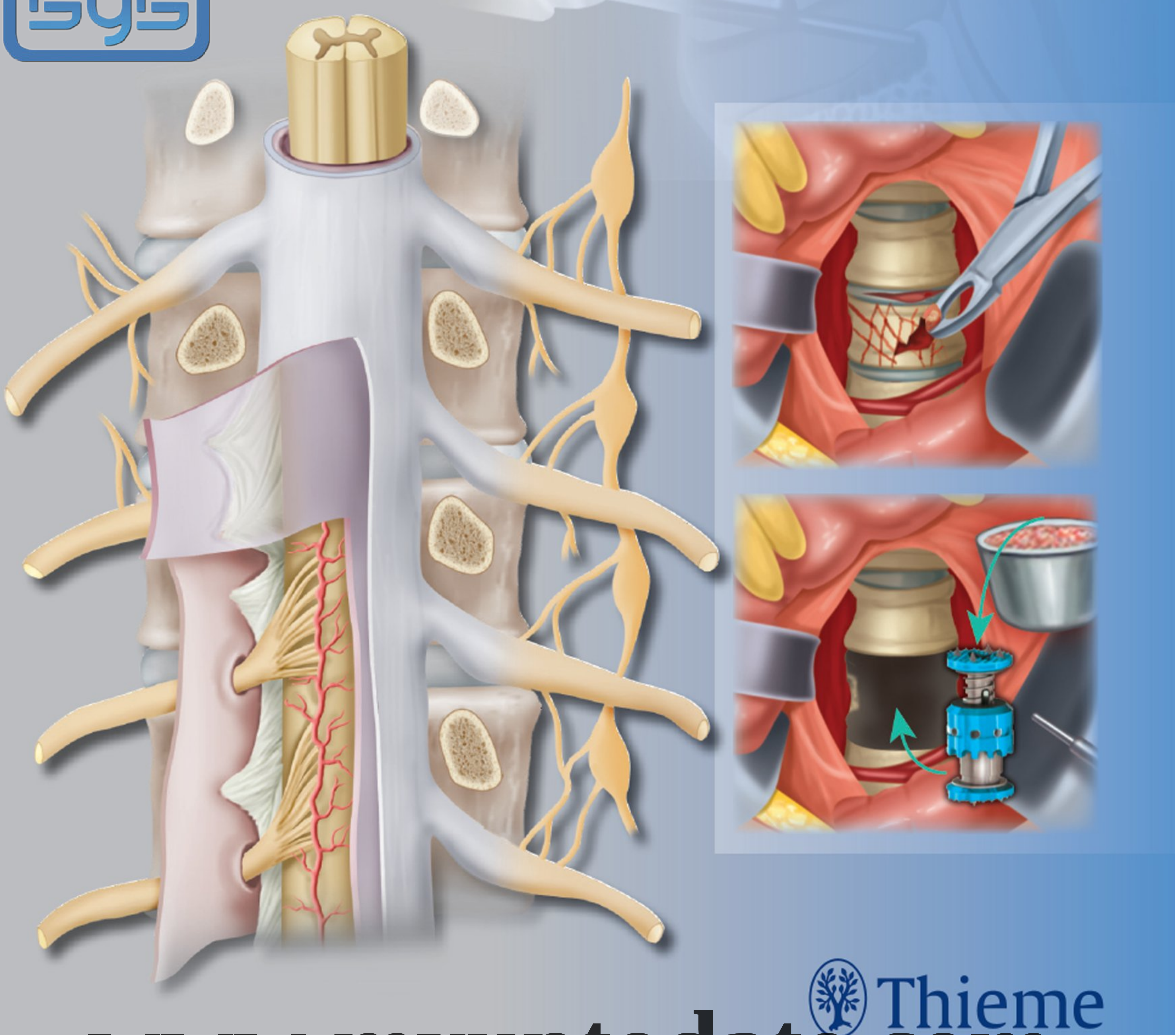
Synopsis of Spine Surgery

Third Edition

Howard S. An
Kern Singh



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I would like to dedicate this book to my mother, father, my wife, and my children, who have given me unconditional love and support throughout my life.

Howard S. An

I dedicate this book to my mother and father. Thank you both for always forcing me to be the best I could be. Without your love, patience, and dedication, I would not be the physician, father, or son I am today.

Kern Singh

Contents

Preface

The third edition of the *Synopsis of Spine Surgery* reflects the rapid advances in the field since the first edition—advances that have changed our approach to patient care, particularly involving minimally invasive surgery. In addition, significant advances have been made in bone physiology and biologics. The third edition succinctly summarizes these advances with the reader being able to quickly reference these new spine surgical developments.

The premise behind this book is to concisely summarize information in an outline format. The reader is able to peruse broad topics in an expeditious manner, while being directed to key references that may provide greater detail on topics of particular interest. This book is written primarily for orthopedic surgeons and neurosurgeons-in-training. The easy-to-read format also makes this an excellent resource for practicing spine surgeons as well as nonoperative spine physicians.

We hope that this book will provide our readers with a thorough understanding of the modern field of spine surgery in the most efficacious manner possible, increasing the quality of care given to spine patients.

Howard S. An, MD
Kern Singh, MD

1 Anatomy and Surgical Approaches

1.1 Basic Anatomy of the Spine

I. Vertebral column.
   A. The spinal column has specifically adapted anatomical features to maintain stability, protect neural elements, and allow range of motion.
   B. Stability is augmented by the intervertebral disks, ligaments, and muscles.
   C. There are 33 vertebrae in the spinal column (7 cervical, 12 thoracic, 5 lumbar, 5 sacral, and 4 coccygeal).
   D. There are four sagittal curves in the vertebral column.
      1. Cervical lordosis (20–40°).
      2. Thoracic kyphosis (20–50°).
      3. Lumbar lordosis (31–79°).
      4. Sacral kyphosis.
   E. The kyphotic curves are called primary because they form during the fetal period.
      1. Caused by the wedge-shaped nature of vertebrae.
   F. The lordotic curves are called secondary because they begin to form during the late fetal period and continue to form after birth. Head and body weight contributes to their formation.
      1. Caused by differences in the anteroposterior dimensions of the intervertebral disks.
   G. Each vertebra consists of a posterior bony arch and an anterior body, both of which surround the vertebral canal.
      1. Posterior bony arches between two adjacent vertebrae form a foramen for the spinal nerve roots.
      2. Anterior bodies of the lumbar spine support 80%of the axial load on the spinal column.
   H. Posterior bony arch components **(Table 1.1).**

II. Intervertebral disks **(Table 1.2).**
   A. There are 23 intervertebral disks in the spinal column (6 cervical, 12 thoracic, 5 lumbar).
   B. Each disk is located between adjacent vertebral end plates, which are covered by hyaline and fibrocartilage and supported by subchondral bone.
   C. Vertebral disks make up one quarter of the spinal column height. The disks expand when the column is horizontal because water and nutrients enter the disk, but they collapse under the stress of prolonged standing or sitting.
   D. The disk is a relatively avascular structure with the outer layers receiving nutrients from the end arterioles and the central portions receiving nutrients by diffusion from the vertebral end plates.
   E. Each disk consists of an outer annulus fibrosus, which surrounds a central nucleus pulposus **(Fig. 1.1)**.

Table 1.1 Posterior bony arch components

Components	Function
Pedicles and laminae	Form the borders of the vertebral canal with the posterior border of the vertebral body
Spinous and transverse processes	Attachment sites for supporting ligaments and muscles
Articular processes	The superior articular process of the caudal vertebrae forms a facet joint with the inferior articular process of the cephalad vertebrae bilaterally. The articular facet joints support 20% of the axial load on the spinal column. The pars interarticularis is the bony region between the superior and inferior articular processes of an individual vertebra.

Table 1.2 Histological characteristics of the intervertebral disk

Intervertebral disk component	Characteristics
Annulus fibrosus	• 10–20 layers of concentric lamellae of fibrocartilage • Type I collagen • Lattice made of sheets running in opposite directions that give the disk greater rotational strength • Thickest portion is anterior. Thinnest portion is posterolateral. • Outer portions of the annulus are continuous with the anterior and posterior longitudinal ligaments.
Nucleus pulposus	• Mucoid type II collagen and water-imbibing proteoglycans (e.g., aggrecan) • Cushions axial load • Cannot be compressed under pressure due to its fluid nature, but rather will deform in all directions

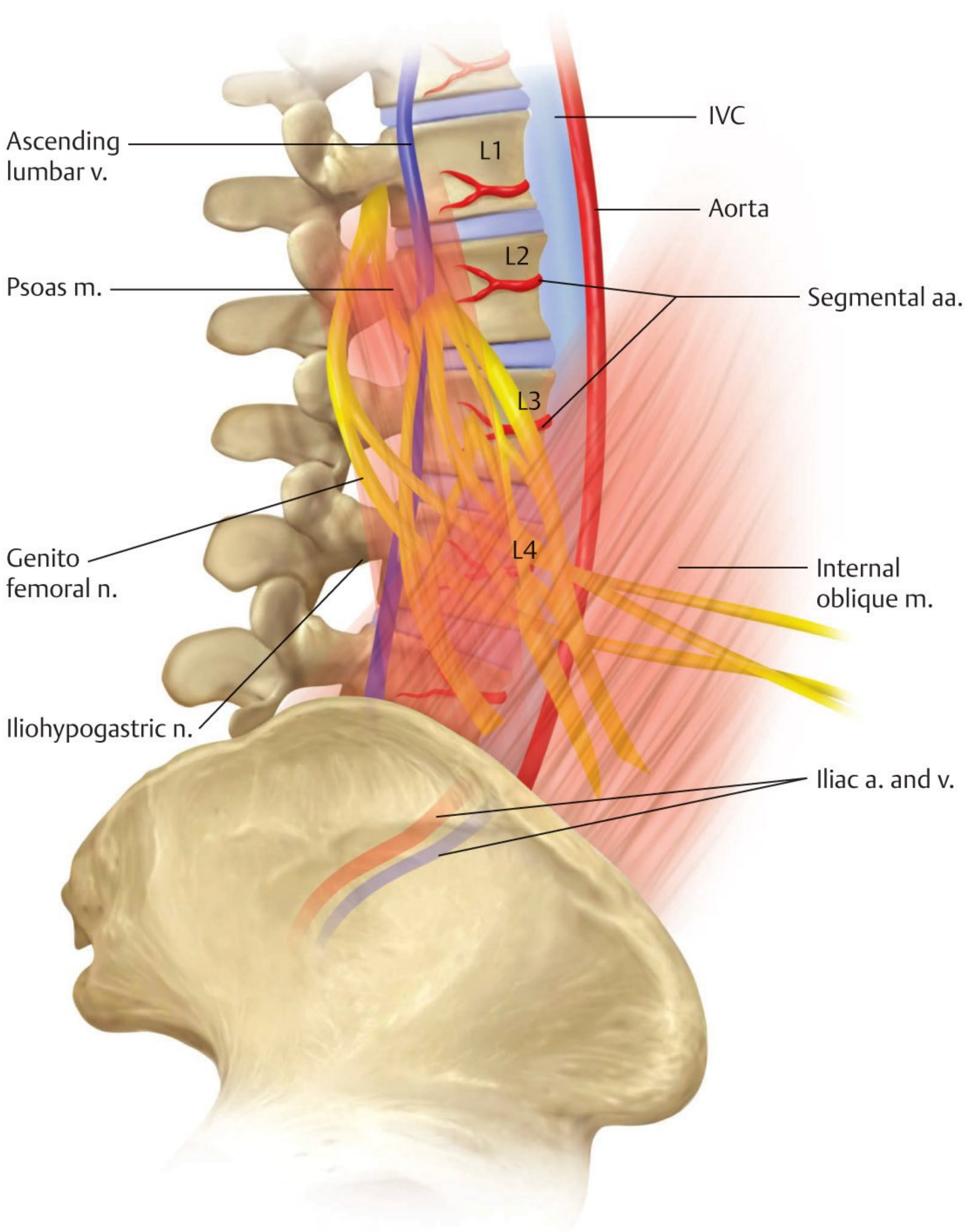


Fig. 1.1 The nerve structures of the lumbar plexus.

1.2 Neuroanatomy

I. Spinal cord.
  A. Gross structure.
    1. The spinal cord typically ends at L1–L2 (conus medullaris).
      a. It may be as high as T12 or as low as L2–L3.
      b. In newborn infants, the cord ends at L2–L3.
    2. Length: 45 cm cord and 25 cm filum terminale (10%increase in length with flexion, mostly at C1, T1, and L1, least at C6 and T6).
    3. Mean diameter (10 mm, transverse diameter greater than sagittal diameter).
    4. Relationship between cord and vertebral segments **(Fig. 1.2) (Table 1.3)**.

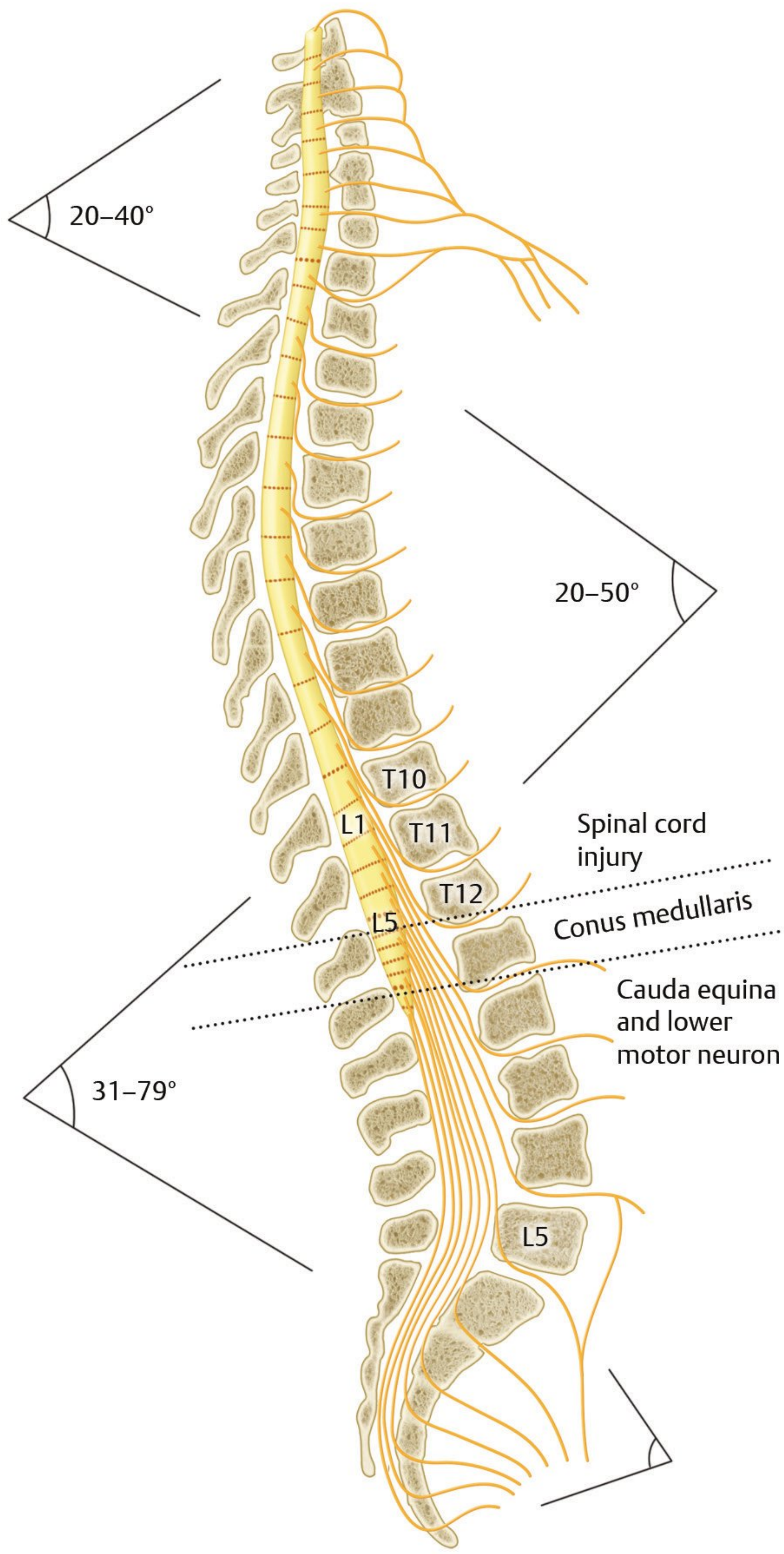


Fig. 1.2 The spinal cord and nerve roots. The spinal cord emerges from the foramen magnum as a continuation of the medulla oblongata and ends in a cone-shaped structure known as the conus medullaris. The location of the conus medullaris is usually the L1–L2 intervertebral disk in adults. The cervical cord enlarges maximally at the C6 vertebra to provide C3–T2 innervation to the upper limbs, and the lumbosacral enlargement is present at T11–L1 vertebral segments to provide L1–S3 cord segments to the lower extremities.

Table 1.3 Relationship between cord and vertebral segments

Cord segment	Vertebral segment
C1	C1
C8	C7
T6	T5
T12	T8
L2	T10
L5	T11
S3	T12

B. Internal structures **(Fig. 1.3) (Table 1.4)**.
   1. The peripheral white matter and central gray matter.
      a. Gray matter: cell bodies of efferent neurons.
         (1) Posterior horns: somatosensory.
         (2) Anterior horns: somatomotor.
         (3) Intermediolateral horns: visceral.
         (4) Reflex somatic centers.
      b. White matter: nerve fibers and glia.
         (1) Posterior funiculus: posterior columns (fasciculus cuneatus laterally and gracilis medially).
         (2) Lateral funiculus: lateral corticospinal and lateral spinothalamic fasciculus.
         (3) Anterior funiculus: anterior spinothalamic tract.
   2. Central ependymal canal: passage of cerebrospinal fluid.
   3. Spinal cord syndromes **(Table 1.5)**.

C. Vascularity of the spinal cord.
   1. Cervical spine.
      a. The anterior spinal artery: the major vessel for anterior and central aspects of the cord:
         (1) Two medullary feeders at the brain stem by the vertebral arteries.
         (2) Other medullary feeders from the vertebral arteries and ascending cervical arteries, particularly C2 and C6 from the left and C2, C5, C6 from the right.
      b. Two posterior spinal arteries from the posterior inferior cerebellar arteries have minimal contribution to the central gray matter.
   2. Thoracolumbar spine.
      a. The anterior spinal artery, two posterior spinal arteries.

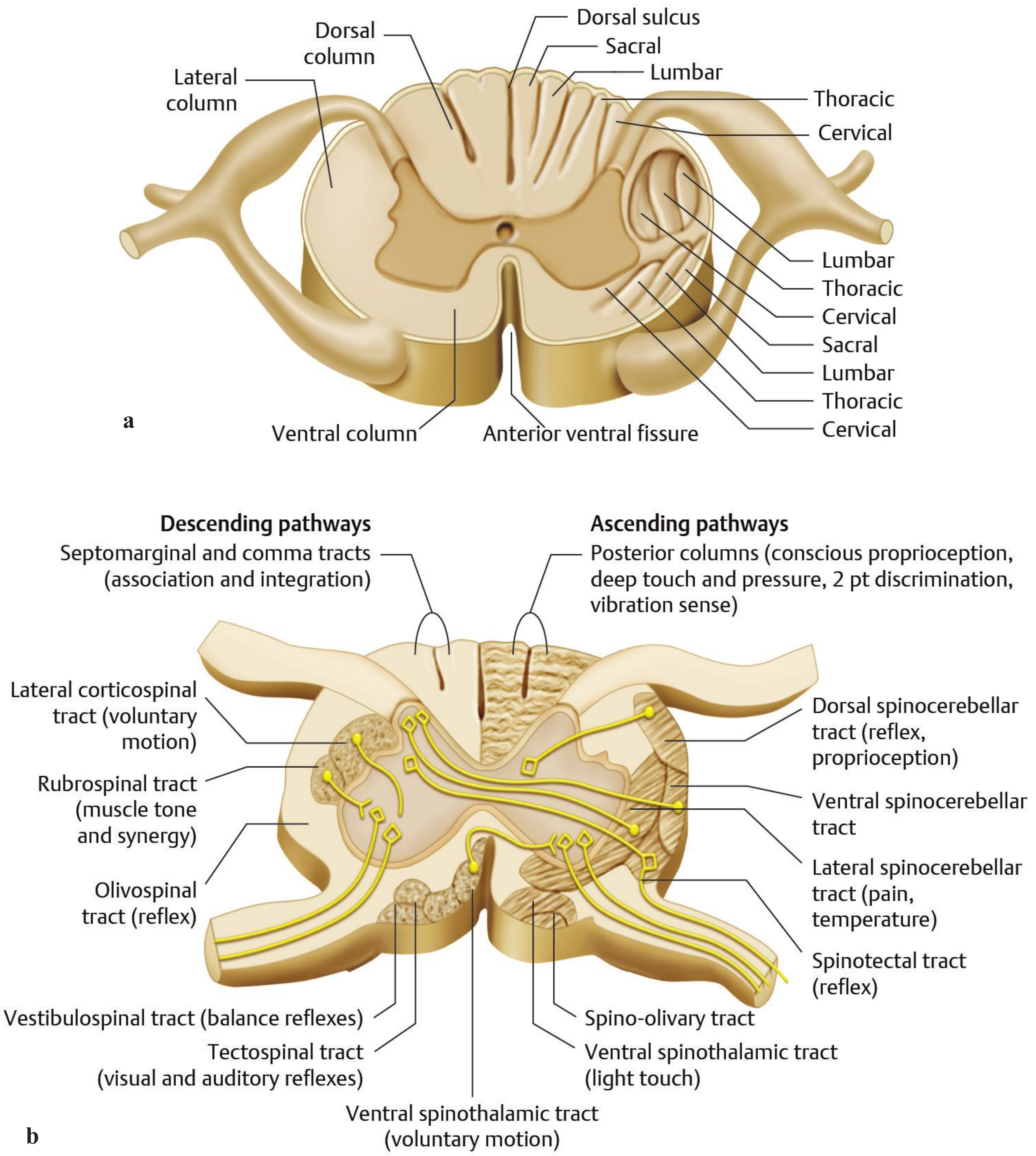


Fig. 1.3 **(a,b)** Cross section of the spinal cord with the outer white matter and the inner gray matter. The white matter of the spinal cord contains nerve fibers and glia and is divided into three columns: posterior, lateral, and anterior. The posterior column includes the fasciculus cuneatus laterally and the fasciculus gracilis medially. The lateral column contains the descending motor lateral corticospinal and lateral spinothalamic fasciculi, and the anterior funiculus contains the ascending anterior spinothalamic tract and other descending tracts. The lateral spinothalamic tracts cross through the ventral commissure to the contralateral side of the cord. The gray matter of the spinal cord contains cell bodies of efferent and internuncial neurons.

Table 1.4 Spinal cord function

	Components	Notes
Motor function	Cerebral cortex Internal capsule Corticospinal tract Pyramidal tract (90% decussate at the medulla to the contralateral lateral corticospinal fasciculus) Anterior horn cells	Lateral corticospinal fasciculus: tracts for the upper extremities are medial to the lower extremities
Sensory function	Fasciculus gracilis: lower limbs and below midthorax Fasciculus cuneatus: upper limbs and above midthorax	Sensory tracts cross to the opposite side in the medulla oblongata and to the sensory cortex Tactile discrimination, proprioception, and vibration sense
	Lateral spinothalamic fasciculus: pain, temperature, and light touch	Most fibers cross through the ventral commissure to the opposite side and ascend through the lateral spinothalamic tract
	Anterior spinothalamic fasciculus: crude touch	Posterior cord syndrome: only crude touch is spared

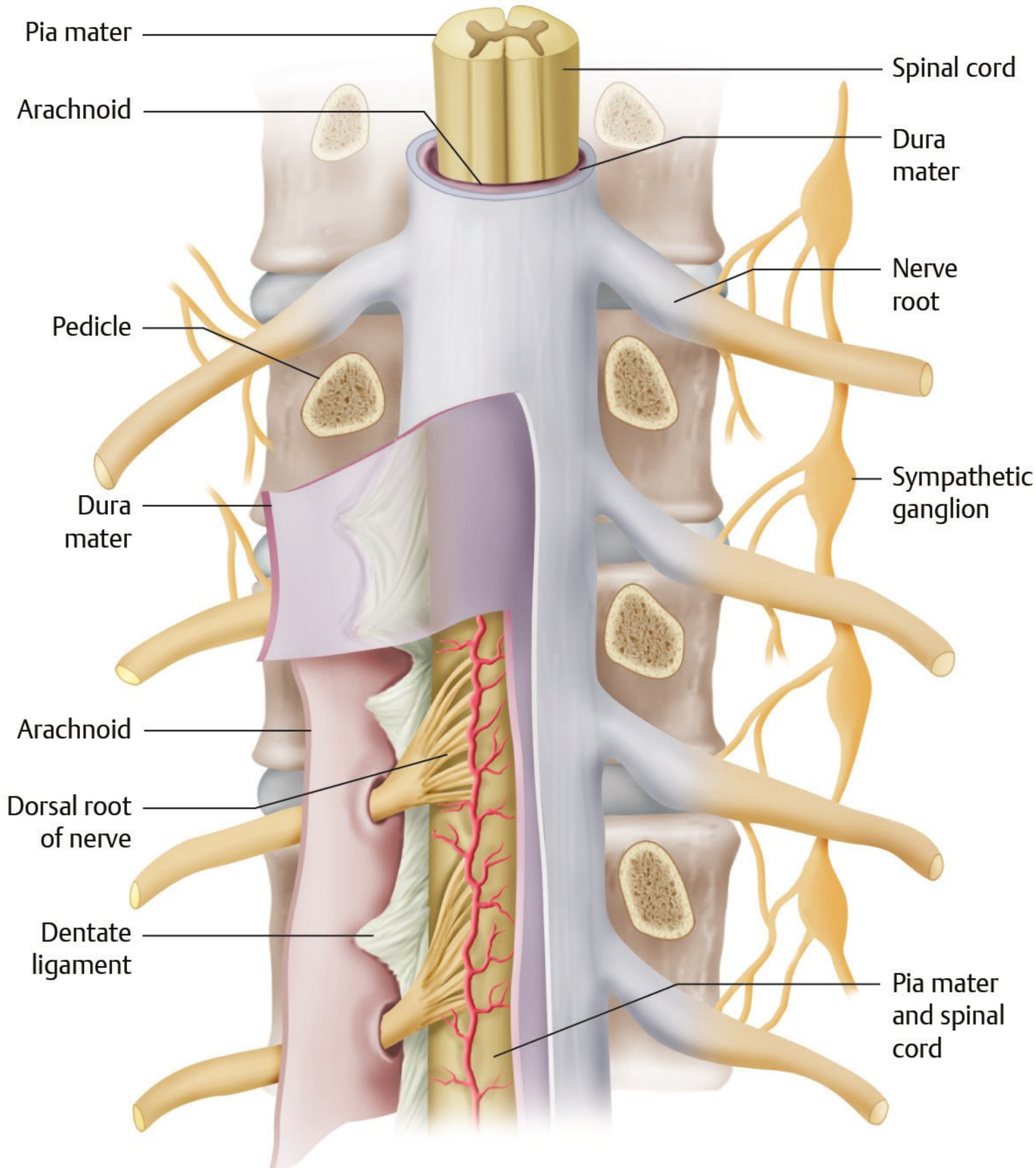
b. Feeders.
   (1) Superior intercostal artery: branch of deep cervical artery, which is a branch of the right subclavian artery, feeds the cord at the cervical–thoracic junction.
   (2) One to five segmental vessels: tenuous blood supply for the upper thoracic cord (watershed critical zone from T4 to T10).
   (3) The artery of Adamkiewicz (80%from T10 from the left, but origin may vary from T5 to L5) supplies the thoracic cord.
   (4) Anastomotic loop of the conus medullaris from aortic segmental and lateral sacral arteries.

3. Venous drainage (azygos and hemiazygos veins).
   a. Veins of the spinal cord anteriorly and posteriorly.
   b. Batson's plexus: from basiocciput to the coccyx.
   c. Clinical significance: metastatic dissemination and infections

II. Spinal meninges **(Fig. 1.4)**.
   A. Dura mater: outer covering of the spinal cord.
   B. Leptomeninx: pia mater (outer lining of the cord) and arachnoid membrane (transparent sheet containing the cerebrospinal fluid).

Table 1.5 Spinal cord syndromes (incomplete cord injury)

Syndrome	Characteristics	Common causes	Recovery
Central cord syndrome (most common)	Greater motor deficit in upper extremities (medial tracts of the lateral corticospinal fasciculus) compared with the lower extremities (lateral tracts).	Extension injury mechanism in patients with spondylosis due to anterior osteophytes and posterior infolded ligamentum flavum (pincer effect)	Good prognosis (full functional recovery is rare) Lower extremity and bladder function recover before upper extremities
Anterior cord syndrome	Motor function and sensation to light touch are impaired. The posterior column functions (pressure and proprioception) are spared.	Direct compression of anterior spinal cord or anterior spinal artery injury from bony spicules	Worst prognosis 10–20%motor recovery
Brown–Séquard syndrome	Pain and temperature loss are contralateral and one or two levels below the injury, whereas motor and proprioception loss are ipsilateral and at the level of the injury. The motor paralysis is flaccid at the level of the injury (lower motor injury). Below the level of injury, the motor paralysis is spastic (upper motor injury).	Penetrating trauma (stab wound to the back)	Excellent prognosis 99%recovery of ambulatory function
Posterior cord syndrome (very rare)	Loss of proprioception and vibration sense only (posterior spinal cord).	Injury to the posterior spinal artery	Rare and not well characterized



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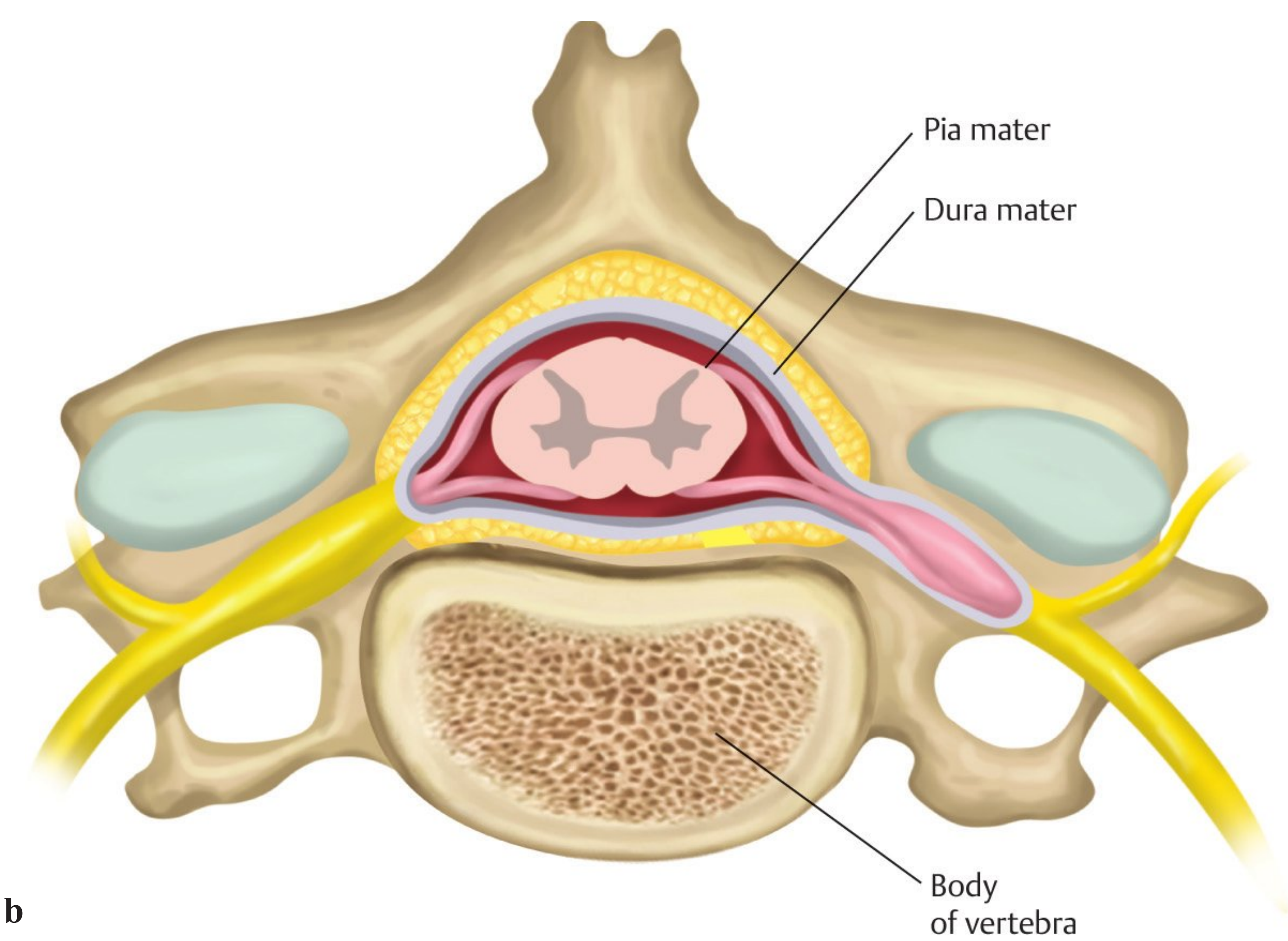

b

Fig. 1.4 (a,b) Cross section of the spinal cord and meninges. The spinal cord is covered by the pia mater, which is the outer lining of the cord, and the transparent arachnoid mater, which contains the cerebrospinal fluid. The dura mater is the outer covering of the spinal cord. The spinal cord is anchored to the dura by the dentate ligaments that project laterally from the lateral side of the cord to the arachnoid and dura midway between the exiting spinal nerves.

C. Dentate ligament: between the dorsal and ventral nerve roots, stabilizes the spinal cord within the dura.
D. Septum posticum between the pia and arachnoid on the dorsal aspect from lower cervical to conus regions.
E. Epidural space: space between the bone and dura.
   1. Space: 2 mm at L3–L4, 4 mm at L4–L5, 6 mm at L5–S1.
   2. Plica mediana dorsalis durae matris: a median fold at the lumbosacral region.
F. Termination of the dura/arachnoid envelope varies from S1–S2 to S2–S3, and the dura invests the filum terminale and attaches to the coccyx.

III. Spinal nerves.
A. Thirty-one pairs of spinal nerves: 8 cervical, 12 thoracic, 5 lumbar, 5 sacral, 1 coccygeal.
B. The spinal root nerve consists of motor and sensory rootlets, the dorsal root ganglion, and the spinal nerve.
   1. Sympathetic connections by preganglionic (white) rami and unmyelinated postganglionic (gray) rami.
   2. Branches: sinuvertebral nerve to the annulus of the disk, and dorsal ramus for facets and posterior muscles.
      a. The sinuvertebral nerve reenters the spinal canal through the foramen to lie anterior to the nerve root and cranial to the disk. The sinuvertebral nerves innervate the posterior longitudinal ligament, the posterior part of the annulus, and the ventral part of the dura. The sinuvertebral nerves typically ascend to innervate the superior disk as well.
      b. The dorsal primary rami gives medial (innervates facet joints above and below, segmental muscles, and interspinous ligament), lateral (innervates iliocostalis muscle), and occasionally intermediate (innervates longissimus muscle) branches.
C. The C1 nerve emerges above the C1 vertebra, and the C8 emerges above the T1 vertebra. In the thoracic and lumbar regions, the spinal nerves emerge beneath the pedicles bearing the same number.
D. Spinal nerves in the intervertebral foramina:
   1. Cervical spine: no intervertebral foramen for C1 and C2, but C3–C8 escape through corresponding foramina occupying ~ 75%of space.
   2. Thoracic spine: thoracic spinal nerves are small and occupy 20%of the foramen and exit below the pedicle.
   3. Lumbar spine: large lumbar nerves occupy 33%of the foramen and exit obliquely below the pedicle.
   4. Sacrum: anterior rami emerge through the anterior sacral foramina, and posterior rami through the posterior sacral foramina.
E. Dermatomes and myotomes **(Fig. 1.5) (Table 1.6)**.
F. Dynamics:
   1. L5 or S1 nerve roots may glide up to 1 cm during flexion–extension.
   2. The spinal cord and nerve roots generally stretch in flexion and relax in extension, but the spinal canal and foramen enlarge in flexion and narrow in extension.
G. Cauda equina:
   1. Lumbar and sacral roots are organized in a specific pattern.
   2. Fifty percent compression of the thecal sac leads to dysfunction.

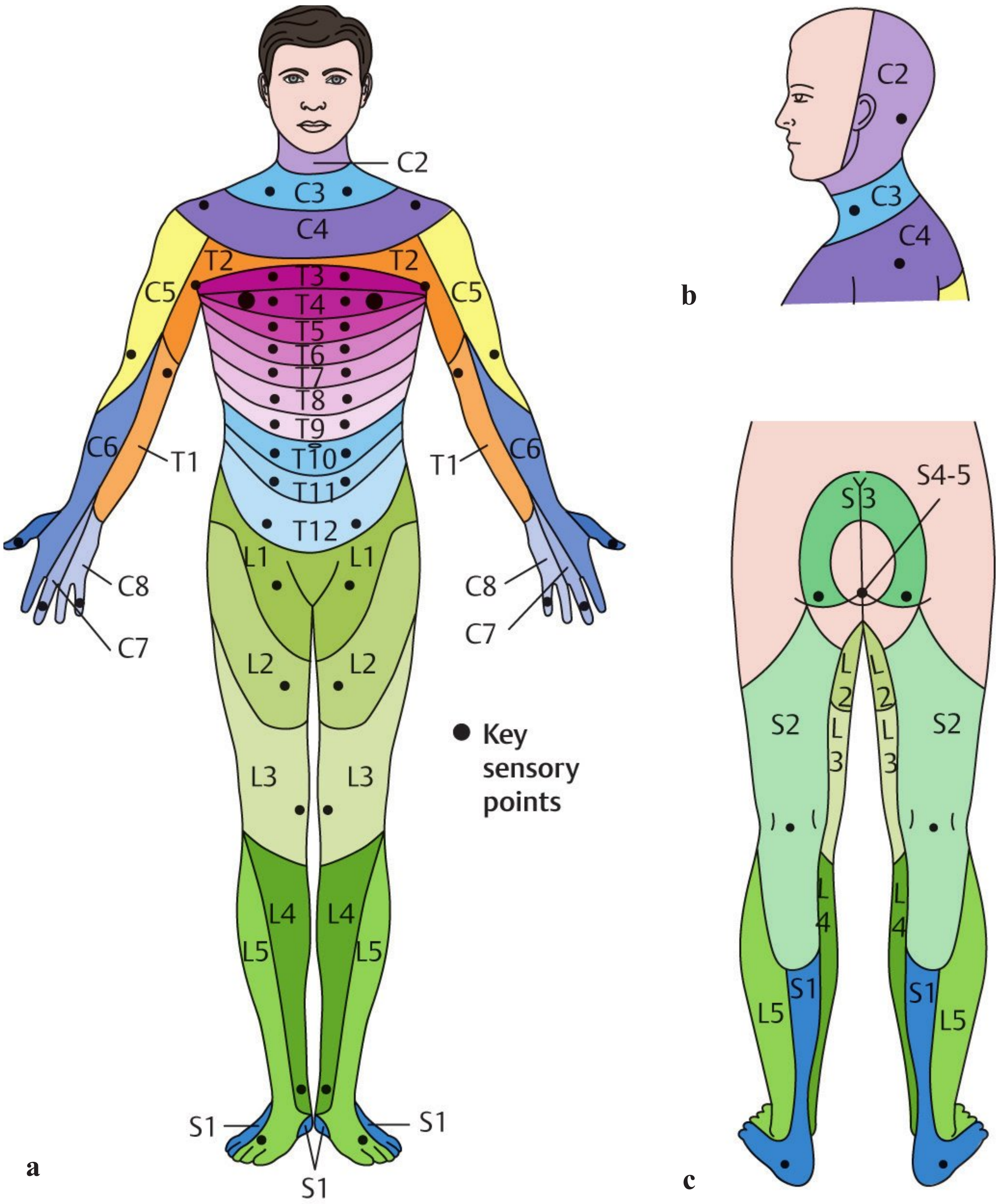

Fig. 1.5 **(a–c)** Anterior and posterior dermatomes.

H. Nerve root anomalies (Kadish and Simmons) **(Fig. 1.6)**:
  1. Type I: intradural anastomosis.
  2. Type II: anomalous origin of nerve roots.
  3. Type III: extradural anastomosis.
  4. Type IV: extradural division.

I. Vasculature of the nerve root:
  1. Proximal and distal radicular arteries anastomose in the proximal third of the root in the foramen, which may be a vascular-deficient area.
  2. Intrinsic vasculature: interfascicular and intrafascicular vessels with compensating coils and arteriovenous anastomosis allow considerable interfascicular motion and stretch of the root.
  3. A thin pia mater allows exchange of metabolites with cerebrospinal fluid.
  4. Mechanical compression causes vascular compression, which manifests in neuroischemic claudication clinically.

J. Plexus:
  1. Cervical and brachial plexus.
     a. The anterior rami of C1–C4 form the cervical plexus.

Table 1.6 Myotome and dermatome distribution

Nerve root	Motor	Sensory	Reflex
C4	Diaphragm and trapezius	Base of neck	None
C5	Deltoid and biceps	Upper outer arm	Biceps brachii
C6	Wrist extensors	Thumb	Brachioradialis
C7	Triceps and wrist flexors	Long finger	Triceps brachii
C8	Finger flexors	Little finger	None
T1	Intrinsic muscles of the hand	Medial forearm	None
L1	Transversus abdominis	Inguinal region	None
L2	Iliopsoas	Upper thigh	None
L3	Quadriceps	Anterior and medial thigh	None
L4	Tibialis anterior	Anterior knee, medial leg, ankle, and foot	Patellar tendon
L5	Extensor hallucis longus	First web space	Hamstring
S1	Gastrocnemius	Posterior thigh, small toe	Achilles tendon
S2	Bladder sphincter	Posterior thigh and leg	Bulbocavernosus (S2–S3)
S3–S5	Anal sphincter (S3)	Perineum, anus	Anal tone (cauda equina syndrome)

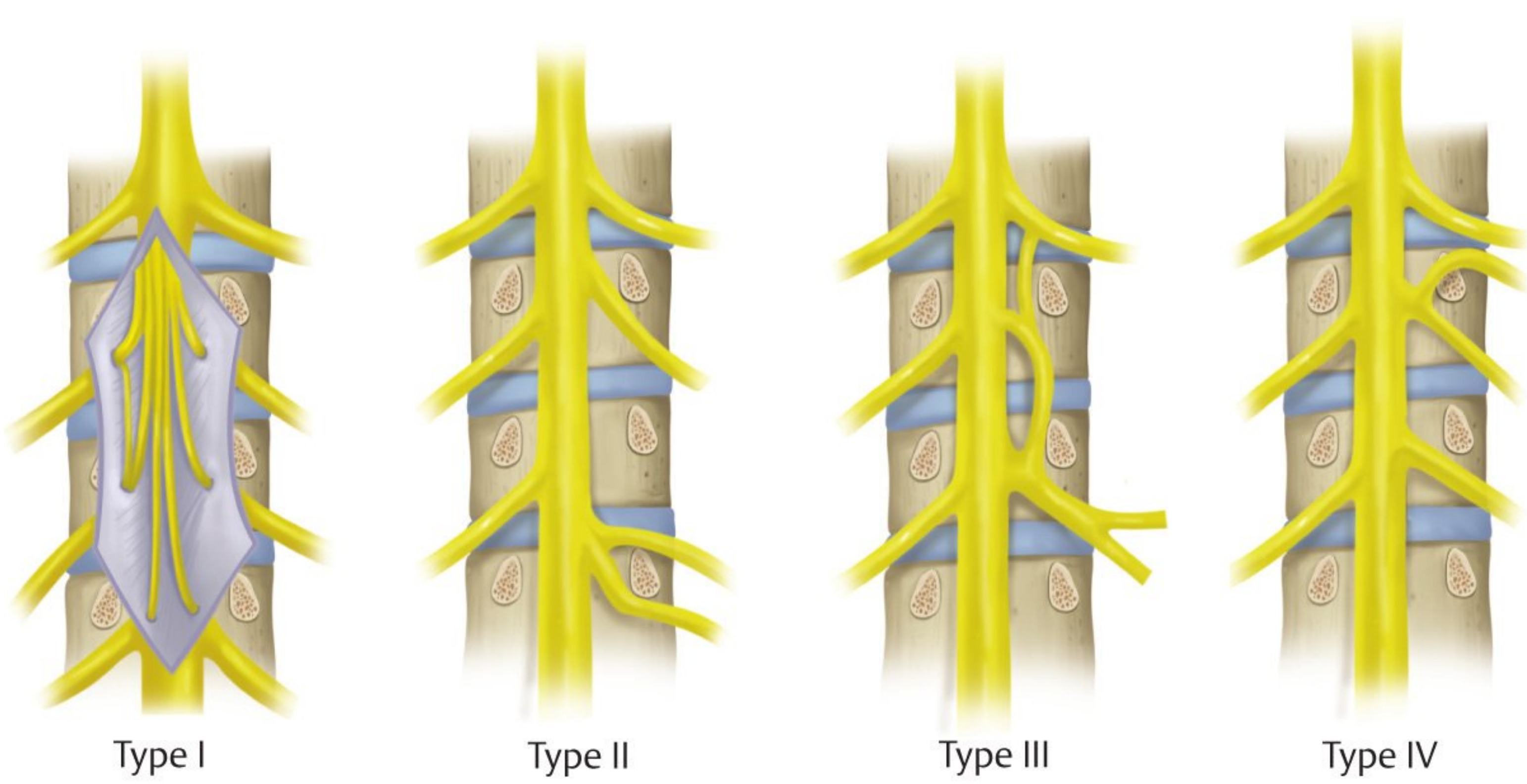

Fig. 1.6 The four types of nerve root anomalies.

b. The anterior rami of C5–T1 form the brachial plexus.
   (1) Branches: suprascapular (C5–C6), subscapular (C5–C6), subclavius (C5–C6), long thoracic (C5–C7), musculocutaneous (C5–C6), median (C5–T1), axillary (C5–C6), radial (C5–T1), medial cutaneous nerve of arm and forearm (C8–T1), medial cutaneous (C8–T1), ulnar (C8–T1).

2. Lumbosacral and coccygeal plexus.
   a. Lumbosacral trunk (L4, L5) and S1, S2, S3, and S4 anterior rami.
   b. Sciatic (L4–S3) and pudendal (S2–S4) nerves.
   c. Branches: superior gluteal (L4–S1), inferior gluteal (L5–S2), nerve to the obturator internus, nerve to the quadratus femoris (L5–S2), and the posterior cutaneous nerve of the thigh (S1–S3).
   d. Anterior coccygeal plexus: S5 and coccygeal anterior rami to become anterior caudal nerve.

IV. Autonomic systems (sympathetic and parasympathetic systems).
A. Sympathetic centers.
1. C8 to L4 spinal cord.
2. Sympathetic trunk and ganglions: cervical to sacral.
3. Cardioaccelerator center, sweat glands, vasomotor, bronchopulmonary, abdominal splanchnic, anorectal/bladder continence, and ejaculation center.
4. Loss of sympathetic system.
   a. Peripheral vasodilation (hypotension), bradycardia, inability to perspire, and hypothermia due to spinal cord injury (injury of the preganglionic fiber and intermediolateral cell column).
   b. Horner’s syndrome: drooping of upper eyelid (ptosis), enophthalmos, contraction of the pupil (miosis), absence of sweating (anhidrosis) (injury to cervical or first thoracic sympathetic chain).
   c. Urogenital problems: retrograde ejaculation due to improper closing of the bladder neck (injury to the hypogastric plexus).
   d. Autonomic dysreflexia.
      (1) Spinal cord injury above sympathetic splanchnic visceral outflow (T6).
      (2) Hypertension, sweating, headache, flushing (return of reflex).

B. Parasympathetic systems: brain stem and sacral spinal cord (visceral and penile erection functions).

1.3 Surgical Anatomy

I. Cervical spine **(Fig. 1.7)**.
A. Bony anatomy **(Fig. 1.8)**.
1. Atlas: no vertebral body, anterior tubercle (longus colli attachment), posterior tubercle (rectus minor and suboccipital membrane attachment), and large transverse processes with transverse foramen (superior and inferior oblique muscle attachment).
   a. The posterior neural arch fuses at 3 years, and anterior neural arch (two sites) fuses at 7 years.
   b. The atlas has large transverse processes, where the superior and inferior oblique muscles attach. The transverse foramen is located within the transverse process, through which the vertebral artery passes.

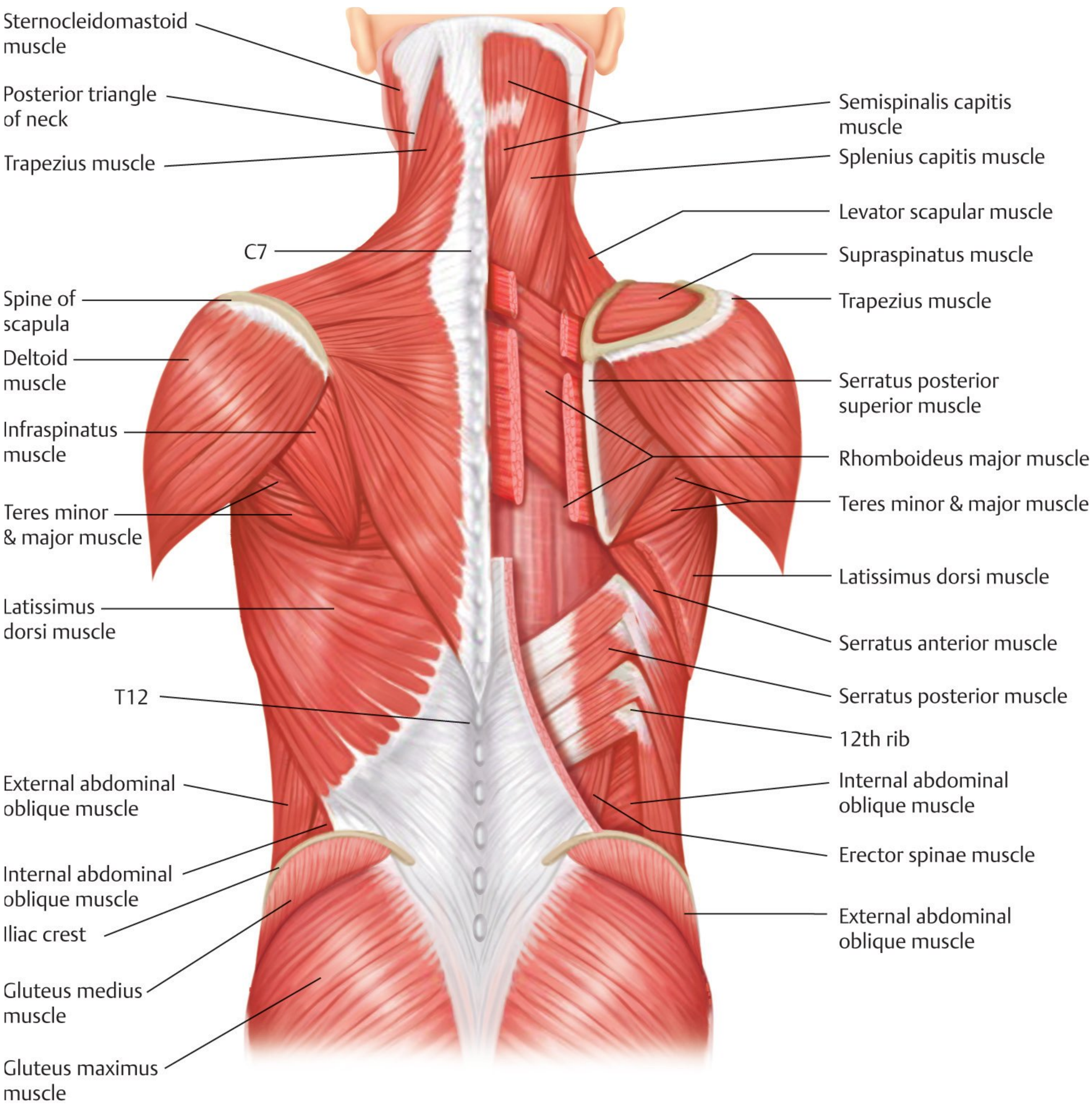

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Fig. 1.7 The human spine. **(a)** Posterior musculature of the spine.

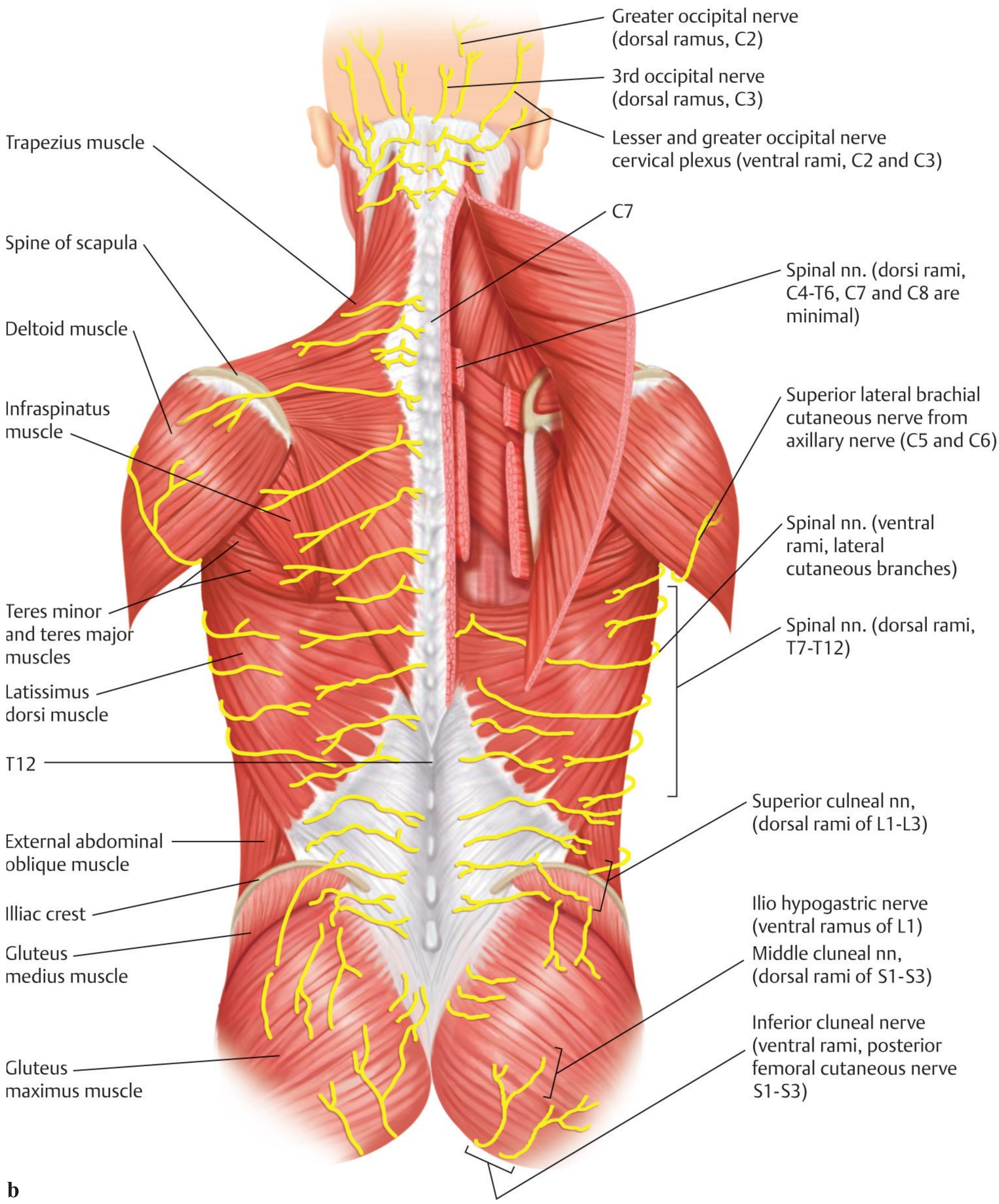


Fig. 1.7 (Continued) The human spine. **(b)** Posterior musculature of the spine and its innervation.

