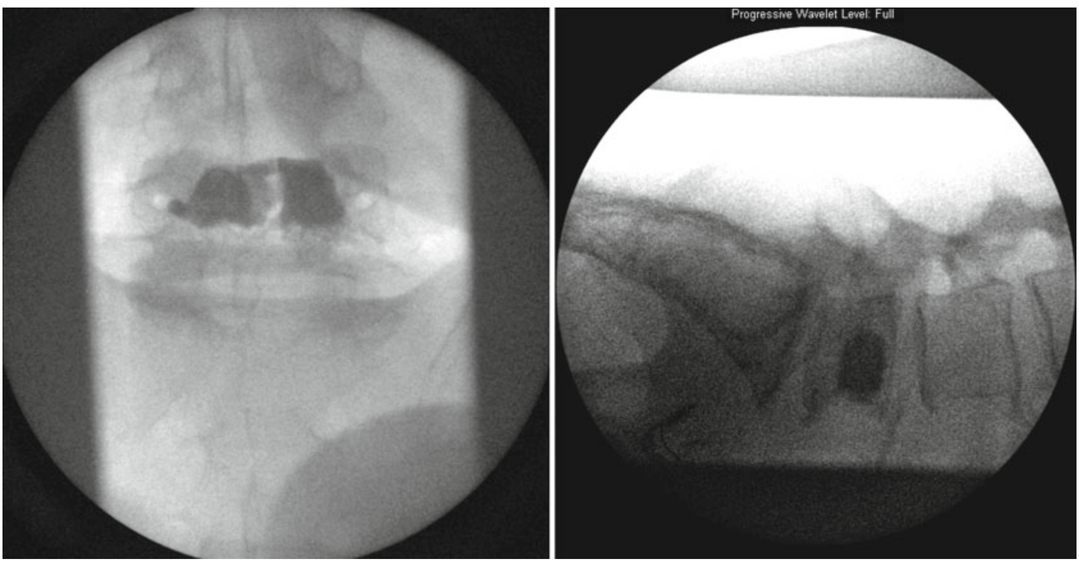


Fig. 37.3 Anterior-posterior and lateral intraoperative images after cement injection of L5 body during L5 kyphoplasty

general, patients with T scores of less than −2.5 should be approached cautiously with instrumentation. Techniques that can be employed by the spine surgeon in the face of poor bone quality include:

- Increasing screw length
- Increasing screw diameter
- Optimal screw trajectory
- Under tapping
- Increasing points of fixation

- Supplementing construct with offset sublaminar hooks
- Augmentation of screw purchase with PMMA injection through the pedicle into the vertebral body

37.9 Summary

With the aging population, today's spine surgeon must have an increasing appreciation of osteoporosis in the spine. The morbidity of vertebral fragility fractures is significant with optimal treatment modalities still needing to be further developed. Furthermore, reconstructive spine surgeries in osteoporotic bone bring many challenges that require modified techniques to address. The ultimate goal is to prevent osteoporosis from becoming symptomatic, and this effort can be supported through proper education, diet, and exercises in childhood and early adulthood to maximize peak bone mineral density. However, when this does not occur, it is essential that the spine surgeon have a thorough understanding of the pathology and the proper treatment options for his/her patient.

Questions

1. Peak bone mineral density occurs during:
   (a) Ages 0–20
   (b) Ages 21–40
   (c) Ages 41–60
   (d) Age >60
2. All of the following are risk factors for the development of osteoporosis except:
   (a) Female gender
   (b) African American ethnicity
   (c) History of steroid use
   (d) Smoking

References

1. Sambrook P, Cooper C (2006) Osteoporosis. Lancet 367(9527):2010–2018
2. Cummings SR, Melton LJ (2002) Epidemiology and outcomes of osteoporotic fractures. Lancet 359:1761–1767
3. Johnell O (1996) Advances in osteoporosis: better identification of risk factors can reduce morbidity and mortality. J Intern Med 239(4):299–304
4. Verbrugge LM, Lepkowski JM, Imanaka Y (1989) Comorbidity and its impact on disability. Milbank Q 67(3–4):450–484
5. Gold D, Lyles K (1999) Fractures: effects on quality of life. Academic, San Diego
6. Gold DT (1996) The clinical impact of vertebral fractures: quality of life in women with osteoporosis. Bone 18(3 Suppl):185S–189S
7. Silverman SL (1992) The clinical consequences of vertebral compression fracture. Bone 13(Suppl 2):S27–S31
8. White AA III, Panjabi MM, Thomas CL (1977) The clinical biomechanics of kyphotic deformities. Clin Orthop Relat Res (128):8–17
9. Lim LS, Hoeksema LJ, Serin K, ACPM Prevention Practice Committee (2009) Screening for osteoporosis in the adult U.S. population. Am J Prev Med 36(4):366–375
10. Shih TT, Huang KM, Li YW (1999) Solitary vertebral collapse: distinction between benign and malignant causes using MR patterns. J Magn Reson Imaging 9(5):635–642
11. Cranney A, Guyatt G, Griffith L et al (2002) Summary of meta-analyses of therapies for postmenopausal osteoporosis. Endocr Rev 23:570–578
12. Esses SI et al (2011) The treatment of symptomatic osteoporotic spinal compression fractures. J Am Acad Orthop Surg 19:176–182
13. Jensen ME et al (1997) Percutaneous polymethylmethacrylate vertebroplasty in the treatment of osteoporotic vertebral body compression fractures: technical aspects. AJNR Am J Neuroradiol 18(10):1897–1904
14. Belkoff SM et al (1999) An in vitro biomechanical evaluation of bone cements used in percutaneous vertebroplasty. Bone 25(2 Suppl):23S–26S
15. Gangi A, Kastler BA, Dietemann JL (1994) Percutaneous vertebroplasty guided by a combination of CT and fluoroscopy. AJNR Am J Neuroradiol 15(1):83–86
16. Mathis JM, Petri M, Naff N (1998) Percutaneous vertebroplasty treatment of steroid-induced osteoporotic compression fractures. Arthritis Rheum 41(1):171–175
17. Garfin S et al (2001) Retrospective analysis of the outcomes of balloon kyphoplasty to treat vertebral body compression fracture (VCF) refractory to medical management. Eur Spine J 10(Suppl 1):S7
18. Lieberman IH et al (2001) Initial outcome and efficacy of "kyphoplasty" in the treatment of painful osteoporotic vertebral compression fractures. Spine 26(14):1631–1638
19. Soshi S et al (1991) An experimental study on transpedicular screw fixation in relation to osteoporosis of the lumbar spine. Spine 16(11):1335–1341

Part V

Complications

Postoperative Spinal Infections

38

Michael Murray and Wellington Hsu

38.1 Introduction

With modern surgical techniques and perioperative antibiotics, spinal infection after surgery is relatively uncommon. Certain patient and surgical treatments are however at higher risk, and should be treated with extra caution. This chapter will review the prevention, diagnosis, and treatment of postoperative spinal infection.

38.2 Incidence and Risk Factors

The rate of infection following spine surgery varies considerably based on the procedure performed as well as patient factors. For example, the infection rate following a lumbar diskectomy has been reported to be 0.7 %, increasing to 1.4 % with the use of a microscope [40]. In elective instrumented cases, the reported rate of infection increases to between 2.8 and 6 % [16, 20, 22, 29, 37]. In addition to the baseline risk of infection following surgery, additional risk is conferred to patients that have a history of smoking, obesity, diabetes, long-term steroid use, alcohol abuse, prior surgical site infection, prior spinal surgery, malnourishment, and a preoperative hospitalization of >1 week [9, 21, 29, 49]. Patient age as a risk factor has been a subject of debate. Some studies have shown a higher infection rate in older patients, while others show an equal infection rate among age groups [21].

The role of diabetes in the development of postoperative infections has been investigated thoroughly. Chen et al. demonstrated a relative risk of 4.10 (95 % CI: 1.37–12.32) for developing a postoperative surgical site infection (either deep or superficial) in diabetic patients [12]. A history of diabetes makes a patient susceptible to infection due to impaired tissue microvascularity, poor antibiotic penetration [18], and immunosuppression secondary to impaired granulocyte function [44]. In addition to these underlying impairments, elevated pre- (>125 mg/dL) and postoperative (>200 mg/dL) blood glucose levels have been identified as an independent risk factor for the development of a postoperative infection [33]. To this end, tight control of perioperative blood glucose levels is critical in preventing postoperative surgical site infections in this patient population.

A history of prior surgical site infection is another risk factor that has been described by multiple investigators [1, 36, 50]. It is proposed that the previous infectious organism may reside in small quantities in the scar tissue caused by

M. Murray, M.D. (✉)
Emory Spine Center,
Department of Orthopaedic Surgery,
59 Executive Park South, Suite 3000,
Atlanta, GA 30329, USA
e-mail: mmurra5@emory.edu

W. Hsu, M.D.
Northwestern University,
Department of Orthopaedic Surgery,
676 N. St. Clair, Suite 1350, Chicago,
IL 60611, USA
e-mail: whsu@nmff.org

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_38, © Springer-Verlag Berlin Heidelberg 2014

prior procedures. The patient may have no signs or symptoms of infection despite this bacterial colonization. Consequently, the antibiotic sensitivities of the prior infectious organism should be considered when choosing a perioperative prophylactic antibiotic in patients with prior surgical site infections [36].

Surgery for spine trauma is associated with an increased rate of infection compared with those for other diagnoses. The rate of a postoperative infection following trauma has been reported to be upward of 10 % [7, 37]. The explanation for this infection rate is believed to be multifactorial. These patients are more likely to have spent time in the ICU and have poor soft-tissue envelopes secondary to significant soft-tissue trauma. They often have a greater number of comorbidities and may be more nutritionally compromised due to their trauma-induced catabolic state [23]. Finally, trauma patients with complete neurologic deficit or cognitive impairment are at an even higher risk of a postoperative infection [37, 43]. Further risk factors specific to this population include multiple levels of surgical involvement and delayed surgical treatment of over 160 h [7].

In addition to patient-specific risk factors, the operative plan and surgical technique may also influence the chances of developing a postoperative infection. Multiple studies have suggested that the anterior surgical approach is associated with a lower rate of postoperative infections when compared to the posterior approach [26, 36]. This phenomenon is hypothesized to be a result of enhanced bacterial clearance due to superior venous and lymphatic drainage of the anterior spine. Therefore, consideration should be made for an anterior surgical approach if the same surgical goals can be accomplished equally through either approach.

Intraoperative risk factors for infection have been reported to include prolonged surgical time and intraoperative blood loss in excess of 1 L [50]. Susceptibility to infection following blood loss has been hypothesized to be due to the association between significant blood loss and subsequent non-autologous blood transfusion. Non-autologous blood transfusions have been hypothesized to cause a relatively immunosuppressed state in the recipient (termed transfusion-associated immunomodulation (TRIM) [6]). The patient may then become susceptible to an increased risk of infection [4, 31, 46]. Therefore, effort should be made to minimize blood loss whenever possible and, thus, the need for non-autologous transfusions.

38.3 Etiology

Postoperative infections may arise from either direct inoculation of the wound or hematogenous seeding. Infections caused by low virulence organisms are thought to be due largely from direct inoculation. These organisms are highly susceptible to clearance by the body's immune system and rarely spread hematogenously. However, virulent organisms, such as Staphylococcus aureus, may cause infection by either route. Staphylococcus aureus has been demonstrated to be the most common causative organism in postoperative spine infections. A study by Massie [29] showed that Staphylococcus aureus was present in over 50 % of the 22 cases of postoperative spinal infections that were analyzed. Other isolated organisms included Staphylococcus epidermidis (coagulase-negative Staphylococcus), Peptococcus, Enterobacter cloacae, and Bacteroides. Gram-negative organisms are less commonly identified and are seen primarily in trauma patients, especially those with neurologic injury [37]. Polymicrobial infections may also occur but are felt to be due to direct inoculation of the wound as opposed to a hematogenous route.

38.4 Prevention

Prophylactic antibiotics have been shown to decrease infection rates in all types of spine surgery [5]. Since Staphylococcus aureus is the most common offending organism in postoperative infections, a first-generation cephalosporin such as cefazolin is generally selected as the antibiotic of choice. Cefazolin is effective against both Staphylococcus aureus and Staphylococcus epidermidis and reaches peak serum concentrations quickly. For patients allergic to penicillin, clindamycin and vancomycin are viable alternatives. If a patient is at high risk for methicillin-resistant Staphylococcus aureus (MRSA), vancomycin is frequently the prophylactic antibiotic of choice [40]. The perioperative antibiotic should be administered within 1 h of skin incision, and cefazolin should be re-dosed if the surgical duration exceeds 4 h. It is also recommended that antibiotics should also be re-dosed if the surgical blood loss exceeds 1,500 mL, based on findings of decreased tissue concentration of cefazolin in settings of blood loss that exceeds that volume [45]. Postoperatively, antibiotics should be given for 24 h after closure.

Perioperative skin preparation products are also utilized for infection prevention. For example, in foot/ankle and shoulder surgery, ChloraPrep (2 % chlorhexidine gluconate and 70 % isopropyl alcohol, Enturia, El Paso, TX, USA) was found to be superior to other cleansing products such as DuraPrep and povidone-iodine scrub in terms of reducing the cutaneous bacterial load [34]. Despite encouraging evidence for chlorhexidine as a superior operative skin preparation, there has not been any evidence to date, showing a decreased clinical infection rate when used in spine surgery.

Other surgical techniques that have been postulated to reduce infection rates but not substantiated by results include release of retractors at least every 2 h, use of antibiotic irrigation, avoidance of drains, and debridement of necrotic tissue at the end of the case. The practice of shaving the surgical site prior to surgery has been examined and suggested to actually increase the rate of postoperative surgical site infection [10].

38.5 Diagnosis

38.5.1 Clinical Presentation

Postoperative infections of the spine can be separated into superficial and deep infections. Superficial infections often present with pain, erythema, edema, warmth, and occasional drainage at the surgical site. These infections are primarily diagnosed by clinical evidence and are frequently managed medically with antibiotic therapy. However, it is essential that a deep infection is not overlooked.

Deep infections are often more challenging to diagnose. Patients often will have indolent infections which do not manifest acutely after surgery. Fevers and other systemic symptoms are often not present, and white blood cell counts may not be elevated, particularly with indolent infections. A persistently painful, draining wound that does not respond to local wound care and conservative measures is one clue to a deep infection (Fig. 38.1). However, the superficial appearance of the wound may appear benign due to the deep nature of the infection and the multilayered closure. Therefore, when pain at the surgical site is unexplained and does not decrease as expected postoperatively, an infection must be ruled out. Drainage from a seroma will be relatively clear, possibly blood tinged, and with low viscosity, whereas that from an infection will likely be more copious, viscous, and may appear purulent. Fluid aspiration and analysis has been deemed particularly useful for the detection of acute infections [43].

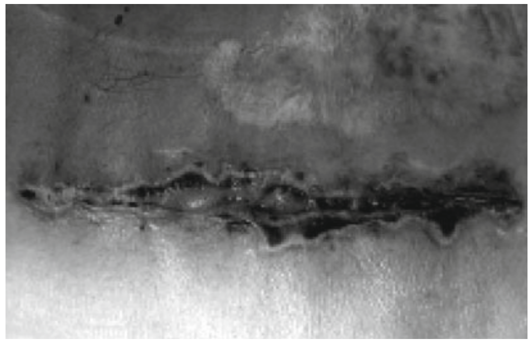


Fig. 38.1 Clinical image of a deep infection of the lumbar spine associated with underlying myonecrosis (Reprinted with permission from Sasso et al. [40])

Patients suspected of infection must be evaluated for constitutional signs of infection as well such as a temperature reading over 39° centigrade. Furthermore, other signs such as chills, sweats, malaise, lethargy, and mental status changes should be noted. These signs warrant urgent intervention as sepsis may lead to multiple organ system failure and even death.

38.5.2 Laboratory Evaluation

As mentioned above, deep infections may exist in the setting of a completely benign appearing surgical site. In these patients, the presence of unexplained increasing pain at the surgical site may be the only clinical finding. In the right clinical setting, leukocyte count, ESR, and CRP could aid the evaluation of these patients. An elevated ESR and CRP warrant increased suspicion for a postoperative infection; however, levels must be considered in respect to their expected postoperative values. ESR does not typically return to baseline values until 6 weeks after surgery, while CRP returns to baseline 2 weeks postoperatively [40].

38.5.3 Imaging

In addition to clinical and laboratory evaluation, imaging studies are often indicated and may be useful in the diagnosis of a postoperative infection. Plain radiographs should be evaluated carefully as the subtle finding of osteolysis around hardware may be suggestive of an infection. However, in the early stages of infection, the utility of plain radiographs is limited as radiographic findings lag considerably behind clinical symptoms.

A CT scan may be performed to examine the vertebral bodies as well as any possible dissolution of the end plates or disk space which may be seen with a diskitis or vertebral osteomyelitis. CT will also show increased detail of the instrumentation-bone interface. Evaluation of this interface is critical as lucency surrounding instrumentation may be indicative of a postoperative infection.

MRI is the most sensitive test for the detection of a postoperative infection; however, findings must be interpreted with caution because of the expected inflammatory response from the surgical insult. Gadolinium contrast can help to increase the sensitivity of the MRI by demonstrating rim enhancement of a large fluid collection, progressive marrow changes, and ascending epidural collections [24, 41].

While the diagnosis of a postoperative infection is challenging, adequate information can frequently be obtained through clinical evaluation, appropriate laboratory studies (including WBC, ESR, CRP), and imaging studies. However, if the laboratory studies and imaging are equivocal, close observation with repeated studies as appropriate is recommended.

38.6 Postoperative Wound Classification

Postoperative wounds may be classified by the Thalgott et al. [47] modification of the Cierny classification [13] for osteomyelitis. This staging system takes into consideration the characteristics of the infection (group 1: single organism, group 2: multiple organism, or group 3: multiple organism plus myonecrosis) as well as the clinical status of the patient (class A: healthy, normal immune system; class B: local or multiple systemic diseases, including smoking; or class C: immunocompromised or injury severity score >18). Thalgott evaluated 32 patients with postoperative infection and found 13 to be infected with a single organism (group 1), 16 with more than one organism (group 2), and two patients with multiple organism and extensive myonecrosis. The average time to diagnosis was 23 days (range 5–110 days). Most patients with single organism infections (group 1) were successfully managed with single irrigation and debridement with closure over suction drains. Patients with multiple organism infections (group 2) required an average of three irrigation and debridements. This study included two patients with group 3 infections. These were difficult to manage and had much worse outcomes than the other two groups. They required an average of six surgeries (range 4–8), and both patients required flaps for closure.

38.7 Management

38.7.1 Non-Instrumented Infections

38.7.1.1 Post-Procedural Diskitis

Percutaneous intradiskal procedures have become popular methods to both diagnose and treat disk pathology. The rate of infection following these procedures has been reported to range between 0.2 and 2.75 % [35, 42]. Clinical manifestations of infection may not be obvious, and suspicion must be heightened in patients with unexplained increasing pain following the procedure. Staphylococcus aureus is the most common causative organism, but anaerobic organisms have been isolated as well [39]. The incision site is often benign appearing, and blood cultures are generally negative. Serum markers of inflammation including CRP and ESR may be helpful, but an MRI is frequently indicated. Findings on an MRI may be nonspecific, and a biopsy is often indicated for definitive diagnosis of an infection. After diagnosis, management with culture-directed intravenous antibiotics and bracing for comfort are generally effective [28]. Surgical intervention is primarily indicated only for cases of failure of medical management and development of neurologic deficit.

38.7.1.2 Post-Diskectomy Diskitis

Patients with a postoperative disk space infection may present with the isolated finding of increasing or non-dissipating low back pain in an otherwise uneventful postoperative course. Persistent elevation of inflammatory markers (ESR, CRP) may also assist in the diagnosis. Imaging using MRI with gadolinium is often performed and may show enhancement of the disk space and adjacent bone marrow on T2 images (Fig. 38.2) and decreased signal from the disk space on T1 images. Treatment of a postoperative disk space infection involves appropriate fluid or tissue culture followed by the administration of intravenous antibiotics. Often, treatment with antibiotics alone may resolve the infection and result in an autofusion of the infected disk space. Surgical indications include failure of medical management (evidenced by progression of infection on MRI), spread of the infection into the spinal canal with abscess formation, or neurologic deficit. Surgical intervention traditionally involves debridement of the disk space from either an anterior or posterior approach. Successful management has also been reported with a posterior interbody fusion with instrumentation [25].

Fig. 38.2 A T2 weighted image of the lumbar spine demonstrating increased signal within the disk space and adjacent vertebral body. These findings are consistent with a diskitis (Reprinted with permission from Sasso et al. [40])

38.7.1.3 Postspinal Decompression

Postoperative infections following a spinal decompression often cause subfascial abscesses with or without associated disk involvement or vertebral osteomyelitis. These subfascial abscesses often do not respond adequately to antibiotics, and surgical intervention is generally indicated. Culture of infected material and debridement of infected necrotic material is recommended. Intravenous antibiotics based on culture results should be initiated. Intravenous antibiotics are maintained for at least 6 weeks, and consideration should be given to serial debridements, particularly those with multiple organism involvement, substantial myonecrosis, and in immunocompromised patients. Timely recognition and management of the infection is absolutely necessary in order to prevent spread of the infection to the disk and vertebral bodies.

38.7.2 Instrumented Infections

38.7.2.1 Post-Instrumented Fusion

Deep infections that occur after instrumentation of the spine are best managed surgically. The goals of managing this type of infection are identification of the offending organism, eradication of the infection, wound healing, and maintenance of the structural integrity of the spine and viability of the bone graft. In the operating room, deep tissue gram stain and cultures should be obtained prior to administration of intravenous antibiotics. Cultures should be maintained for at least 10 days in order to detect less virulent organisms, including Propionibacterium acnes [38].

Meticulous debridement should be performed at all layers of the wound, and all devitalized material should be excised. Pulse lavage may be used but should not be considered a substitute for meticulous excision of necrotic tissue. In subacute cases (<6 months from the time of surgery) and in the absence of a virulent infection, an attempt is made to retain instrumentation and to revise or remove only instrumentation that is found to be loose [48]. Stable hardware should be left in place in order to prevent destabilization of the spine. If loose instrumentation is identified, titanium implants should be used for the revision in the setting of infection. Titanium has been demonstrated in an animal model to be associated with a lower infection rate following a bacterial challenge when compared to steel [2]. The efficacy of titanium in the setting of revision surgery following a spinal infection is also supported in multiple clinical studies [11, 17, 27]. The next step in management depends on the results of the intraoperative gram stain. If the gram stain reveals no organism or a single organism, closure over suction drains is a viable option. With a polymicrobial infection, consideration should be made for multiple debridements until the infection has been eradicated. With extensive myonecrosis, repeat debridement at 48 and 72 h is highly recommended with repeat gram stain and cultures taken at each debridement.

Wound closure in such cases of severe soft-tissue necrosis may be accomplished by granulation tissue from secondary intention, with/without wound vacuum assistance, or coverage by local muscle rotational flap. The use of a wound vacuum has been successful in patients with exposed hardware and considerable tissue loss [3, 51]. Local muscle flaps have also been found to be effective, providing increased vascularity and soft-tissue coverage, thereby providing protection for underlying bone graft and instrumentation [15, 30]. Consideration for a plastic surgery consult should be made for wound management, particularly when substantial soft tissue is debrided [15, 30]. Broad-spectrum antibiotics should be started postoperatively until the intraoperative cultures and organism susceptibilities have returned. Culture-directed intravenous antibiotics are maintained for at least a 6-week period.

Consultation with infectious disease is often indicated for management of these difficult infections. In addition, it is important to consider the nutritional status of the patient. Laboratory markers such as transferrin and albumin can be monitored to assess the patient's nutritional state. For patients with substantial myonecrosis, intravenous hyperalimentation should be considered.

In the setting of extensive infection or in patients with late-onset of infection, antibiotics and surgical irrigation and debridement may not definitively eradicate the infection. Ho et al. demonstrated that hardware was able to be retained in 97 % of patients presenting with infection within 6 months of surgery but only 59 % of patients presenting >6 months of their index procedure [19]. Bose suggested routine removal of hardware in patients with a late presenting infection [8]. However, he further commented that this decision should be made on a case-by-case basis and that if the infection is not communicating with the hardware that an attempt can be made to retain the hardware. If retention of the hardware is attempted, but a patient fails serial debridements, one must be prepared to abandon this plan. The goal then becomes to delay removal of the instrumentation until successful spinal fusion has occurred. Intravenous antibiotic therapy may be used to suppress the infection until a fusion mass has solidified. Once the fusion mass is present, removal of instrumentation should be then performed. However, postoperatively, the spine should be monitored carefully for the increased risk of spinal deformity following removal of instrumentation [14, 32].

Conclusion

As with all spinal infections, diagnosis of a postoperative infection begins with a detailed history and physical exam, followed by appropriate blood tests and imaging. Spinal infections may be challenging as the definitive diagnosis often remains unclear after these diagnostic steps. In this case, a biopsy is indicated. Once the diagnosis has been established, treatment will vary depending on the characteristics of the infection. Variables that are considered in the treatment of all subtypes of infection are the duration of infection, organism, neurologic status, structural integrity, and maintenance of spinal alignment.

References

1. Andreshak TG, An HS, Hall J, Stein B (1997) Lumbar spine surgery in the obese patient. J Spinal Disord 10(5):376–379
2. Arens S, Schlegel U, Printzen G, Ziegler WJ, Perren SM, Hansis M (1996) Influence of materials for fixation implants on local infection. An experimental study of steel versus titanium DCP in rabbits. J Bone Joint Surg Br 78(4):647–651
3. Argenta LC, Morykwas MJ (1997) Vacuum-assisted closure: a new method for wound control and treatment: clinical experience. Ann Plast Surg 38(6): 563–576;discussion 577
4. Banbury MK, Brizzio ME, Rajeswaran J, Lytle BW, Blackstone EH (2006) Transfusion increases the risk of postoperative infection after cardiovascular surgery. J Am Coll Surg 202(1):131–138. doi:10.1016/j.jamcollsurg.2005.08.028, pii:S1072-7515(05)01430-4
5. Barker FG 2nd (2002) Efficacy of prophylactic antibiotic therapy in spinal surgery: a meta-analysis. Neurosurgery 51(2):391–400; discussion 400–391
6. Blajchman MA (2002) Immunomodulation and blood transfusion. Am J Ther 9(5):389–395
7. Blam OG, Vaccaro AR, Vanichkachorn JS, Albert TJ, Hilibrand AS, Minnich JM, Murphey SA (2003) Risk factors for surgical site infection in the patient with spinal injury. Spine (Phila Pa 1976) 28(13):1475–1480. doi:10.1097/01.BRS.0000067109.23914.0A
8. Bose B (2003) Delayed infection after instrumented spine surgery: case reports and review of the literature. Spine J 3(5):394–399, pii:S1529943003000238
9. Capen DA, Calderone RR, Green A (1996) Perioperative risk factors for wound infections after lower back fusions. Orthop Clin North Am 27(1):83–86
10. Celik SE, Kara A (2007) Does shaving the incision site increase the infection rate after spinal surgery? Spine (Phila Pa 1976) 32(15):1575–1577. doi:10.1097/BRS.0b013e318074c39f, pii:00007632-200707010-00002
11. Chang CC, Merritt K (1994) Infection at the site of implanted materials with and without preadhered bacteria. J Orthop Res 12(4):526–531. doi:10.1002/jor.1100120409
12. Chen S, Anderson MV, Cheng WK, Wongworawat MD (2009) Diabetes associated with increased surgical site infections in spinal arthrodesis. Clin Orthop Relat Res 467(7):1670–1673. doi:10.1007/s11999-009-0740-y
13. Cierny G 3rd, Mader JT, Penninck JJ (2003) A clinical staging system for adult osteomyelitis. Clin Orthop Relat Res 414:7–24. doi:10.1097/01.blo.0000088564.81746.62
14. Deckey JE, Court C, Bradford DS (2000) Loss of sagittal plane correction after removal of spinal implants. Spine (Phila Pa 1976) 25(19):2453–2460
15. Dumanian GA, Ondra SL, Liu J, Schafer MF, Chao JD (2003) Muscle flap salvage of spine wounds with soft tissue defects or infection. Spine (Phila Pa 1976) 28(11):1203–1211. doi:10.1097/01.BRS.0000067260.22943.48
16. Esses SI, Sachs BL, Dreyzin V (1993) Complications associated with the technique of pedicle screw fixation. A selected survey of ABS members. Spine (Phila Pa 1976) 18(15):2231–2238; discussion 2238–2239
17. Fayazi AH, Ludwig SC, Dabbah M, Bryan Butler R, Gelb DE (2004) Preliminary results of staged anterior debridement and reconstruction using titanium mesh cages in the treatment of thoracolumbar vertebral osteomyelitis. Spine J 4(4):388–395. doi:10.1016/j.spinee.2004.01.004, pii:S1529943004000191
18. Goodson WH 3rd, Hung TK (1977) Studies of wound healing in experimental diabetes mellitus. J Surg Res 22(3):221–227
19. Ho C, Skaggs DL, Weiss JM, Tolo VT (2007) Management of infection after instrumented posterior spine fusion in pediatric scoliosis. Spine (Phila Pa 1976) 32(24):2739–2744. doi:10.1097/BRS.0b013e31815a5a86, pii:00007632-200711150-00018
20. Hodges SD, Humphreys SC, Eck JC, Covington LA, Kurzynske NG (1998) Low postoperative infection rates with instrumented lumbar fusion. South Med J 91(12):1132–1136
21. Khan IA, Vaccaro AR, Zlotolow DA (1999) Management of vertebral diskitis and osteomyelitis. Orthopedics 22(8):758–765
22. Klein JD, Garfin SR (1996) Nutritional status in the patient with spinal infection. Orthop Clin North Am 27(1):33–36
23. Klein JD, Hey LA, Yu CS, Klein BB, Coufal FJ, Young EP, Marshall LF, Garfin SR (1996) Perioperative

nutrition and postoperative complications in patients undergoing spinal surgery. Spine (Phila Pa 1976) 21(22):2676–2682
24. Kothari NA, Pelchovitz DJ, Meyer JS (2001) Imaging of musculoskeletal infections. Radiol Clin North Am 39(4):653–671
25. Lee JS, Suh KT (2006) Posterior lumbar interbody fusion with an autogenous iliac crest bone graft in the treatment of pyogenic spondylodiscitis. J Bone Joint Surg Br 88(6):765–770. doi:10.1302/0301-620-X.88B6.17270, pii:88-B/6/765
26. Levi AD, Dickman CA, Sonntag VK (1997) Management of postoperative infections after spinal instrumentation. J Neurosurg 86(6):975–980. doi:10.3171/jns.1997.86.6.0975
27. Liljenqvist U, Lerner T, Bullmann V, Hackenberg L, Halm H, Winkelmann W (2003) Titanium cages in the surgical treatment of severe vertebral osteomyelitis. Eur Spine J 12(6):606–612. doi:10.1007/s00586-003-0614-z
28. Lindholm TS, Pylkkanen P (1982) Discitis following removal of intervertebral disc. Spine (Phila Pa 1976) 7(6):618–622
29. Massie JB, Heller JG, Abitbol JJ, McPherson D, Garfin SR (1992) Postoperative posterior spinal wound infections. Clin Orthop Relat Res 284:99–108
30. Mitra A, Harlin S (2004) Treatment of massive thoracolumbar wounds and vertebral osteomyelitis following scoliosis surgery. Plast Reconstr Surg 113(1): 206–213. doi:10.1097/01.PRS.0000097440.15013.5C
31. Murphy GJ, Reeves BC, Rogers CA, Rizvi SI, Culliford L, Angelini GD (2007) Increased mortality, postoperative morbidity, and cost after red blood cell transfusion in patients having cardiac surgery. Circulation 116(22):2544–2552. doi:10.1161/CIRCULATIONAHA.107.698977, pii:CIRCULATIONAHA.107.698977
32. Muschik M, Luck W, Schlenzka D (2004) Implant removal for late-developing infection after instrumented posterior spinal fusion for scoliosis: reinstrumentation reduces loss of correction. A retrospective analysis of 45 cases. Eur Spine J 13(7):645–651. doi:10.1007/s00586-004-0694-4
33. Olsen MA, Nepple JJ, Riew KD, Lenke LG, Bridwell KH, Mayfield J, Fraser VJ (2008) Risk factors for surgical site infection following orthopaedic spinal operations. J Bone Joint Surg Am 90(1):62–69. doi:10.2106/JBJS.F.01515, pii:90/1/62
34. Ostrander RV, Botte MJ, Brage ME (2005) Efficacy of surgical preparation solutions in foot and ankle surgery. J Bone Joint Surg Am 87(5):980–985. doi:10.2106/JBJS.D.01977, pii:87/5/980
35. Petri A, Jensen IP (1996) Postoperative lumbar discitis. Types, diagnosis and treatment. Ugeskr Laeger 158(38):5281–5285
36. Pull ter Gunne AF, Cohen DB (2009) Incidence, prevalence, and analysis of risk factors for surgical site infection following adult spinal surgery. Spine (Phila Pa 1976) 34(13):1422–1428. doi:10.1097/BRS.0b013e3181a03013, pii:00007632-200906010-00016
37. Rechtine GR, Bono PL, Cahill D, Bolesta MJ, Chrin AM (2001) Postoperative wound infection after instrumentation of thoracic and lumbar fractures. J Orthop Trauma 15(8):566–569
38. Richards BR, Emara KM (2001) Delayed infections after posterior TSRH spinal instrumentation for idiopathic scoliosis: revisited. Spine (Phila Pa 1976) 26(18):1990–1996
39. Saeed MU, Mariani P, Martin C, Smego RA Jr, Potti A, Tight R, Thiege D (2005) Anaerobic spondylodiscitis: case series and systematic review. South Med J 98(2):144–148
40. Sasso RC, Garrido BJ (2008) Postoperative spinal wound infections. J Am Acad Orthop Surg 16(6):330–337, pii:16/6/330
41. Seabold JE, Nepola JV (1999) Imaging techniques for evaluation of postoperative orthopedic infections. Q J Nucl Med 43(1):21–28
42. Silber JS, Anderson DG, Vaccaro AR, Anderson PA, McCormick P (2002) Management of postprocedural discitis. Spine J 2(4):279–287, pii:S1529943002002036
43. Sponseller PD, LaPorte DM, Hungerford MW, Eck K, Bridwell KH, Lenke LG (2000) Deep wound infections after neuromuscular scoliosis surgery: a multicenter study of risk factors and treatment outcomes. Spine (Phila Pa 1976) 25(19):2461–2466
44. Subbaiah PV, Bagdade JD (1978) Demonstration of enzymatic conversion of lysolecithin to lecithin in normal human plasma. Life Sci 22(22):1971–1977
45. Swoboda SM, Merz C, Kostuik J, Trentler B, Lipsett PA (1996) Does intraoperative blood loss affect antibiotic serum and tissue concentrations? Arch Surg 131(11):1165–1171; discussion 1171–1162
46. Tartter PI (1988) Blood transfusion and infectious complications following colorectal cancer surgery. Br J Surg 75(8):789–792
47. Thalgott JS, Cotler HB, Sasso RC, LaRocca H, Gardner V (1991) Postoperative infections in spinal implants. Classification and analysis--a multicenter study. Spine (Phila Pa 1976) 16(8):981–984
48. Weiss LE, Vaccaro AR, Scuderi G, McGuire M, Garfin SR (1997) Pseudarthrosis after postoperative wound infection in the lumbar spine. J Spinal Disord 10(6):482–487
49. Wimmer C, Gluch H (1996) Management of postoperative wound infection in posterior spinal fusion with instrumentation. J Spinal Disord 9(6):505–508
50. Wimmer C, Gluch H, Franzreb M, Ogon M (1998) Predisposing factors for infection in spine surgery: a survey of 850 spinal procedures. J Spinal Disord 11(2):124–128
51. Yuan-Innes MJ, Temple CL, Lacey MS (2001) Vacuum-assisted wound closure: a new approach to spinal wounds with exposed hardware. Spine (Phila Pa 1976) 26(3):E30–E33

Management of Dural Tears in Spinal Surgery

39

Sheeraz A. Qureshi, Steven M. Koehler, and Michael C. Gerling

39.1 Introduction

Dural tears are one of the most common complications of spinal surgery. Although unintended durotomies can occur during surgery at any level of the spine, they most frequently occur during lumbar spine surgery. The incidence of unintended durotomy varies widely throughout the literature. In a retrospective review, Cammisa et al. reported a 3.1 % overall incidence of durotomy for all level spine surgery [7]. Wang et al. reported the incidence of dural tears during lumbar spine surgery to be 14 %, with a higher rate seen in complex revision surgery as compared to primary surgery [47]. Higher rates in revision have been attributed to loss of anatomic landmarks and/or postoperative adhesions. Hannallah et al. found the overall rate of cerebrospinal fluid leak during cervical spine surgery to be 1 % [16]. And a recent review of cases submitted by members of the Scoliosis Research Society suggested a 2.2 % rate of unintended durotomy during thoracic spine surgery [49].

Deleterious consequences of unrecognized or inadequately treated dural leaks include dural-cutaneous fistulas that can lead to meningitis, arachnoiditis or epidural abscess, impaired wound healing, wound infection, pseudomeningocele formation, nerve root entrapment, and headache.

The management of unintended durotomy varies based on the location of the dural violation. For example, primary watertight closure is the most important aspect of treatment of durotomies that occur during posterior lumbar spine surgery. On the other hand, cerebrospinal fluid leaks that occur during anterior cervical spine surgery can often be treated with observation alone.

This chapter will provide an overview of the anatomy and physiology of the dura mater and cerebrospinal fluid. We will then review the relevant literature and treatment recommendations for unintended durotomies that occur during lumbar and cervical spine surgery. Finally, we will outline the current options for surgical repair of unintended durotomies.

S.A. Qureshi, M.D., MBA (✉) • S.M. Koehler, M.D.
Department of Orthopaedic Surgery,
Mount Sinai Hospital, Mount Sinai School of Medicine,
5 East 98th Street, 9th Floor, 1188,
New York, NY 10029, USA
e-mail: sheeraz.qureshi@mountsinai.org;
steven.koehler@mssm.edu

M.C. Gerling, M.D.
Assistant Professor of Orthopaedic Surgery,
Director of Spine Service,
SUNY Downstate Medical Center,
Brooklyn, NY, USA

Lutheran Medical Center,
150 55th Street, Brooklyn, NY 11220, USA
e-mail: michael.gerling@lmcmc.com

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_39, © Springer-Verlag Berlin Heidelberg 2014

39.2 Anatomy and Physiology of the Dura Mater and Cerebrospinal Fluid

Knowledge of CSF physiology is important in the management of dural tears, especially when subarachnoid drains are needed to manage a complex tear or leak.

The meninges that cover the brain and spinal cord consist of the dura, arachnoid, and pia mater. The dura mater has three distinct layers: a cellular inner layer, a fibrous middle layer, and a fibroelastic outer layer [44]. Although electron microscopic studies have found that dural fibers can run in a variety of directions, the strongest fibers are directed longitudinally with respect to the long axis of the spinal cord.

Human cerebrospinal fluid is produced in the third, fourth, and lateral ventricles of the brain by the choroid plexus. Blood plasma is ultrafiltrated by permeable capillaries of the choroid plexus and then modified by a variety of transporters and channels to create cerebrospinal fluid. Cerebrospinal fluid is formed at a rate of 0.3–0.6 mL/min or 500–600 mL daily [18]. The total amount of cerebrospinal fluid is turned over three to four times per day. The path of flow of cerebrospinal fluid starts in the lateral ventricles and then flows through the third and fourth ventricles, before entering the basal cistern. From the basal cistern, cerebrospinal fluid enters the cortical and spinal subarachnoid space.

39.3 Mechanism of Dural Tears

Most frequently, direct laceration causes dural tears during spinal surgery. Because of the fact that the tear is often detected and repaired intraoperatively, this is the most reported and studied type of tear. As introduced previously, and expanded upon below, the incidence of dural tears differs depending on the procedure and the level of spinal surgery. Universally, however, dural entry is much more common in revision procedures due to adhesion in the epidural space and dural scarring/fibrosis.

In addition to the level of spinal surgery, Sin et al. determined risk factors for dural tears in a prospective study [37]. Two factors were statistically significant: surgeon inexperience ($P=0.044$) and advanced patient age ($P=0.02$). In addition to lack of experience, the authors found that signs of aging, such as narrowing of the spinal canal, thickening of the ligamentum flavum, and osteophyte formation, contributed significantly to increased risk of tears. Interestingly, the authors also found that the primary tool involved in dural tear was the Kerrison rongeur. This was explained by the fact that the Kerrison is the most commonly used tool in decompression of the lumbar spine. Furthermore, the authors also hypothesized that shortening of the spine by degenerative processes in elderly patients may also cause redundancy of the dura, which is more easily caught in the jaws of a Kerrison rongeur.

Intraoperative mechanisms other than direct laceration of the dura include excessive nerve root traction and instrumentation. Excessive traction is easily prevented by meticulous surgical technique. Faulty screw placement has been reported to cause tears, and instrumentation can increase dead space, preventing tamponade of small dural tears by paraspinal muscles.

Occasionally, durotomies can be encountered incidentally. The classic situation in which this has been found to occur is in patients who sustain a burst fracture with a concomitant fracture of the lamina. This has been described in detail by Cammisa et al., who found that a burst fracture with an associated laminar fracture was 100 % sensitive and 74 % specific for the presence of a dural tear [6]. They theorized that vertical loading caused the pedicles to splay laterally, resulting in retropulsion of bone from the vertebral body that impinges the dural sac. When the load dissipates, the bone retracts and the dura and nerve roots remain trapped in the bony fragments. In this situation, it is important to recognize the injury pattern preoperatively so that the surgeon can be prepared intraoperatively, as there can often be nerve rootlets trapped within the laminar fracture.

Patients undergoing CT myelogram can experience a postdural puncture CSF leak. When measured by incidence of post-procedure headache, the incidence of CSF leak after CT myelogram has been found to be as high as 75 % [32]. Durotomies secondary to lumbar puncture are usually millimeters in size and contained by intact lumbar anatomy and thus can be managed by observation, hydration, and supine positioning for 24 h.

Less commonly, other mechanisms may lead to CSF leaks postoperatively. These include residual bone spikes puncturing the dural sac, breakdown of dural tissue in the setting of infection, and patient-induced increased intrathecal pressure. Patients may have coughing, violent awakening from anesthesia, or postoperative seizures that can increase the pressure in the epidural space, distending the dura and possibly disrupting any dural repair.

39.4 Lumbar Dural Tears

The incidence of unintended durotomy during lumbar spine surgery has varied widely in the literature. Wang et al. reported that 14 % of patients undergoing lumbar spine surgery for degenerative conditions sustained a dural tear, and Cammisa et al. found an overall rate of 3.1 % with a rate of 8.1 % in lumbar revision cases [7, 47]. In the largest series to date, Khan et al. reported a 7.6 % dural tear rate during primary lumbar spine surgery and 15.9 % rate during revision procedures [22]. Epstein et al. found that the three most common factors associated with unintended durotomy were ossification of the yellow ligament, synovial cysts, and prior surgery [12].

The signs and symptoms of dural tears in the lumbar spine are secondary to persistent leakage of cerebrospinal fluid. The most common findings are persistent headaches that are exacerbated by head elevation and relieved by Trendelenburg position. Photophobia or the presence of clear drainage can signify persistent leakage.

To date, studies have failed to demonstrate acceptable results using nonoperative management of unrepaired dural tears. Eismont et al. advocated several principles in the management of dural tears, the most important of which was primary repair [11]. Other recommendations included appropriate visualization by using loupe magnification or an operating microscope and abstaining from use of subfascial drains to prevent the formation of a spinocutaneous fistula.

Khan et al. used a postoperative protocol that resulted in 98.2 % success in the management of unintended durotomy after lumbar spine surgery [22]. After primary repair, patients were kept supine for 24 h. Subfascial drains were taken off suction and put to gravity the morning after surgery, and after 24 h, patients were elevated to 30° for 8 h. If no headache occurred, patients were allowed to ambulate. If patients experienced a headache with head elevation or ambulation, another 24 h of bed rest was trialed. If headaches persisted beyond 72 h, patients were taken back to the operating room for reexploration.

39.5 Cervical Dural Tears

The incidence of dural tears during cervical spine surgery is significantly less than in lumbar spine surgery. In the largest reported series, Hannallah et al. reported a rate of 1 % [16]. In their series, the most common factors associated with cervical durotomy were ossification of the posterior longitudinal ligament (OPLL), revision surgery, and patients undergoing anterior cervical corpectomy. Dural tears in patients with OPLL typically occur with resection of the ligament with a Kerrison rongeur. These patients are 13.7 times more likely to experience dural tear than patients with a normal PLL. The second most common cause of dural tear is revision cervical spine surgery.

Unintended durotomies during anterior cervical surgery can be difficult, if not impossible, to repair primarily, especially when occurring during a discectomy procedure. If the site of a leak is not suturable, a sealant is the preferred method of treatment. Unlike repairs of lumbar dural tears, an upright position postoperatively is preferred for repairs of cervical durotomies because gravity diverts fluid away from the defect, decreasing the local CSF pressure and enabling spontaneous closure.

If a primary repair is not watertight, or in the case of a failed repair, lumbar subarachnoid catheter drainage is utilized to reduce intrathecal pressure and help the durotomy site heal. The subarachnoid drain is generally left in place for 4 or 5 days, and prophylactic antibiotics are used during this time. CSF is drained at a rate of 8–12 mL/h. If there is any evidence of infection, the drain must be discontinued immediately.

39.6 Dural Repair

The most important aspect of treatment of a dural tear is prevention. Knowing that dural tears occur more frequently in revisions and the elderly should prompt the surgeon to use careful preoperative planning and meticulous surgical technique. In a revision, we recommend that the surgeon begins dissection in areas of unscarred tissue and proceed toward the areas of scar and adhesions to help prevent unintended durotomy.

Eismont et al. were among the first to report on the management of dural tears in the orthopedic literature [11]. Their recommendations included the importance of visualization. They commented on the necessity of a dry surgical field, having appropriate lighting, and using some form of magnification. They suggested the dura should be repaired primarily with suture and augmented when necessary. After completion of the repair, its integrity should be tested intraoperatively using maneuvers, such as the Valsalva, that increase intrathecal pressure. The wound should be closed in layers, and a watertight closure of the fascial layer is of utmost importance. Postoperatively, a variable amount of bed rest can be used to allow the symptoms to subside. The principles that they advocated have formed the foundation of current intraoperative and postoperative management.

Intraoperatively, an unintended durotomy is most often seen at the time it occurs. If the durotomy is not seen, there are several signs that can be suggestive of a dural violation. The presence of clear fluid in a dry field or lighter fluid within a bloodier field can indicate a dural tear. The presence of significant epidural bleeding, which occurs secondary to the loss of tamponade effect of the epidural veins by the dura, can also be indicative of dural leakage.

Although surgeons often base the necessity of a repair on the size of the tear, there is no literature to guide treatment of a durotomy based on absolute size. Dural tears can range in size from pinholes to massive defects [13]. There are even times when only the outer layer is torn and the two inner meningeal layers remain intact. In this situation, there is often no leakage of fluid. It is our recommendation to repair all violations of the dura that occur during posterior spine surgery, including those in which there is no leakage, as increased postoperative abdominal pressure will often result in bursting of the arachnoid layer and symptomatic spinal fluid leak. Other consequences of persistent dural tear include CSF fistula, pseudocyst, meningitis, nerve root entrapment, and wound complications [4].

In the cervical spine, a dural tear that is amenable to repair (such as that which occurs during an anterior corpectomy procedure or posterior cervical procedure) should be fixed, whereas one that is difficult to access should be treated through adjunctive methods which were briefly discussed previously and will be readdressed later.

39.6.1 Primary Repair

Once a dural tear has occurred, preparations must be made for repair. Depending on the location of the tear, it can be necessary to resect more bone in order to fully visualize the tear and also to accommodate the instruments necessary to repair the defect. Exposure is often the most important step in successful dural repairs. Once the field is large enough to allow for successful placement of the necessary instruments for repair, the focus should turn to obtaining adequate hemostasis. Bipolar cautery is used to control epidural bleeding. A hemostatic agent, such as a flowable sealant, can be used along with cottonoids to assist with maintenance of a dry field. It can help to pack the lateral gutters with Surgifoam® and pad the surrounding intact dura with cottonoids so that there is no leakage of blood into the area of repair. The torn area should be irrigated to remove any blood that has collected within the dural violation to reduce the risk of arachnoiditis. A dura-friendly suction device (e.g., Frazier) should be utilized, and suction should be done through a cottonoid to avoid sucking and injuring any extruded nerve rootlets.

Attention should now be turned to any nerve rootlets that have extruded from the torn area. These should be gently pushed back into the dural sac. Often, it can be necessary to keep the rootlets pushed in with an instrument such as a Penfield 3 elevator and start the suture repair around it. A cottonoid patty can also be used to help hold the rootlets within the dural sac while the repair is being performed. Additionally, use of the Trendelenburg position can assist in allowing the nerve roots to fall anteriorly and relaxing the dural edges. Whatever the method, it is critical not to catch the rootlets with the suture as this will result in trapping the rootlets within the repair and can lead to postoperative pain.

Once hemostasis has been achieved, the entire extent of the dural tear visualized, and all rootlets securely held within the dural sac, repair of the dura can commence. Special instruments, such as fine needle drivers and dural holding forceps, are often necessary and should be available. The type of suture often depends on surgeon preference. There has been no evidence of any one particular suture material or suturing technique being superior to any other. Khan et al. used 4-0 silk suture in a locking fashion without any adjuncts unless a persistent leak was present which resulted in 98.2 % successful management [22]. Our preference is to use a 5–0 Nurolon® (Ethicon, Inc.) (nylon) suture which is tightly braided and handles like silk but has been found to have more strength and elicit less of a tissue reaction than silk [10]. We recommend running the suture in a locking fashion beginning a few millimeters proximal to the cephalad edge of the tear and proceeding to below the distal end of the tear. A watertight closure of the dural defect should be the goal of any repair. Upon appropriate repair, the dura reinflates in a pulsatile manner. The integrity of the repair should be tested by performing a Valsalva maneuver.

If a watertight closure cannot be achieved by suture repair alone, adjunctive methods are available.

39.6.2 Grafts

Fat grafts are among the earliest and least expensive options for enhancing the integrity of dural repair. The benefits of using fat are that it is impermeable to water, easily harvested, and does not adhere to neural structures. Onlay techniques for fat graft have been described, as have techniques for the usage of fat in anterior dural tears during posterior lumbar surgery [2]. When using a fat graft, we generally harvest a piece of fat locally that approximates the size of the dural defect. Simple sutures are then placed at both ends of the defect which has already been approximated by suture. The simple stitches at either end are then passed through the fat graft which is slid down along the suture and onto the dura. The stitches are then tied on top of the fat graft to hold it in place. For anterior tears, fat can be used to fill the disk dead space anteriorly. Addition of another layer of fat posteriorly allows for 360° of fat enclosure.

Fascial grafts can be used when the dural defect is too large to allow direct closure without compression of the neural elements [4]. Fascia lata has been used successfully for 40 years as a dural graft but necessitates a second incision to harvest. During the Korean War, Wallace and Meirowsky used fascia lata in 198 of 590 cases of penetrating wounds of the brain requiring dural grafting [46]. Thammavaram et al. described 37 cases in which fascia lata was used for dural patching [39]. They reported no complications related to grafting. Tachibana et al. demonstrated histologically that fascia grafts tightly adhere to dura mater and display a thick fibrous tissue around the fascial graft, leading to successful tolerance of high CSF pressures [38]. A recent study demonstrated successful repair of intraoperative durotomies with fascial grafts in nine patients with OPLLs using an onlay technique [45]. Fascia lata can be used for large defects and adjacent lumbar fascia for small defects. Eismont described securing these in place with interrupted dural silk sutures [11].

Synthetic collagen grafts are also available and currently more frequently used than fat and fascia to substantiate a dural repair. Narotam et al. reported the successful use of a collagen matrix onlay sutureless graft during primary repair of dural tears, in which the onlay graft is placed over the defect and attaches via surface tension to the dura, providing a low-pressure absorptive surface to diffuse any CSF and acting as a site for biological repair [30]. The collagen's hemostatic properties initiate clot formation, resulting in an immediate seal. In their series of 110 patients with dural lacerations, microfibrillar collagen matrix was applied in 100 % of cases, combined with subfascial drains (in 82 %), fibrin glue (in 7.3 %), lumbar drains (in 2.7 %), or other adjunct, including suture (in 8 %), sealing dural leaks 95 % of the time. Options for suturable grafts and grafts not requiring suture are available depending on the preference of the operating surgeon. In addition to not requiring additional fixation, other advantages of synthetic grafts include the fact that they fully degrade and are replaced with natural collagen in approximately 3 months. Furthermore, there is no increased risk of infection, such as when using fibrin glue from pooled sources.

Because a failure rate of 5–10 % has been reported of primary dural repair, with leakage from suture holes made during the repair implicated as the main cause, a sutureless means of dural closer with grafts alone has been investigated [4, 11, 19, 36]. Although there are some studies suggesting excellent results with use of grafts alone in the treatment of dural tears, we feel the current literature is not sufficient to recommend this as a preferred method in the treatment of posterior durotomies. In dural tears that occur during open posterior lumbar and cervical surgery, we recommend a primary watertight repair with suture that can be further supported with a fat or synthetic graft. It should be noted that with the recent upsurgence of minimally invasive surgery, dural tears are occurring more frequently in locations that are difficult to repair. Further studies are needed to investigate the necessity of performing a primary suture repair in this setting, as tissue plains are better preserved with the presumed benefit of smaller dead space for CSF leakage.

39.6.3 Fibrin Glue

Fibrin glue is another adjunctive method and is composed of purified fibrinogen and thrombin made from cryoprecipitate. The two solutions are injected onto the dural defect simultaneously. The first solution contains human fibrinogen, factor XIII, fibronectin, and plasminogen from the cryoprecipitate. The second solution is a mixture of thrombin powder and calcium chloride. Injecting the two solutions together creates an immediate mechanical seal [8].

The benefits of fibrin glue are that it invokes a minimal inflammatory response and it is resorbed during the healing process; therefore, epidural scarring and fibrosis are inhibited. This is especially important when spinal reoperation may be a consideration. Moreover, Cain et al. showed that using fibrin glue to augment suture repair resulted in a sevenfold increase in bursting pressure compared to suture alone [5]. Nakajima et al. demonstrated the importance of fibrin glue for sealing the suture holes at the repair site to prevent CSF leaks [29]. However, authors unanimously agree that fibrin glue is intended to augment primary repair with suture and should not replace it [17, 36].

39.6.4 Hydrogel Sealant

Hydrogel sealant is composed of a polyethylene glycol ester solution and a trilysine amine solution that is intended as an adjunct to primary dural suture repair. It was approved in 2005 by the Food and Drug Administration [48]. The literature quotes its success rate as 98.2 %, based on intraoperative sealing in a prospective, multicenter, nonrandomized, single-arm clinical investigation that involved 111 patients [43]. Additional studies have been undertaken to validate its effectiveness and safety [9, 48]. However, because the hydrogel may swell up to 50 % of its size in any dimension, there is a contraindication to applying the hydrogel to confined bony structures where nerves are present, since neural compression may occur [42]. A study in canines demonstrated this in vivo expansion, and mass effect occurs in the first 2 weeks after insertion [20]. There have been several reported cases of postoperative cord compression associated with the mass effect of the hydrogel due to its expansion and swelling [3, 25, 40].

39.6.5 Nonpenetrating Titanium Clips

Clips provide the advantage of speed and ease of use in a small, restricted operative field. Additionally, they decrease intradural adhesions due to nonpenetration. Clip closure of the dura was first reported by Marks and Koskuba in a thoracic tumor case [26]. They found that the clips were easily manipulated in a restricted operative field. Timothy et al. reported the use of anastomotic clips to close the dura in 58 adult patients [41]. Eight of the 58 cases had additional methods of dural closure that included suturing (3 cases), fibrin glue (3 cases), fibrin glue and dural patch (1 cases), and a muscle patch (1 case). In their report, there was a 14 % incidence of CSF leakage postoperative, with 3 cases requiring reoperation. Importantly, any closure method must not interfere with any postoperative imaging that may be required. Kaufman et al. reported 27 pediatric cases in which clips were used to close durotomies of <2 cm in length [21]. Clips were augmented with adjuncts in all cases. We agree with Kaufman et al. that when a very limited durotomy is used, clips may be effectively used to close the dura without impairing subsequent imaging and care. However, there is a 20-fold difference in cost compared with suture material versus clips, precluding justification of their use in durotomies >2 cm. Additionally, clips cause significant artifact on MRI and CT imaging.

39.6.6 Subarachnoid Drain

A subarachnoid drain can be placed at the time of surgery if the dural repair is tenuous or in the postoperative period if there are signs of persistent leakage. The drain acts as a CSF shunt and is placed in a manner similar to spinal anesthesia. The CSF should drain preferentially through the catheter thereby taking pressure off of the repair or the tear. This technique may also contribute to healing of a CSF fistula as drainage may decrease the distention of the dural sac, with approximation of the dural edges, facilitating healing. Kitchel et al. described an 82 % success rate in patients that had a catheter placed for 4 days that drained at a rate of 200–300 mL/day [23]. In the event of a cervical dural tear that is not amenable to direct repair, placement of a subarachnoid drain is an acceptable substitute. When using a lumbar subarachnoid drain, we recommend using a silicone catheter and titrating the shunt to drain CSF at a rate of 10 mL/h. If this results in a positional headache, the rate is further decreased. In general, a lumbar subarachnoid catheter can be in place for approximately 5 days. The use of antibiotics while the drain is in place is based on surgeon preference, but if there are any signs of infection, the drain should be immediately removed.

39.7 Wound Drains

The use of subfascial drains in the setting of a dural repair is controversial. Eismont et al. recommended against the use of subfascial drains due to concerns of decreased pressure on the repair and the risk of forming a durocutaneous fistula [11]. Wang et al. reported no increased rate of spinocutaneous fistula formation with the use of subfascial drains as long as a definitive dural closure was performed [47]. Our practice is to use subfascial drains in posterior lumbar and cervical spine surgery and submuscular drains in anterior cervical surgery in the setting of dural repair. The drainage is inspected closely in the postoperative period to ensure no clear fluid is being pulled out.

39.8 Postoperative Management

First and foremost, a smooth reversal of anesthesia is critically important to prevent coughing and retching.

Postoperative management of a patient after dural repair involves a decision of whether or not to prescribe bed rest. A variety of protocols have been developed and often depend on surgeon preference.