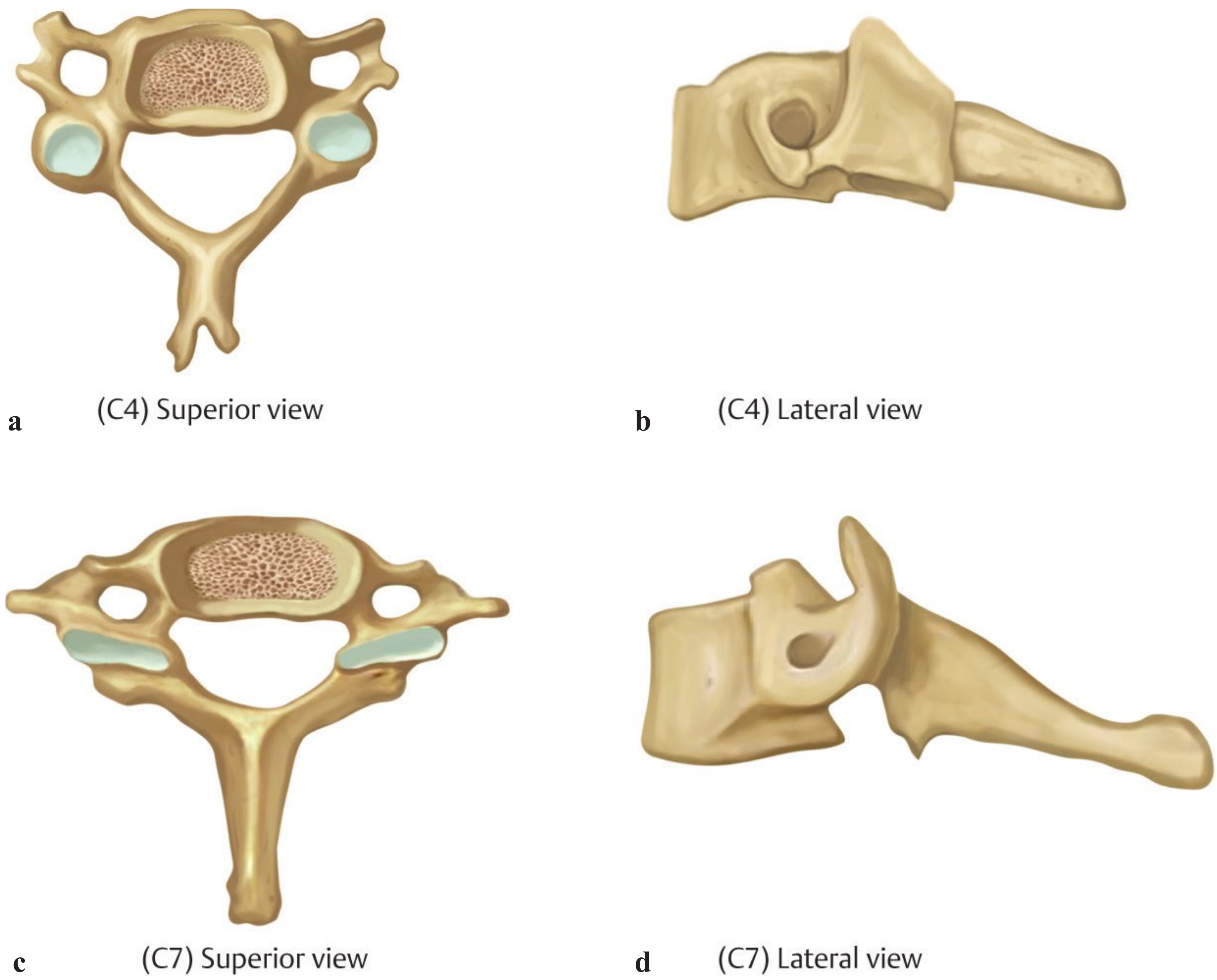


Fig. 1.8 **(a–d)** Morphology of the C4 and C7 vertebrae from superior and lateral views.

c. The posterior arch has a groove along its superior border for the vertebral artery, which runs along it on its way to the foramen magnum of the skull.
d. Superior articular facets form the atlanto-occipital articulation with the occipital condyles. This joint accounts for the majority of flexion and extension of the head.
e. The inferior articular facet contributes to the atlantoaxial joint.
f. The ponticulus posticus (arcuate foramen) is a malformed bony bridge between the posterior superior articular process and the superior margin of the posterior arch of the atlas.
   (1) The arcuate foramen contains the vertebral artery and the suboccipital nerve.
   (2) It can be mistaken for a posterior arch.
   (3) Lateral mass screw placement through the arcuate foramen can cause vertebral artery injury.

2. Axis: odontoid process with oval articular facet anteriorly, making a synovial joint with the anterior arch facet and large bifid spinous process (rectus major and inferior oblique muscle attachment).
   a. The synchondrosis between the dens and arch and the neurocentral cleft between the body and arch fuse at 3 to 6 years.
   b. The pedicle of the axis is large and projects medially at 30° and superiorly at 20°.

   c. The transverse ligament secures the dens to the anterior arch of the atlas. Extension of this ligament superiorly and inferiorly creates the cruciform ligament.
   d. Responsible for the majority of cervical rotation around the axis.
   e. The alar ligaments connect the odontoid to the occipital condyles, further stabilizing the joint.
   f. The transverse processes also contain the transverse foramen with the vertebral artery.
3. C3–C6 vertebrae: bifid spinous processes, pedicle, laminae, articular processes, lateral mass (between the articular processes), transverse processes with anterior and posterior tubercles and transverse foramen (carotid tubercle for C6 anterior tubercle and vertebral artery in the foramen), uncinate processes ("joints of Luschka"), and triangular vertebral foramen.
   a. The superior surfaces of the cervical vertebrae are concave, and the inferior surfaces are convex.
   b. The cervical vertebrae have small bodies compared with the vertebral canal.
   c. The facets gradually become steeper and oriented more sagittally as one progresses down the cervical spine.
      (1) The lateral masses are bony regions between the cervical facets just lateral to the laminae.
   d. The vertebral artery travels within the transverse foramina, dividing it into posterior and anterior tubercles, between which passes the exiting nerve root.
4. C7 vertebra: large, thick spinous process and not bifid.
   a. Transitional vertebra with unique characteristics.
   b. Inferior surface larger than superior surface.
   c. Taller and shallower lateral masses.
   d. The pedicles enlarge starting with C7 and going down the spine.
   e. The spinous process is the site of attachment of the ligamentum nuchae.
   f. It has transverse foramina but the vertebral artery passes through them in only 5%of patients.

B. Ligamentous anatomy and articulation.
1. Atlanto-occipital articulation: articulation between the condyles of the occipital bone superior facets of the atlas, supported by anterior and posterior occipital membranes (continuation of the anterior longitudinal membranes and ligamentum flavum, respectively), and capsule (flexion, extension, and lateral motion).
2. Atlantoaxial articulation **(Fig. 1.9)**.
   a. Rotational movement between the odontoid process and anterior arch of the atlas (responsible for 50%of cervical rotation).
   b. Ligaments.
      (1) Anterior and posterior atlantoaxial ligaments.
      (2) Transverse ligament: across the arch of atlas to hold the dens against the anterior arch of the atlas (cruciform ligament of the atlas: transverse ligament plus superior and inferior extension).

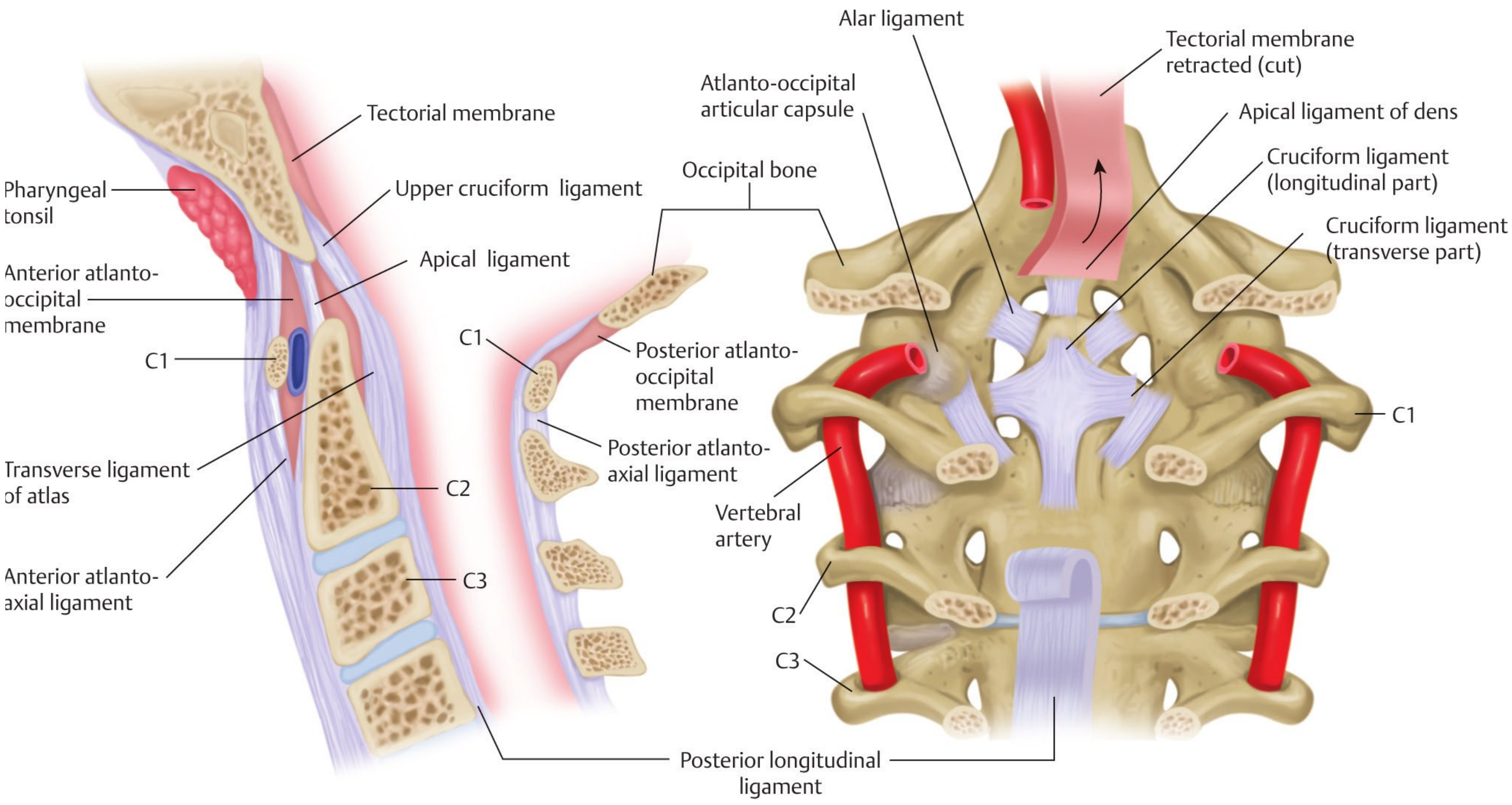


Fig. 1.9 Ligamentous anatomy of the upper cervical spine.

(3) Alar ligament (sides of dens to condyles of occipital bone) and apical ligament (from the apex of the dens to the foramen magnum as a remnant of the notochord in this area).

(4) Tectoral membrane: continuation of the posterior longitudinal membrane.

3. C2–C7 articulation.
   a. Flexion and extension motion.
   b. Facet joint and capsule: horizontal plane (45° oblique) of the joint and weak capsule allow more mobility than lumbar and thoracic vertebrae.
   c. Ligaments.
      (1) Anterior and posterior longitudinal ligaments.
      (2) Ligamentum flavum: from the posterior aspect of the lamina below to the anterior aspect of the lamina above with deficiency in the midline.
      (3) Interspinous ligament: oblique orientation from the posterior superior aspect to the anterior inferior aspect.
      (4) Supraspinous ligament.
      (5) Ligamentum nuchae: fibroelastic septum from the occiput to C7.
   d. Intervertebral disks: annulus fibrosus and nucleus pulposus.

C. Muscles.
1. Posterior muscles.
   a. Superficial: trapezius (from the external occipital protuberance and C7 to the T12 spinous processes to insert at the lateral clavicle, acromion, and spine of the scapula).
   b. Intermediate: splenius capitis and cervicis.
   c. Deep: semispinalis capitis, semispinalis cervicis, and multifidus with rotators.

2. Suboccipital muscles.
   a. Rectus capitis posterior major: C2 spinous process to inferior nuchal line.
   b. Rectus capitis posterior minor: C1 posterior tubercle to inferior nuchal line.
   c. Obliquus capitis inferior: C2 spinous process to transverse process of C1.
   d. Obliquus capitus superior: C1 transverse process to occipital bone between superior and inferior nuchal lines.
3. Anterior muscles.
   a. Platysma: from deltoid and pectoral fascia to mandible and skin, innervated by facial (VII) nerve.
   b. Sternocleidomastoid: from sternum and clavicle to mastoid process.
   c. Strap muscles of larynx: sternohyoid and sternothyroid muscles.
   d. Omohyoid: superior and inferior bellies to depress the hyoid bone.
   e. Longus colli: anterior aspect of the vertebral bodies.

II. Thoracolumbar spine, sacrum, and coccyx.
  A. Bony and ligamentous anatomy **(Fig. 1.9).**
    1. Thoracic vertebrae **(Fig. 1.10).**
       a. Mechanically stiffer and less mobile because of rib attachment.
       b. Physiological kyphosis (primary curve).
       c. The upper and middle thoracic vertebrae have stability against anteroposterior translation, and the lower thoracic vertebrae have stability against rotation due to facet joint orientation.
       d. The transverse processes decrease from T1 to T10.
       e. The spinal canal is circular and has less free space for the spinal cord than the cervical and lumbar region.
       f. Articular facets for ribs: body and transverse process and ligaments (radiate and costovertebral ligaments between the body and rib, and costotransverse and intertransverse ligaments between the transverse process and rib).
       g. Connected to the ribs at the junction between the body and the pedicle and also at the transverse costal facet of the transverse process. The transverse processes are posteriorly angulated to leave room for the ribs.
       h. A heart-shaped body with a possible depression due to the descending aorta on the left side. The spinous processes are long, slender, and downward-pointing so that they overlap the vertebral arches of the inferior vertebra.
    2. Lumbar vertebrae **(Fig. 1.11).**
       a. Strong facet joint and capsule for rotational stability and superior articular processes (mammillary process) are lateral and anterior to the inferior articular process below.
       b. Pedicles: strong and directed posteriorly 1 mm inferior to the tip of the inferior articular process in the middle of the transverse processes. They arise from the upper part of the vertebral body.
       c. Triangular spinal canal.
       d. Ligaments:
          (1) Supraspinous ligament: ends around L3.
          (2) Interspinous ligament: oriented obliquely from cephalad to caudad from posterior to anterior.

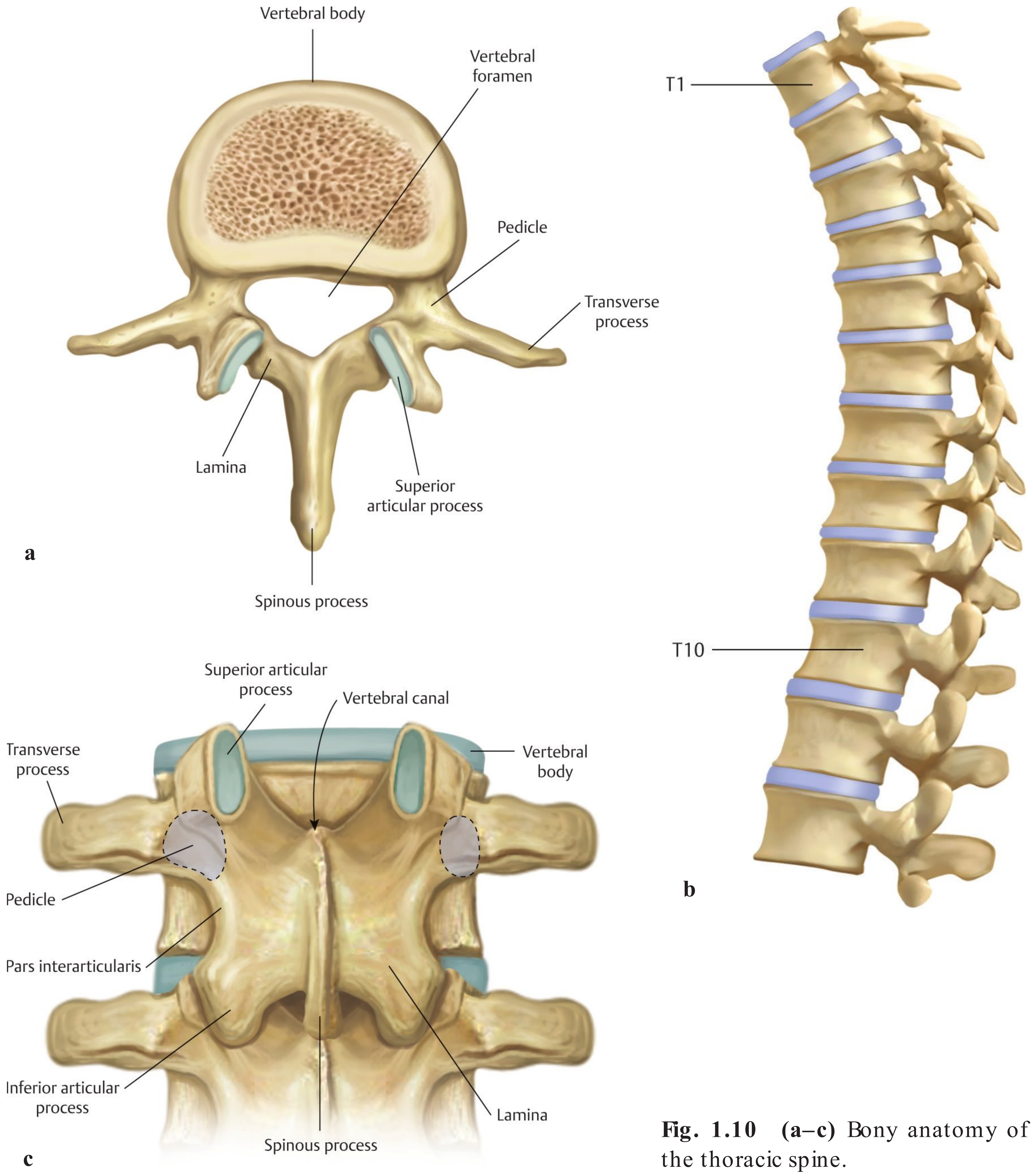


Fig. 1.10 **(a–c)** Bony anatomy of the thoracic spine.

(3) Posterior longitudinal ligament.
(4) Anterior longitudinal ligament.
(5) Ligamentum flavum: from the posterior aspect of the lamina below to the anterior aspect of the lamina above.

e. Kidney-shaped vertebral bodies that are widest transversely.
f. The facets are in a sagittal orientation, limiting the axial rotation. The exception is the L5–S1 facet because it is more coronal to resist anteroposterior translation.
g. More prominent pars interarticularis.
h. Broad and tall spinous process.

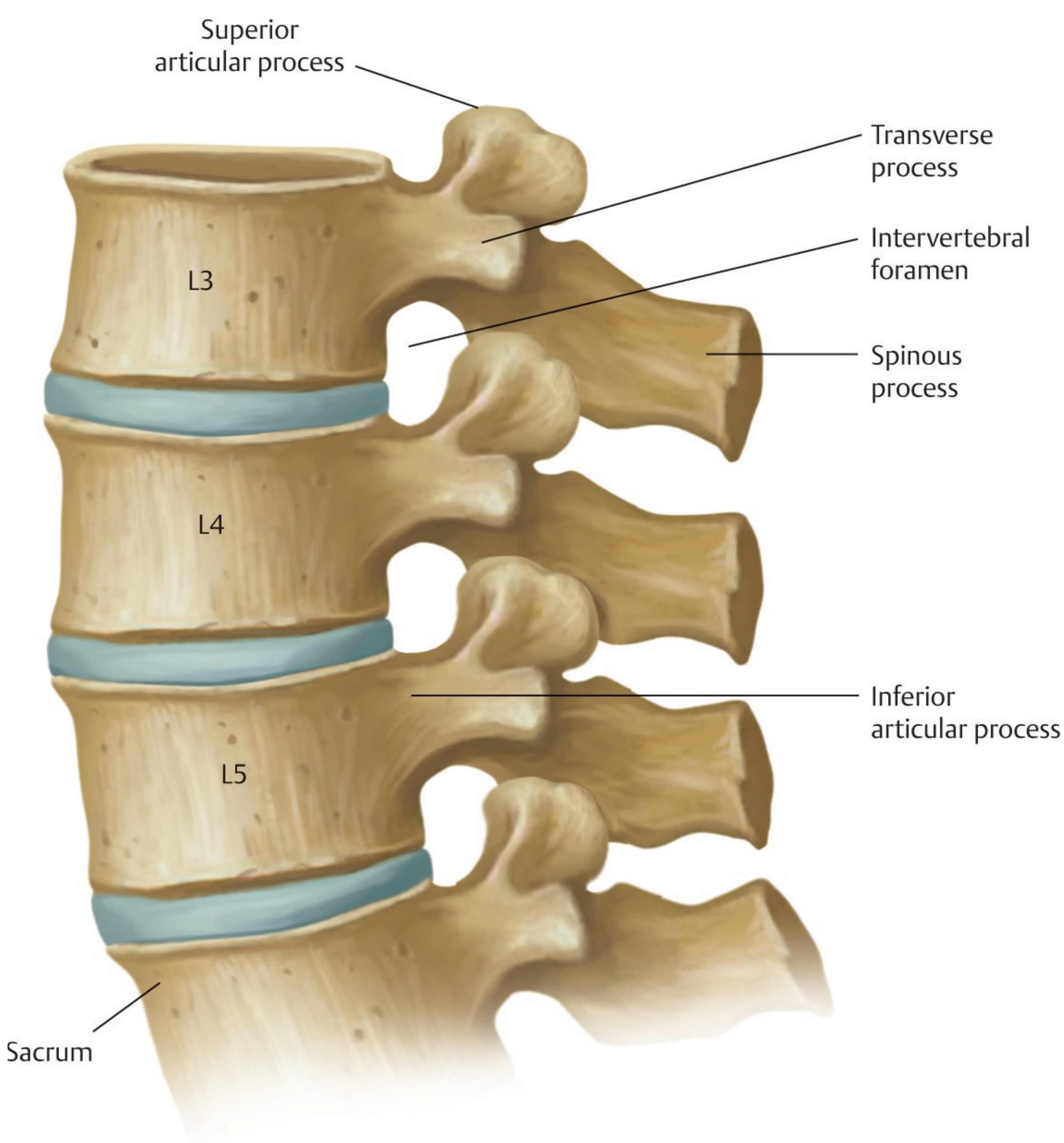
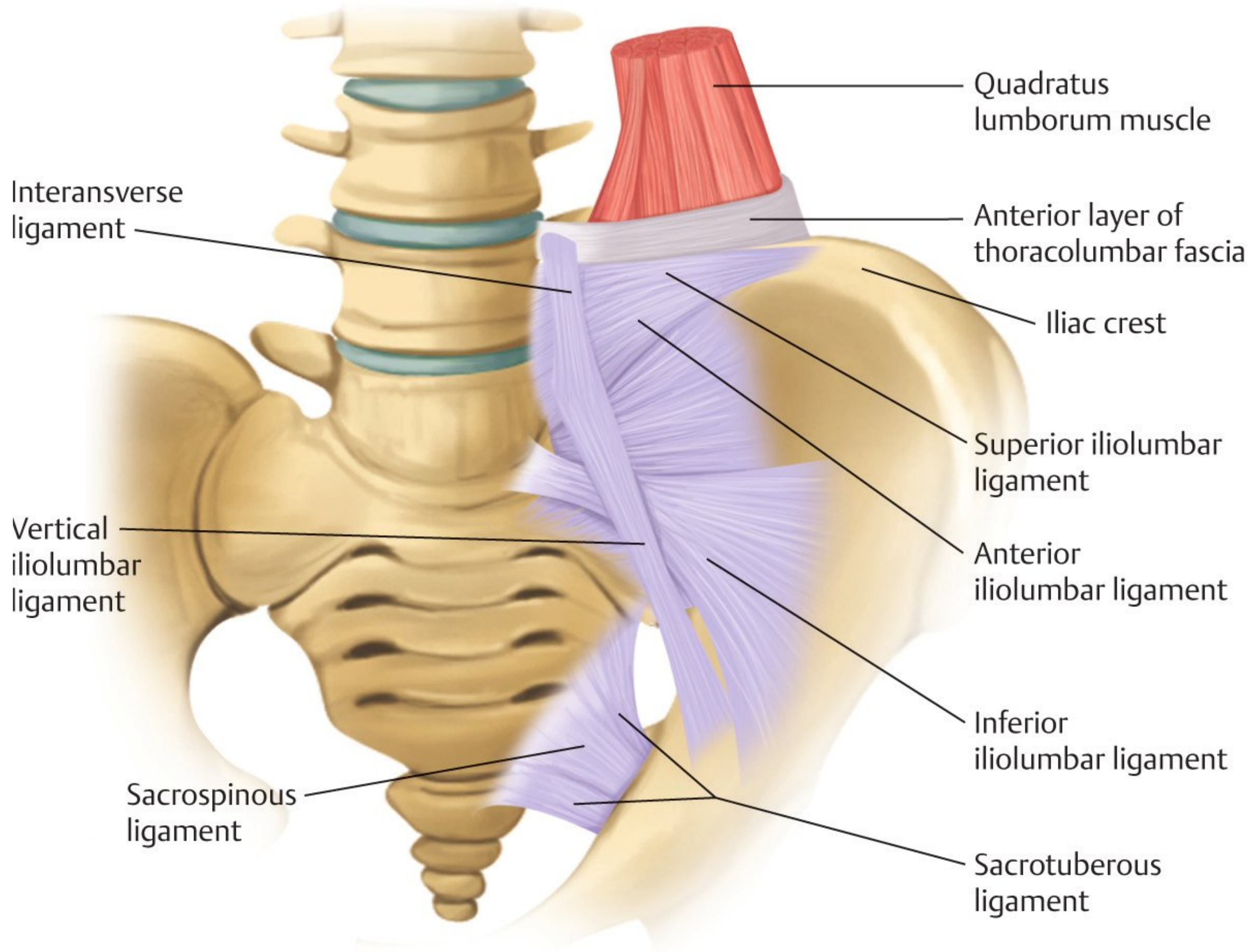


Fig. 1.11 Bony anatomy of the lumbar spine.

i. Smaller transverse process:
   (1) The L5 transverse process attaches to the iliolumbar ligament.
   (2) The accessory process is on the medial aspect of the transverse process where it joins the posterior bony arch.

3. Sacrum and coccyx **(Fig. 1.12).**
   a. Bony structures of the sacrum: ala, promontory, median sacral crest, sacral foramina, articular surface **(Fig. 1.13)**.
   b. Coccyx: three or four elements and the last two to three segments are fused.
      (1) Attachment site for the muscles of the pelvic floor.
   c. Sacroiliac joint.
      (1) Articular process: sacral hyaline cartilage and iliac fibrocartilage.
      (2) Ligaments: interosseous sacroiliac ligament, posterior sacroiliac ligament, and anterior sacroiliac ligament.
   d. Connecting ligaments.
      (1) Sacrotuberous ligament: sacrum to ischial tuberosity.
      (2) Sacrospinous ligament: divides pelvis into greater and lesser sciatic notches.
      (3) Iliolumbar ligaments: L5 transverse processes to ala of sacrum.



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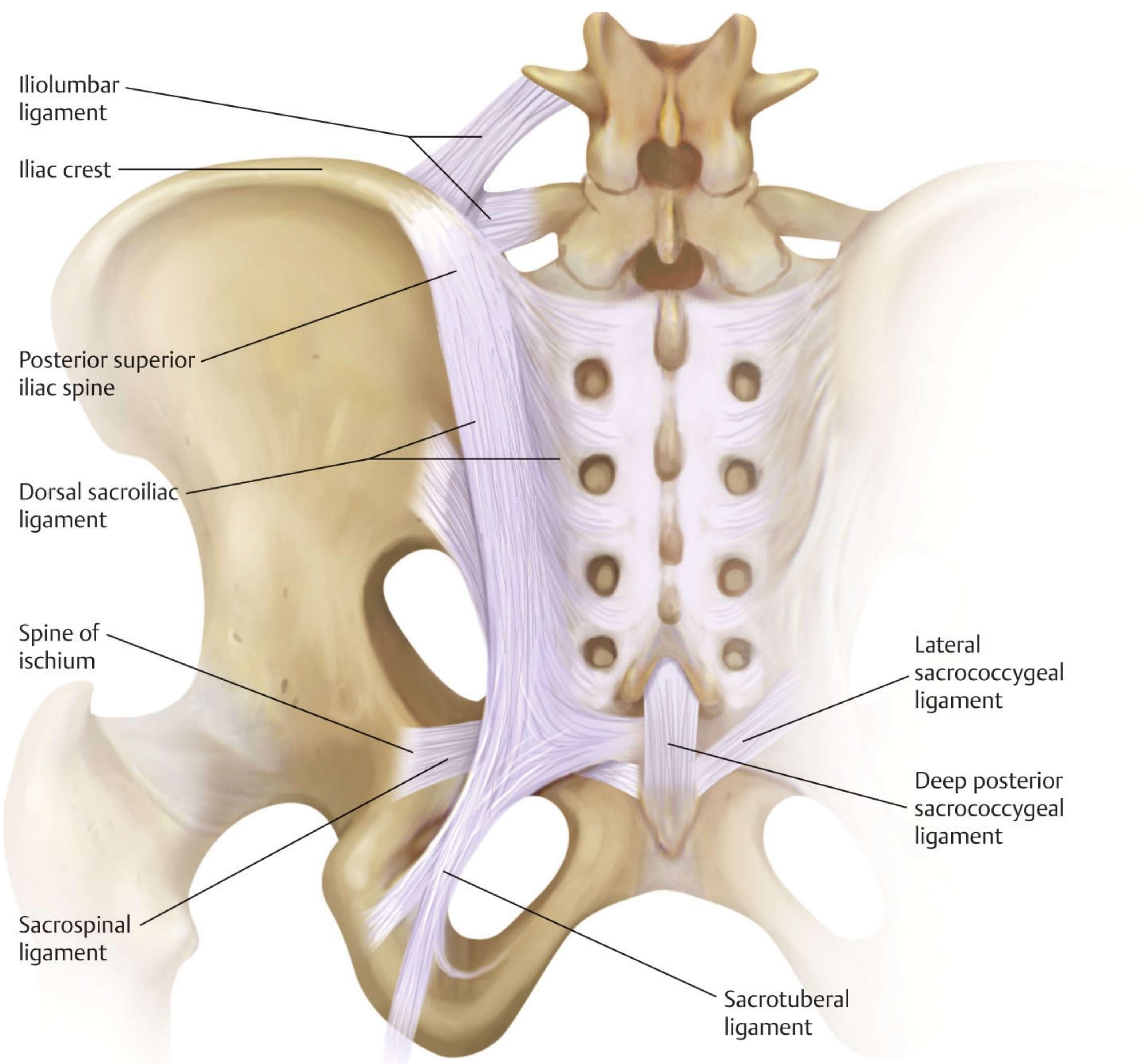


b

Fig. 1.12 **(a)** Anterior and **(b)** posterior anatomy of the sacrum.

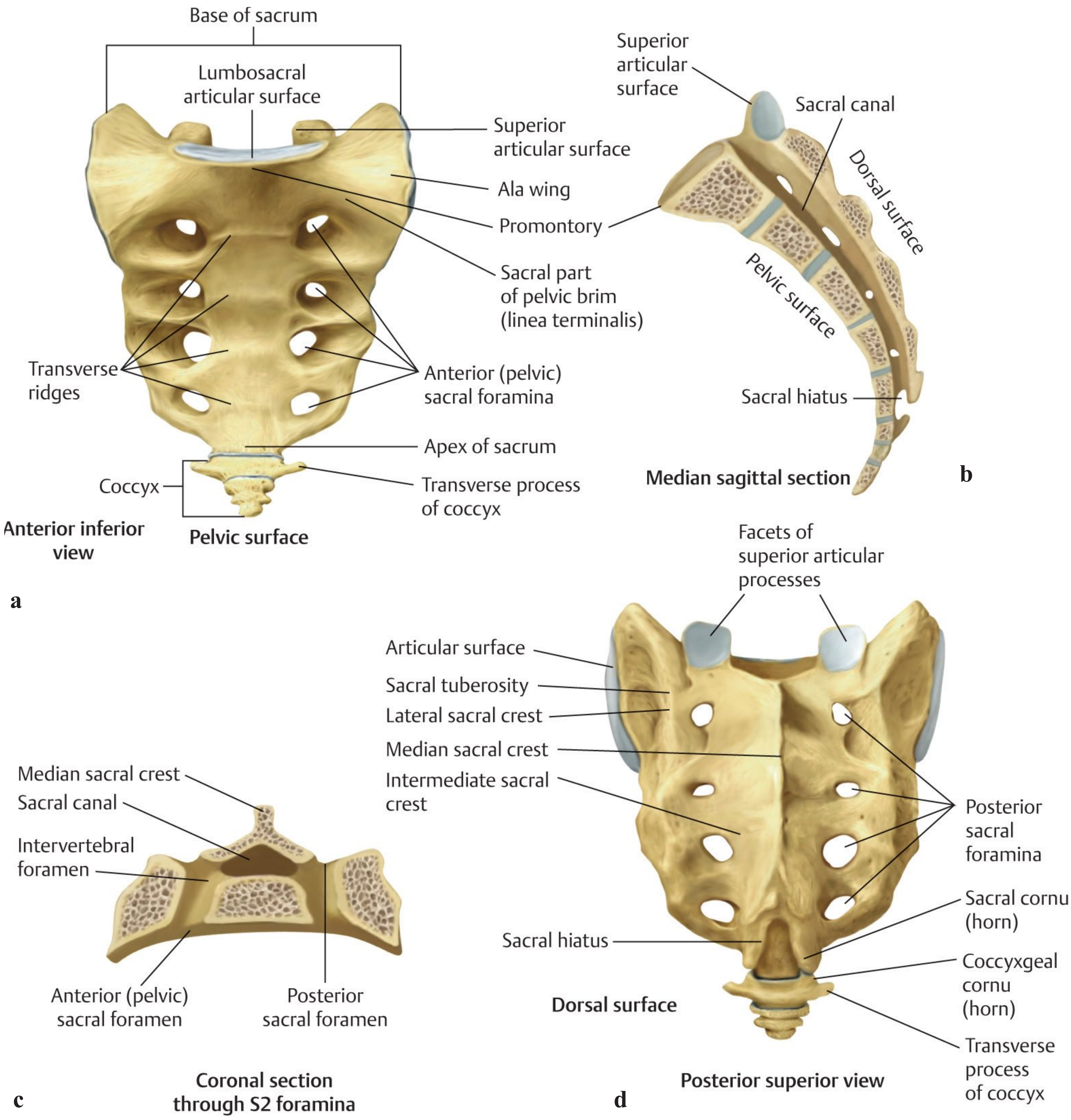


Fig. 1.13 **(a–d)** Bony anatomy of the sacrum.

B. Soft tissue structures.
  1. Muscles.
    a. Superficial.
      (1) Latissimus dorsi: origin from T6 to T12, lumbar spine, sacrum, and posterior iliac crest and lower four ribs to insert at the bottom of the intertubercular groove of the humerus.
      (2) Levator scapulae: origin from C1 to C4 transverse processes to insert at the medial border of the scapula above the spine.
      (3) Rhomboid minor: origin from C7 to T1 to insert at medial border of the scapula at the root of the spine.
      (4) Rhomboid major: origin from T2 to T5 and insertion at the medial border of the scapula below the spine.

b. Deep muscles of the back.
   (1) Superficial layer (transversocostal group or erector spinae): iliocostalis, longissimus, and spinalis.
   (2) Deeper layer (transversospinal group): semispinalis, multifidus, and rotators.
   (3) Deepest layer: interspinales and intertransverse muscles.

1.4 Surgical Approaches

I. Cervical spine.
  A. Posterior approaches: reverse Trendelenburg position and Mayfield tongs help to secure the head and minimize venous bleeding in the surgical field.
    1. Posterior approach to occiput to C1–C2 **(Fig. 1.14)**:
      a. Midline incision from the external occipital protuberance to C2 spinous process (6–8 cm).

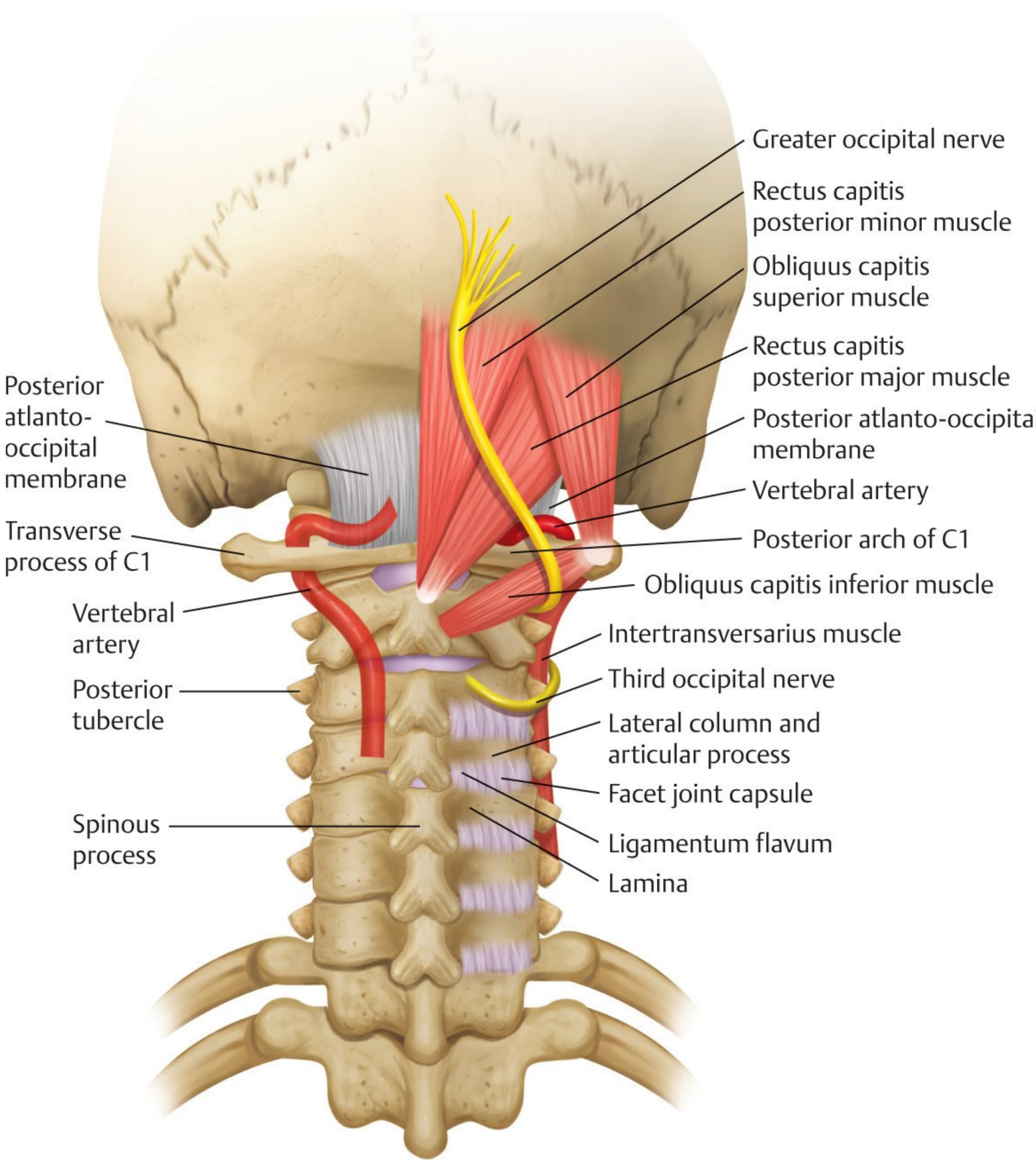


Fig. 1.14 The course of the vertebral artery and the greater occipital nerve in relation to the posterior midline.

b. Ligamentum nuchae and paravertebral muscle dissection to the posterior elements of C1 and C2.
c. Lateral exposure should not go beyond 1.5 cm on the C1 ring (cervical ganglion and vertebral artery), with care not to fracture the C1 ring.
   (1) Aberrant anatomy should be identified (ponticulus posticus).
d. Separate occipitoatlantal and atlantoaxial membranes from the bone and wiring.
e. Occiput: make drill holes above the foramen magnum and remove bone distally for decompression. External occipital protuberance can be used for wiring for fusion.
f. Neurovascular structures:
   (1) Suboccipital nerve (C1): within the suboccipital triangle (motor).
   (2) Greater occipital nerve (C2): beneath and over the inferior oblique muscle (sensory).
   (3) Third occipital nerve (lateral to the suboccipital triangle [sensory]).
   (4) Vertebral artery: from C6 transverse foramen to atlas transverse foramen and pierces the lateral angle of the posterior atlanto-occipital membrane.

2. Posterior approach to the lower cervical spine:
   a. Midline incision down to the spinous processes and lamina (interlaminar space is wide, and caution should be taken to avoid penetration to the dura).
   b. Lateral exposure to the transverse processes, exposing the facets and lateral masses.
   c. Laminectomy, foraminotomy (resection of the medial aspect of superior and inferior facets), or excision of disk or osteophytes of the joints of Luschka to decompress the nerve roots.
      (1) The C5 nerve root forms ~45° with the spinal cord; this angle increases as one descends and is ~90° at the C8 level.
      (2) Nerve roots (foramen borders): disks and joints of Luschka anteriorly, zygapophyseal joints posteriorly, pedicles superiorly and inferiorly. Also, the vertebral artery is anterior to the roots.

B. Anterior approaches: Gardner–Wells tongs are used for traction and to keep the neck slightly extended **(Fig. 1.15)**.
   1. Anterior medial approach to the midcervical spine (Smith–Robinson).
      a. Landmarks:
         (1) Hard palate: arch of the atlas.
         (2) Lower border of mandible: C2–C3.
         (3) Hyoid bone: C3.
         (4) Thyroid cartilage: C4–C5.
         (5) Cricoid cartilage: C6.
         (6) Carotid tubercle: C6.
      b. Use a transverse incision from the midline to the anterior border of the sternocleidomastoid.
      c. Split the platysma longitudinally or transversely.

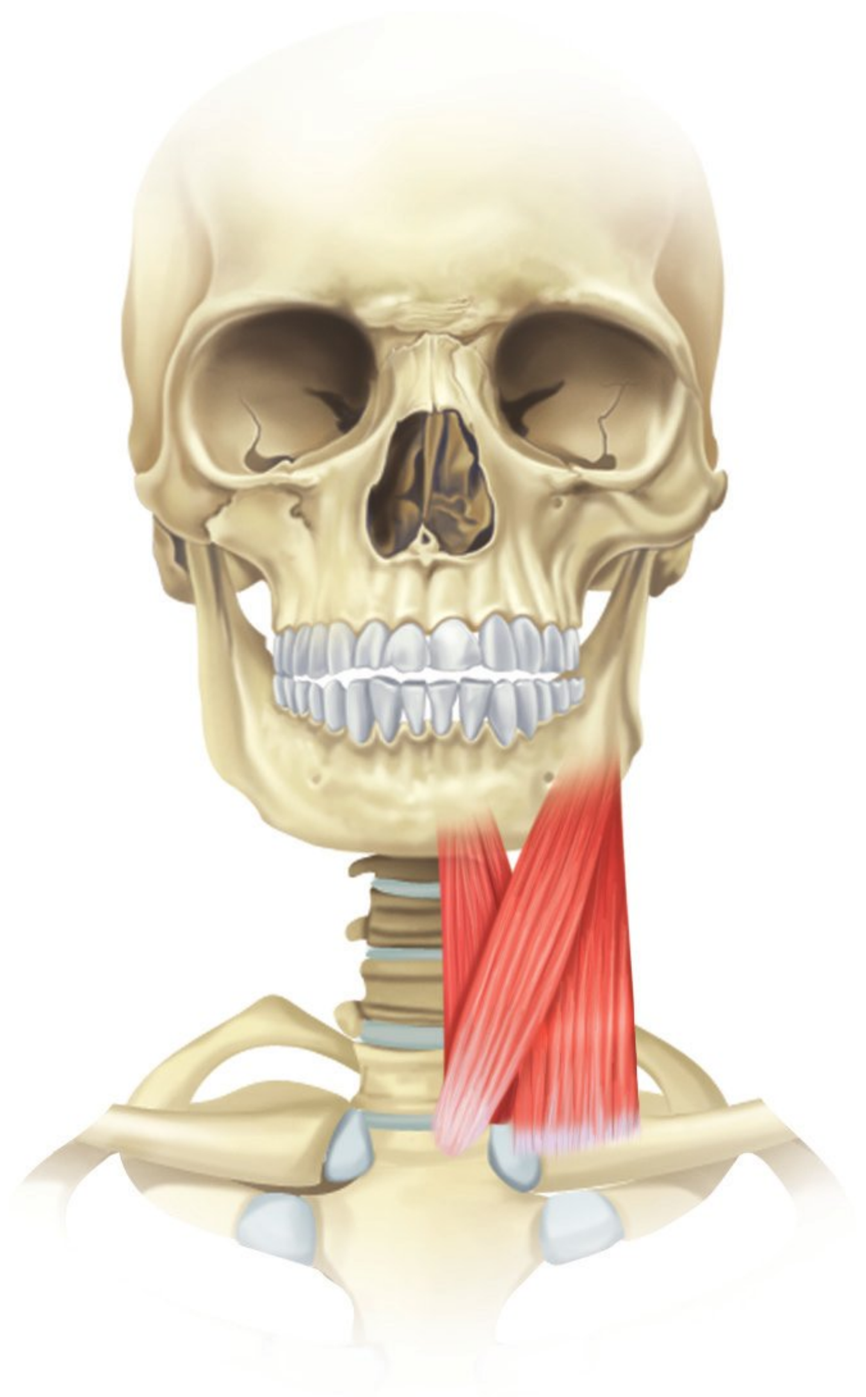


Fig. 1.15 The infrahyoid and sternocleidomastoid muscles.

d. Incise the pretracheal fascia immediately anterior to the sternocleidomastoid, followed by blunt finger dissection to the vertebral bodies, retracting the carotid sheath (carotid artery, internal jugular vein, and vagus nerve) laterally, and retract the strap muscles, trachea, and esophagus medially **(Fig. 1.16)**.
e. The superior thyroid arteries may limit dissection above C3–C4 and the inferior thyroid artery below C6 (may ligate and divide).
f. Divide the prevertebral fascia and anterior longitudinal ligament in the midline, retracting the longus colli laterally.
g. Neurovascular and vital structures **(Fig. 1.17)**:
   (1) Recurrent laryngeal nerve: ascends in the neck between the trachea and esophagus from the arch of the aorta on the left side and runs along the trachea after hooking around the subclavian artery on the right side. It crosses from lateral to medial to the midline trachea in the lower part of the neck, making the right-sided approach slightly more vulnerable. Protect it by placing the retractor below longus colli muscles.
   (2) Sympathetic nerves and stellate ganglion: avoid dissection out onto the transverse processes and keep dissection subperiosteal.
   (3) Carotid sheath contents: from the medial to lateral carotid artery, internal jugular vein, and vagus nerve anterior to the sternocleidomastoid muscle.
   (4) Esophagus: take precaution on deep medial retraction.

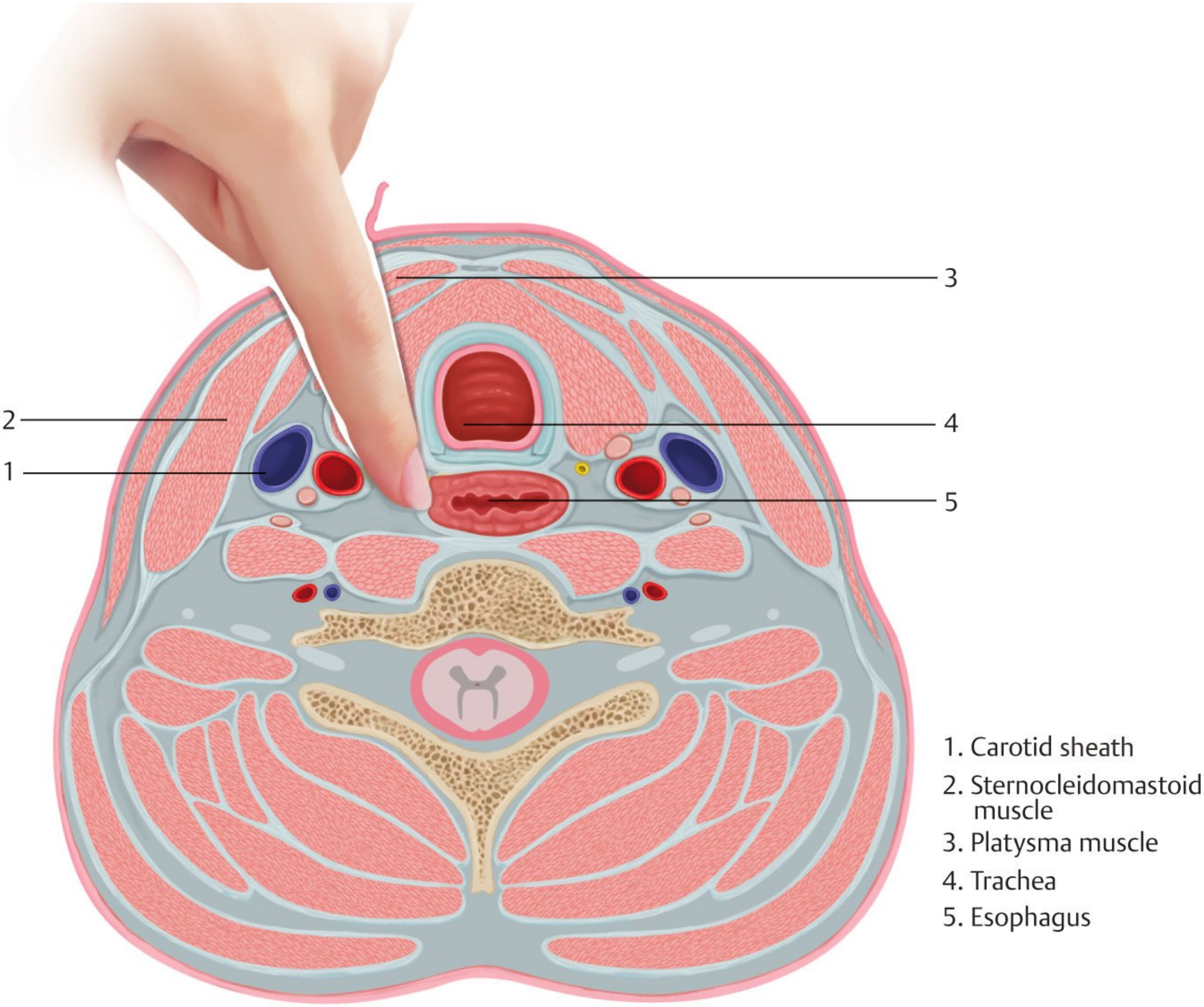


Fig. 1.16 Blunt finger dissection is done through the Smith–Robinson interval toward the anterior cervical spine.

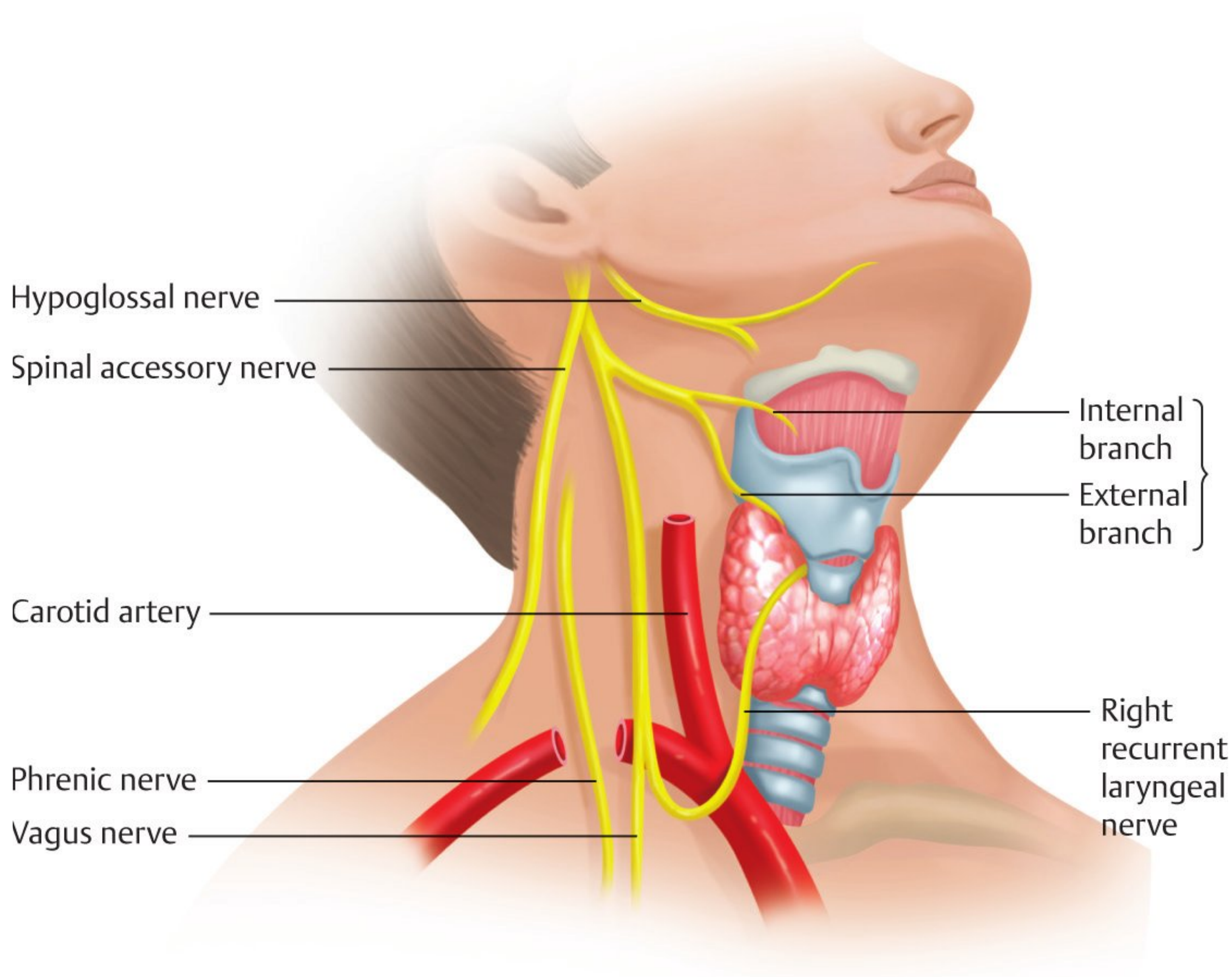
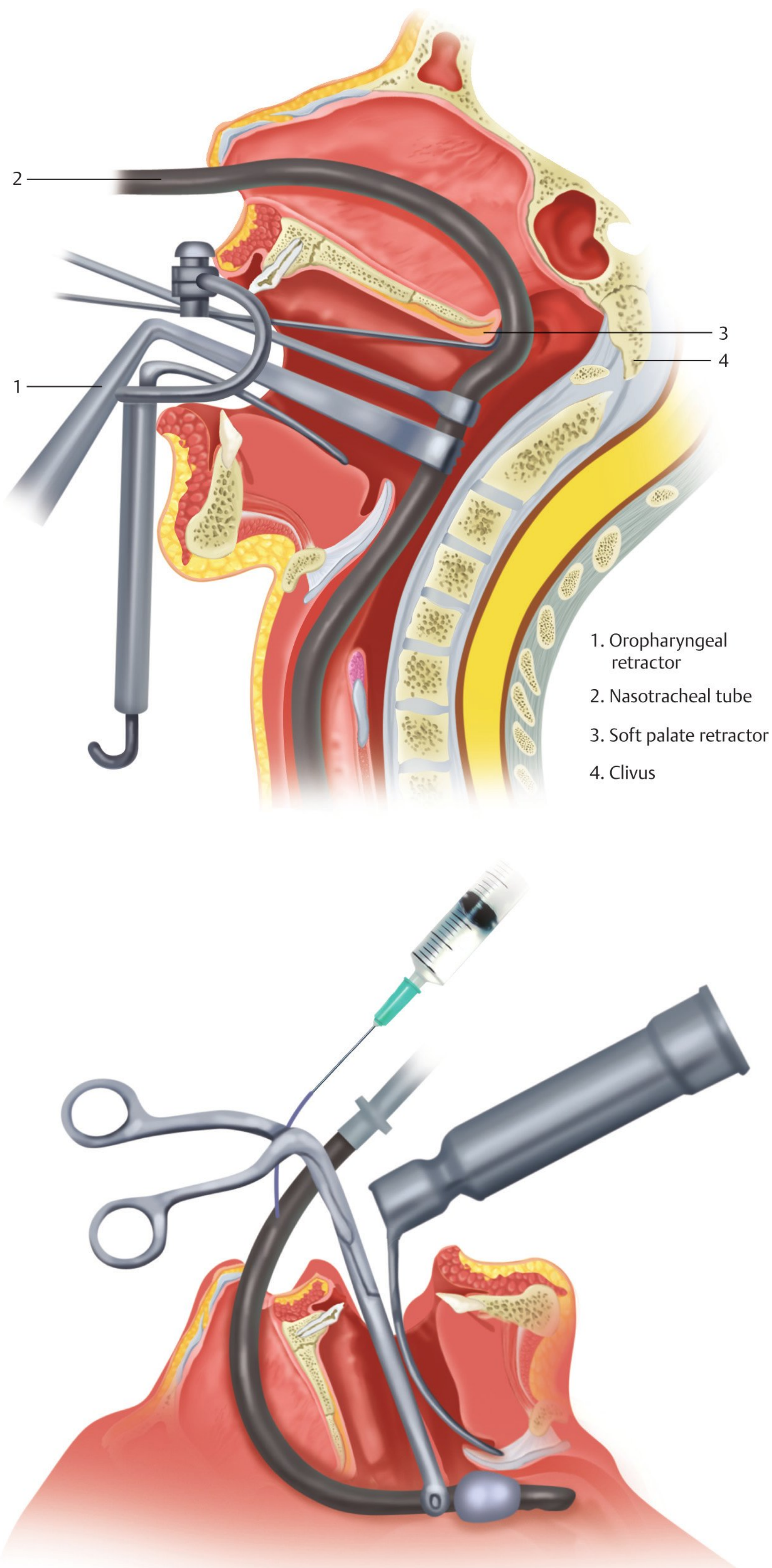


Fig. 1.17 The neural and vascular structures of the neck.