For lumbar dural tears, patients are generally kept flat for 24 h, as the supine position minimizes fluid pressure at the durotomy site in the lumbar spine. After 24 h, patients are elevated to a sitting position for approximately 8 h, and if this is tolerated, they are then allowed to ambulate. If the sitting position results in symptoms such as headache, another 24 h of supine positioning is recommended. If after the second 24 h there are still symptoms of persistent dural leakage, consideration should be given to taking the patient back to the operating room for re-repair.

In dural repairs of the cervical spine, patients are kept upright in the postoperative period as this is the position that places the least amount of pressure on the repair. Antiemetics to prevent Valsalva during vomiting and adequate pain control are critical so that there is no increase in pressure on the repair that can lead to persistent or recurrent leakage of cerebrospinal fluid.

Signs of a successful repair include lack of classic CSF leak symptoms, such as positional headache, nausea, and/or photophobia, and lack of clear drainage from the incision. Symptoms of persistent CSF leak may continue for a few days postoperatively despite successful repair. Pain and nausea can be relieved with opioids, nonsteroidal anti-inflammatory drugs, and antiemetics. In the event of postdural puncture headache (PDPH), the decreased intracranial CSF with resulting venodilation can be managed with caffeine or theophylline – a methylxanthine agent that produces vasoconstriction. An epidural blood patch can also be used in some circumstances, as outlined and described below.

39.9 Missed or Inadequate Repair of a Dural Tear

The signs and symptoms of a missed or inadequate repair of a dural tear are the same as a PDPH, which include headache (that increases in severity with standing), nausea, vomiting, and dizziness. In some instances, clear fluid can be seen exiting the surgical wound or is collected within the surgical drain.

Serious sequelae of a missed tear include pseudomeningocele and myelocutaneous fistula formation, both of which could lead to a superficial or deep infection or meningitis. A CSF leak can be detected with a β-2 transferrin assay on the collected fluid. The assay has been shown to have a high sensitivity and specificity [4]. Additionally, imaging can be very helpful in the diagnosis (Fig. 39.1). MRI or CT cisternography can demonstrate CSF accumulation. However, CT cisternography is time-consuming, contraindicated in patients with intracranial mass effect, and insensitive to CSF fistulas that are not actively draining. Radionuclide cisternography is a newer and promising imaging modality that has shown a sensitivity of 84 % and specificity of 98 % in detecting CSF leaks in patients with CSF rhinorrhea [1].

In the event of a missed or inadequate repair of a dural tear, the surgeon has several options. Bed rest alone may not be sufficient [11]. If CSF leakage is confirmed, reoperation and primary repair is indicated using imaging to identify the level of CSF leak. However, prior to reoperation, the surgeon can utilize nonoperative alternatives to minimize invasiveness and avoid morbidity associated with surgery and revision procedures. Other options, previously described above, include the placement of a subarachnoid drain or the use of an epidural blood patch. 20 mL of autologous venous blood is injected in the epidural space near the CSF leak. The injected blood spreads cephalad and caudally in the epidural space and adheres to the dural defect, forming a gelatinous seal over the dura [33]. One study reported an overall success rate of 97.5 % in 118 patients, and several other reports have confirmed its efficacy and safety in patients having spinal surgery [24, 27, 28]. Fibrin glue also has the potential for use in postoperative treatment. Patel et al. described treating 6 patients with postoperative CSF leaks who were treated with percutaneous fibrin sealant [31]. Spinal fluid was aspirated under CT guidance, and a cryoprecipitate solution and a calcium chloride and thrombin solution were injected simultaneously; CT imaging confirmed the fibrin

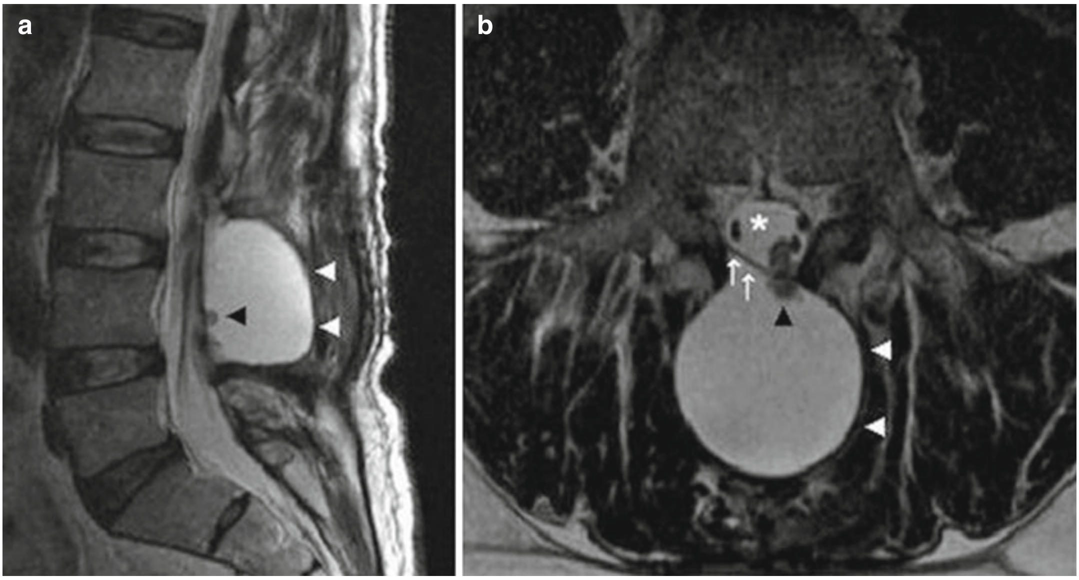


Fig. 39.1 Postoperative T2-weighted MRI of the lumbar spine in the sagittal plane (**a**) and the transversal plane (**b**) showing a large pseudomeningocele (*white arrowheads*) at L3–L4 level, containing herniated cauda equina fibers (*black arrowhead*). *White arrows* indicate dura and *white asterisk* indicates spinal canal

adherence. Since the initial description, technique refinement and additional studies have further described this nonsurgical option [14, 35].

39.10 Outcomes After Dural Tear

The literature is mixed in terms of patient outcomes following spinal surgery complicated by dural tears. Although there are studies that suggest poorer outcome following surgery complicated by a dural tear, Cammisa et al. found no long-term sequelae of unintended durotomy in a study of 2,144 patients [7, 34]. Wang et al. and Jones et al. also reported that there were no long-term effects of dural tears when appropriately repaired [19, 47].

Conclusions

Dural tears occur after puncture for CT myelography and during anterior or posterior surgery on the spine. Although prevention is always paramount, revision surgery and presence of ossified ligaments are associated with increased rate of unintended durotomy.

If a dural tear is encountered during posterior spine surgery, a direct suture repair is recommended. After direct repair is completed, the integrity of the repair should be tested by using a Valsalva maneuver. If the repair is tenuous or if suture repair is not possible due to location or tissue quality, use of grafts or fibrin glue should be considered. Lumbar subarachnoid drainage catheter can also be placed.

When an unintended durotomy occurs during anterior spine surgery, a repair should be attempted if possible. If a direct suture repair is not possible, primary use of grafts or fibrin glue is recommended.

Postoperatively, patients with lumbar dural tears should be kept supine for 24 h, and patients with cervical dural tears should be kept upright, followed by a return to normal activity. The protocol is repeated once or twice thereafter if symptoms persist. If a patient continues to have symptoms thereafter, a subarachnoid drain or blood patch can be placed or the patient is taken back to the operating room to evaluate the repair.

Although dural tears are the second most cited cause of malpractice suits in spinal surgery, most of the literature indicates that proper treatment should lead to no long-term sequelae or worsened outcomes [15].

References

1. Aydin K, Terzibasioglu E, Sencer S, Suoglu Y, Karasu A, Kiris T, Turantan MI (2008) Localization of cerebrospinal fluid leaks by gadolinium-enhanced magnetic resonance cisternography: a 5-year single-center experience. Neurosurgery 62:584–589
2. Black P (2002) Cerebrospinal fluid leaks following spinal surgery: use of fat grafts for prevention and repair. J Neurosurg 96:250–252
3. Blackburn SL, Smyth MD (2007) Hydrogel-induced cervicomedullary compression after posterior fossa decompression for Chiari malformation. J Neurosurg 106:302–304
4. Bosacco SJ, Gardner MJ, Guille JT (2001) Evaluation and treatment of dural tears in lumbar spine surgery: a review. Clin Orthop Relat Res 389:238–247
5. Cain JE Jr, Dryer RF, Barton BR (1988) Evaluation of dural closure techniques: suture methods, fibrin adhesive sealant, and cyanoacrylate polymer. Spine 13: 720–725
6. Cammisa FP Jr, Eismont FJ, Green BA (1989) Dural laceration occurring with burst fractures and associated laminar fractures. J Bone Joint Surg Am 71: 1044–1052
7. Cammisa FP Jr, Girardi FP, Sangani PK, Parvataneni HK, Cadag S, Sandhu HS (2000) Incidental durotomy in spine surgery. Spine 25:2663–2667
8. Chen MN, Kang JD (2009) Cerebrospinal fluid leaks in anterior and posterior cervical spine surgery. Semin Spine Surg 21:161–166
9. Chin CJ, Kus L, Rotenberg BW (2010) Use of duraseal in repair of cerebrospinal fluid leaks. J Otolaryngol Head Neck Surg 39:594–599
10. Chu CC (1981) Mechanical properties of suture materials: an important characterization. Ann Surg 193: 365–371
11. Eismont FJ, Weisel SW, Rothman RH (1981) Treatment of dural tears associated with spinal surgery. J Bone Joint Surg Am 63:1132–1136
12. Epstein N (2007) The frequency and etiology of intraoperative dural tears in 110 predominantly geriatric patients undergoing multilevel laminectomy with noninstrumented fusions. J Spinal Disord Tech 20: 380–386
13. Espiritu M, Rhyne A, Darden B (2010) Dural tears in spine surgery. J Am Acad Orthop Surg 18:537–545
14. Gladstone JP, Nelson K, Patel N, Dodick DW (2005) Spontaneous CSF leak treated with percutaneous CT-guided fibrin glue. Neurology 64:1818–1819
15. Goodkin R, Laska LL (1995) Unintended "incidental" durotomy during surgery of the lumbar spine: medicolegal implications. Surg Neurol 43:4–12
16. Hannallah D, Lee J, Khan M, Donaldson WF, Kang JD (2008) Cerebrospinal fluid leaks following cervical spine surgery. J Bone Joint Surg Am 90:1101–1105
17. Hodges SD, Humphreys C, Eck JC et al (1999) Management of incidental durotomy without mandatory bed rest. Spine 24:2062–2064
18. Johanson CE, Duncan JA III, Kinge PM, Brinker T, Stopa EG, Silverberg GD (2008) Multiplicity of cerebrospinal fluid functions: New challenges in health and disease. Cerebrospinal Fluid Res 5:10
19. Jones AA, Stambough JL, Balderston RA, Rothman RH, Booth RE Jr (1989) Long-term results of lumbar spine surgery complicated by unintended incidental durotomy. Spine 14:443–446
20. Kacher DF, Frerichs K, Pettit J, Campbell PK, Meunch T, Norbash AM (2006) DuraSeal magnetic resonance and computed tomography imaging: evaluation in a canine craniotomy model. Neurosurgery 58:ONS140–ONS147
21. Kaufman BA, Matthews AE, Zwienenberg-Lee M, Lew SM (2010) Spinal dural closure with nonpenetrating titanium clips in pediatric neurosurgery. J Neurosurg Pediatr 6:359–363
22. Khan MH, Rihn J, Steele G, Davis R, Donaldson WF 3rd, Kang JD, Lee JY (2006) Postoperative management protocol for incidental dural tears during degenerative lumbar spine surgery. Spine 31:2609–2613
23. Kitchel SH, Eismont EJ, Green BA (1989) Closed subarachnoid drainage for management of cerebrospinal fluid leakage after an operation on the spine. J Bone Joint Surg Am 71:984–987
24. Lauer KK, Haddox JD (1992) Epidural blood patch as treatment for surgical durocutaneous fistula. J Clin Anesth 4:45–47
25. Lee G, Lee CK, Bynevelt M (2010) DuraSeal-hematoma: concealed hematoma causing spinal cord compression. Spine 35:E1522–E1524
26. Marks P, Koskuba D (2000) Use of a non-penetrating staple device for spinal dural closure. Br J Neurosurg 14:468
27. Maycock NF, van Essen J, Pfitzner J (1994) Post-laminectomy cerebrospinal fluid fistula treated with epidural blood patch. Spine 19:2223–2225
28. McCormack BM, Taylor SL, Heath S, Scanlon J (1996) Pseudomeningocele/CSF fistula in a patient with lumbar spinal implants treated with epidural blood patch and a brief course of subarachnoid drainage. Spine 21:2273–2276
29. Nakajima S, Fukuda T, Hasue M, Sengoku Y, Haraoka J, Uchida T (2001) New technique for application of fibrin sealant: rubbing method devised to prevent cerebrospinal fluid leakage from dura mater sites repaired with expanded polytetrafluoroethylene surgical membranes. Neurosurgery 49:117–123
30. Narotam PK, Jose S, Nathoo N, Taylon C, Vora Y (2004) Collagen matrix (DuraGen) in dural repair: analysis of a new modified technique. Spine 29:2861–2869

31. Patel MR, Louie W, Rachlin J (1996) Postoperative cerebrospinal fluid leaks of the lumbosacral spine: management with percutaneous fibrin glue. AJNR Am J Neuroradiol 17:495–500
32. Peterman SB (1996) Postmyelography headache: a review. Radiology 200:765–770
33. Rupp SM, Wilson CB (1989) Treatment of spontaneous cerebrospinal fluid leak with epidural blood patch. J Neurosurg 70:808–810
34. Saxler G, Kramer J, Barden B, Kurt A, Pfoertner J, Bernsmann K (2005) The long-term clinical sequelae of incidental durotomy in lumbar disc surgery. Spine 30:2298–2302
35. Schievink WI, Maya M, Moser FM (2004) Treatment of spontaneous intracranial hypotension with percutaneous placement of fibrin sealant. J Neurosurg 100:1098–1100
36. Shaffrey CI, Spotnitz WD, Shaffrey ME et al (1990) Neurosurgical applications of fibrin glue: augmentation of dural closure in 134 patients. Neurosurgery 26:207–210
37. Sin AH, Caldito G, Smith D, Rashidi M, Willis B, Nanda A (2006) Predictive factors for dural tear and cerebrospinal fluid leakage in patients undergoing lumbar surgery. J Neurosurg Spine 5:224–227
38. Tachibana E, Saito K, Fukuta K, Yoshida J (2002) Evaluation of the healing process after dural reconstruction achieved using a free fascial graft. J Neurosurg 96:280–286
39. Thammavaram KV, Benzel EC, Kesterson L (1990) Fascia lata graft as a dural substitute in neurosurgery. South Med J 83:634–636
40. Thavarajah D, De Lacy P, Hussain R, Redfern RM (2009) Postoperative cervical cord compression induced by hydrogel (DuraSeal). Spine 35:E25–E26
41. Timothy J, Hanna SJ, Furtado N, Shanmuganathan M, Tyagi A (2007) The use of titanium non-penetrating clips to close the spinal dura. Br J Neurosurg 21:268–271
42. U.S. Food and Drug Administration (2005) Instructions for use of DruaSeal. http://www.fda.gov/ohrms/dockets/ac/04/briefing/2004-4083b1_04_IFU%20PMA%20P040034.pdf. Accessed 3 Aug 2011
43. U.S. Food and Drug Administration (2005) Summary of safety and effectiveness data: DuraSeal dural sealant system. http://www.accessdata.fda.gov/cdrh_docs/pdf4/P040034b.pdf. Accessed 3 Aug 2011
44. Vandenabeele F, Creemers J, Lambrichts I (1996) Ultrastructure of the human spinal arachnoid mater and dura mater. J Anat 189:417–430
45. Vivek J, Kumar GSS, Rajshekhar V (2009) Cerebrospinal fluid leak during cervical corpectomy for ossified posterior longitudinal ligament. Spine 34:491–494
46. Wallace PB, Meirowsky AM (1960) The repair of dural defects by graft, an analysis of 540 penetrating wounds of the brain incurred in the Korean War. Ann Surg 151:174–180
47. Wang JC, Bohlman HH, Riew KD (1998) Dural tears secondary to operations on the lumbar spine. Management and results after a Two-year-minimum follow-up of eighty-eight patients. J Bone Joint Surg Am 80:1728–1732
48. Weinstein JS, Liu KC, Delashaw JB Jr, Burchiel KJ, van Loveren HR, Val FL, Agazzi S, Greenberg MS, Smith DA, Tew J Jr (2010) The safety and effectiveness of a dural sealant system for use with nonautologous duraplasty materials. J Neurosurg 112:428–433
49. Williams BJ, Sansur CA, Smith JS, Berven SH, Broadstone PA, Choma TJ, Goytan MJ, Noordeen HH, Knapp DR Jr, Hart RA, Zeller RD, Donaldson WF 3rd, Polly DW Jr, Perra JH, Boachie-Adjei O, Shaffrey CI (2011) Incidence of unintended durotomy in spine surgery based on 108478 cases. Neurosurgery 68:117–124

40 Complications: Neurological Injury

Shannon Hann, Nelson Saldua, and James S. Harrop

40.1 Case Presentation

A 42-year-old morbidly obese man presented with 1-week history of severe mid-thoracic back pain and worsening bilateral lower extremity weakness to the point of not moving his lower extremities. Due to the patient's body habitus, he required an open MRI (x-ray and CT of spine were unremarkable), which showed a hemorrhaged cavernoma in the T7 level of the spinal cord (Fig. 40.1a). His examination showed he was only able to move his bilateral toes (EHL3/5) with all other lower extremity muscle groups 0/5. The patient had no bladder and bowel dysfunction. He was taken to the operating room for a decompressive laminectomy and intraparenchymal resection of the cavernoma. Postoperatively, his back pain improved with isolated EHL function of 3/5 in his lower extremities. However, on postoperative day 1, his back pain increased and his EHL function deteriorated to 0/5. Emergent CT of the thoracic and lumbar spine showed a large hematoma (arrow) at the postoperative site extending into the canal space (Fig. 40.1b), even though a drain was in place. Patient was emergently taken back to the operating room for evacuation of this epidural hematoma. Postoperatively, the patient regained his bilateral toes mobility and was discharged to a rehabilitation facility.

A 69-year-old man presented with neurogenic claudication when standing or walking with resolution of symptoms upon sitting or lumbar flexion. An MRI showed a grade I spondylolisthesis of L4 over L5 causing lumbar stenosis at this level as well as stenosis at the level above (L3–4) (Fig. 40.2a). Standing flexion extension of lumbar x-ray showed 5 mm of translation. There was no evidence of a pars defect which was confirmed by CT of the lumbar spine. The patient was taken to the OR for L3–4 and L4–5 posterior lumbar interbody fusion. Postoperatively, he was found to have a new left-sided foot drop. Lumbar spine x-ray and CT were obtained which showed the left L5 pedicle screw with a medial trajectory (Fig. 40.2b). He was taken back to the OR on the same day for revision of the screw. His foot drop resolved after screw revision/removal but he continued to have a degree of neuropathic pain.

A 47-year-old electrician presented with progressive neurological decline with weakness, paresthesia, and neuropathic pain in his hands after a motorcycle collision 6 months prior. Imaging showed cervical cord compression and stenosis from C3–6 level (Fig. 40.3a). On exam, the bilateral upper extremities were weak with

S. Hann, M.D.
Department Neurousrgery, Thomas Jefferson University Hospital, Philadelphia, PA, USA

N. Saldua, M.D.
Department of Orthopaedic Surgery, Naval Medical Center San Diego, 34800 Bob Wilson Drive, suite 112, San Diego, CA 92134, USA

J.S. Harrop, M.D. (✉)
Department of Neurosurgery, Jefferson Medical College, 909 Walnut Street, Second Floor, Philadelphia, PA 19107, USA
e-mail: james.harrop@jefferson.edu

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_40, © Springer-Verlag Berlin Heidelberg 2014

Fig. 40.1 (**a**) Sagittal MRI of T-spine demonstrating hemorrhaged cavernoma at T7 level (*arrow*). (**b**) Sagittal CT of T-spine demonstrating hematoma at post-laminectomy site compressing spinal cord (*arrow*)

4/5 strength proximally and 4/5 distally. In addition, there was decreased sensation bilaterally in a C5–C7 dermatomal distribution and diminished sensation to temperature. This patient underwent a C3–7 laminectomy and fusion as well as bilateral C4–5 foraminotomy. On postoperative day 1, he developed a delayed C5 palsy on the left with deltoid strength of 2/5 and biceps strength of 4/5 but no change in sensation. An MRI of the C-spine taken at the time showed excellent decompression (Fig. 40.3b). However, on postoperative day 5, he developed new right side weakness consistent with bilateral delayed C5 palsies. An MRI at this time again showed no mass effect on the spinal cord or foramen (Fig. 40.3c). He had difficulty elevating his arms and was seen by physical and occupational therapy. A sling was applied to his left arm when he was out of bed ambulating. Patient was discharged to attend 6 weeks of outpatient rehabilitation. During this period, his bilateral C5 palsy gradually resolved.

Fig. 40.2 (**a**) Sagittal MRI of L-spine showing grade I spondylolisthesis of L4 over L5. (**b**) Postoperative axial CT of L-spine showing medially placed left screw at L5 pedicle

40.2 Pathology

The pathology of neurological damage from spine surgery can be categorized by the timing (preoperative, intraoperative, or postoperative), the location (cervical, thoracic, and lumbar), and the mechanism (positioning, traction, direct mechanical, or ischemic). This section will discuss these common causes of iatrogenic neurological injury based on the above three referenced cases.

Case 1: Transient SSEP (Somatosensory Evoked Potential) Change Due to Intraoperative Hypotension-Induced and Postoperative Spinal Epidural Hematoma

There are multiple etiologies of intraoperative neurologic losses and neuromonitoring alerts. One cause may be spinal cord hypoperfusion due either to direct blood supply interruption or hypotension. This appears to be much more common in the thoracic spinal cord region than in the cervical or lumbar spinal cord [1]. This is because

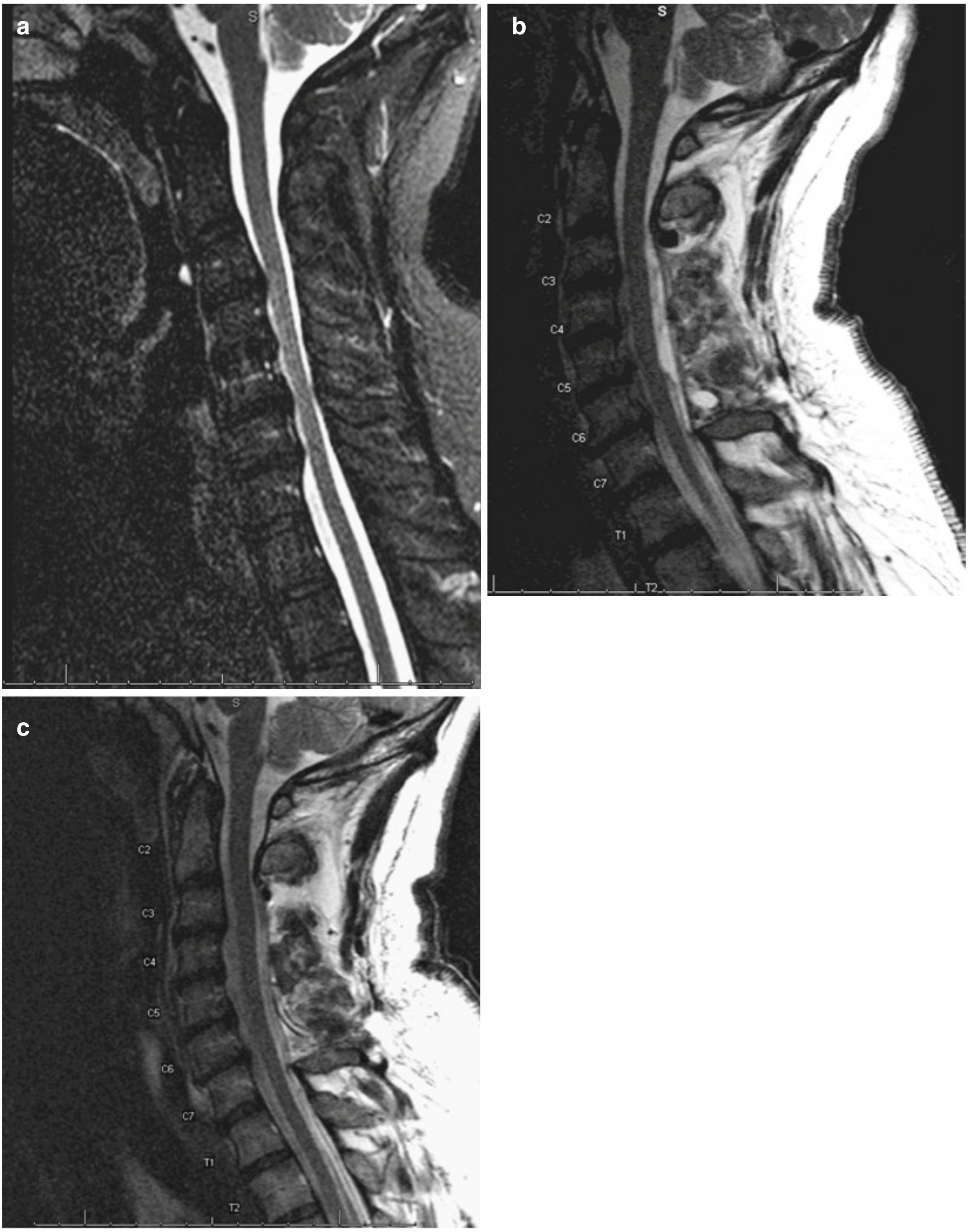


Fig. 40.3 (**a**) Sagittal MRI of C-spine showing stenosis of C3–6 level with cord compression. (**b**) POD1 C-spine MRI after developing left C5 palsy demonstrating well-decompressed spinal cord. (**c**) POD5 C-spine MRI after right C5 palsy development again shows excellent decompression

the mid-thoracic area has decreased vascular supply due to a lack of contribution from radiculomedullary arteries as well as the artery of Adamkiewicz which normally arises below T9 [1, 2]. This creates a "watershed" region of the spinal cord where blood flow is lowest.

Diminution of blood supply to this variable area may lead to spinal cord ischemia and infarction. The risk increases significantly in the presence of preoperative hypertension, hypocapnia, anemia, rapid blood loss, or a sudden drop in the blood pressure [1, 3]. Most importantly, neuromonitoring changes should not be ignored. Rather, sources should be sought and treated such as increasing the blood pressure, reposition of the patient, releasing distraction or correction, and looking for any compression.

Postoperatively, any new neurologic deficit questions the presence of a spinal epidural hematoma. If symptomatic, the patient may require urgent evacuation of the hematoma as soon as the diagnosis is established or suspected. The mechanism is thought to be bleeding from disrupted epidural veins or arteries diseased hypervascular epidural soft tissue, or fractured bone, which leads to an expanding hematoma confined within a rigid spinal canal [1, 3]. Symptomatic postoperative subdural hematoma has been commonly attributed to multilevel procedures, preoperative coagulopathy, preoperative NSAID use, large intraoperative blood loss volumes, advanced age, Rh-positive blood types, intraoperative hemoglobin values <10 g/dl, and elevated INR values [4–6]. Clinically, the hematoma follows a rapid course, manifesting as an incomplete cord injury with rapidly deteriorating neurological status. Common signs typically begin with increased pain, more typical for the procedure followed by weakness and numbness within several hours after injury as a result of external compression from the enlarging hematoma [2, 6]. In rare cases, cauda equina symptoms have been reported with epidural hematoma in the lumbar spine [7].

Case 2: Lumbar Spine Instrumentation-Induced Foot Drop

Some neurological injuries are unique to the lumbar spine, and special caution to these hazard areas is warranted. Possibilities include nerve root injury from a pedicle screw, excessive nerve root retraction, and neural injury due to malpositioned interbody devices. The use of pedicle screw fixation as adjuncts for lumbar degenerative disease has increased significantly in the past decade, and the incidence of neurologic injury from misplaced screws has been reported to range from 0 to 12 % [8]. Optimal placement involves a thorough knowledge of pedicular anatomy, specifically the angle, trajectory, and diameter of the pedicle. When placed too medially or into foramen, it can cause direct mechanical damage to nerve roots or cord causing reversible or irreversible damage despite emergent reoperation to remove the screw [8, 9]. Another potential mechanism of neurological compromise from lumbar interbody fusions arises from a malpositioned graft. A graft that is in the canal space may cause cord compression or cauda equina syndrome [3, 10]. Conventionally, anatomic landmarks are determined by plain x-ray or fluoroscopy during operation to aid in placement of pedicle screws. Intraoperative evoked potentials (SSEP or MEP) are also commonly used to enable surgeons to determine the accuracy of pedicle screw placement.