C. Other anterior approaches to the cervical spine:

1. Transoral approach to C1–C2 (**Fig. 1.18**):



a

b

Fig. 1.18 (a,b) The transoral approach, which allows exposure of the midline between the arch of the atlas and C2. The exposure may be extended cephalad by dividing the soft and hard palate to allow access to the foramen magnum and lower half of the clivus.

a. Fiberoptic nasotracheal intubation and nasogastric tube are used.
b. The patient is placed in the supine position with the head held in slight extension using the Mayfield frame.
c. The oral cavity is cleansed with chlorhexidine, and perioperative antibiotics with an intravenous cephalosporin and metronidazole are instituted for 72 hours as prophylaxis against wound infection.
d. The key surgical landmark is the anterior tubercle on the atlas to which the anterior longitudinal ligament and longus colli muscles are attached.
e. The transoral retractors are inserted, exposing the posterior oropharynx. The area of the incision is infiltrated with 1:200,000 epinephrine.
f. A midline 3 cm vertical incision centered on the anterior tubercle is made through the pharyngeal mucosa and muscle.
g. The tubercle of the atlas and anterior longitudinal ligament are exposed superiosteally, and the longus colli muscles are mobilized laterally.
h. A high-speed bur may be used to remove the anterior arch of the atlas to expose the odontoid process.

2. An anteromedial approach to the upper cervical spine by de Andrade and Macnab:
   a. The neck is hyperextended, and the chin is turned to the opposite side.
   b. A skin incision is made along the anterior aspect of the sternocleidomastoid muscle and curved toward the mastoid process.
   c. The platysma and the superficial layer of the deep cervical fascia are divided in the line of the incision to expose the anterior border of the sternocleidomastoid.
   d. The sternocleidomastoid muscle is retracted anteriorly and the carotid artery laterally.
   e. The superior thyroid artery and lingual vessels are ligated. The facial artery is identified at the upper portion of the incision, which helps to find the hypoglossal nerve adjacent to the digastric muscle.
   f. The superior laryngeal nerve is in close proximity to the superior thyroid artery, and excessive retraction of this nerve should be avoided.
   g. Stripping of the longus colli muscle exposes the anterior aspect of the upper cervical spine and basiocciput.
3. Anterior retropharyngeal exposure of the upper cervical by McAfee:
   a. A right-sided submandibular transverse incision and division of the platysma leads to the sternocleidomastoid muscle and its deep cervical fascia.
   b. The mandibular branch of the facial nerve should be identified with the aid of a nerve stimulator, and the retromandibular vein is ligated during the initial stage of dissection.
   c. The anterior border of the sternocleidomastoid muscle is mobilized. The submandibular salivary gland and the jugular digastric lymph nodes are resected.
   d. Care should be taken to suture the duct in the salivary gland to prevent a salivary fistula.
   e. The digastric tendon is divided and tagged for later repair.
   f. The hypoglossal nerve is next identified and mobilized. The carotid sheath is opened, and arterial and venous branches are ligated, including the superior thyroid artery and vein, lingual artery and vein, ascending

pharyngeal artery and vein, and facial artery and vein, beginning inferiorly, progressing superiorly.

g. The superior laryngeal nerve is also identified and mobilized.

h. The prevertebral fasciae are transected longitudinally to expose and dissect the longus colli muscles.

4. The anterolateral retropharyngeal approach by Whitesides and Kelley:

   a. The skin incision is made from the mastoid along the anterior aspect of the sternocleidomastoid.

   b. The external jugular vein is ligated, and the greater auricular nerve is spared if possible.

   c. The sternocleidomastoid and splenius capitus muscles are detached from the mastoid, leaving a fascial edge for later repair. The spinal accessory nerve should be identified and protected.

   d. Retract the carotid contents along with the hypoglossal nerve anteriorly, while retracting the sternocleidomastoid posteriorly. Blunt dissection leads to the transverse processes and anterior aspect of C1–C3.

5. Lateral approach to the cervical spine by Verbiest:

   a. The exposure is achieved by dissecting anterior to the carotid sheath and exposing the vertebral artery and nerve roots posterior to the transverse processes.

   b. This lateral approach may be used for lesions that are localized laterally or if the vertebral artery must be exposed.

6. Cervicothoracic junction: anterior exposure of the upper thoracic vertebrae may be accomplished through the low cervical, supraclavicular approach, sternum-splitting approach, or transthoracic approach **(Fig. 1.19, Fig. 1.20, Fig. 1.21, Fig. 1.22,** and **Fig. 1.23)**.

   a. Low cervical approach to C6–T2: an extension of the anteromedial approach to the lower cervical spine.

   b. The supraclavicular approach to C6–T2.

      (1) A transverse incision above the clavicle and a dissection posterior to the carotid sheath.

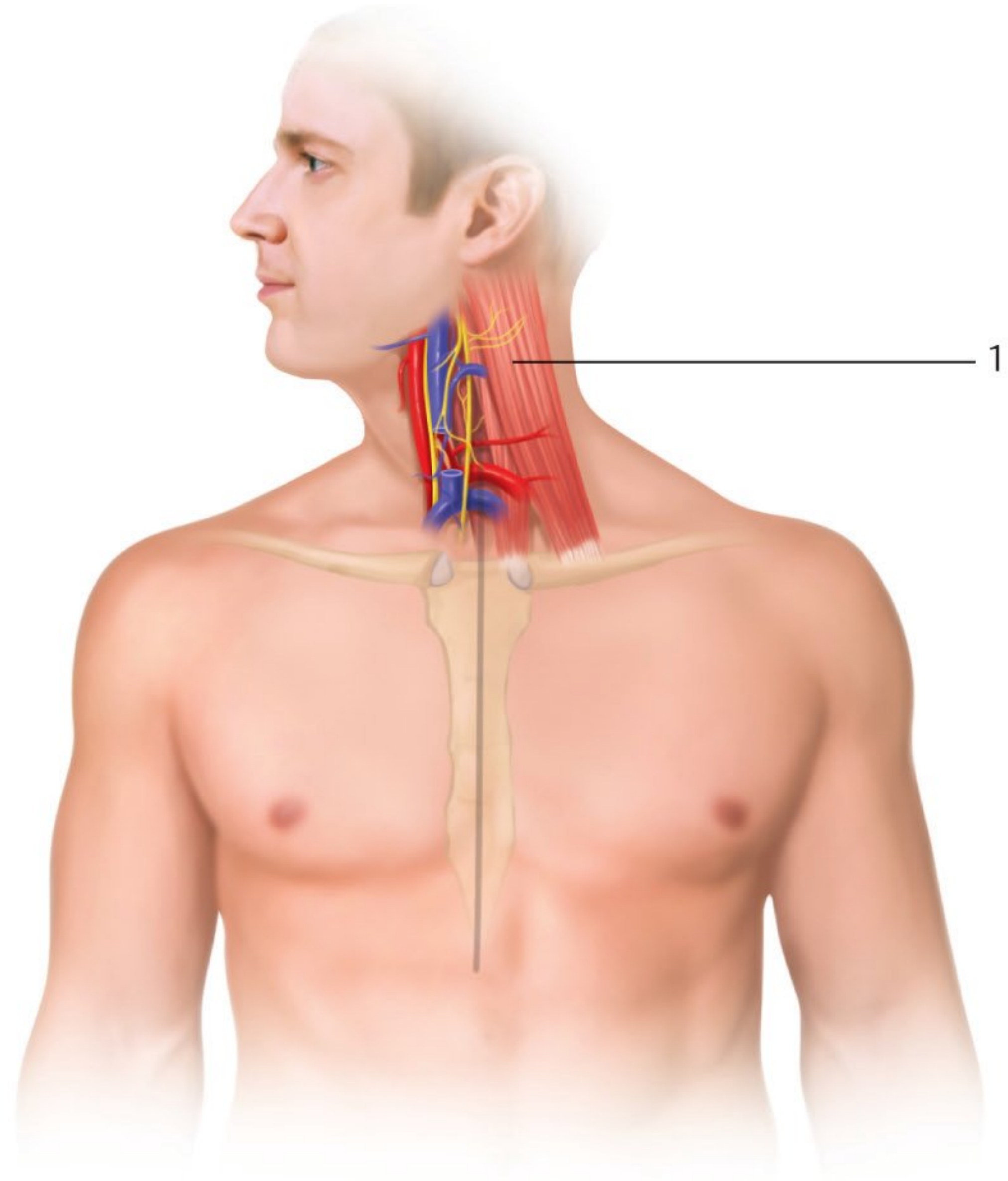
      (2) After incision of the platysma muscle, the clavicular head of the sternocleidomastoid is divided. The fascia beneath is divided to release the omohyoid from its pulley.

      (3) The subclavian artery and its branches, which include the thyrocervical trunk, suprascapular artery, and transcervical artery, must be identified. The dome of the lung and the phrenic nerve are in close proximity to the scalenus anterior muscle.

      (4) Division of the scalenus anterior muscle exposes the Sibson's fascia in the floor of the wound, which covers the dome of the lung.

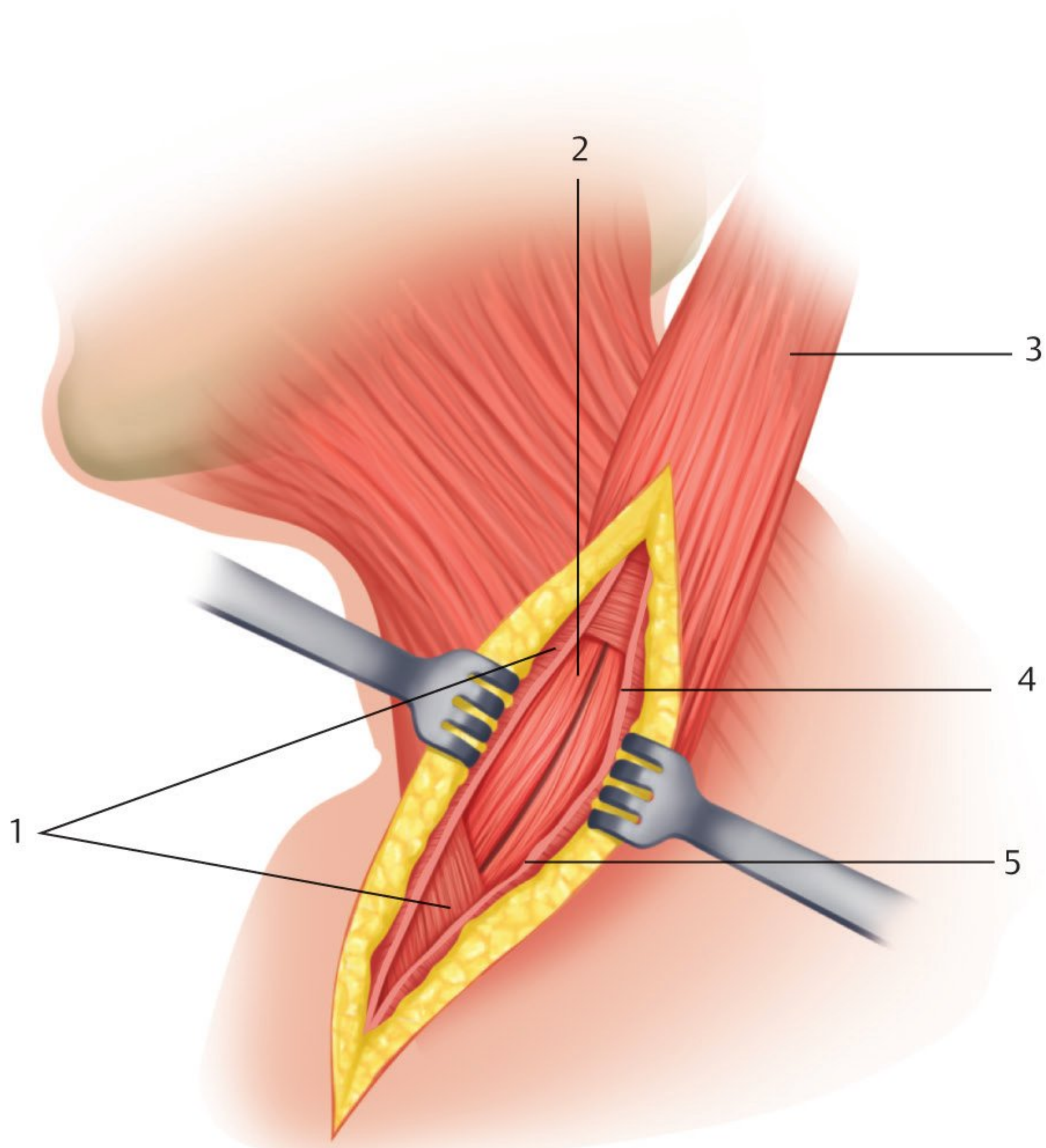
      (5) Sibson's fascia is divided transversely using scissors, and the visceral pleura and lung should be retracted inferiorly.

      (6) The trachea, the esophagus, and the recurrent laryngeal nerve must be protected during medial retraction. The posterior thorax, stellate ganglion, and upper thoracic vertebral bodies are now visible looking from above downward through the thoracic inlet. The recurrent laryngeal nerve should be identified and protected. Likewise, the inferior thyroid artery and vertebral artery should be identified. The thoracic duct should be identified if approached from the left.



a

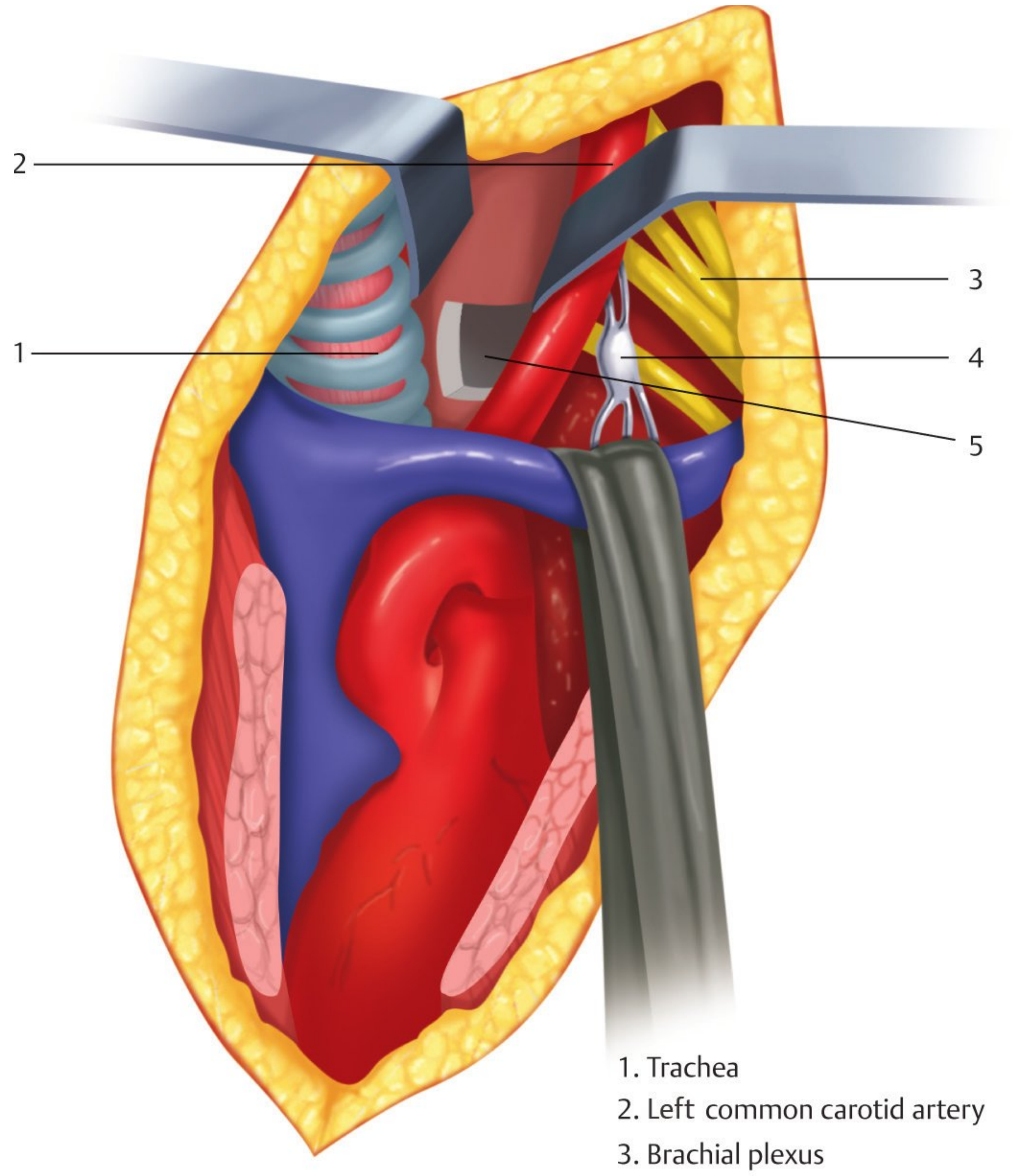
1. Sternocleidomastoid muscle



1. Platysma muscle
2. Strap muscle
3. Sternocleidomastoid muscle
4. Fascia over platysma muscle
5. Deep cervical fascia over sternocleidomastoid muscle

b

Fig. 1.19 **(a)** The sternal-splitting approach. **(b)** After division of the platysma, the deep cervical fascia is divided sharply.



1. Trachea
2. Left common carotid artery
3. Brachial plexus
4. Sympathetic chain
5. Dura mater

Fig. 1.20 To complete the exposure, the esophagus, trachea, and brachiocephalic trunk are gently retracted to the right, and the thoracic duct is retracted to the left.

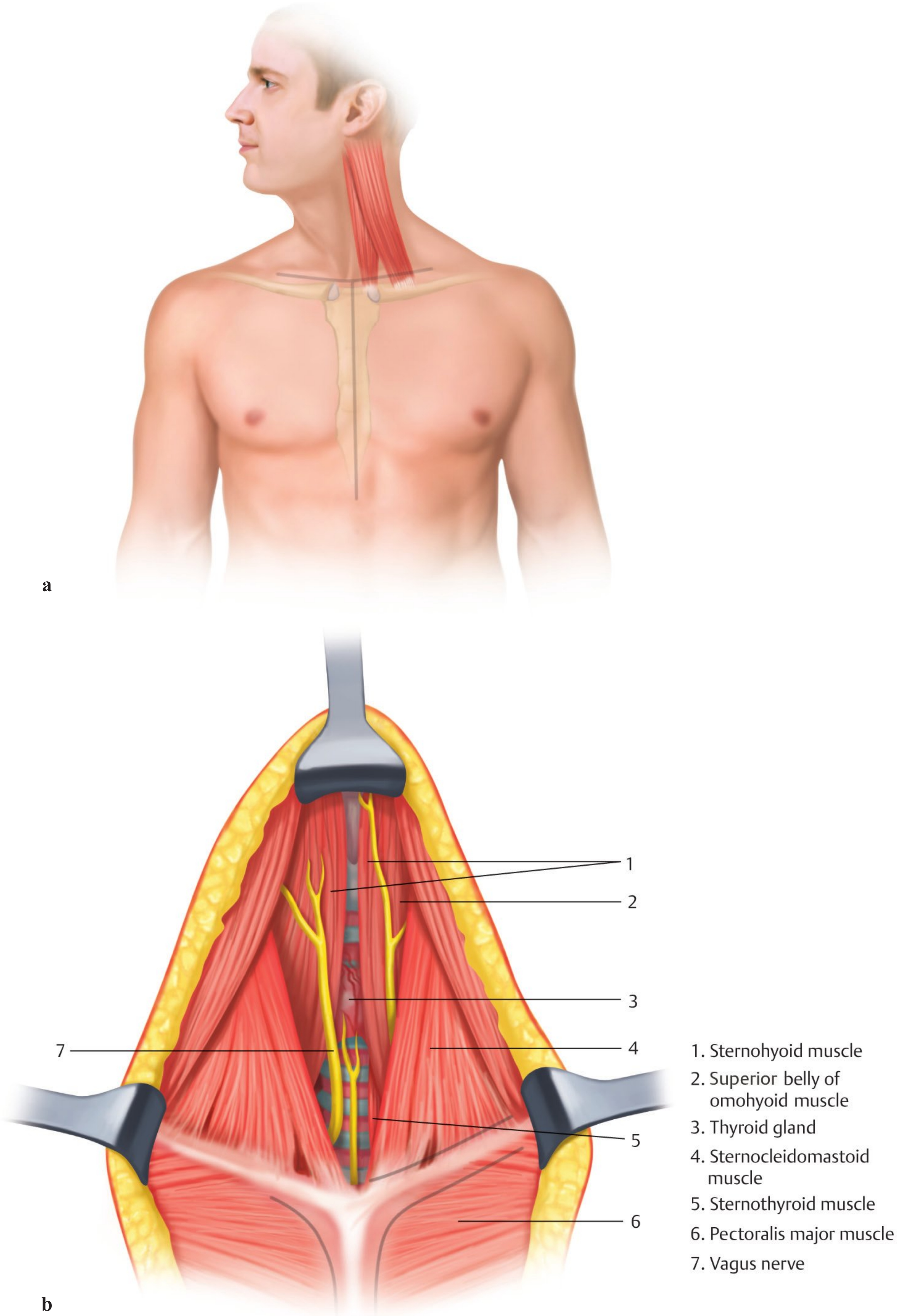


Fig. 1.21 **(a)** A T-shaped incision is performed during the sternal splitting approach. **(b)** Deeper exposure reveals the sternocleidomastoid and pectoralis major muscles.

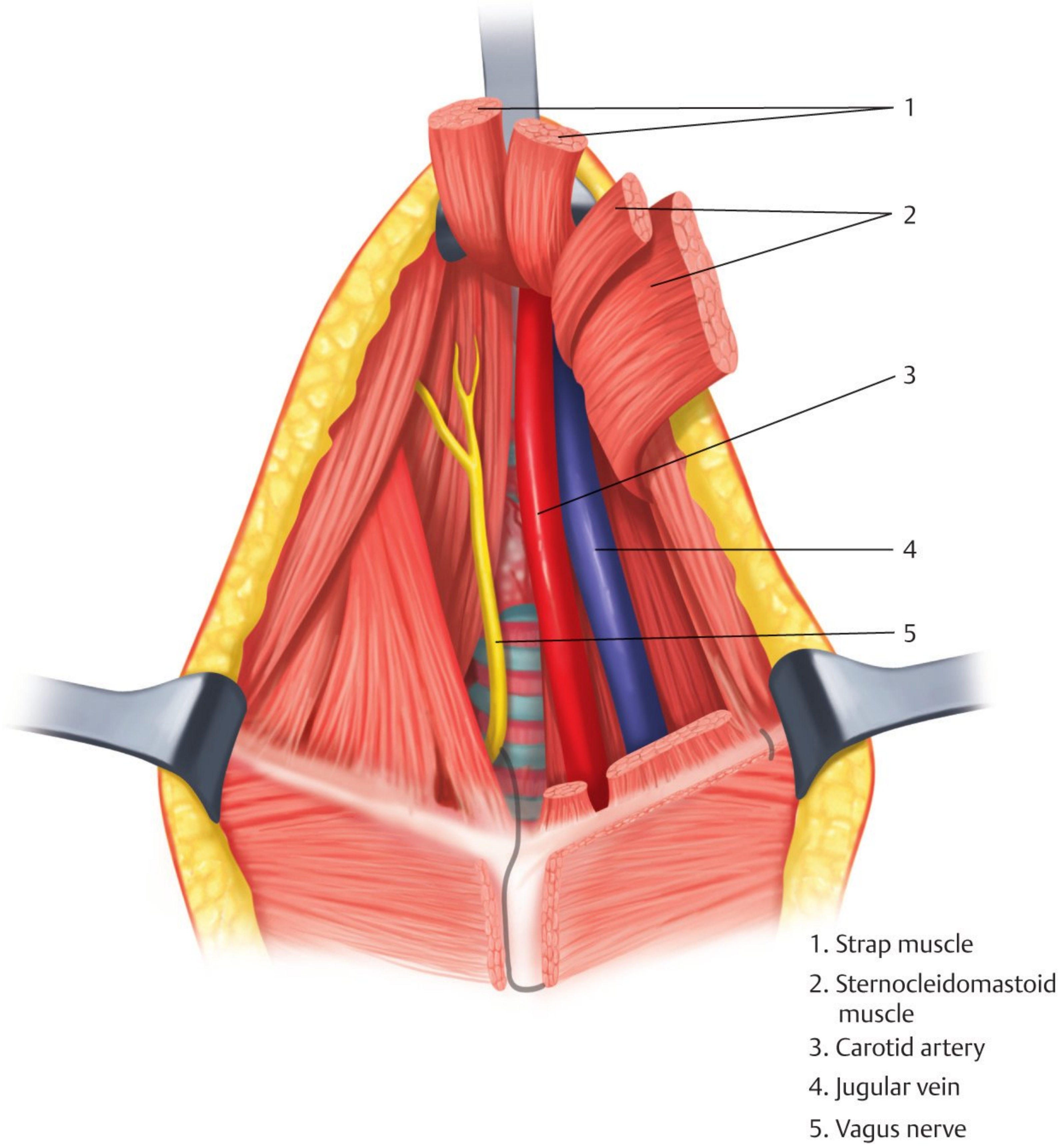
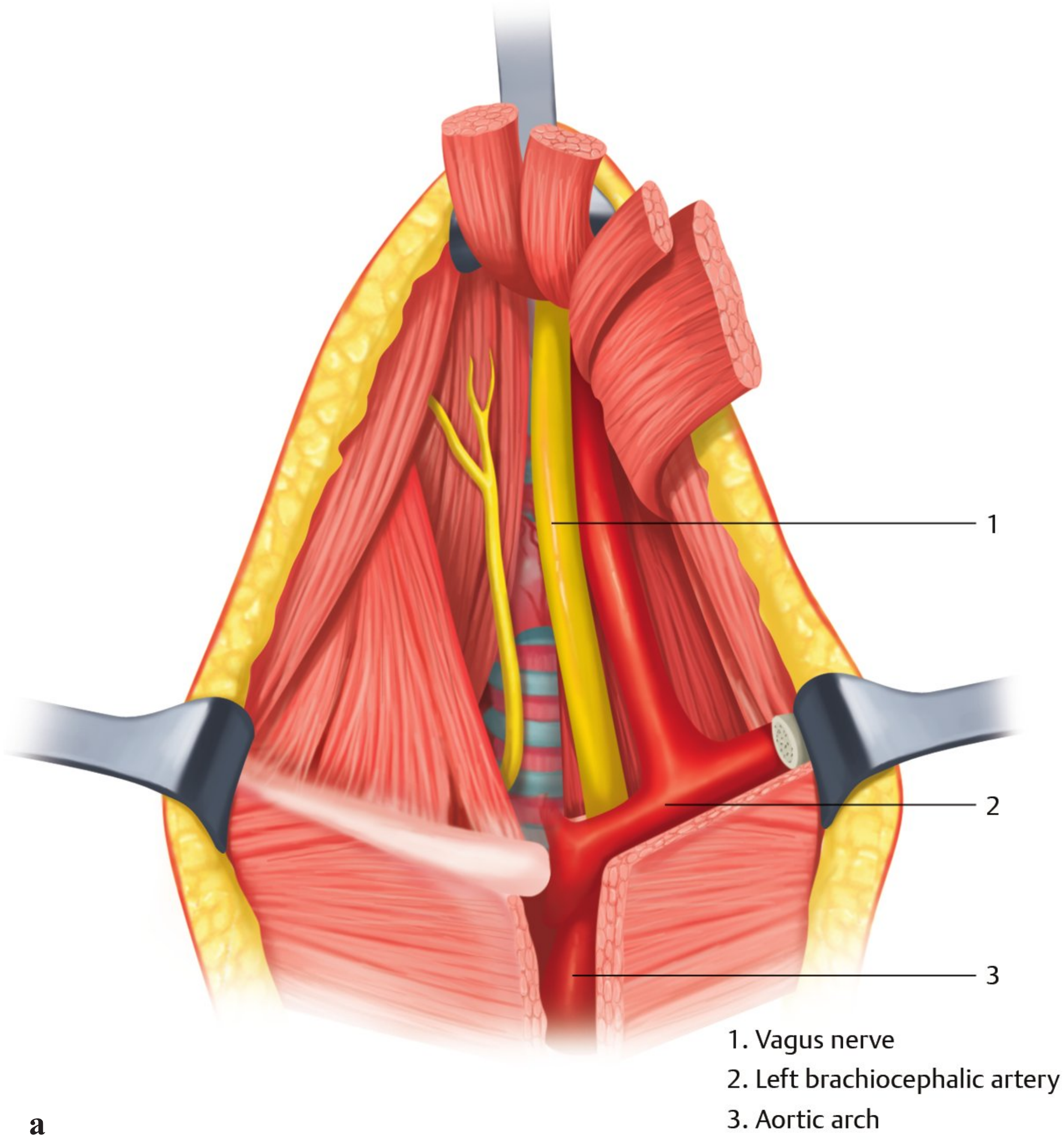


Fig. 1.22 The sternal and clavicular heads of the sternocleidomastoid are detached at the level of the manubrium.



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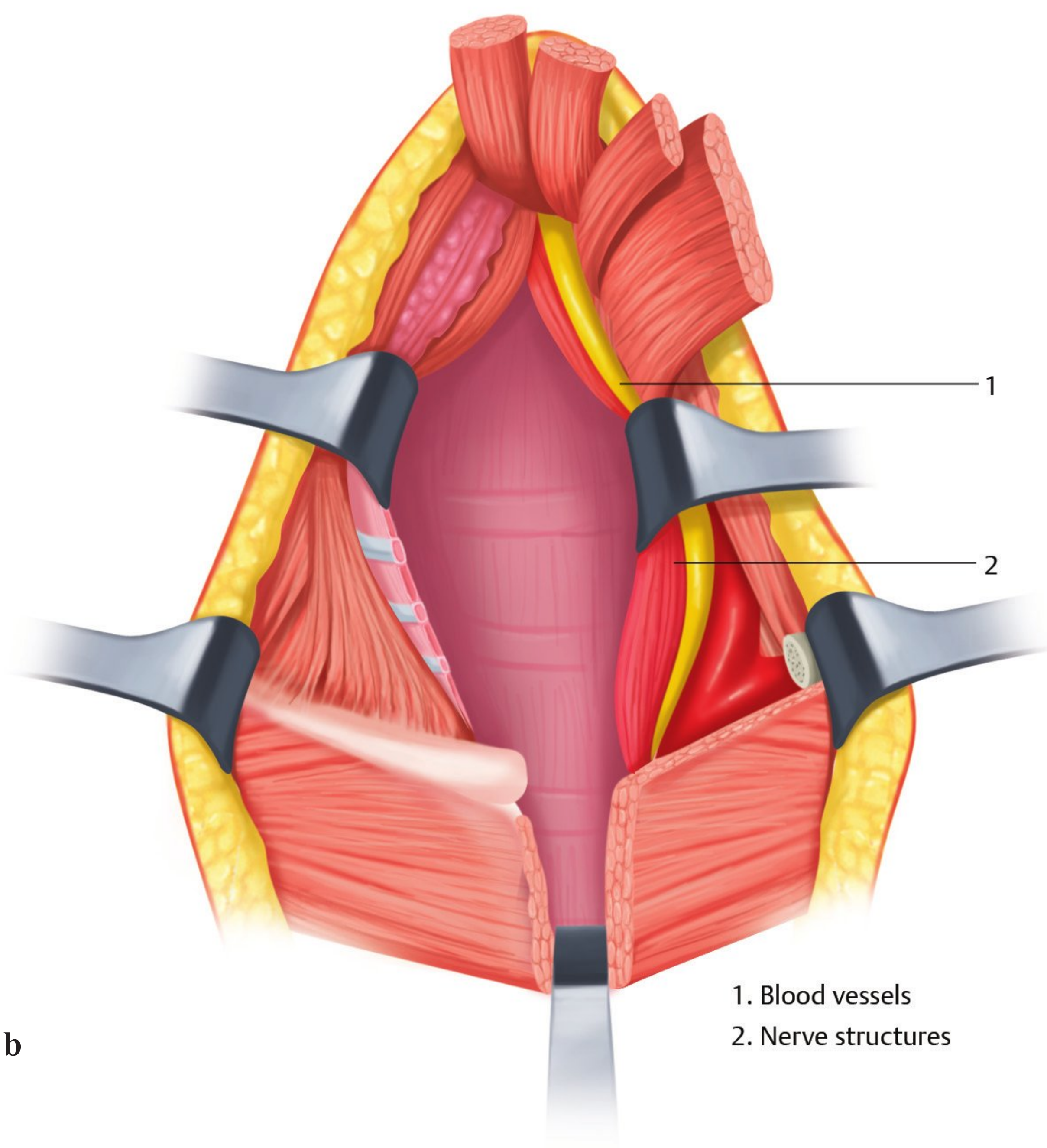


b

Fig. 1.23 **(a)** The medial third of the clavicle and a rectangular piece of the manubrium are removed. **(b)** Retraction of the vessels and trachea exposes the cervicothoracic junction.

c. Thoracotomy to T1–T4 **(Fig. 1.24, Fig. 1.25, Fig. 1.26, Fig. 1.27, Fig. 1.28,** and **Fig. 1.29).**

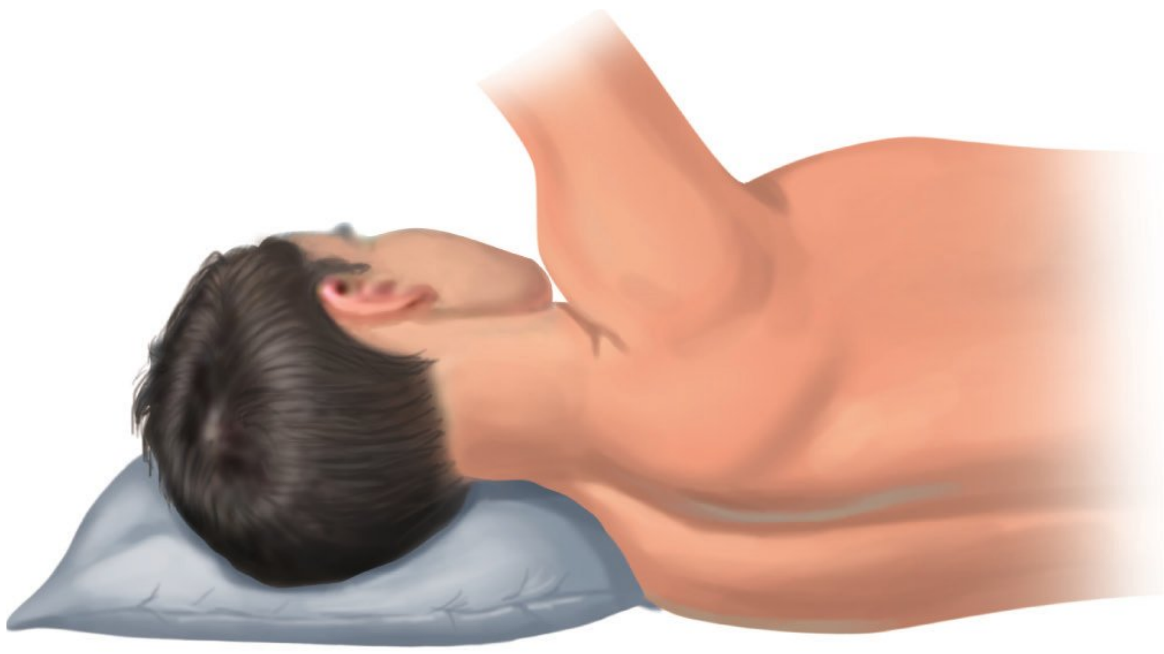
(1) The right-sided approach is preferred to avoid the left subclavian artery, which is more curved than the right brachiocephalic artery.

(2) The incision is medial and inferior to the scapula. The scapula is retracted laterally by dividing the trapezius, latissimus dorsi, rhomboids, and levator scapulae muscles.

(3) The chest is entered through the third rib.

(4) The posterior 7 to 10 cm of each of the second, third, fourth, and fifth ribs may be removed.

(5) Exposure of the vertebrae is made with an **L**-shaped incision in the pleura.



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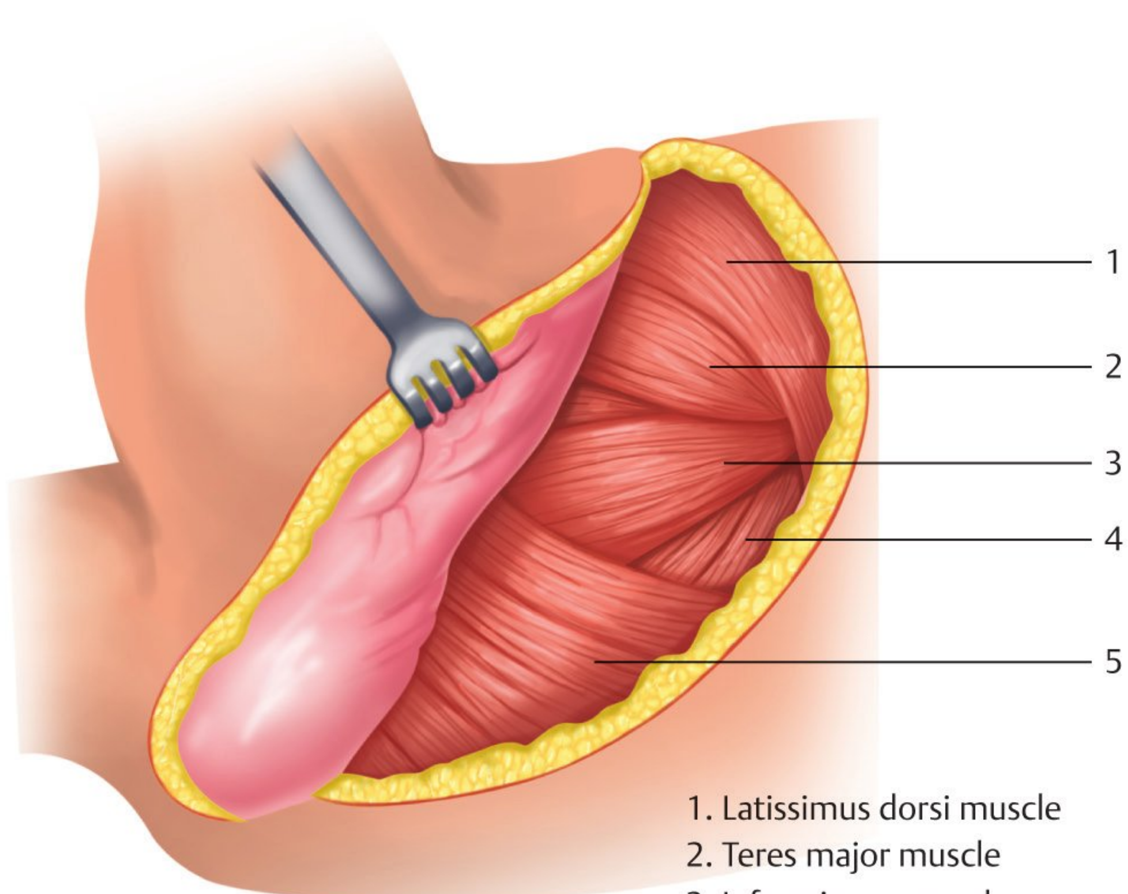
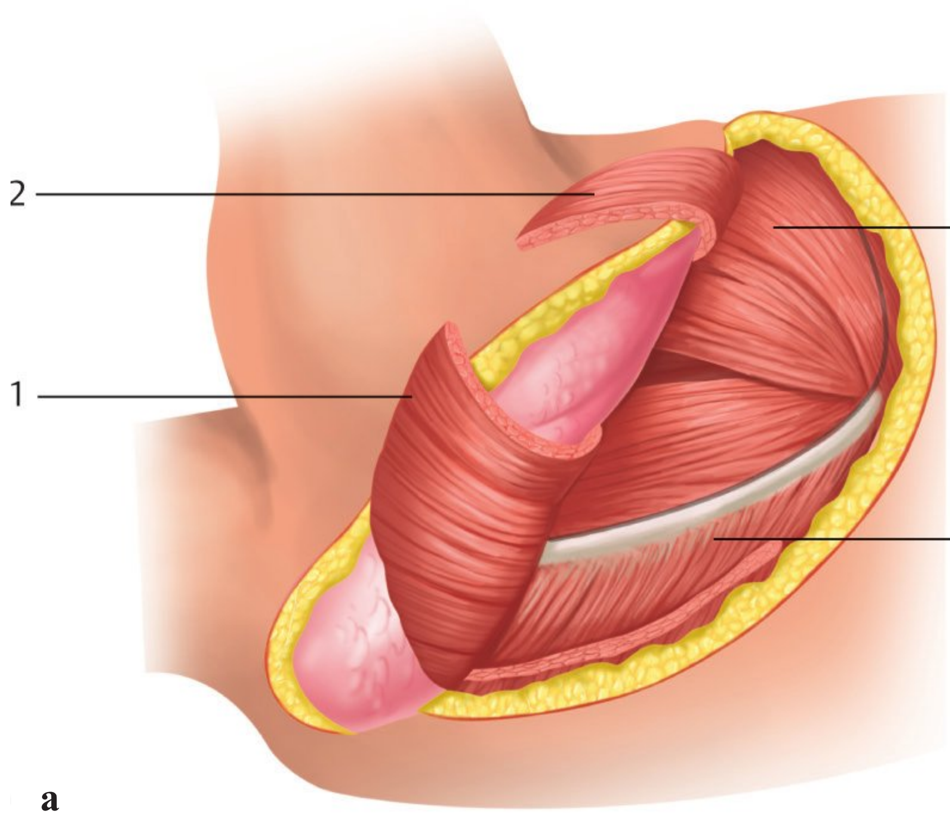


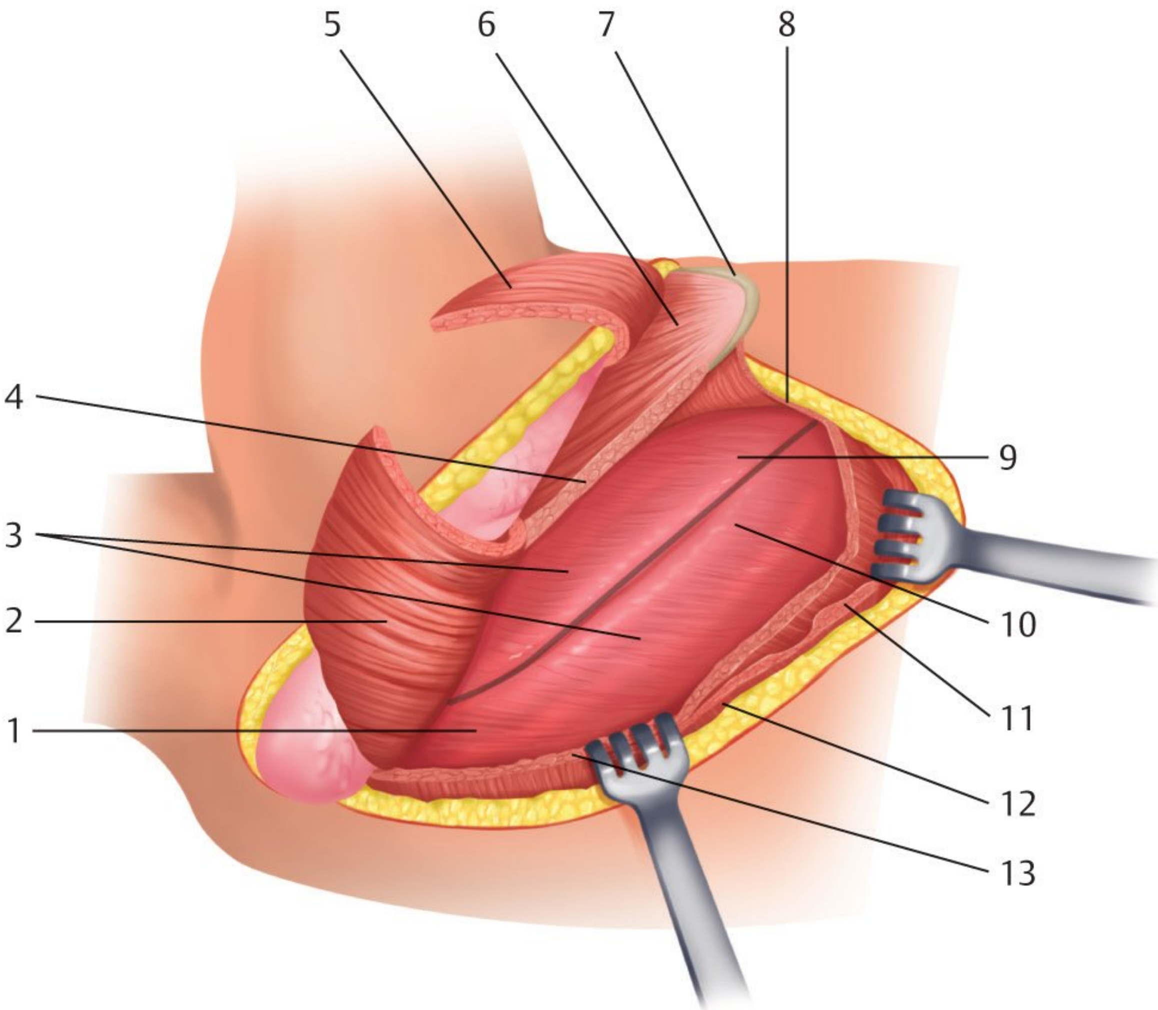
b

Fig. 1.24 **(a)** High transthoracic approach to the upper cervicothoracic spine. **(b)** The trapezius muscle is divided close to the spinous processes and parallel to the skin.



a

1. Trapezius muscle
2. Latissimus dorsi muscle
3. Serratus anterior muscle
4. Rhomboideus major muscle



1. Iliocostal muscle
2. Trapezius muscle
3. External intercostal muscle
4. Cut edge of rhomboideus major muscle
5. Latissimus dorsi muscle
6. Teres major muscle
7. Inferior angle of scapula
8. Serratus anterior muscle
9. Third rib
10. Fourth rib
11. Latissimus dorsi muscle
12. Trapezius muscle
13. Rhomboideus major muscle

b

Fig. 1.25 **(a)** The rhomboid major is divided near its insertion, and the serratus anterior muscle is divided as caudally as possible. **(b)** The scapula can then be retracted superolaterally, and the periosteum can be incised.

d. The sternum-splitting approach to C4–T4.
   (1) The skin incision is made anterior to the left sternocleidomastoid muscle and extends along the midsternal area down to the xiphoid process.
   (2) After division of the platysma muscle and superficial cervical fascia, blunt dissection is done between the laterally situated neurovascular bundle and medial visceral structures.
   (3) The retrosternal adipose and thymus tissues are retracted from the manubrium.

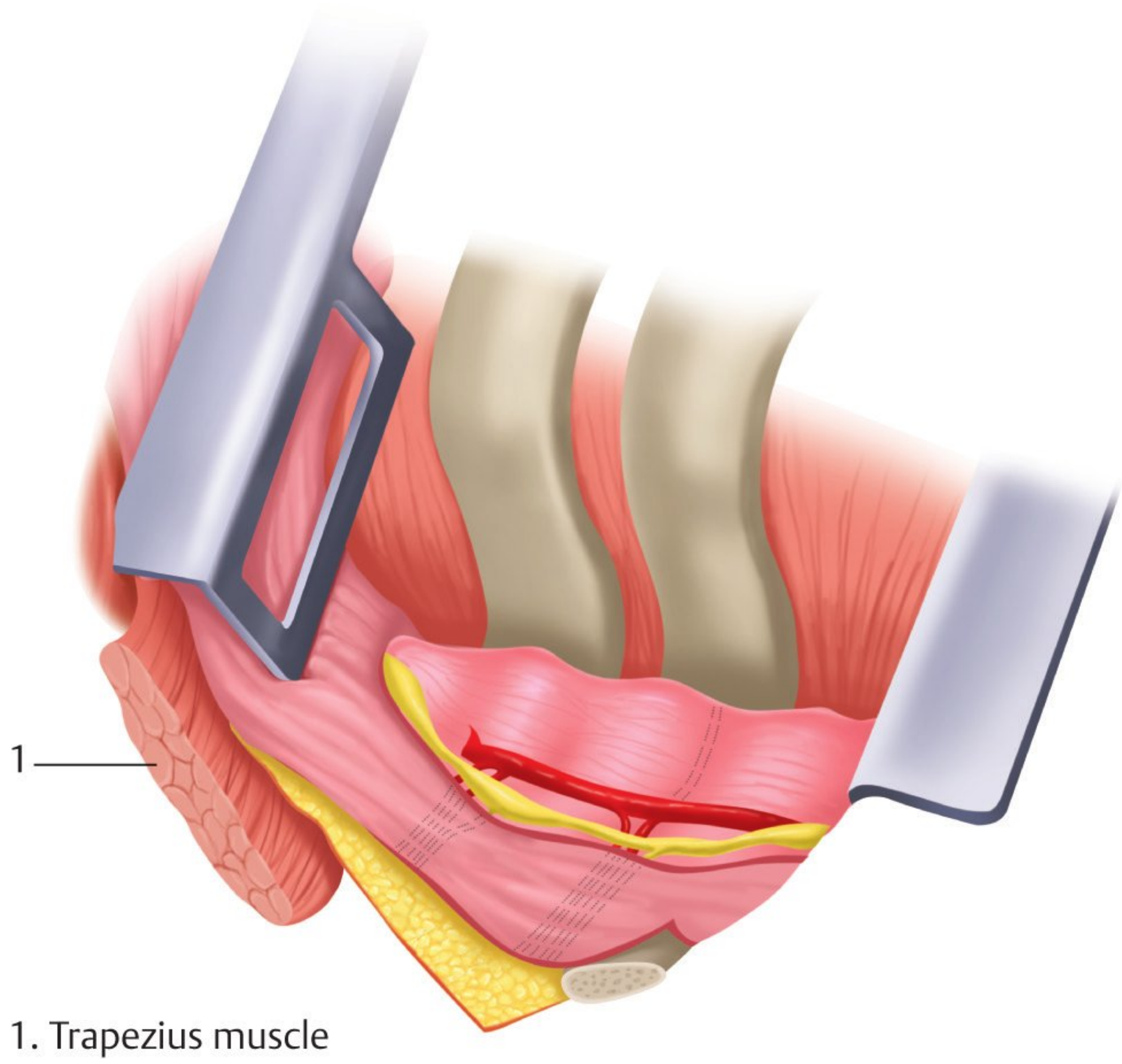


Fig. 1.26 Retractors are positioned and the upper thoracic spine is exposed.

(4) A median sternotomy should be performed carefully to prevent injury to the pleura. The sternohyoid, sternothyroid, and omohyoid muscles are identified and transected as necessary.

(5) The inferior thyroid artery is ligated and transected.

(6) Blunt dissection is performed from the cranial toward the caudal portion until the left brachiocephalic vein is exposed.

(7) Retraction of the carotid artery laterally, brachiocephalic vein inferiorly, and trachea medially exposes the vertebrae.

II. Thoracolumbar spine.

A. Posterior approaches: the patient is usually positioned on the four-poster or Relton-Hall frame (Surgmed, Dorval, Quebec, Canada) for the thoracolumbar spine and kneeling position for the lumbar spine.

1. Thoracic spine.

a. Posterior: midline exposure of the posterior elements (spinous process, lamina, facets, pedicle, and transverse processes).

(1) Transpedicular approach: the thoracic pedicle is located by crossing a horizontal line at the midportion of the transverse process and a vertical line at the junction between the lamina and transverse process.

(2) Posterolateral: costotransversectomy approach:

(a) A C-shaped curved incision is made along the paraspinous muscles, spanning about four to five ribs.

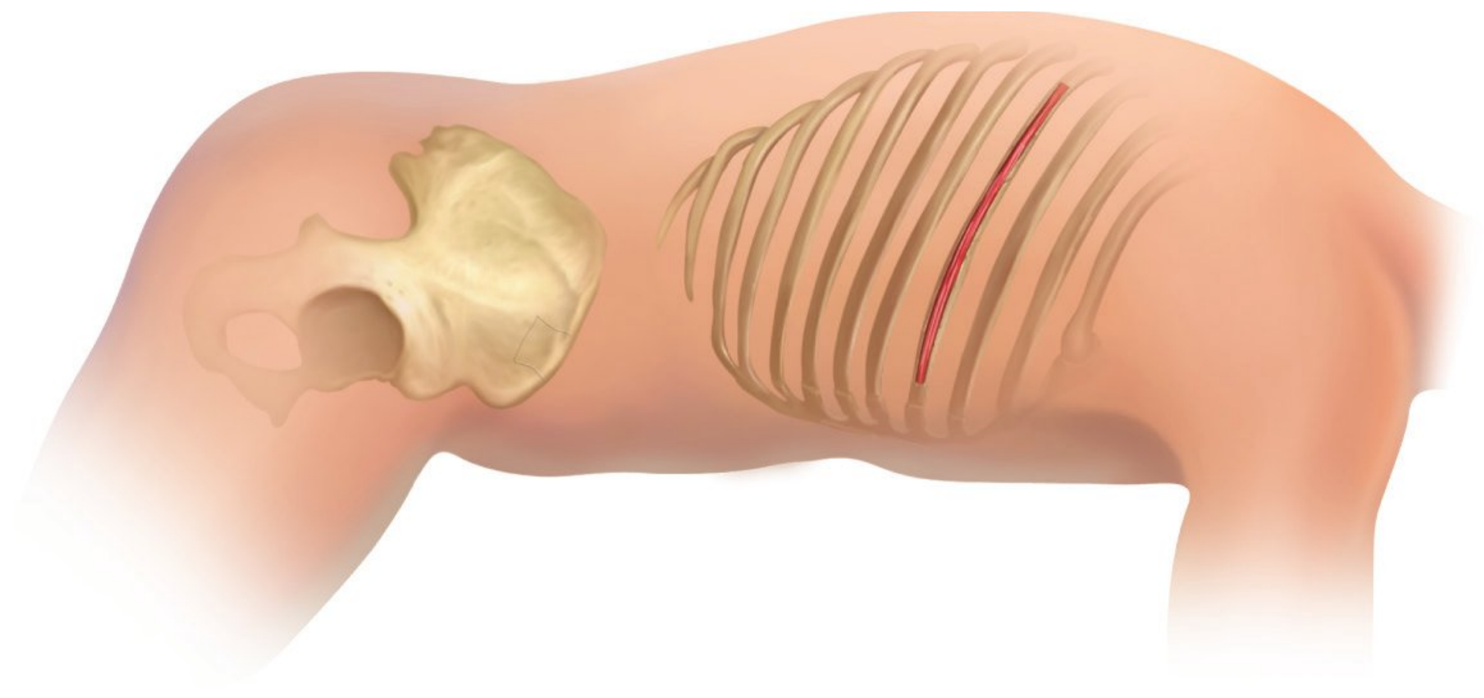


Fig. 1.27 The incision employed for a transthoracic approach to the spine.

(b) The middle part of the incision should be ~ 2.5 in from the midline.

(c) By undermining the skin and subcutaneous tissue, exposure of the paraspinous muscles and posterior elements of the spine is completed.

(d) The trapezius and latissimus dorsi muscles are divided either longitudinally or transversely.

(e) The rib and transverse process are resected at one to four levels, depending on the extent of the lesion. The rib is exposed subperiosteally and excised ~ 3.5 inches lateral to the vertebra and disarticulated at the costovertebral junction.

(f) Careful retraction of the pleura will lead to the vertebrae.

2. Lumbar spine.
   a. Laminectomy or laminotomy:
      (1) Expose the spinous process, lamina, and ligamentum flavum.
      (2) Excise the ligamentum flavum to enter the epidural space.
      (3) Remove part of the superior facet to decompress the lateral recess.
      (4) Retract the nerve root medially to remove the offending disk material.
   b. Transpedicular approach **(Fig. 1.26)**: the pedicle is located by crossing a horizontal line at the midportion of the transverse processes and a vertical line at the lateral edge of the superior facet.

B. Anterior approaches:
   1. Thoracic spine:
      a. Transthoracic approach by removing a rib and dividing the pleura **(Fig. 1.27)**.
         (1) The skin incision is made along the rib intended for removal from the anterior margin of the latissimus muscle anteriorly to the costochondral junction.

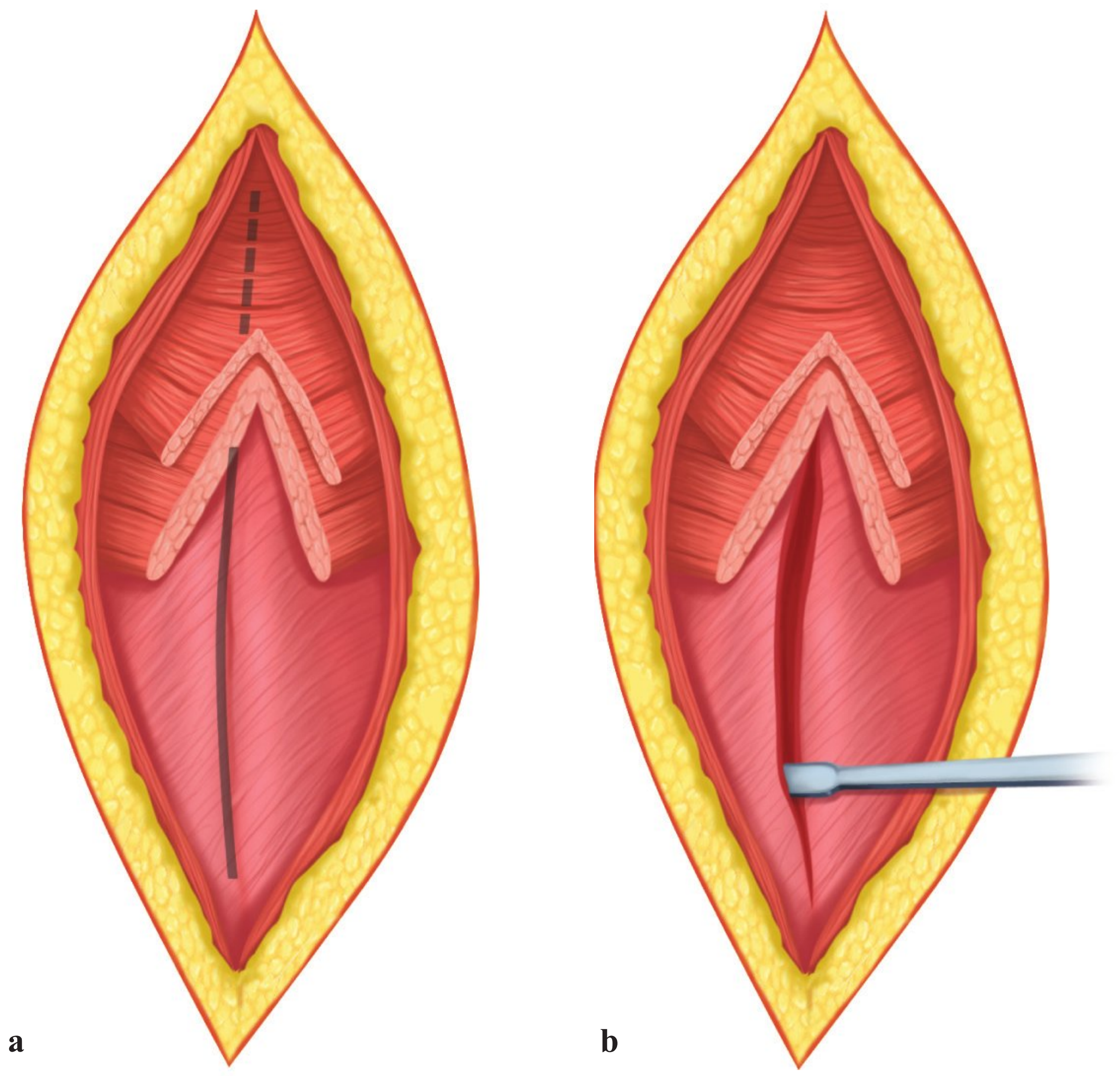


Fig. 1.28 **(a)** The anterior aspect of the latissimus is divided, exposing the underlying rib. **(b)** The underlying rib is dissected free of the periosteum.

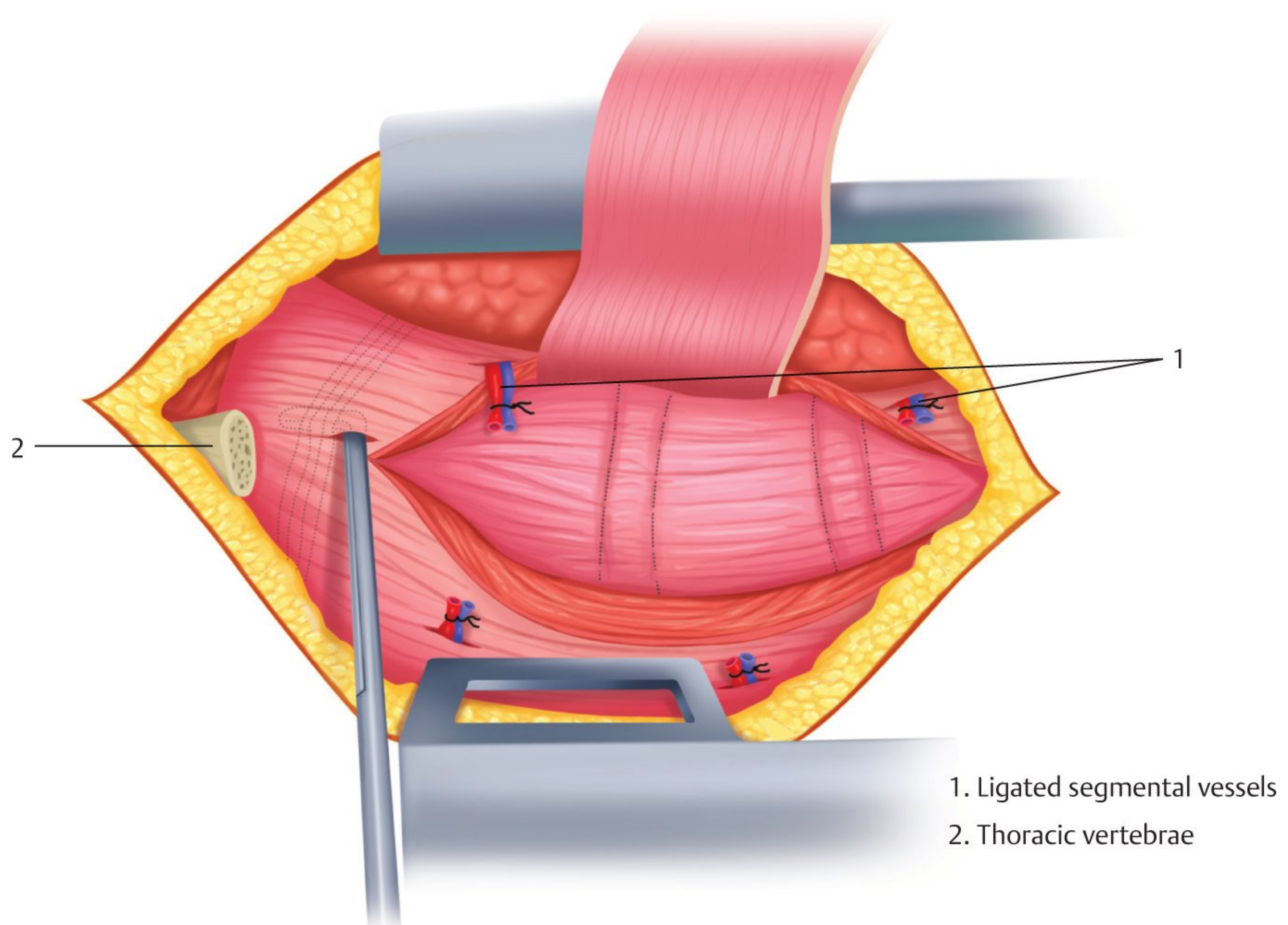


Fig. 1.29 The overlying rib is resected near its articulation with the costovertebral junction. The parietal pleura is incised, and the overlying prevertebral fascia is identified. Shown are the ligated segmental vessels overlying the thoracic vertebrae.

(2) The anterior aspect of the latissimus muscle can be undermined or minimally incised, and the posterior border of the serratus anterior muscle is mobilized or transected.
(3) The lateral margin of the trapezius muscle is mobilized and transected if necessary.
(4) Rib resection is then performed by first incising the overlying periosteum in the midportion of the rib using electrocautery. A rib stripper is then used to dissect off the intercostal musculature.
(5) The rib is divided at the costochondral junction anteriorly, elevated and resected as far posteriorly as the exposure will allow.
(6) The chest is then sharply entered in the center of the rib bed, and the lung retracted anteriorly and inferiorly.
(7) The pleura overlying the vertebral bodies is then incised and the segmental vessels ligated as needed in the middle of the vertebral bodies.

b. Thoracoabdominal approach by removing the tenth rib and dividing the diaphragm and entering through the retroperitoneal space.
(1) A skin incision is made over the tenth rib from the lateral border of the paraspinous musculature to the costal cartilage. The incision is curved anteriorly to the edge of the rectus sheath.
(2) The dissection is extended down to the muscle layers to remove the 10th rib.
(3) The costal cartilage is split after removal of the 10th rib. The pleura is incised and the lung is retracted, and the retroperitoneal space is identified by the light areolar tissue.
(4) Blunt dissection is performed to mobilize the peritoneum from the undersurface of the diaphragm and abdominal wall.
(5) After the peritoneum is retracted, the external oblique, internal oblique, and transverse abdominis muscles of the abdomen are divided one layer at a time.
(6) The diaphragm is incised circumferentially 1 in from its peripheral attachment to the chest wall. Marker stitches or clips are placed for resuturing the diaphragm later.
(7) For the exposure of the T12–L1 region, the crus of the diaphragm is cut and mobilized.
(8) The segmental vessels are tied and ligated as necessary to mobilize the aorta.

2. Lumbar spine.
a. Anterolateral retroperitoneal approach: lateral decubitus position.
(1) Dissection is through the external oblique, internal oblique, and transverse abdominis muscles (skin incision depends on the level of exposure).
(2) The retroperitoneal space is entered laterally by identifying the retroperitoneal fat, taking care to avoid penetration of the peritoneum just lateral to the rectus sheath.
(3) Blunt dissection anterior to the psoas muscle should lead to the spine.

(4) One should identify the genitofemoral nerve on the anterior surface of the psoas muscle and the sympathetic chains medial to the muscle **(Fig. 1.30)**:
   (a) The L4–L5 level is at most risk for neural and vascular injury.
   (b) Prevention: direct visualization while dissecting the psoas muscle with concurrent neuromonitoring.
(5) The ureter is under the peritoneum anteriorly.
(6) Vessels (aorta or vena cava) are mobilized, and segmental vessels are identified in the middle portion of the vertebral bodies and ligated as necessary.
(7) Dissection of the psoas muscle should be accompanied with triggered electromyography.

b. Anterior muscle-splitting approach.
   (1) A vertical paramedian incision is made from the umbilicus to the pubis at the edge of the rectus.
   (2) The fascia of the rectus abdominis is divided, and the muscle is retracted medially.
   (3) The posterior rectus sheath is carefully divided along the peritoneal attachment.
   (4) Blunt finger dissection of the peritoneum leads to the lower lumbar spine (L3–S1).

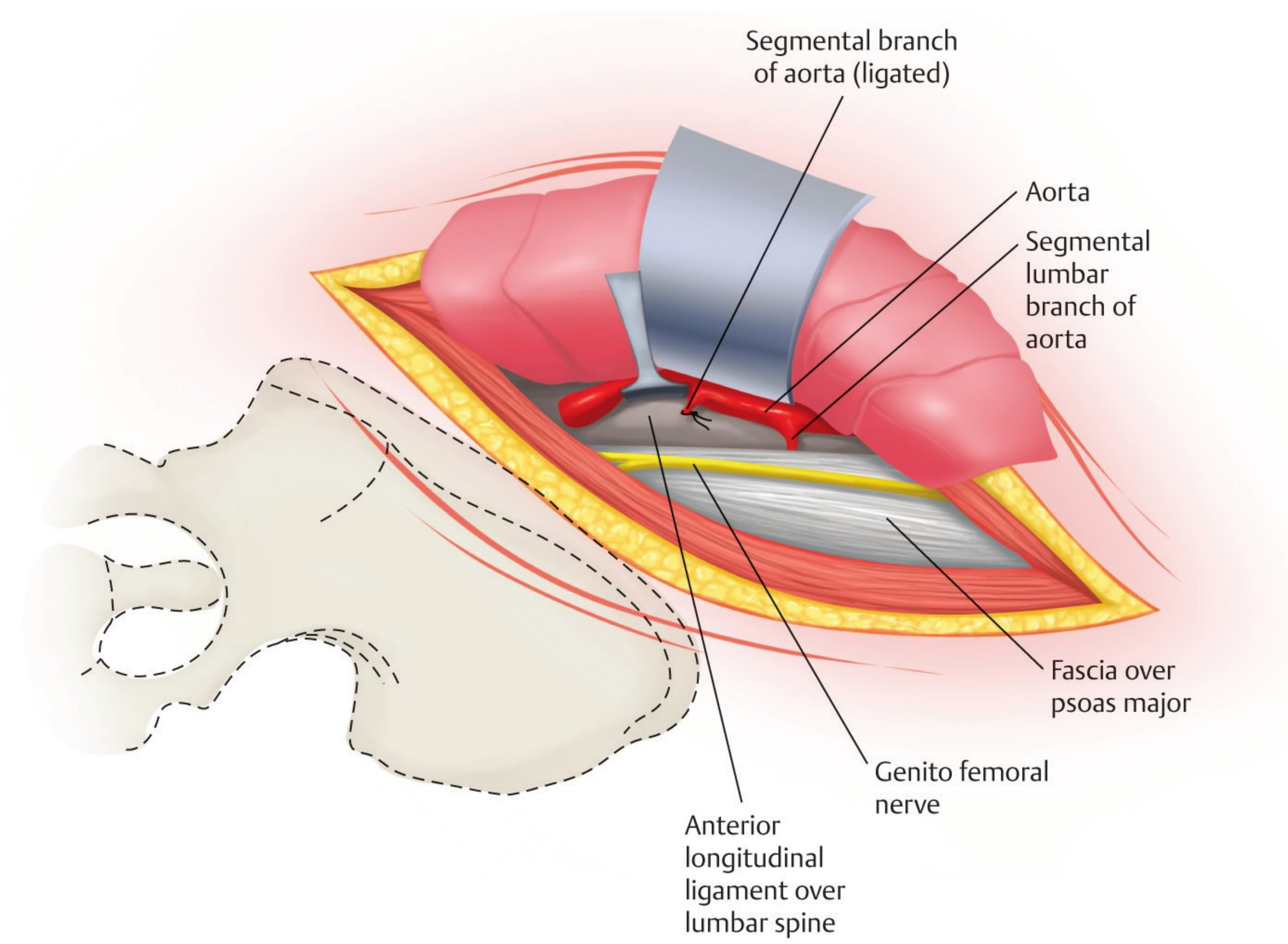


Fig. 1.30 The anterolateral peritoneal approach demonstrating the genitofemoral nerve on the anterior surface of the psoas muscle and the sympathetic chains.

c. Transperitoneal approach to lumbosacral junction.
   (1) A vertical or transverse incision is made above to the pubis **(Fig. 1.31** and **Fig. 1.32)**.
   (2) The peritoneum is entered, and the bowel structures are retracted **(Fig. 1.33a)**.
   (3) The posterior peritoneum is lifted and divided.
   (4) The iliac vessels are mobilized to expose L4–S1 **(Fig. 1.33b)**.

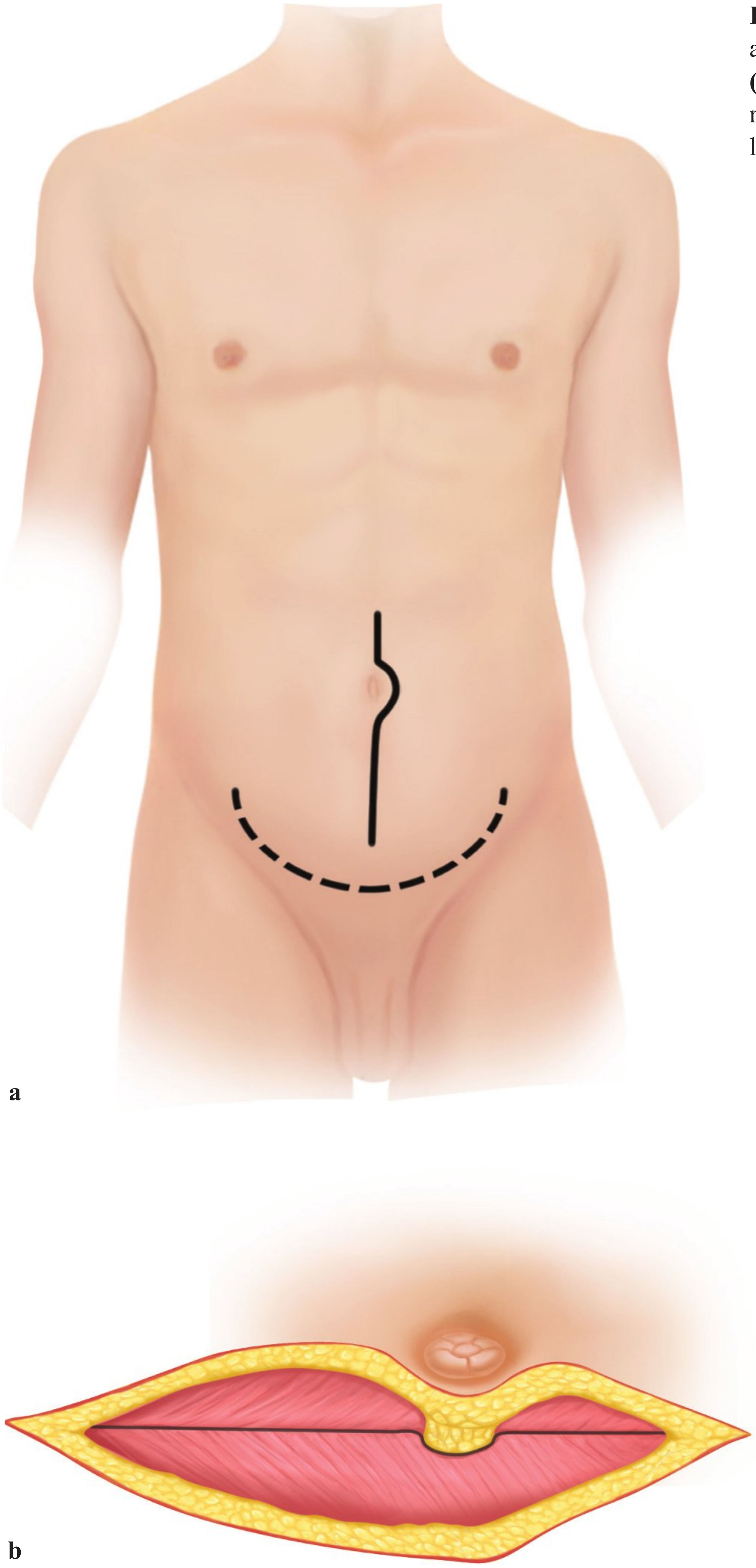


Fig. 1.31 **(a)** The transperitoneal approach to the lumbar spine. **(b)** The vertical incision splits the rectus abdominis in the midline linea alba.

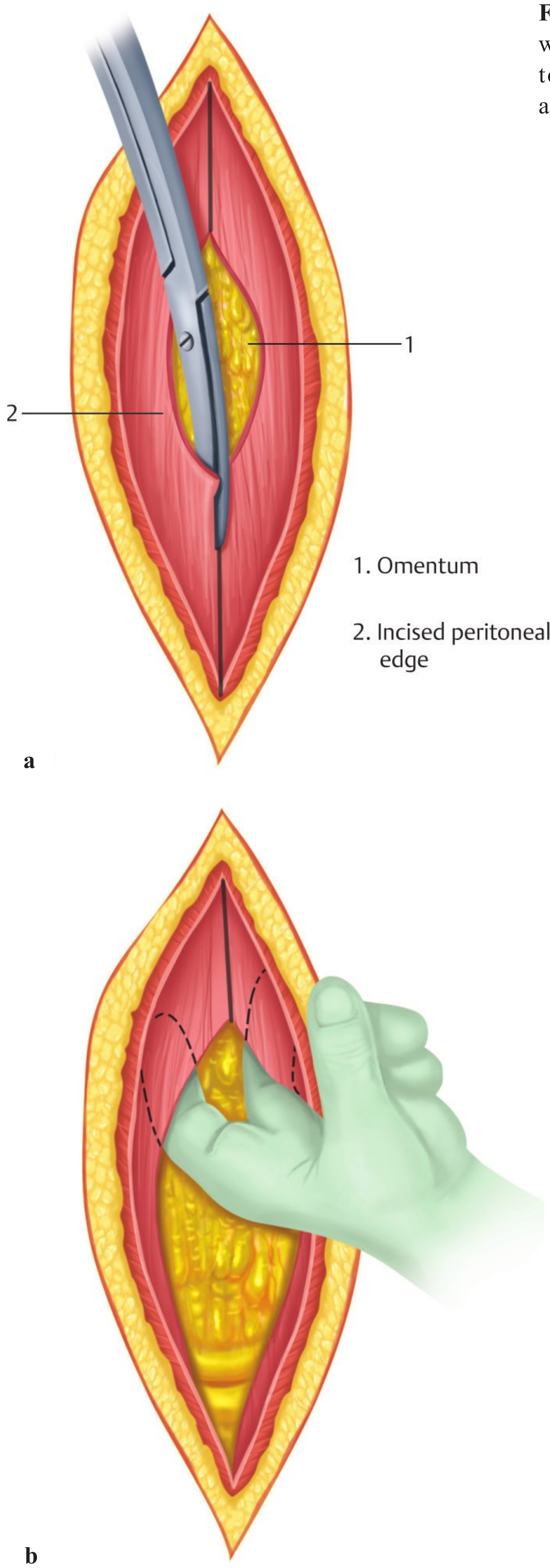
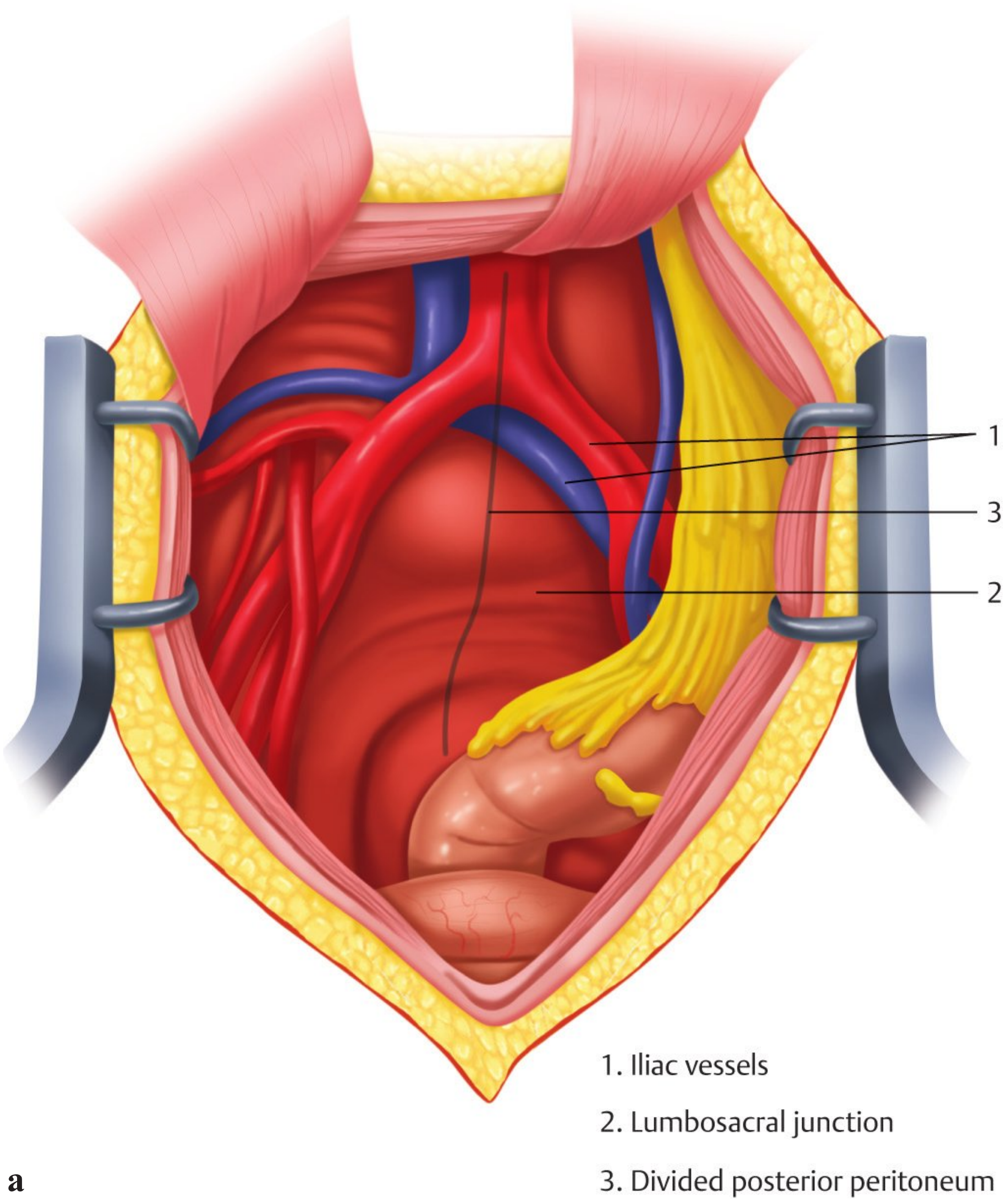


Fig. 1.32 **(a)** The overlying peritoneum is incised, with care to avoid damaging the underlying peritoneum. **(b)** The abdominal viscera are retracted, and the underlying vertebral bodies are exposed.



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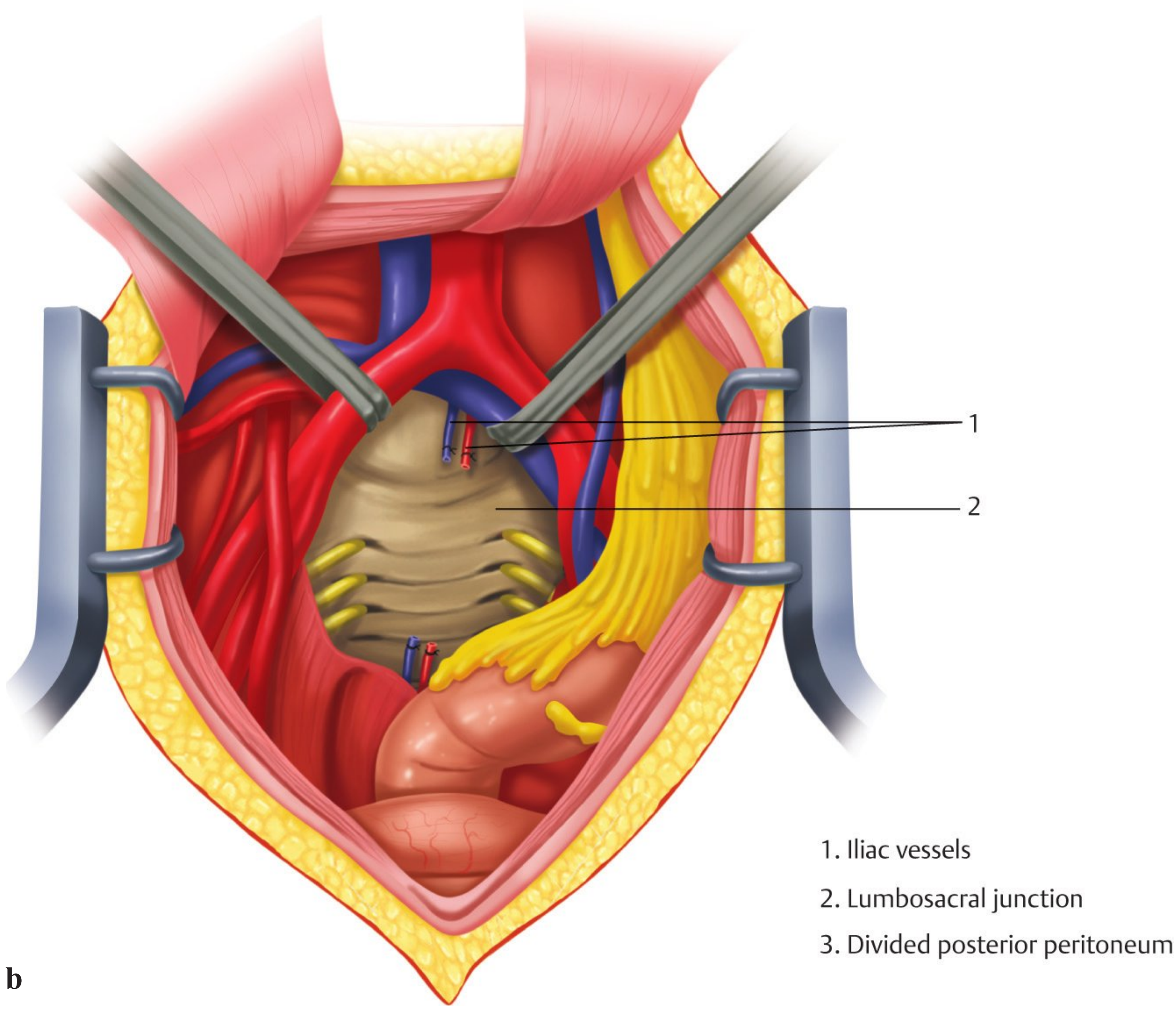

b

Fig. 1.33 **(a)** The omentum and peritoneal contents are reflected, exposing the bifurcation of the aorta. **(b)** Retractors are placed along the common iliac arteries bilaterally, exposing the L5–S1 interspace.

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2 History and Physical Examination

2.1 History

I. Degenerative disorders of the spine.
  A. History is the most important component of the patient evaluation.
    1. Establishes the initial differential diagnoses.
    2. Guides physical examination and selective diagnostic tests.
  B. Spinal pain can be described as mechanical versus nonmechanical, or axial versus radicular.
    1. Mechanical versus nonmechanical.
      a. Mechanical pain tends to be associated with activity.
        (1) Relieved by rest.
        (2) Progressively worse over the course of the day.
      b. Nonmechanical pain is typically due to tumors or infections.
        (1) Independent of activity.
        (2) Worse at night.
        (3) Not relieved by rest or immobilization.
    2. Axial versus radicular.
      a. Axial pain is usually diffuse.
        (1) Referred pain to the scapula or shoulder in cervical spine disorders.
        (2) Referred pain to the buttock or posterior thigh in lumbar spine disorders.
      b. Radicular pain is typically associated with paresthesia, numbness, or weakness in a dermatomal distribution **(Fig. 2.1)**.
        (1) Associated with tension signs **(Table 2.1).**
  C. Myelopathy **(Fig. 2.2):**
    1. Presents with poorly characterized pain.
      a. Vague sensory and motor symptoms over a long period of time are also common.
    2. Associated with neck, arm, or leg pain in a nondermatomal pattern or with pain in a cervical dermatome.
    3. Characterized by a slow and broad-based gait.
    4. Problems with upper-extremity fine motor functions.
      a. Difficulty with fastening buttons is noted early.
      b. Lower-extremity dysfunction and spasticity.
      c. Bowel and bladder dysfunction are noted later.
    5. Associated with pathological long tract signs **(Table 2.2).**

II. Traumatic disorders.
  A. The airway, breathing, and circulation (ABC) should be checked first in any trauma patient.
  B. The mechanism of injury should be sought.
  C. Pain and neurological symptoms should be documented.

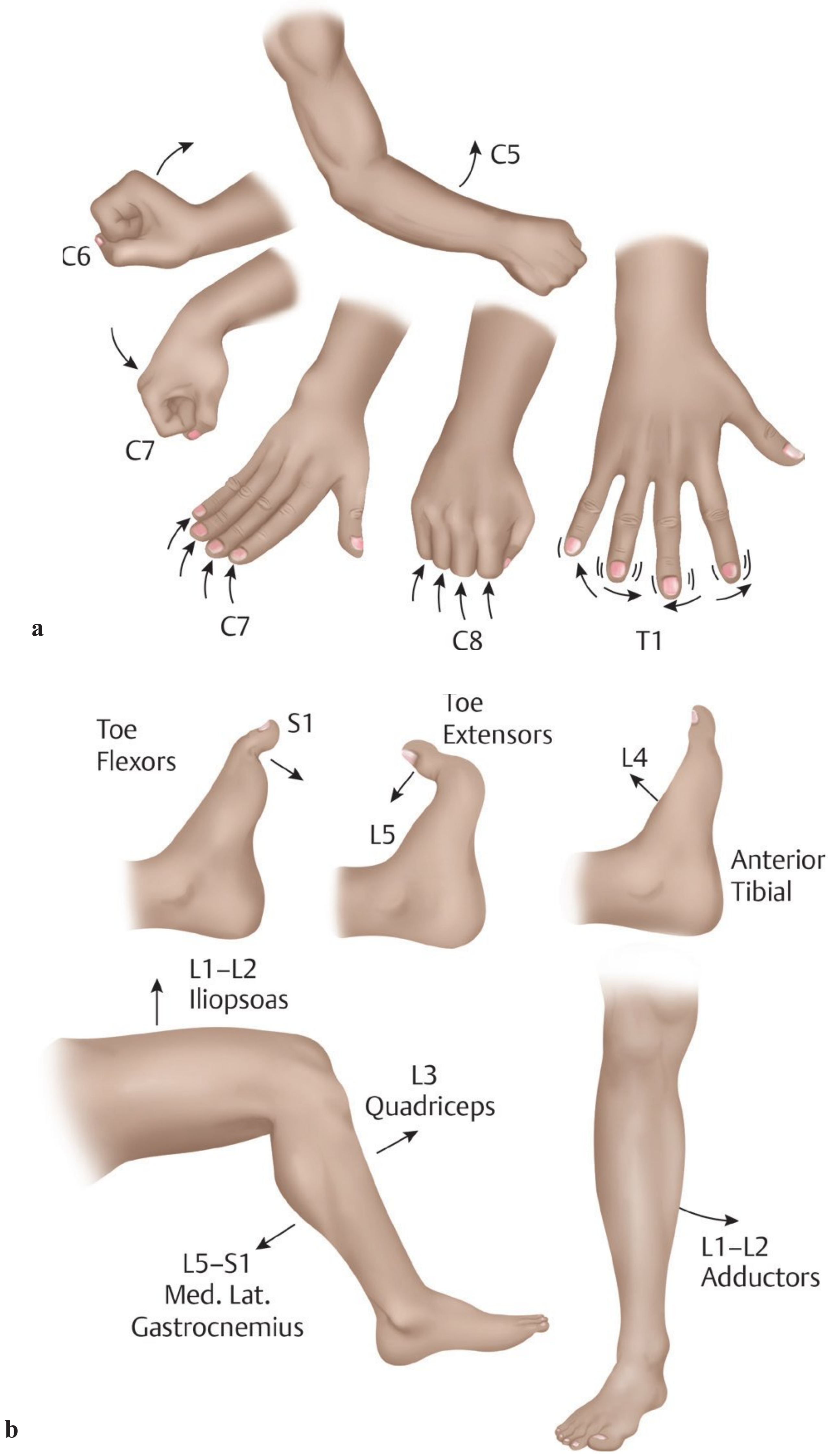

Fig. 2.1 **(a,b)** Motor examination for cervical and lumbar nerve roots.

III. Spinal deformity.
- A. Deformity and pain are the two most common presenting complaints.
  - 1. Pain is a more ominous sign in the child.
    - a. There are several possible etiologies:
      - (1) Spinal cord or bony tumor.
      - (2) Scheuermann's disease.
      - (3) Spondylolisthesis.

Table 2.1 Nerve tension signs

Cervical tension signs	**Findings**
Spurling's	Neck extension and rotation toward the painful side causes radicular limb pain.
Compression	Axial loading on the head reproduces pain.
Distraction test	Skull traction relieves pain.
Shoulder abduction	Elevation of painful limb relieves pain.
Lumbar tension signs	**Findings**
Lasegue's (straight leg raise [SLR])	Elevation of painful limb causes radicular limb pain, not back pain. Pain should be reproduced with <60° of hip flexion.
Bowstring	After reproducing the patient's pain and obtaining a positive Lasegue's sign, the knee is flexed. This is positive if the patient's pain resolves with flexion of the knee.
Fajersztajn's (contralateral SLR)	Elevation of the nonpainful limb causes back and limb pain on the opposite side (usually means sequestered or large extruded herniated disk).
Femoral stretch (reverse SLR)	Hip extension in either the lateral decubitus or prone position stretches the femoral nerve and reproduces pain in the L3 or L4 distribution.

2. In adults, pain associated with deformities tends to be present at the convexity.
   a. Due to muscle fatigue early.
   b. Localizes to the concavity when degenerative changes have occurred later.

B. Medical history, family history, onset of menarche, time of curve detection, and progression should be obtained in the adolescent scoliotic patient.

2.2 Physical Examination

I. Inspection.
   A. Look for obvious deformities in both the coronal and the sagittal plane.
      1. Coronal plane **(Fig. 2.3)**.
         a. Scoliosis evaluated by a plumb line dropped from the seventh cervical vertebra.
         b. Pelvic obliquity.
         c. Shoulder imbalance.
         d. Scapular protuberance.
         e. Rib prominence.

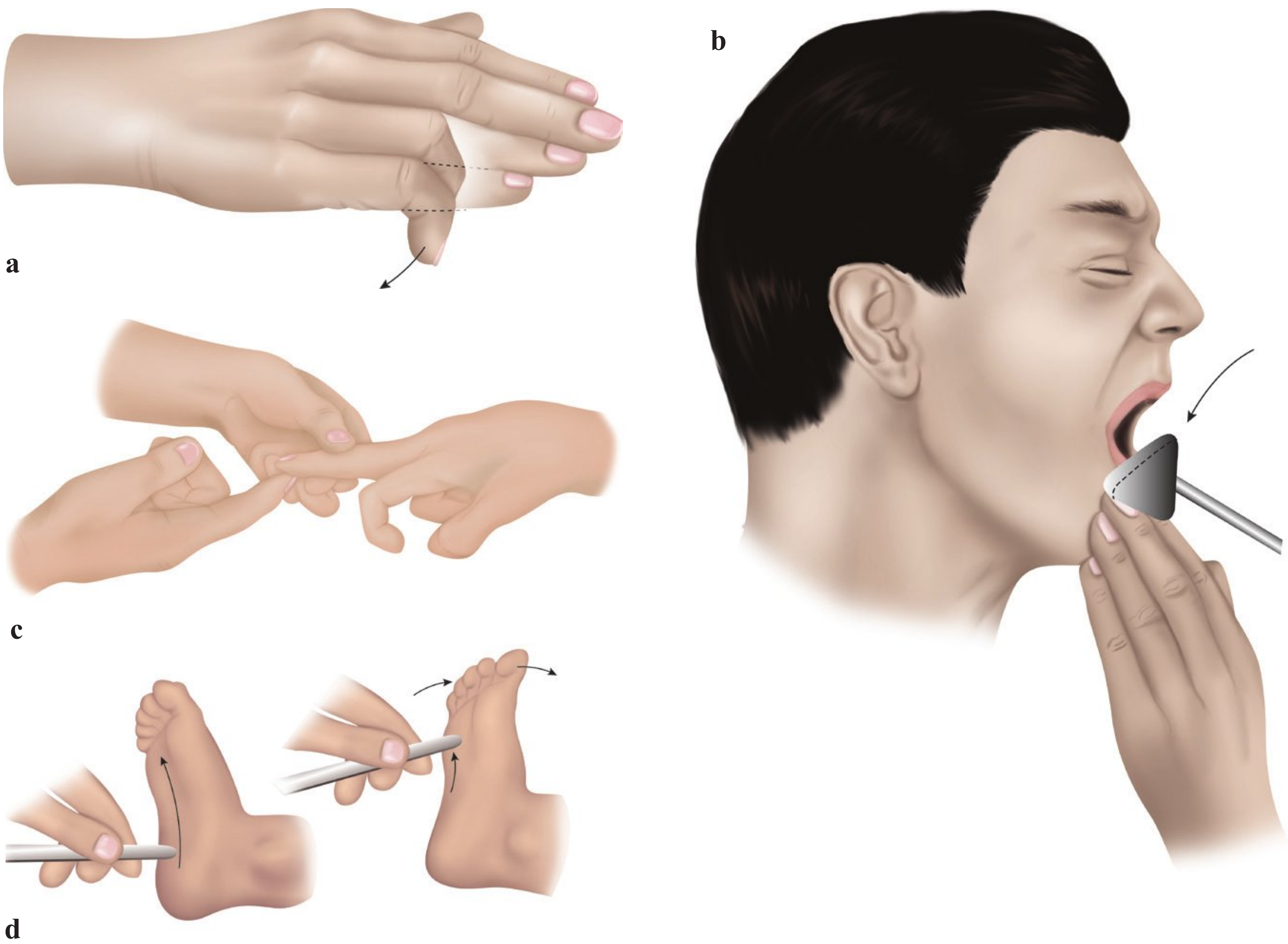

Fig. 2.2 Myelopathic motor examination findings. **(a)** Finger escape sign. **(b)** Jaw-jerk reflex. Myelopathic motor examination findings. **(c)** Hoffman's sign. **(d)** Babinski's sign.

2. Sagittal balance and regional deformities.
   a. Normal cervical lordosis: 20 to 40°.
   b. Normal thoracic kyphosis: 20 to 45°.
   c. Normal lumbar lordosis: 40 to 60°.

B. Look for skin or subcutaneous lesions.
   1. Café au lait spots in neurofibromatosis patients.
   2. Midline tufts of hair, dimples, or rosy spots may indicate occult spinal dysraphism.

C. Muscle atrophy should be observed in neurologically impaired patients.

II. Palpation.

A. Bony palpation.
   1. Spinous processes.
   2. Posterior superior iliac spines: "dimples".
   3. Scapula and ribs.
   4. Iliac crests.
   5. Sacrum and coccyx.
   6. Trochanter and ischial tuberosity.

B. Soft tissue palpation for spasm or trigger point tenderness.
   1. Trapezius muscle.
   2. Rhomboid/levator muscles.

Table 2.2 Myelopathic signs

Long tract sign/reflex	Findings/provocative maneuver
Lhermitte's grip release	Neck flexion causes electric shock sensation or paresthesias radiating into the upper and lower extremities. Patient has trouble making a fist and fully extending fingers repeatedly—normal 20 times in 10 seconds.
Finger escape	Ask the patient to keep the fingers in full extension and the ulnar digits tend to gradually flex and abduct.
Jaw jerk	Hyperreflexia on tapping the jaw suggests an upper motor neuron lesion at the level of the brain stem; involves the masseter and temporalis muscles and the fifth cranial nerve.
Shimizu (scapulohumeral)	Tapping the tip of the spine of the scapula and the acromion elicits elevation of the humerus. Reflex suggests spinal cord compression at the upper cervical region.
Inverted radial	Tapping of the brachioradialis tendon causes spastic finger flexor contraction instead of normal extension of the wrist. Positive reflex suggests spinal cord compression at the C6 region.
Hoffman's	Holding the middle finger extended and suddenly extending the distal interphalangeal joint (DIP) will produce finger and thumb flexion.
Babinski's	Gentle stimulus applied to the lateral aspect of the sole of the foot starting over the heel extending toward the fifth digit. A positive Babinski's sign refers to the initial dorsiflexion of the great toe upward and the spreading of the other toes.
Clonus	Rhythmic, nonvoluntary movements of the muscle with firm passive continuous stretch

3. Paravertebral muscles.
4. Gluteus muscles.
5. Piriformis muscle.
6. Sciatic nerve.

III. Range of motion.

A. Cervical spine.

1. Flexion: 45° (the chin touches the chest).
2. Extension: 75°.
3. Lateral bending: 40°.
4. Rotation: 75°.

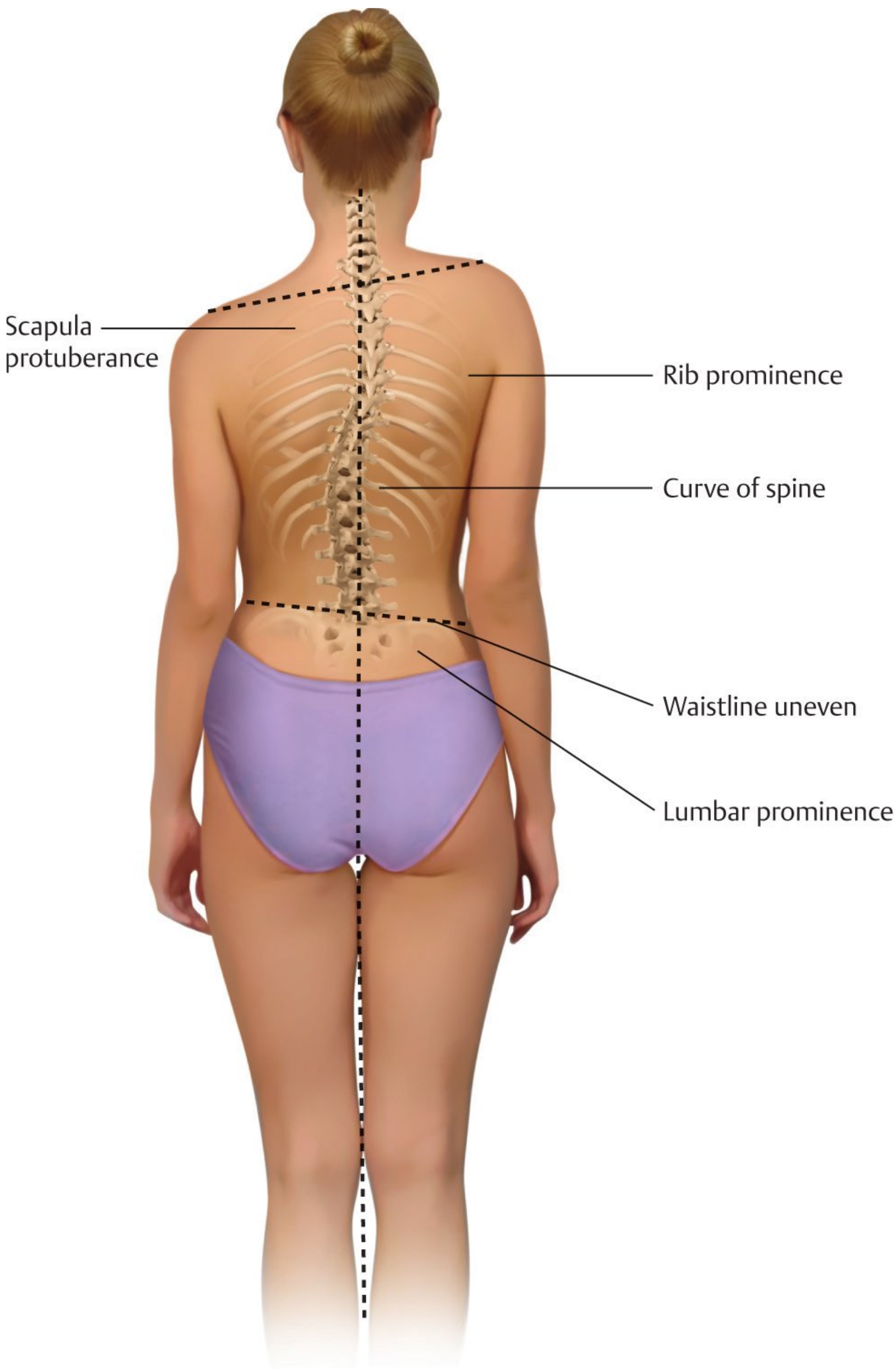

Fig. 2.3 Pelvic obliquity, shoulder imbalance, scapular protuberance, and rib prominence in the coronal plane in a scoliosis patient.

B. Thoracolumbar spine.
   1. Flexion: 80° (measure the distance from the tip of hands to the floor).
   2. Extension: 40°.
   3. Lateral bending: 40°.
   4. Rotation: 45°.

IV. Neurological examination of individual roots.
   A. Sensory tests.
      1. Four distinct sensations with defined anatomical pathways in the spinal cord:
         a. Pain perception may be tested with defined anatomical pathways of the spinal cord.

   b. Light touch may be tested with a cotton swab.
   c. Temperature may be tested with two test tubes containing either a hot or a cold solution.
   d. Proprioception begins distally at the distal phalanx or great toe and proceeds proximally to each larger joint.
2. The aim of sensory testing is to identify whether there is a dermatomal pattern of sensory dysfunction, which would suggest spinal root pathology, or a possible glove/stocking distribution that would suggest a neuropathy **(Table 2.3) (Fig. 2.4)**.

B. Motor tests.
1. Muscle tone—resistance to passive range of motion.
   a. Hypertonia may indicate an upper motor nerve lesion.
   b. Hypotonia may indicate a lower motor nerve lesion.
2. Muscle strength.
   a. Grade 5: normal.
   b. Grade 4: weak against resistance.
   c. Grade 3: motion against gravity.
   d. Grade 2: motion with gravity eliminated.

Table 2.3 Anatomy of dermatomal distribution

Nerve root	Dermatomal distribution
C5	Upper outer arm
C6	Thumb
C7	Long finger
C8	Little finger
T1	Medial forearm
T4	Nipple
T10	Umbilicus
L1	Groin
L2	Anterior thigh
L3	Knee
L4	Medial malleolus
L5	Great toe
S1	Small toe
S2	Posterior thigh
S3–S5	Anal

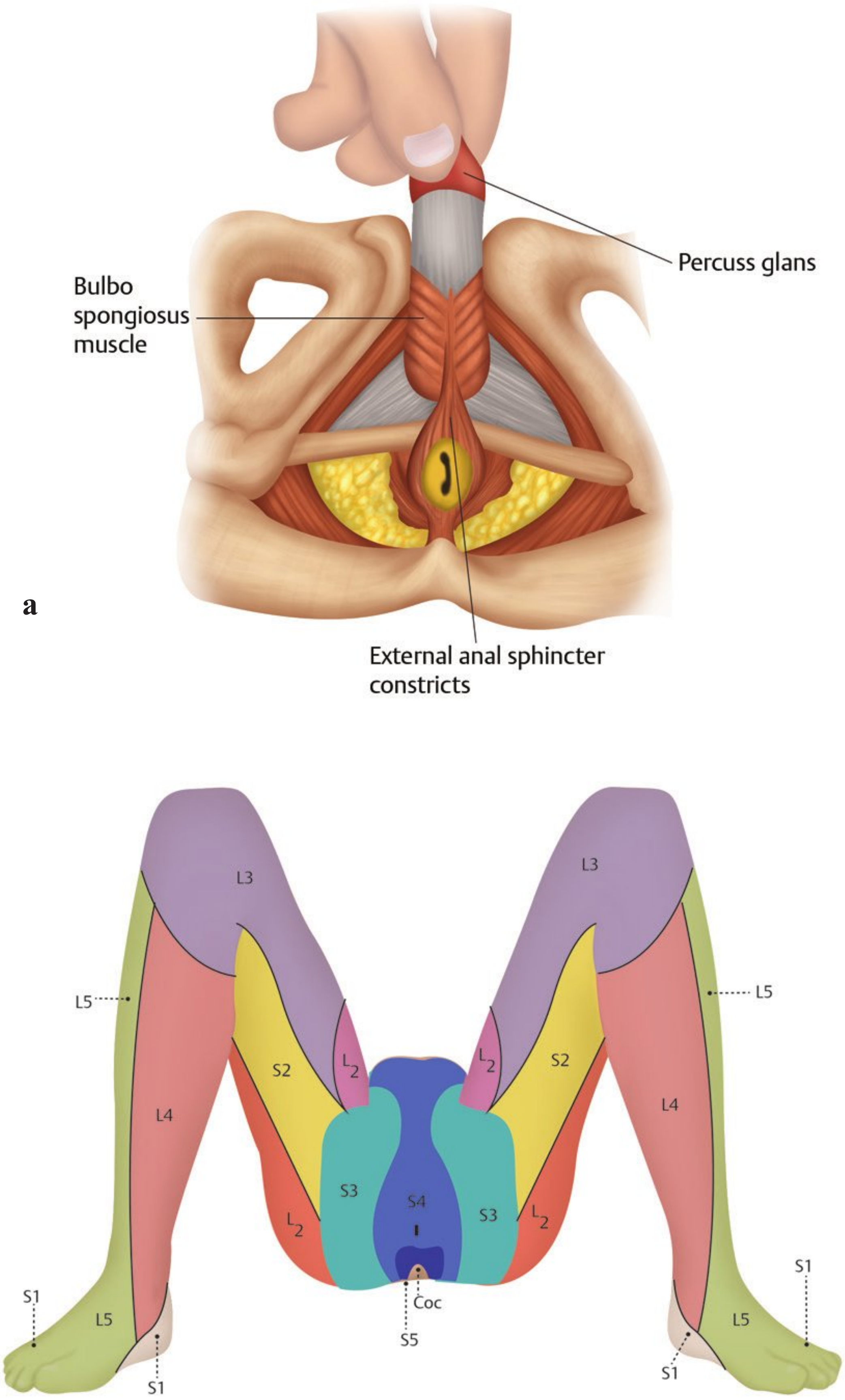

Fig. 2.4 **(a,b)** Normal distribution of dermatomal levels.

e. Grade 1: evidence of contractility.

f. Grade 0: no evidence of contractility.

3. Motor root testing/reflexes **(Table 2.4).**

V. Special provocative tests **(Fig. 2.5).**

A. Adson's test.

1. Test to evaluate thoracic outlet syndrome.
2. Abduct, extend, and externally rotate the arm while feeling the radial pulse. Also rotate the head toward the testing arm.
   a. If the pulse disappears with reproduction of symptoms, the test is positive.

B. Sacroiliac tests.

1. Patrick's test: flexion, abduction, and external rotation of the hip cause pain referred from the sacroiliac joint.
2. Gaenslen's test: dropping the leg on the table (extension of the hip) causes pain in the ipsilateral sacroiliac joint.

Table 2.4 Motor strength testing

Root	Muscles	Reflex
C5	Deltoid, biceps	Biceps
C6	Biceps, wrist extensors	Brachioradialis
C7	Triceps, wrist flexion	Triceps
C8	Finger flexors	
T1, T2	Hand intrinsics	
T2–T12	Intercostals, rectus abdominis	Beevor's sign (abdominal)—asymmetric contraction of the umbilicus with stimulation of the abdomen
L1–L3	Iliopsoas	
L4	Tibialis anterior	Patellar tendon
L5	Extensor hallucis longus	Posterior tibial tendon
S1	Peronealis, gastrocnemius	Achilles

C. Bulbocavernous reflex **(Fig. 2.4)**:
   1. Monitoring the anal sphincter contraction in response to squeezing the glans penis or clitoris or pulling an indwelling Foley catheter.