Case 3: Postdecompressive C5 Nerve Palsy

Postdecompressive C5 palsy is typically considered to be a result of nerve root injury or segmental spinal cord disorder and has been more frequently reported after the posterior approach to the cervical spine [11–14]. There are five proposed mechanisms for its cause:

1. Nerve root traction caused by posterior shifting of the cord following decompression surgery.
   Many have presumed that tethering of the nerve root might cause a C5 palsy as a result of a posterior shift of the spinal cord in association with anchoring of the nerve root at the edge of the superior facet [10, 15, 16]. The C5 root is particularly susceptible because the facet joint at C4–5 protrudes more, the C5 root is shorter, and the C5 root is usually at the apex of the decompression area resulting in greater posterior shifting of the cord at the C5 level [13, 14].
2. Spinal cord ischemia due to decreased blood supply from radicular arteries
   This is supported by the fact that C5 palsy generally has a good prognosis for a functional recovery [17]. Patients with cervical spondylotic myelopathy, as in Case 1, may have impairment of anterior spinal cord circulation due to

compression from ventral osteophytes or OPLL [10]. In this setting of already compromised cord perfusion, intraoperative drops in blood pressure can have catastrophic neurological consequences. Hypotension can occur during surgery for a number of reasons, but it is most commonly encountered during posterior cervical operations as the patient is positioned prone and put into reverse Trendelenburg [10, 15, 16].

3. Segmental spinal cord disorder
   Some researchers have postulated that C5 palsy might be associated with pathology of the gray matter of the cord because high intensity areas in the spinal cord on postoperative T2-weighted MRI were found to be present and expanded after surgery on C3–4 and C4–5 in patients with C5 palsy. However, it should be noted that C5 palsy does not always develop in patients whose preoperative T2 MRI shows high intensity areas at the C5 segment [14, 18].
4. Reperfusion injury of the spinal cord
   Chiba et al. found that high signal on postoperative T2 MRI appeared significantly more frequently in patients with postoperative paresis of the arm than in patients without [9]. Furthermore, affected muscles corresponded to segments containing high intensity regions on T2-weighted images. They proposed that postdecompressive C5 paresis might be caused by deterioration of gray matter and that local reperfusion in the spinal cord could be the mechanism [9, 19, 20].
5. Inadvertent injury to the nerve root during surgery from kinking of the nerve root at the lateral part of the residual OPLL [11–13]. Displacement of the bone graft into the neural foramen can cause compression of the nerve root within it [11]. In the case of laminoplasty, the downward displacement of the elevated lamina into the spinal canal might injure the nerve root [12, 17].

40.3 Physical Exam Findings

A relevant neurological examination should be performed on postoperative patients as soon as possible. If a patient wakes up with a new deficit, several questions must be addressed. Is the deficit partial or complete? Spinal cord or nerve root level? What is the etiology? Are there any imaging studies indicated for determining the cause? Can it be corrected with reoperation? Thorough and timely postoperative checks and urgent diagnosis of the cause are the key to correcting further neurological insults. Especially high index of suspicion for spinal epidural hematoma is needed, and any new neurologic deficit should prompt rapid evaluation of this diagnosis [3, 10].

40.4 Differential Diagnosis

- Brachial/lumbosacral plexus injury
- Nerve root trauma or traction
- Injury from hypoperfusion (ischemic)
- Spinal cord injury (traumatic)
- Spinal epidural hematoma at surgical site
- Neck hematoma
- Lingering effect of anesthesia [21]

40.5 Investigations

In a patient with a new post-op deficit, postoperative spinal radiographs should be obtained to assure there is no gross malpositioning of instrumentation or disruption of spinal column alignment. If there is any doubt, a CT scan can confirm that all instrumentation is located on the correct position. There must be a high index of suspicion for the diagnosis of a spinal epidural hematoma. After postoperative radiographs have ruled out graft or hardware malposition or change in alignment, MRI is the diagnostic test of choice. Postoperative MRI is helpful in delineating abnormal cord signal and soft tissue edema that may not be apparent on another mode of imaging. If the patient has contraindication for MRI, CT/myelography may be indicated to further confirm the screw purchase or detecting bony abnormalities [10].

40.6 Nonsurgical Treatment

If the patient wakes up with a partial deficit, post-operative x-rays are indicated to evaluate spinal alignment and hardware, and possibly followed by an MRI or CT/myelogram to rule out spinal cord pathology. If there is no hematoma or disk or structural disorder, it is likely that the treatment will be nonoperative, such as blood pressure support and possible corticosteroids [9, 21]. A good example of this is the treatment of post-operative C5 palsy. Usually there is no indication for surgical intervention for post-op C5 palsy. It was reported that rigid external fixation has been effective for the treatment of C5 palsy after cervical laminectomy [11, 14]. It is also common to be treated medically with steroids [21, 22], although the benefit of this medication is not clear. In Case 1, the patient was prescribed physical therapy to include cervical traction, muscle strengthening exercises, low frequency wave therapy, and range of motion exercises of the shoulder joint in order to prevent contracture [3].

40.7 Surgical Emergencies

If a patient wakes up with a significant injury and has undergone a posterior decompression only, rapid investigation to detect possible epidural hematoma or a retained disk fragment is required. Both of which may be reversible. Immediate return to the operating room to treat those causes following appropriate imaging is necessary. If a retained disk fragment is found, it should be removed and the disk space re explored. If, as in Case 2, an epidural hematoma is discovered, it should be evaluated and all bleeding sites identified. Further exploration of whether the hematoma has spread beyond the space of the original surgery is also crucial [9].

If a patient wakes up with a significant deficit and has undergone bone grafting or fixation, such as in Case 2, immediate plain radiographs are useful for assessing the status of the bone graft and hardware. If there is an obvious problem, the patient should be taken to the OR to explore the surgical site in hopes of correcting the misplacement [10]. Depending on the type of surgery, further imaging studies may be required. Returning to OR and ruling out hematoma by wound exploration before any imaging is another option if the neurological deterioration is pressing.

If the patient wakes up with a normal neurological exam and then has a progressive neurological deficit as in Case 1, the most likely cause is an expanding hematoma.

40.8 Considerations to Avoid Complications

40.8.1 Role of Intraoperative Spinal Cord Monitoring

In recent years, intraoperative spinal cord monitoring such as motor evoked potentials (MEPs), either alone or in combination with somatosensory evoked potentials (SSEPS), has been used extensively as a means of reducing intraoperative spinal cord injury [1].

Initially, the intraoperative monitoring was attempted by the Stagnara wake-up test, which involved waking the patient during the course of surgery and performing a limited neurological exam [5]. Though it serves the purpose of assuring a patient's neurological status, there are obvious limitations including the ability to perform the test during an operation, the need to wake the patient, and discontinuing the muscle relaxant intraoperatively, as well as increased risk of air embolism and infection [5, 9].

SSEPs were developed to overcome the problems associated with the wake-up test. SSEPs monitor the sensory pathways located in the posterior white matter of the spinal cord, and thus are most useful in dorsal spinal surgery [5, 9]. SSEPs involve stimulating nerves in the upper and lower extremity (such as the peroneal, ulnar, median, or posterior tibial) and then measuring the evoked potentials, usually at cortical sites [5]. Through a continuous recording of the amplitude and latency of these nerve potentials, possible injury to the spinal cord can be detected and, hopefully, reversed [9]. A significant change in SSEPs recording involves an amplitude signal decrease by 50 % or slowing of the latency by 10 % [5].

In addition, SSEPs can help distinguish the cause of insult. Ischemic injuries to the spinal cord occur at a slower rate than mechanical insults, which tend to be immediate. Ischemia may occur as a result of systemic or local hypotension or reduced spinal cord perfusion secondary to over distraction [1, 10]. Mechanical injuries may occur secondary to direct compression or over distraction of the spinal cord. In ischemic injuries, the amplitude is often significantly reduced, while the latency is unchanged. The latency is significantly prolonged, and the amplitude is significantly reduced in mechanical injuries [23].

The shortcomings of SSEPs are that there may be a false-positive rate as high as 1.5 % and more importantly a false negative rate of 0–13 %. Anesthetic agents alone may alter the SSEPs. Also, because SSEPs are a measure of sensory spinal cord injury functions, rather than motor function, there could be injury to the motor tracts without changes in the SSEPs. For this reason, most intraoperative monitoring today involves MEPs [5, 6]. MEPs can be elicited by stimulating either the spinal cord or the motor cortex of the brain. There are two types of MEPs: a compound nerve action potential, and the compound muscle action potential which is recorded on an EMG [6, 9]. The compound muscle action potential has several disadvantages of less reliable recordings during surgery and less predictability of patients' postoperative deficit [6]. Thus, compound nerve action potentials have been utilized more and more frequently and are considered highly reliable. A 10 % change in latency or an 80 % decrease in amplitude has been defined as a significant alteration and correlates with new-onset postoperative neurological deficit [24].

If changes in SSEP or MEP monitoring occur, steps must be taken to investigate the cause. Alterations may be due to anesthetic changes, technical aspects of the monitoring apparatus, or true neurological injury [2, 5, 6]. The test should be performed again, all leads checked, and assessment of whether anesthetic changes have occurred. If there was a preceding spinal cord manipulation, this should be stopped or reversed, usually in the form of hardware removal, support of blood pressure, or realigning the spinal cord [9]. MEPs also can help distinguish the mode of damage: a reduction of spinal cord blood flow has been shown to cause deterioration of motor evoked potentials and resultant spinal cord damage [6]. Hypoperfusion causes a slow loss of evoked potentials (>15 min), while cord compression causes a rapid loss (<5 min) of signals. A spinal cord blood flow of at least 65 % of baseline is required to maintain the physiological integrity of the spinal cord, and a decrease in blood flow to 12 % of baseline is associated with the potential for paralysis [3, 24].

40.8.2 Avoiding Direct Mechanical Trauma

Operative trauma to the spinal cord is seen more often in posterior surgery than anterior, and most often occurs during laminectomy [11, 14]. This may be caused by inadvertent pressure on the spinal cord with the use of rongeurs, particularly in patients with very significant stenosis [25]. Some surgeons avoid this by performing laminectomies by initially using a high-speed drill, making a laminotomy at the most caudal level to be removed and then inserting the footplate of a Kerris on punch and proceeding cephalad [10].

40.8.3 Prevention of Hypoperfusion Injury

This is especially pertinent when operating ventrally in the thoracic and thoracolumbar region. The major arterial supply to the spinal cord is through this region, and in some individuals through the artery of Adamkiewicz with its variable appearance from T9 to L2 [26]. There exists a "watershed" region of the spinal cord where blood flow is lowest; diminution of blood supply to this variable area may lead to spinal cord ischemia and infarction [1, 2, 4, 9]. Therefore, it is important to preserve the spinal arteries although there is literature that illustrated the redundancy of the spinal vasculature [3].

40.8.4 Prevention of Nerve Root Retraction Injury

Nerve root and the cal sac retraction are performed more commonly in the lumbar spine, due to the length of these nerve roots compared to thoracic nerves. However, over-retraction of the nerve roots of the cal sac may cause neurologic injury, and unfortunately there is no good contemporary intraoperative method of detecting this problem [10]. The only means of avoiding it is to prevent by gentle intermittent retraction with shorter total retraction time [27]. Removing more bone or ligament so that less traction is placed on the nerve may assist during surgical dissection [9]. Also, ensuring adequate contralateral decompression first avoids compressing the nerves during retraction.

40.8.5 Avoiding Instrumentation Injury

Intraoperative radiographs are useful for confirmation of the pedicle screw placement [10, 21]. Recent advances in pedicle screw placement are intraoperative frameless navigation, which illustrates the pedicle in three dimensions, and intraoperative EMG, which lets the surgeon know if a nerve root is irritated [10]. Posterior migration of an interbody graft (Fig. 40.4) causing nerve root compression or cauda equina symptom can be avoided by proper sizing and shaping of the implant and proper placement within the disk space [10]. Use of pedicle screws, with the potential for intraoperative compression of the interbody device such as bone or cage, enhances the stability of the construct [10].

Experience of a surgeon is another important factor affecting spinal surgical outcomes. However, even a technically skilled, experienced spine surgeon will develop complications. Proper patient selection, realistic outcome expectations, appropriate preoperative planning, and meticulous surgical technique are all keys to minimizing complications.

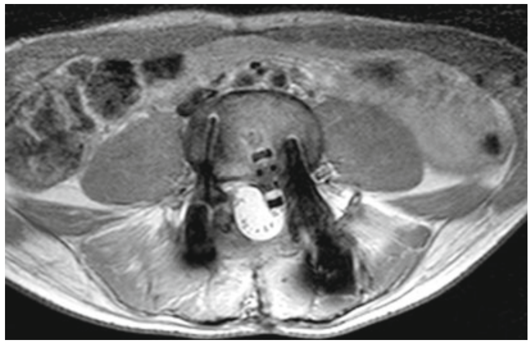


Fig. 40.4 Axial MRI of L-spine showing extruded interbody graft causing canal compromise

Questions

1. Which nerve root is most susceptible to traction injury during cervical surgery?
   (a) C5
   (b) C6
   (c) C7
   (d) C8
   (e) T1
   The correct answer is (a). C5 is a short nerve root and especially susceptible to injury with over distraction, especially during cervical corpectomy.
2. When performing laminoplasty hypotensive anesthesia is:
   (a) Beneficial in that it reduces blood loss
   (b) Up to the surgeons discretion to use assuming that anesthesiologist is able to perform it
   (c) Has been shown to decrease the likelihood of coronary events
   (d) None of the above
   The correct answer is (d). Need to keep blood pressure reasonably high to maintain cord perfusion, i.e., SBP > 100 or MAP > 80.
3. All of the following are true regarding cervical postoperative root palsies except:
   (a) They can occur with anterior corpectomy surgery.
   (b) Most tend to be motor dominant.

(c) Surgical treatment is mandatory in patients who develop postoperative root palsies.
(d) Postoperative root palsies most commonly involve C5.
(e) They can involve any root.

The correct answer is (c). No indication for surgical intervention. It can be treated medically with steroids or physical therapy.

4. Which of the following is a false statement regarding complications of laminoplasty?
   (a) Laminoplasty has higher a rate of infection than anterior surgery.
   (b) Irreversible segmental palsy of C5 is a common problem, which can be avoided by doing anterior surgery.
   (c) Some loss of lordosis occurs in laminoplasty, although in the vast majority of cases, it is not enough to be clinically evident or of the catastrophic form seen in post-laminectomy kyphosis.
   (d) Persistent worsening axial neck pain is a problem associated with laminoplasty.

   The correct answer is (b). C5 palsy can happen with anterior surgery as well though thought to be less frequent.

References

1. Saldua NS, Harrop JS (in press) Iatrogenic spinal cord injuries. In: The cervical spine, 5th edn. Lippincott Williams & Wilkins
2. Murthy NS, Maus TP, Behrns CL (2010) Intraforaminal location of the great anterior radiculomedullary artery (artery of adamkiewicz): a retrospective review. Pain Med 11(12):1756–1764
3. Greenberg MM (ed) (2010) Handbook of neurosurgery, 7th edn. Thieme, New York
4. Aono H, Ohwada T, Hosono N, Tobimatsu H, Ariga K, Fuji T et al (2011) Incidence of postoperative symptomatic epidural hematoma in spinal decompression surgery. J Neurosurg Spine 15:202–205
5. Awad JN, Kebaish KM, Donigan J, Cohen DB, Kostuik JP (2005) Analysis of the risk factors for the development of post-operative spinal epidural haematoma. J Bone Joint Surg Br 87-B(9):1248–1252
6. Sokolowski MJ, Garvey TA, Perl JII, Sokolowski MS, Cho W, Mehbod AA et al (2008) Prospective study of postoperative lumbar epidural hematoma: incidence and risk factors. Spine 33(1):108–113
7. Kebaish KM, Awad JN (2004) Spinal epidural hematoma causing acute cauda equina syndrome. Neurosurg Focus 16(6):1–4
8. Vaccaro A, Garfin SR (1995) Pedicle-screw fixation in the lumbar spine. J Am Acad Orthop Surg 3(5):263–274
9. Chiba K, Toyama Y, Matsumoto M, Maruiwa H, Watanabe M et al (2002) Segmental motor paralysis after expansive open-door laminoplasty. Spine 27(19):2108–2115
10. Vaccaro AR (2004) Complications of pediatric and adult spinal surgery. Marcel Dekker, New York
11. Sakaura H, Hosono N, Mukai Y, Ishii T, Yoshikawa H (2003) C5 palsy after decompression surgery for cervical myelopathy: review of the literature. Spine 28(21):2447–2451
12. Okada M, Minamide A, Endo T, Yoshida M, Kawakami M et al (2009) A prospective randomized study of clinical outcomes in patients with cervical compressive myelopathy treated with open-door or french-door laminoplasty. Spine 34(11):1119–1126
13. Sasai K, Saito T, Akagi S, Kato I, Ohnari H et al (2003) Preventing C5 palsy after laminoplasty. Spine 28(17):1972–1977
14. David KS, Rao RD (2006) Bilateral C5 motor paralysis following anterior cervical surgery – a case report. Clin Neurol Neurosurg 108(7):675–681
15. Wang MC, Chan L, Maiman DJ, Kreuter W, Deyo RA (2007) Complications and mortality associated with cervical spine surgery for degenerative disease in the united states. Spine 32(3):342–347
16. Romano PS, Campa DR, Rainwater JA (1997) Elective cervical discectomy in California: postoperative in-hospital complications and their risk factors. Spine 22(22):2677–2692
17. Komagata M, Inahata Y, Nishiyama M, Endo K, Tanaka H et al (2007) Treatment of myelopathy due to cervico thoracic OPLL via open door laminoplasty. J Spinal Disord Tech 20(5):342–346
18. Imagama S, Matsuyama Y, Yukawa Y, Kawakami N, Kamiya M, Kanemura T et al (2010) C5 palsy after cervical laminoplasty: a multicentre study. J Bone Joint Surg Br 92-B(3):393–400
19. Chiba K, Ogawa Y, Ishii K, Takaishi H, Nakamura M et al (2006) Long-term results of expansive open-door laminoplasty for cervical myelopathy-average14-year follow-up study. Spine 31(26):2998–3005
20. Hasegawa K, Homma T (2007) Chiba Y upper extremity palsy following cervical decompression surgery results from a transient spinal cord lesion. Spine 32(6):E197–E202
21. Nader R, Sabbagh AJ (2010) Neuro surgery case review: questions and answers. Thieme, New York
22. Park P, Lewandrowski K, Ramnath S, Benzel EC (2007) Brachial neuritis: an under-recognized cause of upper extremity paresis after cervical decompression surgery. Spine 32(22):E640–E644

23. Malhotra NR, Shaffrey CI (2010) Intraoperative electrophysiological monitoring in spine surgery. Spine 35(25):2167–2179
24. Gonzalez AA, Jeyanandarajan D, Hansen C, Zada G, Hsieh PC (2009) Intraoperative neurophysiological monitoring during spine surgery: a review. Neurosurg Focus 27(4):E6
25. Sin AH, Caldito G, Smith D, Rashidi M, Willis B, Nanda A (2006) Predictive factors for dural tear and cerebrospinal fluid leakage in patients undergoing lumbar surgery. J Neurosurg Spine 5(3):224–227
26. Kato S, Kawahara N, Tomita K, Murakami H, Demura S et al (2008) Effects on spinal cord blood flow and neurologic function secondary to interruption of bilateral segmental arteries which supply the artery of adamkiewicz: an experimental study using a dog model. Spine 33(14):1533–1541
27. Bosnjak R, Makovec M (2010) Neurophysiological monitoring of S1 root function during microsurgical posterior discectomy using H-reflex and spinal nerve root potentials. Spine 35(4):423–429

Complications: Pseudoarthrosis/ Nonunion

41

Raj Kullar, Eric Klineberg, and Munish Gupta

41.1 Introduction

Pseudoarthrosis of the spine is defined as significant motion in the area of intended fusion that occurs more than 1 year after the index surgery [1]. The diagnosis can be anticipated but cannot be made definitive before 1 year as the fusion mass continues to mature and may take as long as 1 year [2]. Radiographically, it is characterized by the presence of persistent motion and absence of bridging mature bony trabeculae between adjacent vertebral bodies [2]. Histologically fibrous soft tissue lies adjacent to mobile bone segments, with sclerotic bone at the margins of the fibrous tissue [3]. Furthermore, the cancellous bone has microfractures, which are postulated to be a source of pain. The incidence of pseudoarthrosis from posterior spine fusion surgery may range from 1 to 15 % following lumbar fusion, although this number may be much higher as many pseudoarthroses can be asymptomatic [4]. A recent review found that 23.6 % of revision fusion surgeries were undertaken for pseudoarthrosis [5].

Furthermore, a literature review by Herkowitz and Sidhu delineated the rates of pseudoarthrosis based on subtype of fusion. They found the incidence of pseudoarthrosis from 5 to 25 % for posterolateral fusion, 6–27 % for posterior lumbar interbody fusion, 20–30 % for anterior lumbar interbody fusion, and 6–27 % for posterior lumbar interbody fusion [6].

R. Kullar, M.D.
Department of Orthopaedic Surgery,
UC Davis Medical Center,
4860 Y St, Suite 3800, Sacramento, CA 95817, USA
e-mail: raj.kullar@ucdmc.ucdavis.edu

E. Klineberg, M.D. (✉) • M. Gupta, M.D.
Department of Orthopaedic Surgery,
UC Davis Medical Center,
4860 Y St, Suite 3800, Sacramento, CA 95817, USA

Adult and Pediatric Spinal Surgery,
Department of Orthopaedic Surgery,
UC Davis Health System, 4860 Y Street, Suite 3800,
Sacramento, CA 95817, USA
e-mail: eric.klineberg@ucdmc.ucdavis.edu;
munish.gupta@ucdmc.ucdavis.edu

41.2 Case Example

The Patient is a 15-year-old female with adolescent idiopathic scoliosis diagnosed at age 11. Her menstrual period began at age 13. Although she has been braced since age 11, her curve has progressed. Physical exam showed an elevated right scapula and prominent right thoracic hump and left lumbar hump. Radiographs are pictured demonstrating approximately 60° thoracic and 60° lumbar curve (Figs. 41.1 and 41.2).

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_41, © Springer-Verlag Berlin Heidelberg 2014

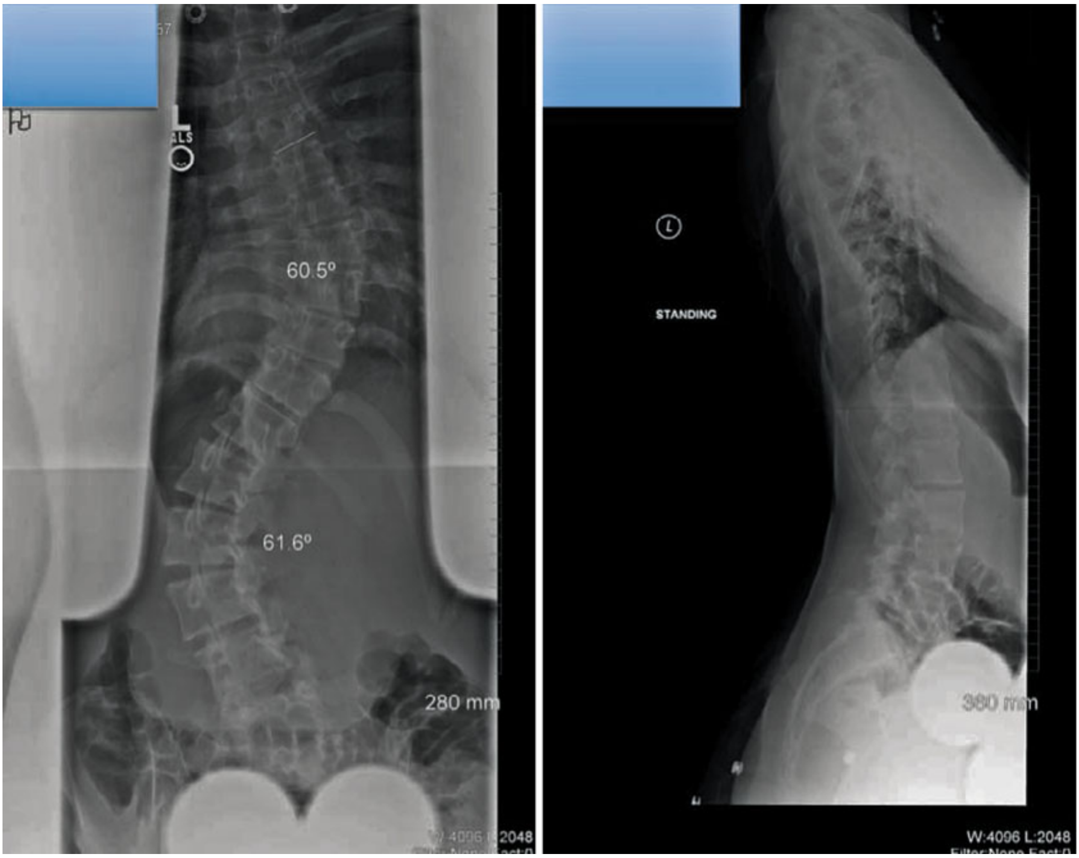


Fig. 41.1 Preoperative radiographs showing double major curves and large deformity

She underwent T4–L4 posterior spine fusion with local autograft and cancellous allograft. She did well until 1 year after surgery, when she was bending forward and heard a pop and noticed a shift in her trunk. Radiographs showed failed instrumentation bilaterally (Fig. 41.3). She is scheduled for revision surgery.

41.3 Classification

In general, nonunions are classified as either hypertrophic or atrophic [7]. Hypertrophic nonunions demonstrate abundant bone formation and are typically thought of as a consequence of inadequate stability in the setting of adequate local biology. In contrast, atrophic nonunions demonstrate little to no bone formation due to poor vascularity and poor local supply of bone forming cells. Thus, the potential treatments for the two types of nonunions should vary. Hypertrophic nonunions that need stability likely will require addition or revision of instrumentation. Atrophic nonunions would be made more likely to fuse and unite with the addition of local biology, either with growth factors or autograft.

Furthermore, Heggeness et al. developed a classification system for spine pseudoarthrosis [3]. Posterolateral lumbar pseudoarthroses were classified into four patterns: atrophic, transverse, shingle, and complex. The atrophic pattern is characterized by an absence of bone formation, with significant graft resorption. Transverse pseudoarthrosis, similar to a hypertrophic nonunion, involves the presence of a large mass of remodeled bone without continuous bridging. This is the most common type of spinal nonunion. Shingle pseudoarthroses are defined by a defect

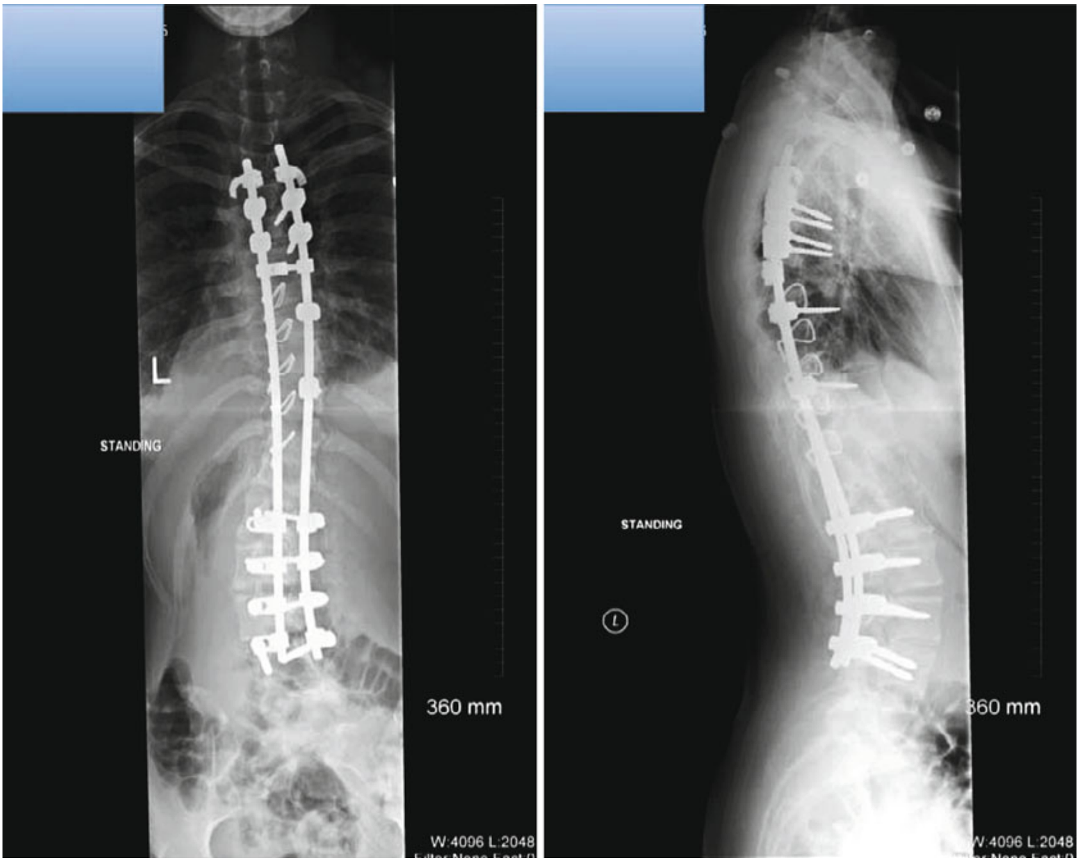


Fig. 41.2 Postoperative radiographs showing correction of deformity and posterior instrumentation

within the fusion mass that passes sagittally and obliquely, leading to a defect within the fusion mass between the graft and the adjacent vertebra. Lastly, complex pseudoarthroses are a mixture of the above types.

41.4 Risk Factors

41.4.1 Patient Factors

Several patient-specific risk factors have been identified that may contribute to the development of pseudoarthrosis. The most often cited is smoking, which can interfere with the development of a mature fusion. Nicotine impairs revascularization and can thus inhibit new bone formation. A study by Brown et al. demonstrated a nonunion rate of 40 % in smokers and 8 % in nonsmokers after two-level laminectomy and fusion in a case–control study of 100 patients [8].