D. Schober's test.
   1. Normal lumbar excursion is usually > 5 cm. Mark 10 cm from the posterior superior iliac spine level when the patient is standing erect, and measure the distance on forward flexion. If it becomes < 15 cm, one should suspect ankylosing spondylitis.

E. Waddell's signs.
   1. Nonorganic physical exam findings.
   2. If three or more signs are found, it is suggestive that the patient's pain complaints may not be anatomical.
      a. Nonanatomical or superficial tenderness that is not proportional to exam findings.
      b. Simulated rotation or compression tests:
         (1) Instruct the patient to stand with the feet together and rotate the patient's pelvis or press on the top of the head. These maneuvers should not cause pain.
      c. Extending the leg in the sitting position is negative but straight leg raising in the supine position is markedly positive.
      d. Weakness and sensory findings that do not correspond to accepted dermatomal distribution.
      e. Verbal or physical overreaction to a particular maneuver.

a
Adson's test

b
Modified Adson's test

Fig. 2.5 **(a)** Adson's and the **(b)** modified Adson's test to evaluate for thoracic outlet syndrome.

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3 Radiographic Anatomy

3.1 General Considerations

I. The ability to properly diagnose and treat spinal pathology requires a thorough understanding of the normal spinal anatomy.

II. Patient symptoms must correlate with positive imaging findings to surgically address the specific pathology.

III. Once a diagnosis is confirmed, the surgeon must plan the best surgical treatment option based on each patient's individual anatomy.

IV. Plain film radiographs are often the first imaging study obtained for most spine-related complaints.

- A. Lateral views are useful to assess spinal alignment and instability.
  1. Cervical spine **(Fig. 3.1).**
  2. Lumbar spine **(Fig. 3.2).**
- B. Open-mouth view: to assess the dens and atlantoaxial joint **(Fig. 3.3).**

Fig. 3.1 Lateral plain film radiograph of **(A)** the anterior border of the vertebral bodies, **(B)** the posterior border of the vertebral bodies, and **(C)** the junction of the laminae and spinous processes.

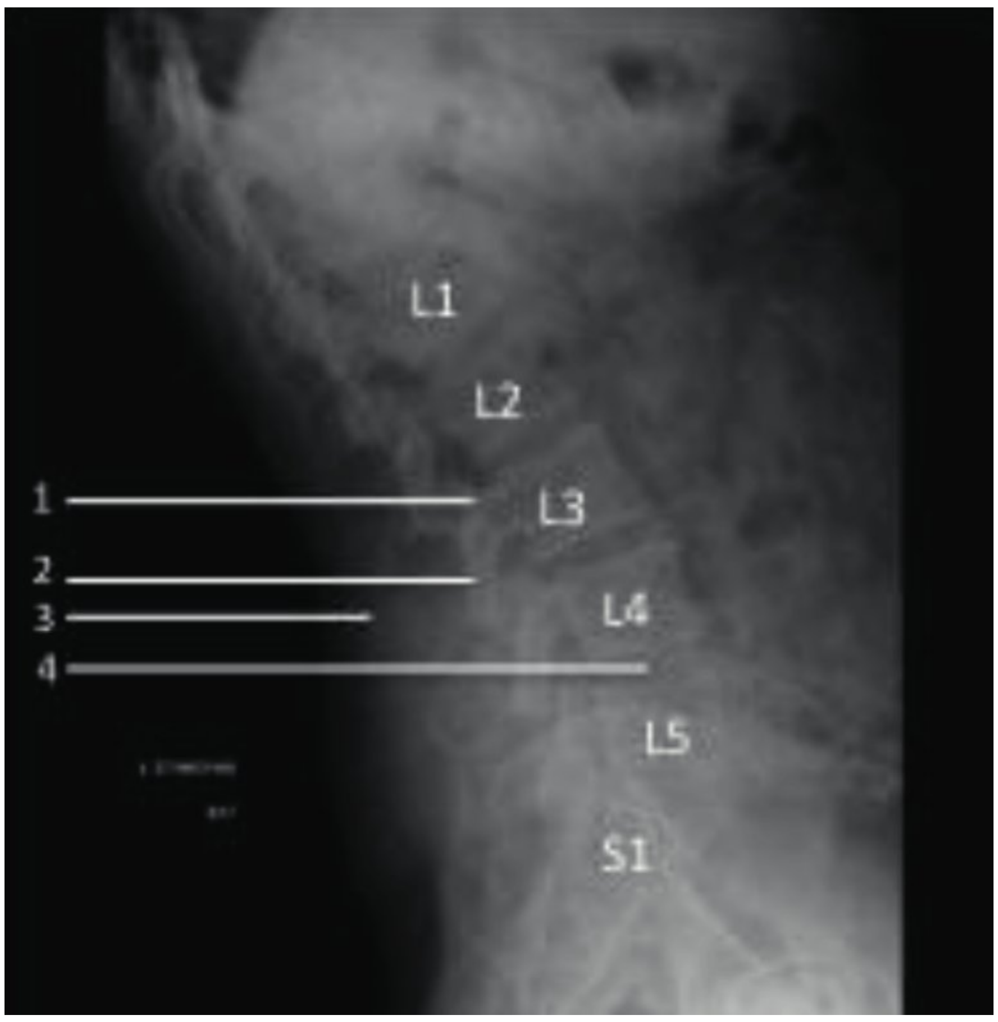

Fig. 3.2 Lumbar spine (lateral plain film radiograph in extension). 1, superior articular process (SAP); 2, inferior articular process (IAP); 3, spinous process; 4, intervertebral disk space (no evidence of anterior slip).

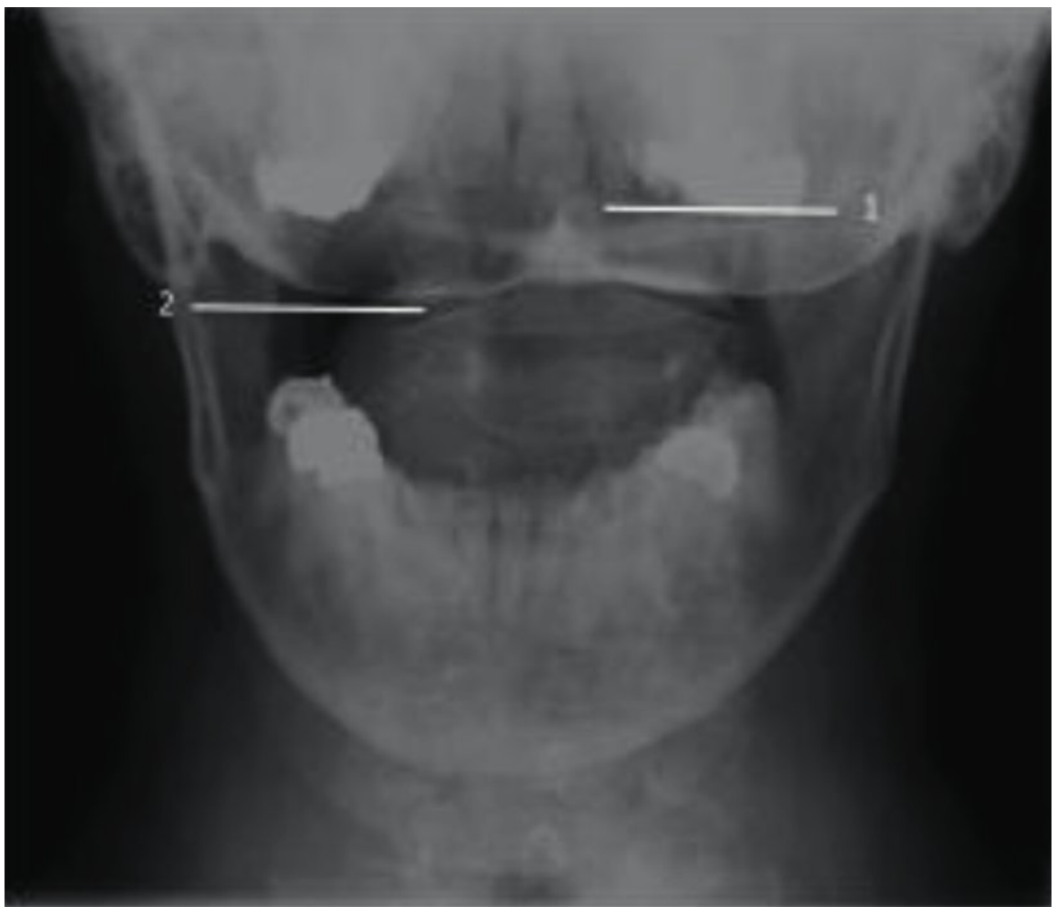

Fig. 3.3 Radiograph, open-mouth view. 1, dens; 2, atlantoaxial joint.

V. Magnetic resonance imaging (MRI) is particularly useful to analyze regional anatomy and assess the safety and feasibility of surgery.
   A. Cervical spine **(Fig. 3.4).**
   B. Lumbar spine **(Fig. 3.5).**

VI. Computed tomography (CT) can also be used for preoperative planning **(Fig. 3.6)**.

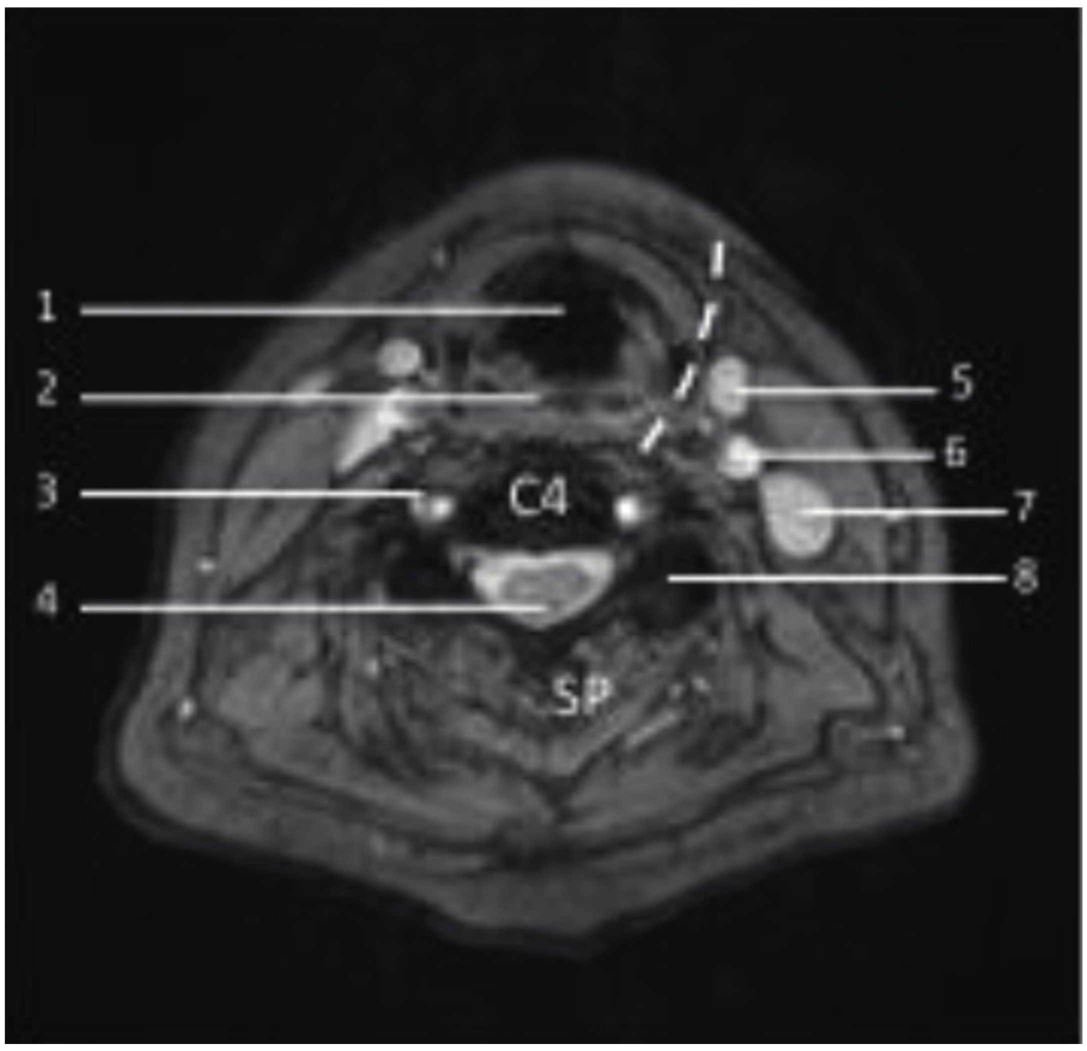

Fig. 3.4 Magnetic resonance imaging axial cut at C4 demonstrating normal anatomy. 1, trachea; 2, esophagus; 3, transverse process (foramina transversaria); 4, spinal cord (note the high-intensity signal surrounding the spinal cord (cerebrospinal fluid); 5, external carotid artery; 6, internal carotid artery; 7, internal jugular vein; 8, facet joint complex; dotted line, surgical plane for ACDF.

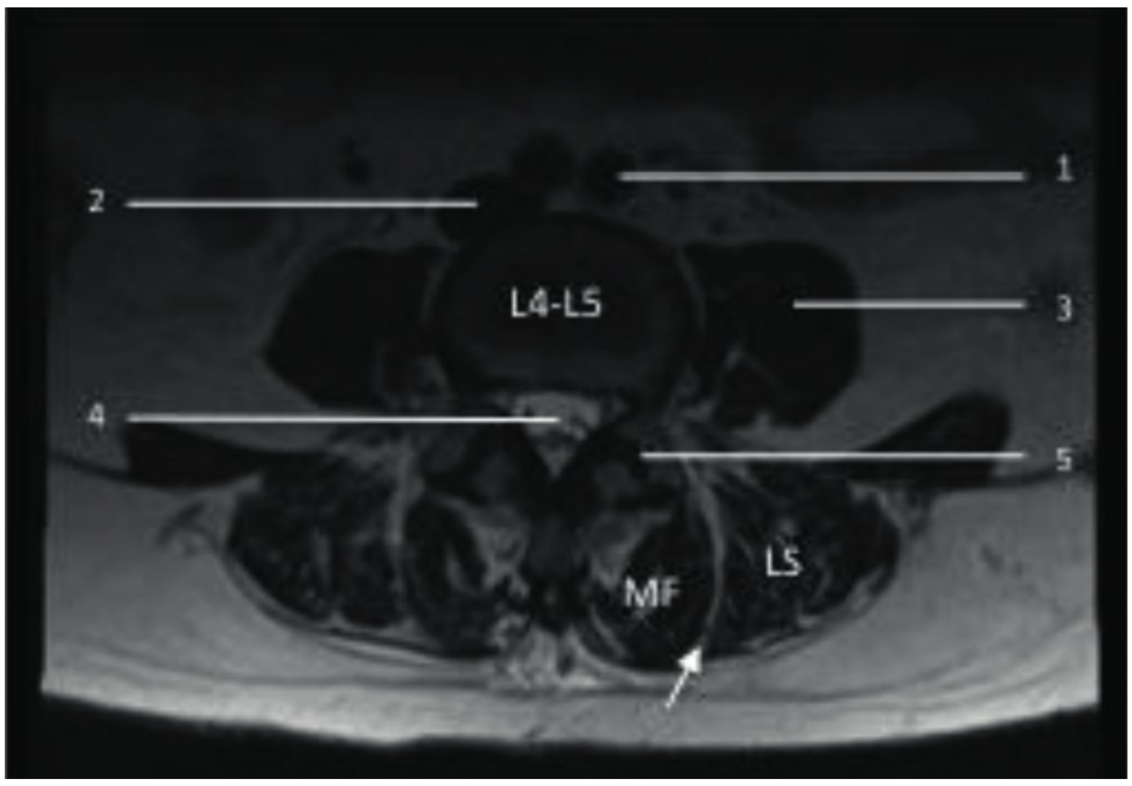

Fig. 3.5 Lumbar spine (magnetic resonance imaging, T2—axial cut L4–L5 disk). 1, left common iliac artery (immediately after bifurcation of the abdominal aorta; 2, inferior vena cava (prior to bifurcation of the left and right common iliac veins; 3, left psoas muscle; 4, spinal canal (cauda equina); 5, facet joint; MF, multifidus muscle; LS, longissimus muscle; arrow, intermuscular Wiltse plane (used in minimally invasive approaches and pedicle screw placement).

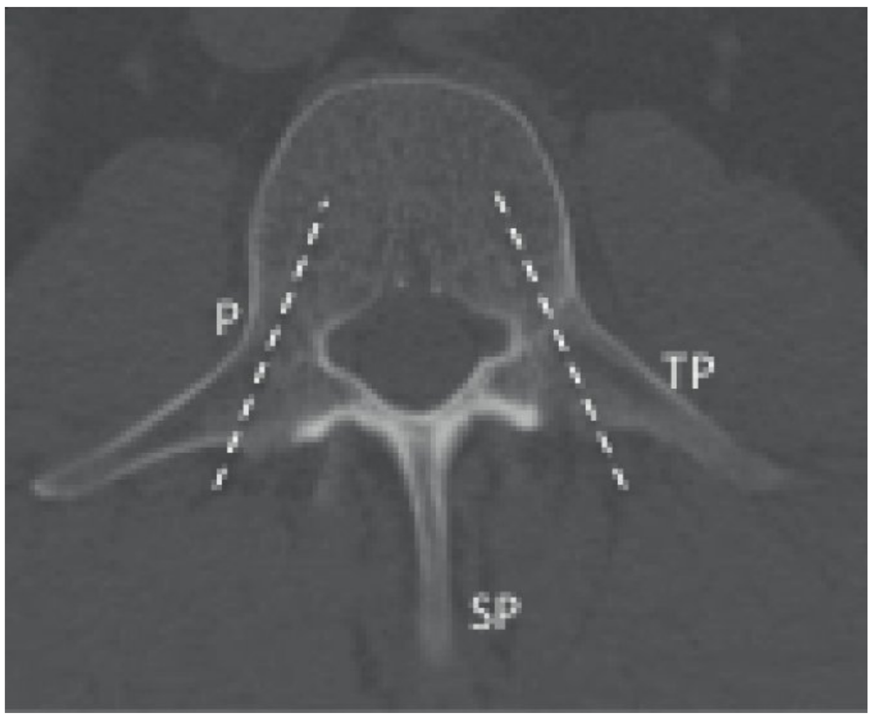

Fig. 3.6 Lumbar spine (axial computed tomography at L3). P, pedicle; SP, spinous process; TP, transverse process; dotted lines, trajectory for spinal access needles and pedicle screws.

3.2 Common Spinal Pathologies

I. Spondylosis: degenerative spinal disease.
  A. Frequent radiographic findings.
    1. Loss of cervical lordosis.
    2. Disk space narrowing **(Fig. 3.7).**
    3. Osteophyte formations **(Fig. 3.8).**
      a. Osteophytes can be observed in plain film radiographs, but CT better delineates the size and extent of osteophytic formation (important for surgical planning).

Fig. 3.7 Lateral plain film radiograph. 1, normal disk height; 2, decreased disk height; solid line, loss of cervical lordosis; dotted line, normal cervical lordosis.

Fig. 3.8 Lumbar spine (sagittal computed tomography). Notice osteophyte formation anteriorly. L1, pedicle; L3, inferior articular process (IAP); L4, superior articular process (SAP); L5–S1, spondylosis.

B. MRI changes.
   1. Reduced disk signal on T2.
      a. T2 is the preferred modality to characterize disk pathology. A normal disk will demonstrate a high-intensity nucleus surrounded by a low-intensity annulus.
         (1) Loss of disk height and darkening of the intervertebral disk are common MRI findings in degenerative disk pathology **(Fig. 3.9** and **Fig. 3.10)**.

Fig. 3.9 Sagittal magnetic resonance imaging cut of cervical spine. 1, posterior arch of C1 (atlas); 2, spinal cord; 3, C5–C6 disk degeneration with posterior disk protrusion; C7, spinous process.

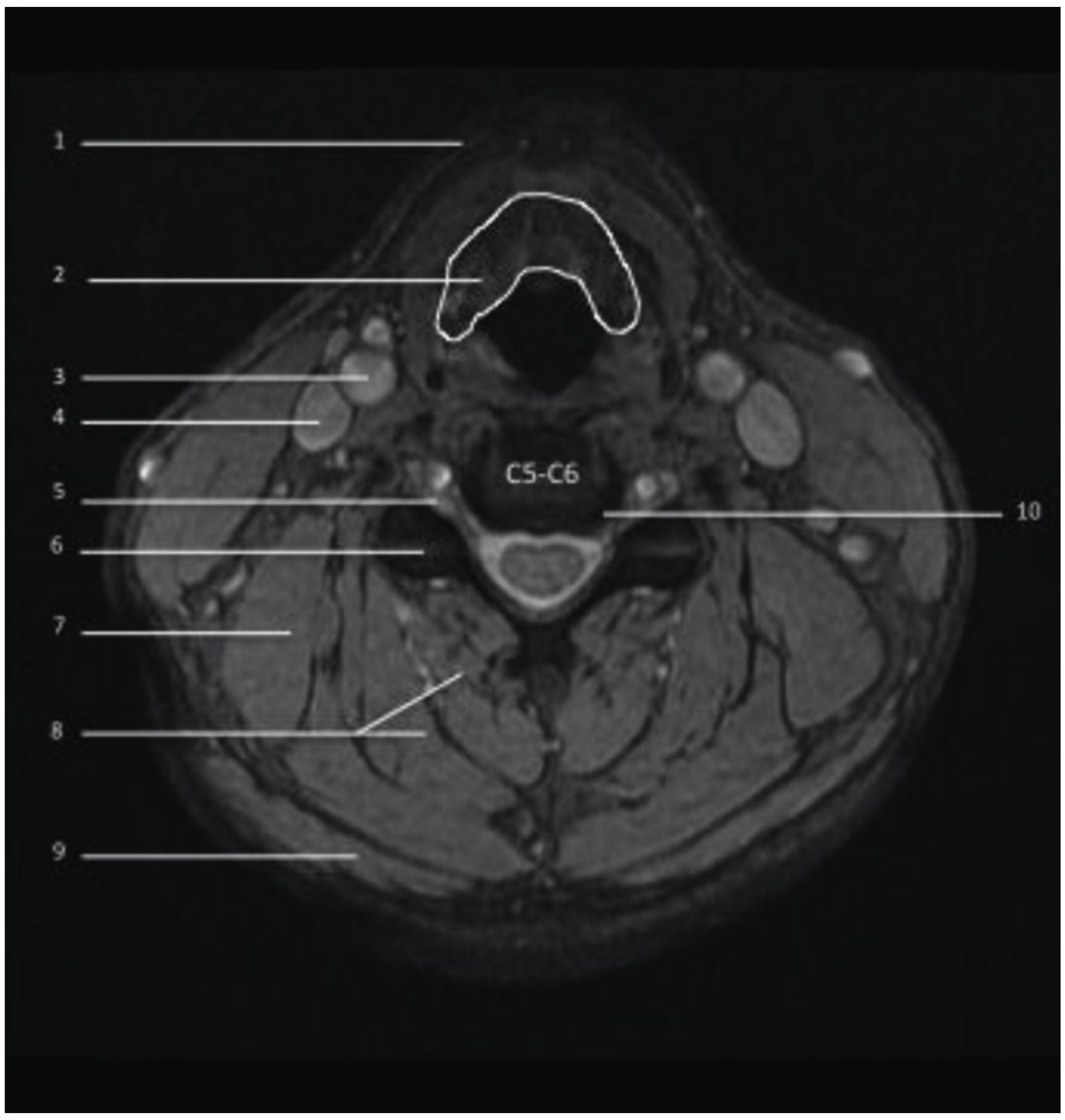

Fig. 3.10 Magnetic resonance imaging, axial cut at C5–C6. 1, platysma muscle; 2, thyroid gland; 3, common carotid artery; 4, internal jugular vein; 5, normal exiting nerve root; 6, facet joint; 7, longus colli muscle; 8, deep cervical muscles; 9, trapezius; 10, left herniated nucleus pulposus (HNP) causing foraminal stenosis.

2. Facet joint hypertrophy.
3. Intervertebral disk bulge or protrusion (herniated nucleus pulposus).
   a. Central.
   b. Paracentral.
   c. Far lateral **(Fig. 3.11** and **Fig. 3.12).**

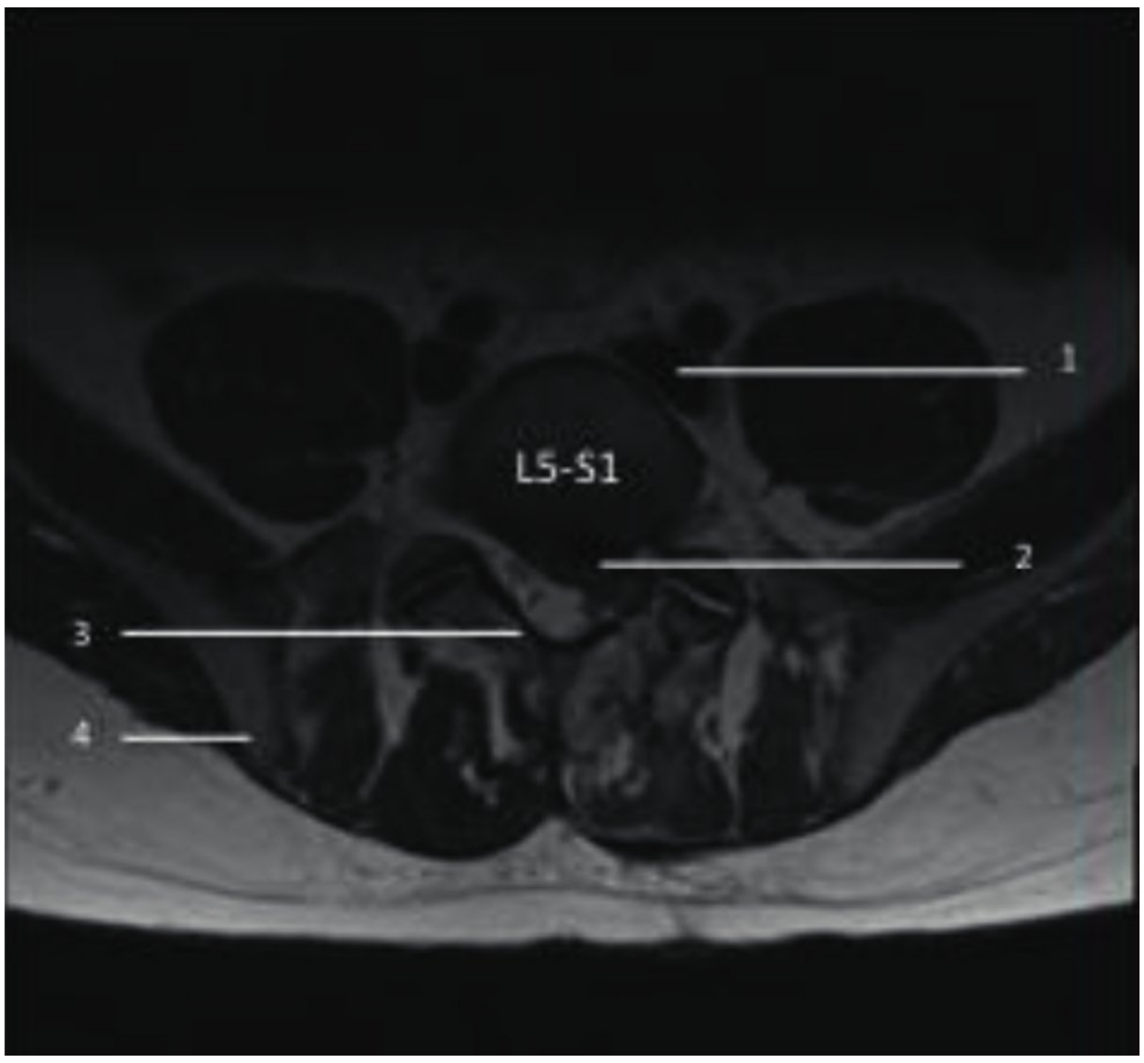

Fig. 3.11 Magnetic resonance imaging axial cut at L5–S1. 1, left common iliac vein; 2, left paracentral herniated nucleus pulposus; 3, left L5 lamina; 4, ilium.

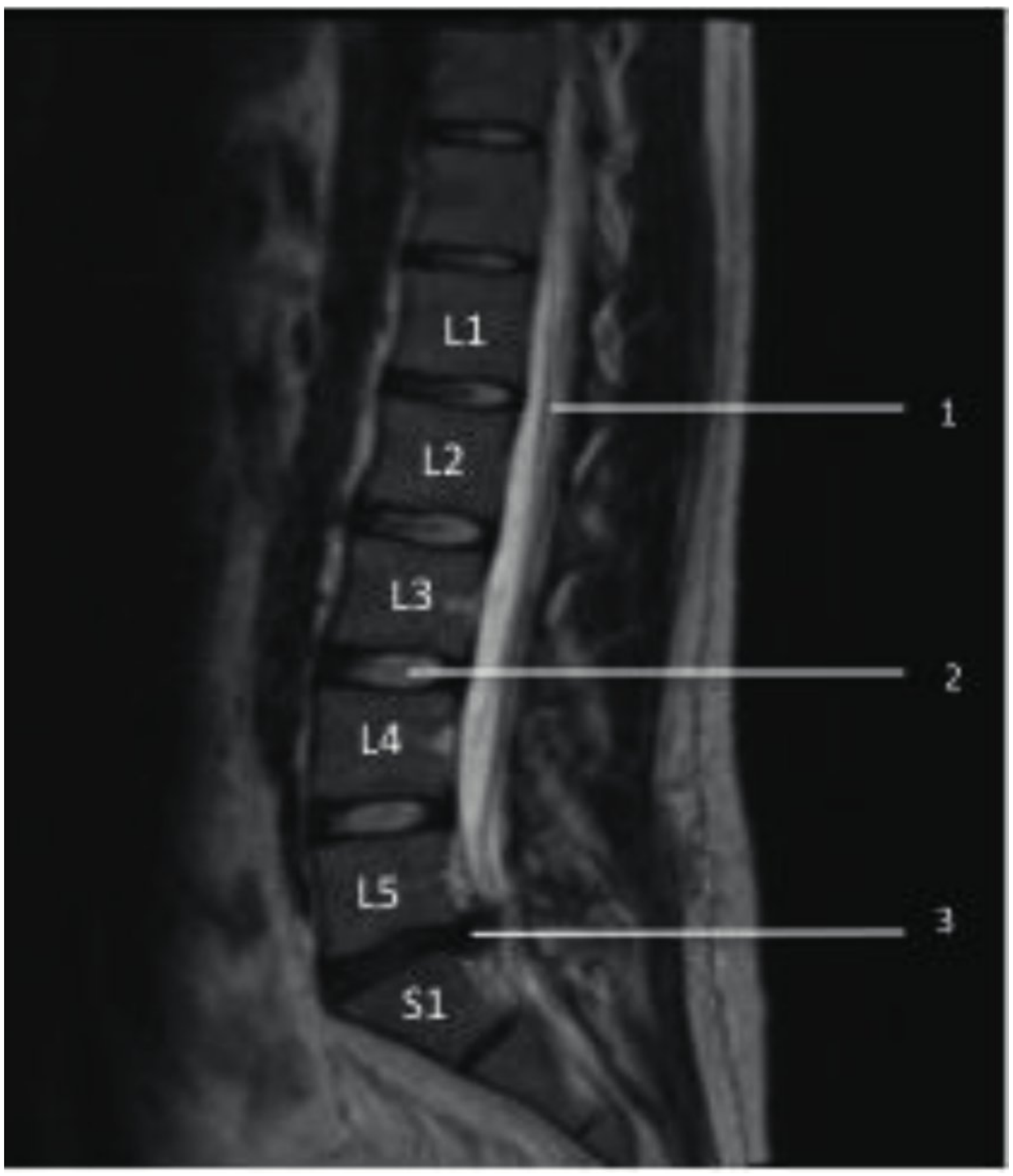

Fig. 3.12 Magnetic resonance imaging sagittal cut through the spinous process. 1, end of spinal cord (conus medullaris); 2, normal L3–L4 intervertebral disk intensity; 3, L5–S1 disk degeneration with posterior herniation.

II. Vertebral body collapse.
   A. Compression fractures in osteoporotic patients.
   B. Pathological fractures from tumors (more often metastatic).
   C. Characterized by loss of vertebral body height with or without segmental kyphosis in lateral plain film radiographs **(Fig. 3.13** and **Fig. 3.14).**
   D. CT is the best imaging study for assessing bony anatomy.

Fig. 3.13 Lateral plain film radiograph. L1 body demonstrates decreased body height with local kyphotic deformity.

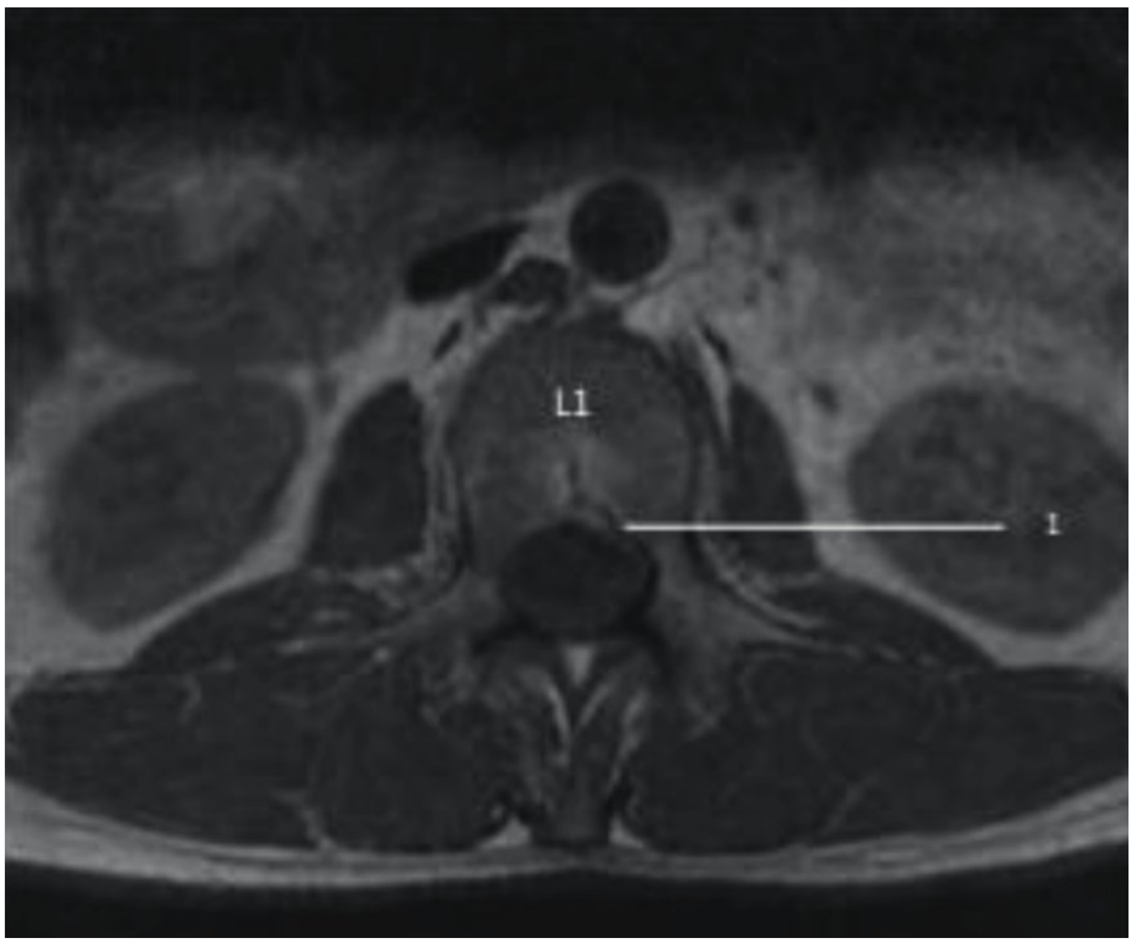

Fig. 3.14 Magnetic resonance imaging axial cut at the L1 body. 1, bony defect consistent with compression fracture.

III. Spinal stenosis.
   A. Narrowing of the spinal canal or neuroforamen can cause neurological symptoms **(Fig. 3.15, Fig. 3.16,** and **Fig. 3.17)**.
   B. Narrowing of the spinal canal more often occurs in elderly patients (> 60 years) due to degenerative changes **(Fig. 3.18)**:
      1. Disk prolapse.
      2. Hypertrophic facet joints or ligamentum flavum.
      3. Degenerative spondylolisthesis.
   C. MRI is the modality of choice for assessing size and shape of the spinal canal.

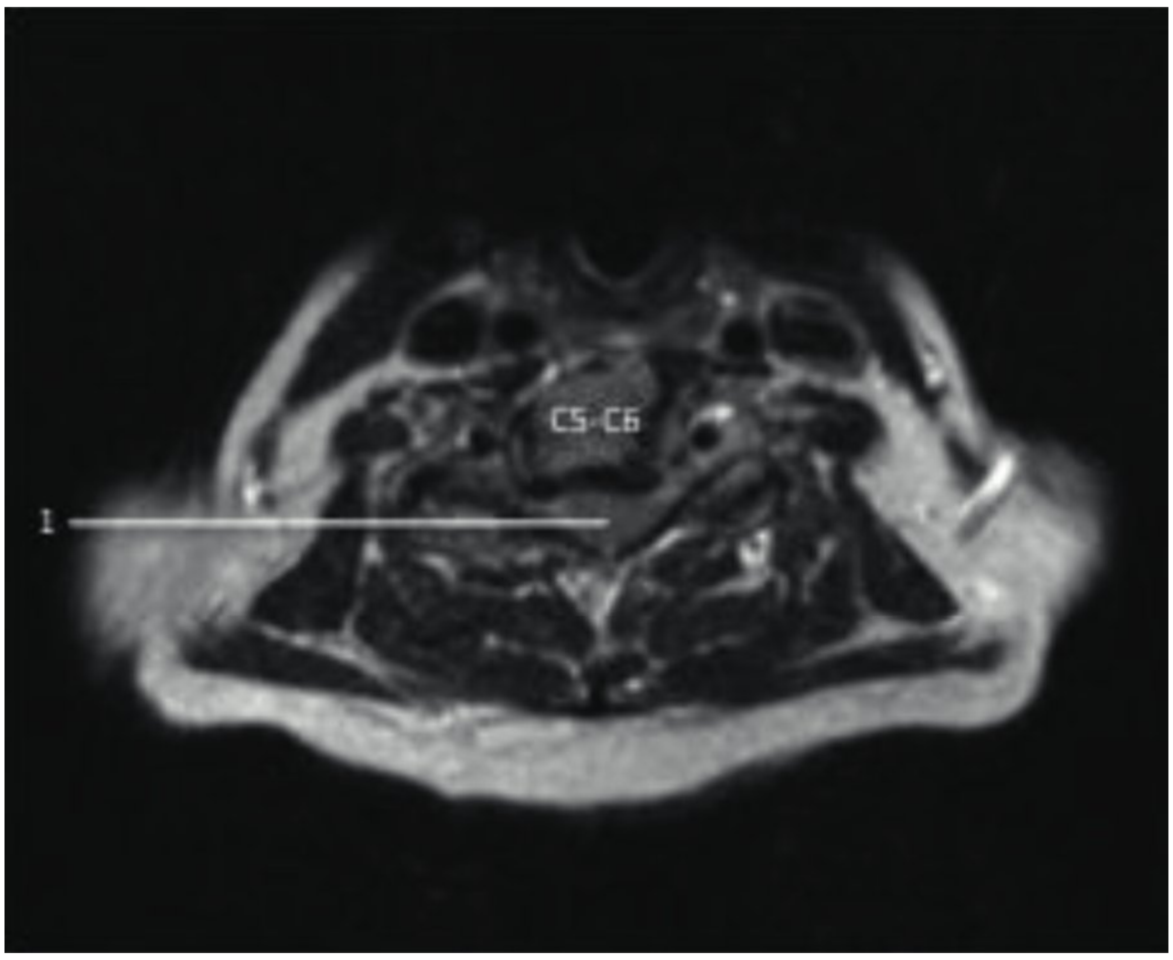

Fig. 3.15 Magnetic resonance imaging axial cut at C5–C6 disk space: severe spinal stenosis. 1, central spinal stenosis (note the absence of high-signal cerebrospinal fluid surrounding the spinal cord).

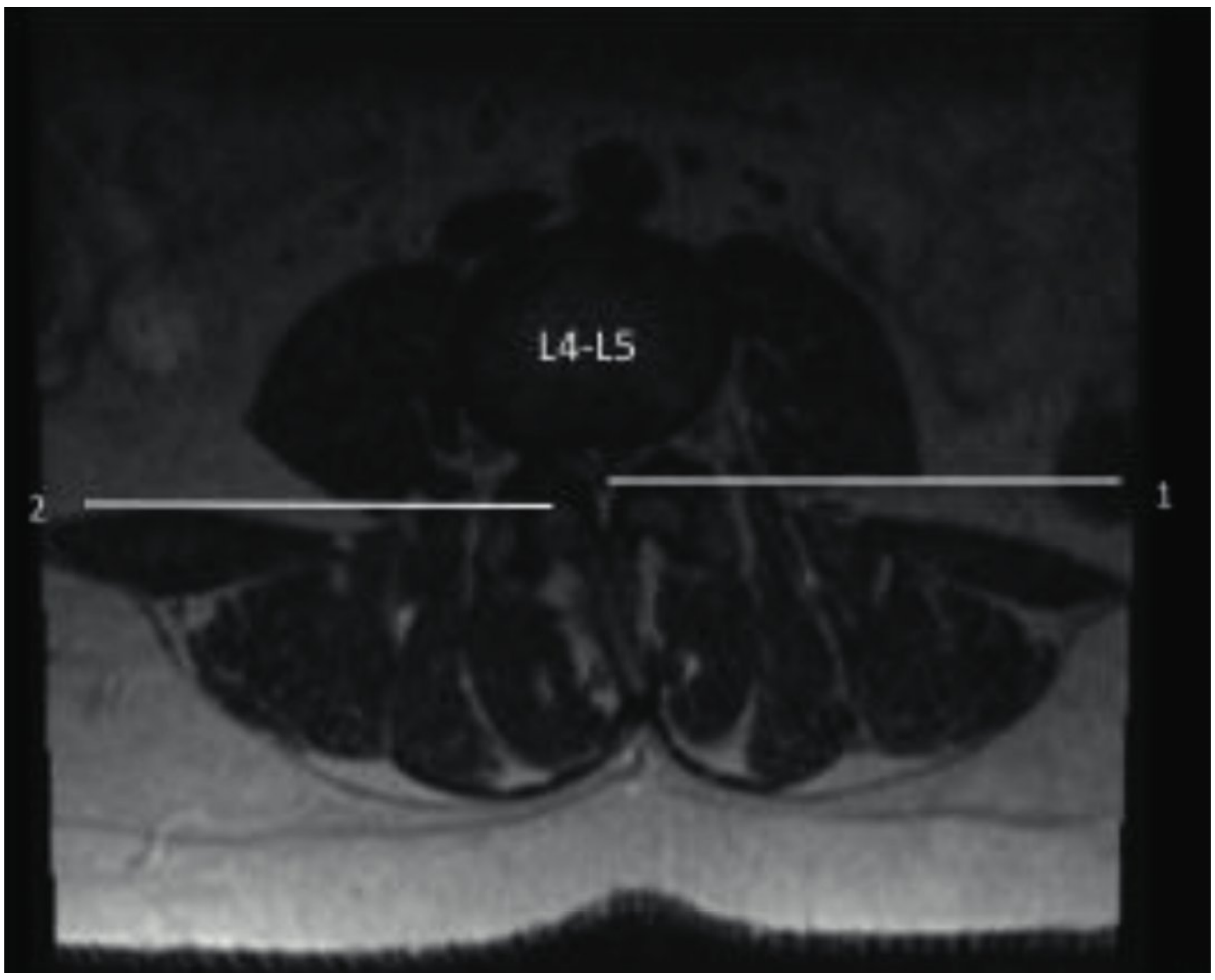

Fig. 3.16 Magnetic resonance imaging axial cut at L4–L5 disk space. 1, severe lumbar spinal stenosis; 2, facet joint hypertrophy.

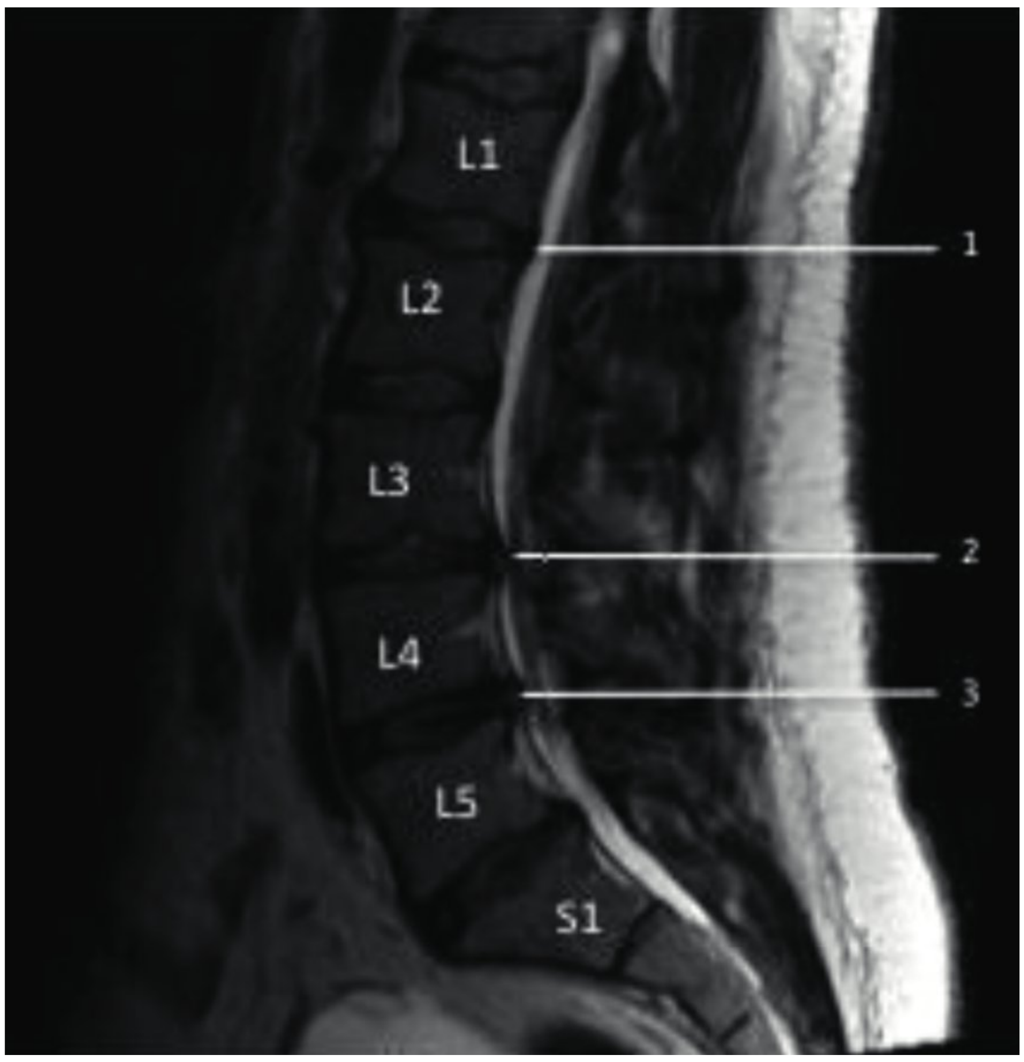

Fig. 3.17 Magnetic resonance imaging (MRI) sagittal cut through the spinous process. 1, end of spinal cord (conus medullaris); 2, L3–L4 disk prolapse; 3, L4–L5 disk prolapse. MRI sagittal cut through right pedicles. 4, L1 pedicle; 5, normal L2 exiting nerve root; 6, stenosed L4 nerve root.

Fig. 3.18 Magnetic resonance imaging sagittal cut through the left-side pedicles. 1, normal nerve root (high-intensity fat surrounding low-intensity nerve root); 2, L3 pedicle; 3, L4 spinous process; 4, L5 foraminal stenosis.

IV. Spondylolisthesis.

A. Refers to the forward slip of a vertebral body on the one below **(Fig. 3.19).**

B. More often occurs as a result of a defect in the pars interarticularis but can also occur as a result of degenerative disk disease.

C. Plain film radiographs are used to determine the degree of the listhesis **(Fig. 3.20)**.

D. MRI allows visualization of neural structures.

1. Spinal cord.
2. Spinal nerve roots.
    a. Foraminal stenosis.

E. CT imaging is the most sensitive for detecting a pars defect.

1. It can also be used for preoperative planning.

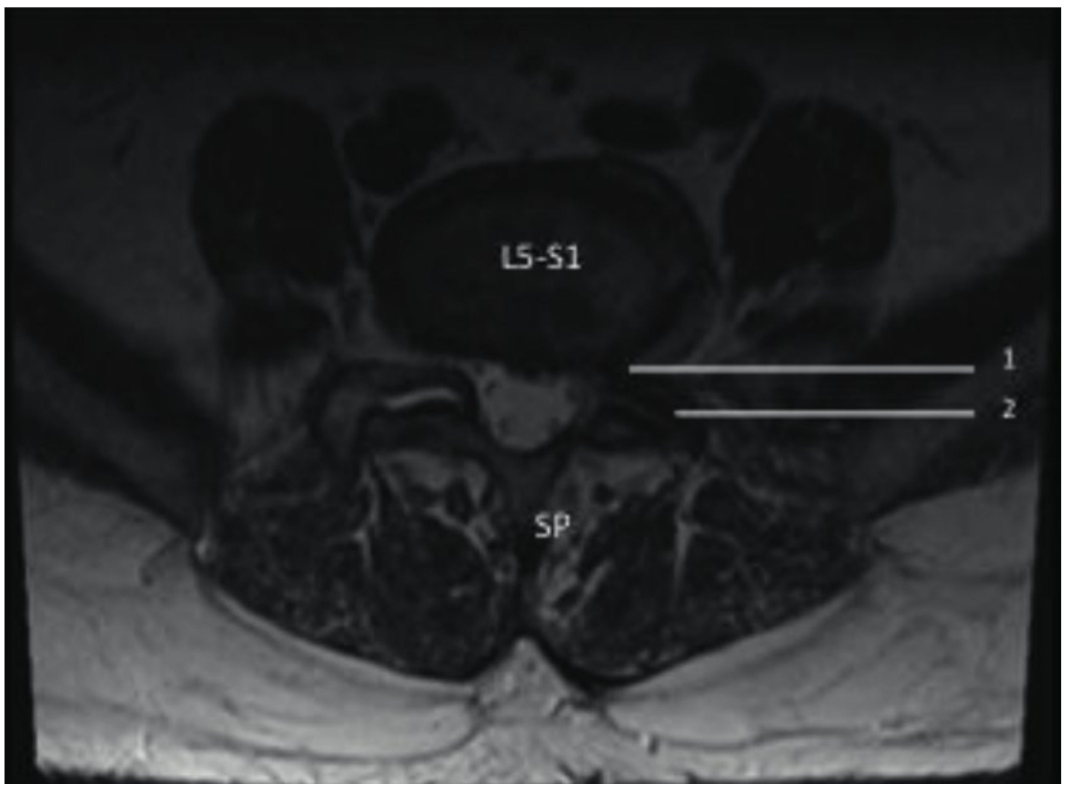

Fig. 3.19 Magnetic resonance imaging axial cut L5–S1 disk. 1, left foraminal stenosis; 2, facet joint arthropathy; SP, spinous process.

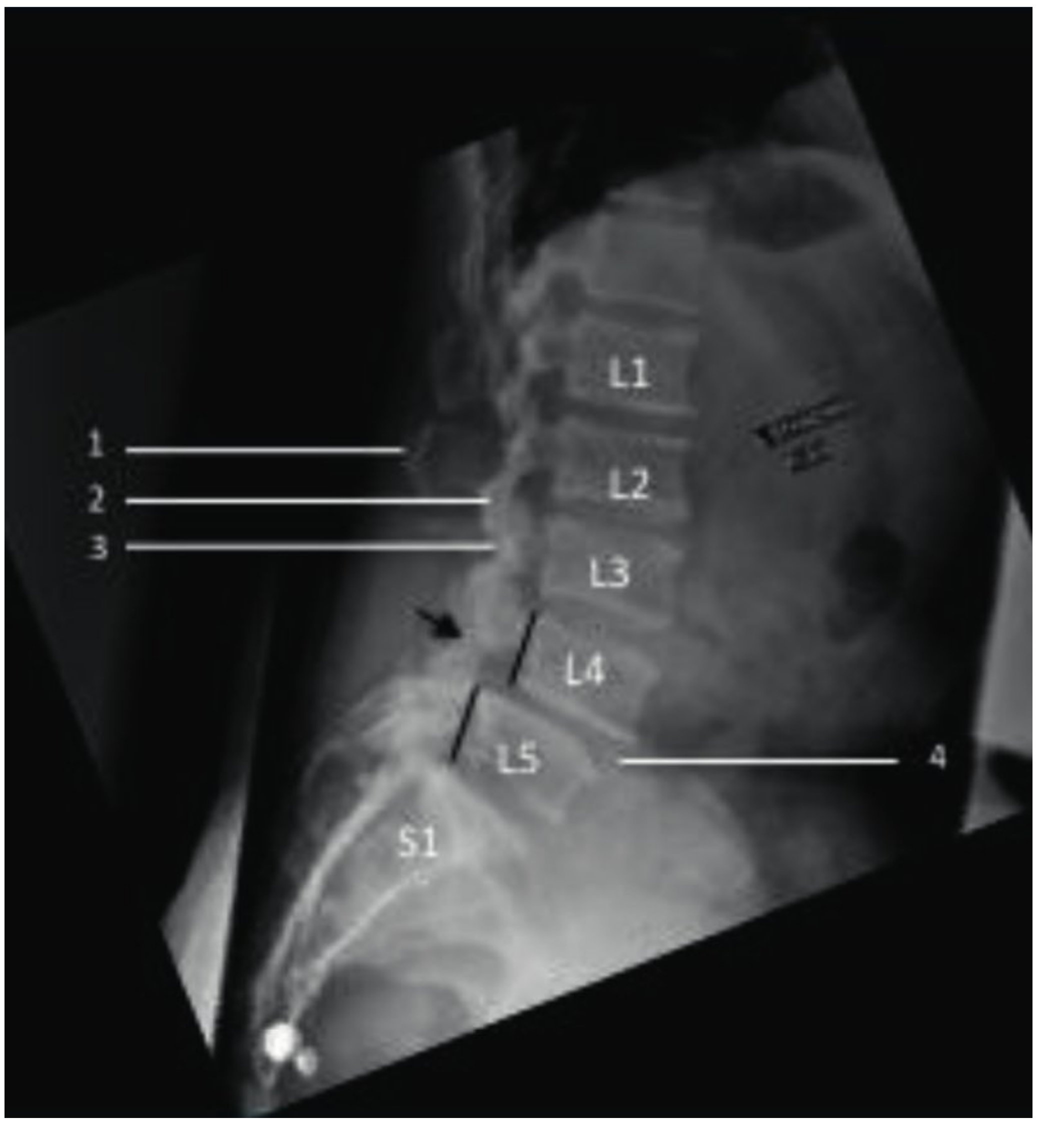

Fig. 3.20 Lateral plain film radiograph (flexion). 1, L2 spinous process; 2, L2 inferior articular process; 3, L3 superior articular process; 4, L4–L5 slip (anterolisthesis); arrow, pars defect.

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4 Spinal Imaging and Diagnostic Tests

4.1 Imaging Modalities

I. General considerations.

   A. Spinal imaging modalities **(Table 4.1)**:

      1. Plain radiographs.
      2. Computed tomography.
      3. Magnetic resonance imaging (MRI).
      4. Bone scintigraphy.
      5. Myelography.
      6. Angiography.
      7. Diskography.

   B. A thorough history and physical examination should lead to a preliminary clinical diagnosis that should predicate both the selection and the timing of imaging tests.

      1. Diagnostic tests should be used to confirm information ascertained during the history and physical examination.

   C. Selection of imaging tests should be based on the appreciation of the sensitivity, specificity, and accuracy of various imaging modalities in conjunction with different disease processes.

      1. Acute neck or back pain and radiculopathy:

         a. The natural history is that of improvement with conservative treatment.

         b. Diagnostic imaging should be delayed until 4 to 6 weeks after the onset of symptoms.

            (1) There are exceptions to an earlier imaging evaluation:

               (a) Trauma.

               (b) Progressive neurological deficit.

               (c) Suspected neoplasm or infection.

      2. Imaging evaluation alone without clinical correlation is associated with an extremely high false-positive rate.

         a. Plain radiographs show aging and degenerative processes in virtually all individuals after the age of 40 years **(Fig. 4.1)**.

         b. MRI findings of the cervical spine demonstrate the following:

            (1) Fourteen percent of asymptomatic individuals < 40 years old and 28% > 40 years old had evidence of a herniated disk.

            (2) Degenerative disk disease is more common in asymptomatic individuals, with an incidence of 25% for < 40 years old and 56% for > 40 years old.

         c. MRI findings of the lumbar spine demonstrate the following:

            (1) In asymptomatic individuals, a herniated disk was noted in 21% of patients between 20 and 39 years of age and 36% of individuals > 60 years of age.

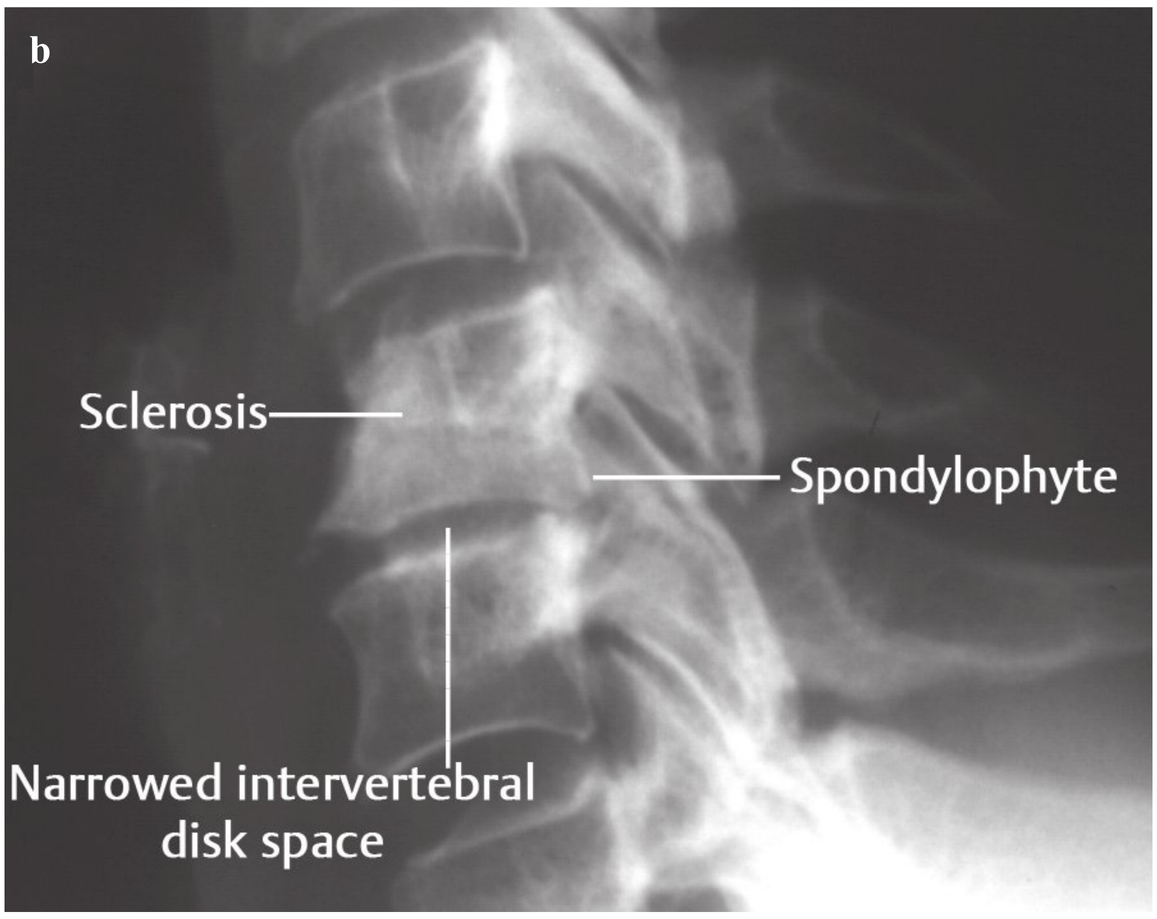

Fig. 4.1 **(a)** Lateral radiograph of the lumbar spine demonstrating a vacuum disk sign at L4–L5. This is suggestive of disk space collapse and disk degeneration. **(b)** Lateral radiograph of the cervical spine demonstrating disk degeneration. (Reproduced from Bohndorf K, Imhof H, Pope TH Jr. Musculoskeletal Imaging: A Concise Multimodality Approach. Stuttgart, Germany: Georg Thieme Verlag; 2001: Figs. 9.61 and 9.62, with permission.)

(2) Spinal stenosis is found in 21% of those > 60 years of age, and bulging disks are found in > 50% of patients in all age groups.

II. MRI **(Fig. 4.2)**.

A. Contraindications:

1. Ferrous metal implants in the brain.
2. Metal debris in the eye.
3. Inner ear implants.
4. Pacemakers.

Fig. 4.2 Magnetic resonance imaging (T2 sagittal image) of the lumbar spine with decreased signal intensity at the L4–L5 and L5–S1 interspace with minimal loss of disk height.

B. Imaging around metal implants is poor unless special techniques are used and if the metal is titanium instead of stainless steel.
C. T1- versus T2-weighted images take advantage of intrinsic tissue properties **(Fig. 4.3) (Table 4.2)**.
   1. Repetition time (TR): time between radiofrequency (RF) pulses.
   2. Echo time (TE): time between RF and recording.
   3. T1-weighted image: short TR (400–600 ms), short TE (5–30 ms).
   4. T2-weighted image: long TR (1,500–3,000 ms), long TE (50–120 ms).
D. Special indications:
   1. Postoperative scar versus recurrent disk herniation.
      a. Use of gadolinium contrast.
      b. The scar is vascular and enhances with gadolinium. The disk does not enhance with contrast agents. This is observed on T1-weighted sequence.
   2. Infection versus tumor.
      a. In spinal osteomyelitis, there is abnormal tissue with decreased signal intensity on T1-weighted images and increased signal intensity on T2-weighted images at the disk margin.
      b. In tumors, the intervertebral disk is spared, and similar changes are noted involving the entire vertebral body.
   3. Compression fractures versus pathological fractures.
      a. More difficult to differentiate.
      b. In pathological fractures.
         (1) Entire vertebral body involvement.
         (2) Frequent involvement of the pedicle.
         (3) Presence of soft tissue masses.
         (4) Canal compromise.

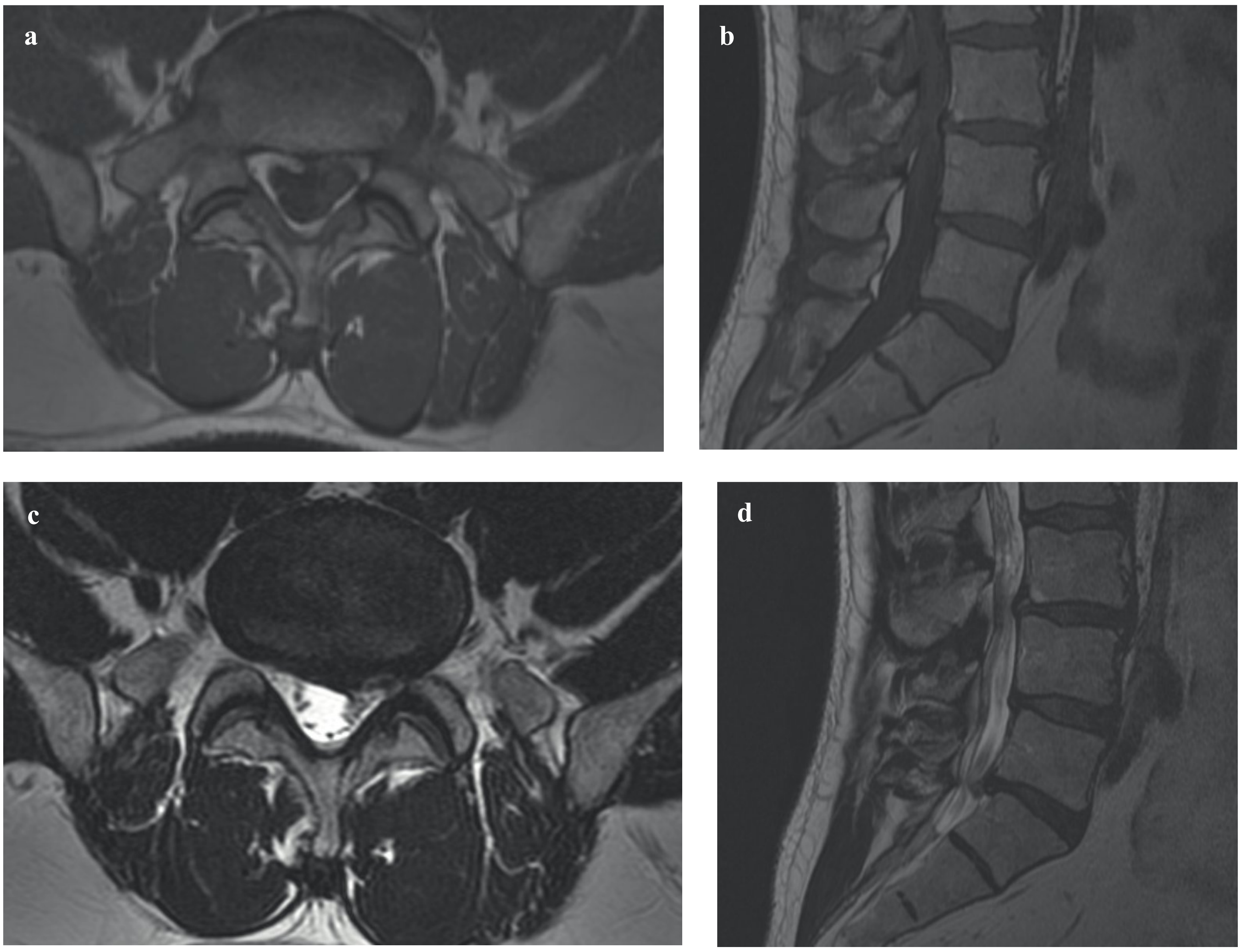

Fig. 4.3 **(a,b)** T1-weighted magnetic resonance imaging (axial and sagittal) of the lumbar spine. **(c,d)** T2-weighted MRI imaging (axial and sagittal) of the lumbar spine.

Table 4.2 Magnetic resonance imaging findings of human tissue

Tissue type	T1 signal	T2 signal
Cortical bone	Low	Low
Tendon/ligament	Low	Low
Hyaline cartilage	Intermediate	Intermediate
Free water	Low	High
Adipose	High	Low
Abscess	Intermediate	High

c. In osteoporotic compression fractures.
   (1) No involvement of the pedicle.
   (2) Partial involvement of the vertebral body.

4. Spinal cord injury.
   a. Distinguishes spinal cord edema versus hemorrhage.
      (1) Edema is bright on T2- and decreased on T1-weighted images **(Fig. 4.4).**

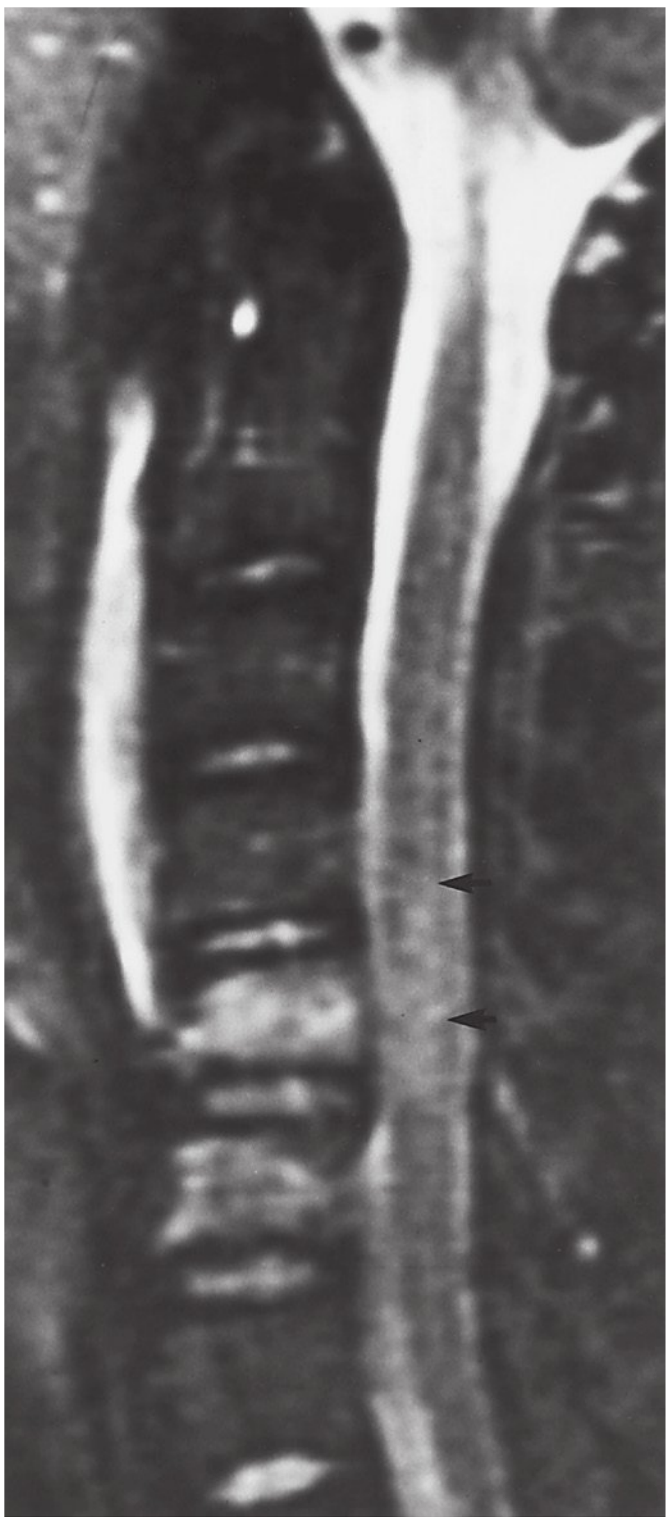

Fig. 4.4 T2-weighted magnetic resonance imaging demonstrating increased signal intensity suggestive of spinal cord edema. (Reproduced from Uhlenbrock D. MR Imaging of the Spine and Spinal Cord. Stuttgart, Germany: Georg Thieme Verlag; 2004: Fig. 7.6, with permission.)

(2) Hemorrhage is bright on T1- and decreased on T2-weighted images **(Fig. 4.5).**

E. Disk degeneration **(Fig. 4.6** and **Fig. 4.7)**:

1. A radial tear of the annulus fibrosus is outlined as a fissure extending from the nucleus to the periphery.
   a. A high-intensity zone in the posterior annulus suggests a radial tear that may be clinically significant.
2. Modic end plate changes:
   a. Type 1 **(Fig. 4.8).**
      (1) Low intensity on T1-weighted image and high intensity on T2-weighted image.
      (2) Associated with segmental spine instability and pain.
   b. Type 2 **(Fig. 4.9).**
      (1) High intensity on T1-weighted image and normal on T2-weighted image
      (2) Fatty marrow changes around the end plates.
      (3) Less likely to be symptomatic.
   c. Type 3 **(Fig. 4.10).**
      (1) Hypointense on T1- and T2-weighted images.
      (2) Sclerotic advanced degenerative changes with less segmental motion.

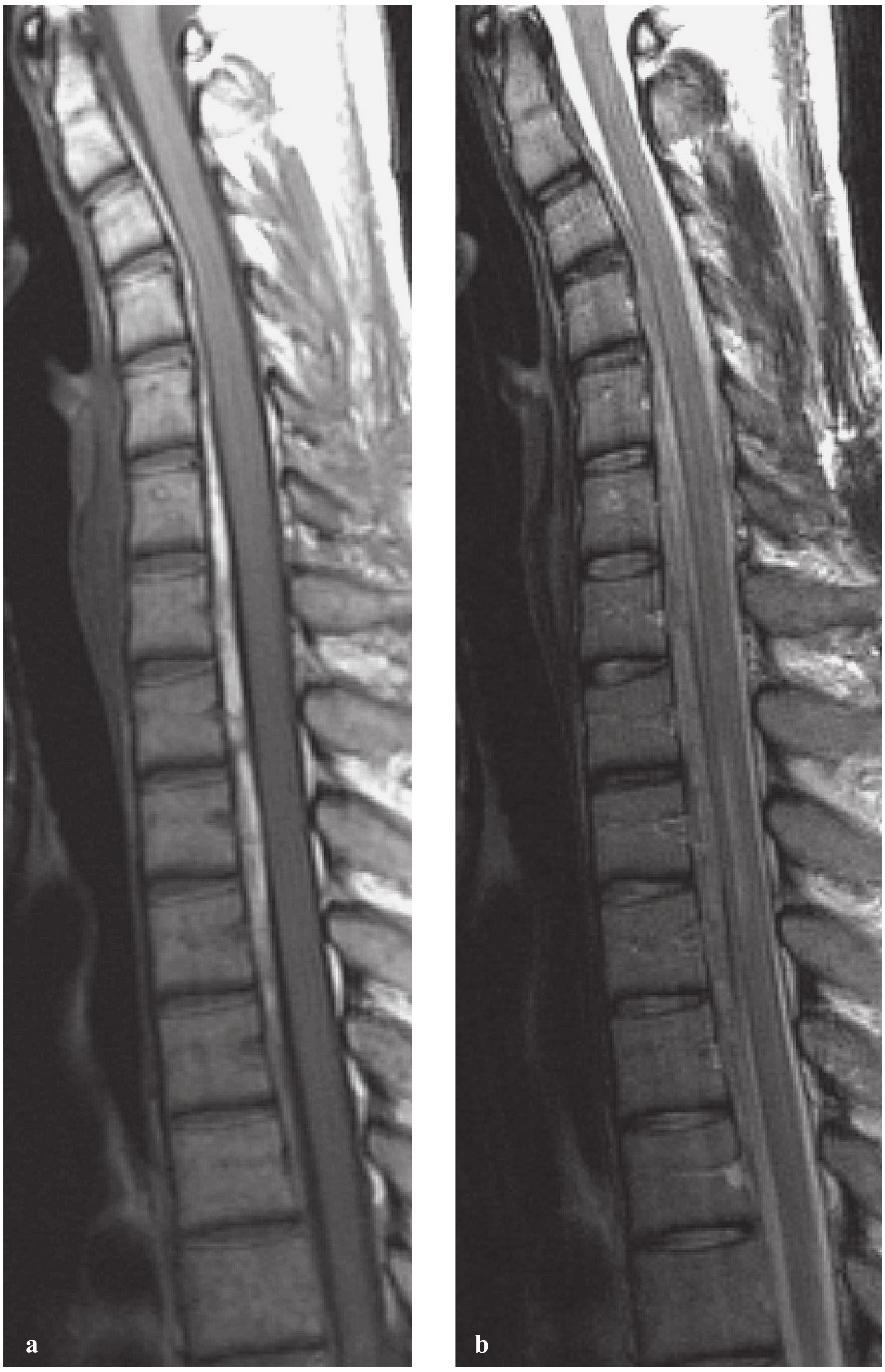

Fig. 4.5 **(a,b)** T1-weighted magnetic resonance imaging demonstrating increased signal intensity suggestive of hemorrhage. (Reproduced from Uhlenbrock D. MR Imaging of the Spine and Spinal Cord. Stuttgart, Germany: Georg Thieme Verlag; 2004: Fig. 8.2a,b, with permission.)

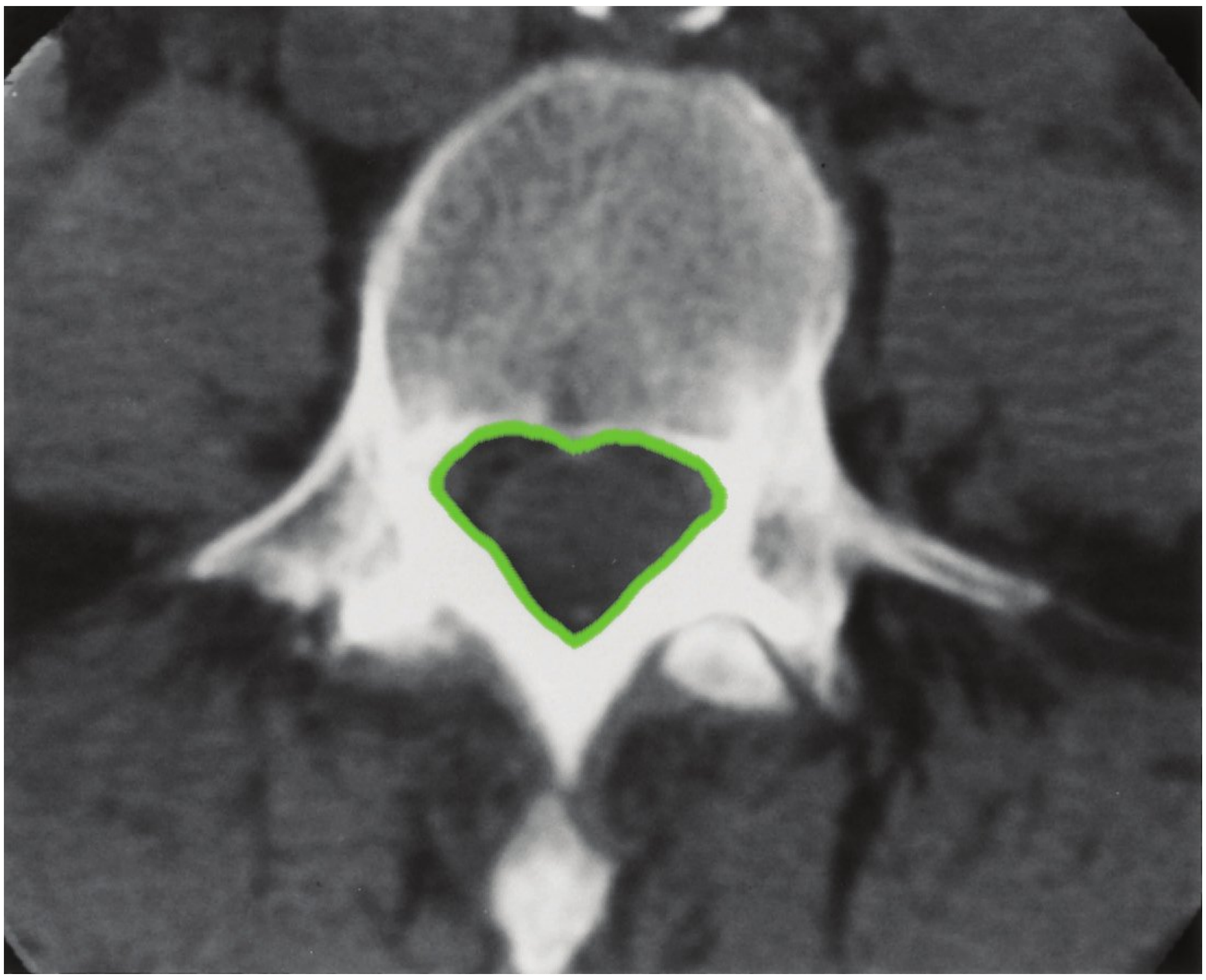

Fig. 4.6 Axial cut (computed tomographic scan) at L4 that demonstrates the pedicles and bony anatomy. Note: the thecal sac can be visualized as well.

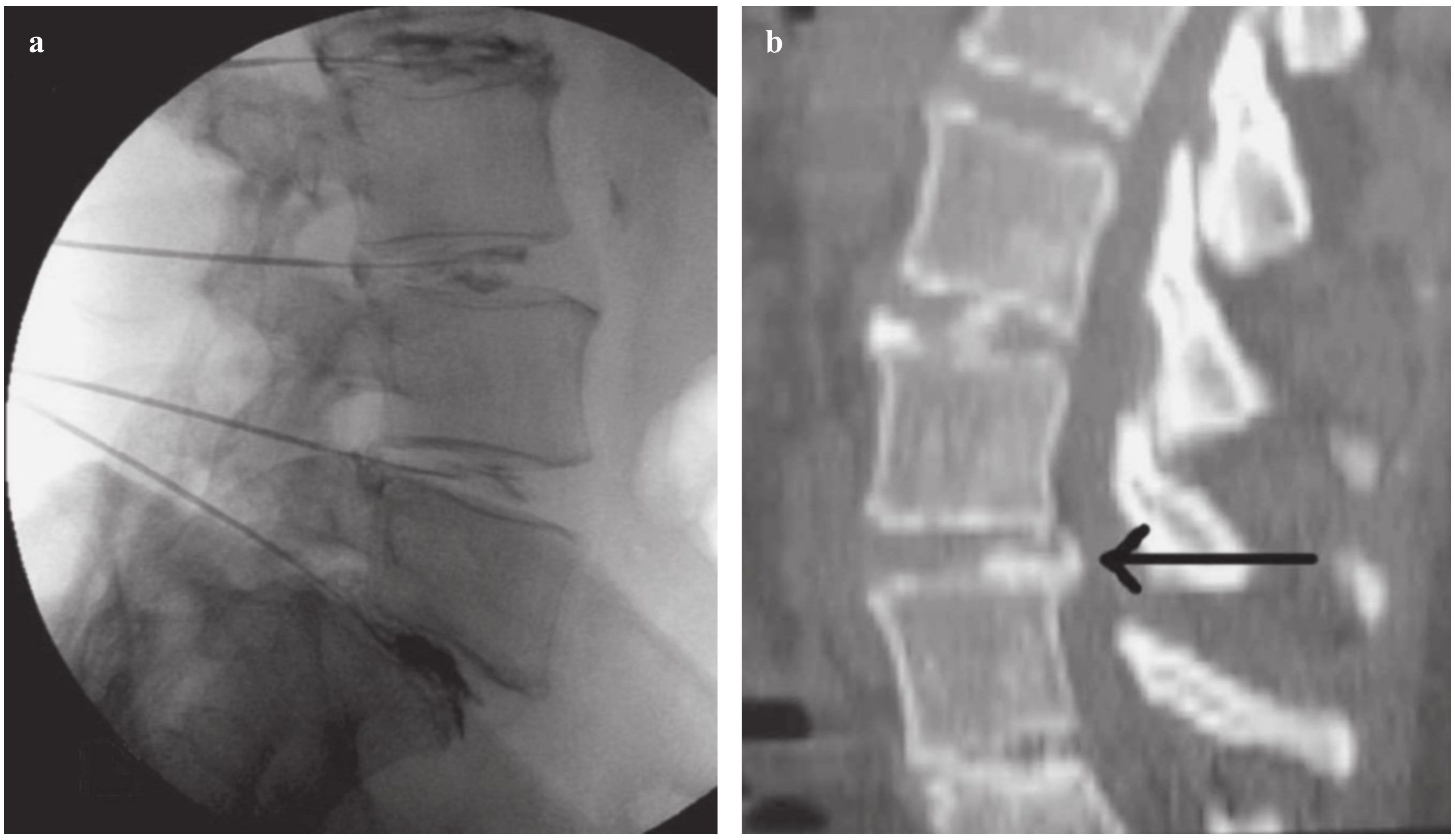

Fig. 4.7 **(a)** Lateral diskogram demonstrating extravasation of dye at the L5–S1 level suggestive of an annular tear. **(b)** Computed tomography postdiskography (sagittal) demonstrating extravasation of dye posteriorly at L4–L5.

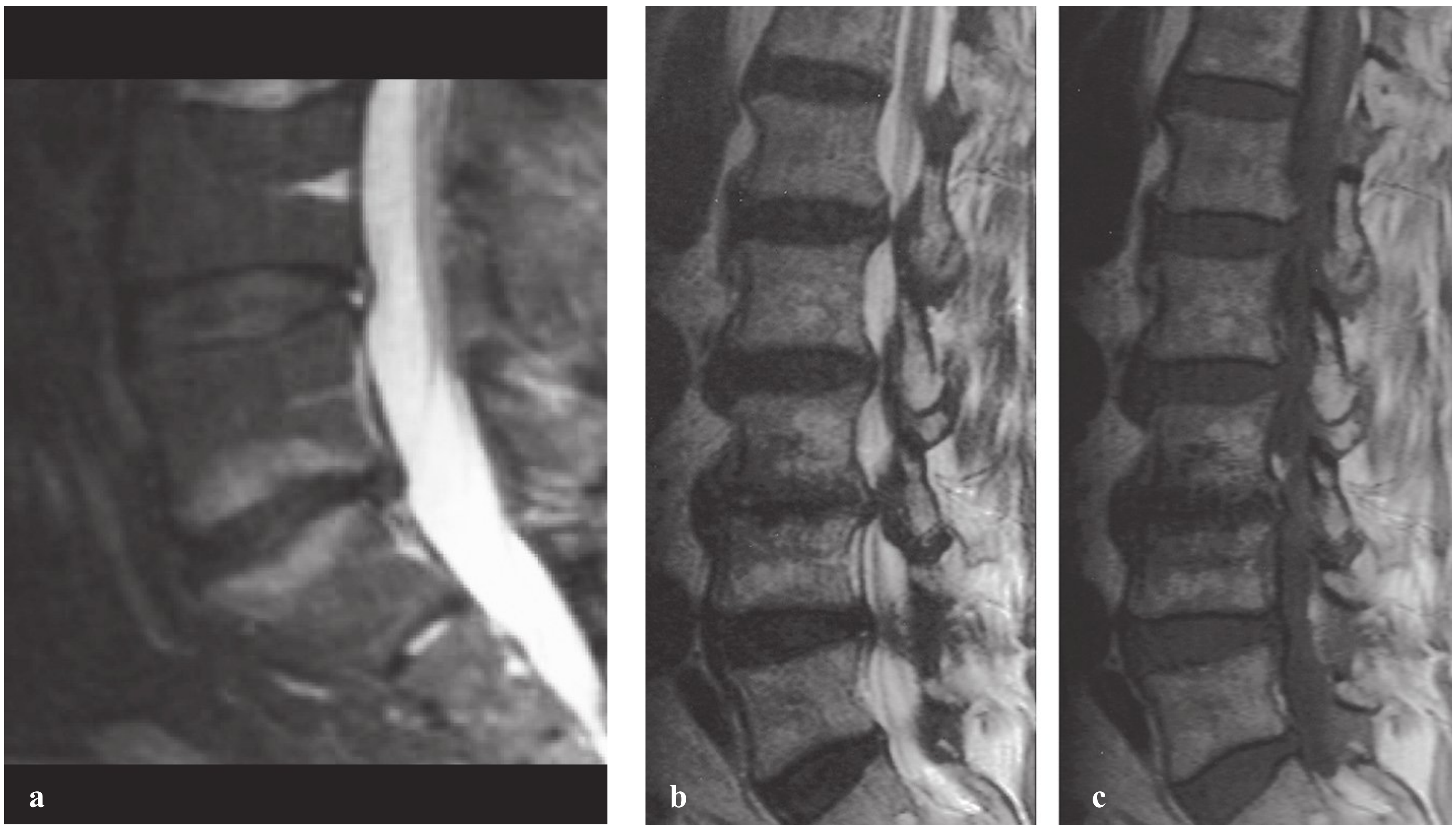

Fig. 4.8 **(a–c)** Sagittal magnetic resonance imaging demonstrating type 1 Modic changes. (Reproduced from Imhof H, et al. Spinal Imaging. Direct Diagnosis in Radiology Series. Stuttgart, Germany: Georg Thieme Verlag; 2008: Figs. 3.4 and 3.5, with permission.)

Fig. 4.9 Sagittal magnetic resonance imaging demonstrating type 2 Modic changes. (Reproduced from Uhlenbrock D. MR Imaging of the Spine and Spinal Cord. Stuttgart, Germany: Georg Thieme Verlag; 2004: Fig. 4.31, with permission.)

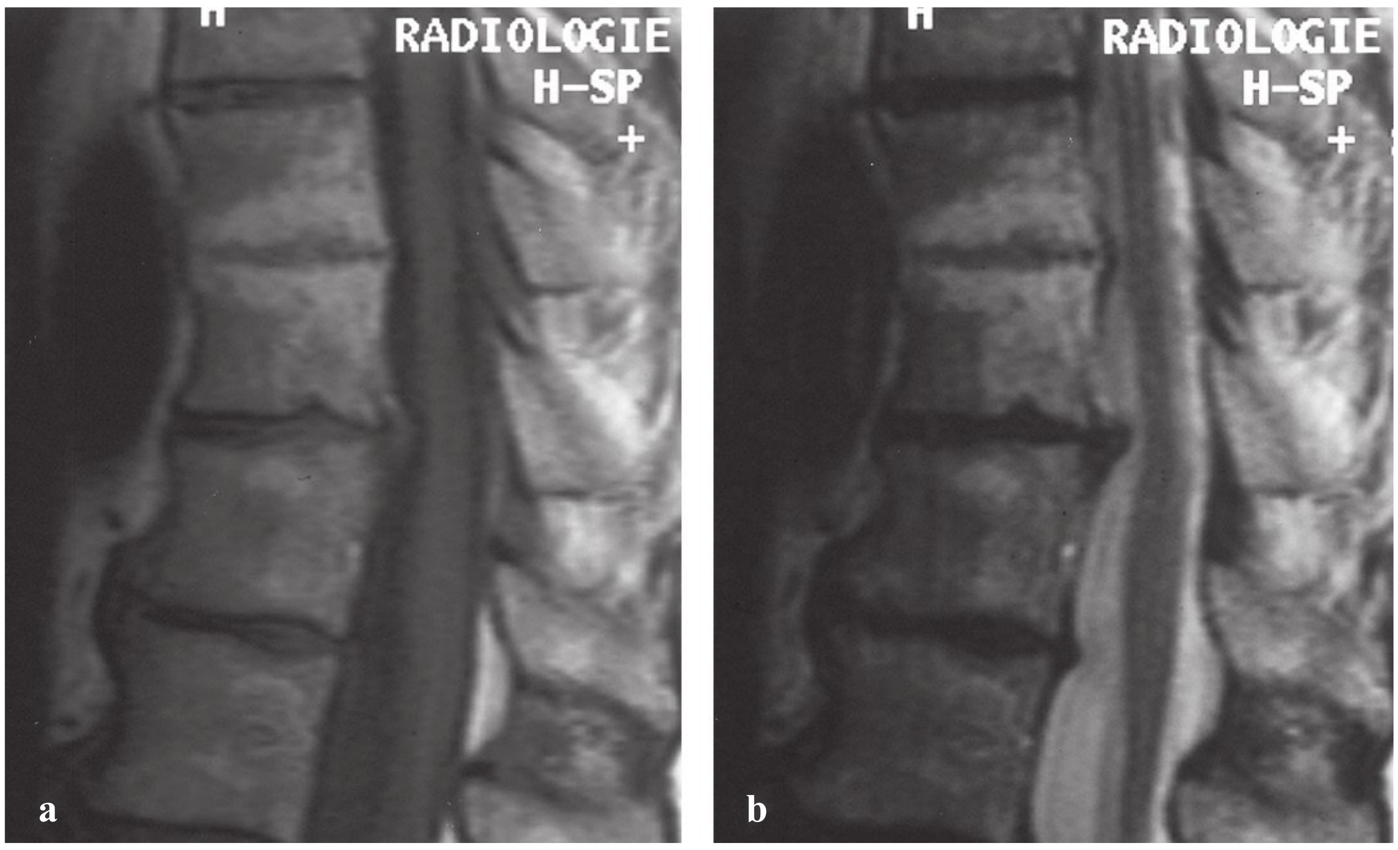

Fig. 4.10 **(a,b)** Sagittal magnetic resonance imaging demonstrating type 3 Modic changes. (Reproduced from Uhlenbrock D. MR Imaging of the Spine and Spinal Cord. Stuttgart, Germany: Georg Thieme Verlag; 2004: Fig. 4.33, with permission.)

4.2 Electrodiagnostic Tests

I. Electromyography (EMG) and nerve conduction studies (NCSs).
   A. EMG/NCS only evaluates the motor tracts of the nerve root.
      1. Radiculopathy may also involve motor, sensory, and autonomic fibers of the nerve root.
   B. Compound muscle action potential in peripheral nerves will show reduced amplitude in proportion to the amount of axonal degeneration that occurs after a compressive nerve root lesion.
   C. Compound muscle action potential in peripheral nerves is more marked when multiple roots are involved, as in lumbar spinal stenosis.
   D. Nerve conduction velocity or latency should *not* be affected by a focal proximal lesion, such as in radiculopathy.
   E. The gold standard for electrodiagnosis of radiculopathy is needle EMG.
      1. The earliest EMG finding in acute radiculopathy is a decrease in the number of motor unit potentials seen on recruitment.
      2. An increase in the number of polyphasic motor unit potentials may be seen early.
      3. Prolonged H reflex latency for C7 or S1 roots and reduced number of F waves in weak muscles may be observed after several days of radiculopathy.
      4. Spontaneous motor activity, fibrillations, and positive F waves are the hallmarks of acute radiculopathy.
      5. Large, long-duration, polyphasic potentials indicate reinnervation.
      6. As the radiculopathy resolves, polyphasic potentials tend to reduce in the number of phases, but the motor unit potentials may remain larger and be of longer duration than the normal motor unit potentials of uninvolved muscles.

F. Indications for EMG/NCS:
   1. Clinical findings suggest other neurological disorders, such as anterior horn cell disease, nerve entrapment syndrome, cervical stenosis, among others.
   2. Imaging studies and clinical findings do not correlate well in patients with suspected radiculopathy.
   3. In cases where neurological progression or deterioration must be documented.

II. Somatosensory evoked potentials (SSEPs) and motor evoked potentials.
   A. Assess sensory tracts from a peripheral nerve to the posterior column of the spinal cord **(Fig. 4.11).**
   B. Most commonly used as an intraoperative monitoring technique to protect the spinal cord during surgery.
   C. Dermatomal SSEPs can be used to monitor nerve root function during surgery.
   D. Motor evoked potentials assess the motor pathway in the spinal cord, particularly during anterior procedures of the spine that may jeopardize the anterior part of the spinal cord.

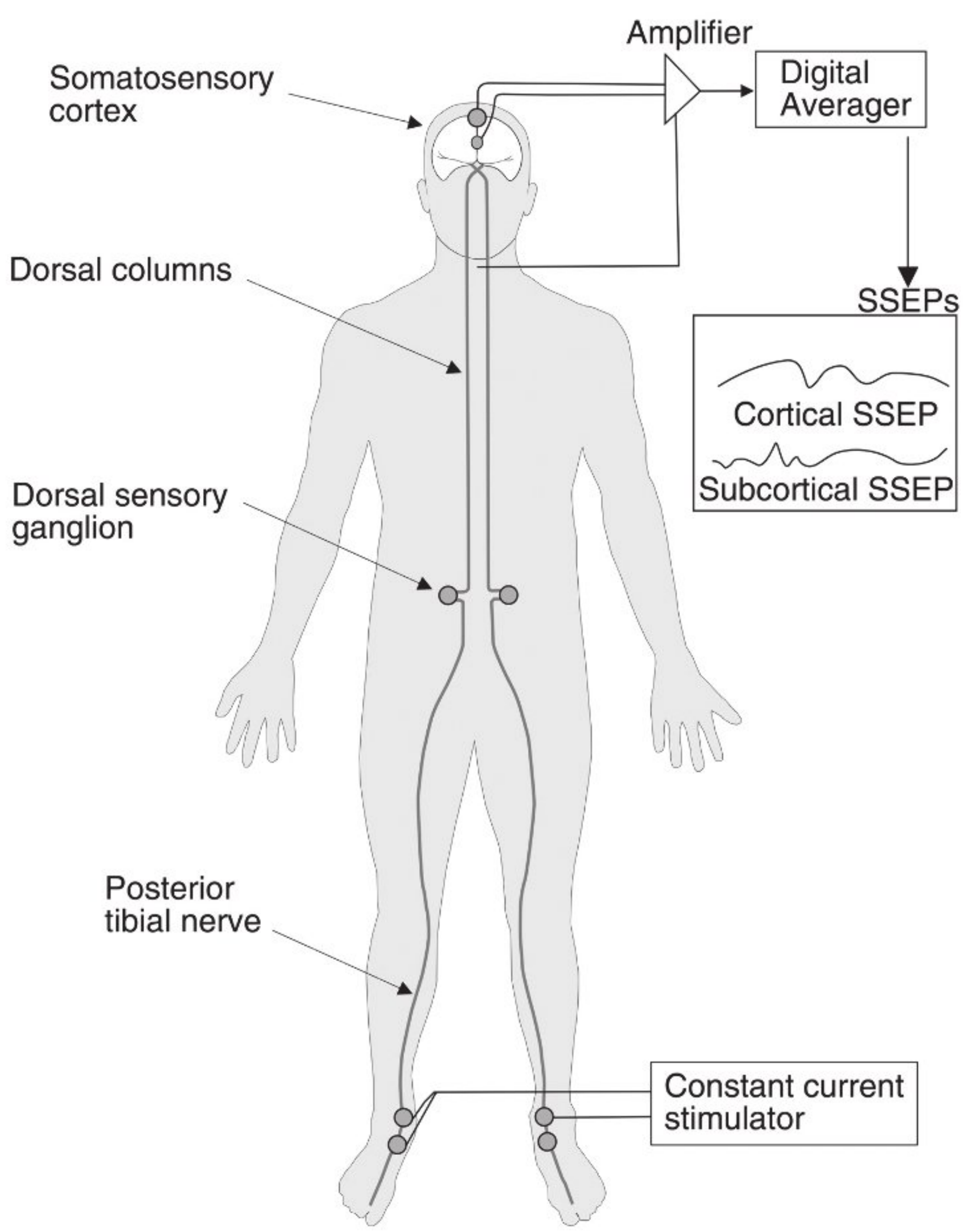

Fig. 4.11 Cortical somatosensory-evoked potential from the posterior columns. (Reproduced from Devlin VJ, Schwartz DM. Intraoperative neurophysiologic monitory during spinal surgery. J Am Acad Orthop Surg 2007;15(9):549–560, with permission.)

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5 Intraoperative Neuromonitoring

5.1 General Considerations

I. Introduction.
   A. Contemporary intraoperative neurophysiological monitoring (IONM) of the spinal tracts stems from advancements made in the 1970s.
   B. Prior to modern modalities, the Stagnara wake-up test was the gold standard for the assessment of global motor function.
      1. Characterized by awakening the patient to assess motor function.
      2. Patient was awakened after critical points during the procedure (e.g., implant placement, curve reduction).
         a. Limited applications outside of scoliosis correction.
      3. Significant limitations of the wake-up test.
         a. Poor patient cooperation.
         b. Lack of real-time monitoring.
         c. False-negatives.
         d. Venous air embolism.
         e. Inability to detect delayed neurological insults.
         f. Inability to take expeditious corrective measures.
   C. Somatosensory evoked potential (SSEP) monitoring was the first advancement in IONM.
      1. Sensory dorsal column–medial lemniscus pathway.
   D. Recent advances include the assessment of transcranial motor evoked potentials (tcMEPs) and electromyography (EMG).
      1. Enable real-time monitoring of the motor tracts and nerve roots and limit signal interference from anesthesia.
   E. Numerous variables confound IONM.
      1. Anesthesia (most common).
      2. Body temperature.
      3. Degree of neural development (age, neuromuscular comorbidities, delayed development).
      4. Mean arterial pressures.
      5. Medications.
      6. Length of procedure.
      7. Personnel expertise.

II. Somatosensory evoked potentials (SSEPs):
   A. Elicited by the stimulation of a peripheral nerve, typically either the posterior tibial, peroneal, ulnar, or median nerves.
   B. The signal traverses the dorsal column medial lemniscus pathway in the spinal cord to the brain.
   C. The ascending signal is captured by an amplifier placed on the head, which then records cortical SSEPs.

1. Subcortical responses can also be tracked by placing the amplifier electrodes on the anterior or posterior neck.
2. Subcortical SSEPs are thought to be less inhibited by the effects of anesthesia and the degree of neural development.

D. Amplitude reduction of > 50% or latency of more than 10% of baseline is cause for concern.

E. Significant limitations of SSEPs:
1. Not true real-time monitoring, because the potentials must summate when they are recorded.
2. Lack of motor track monitoring.
3. Significantly affected by neuroanesthesia.

III. Transcranial motor evoked potentials:

A. Record the descending motor corticospinal tracts.

B. Characterized by transcranial stimulation.

C. A recording electrode is placed in the subdural space of the spine, or a subdermal electrode can be used to record the peripheral musculature.
1. Descending stimulation can summate temporally or spatially to trigger skeletal muscle action, thereby producing compound muscle action potentials (CMAPs).
2. Epidural monitoring is characterized by monitoring the D-wave, which represents direct stimulation of the corticospinal neurons.

D. Myogenic MEPs (mMEPs):
1. Assess the nerve roots and peripheral nerves.

E. A loss of MEPs or a sudden decrease in 75% of amplitude is characteristic of neural insult.

IV. Electromyography:

A. Placement of concentric needle electrodes into the extremity musculature to record amplitude, frequency, duration, and shape of the motor unit action potential.
1. Assesses spinal nerve root insults due to stretch or direct injury.
2. Records changes in the pattern of motor unit action potentials as a result of nerve root injury/irritation.
3. Provides real-time information to the surgeon to reverse any noxious stimuli to the nerve roots.
   a. False negatives are often encountered if nerve root injury results from vascular injury or prolonged stretch injury, which may have a delayed presentation.

B. Triggered EMG (tEMG).
1. Used during implant placement with transpedicular screw fixation.
2. A current is applied to the pedicle screw head after placement, and the CMAPs of the corresponding nerve root musculature are recorded.
3. Assess the signal intensity required to depolarize the musculature:
   a. With pedicle wall breach, the stimulus magnitude is significantly lower than control values, because there is less impedance of current flow to the neural elements.
   b. Less than 10 milliamps may be indicative of a medial wall breach.
4. Continuous EMG is used while the surgeon is traversing the psoas muscle in an effort to prevent lumbosacral plexus injury.

V. Anesthetic effects:
  A. Need to strike a balance to provide adequate anesthesia while ensuring reliable and meaningful IONM.
  B. Inhalational agents, including sevoflurane, isoflurane, and desflurane, are often implicated for producing latency in SSEPs.
  C. Total intravenous anesthesia (TIVA) is often used in conjunction with IONM to prevent suppression of cortical responses.
  D. Neuromuscular blocking agents interfere with the acquisition and reliability of MEPs and EMG recordings.
    1. There is considerable controversy regarding the use of partial neuromuscular blocking agents in conjunction with IONM.

VI. Multimodal monitoring with SSEPs and MEPs is typically used at most centers to account for limitations of each modality.

VII. Corrective measures:
  A. If an intraoperative neuromonitoring alert arises, the first steps involve hemodynamic stabilization.
    1. Maintain MAPs at least around 60 mm Hg and temporarily raise pressures as an initial step.
    2. Check the patient's body temperature.
    3. Check for technical issues.
      a. Patient positioning.
      b. Monitoring equipment malfunction.
      c. Drug infusion dosing.
      d. Lines.
  B. Reverse previous surgical maneuver (implant placement, curve correction, rod placement, distraction).
  C. If none of the corrective measures resolves the issue, an intraoperative wake-up test should be considered.
  D. There is considerable controversy regarding whether the case should be discontinued if neuropotentials cannot be restored.

Suggested Reading

Clark AJ, Ziewacz JE, Safaee M, et al. Intraoperative neuromonitoring with MEPs and prediction of postoperative neurological deficits in patients undergoing surgery for cervical and cervicothoracic myelopathy. Neurosurg Focus 2013;35(1):E7

Fehlings MG, Brodke DS, Norvell DC, Dettori JR. The evidence for intraoperative neurophysiological monitoring in spine surgery: does it make a difference? Spine 2010; 35(9, Suppl):S37–S46

Michael KW, Boden SD. Intraoperative neuromonitoring in spine surgery. Contemp Spine Surg 2012:13

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6 Biomechanics of the Spine and Spinal Instrumentation

6.1 General Considerations

I. Introduction.
  A. Functional spinal unit.
    1. Intervertebral disk.
    2. Adjacent vertebral bodies.
    3. Facet joint complex.
  B. Spinal stability.
    1. Under physiological loading, there is neither abnormal strain nor excessive motion in the functional spinal unit.
    2. This stability is maintained by the bony and ligamentous components of the functional spinal unit, muscular tension, abdominal and thoracic pressures, and rib cage support.
  C. Sagittal balance.
    1. Defined and maintained by the cervical lordosis, thoracic kyphosis, lumbar lordosis, and pelvic tilt.
    2. On a standing lateral plain film radiograph, the weight-bearing axis or plumb line should cross C1, C7, T10, and S2.
    3. Modulation of back extension by the paraspinal muscles can help center the weight-bearing axis over the pelvis and feet.
    4. Additional maintenance of the sagittal balance is achieved by retroversion of the pelvis (pelvic tilt).

II. Kinematics.
  A. Cervical spine.
    1. Occipitoatlantal joint (occiput–C1).
      a. Thirteen degrees flexion/extension.
        (1) Head nod.
      b. Eight degrees lateral bending.
      c. Four degrees axial rotation.
      d. Coupled motion.
        (1) Occipitoatlantal extension with chin-out maneuver.
    2. Atlantoaxial joint (C1–C2) **(Fig. 6.1).**
      a. Approximately 45° of axial rotation.
      b. Ten degrees flexion/extension.
      c. No lateral flexion.
    3. Subaxial cervical spine.
      a. Flexion/extension.
        (1) Greater mobility in the sagittal plane due to the orientation of the facet joints (45° horizontal plane).
          (a) C2–C3 (8°).

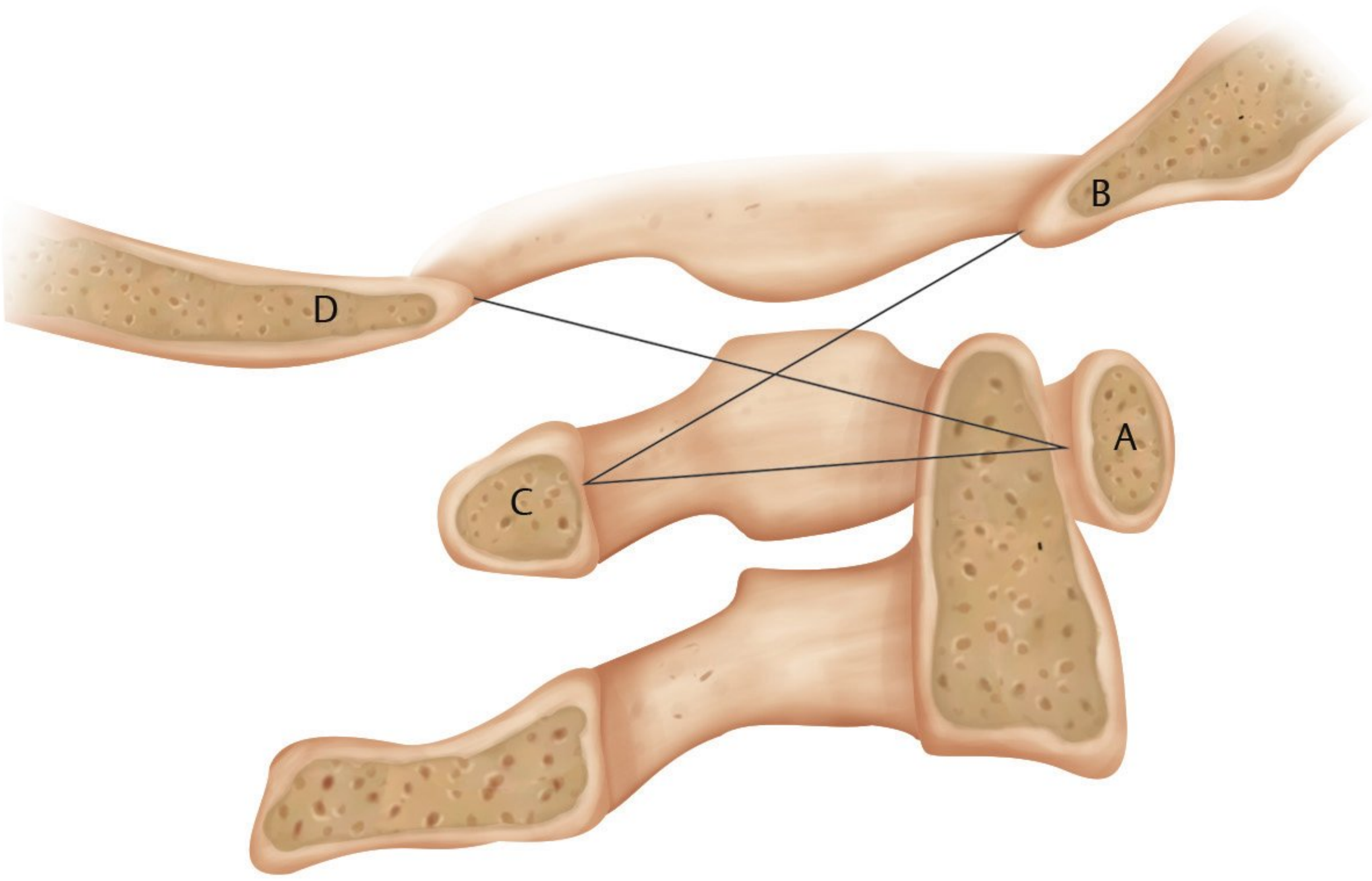


Fig. 6.1 Power's ratio (BC/DA) > 1 signifies anterior occipitoatlantal instability. The distance between the basion and the spinolaminar line of C1 is divided by the distance between the posterior margin of the foramen magnum (opisthion) and the posterior margin of the anterior arch of C1.

(b) C3–C4 (13°).
(c) C4–C5 (12°).
(d) C5–C6 (17°).
(e) C6–C7 (16°).
(f) C7–T1 (9°).

b. Lateral bending.
  (1) Sixty degrees coupled with rotation.
    (a) The spinous processes rotate toward the convexity.

c. Axial rotation.
  (1) Fifty percent of cervical rotation takes place in the subaxial cervical spine.

B. Thoracic spine.

1. The ribs and steep orientation of the facets limit range of motion (ROM).
  a. Flexion/extension.
    (1) Seventy-five degrees combined sagittal motion.
    (2) Flexion is greater than extension.
    (3) Flexion increases caudally.
  b. Axial rotation.
    (1) Seventy degrees axial rotation.
    (2) Rotation decreases caudally.
  c. Lateral bending.
    (1) Seventy degrees lateral bending.
2. More flexion/extension and lateral bending motion is present in the lower vertebral segments, but there is less rotation.

3. Some degree of rotation accompanies lateral bending.
   a. Spinous processes rotate toward the convexity in the upper thoracic region.
   b. In the middle to lower thoracic region, the direction of coupling is not consistent.

C. Lumbar spine.
1. Flexion/extension.
   a. Eighty–five degrees combined flexion/extension ROM.
   b. Flexion is greater than extension.
   c. Motion is greater caudally.
2. Lateral bending.
   a. Thirty degrees ROM.
3. Axial rotation.
   a. Sagittal orientation of the facets limits rotation.
   b. Rotation is least at L5–S1.

III. Biomechanics of spinal instability, orthosis, and instrumentation.

A. Occiput–cervical spine **(Table 6.1).**
1. C2 fractures.
   a. Odontoid fractures produce C1–C2 instability.
   b. C2 pedicle or the hangman’s fracture (traumatic spondylolisthesis of C2) **(Fig. 6.2)**:
      (1) Unstable in flexion.

B. Fixation of the upper cervical spine.
1. Posterior methods.
   a. C1 lateral mass screw/C2 pedicle screw **(Fig. 6.3** and **Fig. 6.4).**
      (1) Strongest biomechanical fixation.
2. Anterior odontoid screws **(Fig. 6.5).**
   a. Biomechanically stronger with two screws.
      (1) One screw may be adequate clinically.
3. C2 translaminar screw.
   a. Used if the posterior elements are intact.

C. Biomechanics of the lower cervical spine.
1. White and Panjabi checklist for clinical instability **(Table 6.2).**
   a. Anatomical components.
      (1) Anterior stability.
         (a) Annulus fibrosus.
         (b) Anterior longitudinal ligament.
         (c) Vertebral body.
      (2) Posterior stability.
         (a) Posterior longitudinal ligament.
         (b) Facet joint and capsules.
         (c) Lamina and interspinous ligaments.
2. Ligamentous disruption of > 3.5 mm or 11° indicates instability **(Fig. 6.6)**.
3. Bony disruption:
   a. Vertical compression and compressive flexion injuries.
      (1) The status of the posterior column influences the overall stability.