Systemic diseases affecting thyroid hormone, growth hormone, or estrogen levels can also impact bone formation and thus increase pseudoarthrosis risk [2]. Corticosteroids can impair bone healing, and chemotherapeutic agents can decrease bone formation. Nonsteroidal anti-inflammatories inhibit the initial inflammatory response, which is necessary for bone healing and thus spine fusion [2]. Age greater than 55 may also play a role in pseudoarthrosis and is noted as a risk factor in several papers [9].

41.4.2 Deformity Specific Factors

Pathology that requires long instrumentation and fusion to the sacrum has an increase in the

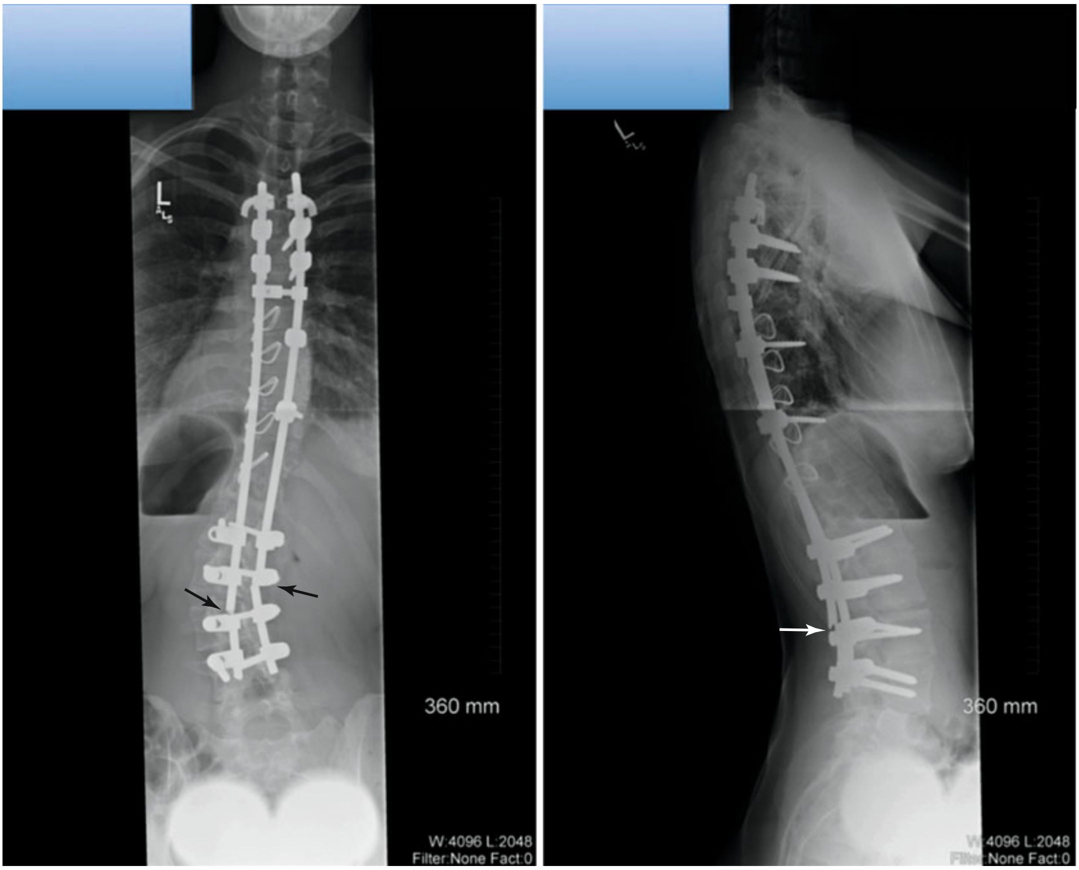


Fig. 41.3 One year postoperatively (*arrows*) and loss of correction

nonunion rate. These deformity characteristics include kyphosis greater than 20°, positive sagittal balance greater than 5 cm, and preexisting hip osteoarthritis [9].

The use of instrumentation reduces the risk of pseudoarthrosis significantly by reducing the amount of micromotion. In a prospective, randomized controlled study of lumbar decompression and posterolateral fusion with and without instrumentation, the incidence of pseudoarthrosis was 45 % versus 82 %, respectively, at 2 years [10]. Extension of the fusion to the pelvis is also challenging, and fusion at the L5–S1 disc space can be difficult. A study by Kim et al. looked at 144 adult patients undergoing fusion to the sacrum for cases of deformity [9]. They observed a 24 % incidence of pseudoarthrosis at minimum 2-year follow-up. Furthermore, a matched cohort study of fusions ending at L5 versus S1 in patients with a "healthy" L5–S1 level showed an incidence of pseudoarthrosis of 4 % to L5 and 42 % to S1 [11]. In thoracolumbar fusions to the sacrum, the incidence of nonunion demonstrating fractured instrumentation may be as high as 83 % [9]. Thus, attempted fusion across the thoracolumbar or lumbosacral junction may enhance risk of pseudoarthrosis. The biological and mechanical burden of a long fusion also impacts bone healing and increases the risk of pseudoarthrosis [1]. Cleveland et al. reported fusion rates of 90.3 % for one level, 77.2 % for two levels, and 65.2 % for three levels for posterolateral lumbar spinal fusion.

Fusion technique can also contribute. Intertransverse process fusions have a lower rate of pseudoarthrosis than standard midline technique [2]. Similarly, anterior or posterior lumbar interbody fusions have higher fusion rates than standard posterior fusions due to a higher surface area available for healing [2].

41.5 Diagnosis

A minimum of 1-year postoperative evaluation is required before a diagnosis of pseudoarthrosis can be made. The time to presentation for patients can vary significantly. A review by Kim et al. of 40 pseudoarthroses found an average time to presentation of 3.5 years [12]. In 23 % of these cases, it was even more delayed at 5–10 years after surgery. The most common presentation of nonunion was instrumentation fracture followed by progression of deformity.

Diagnosis involves a thorough history and physical, followed by advanced imaging studies. The history should include a detailed assessment of patient symptoms. If preoperative symptoms did not change, it is unlikely their symptoms are from pseudoarthrosis but more likely from wrong-level surgery, unaddressed pathology, or symptoms at adjacent levels [2]. If symptoms subside but then return, it may be consistent with nonunion. Progressive symptoms are often also more consistent with pseudoarthrosis.

These changes in patient satisfaction can be more easily detected in outcome measures. Scoliosis Research Society (SRS) questionnaires are utilized by many spinal deformity surgeons to both assess and monitor symptoms related to spinal deformity. The SRS questionnaire has been well validated as an assessment tool in spinal deformity, considered as the standard for determining and comparing treatment outcomes in spinal deformity [13]. Low patient outcome scores are known to correlate with pseudoarthrosis and may deteriorate over time [9]. In addition, physical examination should focus on a thorough neurologic examination to evaluate for deficits, as well as palpation of hardware to discover prominent or painful hardware [2].

The diagnosis of pseudoarthrosis is typically made radiographically in a patient with persistent pain. Several radiographic modalities exist, including flexion–extension radiographs, computed tomography (CT) scans, and magnetic resonance imaging [14]. Plain radiographs may show loss of correction, especially with scoliotic deformities. Although no standard radiographic parameters exist, loss of correction of 10° is agreed as an indicator of possible pseudoarthrosis [15]. Also, segmental motion of greater than 3° in flexion–extension indicates likely nonunion. Plain radiographs have the lowest sensitivity (average 70 %) but high specificity (average 90 %) in detection of presence or absence of fusion [16–18].

CT scans provide more detailed information than plain radiographs, although metal artifact can obscure thorough analysis. Newer 3D CT imaging and instrumentation subtraction technology have shown a better ability to display the fusion mass and assess union [2]. Bone scan has a low sensitivity and positive predictive value for pseudoarthrosis and is considered poorly reliable for diagnosis of pseudoarthrosis [1]. Flexion-extension radiographs may also be helpful in situations of gross instability, but often rigid instrumentation may limit motion. In addition, there are no standardized criteria for assessment of instability [1]. An agreed parameter is >5° of instability, although absence of instability on X-ray does not exclude pseudoarthrosis. Despite this, it is the most affordable and accessible modality, so it is still often ordered first in nonunion evaluation [2]. The gold standard for diagnosis of pseudoarthrosis remains surgical exploration, but even that is not 100 % accurate [19].

41.6 Prevention

Successful treatment of the nonunion involves an understanding of patient symptoms, a thorough radiographic evaluation, and an assessment of the cause of the nonunion. Preoperative radiographs should be evaluated for coronal and sagittal balance, bone quality, and rigidity of previous fixation, and types of operations.

Clearly, prevention of nonunion at the index surgery is the critical first step. Many patient-related risk factors are not modifiable, but lifestyle choices are. Smoking cessation is an important consideration for nonunion prevention. Many spinal deformity surgeons refuse to operate on smokers and require a 3-month nicotine-free interval tested with urine nicotine levels.

Additional steps that can be taken to prevent nonunion include addition of bone grafts and osteobiologics. The gold standard for autogenous bone graft is iliac crest bone graft (ICBG). ICBG is both osteoconductive and osteoinductive. However, there is significant morbidity with its harvest, including bleeding, gait disturbance, fracture, and donor site pain as high as 29 % [20]. A study by Banwart et al. found a 10 % incidence of major complications and 39 % incidence of minor complications after ICBG [20]. However, pseudoarthrosis rates approached 2.7 % with the addition of ICBG with posterior instrumentation and decortication [21].

With the morbidity associated with ICBG harvest, as well as limitation in quantity of graft that can be obtained, particularly with revision surgery, significant investigation has gone into finding a suitable alternative. Options include allograft, demineralized bone matrix, and bone morphogenetic proteins. Several studies have compared allograft versus autograft in idiopathic scoliosis and found that allograft is an acceptable alternative, but limited by its lack of osteoinductive properties [20].

Demineralized bone matrix has both osteoconductive and osteoinductive properties [20]. It is composed of extracellular matrix of noncollagenous bone-inducing proteins. When taken in isolation, several studies have shown acceptable fusion rates, but notably lower than autograft [20]. A retrospective study of 88 patients compared the use of autograft, corticocancellous allograft, and demineralized bone matrix (DBM) in adolescent idiopathic scoliosis [20]. At minimum 2-year follow-up, the rate of pseudoarthrosis was 12.5 % in the ICBG group, 28 % in the corticocancellous allograft group, and 11.1 % in the DBM + bone marrow aspirate group. The authors concluded that DBM with bone marrow aspirate was an acceptable alternative to ICBG in adolescent idiopathic scoliosis. More recently, bone marrow aspirate or bone morphogenetic proteins have been added to DBM to improve its fusion rate.

The use of osteobiologics, most notably bone morphogenetic proteins (BMP), has reduced the incidence of pseudoarthrosis significantly. In a randomized controlled trial of BMP-2 versus iliac crest bone graft in anterior lumbar interbody fusion, patients receiving BMP-2 had 100 % fusion [22]. A study by Dimar et al. comparing fusion rates with iliac crest bone graft versus BMP-2 found a statistically significantly higher fusion rate in the BMP group [23]. BMP use has become standard for many surgeons in adult deformity.

In addition to grafting, surgical approach can be tailored toward the specific deformity. For idiopathic scoliosis, excellent results have been obtained with posterior only fixation, especially with an adequate posterior release and segmental instrumentation. A multicenter analysis of two groups of patients either treated with anterior dual rod versus posterior pedicle fixation for idiopathic scoliosis found no difference in curve correction or loss of correction/pseudoarthrosis at minimum of 2-year follow-up [24]. In contrast, in adult scoliosis, anterior fusion has fared better, leading to solid fusion in 15 patients with anterior instrumentation alone for thoracic and lumbar scoliosis [25].

41.7 Treatment

Once a nonunion has been identified, there are several options for treatment. A study by Pateder et al. detailed an algorithm for treatment [26]. Patients with single-level nonunions without intraoperative instability were treated with posterior only revision with six points of fixation. Patients with coronal and sagittal plane nonunions were treated with anterior stabilization. All L5–S1 nonunions received anterior and posterior fixation. With this algorithm, the investigators reported a 90 % rate of fusion at greater than 3-year follow-up. A second group recently reported their algorithm for nonunion management [2]. In patients with posterior instrumentation, revision posterior instrumentation may be considered unless there is posterior element deficiency, multiple levels involved, or the patient has a number of risk factors for nonunion as detailed above. If patients fail a second surgery or fit the exclusion criteria, they are considered for anterior interbody fusion. The fusion rate for combined anterior and posterior fusion surgery pseudoarthrosis has union rates near 100 % [27–29].

In patients with posterolateral fusion without fixation, the pseudoarthrosis is often secondary to micromotion and instability, and revision fusion with instrumentation is indicated [2]. A retrospective review by Kim et al. found a 55 % fusion rate with repair of pseudoarthrosis with autograft and external immobilization alone [30]. Similarly, 80 % of patients had a solid fusion after revision posterior instrumentation using plate fixation with autograft. Twenty percent needed additional interbody fusion [31].

For nonunions at our institution, determination of the primary cause of nonunion is critical. Nonunion that is hypertrophic is considered a sign of inadequate fixation, and pedicle screw constructs can provide appropriate stability. Rod breakage is a sign of fatigue failure and loss of stability. Nonunions that are atrophic may be the result of poor grafting technique or inadequate biology. For these cases, rigid instrumentation is supplemented by osteoinductive biologics which are important to achieve a solid arthrodesis. Special consideration at L5–S1 includes pelvic fixation for additional posterior stability and interbody grafting if possible. Also as critical is the exploration of the nonunion site. All fibrous tissue must be removed, until bleeding, cancellous bone is exposed. This may require extensive debridement in most cases, but it is extremely important to provide appropriate biology and fusion surface area available for union.

Conclusions

Treatment of patients with spinal deformity includes decompression of neural elements, correction and prevention of deformity progression, and maintenance of coronal and sagittal balance. Unfortunately, one postoperative complication may be the development of a nonunion, even several years after the index procedure. Prevention of a nonunion involves minimizing potential risk factors at the index surgery. Patients should be adequately informed of the known complications, risk stratified, and medically optimized prior to surgery. If, despite these precautions, a nonunion occurs, prompt diagnosis and correct proactive management is appropriate. Symptomatic nonunions are addressed with revision surgery that requires debridement of the nonunion site and additional stability. Osteobiologics and autograft are important considerations for both the primary and revision procedures.

References

1. Raizman NM, O'Brien JR, Poehling-Monaghan KL, Yu WD (2009) Pseudoarthrosis of the spine. J Am Acad Orthop Surg 17(8):494–503
2. Etminan M, Girardi FP, Khan SN, Cammisa FP (2002) Revision strategies for lumbar pseudoarthrosis. Orthop Clin North Am 33(2):381–392
3. Heggeness MH, Esses SI (1991) Classification of pseudoarthrosis of the spine. Spine 16(Suppl 8): S449–S454
4. Hsu WK, Wang JC (2008) The use of bone morphogenetic protein in spine fusion. Spine J 8:419–425
5. Martin BI, Mirza SK, Comstock BA, Gray DT, Kreuter W, Deyo RA (2007) Reoperation rates following lumbar spine surgery and the influence of spinal fusion procedures. Spine 32:382–387
6. Herkowitz HN, Sidhu KS (1995) Lumbar spine fusion in the treatment of degenerative conditions: current indications and recommendations. J Am Acad Orthop Surg 3:123–135
7. Megas P (2005) Classification of non-union. Injury 36(Suppl 4):S30–S37
8. Brown CW, Orme TJ, Richardson HD (1986) The rate of pseudarthrosis (surgical nonunion) in patients who are smokers and patients who are nonsmokers: a comparison study. Spine 11:942–943
9. Kim YJ, Bridwell KH, Lenke LG, Rhim S, Cheh G (2006) Pseudoarthrosis in long adult spinal deformity instrumentation and fusion to the sacrum: prevalence and risk factor analysis in 144 cases. Spine 31(20):2329–2336
10. Fischgrund JS, Mackay M, Herkowitz HN, Brower R, Montgomery DM, Kurz LT (1997) Degenerative lumbar spondylolisthesis with spinal stenosis: a prospective, randomized study comparing decompressive laminectomy and arthrodesis with and without spinal instrumentation. Spine 22:2807–2812
11. Edwards CC, Bridwell KH, Patel A, Rinella AS, Berra A, Lenke LG (2004) Long adult deformity fusions to L5 and the sacrum: a match cohort analysis. Spine 29(18):1996–2005
12. Kim YJ, Bridwell KH, LEnke LG, Cho KJ, Edwards CC, Rinella AS (2006) Pseudoarthrosis in adult spinal deformity following multisegmental instrumentation and arthrodesis. J Bone Joint Surg Am 88:721–728

13. Baldus C, Bridwell K, Harrast J, Shaffrey C, Ondra S, Lenke L, Schwab F, Mardjetko S, Glassman S, Edwards C 2nd, Lowe T, Horton W, Polly D Jr (2011) The scoliosis research society health-related quality of life (SRS-30) age-gender normative data: an analysis of 1346 subjects unaffected by scoliosis. Spine 36(14):1154–1162
14. An HS, Seldomridge JA (2006) Spinal infections: diagnostic tests and imaging studies. Clin Orthop Relat Res 444:27–33
15. Lauerman WC, Bradford DS, Transfeldt EE, Ogilvie JW (1991) Management of pseudoarthrosis after arthrodesis of the spine for idiopathic scoliosis. J Bone Joint Surg Am 73(2):222–236
16. Blumenthal SL, Gill K (1993) Can lumbar spine radiographs accurately determine fusion in postoperative patients? Correlation of routine radiographs with a second surgical look at lumbar fusions. Spine 18(9):1186–1189
17. Brodsky AE, Kovalsky ES, Khalil MA (1991) Correlation of radiologic assessment of lumbar spine fusions with surgical exploration. Spine 16(6 Suppl): S261–S265
18. Kant AP, Daum WJ, Dean SM, Uchida T (1995) Evaluation of lumbar spine fusion. Plain radiographs versus direct surgical exploration and observation. Spine 20(21):2313–2317
19. Deckey JE, Court C, Bradford DS (2000) Loss of sagittal plane correction after removal of spinal implant. Spine 25:2453–2460
20. Price CT, Connolly JF, Carantzas AC, Ilyas I (2003) Comparison of bone grafts for posterior spinal fusion in adolescent idiopathic scoliosis. Spine 28(8): 793–798
21. McMaster MJ, James JI (1976) Pseudoarthrosis after spinal fusion for scoliosis. J Bone Joint Surg Br 58:305–312
22. Burkus JK, Transfeldt EE, Kitchel SH, Watkins RG, Balderston RA (2002) Clinical and radiographic outcomes of anterior lumbar interbody fusion using recombinant human bone morphogenetic protein-2. Spine 27:2396–2408
23. Dimar JR, Glassman SD, Burkus KJ, Carreon LY (2006) Clinical outcomes and fusion success at 2 years of single-level instrumented posterolateral fusions with recombinant human bone morphogenetic protein-2/compression resistant matrix versus iliac crest bone graft. Spine 31:2534–2539
24. Geck MJ, Rinella A, Hawthorne D, Macagno A, Koester L, Sides B, Bridwell K, Lenke L, Shufflebarger H (2009) Comparison of surgical treatment in lenke 5C adolescent idiopathic scoliosis: anterior dual rod versus posterior pedicle fixation surgery: a comparison of two practices. Spine 34(18):1942–1951
25. Smith JA, Deviren V, Berven S, Bradford DS (2002) Does instrumented anterior scoliosis surgery lead to kyphosis, pseudoarthrosis, or inadequate correction in adults? Spine 27:529–534
26. Pateder DB, Park YS, Kebaish KM, Cascio BM, Buchowski JM, Song EW, Shapiro MB, Kostuik JP (2006) Spinal fusion after revision surgery for pseudarthrosis in adult scoliosis. Spine 31(11): E314–E319
27. Butterman GR, Glazer PA, Hu SS, Bradford DS (1997) Revision of failed lumbar fusions: a comparison of anterior autograft and allograft. Spine 22: 2748–2755
28. Gertzbein SD, Hollopeter MR, Hall S (1998) Pseudoarthrosis of the lumbar spine: outcome after circumferential fusion. Spine 23:2352–2356
29. Cohen DB, Chotivichit A, Fujita F, Wong TH, Huckell CB, SEiber AN (2000) Pseudoarthrosis repair: autogenous iliac crest graft versus femoral ring allograft. Clin Orthop 371:46–55
30. Kim SS, Michelsen CB (1992) Revision surgery for failed back surgery syndrome. Spine 17:957–960
31. Thalgott J, LaRocca H, Gardner V, Wetzel T, Lowery G, White J, Dwyer A (1991) Reconstruction of failed lumbar surgery with narrow AO DCP plates for spinal arthrodesis. Spine 16(3 Suppl):S170–S175

42 Medical Complications

Rachid Assina and Robert F. Heary

42.1 Introduction

In spine surgery, the occurrence of complications is inevitable. The complications may occur during the perioperative or postoperative periods and can be either specific to the type of surgery performed or be a general medical complication. The frequency of medical complications is dependent on the following: patient risk factors, the underlying pathology, the complexity of the procedure, the duration of the procedure, and the postoperative care. The lack of a clear definition of perioperative complications, and a standardized method of reporting them, contributes to the wide variability in the reported incidence of complications in spine surgery [1]. In addition, reliance on retrospective studies underestimates the true incidence of complications in spine surgery [2].

Although the highest incidence is between postoperative days 1 and 3, specific complications occur in a distinct sequential pattern: immediate, early, or late [3]. The consequent morbidity and mortality from these complications can reverse the potential benefit from the original surgery. The surgeon and the medical team need to be aware of these potential complications, and the time frame in which they occur, to be able to diagnose and treat them in a timely fashion. Delayed recognition and management of complications leads to anguish for the patient. Constant vigilance is required to recognize, assess, and treat complications before they become serious.

R. Assina, RPH, M.D.
Spine Center, Newark, NJ, USA
e-mail: rassina@yahoo.com

R.F. Heary, M.D. (✉)
Spine Center, Newark, NJ, USA

Department of Neurosurgery, Newark Medical School, University of Medicine and Dentistry of New Jersey, 90 Bergen Street, Suite 8100, Newark, NJ 07103, USA
e-mail: heary@umdnj.edu

42.2 Case Example

A 65-year-old male with past medical history significant for HTN, DM, and CAD who developed neurogenic claudication did not respond to multiple epidural steroid injections and physical therapy. An MRI of his lumbar spine demonstrated an L4–L5 anterolisthesis with bilateral foraminal stenosis. Lumbar flexion and extension X-rays demonstrated dynamic instability. Sequential compression devices were place on his lower extremities before, during, and after surgery. He underwent a posterior lumbar L4–L5 decompression and interbody fusion that was complicated by a dural tear. A lumbar drain was placed. Immediately postoperatively, his heart rate was 140 and he complained of chest pain. For pain control, he was placed on a morphine PCA pump. The patient was kept at bed rest for 5 consecutive days to prevent cerebrospinal fluid leak. On postoperative day 3, he had abdominal distension, and he complained of abdominal pain.

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7_42, © Springer-Verlag Berlin Heidelberg 2014

On postoperative day 5, after removal of the lumbar drain and Foley catheter, he was permitted to get out of bed and ambulate. After 6 h, he had urinary retention and a PVR of 700 cc. The Foley catheter was replaced. While ambulating, he complained of pain in the right calf that was noted to be warmer and larger than the left calf. He was immediately returned to his room, and before getting into his bed, he became short of breath and collapsed. The medical team was unable to resuscitate him, and he expired.

42.3 Vascular Complications: Deep Venous Thrombosis

Deep venous thrombosis (DVT) occurs when a thrombus develops in the large veins of the proximal lower extremities or the pelvic area. This scenario occurs as a result of activation of the coagulation system in areas of reduced blood flow. Contrary to superficial thrombi, clots that form in the deep large veins are more likely to dislodge and travel through the veins, as an embolus, to the lungs and cause a pulmonary embolism (PE). A PE can be a fatal condition if not diagnosed and treated in an expeditious fashion.

42.3.1 Incidence

Deep venous thrombosis is a common and significant potential medical complication after spinal surgery. DVTs occur at a reported rate of between 0.3 and 31 % [4–10]. The rate of DVT formation depends on the patient population, the methods of surveillance, whether DVT prophylaxis is utilized, and the type of DVT prophylaxis. The incidence of DVT is significantly higher in individuals with spinal cord injury (SCI), ranging between 12 and 64 % [11, 12]. This increased DVT rate in SCI patients makes them vulnerable for a venous thromboembolic event, which is one of the major factors that accounts for the 9.7 % mortality rate [13] during the first year following SCI.

42.3.2 Risk Factors

Several clinical risk factors for DVT have been identified, including advanced age, prolonged immobilization, previous DVT or PE, malignancy, obesity, pregnancy, oral contraceptive or estrogen use, smoking, vasculitis, stroke, congestive heart failure, myocardial infarction, and inherited or acquired prothrombotic clotting disorders [14, 15].

According to Virchow's pathogenesis of venous thrombosis [16], three primary factors, referred to as "Virchow's triad", predispose to thrombus formation: (1) stasis or turbulence of blood flow, (2) vessel wall damage (endothelial injury), and (3) an increased tendency of blood to clot (hypercoagulability) [17]. These factors may act independently, or in combination, to cause venous thrombosis. Especially in spine surgery patients who often suffer from extended periods of forced accumbency before surgery secondary to pain or neurological deficit and decreased mobility after surgery, stasis is a major factor in the development of venous thrombi [18]. In addition to the limited mobility, spine surgery patients have additional risk factors which can be related to the lengthy operative time in complex spinal cases, to the manipulation of the great vessels during anterior and lateral approaches to the spine, and to compression of the femoral venous system by certain frames during prone positioning for posterior approaches to the spine [4, 5, 19, 20].

42.3.3 Presentation

DVT can be occult and may occur without any noticeable signs or symptoms. When signs and symptoms do occur, they are usually a sequela of the thrombus that decreases venous blood outflow causing swelling, redness, warmth, tenderness, and pain of the affected extremity. If this progresses to an acute occlusion of a major outflow vein, the affected extremity may become pale and cold with diminished arterial pulses (phlegmasia alba dolens). If there is total occlusion of the venous outflow of the entire affected extremity, extreme pain, petechiae, and cyanosis ensue (phlegmasia cerulea dolens). Although there is not a single pathognomonic physical finding that accurately establishes the diagnosis of DVT [21, 22], the most sensitive and specific physical findings are unilateral edema, unilateral tenderness of the calf muscles along the affected deep vein, a positive Homan's sign (reproducible calf muscle pain upon dorsiflexion of the foot with the knee straight), superficial thrombophlebitis, and low-grade fever [23].

42.3.4 Differential Diagnosis

There are multiple pathological conditions referred to as pseudo-deep venous thrombosis (PDVT) that mimic DVT clinically [24]. PDVT is found in more than half of patients with clinically suspected DVT and is usually caused by calf hematoma, ruptured Baker's cyst, lipoma, muscle or soft tissue abscess, lymphedema, lymphangitis, cellulitis, superficial thrombophlebitis, or acute synovial rupture in rheumatoid arthritis.

42.3.5 Investigation

Although the clinical examination of the postoperative patient is generally considered nonspecific and insensitive, the presence of any signs or symptoms of DVT should prompt further investigation. In order to prevent a PE, the potentially fatal complication of thrombosis, the diagnosis of DVT is time sensitive.

In either asymptomatic or symptomatic patients, the use of the Wells clinical prediction guide [25] (Table 42.1) attempts to quantify the probability of DVT and stratifies patients into high, moderate, or low risk clinical probability groups where the prevalence of DVT is approximately 70, 15, or 5 %, respectively [25, 26].

A fibrinolytic marker, D-dimer fibrin fragment, is a degradation product of cross-linked fibrin in fresh clots and plays an important role in the predictive diagnostic approach of DVT [27].

Table 42.1 Wells clinical prediction guide for DVT

Clinical feature	Score
Active cancer (ongoing treatment or within previous 6 months or palliative)	1
Paralysis, paresis, or recent plaster immobilization of the lower extremities	1
Recently bedridden for more than 3 days or major surgery, within 4 weeks	1
Localized tenderness along the distribution of the deep venous system	1
Entire leg swollen	1
Unilateral calf swelling of greater than 3 cm	1
Pitting edema (greater in the symptomatic leg)	1
Collateral superficial veins (non-varicose)	1
Alternative diagnosis as likely as or more likely than DVT	–2
Total score	–

In patients with symptoms in both legs, the more symptomatic leg is used. Risk score interpretation: less than 1 point, low risk; 1–2 points, moderate risk; 3 or more points, high risk

D-dimer is more than 95 % sensitive and can reasonably rule out acute DVT in patients with low to moderate clinical probability [28–30]. D-dimer assays are nonspecific for DVT [29] because D-dimer level may be elevated in any medical condition where active thrombosis is present. All patients with positive D-dimer assay and high clinical probability require further investigation and should undergo a confirmatory objective diagnostic imaging study.

The previous "gold standard" for evaluating patients for suspected DVT has been contrast venography. Venography visualizes the entire venous system of the lower extremities and pelvis. Because of many disadvantages [31] including invasiveness, allergic reactions, contrast-induced DVT, interobserver variability, technical problems, and lack of availability, venography has been largely replaced by noninvasive imaging studies.