Table 6.1 Biomechanical measurements

Pathology	Measurement	Abnormal	Notes
Occiput–C1 instability	Distance from the tip of the dens to basion of the occiput	Greater than 1 mm translation on flexion–extension is abnormal	
	Powers ratio used to determine anterior atlanto-occipital dislocation	A ratio > 1 signifies anterior atlanto-occipital instability	Distance between the basion (midpoint of the anterior margin of the foramen magnum) and the spinolaminar line of C1 divided by the distance between the posterior margin of the foramen magnum (opisthion) and the posterior margin of the anterior arch of C1
Basilar invagination	McGregor's line	Greater than 4.5 mm odontoid projection above the foramen magnum	
	Ranawat's C1–C2 index	Less than 13 mm is abnormal	
	Redlund–Johnell O–C2 index	Less than 34 mm (men)	
		Less than 29 mm (women)	
Atlantoaxial (C1–C2) instability	Atlantodens interval (ADI)	ADI > 3 mm indicates rupture of the transverse ligament	Transverse ligament is essential for stability
		Greater than 5 mm of ADI indicates rupture of the transverse and alar ligament	
		Greater than 4.5 mm is abnormal in children	
	Space available for the cord (SAC)	SAC < 14 mm impinges on the spinal cord	
	Atlas fracture with > 6.9 mm lateral displacement indicates rupture of the transverse ligament		

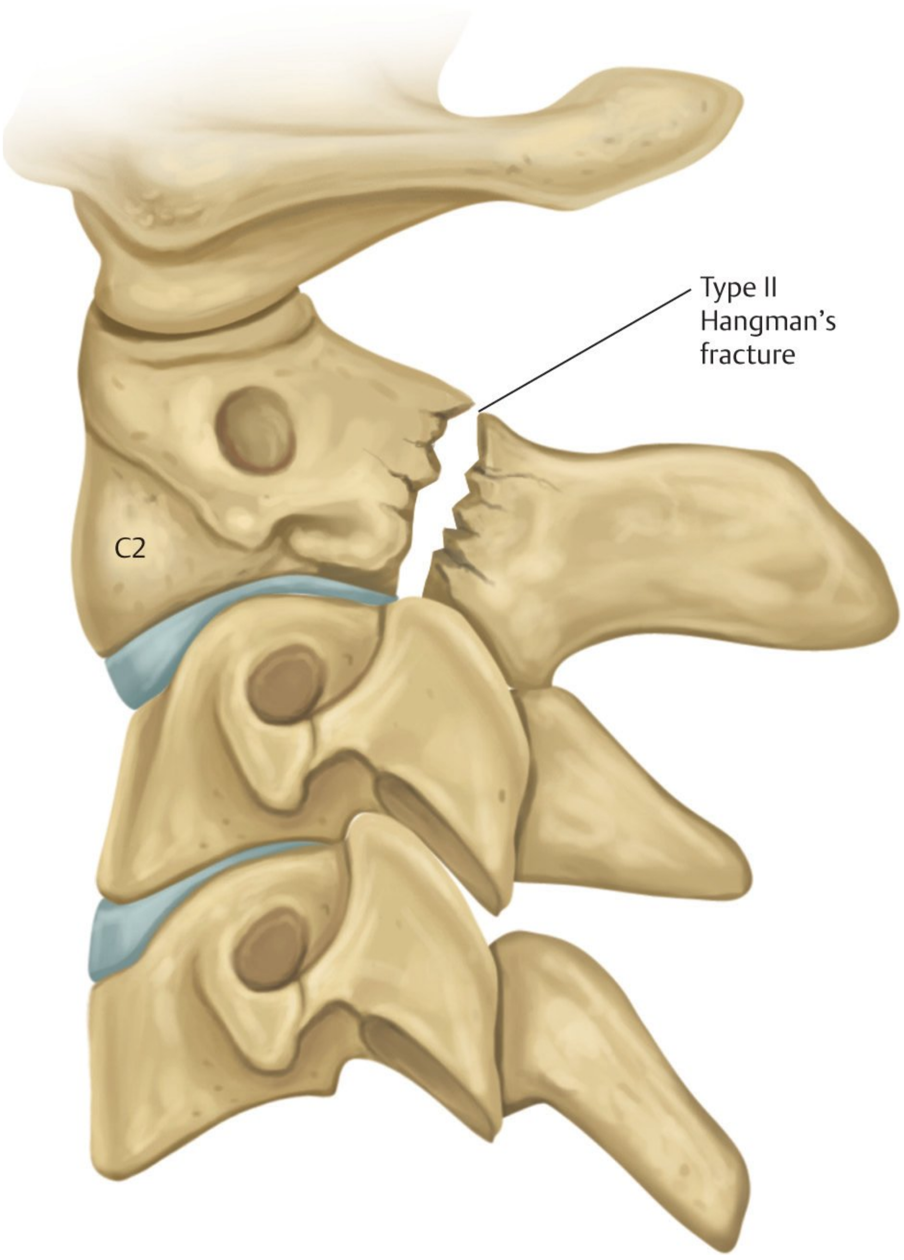


Fig. 6.2 Illustration of type II hangman's fracture.

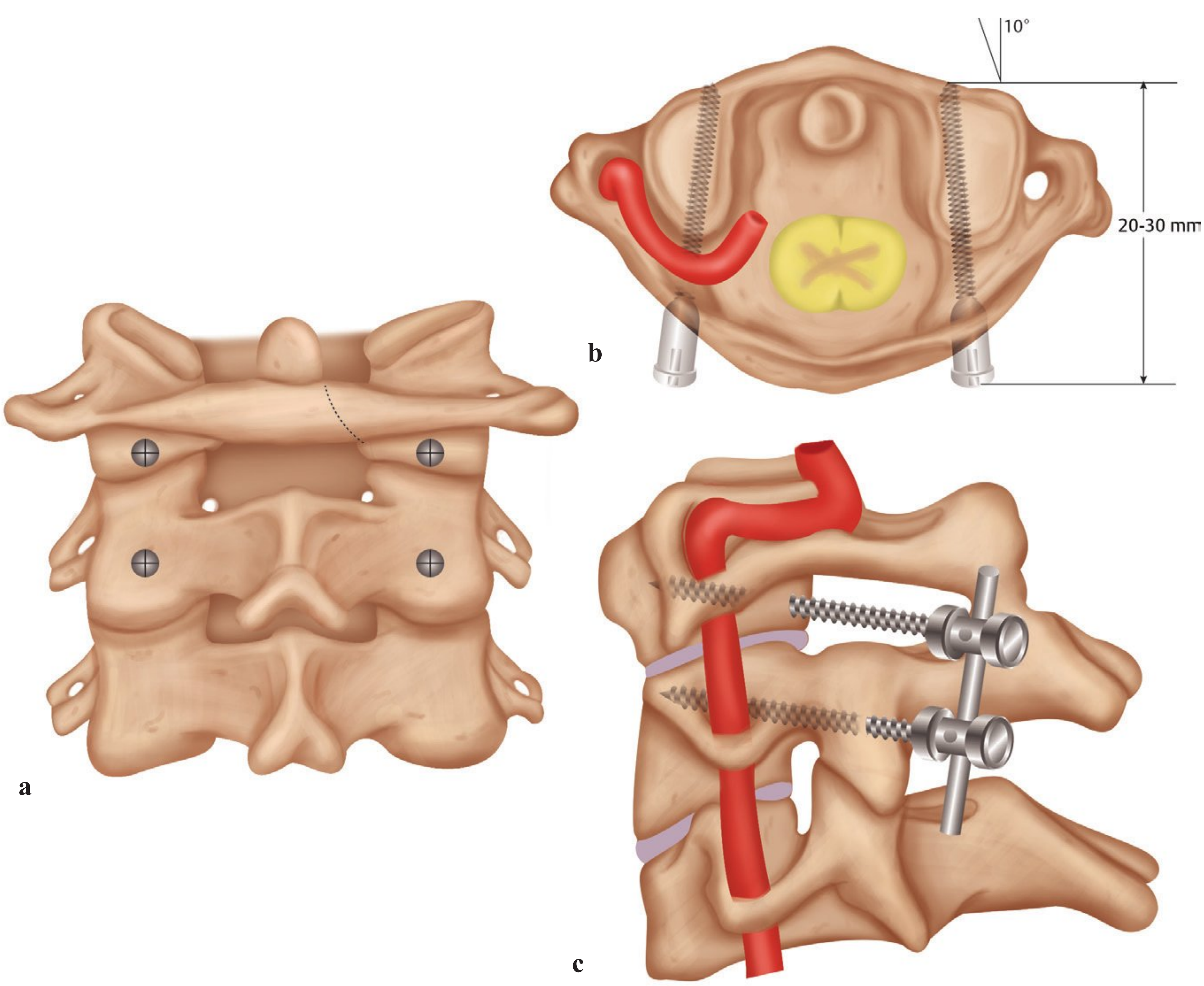


Fig. 6.3 **(a–c)** Starting points and trajectory for a C1 lateral mass screw (Harms technique) and a C2 pedicle screw.

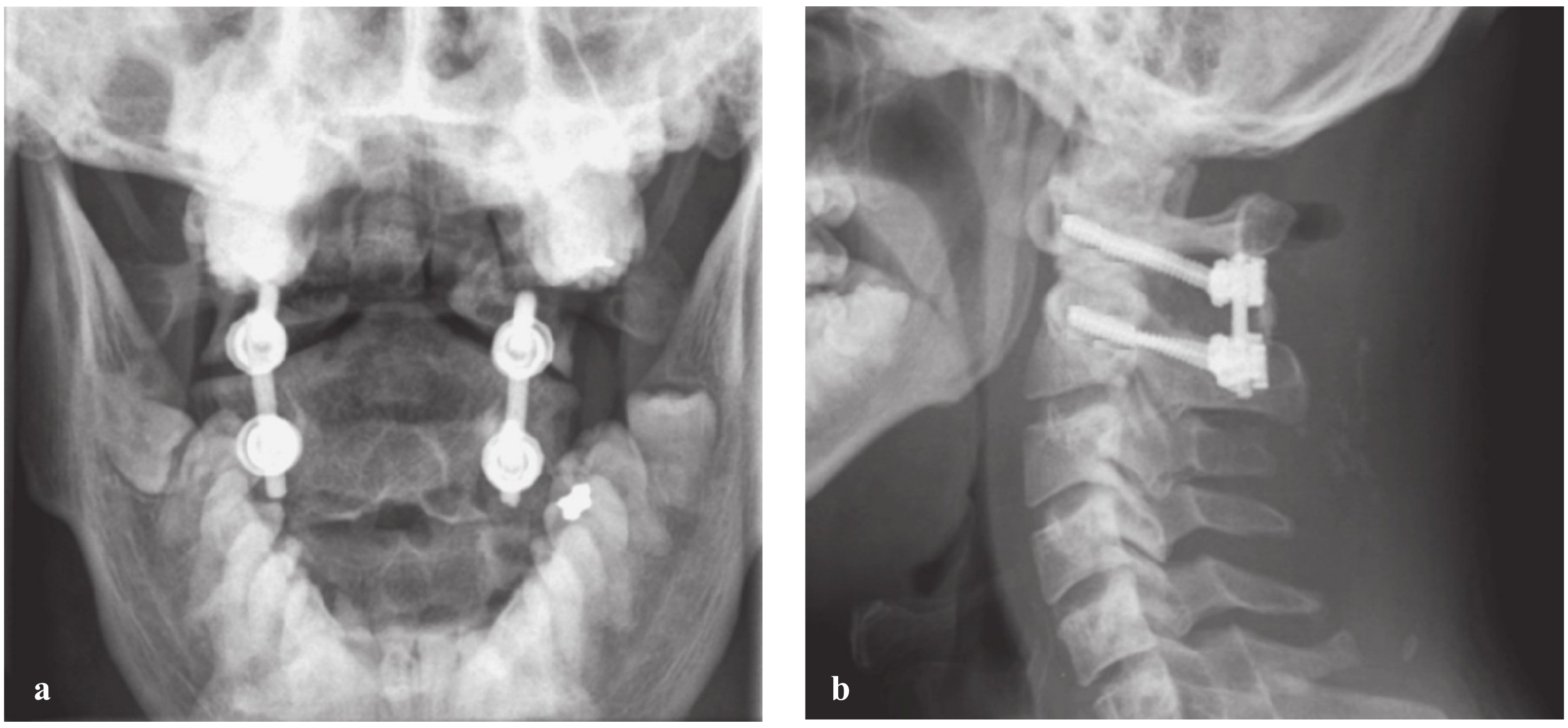


Fig. 6.4 **(a)** Anteroposterior and **(b)** lateral radiographs demonstrating a C1–C2 posterior cervical fusion accomplished via a C1 lateral mass screw and C2 pedicle screw.

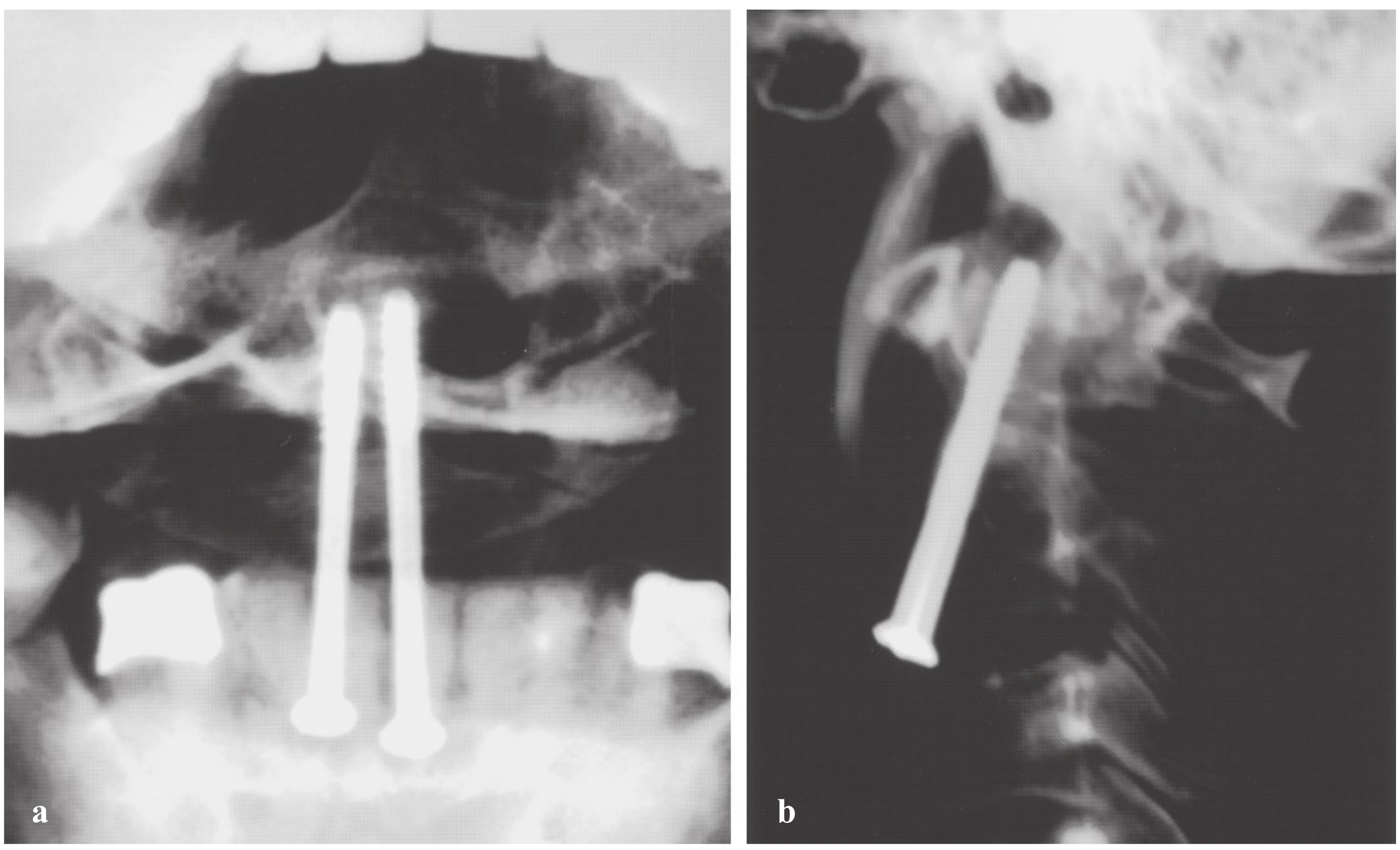


Fig. 6.5 **(a)** Open mouth and **(b)** lateral radiographs demonstrating anterior odontoid screw fixation for a C2 dens fracture.

Table 6.2 White and Panjabi checklist for clinical instability

1. A total of 5 points or more is considered unstable.
   a. Disruption of anterior elements: 2
   b. Disruption of posterior elements: 2
   c. Relative sagittal plane translation > 3.5 mm: 2
   d. Relative sagittal plane rotation > 11°: 2
   e. Positive stretch test: 2
   f. Cord damage: 2
   g. Root damage: 1
   h. Abnormal disk narrowing: 1
   I. Dangerous loading anticipated: 1

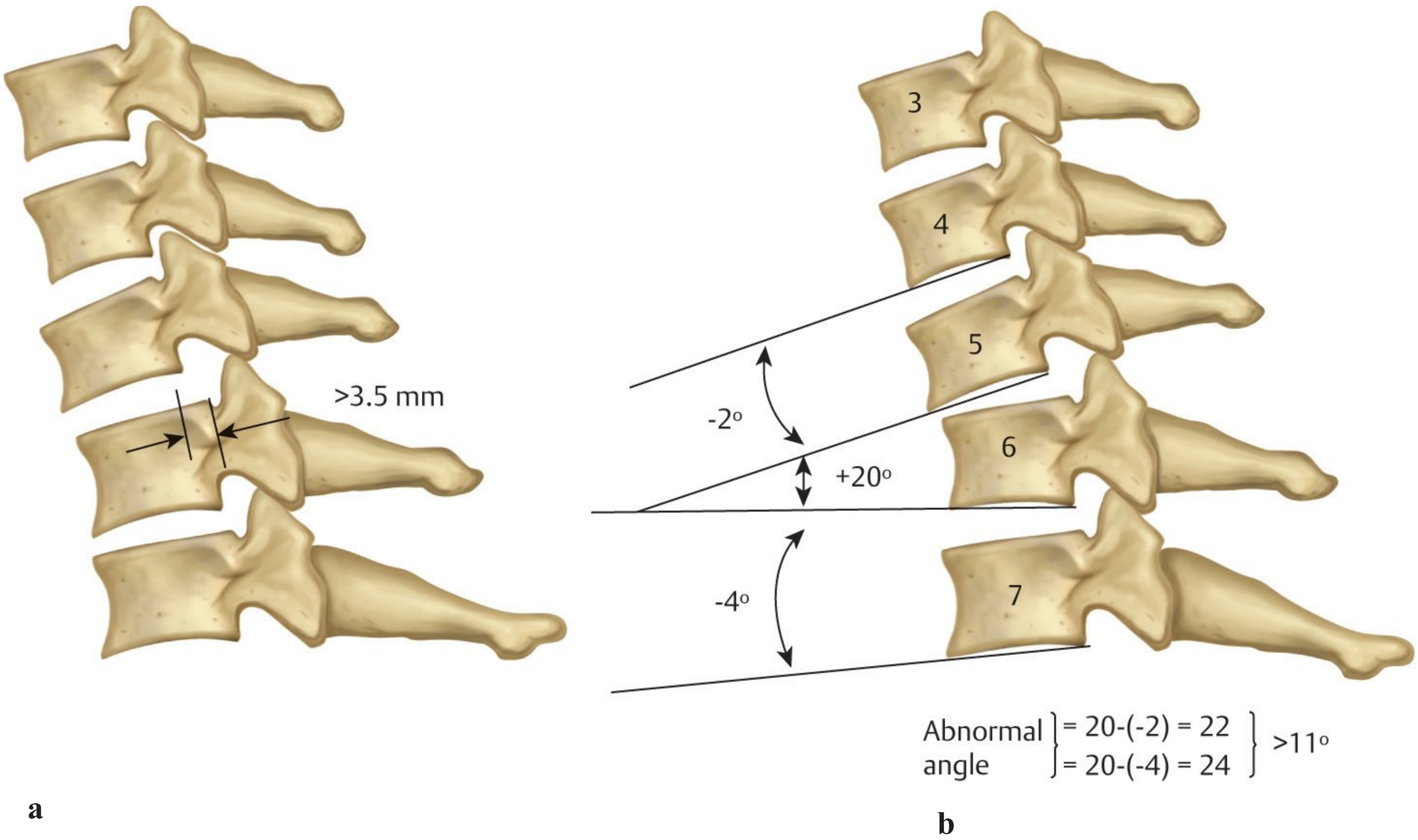


Fig. 6.6 **(a,b)** Ligamentous disruption of > 3.5 mm or 11° indicative of instability.

(a) Laminectomy or facetectomy.
  i. Laminectomy.
    • Eighteen percent loss of stability.
    • Laminectomy at C2 or C7 causes greater instability.
  ii. Laminectomy plus facetectomy.
    • Sixty percent loss of stability.
    • Partial bilateral facetectomy (> 50%) causes instability.

D. Cervical fusion.
  1. Anterior cervical fusion.
    a. Disruption of all anterior ligaments reduces strength by 52%
      (1) Anterior interbody fusion restores stability to 100% of normal in flexion.
      (2) Fifty-five percent restoration in extension (Smith–Robinson–type graft).
      (3) Bone mineral density affects the compressive strength of the graft.
      (4) Anterior interbody fusion plus plating adds strength in extension.
  2. Posterior cervical instrumentation.
    a. Interspinous wiring.
      (1) Thirty-three percent of normal stability in flexion.
        (a) Strength varies with different techniques.
    b. Posterior lateral mass screw–rod **(Fig. 6.7** and **Fig. 6.8).**
      (1) Strongest in both flexion (92%) and extension (60%).
    c. Pedicle screw.
      (1) Greatest level of rigidity in all planes of motion.
      (2) High incidence of medial wall violation.
      (3) Done with either fluoroscopic imaging or laminoforaminotomies or both.

E. Cervical orthosis **(Table 6.3) (Fig. 6.9).**

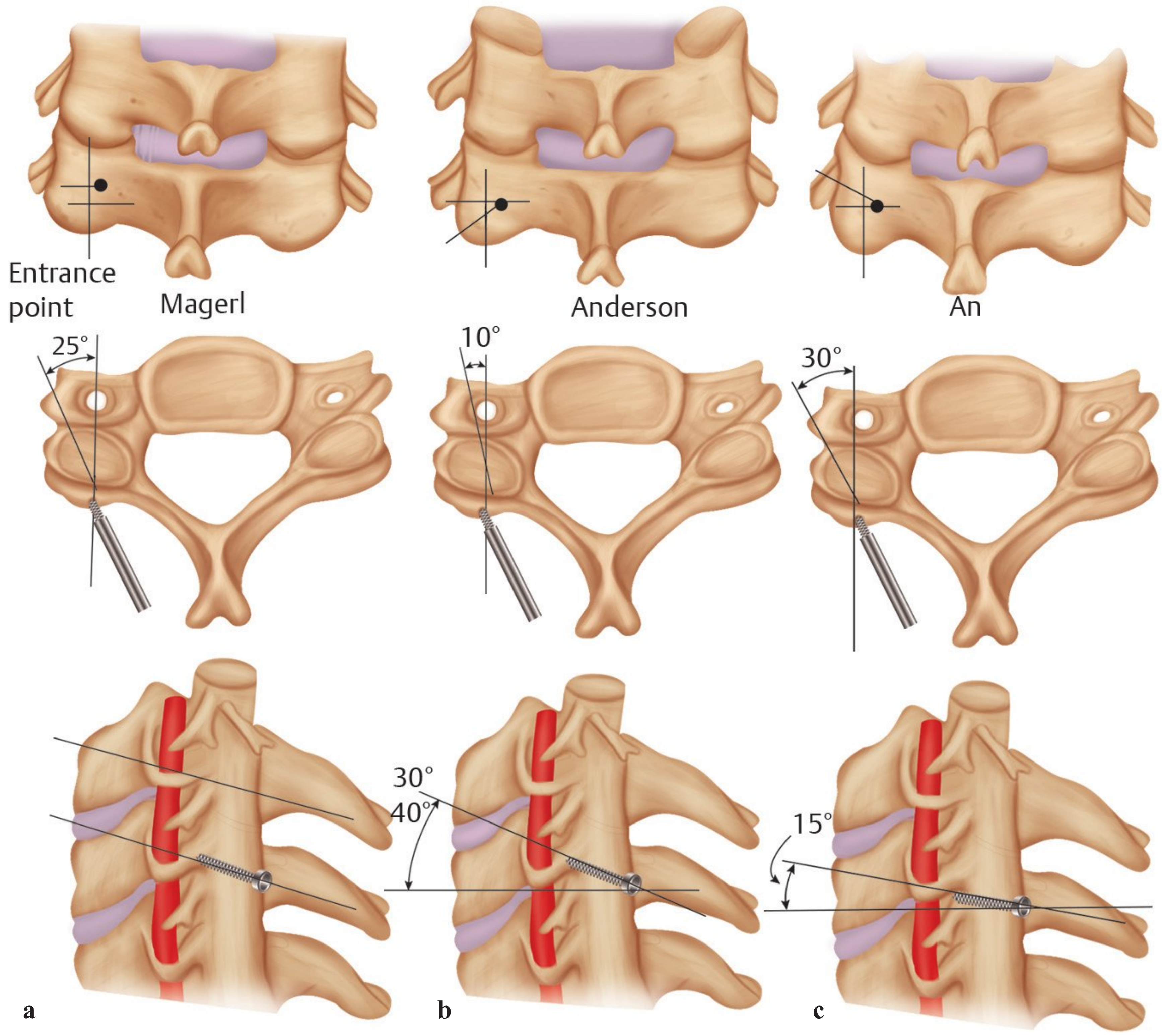


Fig. 6.7 Comparison schematic of **(a)** Magerl, **(b)** Anderson, and **(c)** An methods of lateral mass screw orientation.

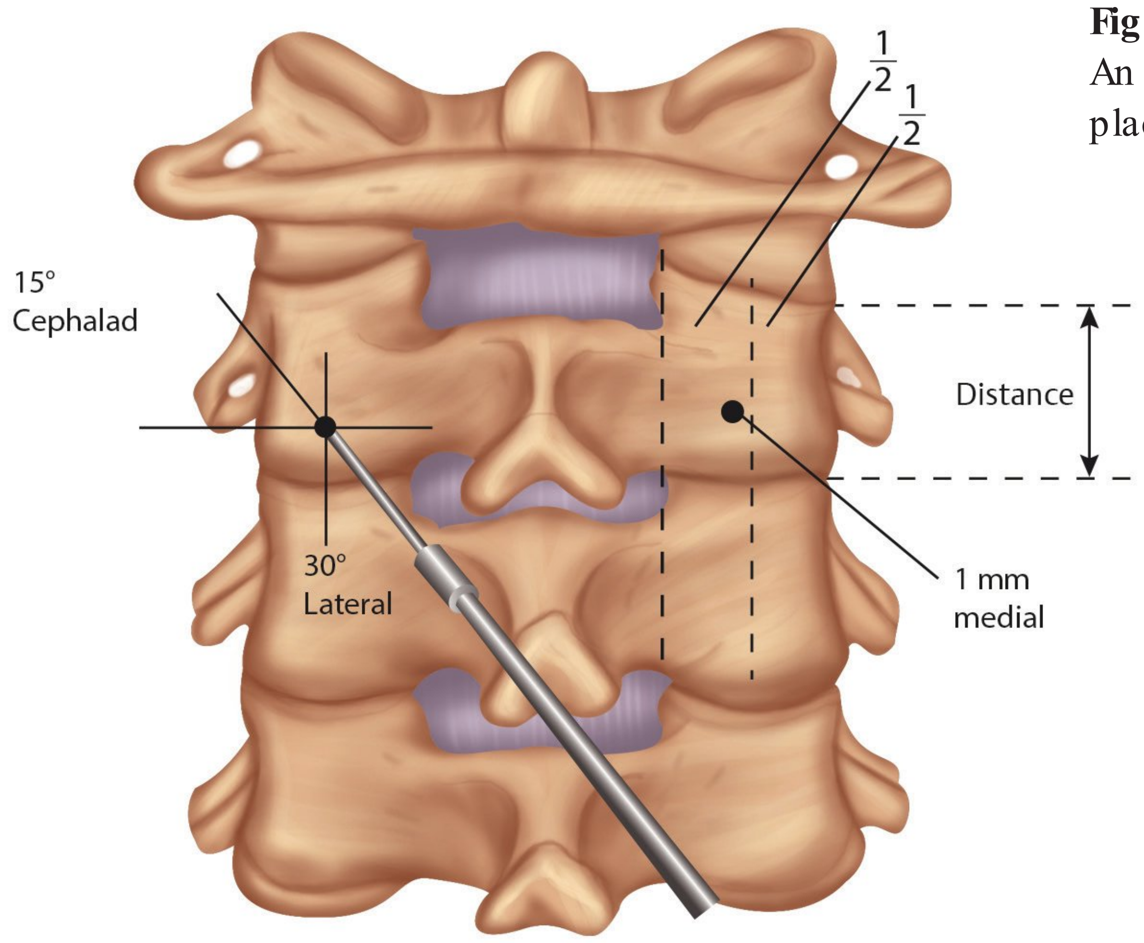


Fig. 6.8 Close-up diagram of the An technique of lateral mass screw placement.

Table 6.3 Cervical orthosis

Type	Notes
Soft collar	– Used only for comfort – Does not maintain stability
Philadelphia (Miami J) collars **(Fig. 6.9)**	– Thirty percent of normal flexion/extension allowed – Ineffective in controlling rotation and lateral bending
Four-poster-type orthosis	– Good for controlling flexion/extension at the midcervical level (20% of normal motion is allowed)
Cervicothoracic-style orthosis Sterno-occipital-mandibular immobilized (SOMI) brace Rigid cervicothoracic braces (Yale type)	– Good for controlling upper cervical spine flexion (C2–C5) – Does not restrict extension effectively – Good for controlling flexion/extension – Controls rotation slightly – Controls bending by only 50%
Halo devices	– Best for restriction of all planes of motion, particularly for the upper cervical spine – Cannot maintain distractive force **(Fig. 6.10)**

Fig. 6.9 Immobilization of the cervical spine.

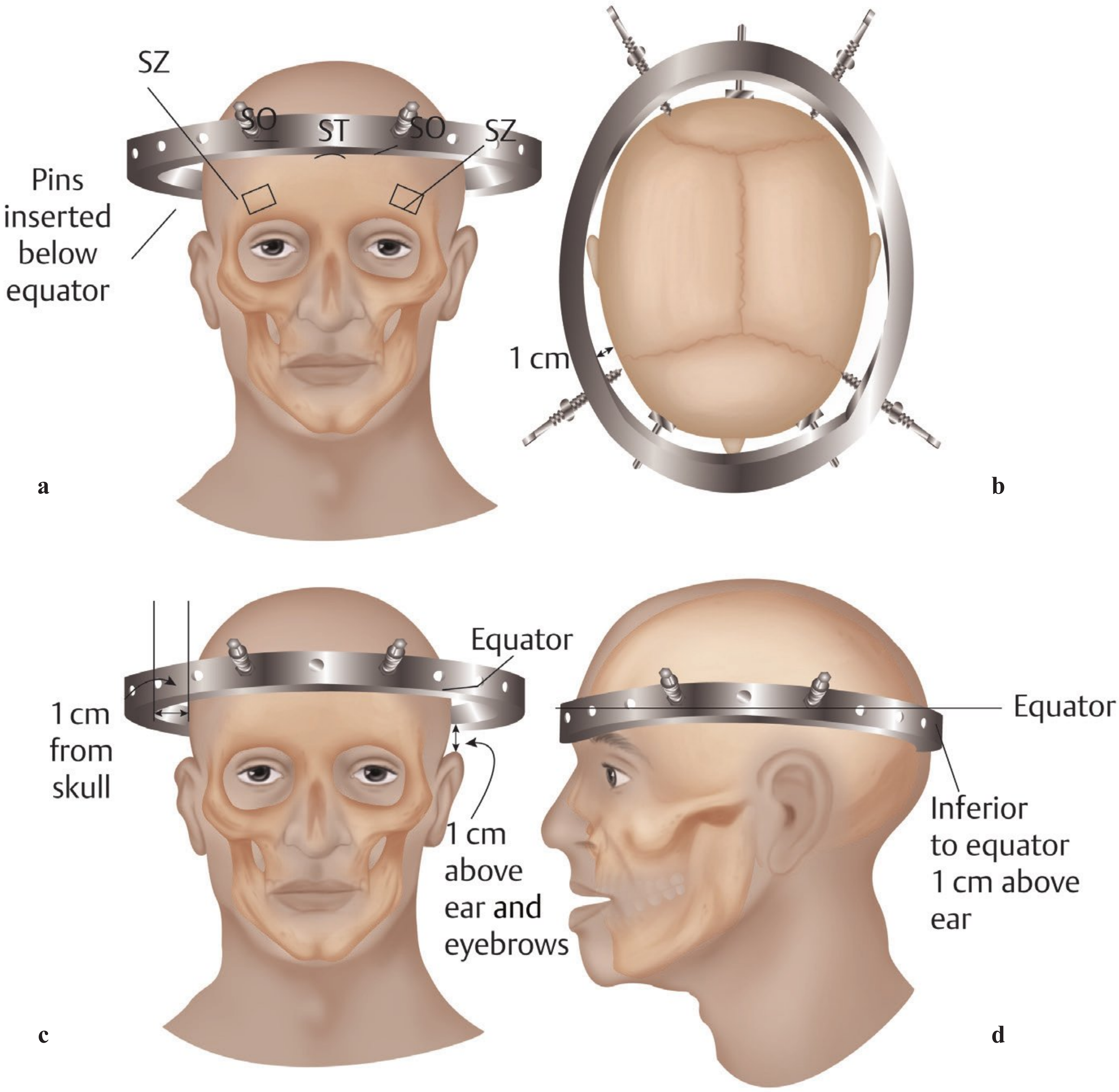


Fig. 6.10 (a–d) Halo application. The safe zone is shown for anterior halo pin placement. Anterior pins should be placed below the equator of the skull, lateral to the supraorbital nerve. The patient's eyes should be closed during halo placement to allow the patient to blink normally once the halo is placed.

F. Thoracic and thoracolumbar spine instrumentation.
   1. Supporting structures:
      a. Anterior.
         (1) Anterior and posterior longitudinal ligaments.
         (2) Intervertebral disk.
         (3) Vertebral body.
      b. Posterior.
         (1) Ligamentum flavum.
         (2) Facet joints.
         (3) Pedicle.
         (4) Costovertebral–transverse complex.

2. The thoracic spine is mechanically stiffer and less mobile than the lumbar spine.
   a. Stress concentration due to change in stiffness at the thoracolumbar junction.
3. Thoracic or thoracolumbar trauma:
   a. Thoracolumbar injury classification and severity (TLICS) score **(Table 6.4).**
      (1) Classification system based on injury morphology, integrity of the diskoligamentous complex, and neurological status.
      (2) Operative management is warranted if the TLICS score is ≥ 5.
         (a) A score of 4 can be managed with nonoperative or operative management (dealer's choice).
         (b) A score < 4 should be treated nonoperatively.
   b. Spine stability.
      (1) Three-column classification of Denis **(Fig. 6.11) (Table 6.5).**
         (a) If two or more columns are disrupted, the spine is considered to be unstable.
         (b) If the middle column is disrupted, the spine is regarded as unstable.
            i. Middle-column disruption is less important above T8 because of the stability provided by the rib cage.
   c. Compression fractures.
      (1) Anterior column failure.

Table 6.4 Thoracolumbar injury classification and severity (TLICS) classification

	Score
Morphology	
Compression fracture	1
Burst fracture	2
Translational/rotational fracture	3
Distraction	4
Neurological involvement	
Intact	0
Nerve root involvement	
Cord, conus medullaris compromise	2
Incomplete	3
Complete	2
Cauda equina	4
Posterior ligamentous complex	
Intact	0
Suspected injury	2
Injured	3

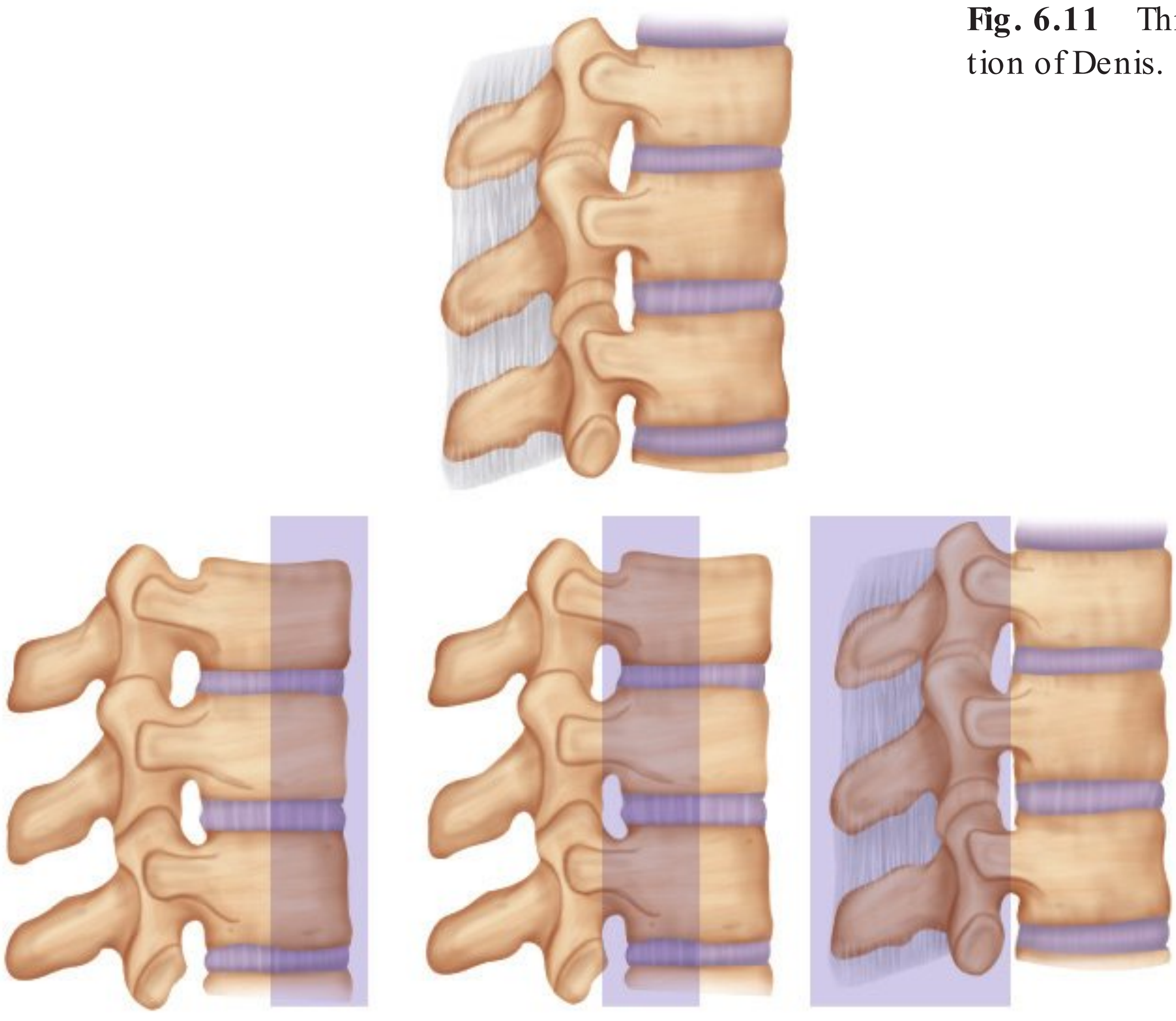


Fig. 6.11 Three-column classification of Denis.

Table 6.5 Denis classification

Anterior column	Anterior longitudinal ligament Anterior annulus fibrosis Anterior half of the vertebral body
Middle column	Posterior longitudinal ligament Posterior annulus fibrosis Posterior half of the vertebral body
Posterior column	Pedicle Facet joints Lamina Spinous processes Interspinous and supraspinous ligaments

(2) End plates are weaker than the intervertebral disks.
   (a) Displacement of the nucleus pulposus into the vertebral body may occur.
(3) Bony fractures are more common in older osteoporotic patients.

d. Burst fractures.
   (1) Anterior and middle column failure.
   (2) Neurological injuries are common secondary to retropulsion of the middle column.

e. Fracture/dislocation.
   (1) Three-column failure.
   (2) Shear/translational, flexion/distraction, or flexion-rotation injuries.
   (3) Posterior stabilization is required.

4. Instrumentation for thoracolumbar fractures:
   a. Purpose:
      (1) Early mobilization.
      (2) Prevent late deformity and pain.
      (3) Indirect decompression by distraction and extension in traumatic injuries.
      (4) Temporary stabilization until fusion matures increases healing rates.
   b. Transpedicular instrumentation **(Fig. 6.12)**:
      (1) Provides the stiffest construct with the shortest segment fusion.
      (2) The gold standard for thoracolumbar fracture management.
   c. Anterior fusion:
      (1) Primary treatment in fractures with neurological deficit.
      (2) Adjunct treatment after posterior instrumentation.
      (3) Anterior plate construct is equal to a construct with an anterior strut graft plus posterior transpedicular instrumentation.
   d. Percutaneous fracture fixation:
      (1) Uses minimally invasive techniques in an effort to lessen intraoperative blood loss, operative time, postoperative pain, and risk of infection, and to lessen the effects on posterior paraspinal musculature and stability
      (2) These benefits are paramount for polytrauma patients, who carry the greatest risk for complications.
      (3) One screw may be adequate clinically.

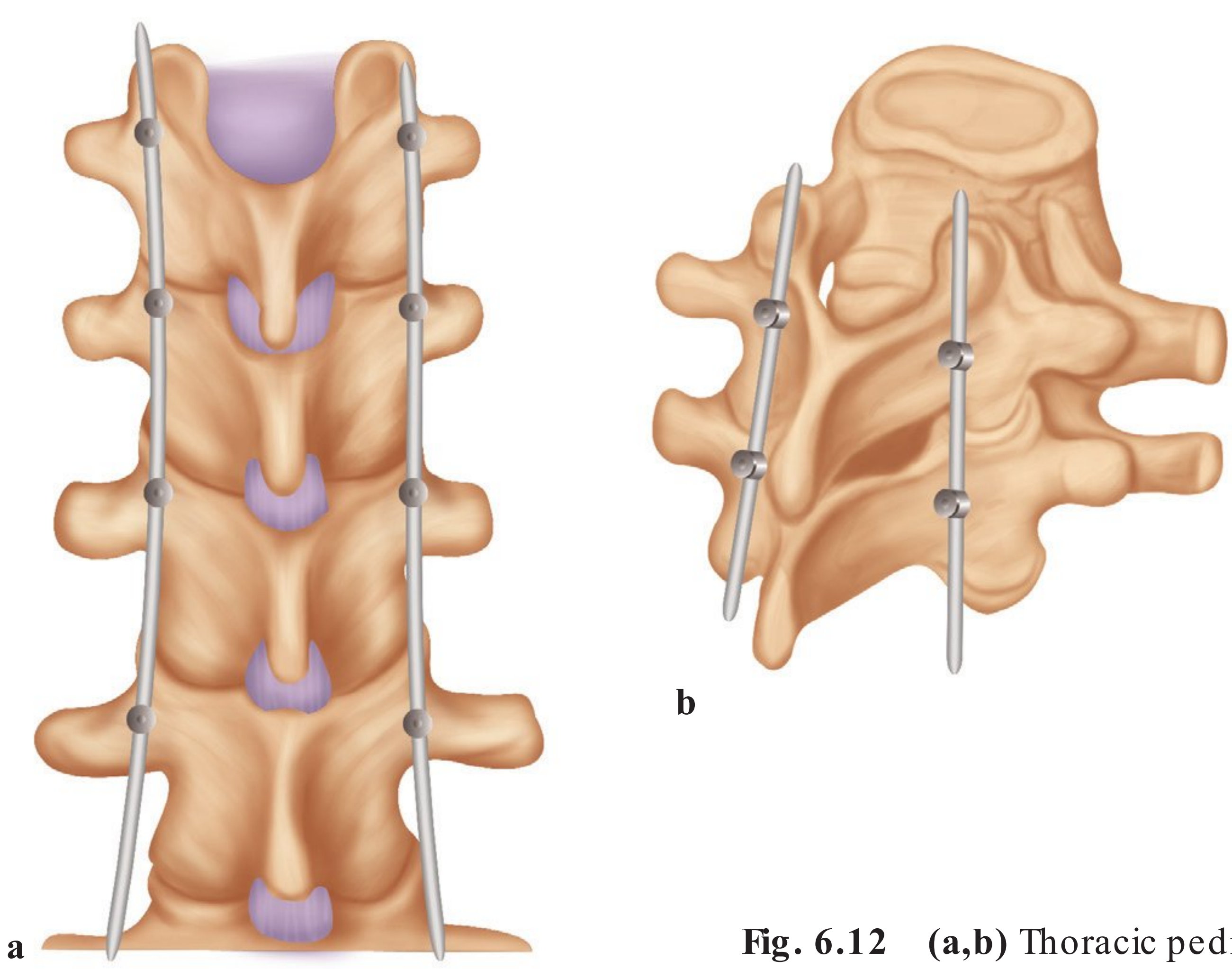


Fig. 6.12 **(a,b)** Thoracic pedicle screw starting points.

e. Some evidence suggests that thoracolumbar burst fractures may not require fusion.
   (1) Surgical management of thoracolumbar fractures involves the restoration of vertebral height and avoidance of kyphosis.
   (2) Often supplemented with transpedicular screw fixation for rigid stabilization.
   (3) In addition, fusion enables early rehabilitation and ambulation.
   (4) Recent, albeit weak, evidence suggests that posterior instrumentation alone provides comparable outcomes to instrumentation and fusion for thoracolumbar fractures.

5. Spinal deformities in the thoracolumbar spine:
   a. Scoliosis.
      (1) Lateral flexion with rotation of the spinous process toward the concavity of the spine.
      (2) Hypokyphosis and posterior wedging of the vertebral body are frequently seen **(Fig. 6.13).**
      (3) Correction.
         (a) Thoracic curve: distraction on the concave side corrects coronal alignment and produces thoracic kyphosis, which is generally desired.
         (b) Lumbar curve: compression on the convex side corrects coronal alignment and restores lumbar lordosis.

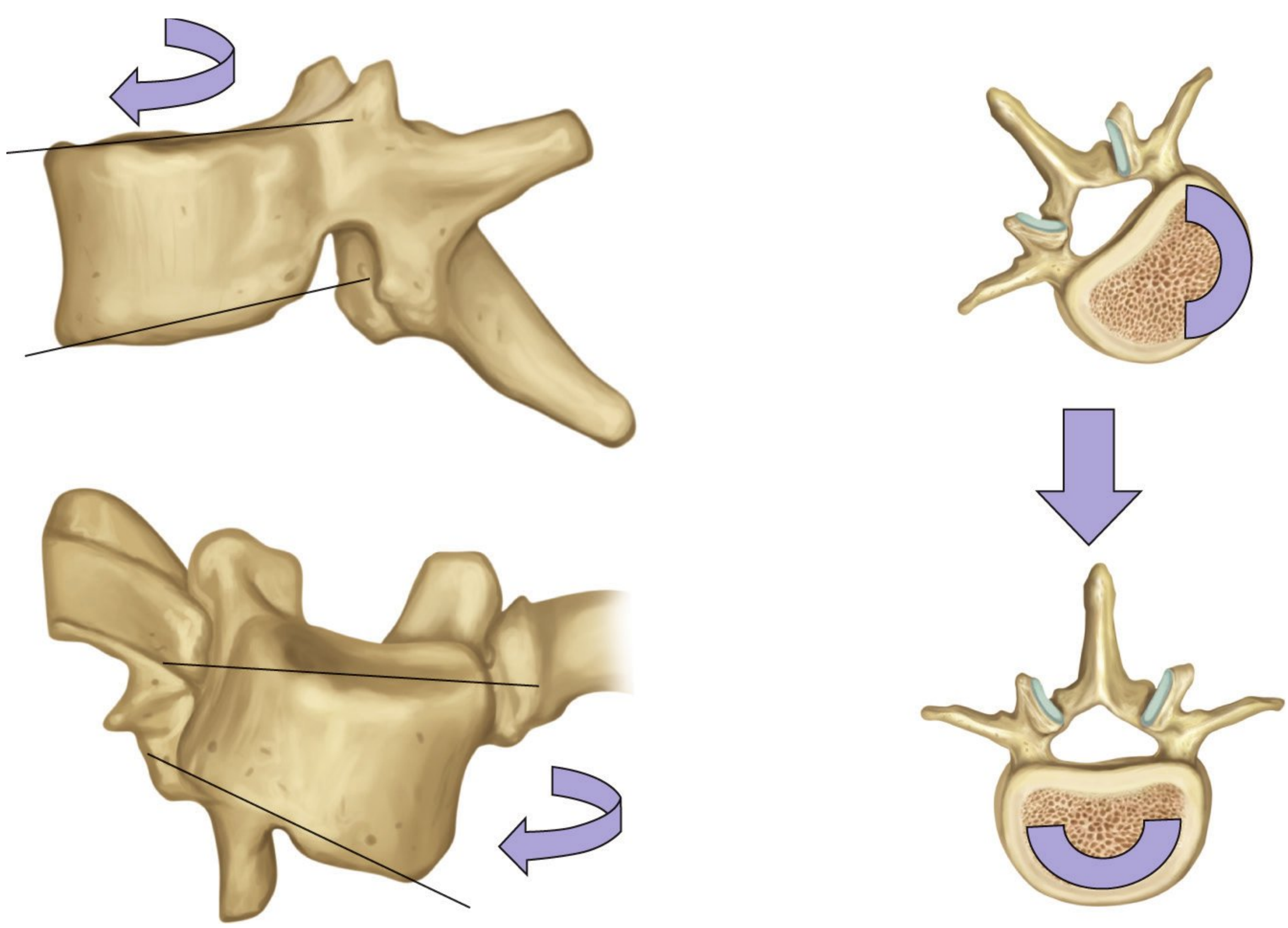
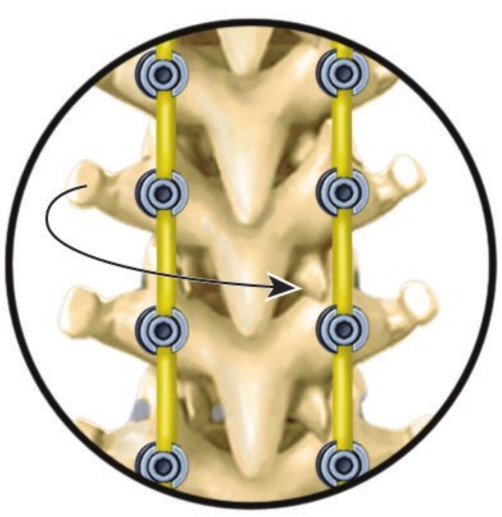


Fig. 6.13 Hypokyphosis and posterior wedging of the vertebral body.

(c) Bending and translation:
   i. Cantilever bending and segmental fixation correct the deformity in the coronal and sagittal plane.
   ii. Rotation–derotation also corrects the deformity in coronal and sagittal planes by shifting the regions of the spine en bloc.

b. Kyphosis.
   (1) The anterior column fails with compression.
   (2) The posterior column fails with tension.
   (3) Deformity increases the moment arm, further increasing deformity.
   (4) Eccentric loading affects cartilaginous growth.
      (a) Compression decreases growth anteriorly.
      (b) Tension increases growth posteriorly.
   (5) Various instrumentations are used to correct kyphotic deformities.
      (a) Posterior compression rods can correct mild and flexible curves **(Fig. 6.14)**.
      (b) Greater curves should be approached by combined anterior and posterior fusion and instrumentation.
         i. Posterior instrumentation is applied with cantilever bending and compression forces.

Fig. 6.14 Posterior rod instrumentation.



G. Lumbar and lumbosacral spine.
  1. Stability **(Fig. 6.15)**:
    a. Anterior stability:
      (1) Anterior longitudinal ligament.
      (2) Vertebral body.
      (3) Annulus fibrosus.
    b. Posterior stability:
      (1) Facet joint.
    c. The role of the muscles, including the erector spinae, abdominal muscles, and psoas, is important in overall stability.

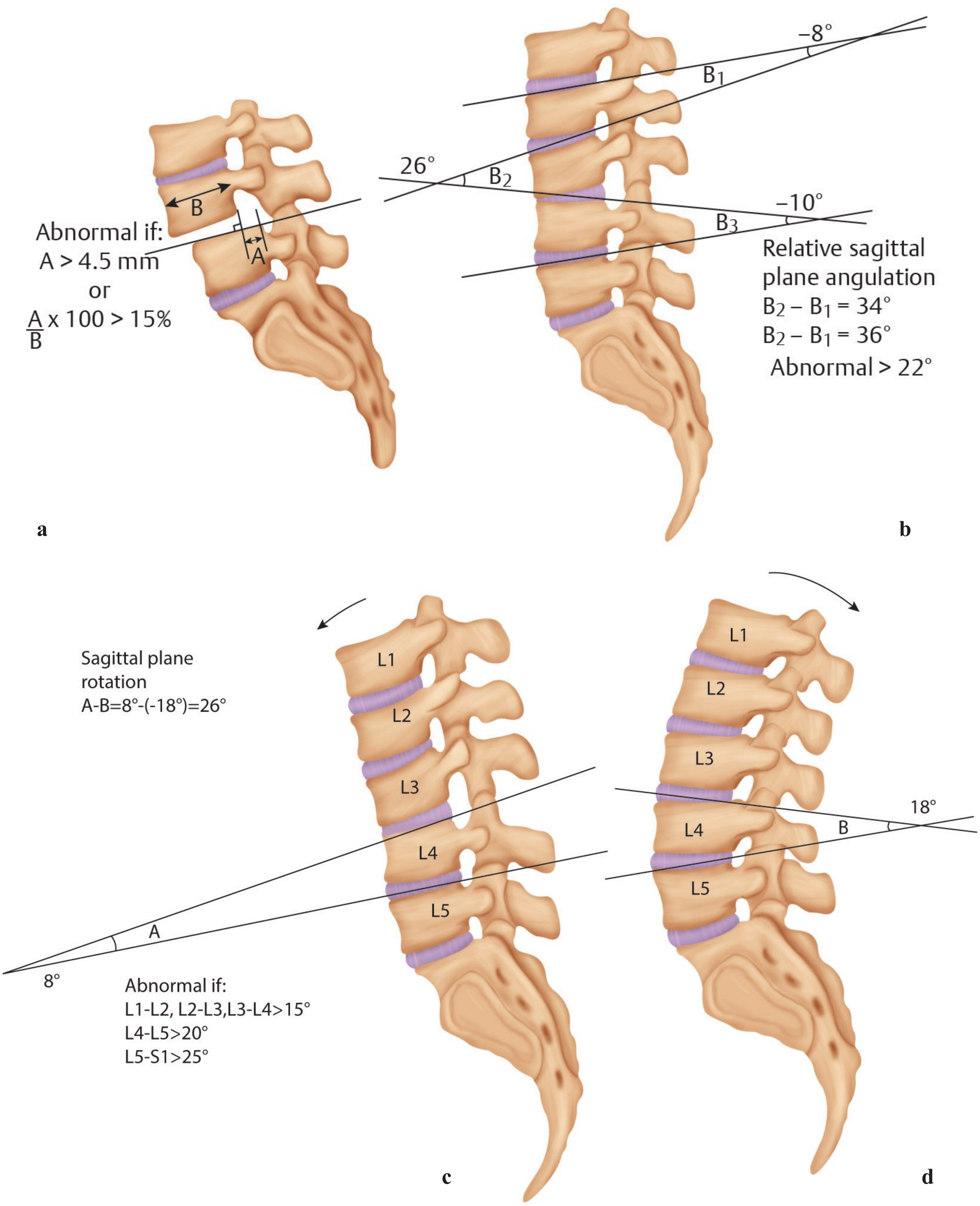


Fig. 6.15 **(a–d)** White and Panjabi's criteria for lumbar segmental instability.