Duplex ultrasonography has become the primary noninvasive diagnostic study for detecting proximal DVT since it has been widely reported to have both high sensitivity (92–95 %) and specificity (97–100 %) [32] (Fig. 42.1). In addition, duplex scanning is noninvasive, readily

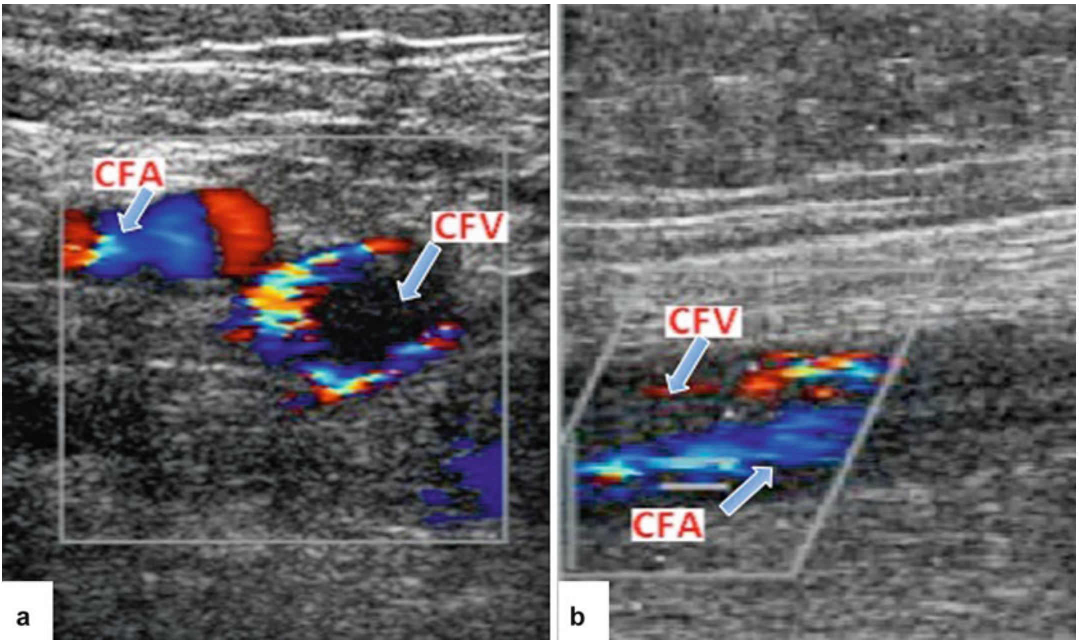


Fig. 42.1 Transverse (**a**) and sagittal (**b**) images of the right lower extremity from color Doppler ultrasound demonstrating an acute common femoral vein (*CFV*) thrombosis and normal blood flow in the common femoral artery (*CFA*)

available, and cost-effective. The major disadvantage of duplex is its limited sensitivity for detecting calf vein (48 %) and pelvic vein (64 %) thromboses [33].

Magnetic resonance venography (MRV) has been found to be as sensitive and specific as contrast venography [33] and ultrasound [34] in the diagnosis of DVT. It is more sensitive than any other noninvasive study in detecting pelvic and below-knee thrombosis, and thus, it is the diagnostic test of choice (100 % sensitive and specific) [35] for iliofemoral DVT. The principle downside of MRV is the prolonged time required to acquire the images.

Computed tomography venography (CTV) has a similar sensitivity, compared to MRV and duplex ultrasonography, for femoropopliteal thrombosis and is 100 % sensitive and 96 % specific in detecting lower extremity DVTs [36]. CTV is emerging as the study of choice for detecting iliofemoral DVT because of its accuracy, availability, and shorter study time [37]. CTV is not feasible in certain clinical situations including iodine allergy, renal failure, or pregnancy.

42.3.6 Treatment

The treatment of DVT is guided by the American College of Chest Physicians (ACCP) Evidence-Based Clinical Practice Guidelines recommendations [38]. The goals of treatment are thrombus resolution and prevention of thrombus propagation/embolization. Treatment is based on whether the diagnosis is of a first episode versus a recurrent episode of DVT, a provoked versus an unprovoked DVT, secondary to transient versus permanent risk factors, and presence versus absence of concurrent cancer.

According to the ACCP guidelines, a confirmed acute DVT should be treated with low molecular weight heparin (LMWH), unfractionated heparin (UFH), or fondaparinux (a factor Xa inhibitor) for at least 5 days with concomitant use of a vitamin K antagonist (VKA), such as warfarin (Coumadin), starting the first day. Once the international normalized ration (INR) is equal to or greater than 2.0, for at least 24 h, the heparin preparation should be discontinued. For DVT secondary to a transient, reversible risk factor, VKA should be used for 3 months. In occurrences of unprovoked DVT, a VKA should be used for at least 3 months. After the initial 3-month treatment, the patient should be evaluated for the risk-benefit of indefinite VKA treatment. Indefinite anticoagulation treatment is recommended for first unprovoked DVT in patients with low bleeding risks and for most patients with a second unprovoked DVT. The target INR for treating a DVT is 2.5 with a range of 2.0–3.0.

Selected patients with lower extremity DVT may be considered for thrombectomy using catheter-based thrombolytic techniques or open surgical removal. Elastic compression stocking use is recommended to prevent post-thrombotic syndrome after proximal DVTs. DVT in the setting of cancer should be treated with LMWH for 3–6 months, then indefinitely with VKA, or until cancer resolves.

For patients with high and/or unacceptable risks of bleeding after systemic anticoagulation, such as patients who recently underwent complex spine surgery, inferior vena cava (IVC) filter placement is an alternative option. IVC filters have been associated with a 98 % success rate in prevention of PE in patients with known DVT [39].

42.3.7 Prophylaxis

The silent nature of DVT is the basis for prophylaxis. The first and only symptom of a DVT could be a fatal pulmonary embolism. Unfortunately, there are no generally agreed upon guidelines for DVT prophylaxis in spine surgery. Recommendations are widely variable and inconsistent. The great deal of variation among spine surgeons regarding thromboembolic prophylaxis is mainly due to the paucity of accurate and consistent scientific evidence concerning the risks of DVT, PE, symptomatic epidural hematoma, the efficacy of a specific prophylactic protocol, and the safety of chemoprophylaxis protocols after spinal surgery.

Spine surgeons are faced regularly with the questions of if, when, and how DVT prophylaxis should be administered. Spine surgeons are reluctant to initiate prophylactic chemical anticoagulation immediately after spinal surgery because of fear of a catastrophic symptomatic epidural hematoma [40] and the risk of wound breakdown [41]. Surgeons have to use their best judgment to balance the risk of DVT, possibly causing a fatal PE, against the risk associated with the use of chemoprophylaxis.

According to the North American Spine Society (NASS), evidence-based clinical guideline recommendations [42], mechanical DVT prophylaxis of any type, pneumatic compression boots (PCB), sequential compression devices (SCD), or thromboembolic disease stockings (TEDS) should be considered for all inpatient spine surgeries before surgery and continued until full ambulation has been demonstrated. Chemoprophylaxis is not recommended after posterior spine approaches as they are accompanied by a definable risk of wound and bleeding complications versus the very low risk of VTE that is associated with posterior approaches. Chemoprophylaxis should be considered for long and complex surgeries, like anterior or combined anterior and posterior spine surgery, and in patients with identifiable high risk factors for thromboembolic disease. The ideal safe timing to begin prophylactic anticoagulation and the duration of therapy is unknown. The recommendation is that the timing and duration of treatment should be considered carefully on an individual case-by-case basis.

42.4 Pulmonary Complications

42.4.1 Pulmonary Embolus

Pulmonary embolism (PE) is a sudden blockage of the main pulmonary artery, or one of its principle branches, by a substance such as thrombus that has embolized from elsewhere in the body. Thrombi can be air, fat, or venous blood. Venous thromboembolism, from a DVT in the legs or the pelvis, is the most common cause of a PE [43].

42.4.1.1 Incidence

The exact prevalence and incidence of PE after spinal surgery is unknown. The incidence is estimated to range from 0 to 13 % [19, 44, 45]. Unfortunately, most of the reported occurrences of PE in the literature do not discriminate between the surgical approaches, the level of surgery, and the use of DVT prophylaxis.

42.4.1.2 Risk Factors

The major risk factor for developing a PE is the presence of a proximal lower extremity or pelvic DVT [43]. DVT and PE are a continuum termed venous thromboembolism (VTE). In spinal surgery, when mentioned in the literature, anterior approaches are associated with an increased risk of developing a PE versus posterior approaches [19]. Although DVT below the knee has less than a 1 % chance of significant embolization, they tend to propagate to the proximal lower extremity deep veins in 30–50 % of the cases [46]. From the proximal lower extremity deep venous system, the risk of a significant PE increases.

42.4.1.3 Presentation

The clinical presentations of PE are diverse and often inconsistent. Patients may present with characteristic symptoms, atypical symptoms, or even no symptoms. Symptoms and signs arise from the obstruction of arterial blood flow to the lungs and the resultant backup pressure on the right ventricle of the heart. Clinical manifestations of PE vary greatly, and it depends on the size of the thrombus, the amount of affected lung, and the presence of underlying lung or heart disease [47]. Common symptoms are shortness of breath, pleuritic chest pain, and cough. The classic clinical description of a PE patient is "air hunger." Less common symptoms are wheezing, excessive sweating, and lightheadedness.

On physical exam, the patient may frequently have tachycardia and tachypnea. In severe PE, such as in a saddle embolism, the patient may present with hypotensive shock, cardiac arrest, or sudden death. Oxygen saturation will be compromised depending on the extent of clot burden [47].

42.4.1.4 Differential Diagnosis

The signs and symptoms of PE overlap significantly with other conditions such as pneumonia, pulmonary edema, pneumothorax, COPD, coronary artery disease, congestive heart failure, myocardial ischemia or infarct, and pericarditis.

42.4.1.5 Investigation

The diagnosis of a pulmonary embolism (PE) remains a major clinical challenge. The investigation starts with the evaluation of the patient's pretest probability of PE by assessing their medical history and clinical presentation. The clinical index of suspension for a PE can be assessed by using one of the validated diagnostic scoring systems such as the modified Wells criteria [48] or the simplified revised Geneva score [49].

The clinical evaluation for a suspected PE starts with chest radiography (CXR), electrocardiography (ECG), arterial blood gas (ABG), and D-dimer measurements. Although CXR and ECG are neither sensitive nor specific for the diagnosis of PE, they are useful in ruling out other possible diagnoses that can mimic PE. CXR and ECG have findings that are nondiagnostic, but suggestive, of PE.

The most common ECG abnormality in PE is sinus tachycardia. Signs of right ventricular strain such as the S1Q3T3 complex, right bundle branch block, or anterior lead T-wave inversion are highly suggestive of PE [50]. CXR has been shown to be normal in only 12 % [51] of patients with an angiographically proven PE and may have suggestive signs such as a Hampton hump (a pleural-based wedge-shaped infiltrate), a Westermark sign (dilatation of pulmonary vessels proximal to the embolism), a knuckle sign (prominent central pulmonary artery with abrupt tapering), or elevation of the ipsilateral diaphragm [50].

An ABG will usually demonstrate a respiratory alkalosis, an increased alveolar-arterial gradient, and a variable partial arterial oxygen pressure secondary to increased alveolar dead space and a ventilation-perfusion (V/Q) mismatch (shunting) [50]. The usual ABG findings will reflect both decreased partial pressures of oxygen (pO_2) and carbon dioxide (pCO_2).

D-dimer levels may be elevated. Although D-dimer is not specific for PE, it is extremely sensitive, and if normal, it has a 99 % negative predictive value when combined with a low to moderate clinical probability index [52]. Advanced imaging

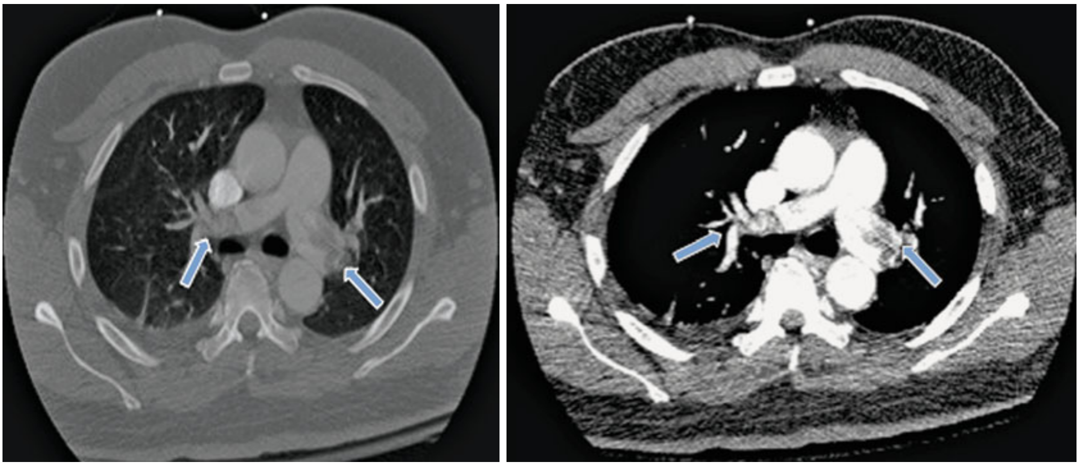


Fig. 42.2 A CT pulmonary angiogram showing a large saddle embolism at the bifurcation of the right pulmonary artery (*arrow*)

is warranted if D-dimer is high or normal with a high clinical suspicion for a PE.

The ideal imaging modality should be accurate, safe, readily available, and cost-effective. CT pulmonary angiogram has become the test of choice in the evaluation of PE because of its comparable sensitivity and specificity to pulmonary digital subtraction angiography (DSA), common availability, demonstration of alternative diagnoses, and rapid interpretation [53, 54]. DSA is the gold standard in the evaluation of PE, but it is seldom performed because of its limited availability, invasiveness, expense, and the need for experienced clinicians to perform and interpret the test [54, 55]. Ventilation-perfusion scan, a nuclear medicine study, has been also replaced by CT pulmonary angiogram in recent years except in patients with iodine contrast allergy, pregnancy, and renal diseases [54] (Fig. 42.2).

42.4.1.6 Treatment

Prompt anticoagulation therapy is the mainstay of treatment and can be lifesaving. Although there is a possibility of a disastrous epidural hematoma occurring in postoperative spine surgery patients after anticoagulation therapy for PE, simple observation of these patients is not an acceptable management. Untreated PE is associated with a very high mortality rate, ranging from 30 to 80 % compared to less than 3 % in patients treated with anticoagulation [56].

Patients with massive PE and cardiogenic shock should be treated urgently with systemic thrombolytic agents such as streptokinase [57]. Although some investigators have shown that catheter-directed therapy for the treatment of massive pulmonary embolism is a relatively safe and effective treatment for acute massive PE [58], others have not identified an advantage of catheter-directed thrombolytic therapy over systemic thrombolytic therapy [59] (Fig. 42.3). Due to its high mortality rate, pulmonary thrombectomy is usually reserved for patients with massive PE and cardiogenic shock who failed thrombolysis [60].

A stable patient with a sub-massive PE and no contraindications for anticoagulation should be started simultaneously on heparin, LMWH, or fondaparinux for 5 days and VKA (Coumadin). The INR goal is between 2 and 3 and should be increased to a range of 2.5–3.5 if another PE occurs while on VKA. Pregnant women and patients with an underlying malignancy should be treated with LMWH instead of VKA [61]. Treatment duration is usually 3–6 months, or lifelong, if it is a first unprovoked or recurrent PE. IVC filter placement is indicated for patients in whom anticoagulation is contraindicated or ineffective and is recommended for prevention of possible new emboli to enter the pulmonary circulation in anticoagulated patients [62].

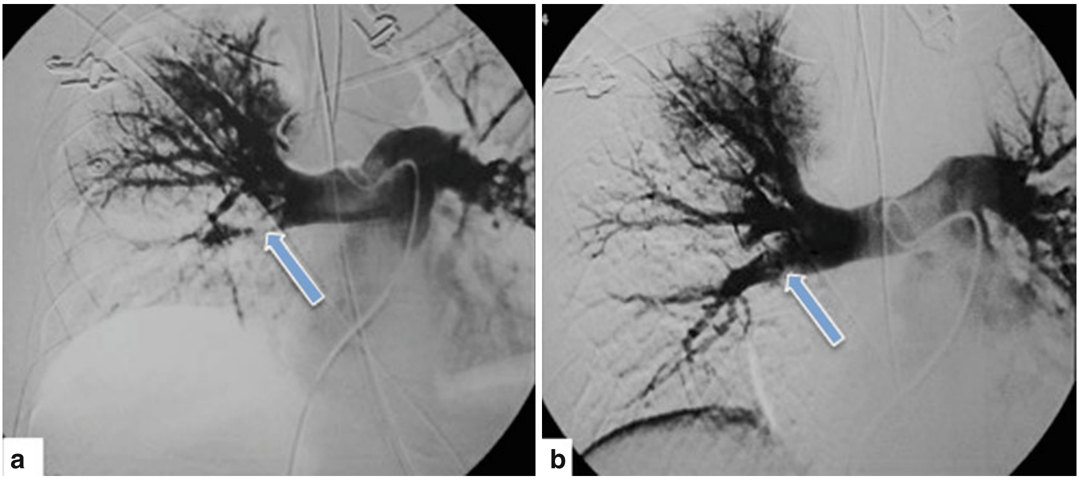


Fig. 42.3 Pulmonary angiogram showing a massive clot in the right pulmonary artery before (**a**) [*arrow*] and after (**b**) [*arrow*] catheter-directed chemical thrombolysis

42.4.1.7 Prophylaxis

DVT and PE are a continuum termed venous thromboembolism (VTE); therefore, any risk factor for DVT will increase the risk of a PE, and the prevention of the latter is directly related to the decrease incidence of DVT. The recommendation for DVT prophylaxis outlined earlier is appropriate for the prevention of PE.

42.4.2 Pneumonia

Pneumonia is an inflammation of the lung parenchyma, especially of the alveoli, usually caused by a microbial organism.

42.4.2.1 Incidence

The incidence of perioperative pulmonary complications, in general, and pneumonia, in particular, after spinal surgery varies widely from 7 to 64 % [63–66] and 1.4 to 12 % [67–70], respectively. It depends on the patient's related determinants (age and comorbidities [especially pulmonary diseases]), the underlying pathology for surgery (trauma, scoliosis, degenerative diseases), the surgical approach (anterior, posterior, lateral, or combined), and the level of surgery (cervical, thoracic, lumbar). Pneumonia is the second most common nosocomial infection after UTI. It is the most common complication in patients with SCI and the leading cause of death among this group irrespective of patient age [13, 71].

42.4.2.2 Risk Factors

Age, smoking, obesity, scoliosis with abnormal pulmonary function tests (PFTs), asthma, COPD, emphysema, prolonged endotracheal intubation, mechanical ventilation, cervical injury, polytrauma, aspiration, and pain are major predisposing factors for developing postoperative pneumonia after spinal surgery. Preoperative and postoperative immobility can lead to atelectasis and an inability to clear respiratory secretions then to pneumonia. The risk is much higher in SCI patients above the C4 level where there is loss of the diaphragmatic function, secondary to phrenic nerve injury, and loss of cough reflexes secondary to the denervation of the intercostal muscles and the accessory muscles of respiration [72]. The risk of aspiration pneumonia is greatly increased when there is a loss of airway reflexes.

42.4.2.3 Presentation

Patients with infectious pneumonia typically present with fever, a productive cough with purulent sputum, shortness of breath, and chest pain upon deep inspiration. In the elderly population, confusion may be the most prominent symptom [73]. Although these clinical findings are helpful in suspecting pneumonia, they are not as useful in ventilator-dependent patient. It has been demonstrated that only 30–40 % of ventilator-dependent patients, with positive clinical findings, have an actual infectious pneumonia on postmortem examination [74].

Physical examination may reveal tachypnea, tachycardia, hypotension, and low oxygen saturation. Upon chest auscultation, bronchial breathing, rales, and crackles may be heard over the affected lung area. Dullness to percussion over the affected lung may also be detected.

42.4.2.4 Differential Diagnosis

Bacterial pneumonia accounts for only one-third of all pulmonary infiltrations. Several other conditions like pulmonary edema, bronchiectasis, ARDS, COPD, emphysema, asthma, lung cancer, and pulmonary embolus have presentations similar to pneumonia, and they account for most of the pulmonary infiltrates in ICU patients [75, 76].

42.4.2.5 Investigation

The diagnosis of pneumonia starts with a high index of suspicion. It should be suspected in any patient when a new pulmonary infiltrate on chest X-ray is present (Fig. 42.4) with two of the following findings: fever, leukocytosis, and purulent tracheal secretions [77]. The definitive diagnosis requires the isolation of the pathogen from the respiratory tract. Respiratory secretions for cultures can be obtained by having the patient expectorate or inducing a cough with a tracheal aspirate or a more invasive bronchoalveolar lavage. Although cultures from the tracheal aspirate are more likely to produce false positive results, and therefore, unnecessary excessive use of antibiotics, it is still the preferred method for obtaining pulmonary cultures. It has been shown that there are no benefits when more invasive methods are used for collecting pulmonary aspirates for cultures [78]. Whenever possible, cultures should be obtained before the initiation of any antibiotics.

42.4.2.6 Treatment

As soon as there is clinical evidence of a suspected pneumonia, empiric antibiotic treatment should be initiated once a specimen is collected

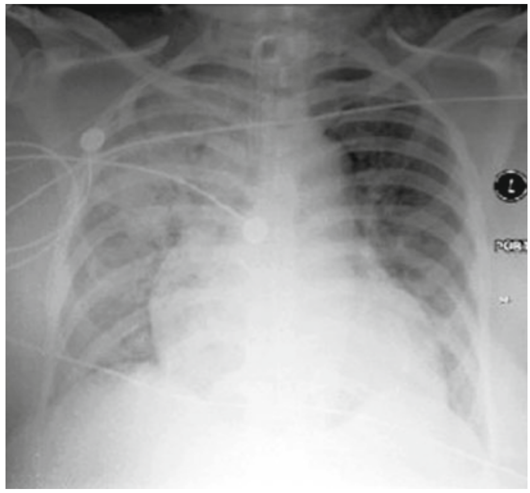


Fig. 42.4 Portable chest X-ray demonstrating right lung consolidation with increased opacity in the right lower lobe more than the right upper lobe. Left lung is clear

from the respiratory tract for gram stain and culture. Empiric antibiotic should be started even before the result of the respiratory specimen is available if the patient is immunocompromised, has sepsis, or is in septic shock. The choice of empiric antibiotic is dictated by the state of the patient's immune system, the patient's location (floor versus ICU), the patient's dependence on the ventilator, and the patient's likelihood of colonization by a pathogen. Empiric treatment should be adjusted according to the culture and sensitivity results and should be continued for 14–21 days [79]. Empiric treatment should be stopped if cultures are negative [80].

42.4.2.7 Prophylaxis

There are many ways to decrease the incidence of perioperative pulmonary complications. It is recommended that patients stop smoking at least 8 weeks prior to elective surgery [81]. Protection of the airway to prevent aspiration is essential in trauma and SCI patients. A good pain control regimen, combined with incentive spirometry and aggressive pulmonary toilet, should be immediately initiated postoperatively. Nasotracheal suctioning, chest physiotherapy, and frequent body rotation by log roll or through an oscillatory bed are all pulmonary hygiene methods that can be used to prevent pneumonia.

In critically ill ICU patients, for the prevention of gastric stress ulcers, it is recommended to use sucralfate instead of antacids or histamine-2 receptor antagonists since it has been shown that sucralfate is associated with a lower incidence of nosocomial pneumonia [82]. In the mechanically ventilated patients, especially those who are unable to cough and clear their own respiratory secretions, mucolytic agents such as acetylcysteine, and expectorants such as guaifenesin, should be started early with frequent suctioning. The application of topical antimicrobials such as methylcellulose paste to the oral mucosa to prevent colonization is associated with a decrease in the incidence of ventilator-associated pneumonia by about 67 % [83]. Early enteral feeding in these patients has also been associated with a reduction in the incidence of pneumonia. In the non-intubated patients, early aggressive incentive spirometry, early mobilization, and, when permitted, aggressive physical and occupational therapy are helpful in preventing pulmonary complications.

42.5 Cardiac Complications

Cardiac events are one of the most critical perioperative complications. Myocardial ischemia, myocardial infarction, and cardiac dysrhythmias are the most common and dangerous cardiac complications after spinal surgery. Myocardial ischemia, or angina pectoris, occurs when the myocardium receives insufficient blood flow secondary to narrowed atherosclerotic coronary arteries, coronary artery vasospasm, or hypotension. Myocardial infarction (MI) is the interruption of blood supply to the myocardium, and its subsequent cell death, secondary to sudden blockage of coronary arteries by an atherosclerotic embolus. Cardiac dysrhythmias encompass a large heterogeneous group of cardiac events in which there is an abnormal electrical activity of the heart.

42.5.1 Incidence

The incidence of cardiac complication after spine surgery varies widely in the literature, and it depends on the patient age, gender, comorbidities, type and duration of surgery performed, and the type of anesthesia. The overall reported incidence of cardiac events ranges from 1.9 to 13 % [65, 66, 68, 70, 84, 85]. The MI incidence ranges from 0.1 to 6 % [12, 67, 86–89]. Cardiac dysrhythmias are the least reported; they are usually reported under "general cardiac" events, and their incidence ranges from 2.4 to 15 % [49, 69, 90, 91].

42.5.2 Risk Factors

Destabilization of a vulnerable atherosclerotic plaque and an imbalance between myocardial oxygen supply and demand are the two major underlying causes of perioperative MI [92–94]. Risk factors for cardiac complications are generally the same risk factors for atherosclerosis. The major ones are, but not limited to, diabetes mellitus, tobacco smoking, age >55 years for males and >65 for females, dyslipidemias, family history of premature cardiac disease, obesity, hypertension, and sedentary lifestyle. Alike any surgical procedure, postoperative cardiac complications after spine surgery are associated with postoperative anemia secondary to blood loss, hypothermia, and pain. They all cause an over activation of the sympathetic system which in turn increases the myocardial workload and oxygen consumption against decreased blood and oxygen delivery [93]

Structural heart diseases are a major risk factor for perioperative cardiac dysrhythmias, and the perioperative transient imbalances in serum electrolytes, oxygenation, ventilation, PH, or temperature are usually the initiating factors [95].

42.5.3 Presentation

Cardiac ischemia may be asymptomatic or may present with chest discomfort rather than pain. Patient often described a squeezing pressure, heaviness, tightness, or a burning sensation that originates in the chest then radiates to the left arm, back, neck, left jaw, or the epigastric area. They may even describe it as an elephant sitting on top of their chest. Shortness of breath (SOB), sweating, and nausea may also be present.

Although some patients with an MI will have no symptoms and others may present with sudden death, classically, they present with sudden chest pain radiating to the left arm with SOB, nausea, vomiting, sweating, and a sense of impending doom. Silent MI, typically not accompanied by pain or other symptoms, is most often seen in elderly, heart transplant, or diabetes mellitus patients.

Patients with cardiac dysrhythmias have a wide range of presentations. They may have no symptoms or merely an appreciation of an abnormal heartbeat. Some may present with lightheadedness, dizziness, syncope, near syncope, or sudden death.

Physical examination findings can vary, from a patient laying comfortably in bed, with normal examination results, to another who may be in severe acute distress. They may have dyspnea, wheezing, diaphoresis, pallor, hypotension, hypertension, tachycardia, bradycardia, or even asystole.

42.5.4 Differential Diagnosis

The postoperative cardiac complications after spinal surgery should always be differentiated from other catastrophic event such as pulmonary embolism. Aortic dissection, pericardial effusion, and cardiac tamponade must be ruled out especially in trauma patients. Tension pneumothorax can be seen in trauma patients and in patients who underwent a thoracic spinal surgery. Esophageal rupture should be differentiated from MI after anterior cervical approaches.

42.5.5 Investigation

Myocardial infarction is defined as myocardial cell injury or necrosis resulting from significant and sustained myocardial ischemia [96]. According to the World Health Organization (WHO) [96], MI is diagnosed when any one of the following criteria is met: (1) rise and/or fall of serum cardiac biomarkers (troponin and creatine kinase-MB fraction) with at least one of the findings of myocardial ischemia such as clinical symptoms of ischemia, EKG changes suggestive of ischemia, pathological Q waves in the EKG, or imaging findings demonstrating loss of viable myocardium or a new wall motion abnormality; (2) sudden unexpected death secondary to cardiac arrest with symptoms suggestive of myocardial ischemia; or (3) histopathological findings of an acute MI in autopsy. The MI workup should start with a good evaluation of the patient's complaints and a thorough physical exam. Serum cardiac biomarkers, EKG, echocardiogram, and coronary angiogram help to confirm the diagnosis. Routine blood tests and chest XR may help in assessing the precipitating causes, the exacerbating factors, and complications of the MI.

The diagnosis of cardiac dysrhythmias is initiated by auscultation of the heart, examination of the peripheral pulses, placement of the patient under continuous telemetry, and laboratory evaluation of ABG and serum electrolytes. An EKG is used to confirm and identify the specific dysrhythmias [95]. A consultation with a cardiologist should be initiated after the initial workup for further management.

42.5.6 Treatment

In general, the initial care of a patient with a suspected MI or cardiac dysrhythmia starts with the assessment of the patient hemodynamic state and treating any airway, breathing, or circulation abnormalities (ABCs). After confirmation of the diagnosis of an MI, the simultaneous administration of morphine to relieve myocardial ischemic pain, oxygen to augment myocardial tissue oxygenation, nitrates to increase myocardial tissue perfusion, aspirin to reduce platelet aggregation, and a beta blocker to reduce myocardial work load should be initiated [97]. Recently, it has been shown that use of supplement oxygen may be harmful, and its use has been discouraged especially in uncomplicated MI patients [98, 99].

Any electrolyte derangement should be corrected. In perioperative spine patients, the spine surgeon and the cardiologist should evaluate the risk-benefit before the use of any antiplatelet agents (including aspirin), anticoagulants, or fibrinolytic agents. The management of perioperative cardiac dysrhythmias is more complex and warrants early involvement of a cardiologist. Depending whether the patient is stable or unstable and the type of dysrhythmia, the treatment may include physical maneuvers such as vagal nerve/carotid massage, medication, electrical conversion, and/or myocardial ablation. Potassium and magnesium depletion has been associated with perioperative dysrhythmias, especially atrial fibrillation [100], and should be corrected immediately.

42.5.7 Prophylaxis

Preoperative cardiac risk stratification [101, 102], intraoperative myocardial ischemia reduction, and perioperative surveillance help in predicting, diagnosing, and treating perioperative cardiac complications [101]. Preoperatively, the authors routinely get a cardiac clearance and cardiac optimization, when applicable, for patients with high risk for cardiac complications. Postoperatively, EKG and serum cardiac biomarkers are obtained for long complicated surgeries, excessive intraoperative blood loss, elderly patients, and any patient with a history of cardiac disease or diabetes mellitus.

Although the use of beta blockers has been considered the standard of care to reduce pre- and postoperative myocardial ischemia and infarction [103], their use has become controversial since it has been shown that they may increase the risk of death and stroke [104].

42.6 Gastrointestinal (GI) Complications

Postoperative abdominal distension is caused by impairment of intestinal content transit secondary to a mechanical or a functional deficiency of enteric propulsion. Adynamic ileus and its variant, acute colonic pseudoobstruction, are well-known gastrointestinal complications of spine surgery. Adynamic ileus, also called paralytic ileus or postoperative ileus (POI), is a disruption of the normal propulsive ability of the GI tract in the absence of a mechanical obstruction causing an impairment of intestinal content transit. Acute colonic pseudoobstruction (ACPO), also known as Ogilvie's syndrome, is a variant of POI characterized by severe functional impairment of colonic content transit and massive colonic dilatation without any mechanical obstruction.

42.6.1 Incidence

The reported incidence of POI after spinal surgery varies from 0.6 to 5.6 % [86, 105–107]. The highest rate has been reported after lumbar spine surgery in comparison to thoracic or cervical. Among the lumbar spine surgical approaches, ALIF is associated with the highest incidence [105, 108] especially if the transperitoneal approach is used versus the retroperitoneal approach [108], or if the assistance of an access surgeon was used for the exposure of the lumbar spine [109]. Classically, ACPO has been considered as a rare syndrome after spinal surgery; in fact, the majority of the reported incidence is limited to case reports and small series of patients [110–112].

42.6.2 Risk Factors

The combination of three major precipitating mechanisms, neurogenic, inflammatory, and pharmacological, has been identified in the literature as the triad of dysmotility [113, 114]. The neurogenic component stems from the pain-induced neuronal reflexes that result in the hyperactivity of the sympathetic system causing GI dysmotility through endogenous neuromuscular inhibitors and endogenous opioids. Surgical manipulations of the bowel, as may occur in ALIF surgeries, trigger dormant macrophages and mast cells to release proinflammatory mediators that inhibit GI motility [114, 115]. Opioids are a major pharmacological inhibitor of GI motility postoperatively. They decrease GI secretions and inhibit peristalsis. This effect is proportional with higher doses and is augmented in patients with opioid tolerance since the GI tract does not develop tolerance [114, 115]. Spinal cord injury is a major risk factor for paralytic ileus especially in those with injuries above the T5 level [116]. Diabetes mellitus, hypothyroidism, and electrolyte imbalances contribute to postoperative ileus [114, 115].

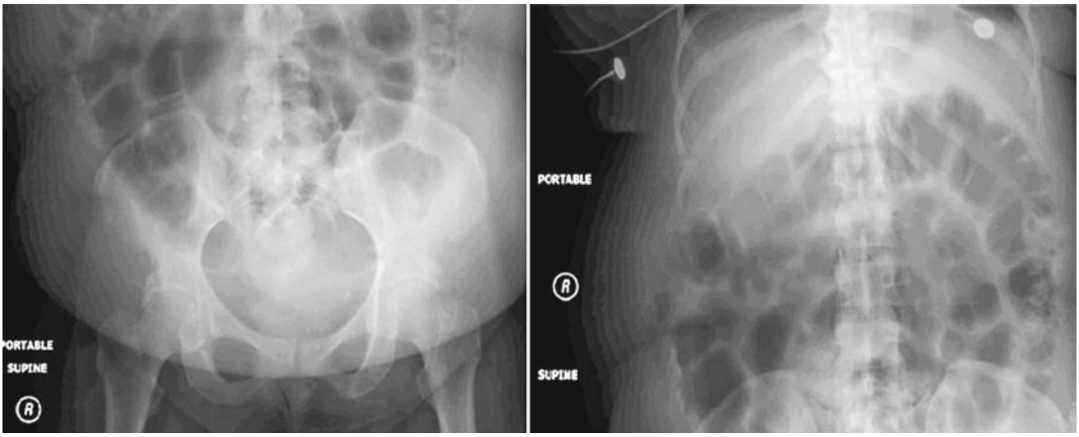


Fig. 42.5 Abdominal X-ray demonstrating several dilated loops of small and large bowel representing an ileus

ACPO has been attributed to an imbalance between sympathetic and parasympathetic colonic innervations [110, 111]. It is associated with increased age, male gender, sepsis, diabetes mellitus, chronic alcoholism, uremia, narcotic addiction, congestive heart failure, trauma, laxative abuse, bed rest, narcotics, tricyclic antidepressants, steroids, phenothiazines, antiparkinsonian drugs, H2 blockers, calcium channel blockers, and electrolyte abnormalities [114, 115].

42.6.3 Presentation

Patients with POI may complain of diffuse abdominal discomfort, moderate nausea and vomiting, oral intake intolerance, constipation, lack of flatulence, excessive belching, and mild shortness of breath. Although the symptoms of ACPO are similar to that of POI, they are more dramatic and may even present with paradoxical diarrhea. Massive painful abdominal distension is the hallmark finding of ACPO.

Physical examination of a patient with POI reveals a distended, mildly tender, tympanic abdomen with hypoactive or absent bowel sounds. They may also present with signs of dehydration such as tachycardia or orthostatic hypotension. The presence of a SCI may confound the diagnosis of POI as pain is not a useful finding. In addition to these physical findings, patients with ACPO may have normal or hyperactive bowel sounds and may present with fever if bowel ischemia or perforation is present.

42.6.4 Differential Diagnosis

ACPO is a variant of POI, and they should be differentiated from each other, as they require different treatment strategies. ACPO is characterized by its significant abdominal distension secondary to right colonic dilatation and the lack of response to POI treatment measures. POI and ACPO should be distinguished from a wide range of GI dysmotility disorders.

POI must be differentiated from mechanical small bowel obstruction (SBO) where patients tend to be more ill, have more severe colicky abdominal pain, and more often have obstipation. Similarly, ACPO must be differentiated from mechanical large bowel obstruction (LBO). In addition, ACPO must be differentiated from toxic megacolon, ischemic colitis, and cecal volvulus through radiographic studies or colonoscopy.

42.6.5 Investigation

The diagnosis of POI and ACPO is based on signs, symptoms, laboratory tests, and abdominal radiographic imaging (Fig. 42.5). Laboratory tests should include serum electrolytes, complete blood count, creatinine, blood urea nitrogen, amylase, lipase, amylase, and liver function tests.

Although abdominal plain X-rays may reveal diffuse gas-filled distended small bowels with air-fluid levels, it is not specific for POI. CT scans are the gold standard radiographic examinations for the diagnosis of POI, as they will differentiate it from SBO in more than 98 % of the cases [117]. Oral contrast passage into the colon is completely blocked in SBO compared to POI where it is only delayed. Leukocytosis is present in 25 % of patients with uncomplicated ACPO and nearly 100 % if colonic ischemia or perforation is present [118].

Proximal colonic dilatation in ACPO is easily seen in the plain X-ray of the abdomen, but it may be difficult to distinguish it from cecal volvulus or LBO. Barium or water-soluble contrast enemas, colonoscopy, abdominal CT scans, or abdominal MRI can help to differentiate these entities. Contrast enemas and colonoscopy may also be therapeutic. Abdominal CT scans are 96 % sensitive and 93 % specific for ACPO diagnosis [119]. The cecal diameter should be measured to predict potential cecal perforation and to initiate treatment. Normal cecal diameter is usually less than 8.5 cm, and intervention is warranted if it reaches more than 9 cm [110]. It has been shown that a cecal diameter less than 12 cm is less likely to cause perforation, whereas a diameter more than 14 cm has a higher incidence of perforation [111].

42.6.6 Treatment

The treatment of POI is largely supportive and starts with restriction of oral intake, intravenous fluid resuscitation, and correction of electrolyte unbalances. Causative pathology, if any, should be treated, and any dysmotility drugs should be discontinued immediately. Although, nothing by mouth should be continued until either flatus or stool is passed, it has been suggested recently that early enteral feeding may shorten the POI duration [114, 115]. Gum chewing, early ambulation, bowel stimulants, enemas, and laxatives can be used to promote motility. Metoclopramide, a dopamine antagonist, and erythromycin by mouth are routinely used to promote GI motility, but they both failed to show, in multiple trials, any proven improvement of bowel function when compared to placebo [114, 115]. Nasogastric suctioning for GI decompression is not beneficial; in fact, it is associated with prolongation of ileus and is only reserved for patients with intractable vomiting or severe gastric dilatation [114, 115]. Normal POI duration is unclear, but many surgeons suggest that bowel function should return after 3–5 days and total parental nutrition should be considered if longer duration is anticipated especially in SCI patients who are prone to rapid catabolism [115].

Although POI is not a life-threatening condition, ACPO, on the other hand, can be. As a result, early recognition, differentiation, and prompt appropriate treatment of the latter will prevent significant morbidity and mortality secondary to cecal ischemia and perforation. Spontaneous perforations can occur in about 3 % of patients with ACPO leading to a 50 % mortality rate [120]. Treatment of ACPO starts with conservative and supportive management similar to POI treatment. Laxatives and enemas should be avoided as they can cause accumulation of fluid in the sluggish bowel and increases the risk of colonic perforation, respectively [114, 115]. Nasogastric tube is of no benefit since it has inadequate efficacy in colonic decompression [114, 115]. Placement of a rectal tube will help decompress the distal colon but has very limited usefulness in decompressing the proximal colon [114, 115]. Conservative management should be continued for at least 3 days as long as there are no signs or symptoms of bowel ischemia or perforation and serial abdominal XR every 12 h showing a cecal diameter less than 12 cm. Most cases resolve after 4 days of conservative therapy [114, 115]. Patients who failed conservative measurements can be given rapid intravenous neostigmine infusion, an acetylcholinesterase inhibitor that has a cure rate of 86–96 % in the treatment of refractory ileus in spine surgery patients [121, 122]. Neostigmine should be administered under continued telemetry and may be repeated in 4 h if no response or recurrence. It is contraindicated if the patient has bradycardia, bronchospasm, or a suspected LBO. Colonoscopic decompression is an option if pharmacological treatment fails or is contraindicated. Although it is associated with a success rate of 70–80 % [118, 123], it has a higher complications rate secondary to an unprepared distended colon. Because it is associated with high morbidity and mortality rates [118], invasive surgical options, such as percutaneous transperitoneal cecostomy or laparotomy with tube cecostomy, should be used as the last resort and should be reserved for the sicker patient who has failed medical and colonoscopic treatments or has evidence of colonic ischemia or perforation [118].

42.6.7 Prophylaxis

Preventive measures include minimizing the triad of dysmotility. Avoiding narcotics, early ambulation, and early by mouth intake postoperatively significantly decrease the duration of the ileus. Serial abdominal exams to monitor for signs of ischemia or LBO, serial abdominal X-rays to monitor for cecal diameter, serial labs to monitor for leukocytosis and metabolic acidosis, and serial temperature checks to monitor for pyrexia are necessary measures for early diagnosis and treatment of any complications from the ileus.

42.7 Genitourinary Complications

Three major genitourinary complications are encountered perioperatively in the spine surgery patients: urinary tract infection (UTI), urinary retention, and retrograde ejaculation.

42.7.1 UTI

Urinary tract infection is an infection of the urinary tract and is named for the anatomical part involved, such as urethritis (urethra), cystitis (bladder), ureteritis (ureter), and pyelonephritis (kidney).

42.7.1.1 Incidence

UTI is a major nosocomial infection especially in ICU patients with an indwelling urethral catheter where it accounts for more than 30 % of ICU-acquired infections [124]. The incidence of UTI in the perioperative spine surgery patients ranges widely from 4 to 34 % [64, 125–127] and depends on sex, age, BMI, DM, length of surgery, and duration of indwelling urinary catheterization. This incidence is reported to be much less, in the vicinity of 0.5 %, in minimally invasive spine surgery [128].

42.7.1.2 Risk Factors

Adult women are more prone to UTI than men because of a shorter urethra that is also much closer to the anus. Among elderly, this difference is nonexistent. Older men develop enlargement of their prostate, which can cause urethral obstruction leading to urinary retention, which will increase the risk of developing a UTI. Urinary catheterization is a major risk factor for developing a UTI, and the presence of an indwelling urethral catheter is associated with a 4–7 % increased risk [129]. Other risk factors include DM [130, 131], family history, immunodeficiency, and urinary retention secondary to SCI or excessive narcotic use [131].

42.7.1.3 Presentation

The clinical presentation of UTI varies with age and the affected anatomical part of the urinary tract. In adults, the most common symptoms and signs for lower UTI (cystitis) are urgency, frequency, dysuria, low-grade fever, cloudy foul smelling urine, and suprapubic tenderness. Hematuria may be present at times. In elderly patients, the most common presentations, besides new or increased incontinence, are nonspecific signs such as altered mental status, delirium, and lethargy, or in extreme cases, sepsis or septic shock [132]. In addition to the above-mentioned signs and symptoms, patients with pyelonephritis may also present with high-grade fever, chills, nausea, vomiting, and flank pain [132]. The presence of an indwelling urinary catheter eliminates the discriminatory value of many of the clinical findings, leaving only the suprapubic tenderness and fever as clues for suspecting the presence of UTI.

42.7.1.4 Differential Diagnosis

Other diagnoses should be considered when there is clinical suspicion of a UTI in view of negative urinalysis and culture. Prostatitis as well as sexually transmitted diseases may present with dysuria, frequency, and urgency. Asymptomatic bacteriuria should be considered when there is bacteriuria in the absence of signs and symptoms of an infection.

42.7.1.5 Investigation

In uncomplicated, straightforward cases, a diagnosis may be obtained based on the signs and symptoms alone without any further laboratory confirmation. In complicated cases, the diagnosis must be confirmed by urinalysis to look for nitrates (a measure of bacteriuria) or leukocyte esterase (a measure of pyuria); urine microscopy to look for red blood cells, white blood cells, or bacteria; and urine culture to isolate the organism. The presence of either nitrates or leukocyte esterase is predictive of UTI with 67–100 % sensitivity and 67–98 % specificity. The specificity improves to 98–100 % when both are positive [132]. Nitrate usefulness is limited when the infecting pathogen is a non-nitrate-reducing bacteria such as Pseudomonas sp. or Enterococci. Leukocyte esterase is useless as well when there is high level of protein, glucose, or bacterial contamination of the urine sample [132].

Since 50 % and nearly 100 % of the patients with indwelling urinary catheters will have positive urine cultures after 5 and 30 days of urethral catheterization, respectively, the diagnosis of a UTI in this group of patients cannot be based on urine culture alone. A combination of positive clinical findings, positive urinalysis, and positive urine cultures must be present in order to make the diagnosis [133].

42.7.1.6 Treatment

According to the Infectious Diseases Society of America, uncomplicated cystitis can be treated, based on signs and symptoms alone, with oral nitrofurantoin monohydrate (Macrobid) twice daily for 5 days or oral trimethoprim-sulfamethoxazole (Bactrim DS) twice daily for 3 days in communities where the rate of resistance is less than 10 and 20 %, respectively [134]. Fluoroquinolones are the treatment of choice, for 3 days, in communities with high resistance rates [134].

There are very limited data regarding the initial treatment of complicated cystitis, so an empiric broad spectrum antibiotic therapy should be initiated as early as possible especially in immunocompromised patients, patients with prosthetic heart valves, and patients with multiple organ failure. The Gram stain should initially guide the antibiotic selection that later on should be tailored based on the urine culture and sensitivity and continued for 7–14 days. Imipenem and ciprofloxacin are good empiric coverage for Gram-negative bacilli and Gram-positive cocci, respectively.

For upper UTI, fluoroquinolones are the standard first-line treatments for 7 days followed by Bactrim DS for 14 days as a second-line agent [134].

42.7.1.7 Prophylaxis

An indwelling urinary catheter is a major risk factor for developing UTIs. The amount of bacteria rises once it gains access to the urinary tract within 24–48 h [135] and continues to rise at a rate of 5 % per day [136]. The key to preventing postoperative UTI is to limit the use of indwelling urinary catheters and, once used, to decrease the duration of their use [137]. Other measures to help prevent postoperative UTIs following spine surgeries are tight blood sugar control and judicious use of narcotics.

42.7.2 Retrograde Ejaculation

Retrograde ejaculation occurs when semen is redirected to the urinary bladder instead of being ejaculated through the urethra secondary to the inability of the internal vesical sphincter, the muscles at the base of the bladder, to contract during ejaculation. The internal vesical sphincter is innervated by the superior hypogastric plexus, a fine thin plexus of the sympathetic chain that is located in the retroperitoneal space overlying the lumbosacral area. Damage to this plexus during anterior exposure of the lumbosacral area causes denervation of the internal vesical sphincter resulting in retrograde ejaculation.

42.7.2.1 Incidence

The reported incidence of retrograde ejaculation following ALIF varies wildly and ranges from 0.1 to 24 % [86, 105]. Incidence as high as 45 % has been reported when a laparoscopic approach is used to access the anterior lumbar spine [138]. This diagnosis can be difficult to make in men who are not attempting to achieve fertilization.

42.7.2.2 Risk Factors

Many factors have been reported in the literature, when the ALIF surgical option is used, to be associated with an increased risk of retrograde ejaculation. The risk is increased with laparoscopic approaches versus standard open laparotomy, transperitoneal approach versus standard retroperitoneal, the left retroperitoneal approach versus right retroperitoneal, and the use of an access vascular surgeon versus none. Recently, it has been reported that the use of recombinant human bone morphogenetic protein-2 (rhBMP2), with an ALIF procedure, is associated with a four to fivefold higher incidence of retrograde ejaculation [139].

42.7.2.3 Presentation

Some patients will complain of an inability to ejaculate, and others may complain that their orgasms are not as pleasurable as they used to be. Inability to procreate is a major complaint. Physical exam is completely normal.

42.7.2.4 Investigation

Diagnosis is performed by urinalysis of a urine specimen that is collected after ejaculation. The specimen will contain an abnormal level of sperm if retrograde ejaculation is present.

42.7.2.5 Treatment

Treatment is aimed toward tightening the bladder neck muscles to prevent the retrograde flow of semen to the bladder. Imipramine, chlorphenamine, ephedrine, and phenylpropanolamine have been used to treat cases of mild nerve damage with some success [140].

42.7.2.6 Prophylaxis

Patient selection is critical when using the ALIF as a surgical option. This procedure should be avoided in males of reproductive age. If there is no alternative for these patients, it is recommended to advise the patients who plan to have children to store sperm before undergoing this procedure. It has been suggested that the incidence of retrograde ejaculation could be eliminated by reclining the presacral tissue and the nerve plexus from right to left through a right anterior approach instead of a left anterior approach [141].

42.7.3 Urinary Retention

Postoperative urinary retention (POUR) is the inability to pass any urine, ischuria, in the presence of a full bladder after a surgical procedure.

42.7.3.1 Incidence

The incidence of POUR varies widely in the literature, from 4 to 29 % [142], depending on the patient age, gender, comorbidities, type and duration of surgery performed, type of anesthesia, use of indwelling urinary catheter, and the use of narcotics. The reported incidence of POUR after spinal surgery is low 0.3–2 % [128, 143], but it is believed to be underreported and that it does not differ greatly from the general incidence of POUR.

42.7.3.2 Risk Factors

Gender plays a significant role as a risk factor in the elderly patients but not so much in the younger patients. Although some studies have reported that young females, less than 40 years old, are more predisposed to POUR than males [144], many other studies have shown no difference [145, 146]. In general, elderly are more prone to POUR secondary to deceased detrusor muscle function with advanced age [142]. Postoperative pain is associated with increase sympathetic outflow which promotes detrusor muscle relaxation and bladder sphincter contraction [142]. Opioid narcotics promote bladder filling and decrease bladder sensitivity by inhibiting the parasympathetic outflow [142]. High pre- and intraoperative intravenous fluid administration may cause bladder overdistention thus decreasing the contractility of the detrusor muscles [142]. General anesthetics cause bladder atony by disrupting the autonomic regulation of the detrusor muscle tone. Some anesthetics, such as halothane, cause great increase in the bladder capacity and decrease its response to stimulus [147]. Length of surgery is associated with POUR. Longer operations necessitate higher fluid and opiate administration thus increasing the risk of POUR [144]. Diabetes mellitus is associated with decrease bladder sensation causing increased filling capacity and decreased contractility [142, 148].

42.7.3.3 Presentation

In early stages, most patients have no symptoms. As the bladder volume increases, they may complain of abdominal discomfort, pain and distension, inability to urinate, urgency, shivering, diaphoresis, and headache. Patients with SCI will usually have no symptoms.

Upon physical examination, a distended abdomen may be noted, and the full bladder can be palpable in the suprapubic area. A digital rectal exam may reveal an enlarged prostate in male patients. A post-void catheterization of the bladder will show a post-void residual (PVR) greater than 50 cc. More recently, the use of ultrasound has been shown to accurately assess PVRs in a noninvasive manner [149]. In extreme cases, the painful stimuli from an overdistended bladder may cause hypertension, hypotension, cardiac dysrhythmias, bradycardia, or even asystole [150]. This is particularly problematic in SCI patients.

42.7.3.4 Investigation

Diagnosis can be established by one of the three methods: physical examination and history, ultrasonographic bladder volume measurement, or post-void residual urine measurement. History of lower abdominal pain and discomfort has been historically used as hallmark indicators for early POUR, but it is unhelpful in patients who cannot feel or verbally communicate these symptoms such as SCI, stroke, or sedated patients. Physical examination by palpation and percussion of the suprapubic area is used for diagnosis, but it does not provide information about the residual urinary volume. Ultrasound bladder scanning is a good noninvasive diagnostic method for POUR. It permits rapid and accurate measurement of bladder volume, which helps distinguish between a failure to void secondary to POUR versus under resuscitation. In the literature, there is no standard definition of POUR [151]. Normal bladder capacity in an adult ranges from 400 to 500 cc [152], and the authors consider any volume above 600 cc as diagnostic for POUR. Post-void residual urine is the volume of urine in the bladder, obtained by straight catheterization within 20 min after voiding. A PVR of more than 200 cc or more than 25 % of the total bladder capacity, voided plus residual volume, is considered diagnostic for POUR by the authors.

42.7.3.5 Treatment

The initial treatment is bladder catheterization to prevent overdistention of the bladder and long-term damage to the detrusor muscle. Although this is the standard of treatment for POUR, it is unknown which patients will benefit from indwelling catheterization versus clean intermittent catheterization. Phenoxybenzamine, an irreversible alpha antagonist, has been used to treat POUR. It has been shown to reduce the time to initial void and to decrease the incidence of bladder catheterization. It decreases urethral outflow resistance and enhances intravesical pressure [153, 154]

42.7.3.6 Prophylaxis

Prevention of POUR starts with the identification of patients with high risk factors. Low intraoperative IVF volume, shorter procedures, choice of anesthetic, and reduction of opiate usage are associated with reduced incidence of POUR. Prophylactic use of phenoxybenzamine has also been shown to decrease the incidence of POUR [142].

42.8 Decubitus Ulcers

Decubitus ulcers or pressure ulcers (PU) are a localized damage to the skin and the underlying tissue that is caused by shear force, unrelieved

Table 42.2 Decubitus ulcer staging

Stage	Description
Stage 1	The most superficial. A localized, discolored, non-blanchable, erythematous intact skin
Stage 2	Epidermal damage. Blistering or abrasion of the skin
Stage 3	Full thickness skin damage. Skin breakdown with visible subcutaneous fat
Stage 4	Full thickness tissue loss with exposed bone, tendon, or muscle

pressure, or friction of any dependent area of the body, especially over bony prominences such as the sacrum, skull, elbows, and ankles. Pressure ulcers are classified into four stages depending on the depth and tissue involvement (Table 42.2). Appropriate staging of pressure ulcers helps in the management and prognosis of the ulcer.

42.8.1 Incidence

Pressure ulcers are a serious medical complication that affect the lives of many spine surgery patients, especially those with SCI, and may ultimately result in death through fatal septic infections [155]. It is one of the leading iatrogenic causes of death in the developed countries. The reported annual prevalence of PU in SCI patients is between 30 and 50 %. The overall incidence in non-SCI patients after spinal surgery is low, 1–2 % [64, 127], except in the elderly patients, over 65, where it is up to 12 % [156].

42.8.2 Risk Factors

Especially among elderly and SCI patients, immobility and limited mobility are the major risk factors for developing PU. Many medical conditions such as diabetes mellitus, peripheral vascular disease, peripheral neuropathy, smoking, renal disease, and malnutrition are intrinsic risk factors for developing PU. Extrinsic risk factors include unrelieved pressure from hard surfaces, shear by sliding down the bed, excessive moisture from perspiration or soiled sheets, friction from agitation, dragging across sheets, or pushing up the bed with heels.

42.8.3 Presentation

In early stages, most PU are asymptomatic, and they are mostly discovered by vigilant medical staff or caregivers. If symptomatic, they will present with pain, fever, leukocytosis, or local erythema. Local skin infection, osteomyelitis, and sepsis are common in advanced stages.

On physical examination, PU appearance depends on the stage of the ulcer. Stage one appears as non-blanching, reactive, hyperemia that does not disappear after relief of pressure. Stage two appears as a blister or an abrasion where there is damage to the epidermis. Stage three is an open wound where the damage involves the dermis with possible subcutaneous tissue involvement. Stage four appears as a deep wound where muscle, tendon, or even bone are visible. Unstageable PU is an ulcer that is covered by an eschar and or exudates. The assessment of depth and therefore the staging of these types of ulcers are very difficult and require the removal of the eschar in order to stage them.

42.8.4 Differential Diagnosis

Venous stasis ulcers, arterial ischemic ulcers, sickle cell ulcers, diabetes mellitus ulcers, gangrene, or trauma can mimic PU appearance.

42.8.5 Investigation

PU is diagnosed by the location and the physical exam of the ulcer. The ulcer is evaluated by assessing size, depth, odor, and presence of dead tissue, blood, pus, or debris.

42.8.6 Treatment

A well-developed individualized plan of care, based on the PU stage and the patient condition, should be designed with the wound care team to guide the treatment. Dressings should be chosen based on the ulcer characteristics and the condition of the peri-wound skin and must be geared toward managing fluid balance in the wound. In nonhealing PU, surgical debridement or negative pressure wound therapy may be considered as adjuvant treatments. The bacterial load must be managed by the use of topical therapy such as silver dressing or systemic antibiotics if there are signs or symptoms of cellulitis or sepsis.

42.8.7 Prophylaxis

Prevention of PU starts with risk assessment. Patients can be assessed for risk by using one of the PU risk assessment scales such as the Braden Scale [157] or the Norton Scale [158]. Based on each individual's risks, a well-defined, specific prevention plan should be drafted to reduce or control the intrinsic risk factors, such as tight blood sugar control, and to eliminate extrinsic risk factors by the use of soft support surfaces in the bed and the chair, by implementing a repositioning schedule to reduce prolonged pressure on the dependent areas, and by the use of heel supports to reduce friction.

42.9 Mortality

Mortality is the worst and the most profound complication of spine surgery. It is a major concern for the patient, the patient's family, and the surgeon equally. Its prevention is the number one goal of the surgical team, and it starts with the knowledge of the rate and causes of mortality associated with specific age groups, population groups, morbidities, and surgical procedures. This knowledge empowers the surgeon to make better surgical decisions, patient selection, and patient counseling. Due to many confounding factors, accurate and meaningful mortality rates are inherently difficult to obtain. Ideally, data should be collected from a broad spectrum of spine surgeons covering a wide range of spine pathology across a large demographic area. The reported rates of mortality in spine surgery vary wildly between 0 and 7 % [65, 70, 159, 160]. Advance age is a major independent predictor for mortality with or without adjustment for the presence of comorbidities [160–162]. Other independent risk factors for mortality after spine surgery, particularly after spine fusion surgery are the following: male gender [160, 162, 163], comorbidity burden [160–163], anterior and anterior/posterior approaches [160], and traumatic or malignancy indications [160]. Thoracic spine surgery, especially via an anterior approach, has one of the highest mortality rates [160, 163]. Pulmonary disease, congestive heart failure, coagulopathy, malignancy, and renal disease increase the comorbidity burden and are associated with the highest increases in the odds for mortality [160, 163]. Postoperative medical complications are associated with a great increase for the risk of mortality, especially pulmonary embolism and cardiac complications where the risk of death can increase by as much as 8.2-fold and 6.9-fold, respectively [160]. The American Society of Anesthesiologists (ASA) grade [164], the Charlson comorbidity indices [165], and the Goldman multifactorial cardiac risk index (CRI) [166] are used for prognostication and for risk stratification. Although the ASA classification of physical status is not a predictor of operative risk by definition, it has been used as such. The Goldman multifactorial index calculates a predictive score for cardiac complication that has a powerful predictive value for perioperative mortality [167]. It has been shown that the perioperative mortality prediction accuracy can be increased if the ASA grade and the CRI score are used in combination [168].

Questions

1. According to the Wells clinical prediction guide, what is the patient DVT pretest probability score? What is the most appropriate next step?
   (a) DVT pretest probability score is 2, so no further action is needed.

(b) DVT pretest probability score is 4, and IV low molecular weight heparin should be started immediately.
(c) DVT pretest probability score is 2, and a lower extremity duplex ultrasound should be done.
(d) DVT pretest probability score is 5, and a D-dimer test would confirm the presence of a lower extremity DVT.

2. A duplex ultrasound confirms the presence of an acute right common femoral vein thrombosis. Which of the following statements is true?
   (a) The patient can expect asymptomatic recovery once he becomes more ambulatory.
   (b) The patient is at a significant risk for developing a fatal pulmonary embolism.
   (c) The patient may be effectively treated with low-dose heparin.
   (d) This condition may be effectively treated with acetylsalicylic acid.
3. Indications for placement of an IVC filter include all the following except:
   (a) Acute axillary vein thrombosis
   (b) Presence of an acute epidural hematoma on postoperative lumbar MRI
   (c) Recurrent pulmonary embolus despite adequate anticoagulation therapy
   (d) Recurrent DVT despite therapeutic anticoagulation
4. What is the most likely cause of chest pain and tachycardia in a postoperative spine surgery patient?
   (a) Pain from surgery
   (b) Anemia secondary to excessive blood loss during surgery
   (c) Myocardial infarction
   (d) Pulmonary embolus
5. What is the initial step in the management of a patient with postoperative chest pain following spine surgery?
   (a) Stat CT pulmonary angiogram
   (b) Stat blood transfusion
   (c) Increase morphine dose
   (d) Stat EKG, ABG, CXR, and cardiac enzymes

Answers: 1—(c), 2—(b), 3—(a), 4—(c), 5—(d).

References

1. Lebude B et al (2010) Defining “complications” in spine surgery: neurosurgery and orthopedic spine surgeons’ survey. J Spinal Disord Tech 23(8):493–500
2. Nasser R et al (2010) Complications in spine surgery. J Neurosurg Spine 13(2):144–157
3. Thompson JS et al (2003) Temporal patterns of postoperative complications. Arch Surg 138(6):596–602; discussion 602–603
4. Ferree BA et al (1993) Deep venous thrombosis after spinal surgery. Spine (Phila Pa 1976) 18(3):315–319
5. Ferree BA, Wright AM (1993) Deep venous thrombosis following posterior lumbar spinal surgery. Spine (Phila Pa 1976) 18(8):1079–1082
6. Glotzbecker MP et al (2009) Thromboembolic disease in spinal surgery: a systematic review. Spine (Phila Pa 1976) 34(3):291–303
7. Oda T et al (2000) Deep venous thrombosis after posterior spinal surgery. Spine (Phila Pa 1976) 25(22):2962–2967
8. Rokito SE, Schwartz MC, Neuwirth MG (1996) Deep vein thrombosis after major reconstructive spinal surgery. Spine (Phila Pa 1976) 21(7):853–858; discussion 859
9. Sansone JM, del Rio AM, Anderson PA (2010) The prevalence of and specific risk factors for venous thromboembolic disease following elective spine surgery. J Bone Joint Surg Am 92(2):304–313
10. West JL 3rd, Anderson LD (1992) Incidence of deep vein thrombosis in major adult spinal surgery. Spine (Phila Pa 1976) 17(8 Suppl):S254–S257
11. Myllynen P et al (1985) Deep venous thrombosis and pulmonary embolism in patients with acute spinal cord injury: a comparison with nonparalyzed patients immobilized due to spinal fractures. J Trauma 25(6):541–543
12. Weingarden SI (1992) Deep venous thrombosis in spinal cord injury. Overview of the problem. Chest 102(6 Suppl):636S–639S
13. DeVivo MJ, Krause JS, Lammertse DP (1999) Recent trends in mortality and causes of death among persons with spinal cord injury. Arch Phys Med Rehabil 80(11):1411–1419
14. Abad Rico JI, Llau Pitarch JV, Rocha E (2010) Overview of venous thromboembolism. Drugs 70(Suppl 2):3–10
15. McManus R et al (2011) Thromboembolism. American Family Physician. Clin Evid 85(2):188–190. PubMed 22335219 (Print)
16. Virchow RLK (1998) Thrombosis and emboli (1846–1856). Science History Publications, Canton, p 234, vi
17. Mammen EF (1992) Pathogenesis of venous thrombosis. Chest 102(6 Suppl):640S–644S
18. Pearson JD (1999) Endothelial cell function and thrombosis. Baillieres Best Pract Res Clin Haematol 12(3):329–341
19. Dearborn JT et al (1999) Thromboembolic complications after major thoracolumbar spine surgery. Spine (Phila Pa 1976) 24(14):1471–1476

20. Smith MD et al (1994) Deep venous thrombosis and pulmonary embolism after major reconstructive operations on the spine. A prospective analysis of three hundred and seventeen patients. J Bone Joint Surg Am 76(7):980–985
21. Cranley JJ, Canos AJ, Sull WJ (1976) The diagnosis of deep venous thrombosis. Fallibility of clinical symptoms and signs. Arch Surg 111(1):34–36
22. Talbot S (1975) The value of clinical signs of venous thrombosis. Br J Clin Pract 29(9):221–226
23. Diamond PT, Macciocchi SN (1997) Predictive power of clinical symptoms in patients with presumptive deep venous thrombosis. Am J Phys Med Rehabil 76(1):49–51
24. Ansari A (1989) Deep vein versus pseudo deep vein thrombosis: the role of imaging techniques in differential diagnosis and management. Tex Heart Inst J 16(4):280–285; discussion 286
25. Wells PS et al (1997) Value of assessment of pretest probability of deep-vein thrombosis in clinical management. Lancet 350(9094):1795–1798
26. Anand SS et al (1998) Does this patient have deep vein thrombosis? JAMA 279(14):1094–1099
27. Linkins LA et al (2004) Use of different D-dimer levels to exclude venous thromboembolism depending on clinical pretest probability. J Thromb Haemost 2(8):1256–1260
28. Buller HR et al (2009) Safely ruling out deep venous thrombosis in primary care. Ann Intern Med 150(4):229–235
29. Heim SW et al (2004) D-dimer testing for deep venous thrombosis: a metaanalysis. Clin Chem 50(7): 1136–1147
30. Righini M et al (2008) D-dimer for venous thromboembolism diagnosis: 20 years later. J Thromb Haemost 6(7):1059–1071
31. Douketis JD, Ginsberg JS (1996) Diagnosis of deep vein thrombosis. Can Fam Physician 42:497–503
32. White RH et al (1989) Diagnosis of deep-vein thrombosis using duplex ultrasound. Ann Intern Med 111(4):297–304
33. Kelly J et al (2001) Screening for subclinical deep-vein thrombosis. QJM 94(10):511–519
34. Sampson FC et al (2007) The accuracy of MRI in diagnosis of suspected deep vein thrombosis: systematic review and meta-analysis. Eur Radiol 17(1):175–181
35. Fraser DG et al (2002) Diagnosis of lower-limb deep venous thrombosis: a prospective blinded study of magnetic resonance direct thrombus imaging. Ann Intern Med 136(2):89–98
36. Baldt MM et al (1996) Deep venous thrombosis of the lower extremity: efficacy of spiral CT venography compared with conventional venography in diagnosis. Radiology 200(2):423–428
37. Haage P, Krings T, Schmitz-Rode T (2002) Nontraumatic vascular emergencies: imaging and intervention in acute venous occlusion. Eur Radiol 12(11):2627–2643
38. Kearon C et al (2008) Antithrombotic therapy for venous thromboembolic disease: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines (8th edition). Chest 133(6 Suppl): 454S–545S
39. Becker DM, Philbrick JT, Selby JB (1992) Inferior vena cava filters. Indications, safety, effectiveness. Arch Intern Med 152(10):1985–1994
40. Glotzbecker MP et al (2010) Postoperative spinal epidural hematoma: a systematic review. Spine (Phila Pa 1976) 35(10):E413–E420
41. Glotzbecker MP et al (2008) Surgeon practices regarding postoperative thromboembolic prophylaxis after high-risk spinal surgery. Spine (Phila Pa 1976) 33(26):2915–2921
42. Bono CM et al (2009) An evidence-based clinical guideline for the use of antithrombotic therapies in spine surgery. Spine J 9(12):1046–1051
43. Goldhaber SZ, Bounameaux H (2012) Pulmonary embolism and deep vein thrombosis. Lancet 379(9828): 1835–1846
44. Schizas C, Neumayer F, Kosmopoulos V (2008) Incidence and management of pulmonary embolism following spinal surgery occurring while under chemical thromboprophylaxis. Eur Spine J 17(7):970–974
45. Pateder DB et al (2008) Pulmonary embolism after adult spinal deformity surgery. Spine (Phila Pa 1976) 33(3):301–305
46. Hamilton MG, Hull RD, Pineo GF (1994) Venous thromboembolism in neurosurgery and neurology patients: a review. Neurosurgery 34(2):280–296; discussion 296
47. Robbins SL, Kumar V (2010) Robbins and cotran pathologic basis of disease, 8th edn. Saunders/Elsevier, Philadelphia, p 1450, xiv
48. Wells PS et al (2000) Derivation of a simple clinical model to categorize patients probability of pulmonary embolism: increasing the models utility with the SimpliRED D-dimer. Thromb Haemost 83(3):416–420
49. Klok FA et al (2008) Simplification of the revised Geneva score for assessing clinical probability of pulmonary embolism. Arch Intern Med 168(19):2131–2136
50. Beers MH et al (1999) The Merck manual of diagnosis and therapy. Merck, Whitehouse Station
51. Value of the ventilation/perfusion scan in acute pulmonary embolism. Results of the prospective investigation of pulmonary embolism diagnosis (PIOPED). The PIOPED Investigators (1990) JAMA 263(20):2753–2759. PupMed 2332918
52. Wells PS et al (2003) Evaluation of D-dimer in the diagnosis of suspected deep-vein thrombosis. N Engl J Med 349(13):1227–1235
53. Schoepf UJ, Costello P (2004) CT angiography for diagnosis of pulmonary embolism: state of the art. Radiology 230(2):329–337
54. Henzler T et al (2011) CT imaging of acute pulmonary embolism. J Cardiovasc Comput Tomogr 5(1):3–11
55. Stein PD et al (1992) Complications and validity of pulmonary angiography in acute pulmonary embolism. Circulation 85(2):462–468
56. Prasad V, Rho J, Cifu A (2012) The diagnosis and treatment of pulmonary embolism: a metaphor for

medicine in the evidence-based medicine era. Arch Intern Med 172(12):955–958
57. Jerjes-Sanchez C et al (1995) Streptokinase and heparin versus heparin alone in massive pulmonary embolism: a randomized controlled trial. J Thromb Thrombolysis 2(3):227–229
58. Kuo WT et al (2009) Catheter-directed therapy for the treatment of massive pulmonary embolism: systematic review and meta-analysis of modern techniques. J Vasc Interv Radiol 20(11):1431–1440
59. Verstraete M et al (1988) Intravenous and intrapulmonary recombinant tissue-type plasminogen activator in the treatment of acute massive pulmonary embolism. Circulation 77(2):353–360
60. Dauphine C, Omari B (2005) Pulmonary embolectomy for acute massive pulmonary embolism. Ann Thorac Surg 79(4):1240–1244
61. Lee AY et al (2003) Low-molecular-weight heparin versus a coumarin for the prevention of recurrent venous thromboembolism in patients with cancer. N Engl J Med 349(2):146–153
62. Imberti D et al (2012) Interventional treatment of venous thromboembolism: a review. Thromb Res 129(4):418–425
63. Jules-Elysee K et al (2004) Pulmonary complications in anterior-posterior thoracic lumbar fusions. Spine J 4(3):312–316
64. Dai LY et al (2004) Thoracolumbar fractures in patients with multiple injuries: diagnosis and treatment-a review of 147 cases. J Trauma 56(2):348–355
65. Deyo RA et al (1992) Morbidity and mortality in association with operations on the lumbar spine. The influence of age, diagnosis, and procedure. J Bone Joint Surg Am 74(4):536–543
66. Patil CG et al (2007) National inpatient complications and outcomes after surgery for spinal metastasis from 1993–2002. Cancer 110(3):625–630
67. Bianchi C et al (2003) Anterior retroperitoneal lumbosacral spine exposure: operative technique and results. Ann Vasc Surg 17(2):137–142
68. Karadimas EJ et al (2008) Spondylodiscitis. A retrospective study of 163 patients. Acta Orthop 79(5): 650–659
69. Cloyd JM, Acosta FL Jr, Ames CP (2008) Effect of age on the perioperative and radiographic complications of multilevel cervicothoracic spinal fusions. Spine (Phila Pa 1976) 33(26):E977–E982
70. White AP et al (2010) Morbidity and mortality related to odontoid fracture surgery in the elderly population. Spine (Phila Pa 1976) 35(9 Suppl):S146–S157
71. DeVivo MJ, Black KJ, Stover SL (1993) Causes of death during the first 12 years after spinal cord injury. Arch Phys Med Rehabil 74(3):248–254
72. Schilero GJ et al (2009) Pulmonary function and spinal cord injury. Respir Physiol Neurobiol 166(3):129–141
73. Hoare Z, Lim WS (2006) Pneumonia: update on diagnosis and management. BMJ 332(7549):1077–1079
74. Wunderink RG (2000) Clinical criteria in the diagnosis of ventilator-associated pneumonia. Chest 117(4 Suppl 2):191S–194S
75. Singh N et al (1998) Pulmonary infiltrates in the surgical ICU: prospective assessment of predictors of etiology and mortality. Chest 114(4):1129–1136
76. Louthan FB, Meduri GU (1996) Differential diagnosis of fever and pulmonary densities in mechanically ventilated patients. Semin Respir Infect 11(2):77–95
77. Fabregas N et al (1999) Clinical diagnosis of ventilator associated pneumonia revisited: comparative validation using immediate post-mortem lung biopsies. Thorax 54(10):867–873
78. Shorr AF et al (2005) Invasive approaches to the diagnosis of ventilator-associated pneumonia: a meta-analysis. Crit Care Med 33(1):46–53
79. American Thoracic Society, Infectious Diseases Society of America (2005) Guidelines for the management of adults with hospital-acquired, ventilator-associated, and healthcare-associated pneumonia. Am J Respir Crit Care Med 171(4):388–416. PubMed 15699079
80. Singh N et al (2000) Short-course empiric antibiotic therapy for patients with pulmonary infiltrates in the intensive care unit. A proposed solution for indiscriminate antibiotic prescription. Am J Respir Crit Care Med 162(2 Pt 1):505–511
81. Nakagawa M et al (2001) Relationship between the duration of the preoperative smoke-free period and the incidence of postoperative pulmonary complications after pulmonary surgery. Chest 120(3):705–710
82. Cook DJ, Reeve BK, Scholes LC (1994) Histamine-2-receptor antagonists and antacids in the critically ill population: stress ulceration versus nosocomial pneumonia. Infect Control Hosp Epidemiol 15(7): 437–442
83. Bergmans DC et al (2001) Prevention of ventilator-associated pneumonia by oral decontamination: a prospective, randomized, double-blind, placebo-controlled study. Am J Respir Crit Care Med 164(3):382–388
84. Lee MJ et al (2012) Risk factors for medical complication after spine surgery: a multivariate analysis of 1,591 patients. Spine J 12(3):197–206
85. Lee MJ et al (2011) Risk factors for medical complication after lumbar spine surgery: a multivariate analysis of 767 patients. Spine (Phila Pa 1976) 36(21): 1801–1806
86. Brau SA (2002) Mini-open approach to the spine for anterior lumbar interbody fusion: description of the procedure, results and complications. Spine J 2(3): 216–223
87. Yadla S et al (2010) Early complications in spine surgery and relation to preoperative diagnosis: a single-center prospective study. J Neurosurg Spine 13(3):360–366
88. Daubs MD et al (2007) Adult spinal deformity surgery: complications and outcomes in patients over age 60. Spine (Phila Pa 1976) 32(20):2238–2244
89. Macdonald RL et al (1997) Multilevel anterior cervical corpectomy and fibular allograft fusion for cervical myelopathy. J Neurosurg 86(6):990–997
90. Rhee JM et al (2003) Staged posterior surgery for severe adult spinal deformity. Spine (Phila Pa 1976) 28(18):2116–2121

91. Raffo CS, Lauerman WC (2006) Predicting morbidity and mortality of lumbar spine arthrodesis in patients in their ninth decade. Spine (Phila Pa 1976) 31(1):99–103
92. Thygesen K et al (2007) Universal definition of myocardial infarction. Circulation 116(22):2634–2653
93. Nagele P, Liggett SB (2011) Genetic variation, beta-blockers, and perioperative myocardial infarction. Anesthesiology 115(6):1316–1327
94. White HD, Chew DP (2008) Acute myocardial infarction. Lancet 372(9638):570–584
95. Atlee JL (1997) Perioperative cardiac dysrhythmias: diagnosis and management. Anesthesiology 86(6): 1397–1424
96. Mendis S et al (2011) World Health Organization definition of myocardial infarction: 2008–09 revision. Int J Epidemiol 40(1):139–146
97. Shannon AW, Harrigan RA (2001) General pharmacologic treatment of acute myocardial infarction. Emerg Med Clin North Am 19(2):417–431
98. Cabello JB et al (2010) Oxygen therapy for acute myocardial infarction. Cochrane Database Syst Rev (6):CD007160
99. Wijesinghe M et al (2009) Routine use of oxygen in the treatment of myocardial infarction: systematic review. Heart 95(3):198–202
100. Chelazzi C, Villa G, De Gaudio AR (2011) Postoperative atrial fibrillation. ISRN Cardiol 2011:203179
101. Williams FM, Bergin JD (2009) Cardiac screening before noncardiac surgery. Surg Clin North Am 89(4):747–762, vii
102. Eagle KA et al (1996) 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 93(6):1278–1317
103. Fleisher LA et al (2007) 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 116(17):1971–1996
104. Devereaux PJ et al (2008) Effects of extended-release metoprolol succinate in patients undergoing non-cardiac surgery (POISE trial): a randomised controlled trial. Lancet 371(9627):1839–1847
105. Rajaraman V et al (1999) Visceral and vascular complications resulting from anterior lumbar interbody fusion. J Neurosurg 91(1 Suppl):60–64
106. Kuslich SD et al (1998) The Bagby and Kuslich method of lumbar interbody fusion. History, techniques, and 2-year follow-up results of a United States prospective, multicenter trial. Spine (Phila Pa 1976) 23(11):1267–1278; discussion 1279
107. Linville DA et al (1999) Complications in the adult spinal deformity patient having combined surgery. Does revision increase the risk? Spine (Phila Pa 1976) 24(4):355–363
108. Scaduto AA et al (2003) Perioperative complications of threaded cylindrical lumbar interbody fusion devices: anterior versus posterior approach. J Spinal Disord Tech 16(6):502–507
109. Smith MW et al (2011) Comparison of perioperative parameters and complications observed in the anterior exposure of the lumbar spine by a spine surgeon with and without the assistance of an access surgeon. Spine J 11(5):389–394
110. Feldman RA, Karl RC (1992) Diagnosis and treatment of Ogilvie's syndrome after lumbar spinal surgery. Report of three cases. J Neurosurg 76(6):1012–1016
111. Caner H et al (2000) Ogilvie's syndrome as a rare complication of lumbar disc surgery. Can J Neurol Sci 27(1):77–78
112. Cakir E et al (2001) Ogilvie's syndrome after cervical discectomy. Clin Neurol Neurosurg 103(4): 232–233
113. Senagore AJ (2007) Pathogenesis and clinical and economic consequences of postoperative ileus. Am J Health Syst Pharm 64(20 Suppl 13):S3–S7
114. Doorly MG, Senagore AJ (2012) Pathogenesis and clinical and economic consequences of postoperative ileus. Surg Clin North Am 92(2):259–272
115. Batke M, Cappell MS (2008) Adynamic ileus and acute colonic pseudo-obstruction. Med Clin North Am 92(3):649–670, ix
116. Karlsson AK (2006) Autonomic dysfunction in spinal cord injury: clinical presentation of symptoms and signs. Prog Brain Res 152:1–8
117. Frager DH et al (1995) Distinction between postoperative ileus and mechanical small-bowel obstruction: value of CT compared with clinical and other radiographic findings. AJR Am J Roentgenol 164(4):891–894
118. Vanek VW, Al-Salti M (1986) Acute pseudo-obstruction of the colon (Ogilvie's syndrome). An analysis of 400 cases. Dis Colon Rectum 29(3):203–210
119. Beattie GC et al (2007) Computed tomography in the assessment of suspected large bowel obstruction. ANZ J Surg 77(3):160–165
120. Rex DK (1994) Acute colonic pseudo-obstruction (Ogilvie's syndrome). Gastroenterologist 2(3): 233–238
121. Althausen PL et al (2001) The use of neostigmine to treat postoperative ileus in orthopedic spinal patients. J Spinal Disord 14(6):541–545
122. Amaro R, Rogers AI (2000) Neostigmine infusion: new standard of care for acute colonic pseudo-obstruction? Am J Gastroenterol 95(1):304–305

123. Rex DK (1997) Colonoscopy and acute colonic pseudo-obstruction. Gastrointest Endosc Clin N Am 7(3):499–508
124. Richards MJ et al (1999) Nosocomial infections in medical intensive care units in the United States. National Nosocomial Infections Surveillance System. Crit Care Med 27(5):887–892
125. Butt MF et al (2007) Management of unstable thoracolumbar spinal injuries by posterior short segment spinal fixation. Int Orthop 31(2):259–264
126. Carreon LY et al (2003) Perioperative complications of posterior lumbar decompression and arthrodesis in older adults. J Bone Joint Surg Am 85-A(11): 2089–2092
127. McLain RF, Burkus JK, Benson DR (2001) Segmental instrumentation for thoracic and thoracolumbar fractures: prospective analysis of construct survival and five-year follow-up. Spine J 1(5): 310–323
128. Park P et al (2008) The impact of minimally invasive spine surgery on perioperative complications in overweight or obese patients. Neurosurgery 62(3):693–699; discussion 693–699
129. Amin M (1992) Antibacterial prophylaxis in urology: a review. Am J Med 92(4A):114S–117S
130. Patterson JE, Andriole VT (1997) Bacterial urinary tract infections in diabetes. Infect Dis Clin North Am 11(3):735–750
131. Foxman B (2002) Epidemiology of urinary tract infections: incidence, morbidity, and economic costs. Am J Med 113(Suppl 1A):5S–13S
132. Lane DR, Takhar SS (2011) Diagnosis and management of urinary tract infection and pyelonephritis. Emerg Med Clin North Am 29(3):539–552
133. Calandra T, Cohen J (2005) The international sepsis forum consensus conference on definitions of infection in the intensive care unit. Crit Care Med 33(7):1538–1548
134. Gupta K et al (2011) International clinical practice guidelines for the treatment of acute uncomplicated cystitis and pyelonephritis in women: a 2010 update by the Infectious Diseases Society of America and the European Society for Microbiology and Infectious Diseases. Clin Infect Dis 52(5):e103–e120
135. Stark RP, Maki DG (1984) Bacteriuria in the catheterized patient. What quantitative level of bacteriuria is relevant? N Engl J Med 311(9):560–564
136. Saint S, Lipsky BA (1999) Preventing catheter-related bacteriuria: should we? Can we? How? Arch Intern Med 159(8):800–808
137. Wong ES (1983) Guideline for prevention of catheter-associated urinary tract infections. Am J Infect Control 11(1):28–36
138. Kaiser MG et al (2002) Comparison of the mini-open versus laparoscopic approach for anterior lumbar interbody fusion: a retrospective review. Neurosurgery 51(1):97–103; discussion 103–105
139. Carragee EJ, Hurwitz EL, Weiner BK (2011) A critical review of recombinant human bone morphogenetic protein-2 trials in spinal surgery: emerging safety concerns and lessons learned. Spine J 11(6):471–491
140. Kamischke A, Nieschlag E (2002) Update on medical treatment of ejaculatory disorders. Int J Androl 25(6):333–344
141. Edgard-Rosa G et al (2012) Midline anterior approach from the right side to the lumbar spine for interbody fusion and total disc replacement. A new mobilization technique of the vena cava. Spine (Phila Pa 1976) 37(9):E562–E569. PubMed 22517482 (Print)
142. Darrah DM, Griebling TL, Silverstein JH (2009) Postoperative urinary retention. Anesthesiol Clin 27(3):465–484, table of contents
143. Koch CA, Layton KF, Kallmes DF (2007) Outcomes of patients receiving long-term corticosteroid therapy who undergo percutaneous vertebroplasty. AJNR Am J Neuroradiol 28(3):563–566
144. Tammela T, Kontturi M, Lukkarinen O (1986) Postoperative urinary retention. I. Incidence and predisposing factors. Scand J Urol Nephrol 20(3): 197–201
145. Petros JG, Bradley TM (1990) Factors influencing postoperative urinary retention in patients undergoing surgery for benign anorectal disease. Am J Surg 159(4):374–376
146. Kumar P et al (2006) Urinary retention and the role of indwelling catheterization following total knee arthroplasty. Int Braz J Urol 32(1):31–34
147. Doyle PT, Briscoe CE (1976) The effects of drugs and anaesthetic agents on the urinary bladder and sphincters. Br J Urol 48(5):329–335
148. Kebapci N et al (2007) Bladder dysfunction in type 2 diabetic patients. Neurourol Urodyn 26(6):814–819
149. Park YH, Ku JH, Oh SJ (2011) Accuracy of post-void residual urine volume measurement using a portable ultrasound bladder scanner with real-time pre-scan imaging. Neurourol Urodyn 30(3): 335–338
150. Kamphuis ET et al (1998) Recovery of storage and emptying functions of the urinary bladder after spinal anesthesia with lidocaine and with bupivacaine in men. Anesthesiology 88(2):310–316
151. Kaplan SA et al (2008) Urinary retention and post-void residual urine in men: separating truth from tradition. J Urol 180(1):47–54
152. Lamonerie L et al (2004) Prevalence of postoperative bladder distension and urinary retention detected by ultrasound measurement. Br J Anaesth 92(4): 544–546
153. Te AE (2002) A modern rationale for the use of phenoxybenzamine in urinary tract disorders and other conditions. Clin Ther 24(6):851–861; discussion 837
154. Evron S et al (1984) The effect of phenoxybenzamine on postoperative urinary complications during extradural morphine analgesia. Eur J Anaesthesiol 1(1):45–54
155. Redelings MD, Lee NE, Sorvillo F (2005) Pressure ulcers: more lethal than we thought? Adv Skin Wound Care 18(7):367–372
156. Sever R et al (2012) Admission Norton scale scores (ANSS) are associated with post-operative complications following spine fracture surgery in the elderly. Arch Gerontol Geriatr 55(1):177–180

157. Bergstrom N et al (1987) The Braden scale for predicting pressure sore risk. Nurs Res 36(4):205–210
158. Norton D (1980) Norton scale for decubitus prevention. Krankenpflege (Frankf) 34(1):16
159. Smith JS et al (2012) 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) 37(23):1975–1982. PubMed 22498991
160. Memtsoudis SG et al (2011) Perioperative morbidity and mortality after anterior, posterior, and anterior/posterior spine fusion surgery. Spine (Phila Pa 1976) 36(22):1867–1877
161. Li G et al (2008) Effects of age and comorbidities on complication rates and adverse outcomes after lumbar laminectomy in elderly patients. Spine (Phila Pa 1976) 33(11):1250–1255
162. Oldridge NB et al (1994) Lumbar spine surgery and mortality among Medicare beneficiaries, 1986. Am J Public Health 84(8):1292–1298
163. Shen Y, Silverstein JC, Roth S (2009) In-hospital complications and mortality after elective spinal fusion surgery in the united states: a study of the nationwide inpatient sample from 2001 to 2005. J Neurosurg Anesthesiol 21(1):21–30
164. Dripps RD (1963) New classification of physical status. Anesthesiology 24:111
165. Charlson ME et al (1987) A new method of classifying prognostic comorbidity in longitudinal studies: development and validation. J Chronic Dis 40(5):373–383
166. Goldman L et al (1977) Multifactorial index of cardiac risk in noncardiac surgical procedures. N Engl J Med 297(16):845–850
167. Detsky AS et al (1986) Predicting cardiac complications in patients undergoing non-cardiac surgery. J Gen Intern Med 1(4):211–219
168. Prause G et al (1997) Can ASA grade or Goldman's cardiac risk index predict peri-operative mortality? A study of 16,227 patients. Anaesthesia 52(3):203–206

Index

V.V. Patel et al. (eds.), *Spine Surgery Basics*,
DOI 10.1007/978-3-642-34126-7, © Springer-Verlag Berlin Heidelberg 2014

T