2. Lower lumbar and lumbosacral spine.
   a. Degenerative disk and facet diseases.
      (1) Intervertebral disk.
         (a) Annulus fibrosus.
            i. Collagen fibers arranged in an oblique direction.
            ii. Provides axial loading stability and 40 to 50%torsional stability.
         (b) Nucleus pulposus.
            i. The gel-like core acts as a ball bearing, changing the center of rotation.
      (2) Intradiskal pressure.
         (a) The load on the disk is about twice the body weight when sitting.
         (b) Thirty percent lower disk pressure when standing.
         (c) Fifty percent lower disk pressure when lying on the side.
         (d) Eighty to 90%lower disk pressure when lying supine compared with sitting.
      (3) Disk degeneration.
         (a) Shifts the instant center of rotation posteriorly.
         (b) Increases stress to the facet joint.
         (c) Disk degeneration affects the motion of the functional spinal unit.
            i. Early disk degeneration with radial tears of the annulus fibrosus decreases stiffness in flexion, lateral bending, and rotation.
            ii. Advanced disk degeneration with loss of disk height and osteophytes increases the stiffness.
         (d) Pfirrmann classification of disk degeneration **(Fig. 6.16)**:
            i. Based on T2-weighted MRI findings.
            ii. Provides a standard nomenclature to classify disk degeneration.
      (4) Facet joints.
         (a) Provide torsional stability.
         (b) Support < 20%of load with weight bearing.
   b. Spondylolisthesis.
      (1) Abrupt change in stiffness across the lumbosacral junction.
      (2) The pars interarticularis is strong but susceptible to fatigue fractures, especially with extension injuries.
      (3) Shear stress at the pars interarticularis:
         (a) Physiological flexion contracture of the hip and secondary hyperlordosis create a pincerlike effect from the superior articular process of S1 and the inferior articular process of L4.

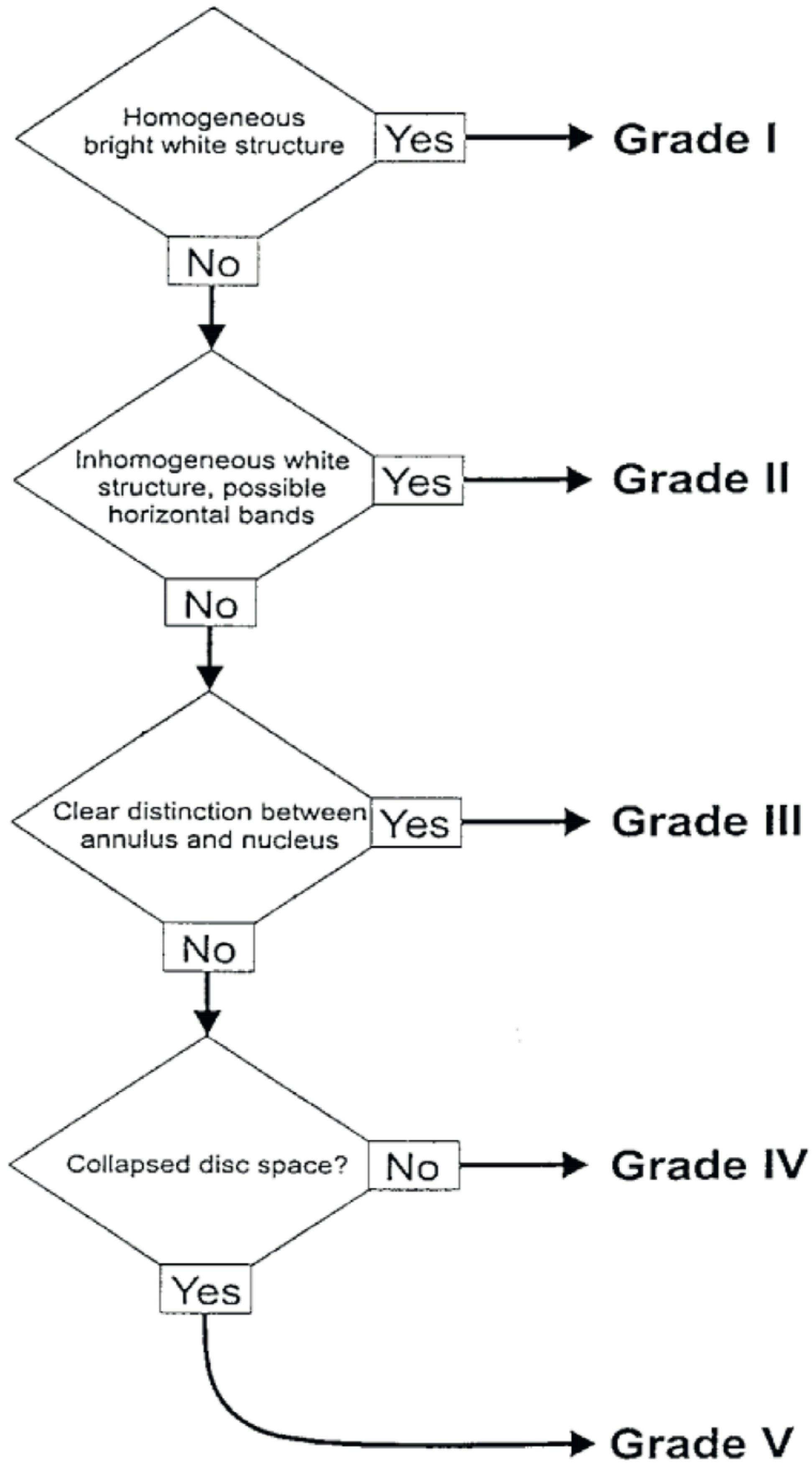


Fig. 6.16 Algorithm for a grading system and assessment of lumbar disk degeneration. (Pfirrmann CWA, Metzdorf A, Zanetti M, et al. Magnetic resonance classification of lumbar intervertebral disc degeneration. Spine 2001;26(17):1873. Lippincott Williams & Williams, Inc. Used with permission.)

H. Biomechanics of transpedicular instrumentation.
   1. Anatomy.
      a. The pedicle is a cylinder of cortical bone.
         (1) The horizontal diameter from T9 to L5 increases from 7 mm to 1.5 cm **(Fig. 6.17)**.
            (a) The vertical diameter is ~ 1.5 cm.
            (b) The inner diameter is < 80% of the outer diameter.
            (c) The pedicle screw diameter should be smaller than the inner diameter.
         (2) The pedicle depth is ~ 45 to 50 mm from the entrance point to the anterior vertebral margin.
      b. Entrance points and directions **(Fig. 6.12):**

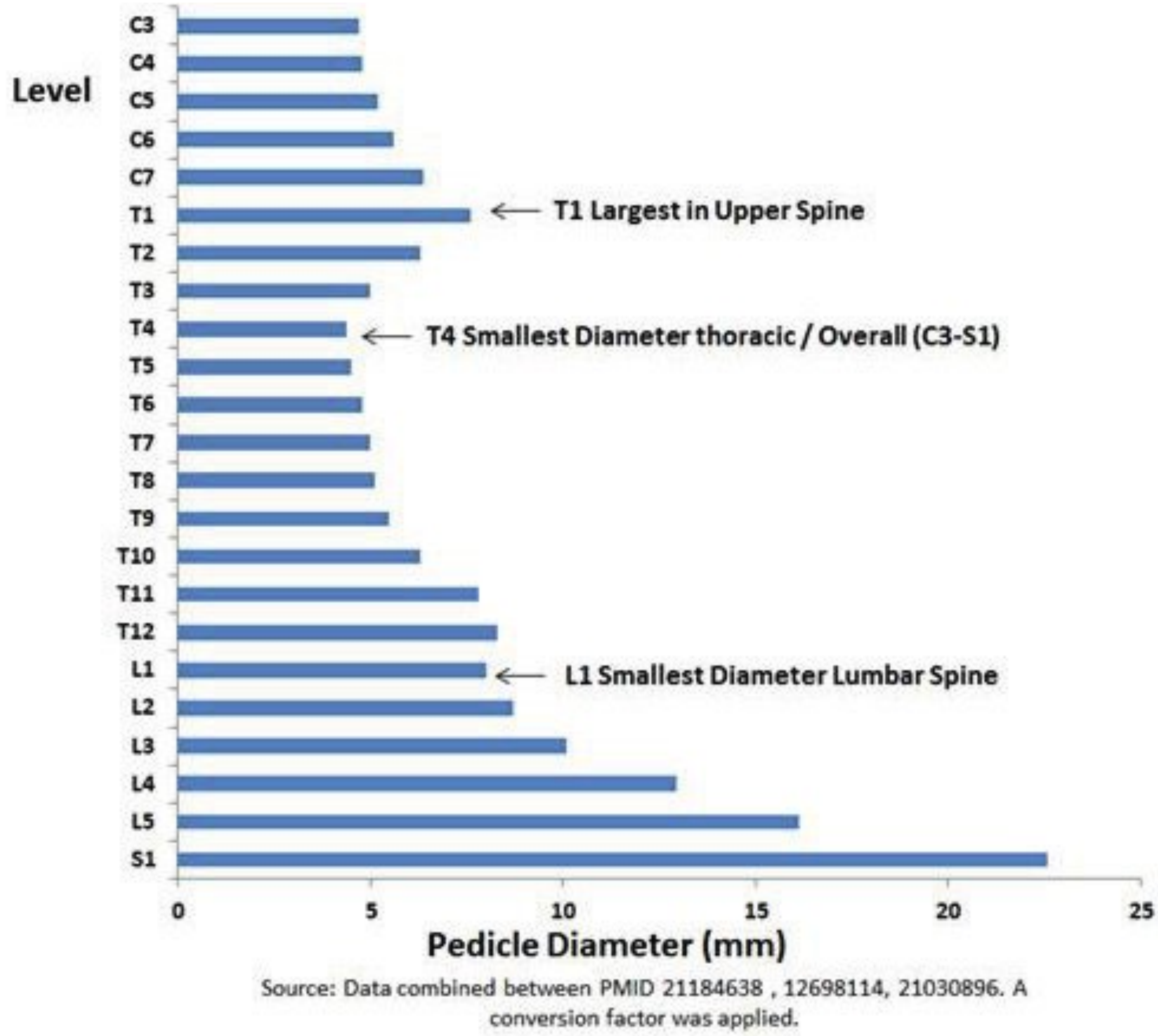


Fig. 6.17 Pedicle diameter (mm) as a function of spinal level. (Courtesy of Orthobullets.)

2. Pedicle screw design and biomechanics.
   a. Structural characteristics.
      (1) The most significant factor for pullout strength is the outer (major) diameter.
      (2) Deeper threads increase the pullout strength.
         (a) Bending strength diminishes due to a smaller minor diameter **(Fig. 6.18)**.
         (b) Pullout strength is also increased by the depth of penetration.
         (c) Not significantly affected by the shape of thread.
3. Transverse connectors.
   a. Important for less rigid systems.
   b. Important if triangulation technique is to be used.
   c. Important in the osteoporotic spine.
   d. Improves torsional stability of the construct.

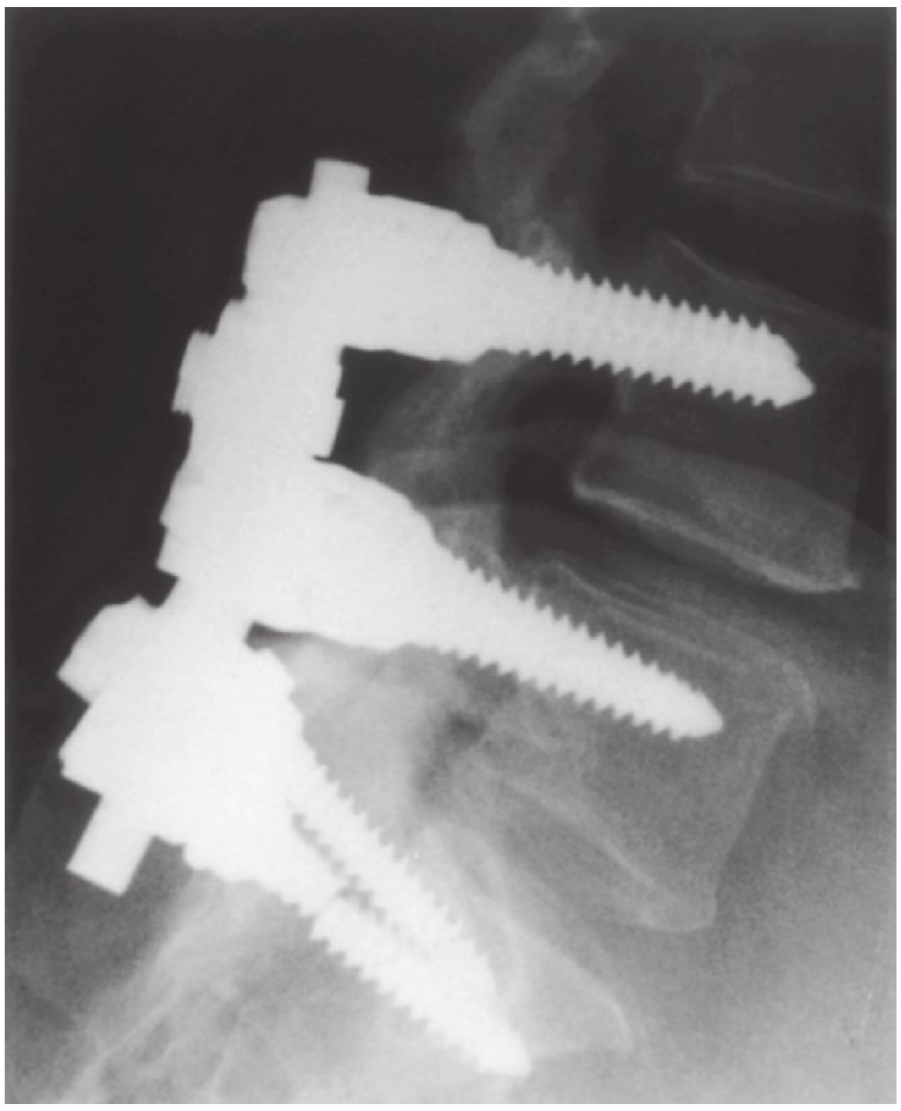


Fig. 6.18 Lateral radiograph of the lumbar spine. Note that the sacral screw has broken because fusion has not occurred. Pedicle screws will eventually fail with cyclical loading if a solid arthrodesis is not obtained.

I. Sacral/pelvic fixation.
 1. Types:
  a. Galveston technique.
  b. Iliosacral screw.
  c. Sacral screws.
  d. Sacral alar screws.
  e. Iliac screws (iliac bolts).
  f. Transiliac bar.
  g. S2 alar iliac screw (S2AI).
  h. Intrasacral rods (Jackson).
  i. Dunn–McCarthy rods (through S1 foramen).
 2. Screws are generally better than hooks in the sacrum.
  a. Sacral fixation with a single sacral screw has a high failure rate (pullout).
  b. S1 screw:
   (1) Anteromedial direction toward the sacral promontory below the superior sacral end plate.
    (a) Safest and biomechanically acceptable.
  c. S2 screw:
   (1) Weak but may enhance overall stability.
   (2) The screw is directed 30 to 40° laterally.
    (a) Avoid penetrance of the anterior cortex if the screw is directed laterally to avoid injuries to the iliac vein, lumbosacral trunk, and sigmoid colon.

J. Instrumentation rigidity and stiffness.
   1. A rigid implant construct increases fusion mass.
   2. May also cause device-related osteoporosis (stress shielding):
      a. Stress shielding by rigid instrumentation is ~ 15%
      b. The benefit of rigid instrumentation outweighs the stress-shielding phenomenon.

Suggested Reading

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7 Physiology of Bone Healing and Fusion

7.1 General Considerations

I. Bone cells and extracellular matrix.
   A. Cells **(Fig. 7.1).**
      1. Osteoblasts.
         a. Responsible for forming the structural bone matrix and regulating osteoclast activity.
         b. Derived from the bone marrow stromal cells and periosteal membrane cells.
         c. Secrete type I collagen.
         d. Express parathyroid hormone receptors and alkaline phosphatase.
            (1) Critical for regulating bone production.
      2. Osteocytes.
         a. Active osteoblasts embedded within the mineralized matrix.
         b. Do not express alkaline phosphatase.
         c. Communicate via canaliculi and regulate bone homeostasis.

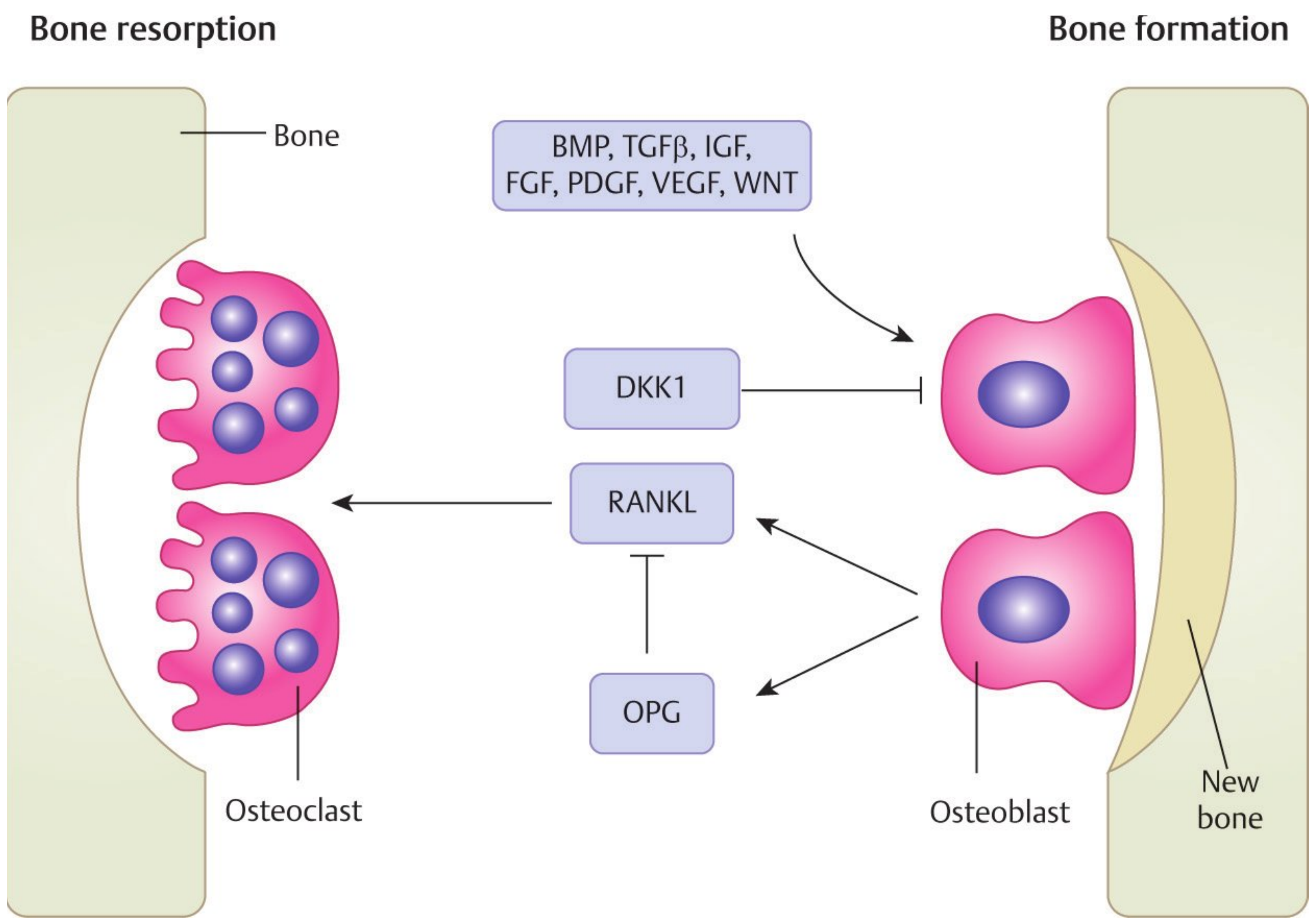

Fig. 7.1 Bone resorption and bone formation and associated factors.

3. Osteoclasts.
   a. Multinucleated bone resorbing cells.
   b. Hematopoietic cells that derive from the monocyte/macrophage family.
   c. These cells have a ruffled border that secretes proteases and ions, which help dissolve the bony matrix (Howship's lacunae) **(Fig. 7.2).**
   d. Tightly regulated by the receptor activator of nuclear factor kB ligand (RANKL).

B. Extracellular matrix (ECM).
   1. The ECM consists of 60 to 70% mineral matrix and 20 to 25% organic matrix.
      a. Mineral matrix.
         (1) Provides the compressive strength of bone.
         (2) Calcium (hydroxyapatite) and phosphate (tricalcium phosphate) make up the majority of the mineral matrix.
      b. Organic matrix.
         (1) Composed primarily of type I collagen (90%).
            (a) Its triple helical conformation contributes tensile strength to the ECM.

II. Bone formation.
   A. Ossification **(Fig. 7.3).**
      1. Intramembranous (e.g., pelvic bones): formation of bone directly from mesenchymal tissue.
      2. Endochondral (e.g., vertebrae): mesenchymal tissue is first replaced by a cartilage model, which then undergoes ossification.
   B. Key growth factors and cytokines.
      1. Bone morphogenetic proteins (BMPs).
      2. Transforming growth factor-b (TGF-b).
      3. Basic fibroblast growth factor (bFGF).
      4. Insulin growth factor (IGF).
      5. Interleukins (ILs).

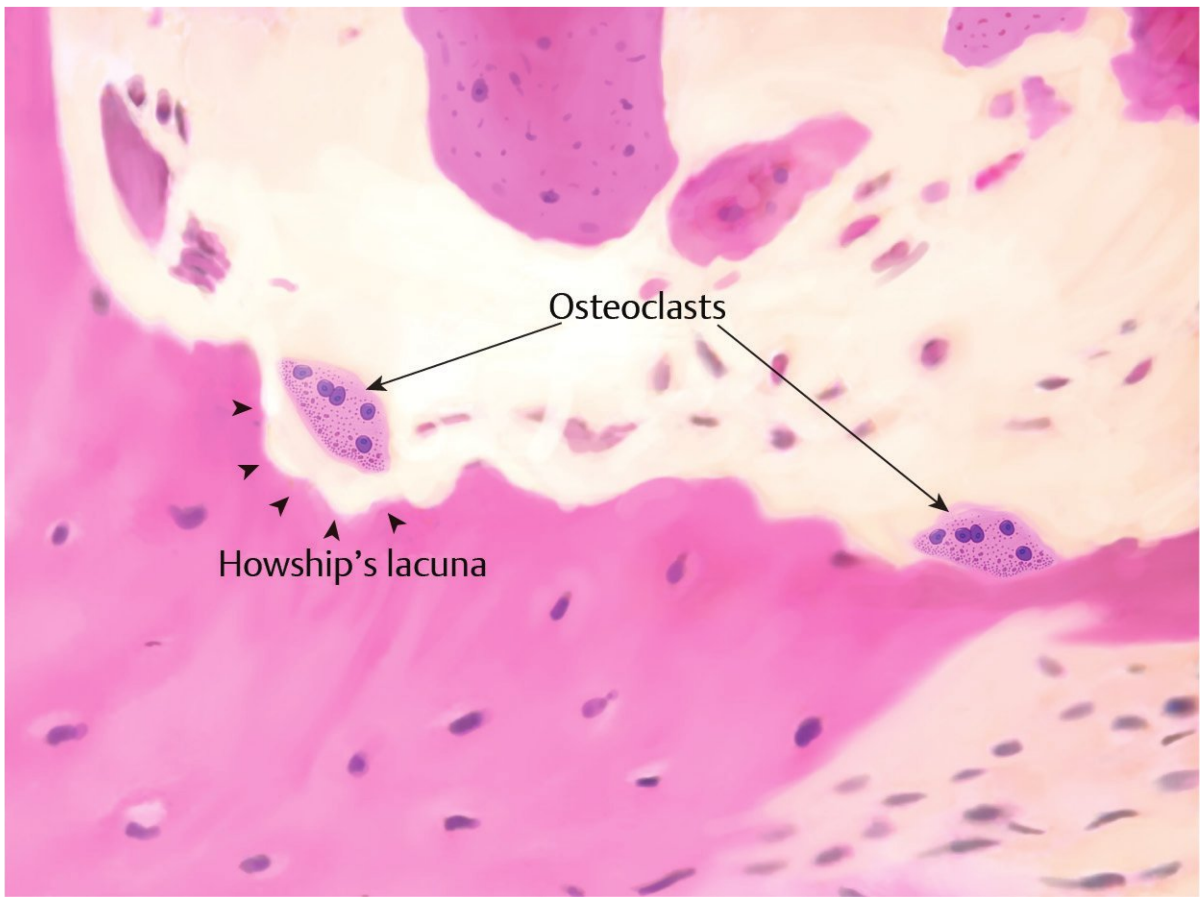

Fig. 7.2 Microscopic hematoxylin-eosin image of osteoclasts and Howship's lacunae.

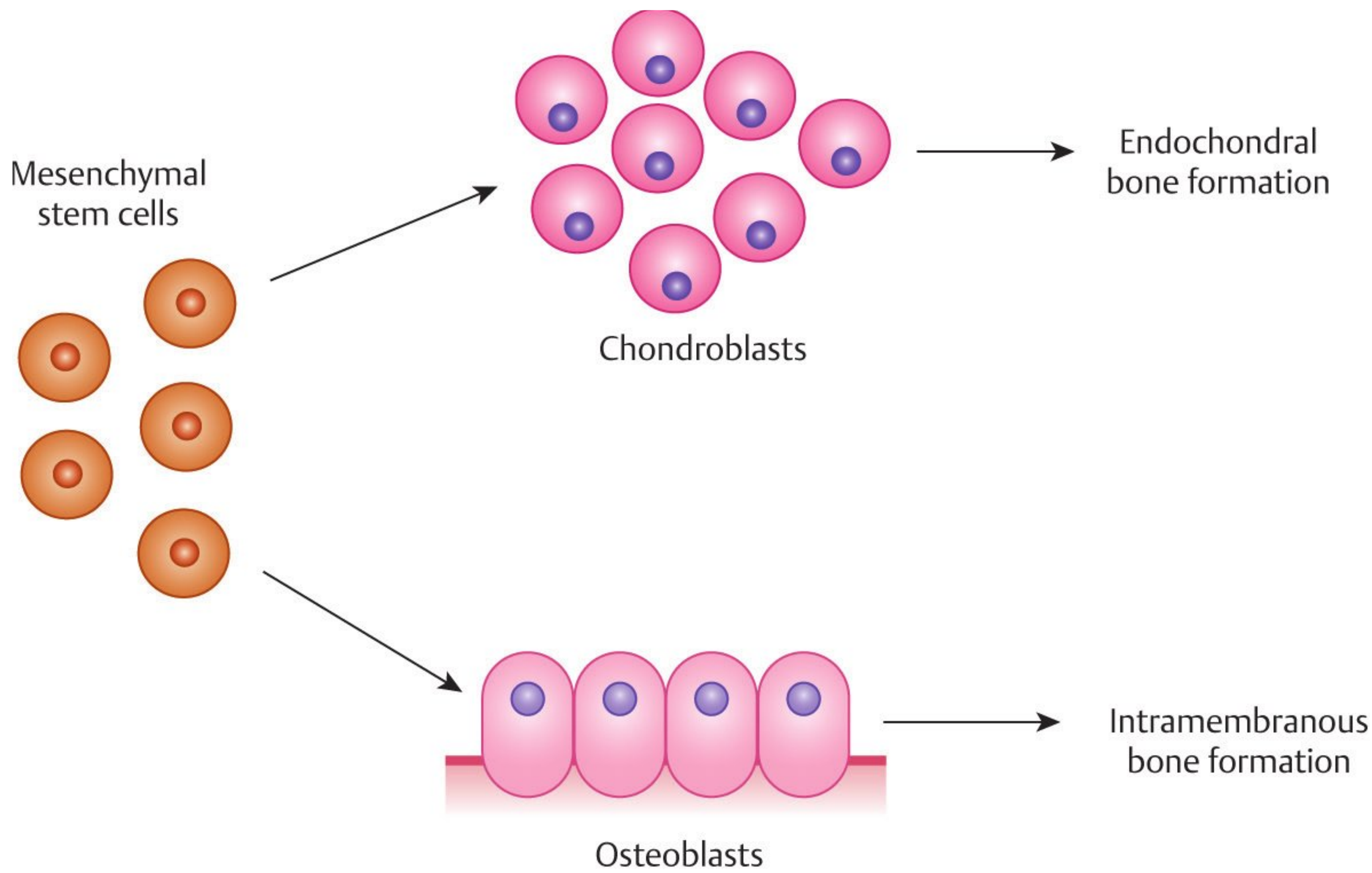

Fig. 7.3 Mesenchymal stem cell differentiation into endochondral versus intramembranous bone formation.

III. Bone repair and remodeling **(Fig. 7.4).**
 A. Hematoma and inflammatory response.
  1. Predominant cell types.
   a. Macrophages.
   b. Platelets.
  2. Cytokines.
   a. IL-1, IL-6.
   b. TGF-b.
   c. Prostaglandin E2.
 B. Early stage.
  1. Predominant cell types.
   a. Mesenchymal cells.
   b. Fibroblasts.
  2. Protein expression.
   a. BMP.
   b. TGF-b.
 C. Hematoma maturation.
  1. New collagenous matrix is produced and slowly replaced by cartilage formation (endochondral ossification).
  2. Collagen types I and II are predominant.
 D. Conversion of cartilage to bone.
  1. The formed hypertrophic cartilage is slowly replaced by bone.

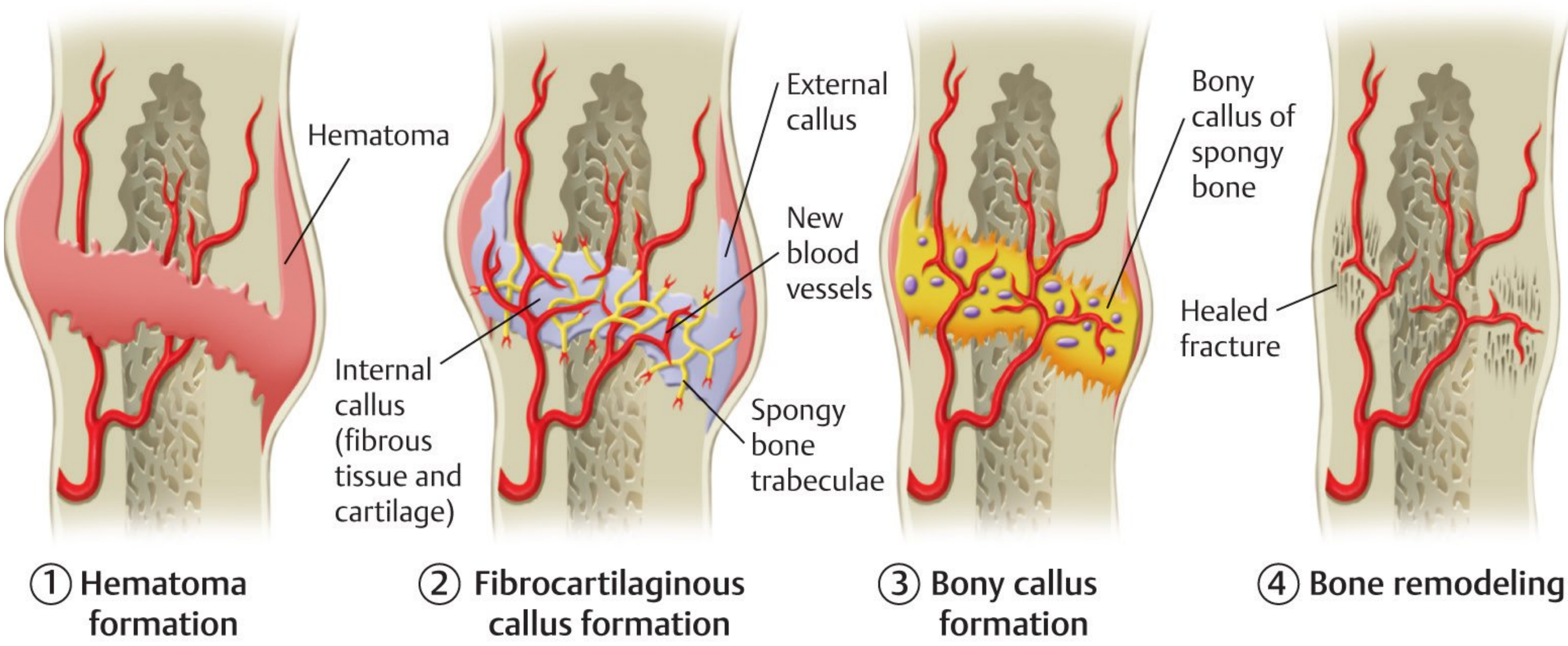

Fig. 7.4 Process of bone repair and remodeling.

2. Protein expression:
   a. BMP.
   b. TGF-b.
   c. IGFs.
   d. Collagen types I and V.
   e. Osteocalcin.

E. Bone remodeling.
   1. Newly formed bone (woven bone) is then remodeled through tight regulation between osteoblasts and osteoclasts.

7.2 Physiology of Bone Grafts

I. Types of grafts.
   A. Autograft.
      1. Iliac crest bone graft (ICBG) is the gold standard.
   B. Allografts.
      1. Fresh, frozen.
      2. Freeze dried.
      3. Cortical cancellous chips.
      4. Demineralized bone matrix (DBM).

II. Bone graft use in spine surgery **(Fig. 7.5).**
   A. Induces fusion between vertebral segments.
   B. Replaces bone defects secondary to trauma, tumor, or infections.

III. Bone graft incorporation.
   A. Undifferentiated progenitor cells are recruited from the host bed and the implanted autograft.
      1. Osteogenic cells from the autograft help to form the initial bone matrix (osteogenesis).

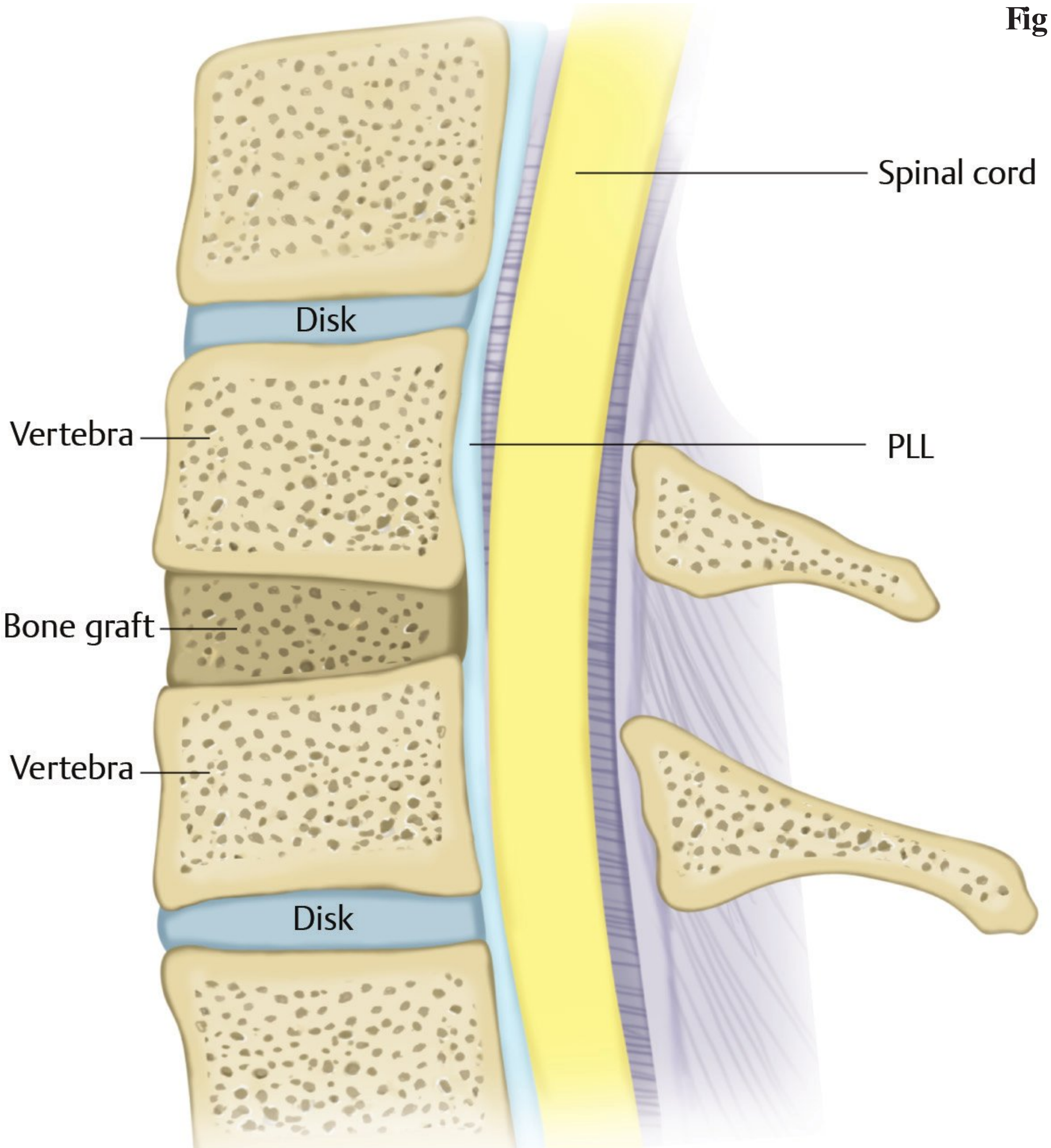

Fig. 7.5 Use of bone graft.

B. Chemotaxis of these progenitor cells is induced by the release of intracellular cytokines and protein expression as a result of the following:
   1. Cell death.
   2. Surgical trauma.
   3. Decortication.
   4. Low oxygen tension and low pH.

C. Undifferentiated progenitor cells become chondroblasts and osteoblasts mediated by growth factors and cytokines (endochondral ossification).

D. The graft's protein matrix acts as a scaffold for bony ingrowth.

IV. General categories of bone grafts.

A. Substitutes: general terms for materials or composite grafts to be intended to replace autografts (e.g., allograft, DBM, BMP).

B. Extenders: used in combination with autograft to increase the amount of osteoconductive and osteoinductive factors for fusion (e.g., calcium phosphate ceramics, allograft, DBM).

C. Enhancers: materials used in conjunction with autograft to increase the rate of fusion; should not be used alone (e.g., DBM, BMP, stem cells).

V. Factors affecting spinal fusion.

A. Patient factors.
   1. Age.

2. Smoking (nicotine consumption).
3. Diabetes mellitus.
4. Metabolic bone disease.
5. Vitamin D deficiency.

B. Anatomical regions.
   1. Spine segment.
      a. Cervical spine.
         (1) Less body mass to support.
         (2) Minimal intervertebral displacement and micromotion due to larger contact area between adjacent vertebral bodies.
         (3) Low strain environment optimizes bone formation.
         (4) Fusion rates are 82 to 100%
      b. Thoracic spine.
         (1) Rib attachments provide additional stability and minimize flexion, bending, and rotation of vertebral segments.
            (a) Associated with high fusion rates.
         (2) The thoracolumbar transition (T12–L1), however, is highly mobile.
            (a) Susceptible to micromotion, hardware failure, and fusion failure (pseudarthrosis).
      c. Lumbar spine.
         (1) High joint reaction forces.
         (2) The primary motion is flexion and extension, with minimal lateral flexion and rotation.
         (3) Posterior elements demonstrate greater excursion than the anterior column.
         (4) Instrumentation that counteracts the large forces of the lumbar spine is critical to promote spinal fusion.
         (5) Fusion rates range from 70 to 100%

C. Surgical procedures.
   1. Primary or revision surgery.
   2. Levels of fusion.
   3. Instrumentation.
   4. Surgical techniques.
      a. Meticulous decortication.
      b. Graft preparation.

D. Types and quantity of bone graft.

E. Medications.
   1. Nonsteroidal anti-inflammatory drugs.
   2. Chemotherapy.
   3. Corticosteroid exposure.

F. Radiation.

G. Electrical stimulation.

H. Ultrasonography.

I. Spinal alignment.

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8 Bone Grafts, Substitutes, and Biologics

8.1 General Considerations

I. Types of bone grafts and biologics.
   A. Autograft.
      1. Iliac crest bone graft (gold standard).
      2. Local bone graft.
   B. Allografts.
      1. Fresh, frozen, or freeze dried.
      2. Cortical cancellous bone chips.
      3. Demineralized bone matrix (DBM).
   C. Ceramics.
   D. Bone morphogenetic proteins (BMP-2, BMP-7).
   E. Bone marrow aspirate and stem cells.
II. Properties of bone grafts.
   A. Osteogenic: directly provide cells that go on to produce bone.
      1. Examples include bone marrow aspirate and autologous bone grafts.
   B. Osteoinductive: contain factors that induce progenitor cells into bone-forming cells.
      1. Examples include BMPs (BMP-2 and BMP-7).
   C. Osteoconductive: provide a scaffold for new bone formation.
      1. Examples include DBMs.
III. Autograft versus allograft **(Table 8.1).**

8.2 Allografts

I. DBM.
   A. Properties.
      1. Allograft bone that is treated with acid extraction to isolate growth factors and structural proteins (collagen). The resulting bone matrix contains < 8%calcium.
         a. DBM does not contain osteoprogenitor cells.
         b. Good osteoconductive properties.
      2. Biologic effects vary widely among commercially available DBMs.
         a. Preparation.
            (1) Demineralization.
            (2) Sterilization.
            (3) Carrier.
               (a) Glycerol.
               (b) Calcium hyaluronate.
               (c) Cellulose.
            (4) Amount and ratios of BMPs.

Table 8.1 Autograft versus allograft

Type of graft	Autograft	Allograft
Osteogenic	+	–
Osteoinductive	+	±
Osteoconductive	+	+
Donor site morbidity	+	–
Immune reaction	–	±
Disease transmission	–	±
Types	• Cortical • Corticocancellous • Cancellous • Vascularized • Bone marrow aspirate	• Fresh • Frozen • Freeze dried • Cortical cancellous chips • Demineralized bone matrix
Limitations	• Limited supply • Large defects (tumor, infection)	• Slow incorporation • High incidence of resorption in posterolateral fusions

b. Donor characteristics.
(1) Patient age and gender.
(2) Bisphosphonate utilization.

B. Urist reported on DBM-induced bone formation in 1965.

8.3 Synthetic Substitutes

I. Ceramics.
A. Properties.
1. Combination of metallic and nonmetallic inorganic elements held together by ionic or covalent bonds.
2. Do not provide osteogenic or osteoinductive properties.
3. Provide immediate structural support and are osteoconductive.
B. Biomechanical strength.
1. Low fracture resistance and tensile strength.
2. Questionable indication for anterior grafting without supplemental fixation.
C. Bonding and release of BMPs.
D. Calcium–based ceramics.
1. Hydroxyapatite, tricalcium phosphate.
2. Calcium sulfate.

3. Calcium phosphate cements.
4. Calcium phosphate ceramics.

E. Clinical use of ceramics.
1. Anterior spinal application.
a. Combination with cages or plates.
2. Filling bony defects (vertebroplasty/kyphoplasty).
3. Graft extenders.

8.4 Growth Factors (Biologics)

I. Bone morphogenetic proteins (BMP-2 and BMP-7) **(Fig. 8.1).**

A. Properties.
1. Members of the transforming growth factor-b superfamily.
2. Potent osteoinductive properties.
3. Associated with increased fusion rates compared with allograft.
4. BMPs are soluble, locally acting, and naturally occurring signaling proteins.
a. Induce mesenchymal cells to differentiate into cartilage and bone-forming cells:
(1) Have the ability to induce bone formation in both soft tissue (muscle, tendons) and bone.
b. Tightly controlled regulatory mechanisms cause bone induction only at the site of BMP and only while BMP is present.
c. The biologic effect of commercially available recombinant human BMPs will depend on the dose and method of delivery.

B. Food and Drug Administration–approved BMPs.
1. Infuse (BMP-2; Medtronic).
a. Approved for a single-level anterior lumbar interbody fusion (ALIF) from L4–S1 to treat degenerative disk disease.
(1) To be used with a tapered interbody fusion device.
b. Approved for acute, open tibial shaft fractures after initial intramedullary nail fixation.

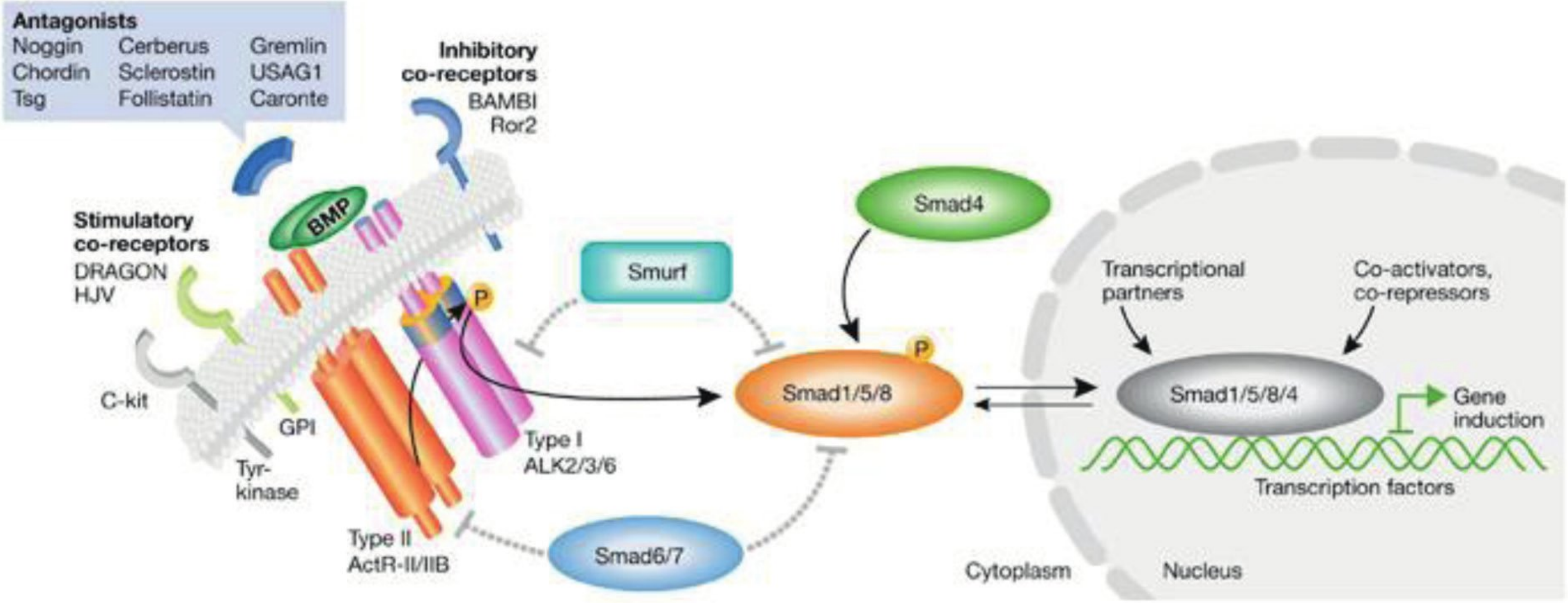

Fig. 8.1 Regulatory mechanisms in the action of bone morphogenetic proteins.

2. OP-1 (BMP-7; Olympus).
    a. Approved for the repair of symptomatic, posterolateral (intertransverse) lumbar spine pseudarthrosis in patients for whom autologous bone and/ or bone marrow harvest are not feasible.
    b. Patients must have at least one of the following comorbidities: osteoporosis, diabetes, or nicotine use.
    c. Approved for recalcitrant long bone nonunions where the use of autograft is unfeasible and alternative treatments have failed.
    d. Available as sterile dry powder and putty.

C. Controversies with BMPs.
1. Majority of BMP use is off-label.
2. Reported adverse effects.
    a. Neuroforaminal bone growth.
    b. Osteolysis.
    c. Retrograde ejaculation.
    d. Dysphagia.
    e. Airway compromise.
    f. Cancer.
3. Biased and conflicted reports in the literature.
    a. Yale Open Data Access (YODA) project.
        (1) Questionable underreporting of complications.
        (2) Limited randomized, controlled prospective studies.
        (3) Lack of consistent reporting and underpowered analyses.

II. Bone marrow aspirate and stem cells **(Fig. 8.2).**

A. Most common sources.
1. Iliac crest and vertebral body.
2. The number of stem cells in the bone marrow.
    a. One in 50,000 in young individuals.
    b. One in 2,000,000 in the elderly.
    c. Potency of marrow aspirate may be increased via centrifugation or clonal expansion.
3. Commercially available stem cell–derived bone graft substitute.
    a. Provides a scaffold for bony ingrowth.
    b. Contains adult mesenchymal cells and osteoprogenitor cells to promote bone formation.
    c. Screened cellular allograft that underwent selective removal of immunogenic elements (blood cells, white blood cells, osteoclasts).
    d. Undergoes antimicrobial and antifungal treatment.
    e. The processed tissue has a 5-year shelf life, because it is cryopreserved.

B. Potential benefits of mesenchymal stem cells (MSCs).
1. Oxidative stress modulation.
2. Autogenous bone contains osteoblastic cells.
    a. Spinal fusion is largely mediated by these cells.
    b. In patients with reduced cellular stores (chronic illness, elderly, osteoporosis), MSC may aid in obtaining a solid fusion.

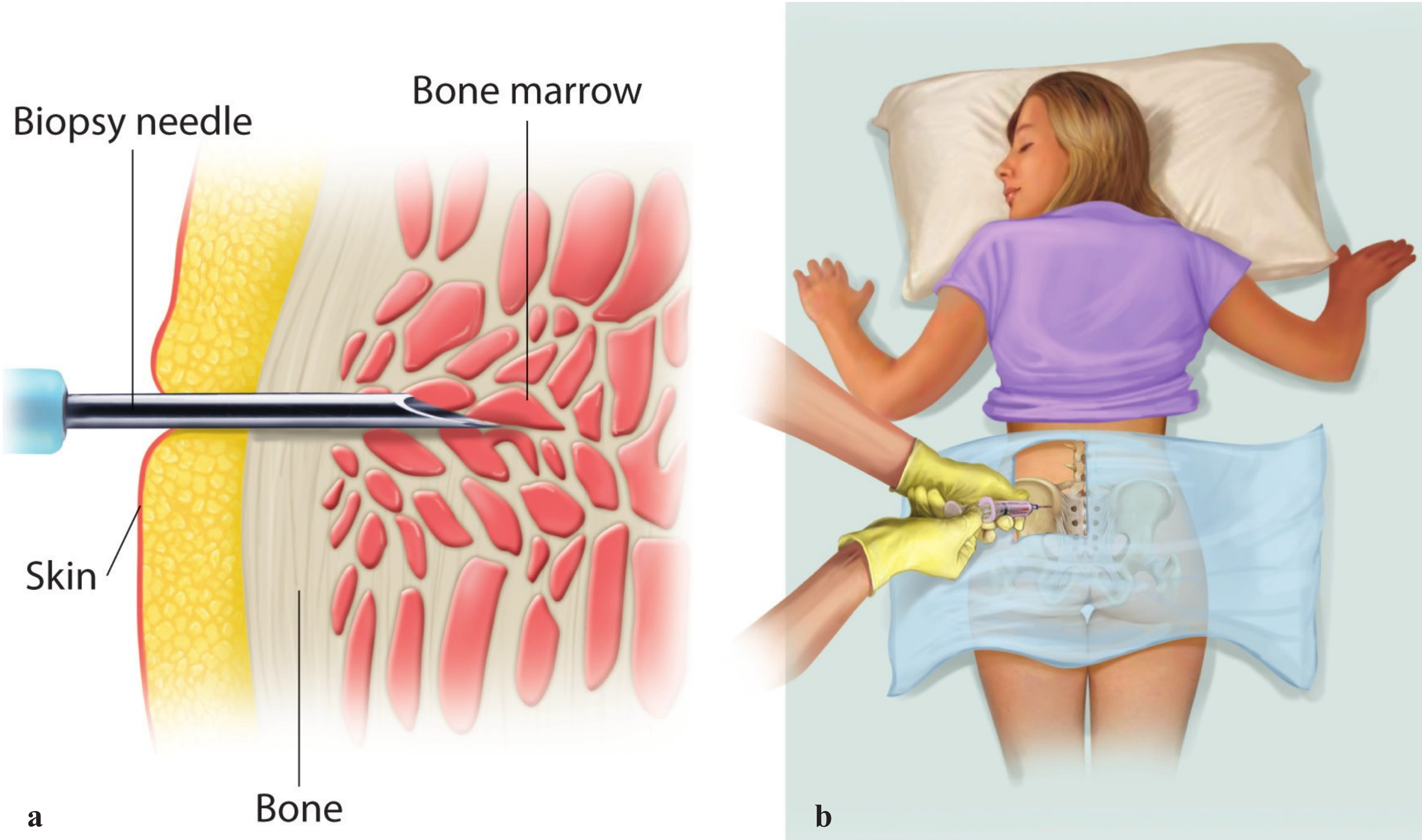

Fig. 8.2 **(a,b)** Bone marrow aspiration.

3. Secretion of cytokines and growth factors:
   a. Immunomodulation.
   b. Anti-inflammatory effects.
   c. Angiogenesis.
   d. Antiapoptotic effects.
   e. Osteogenic properties.
4. Self-renewing.

Suggested Reading

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9 Evaluation and Management of Spinal Cord Injury

9.1 General Considerations

I. Introduction.
   A. Approximately 12,000 to 14,000 spinal cord injuries (SCIs) occur in North America per year.
      1. They most commonly occur in adolescent males.
      2. Most are related to motor vehicle collisions.

II. Prehospital evaluation period.
   A. Care of any trauma patient begins with immobilization of the spine at the scene of injury.
   B. American College of Surgeons mnemonic for in-field management of a trauma patient:
      1. A—airway.
      2. B—breathing.
      3. C—circulation.
      4. D—disability.
      5. E—exposure and environment.
   C. All trauma patients should be immobilized with a rigid cervical orthosis and transferred using lateral bolsters.
      1. The patient should be placed on a long backboard secured with tapes or straps.
      2. Helmeted athletes:
         a. Helmet and shoulder pads should *not* be removed.

III. Emergency room management.
   A. Polytrauma patients may have an altered level of consciousness and are vulnerable to further worsening of their neurological injuries.
   B. Once the ABCs have been established, a thorough but focused physical examination should assess the patient's neurological function.
      1. The entire spinal column should be palpated for "step-off" or misalignment of the spinous processes.
   C. Head trauma:
      1. Glasgow Coma Scale.
         a. Total score can range from 15 (normal responses to stimuli) to 3 (no response or comatose) **(Table 9.1)**.
   D. Evaluation of airway and breathing:
      1. Elective intubation should be performed in patients with severe head injuries or who are unable to protect the airway because of a depressed level of consciousness (Glasgow Coma Scale score < 8).
      2. Patients with SCIs (particularly above C5) having difficulty with respiration should be considered for elective intubation.
      3. Manual in-line stabilization of the cervical spine during orotracheal intubation minimizes motion of the unstable cervical spine.

Table 9.1 Glasgow Coma Scale

Eye opening	Spontaneous	4
	Voice	3
	Pain	2
	None	1
Verbal response	Oriented	5
	Confused	4
	Inappropriate	3
	Incomprehensible	2
	None	1
Motor response	Obeys commands	6
	Localized pain	5
	Withdraws	4
	Decorticate	3
	Decerebrate	2
	None	1

E. Neurological examination **(Fig. 9.1)**:
  1. The American Spinal Injury Association (ASIA) standard of neurological testing provides a concise and detailed method of evaluating spinal cord and peripheral nerve root function.
     a. Sensation is determined in all 28 dermatomes bilaterally by the patient's ability to detect the sharp end of a pin **(Fig. 9.2)**.
     b. Motor function is documented and graded 1 to 5 based on resistance to physical manipulation or gravity **(Table 9.1)**.
     c. Based on both the motor and the sensory examination, the patient is further classified or graded using the ASIA modification of the Frankel neurological classification system **(Table 9.2)**.

F. SCIs:
  1. Complete injury.
     a. No functional motor (less than grade III motor strength) or sensory activity below the zone of injury.
  2. Incomplete injury.
     a. Partial preservation of motor or sensory function below the zone of injury.

G. Imaging evaluation:
  1. Initial radiographs.
     a. Standard anteroposterior/lateral views of the cervical, thoracic, and lumbosacral spine.
        (1) Ten to 15%of patients have noncontiguous spinal column fractures.

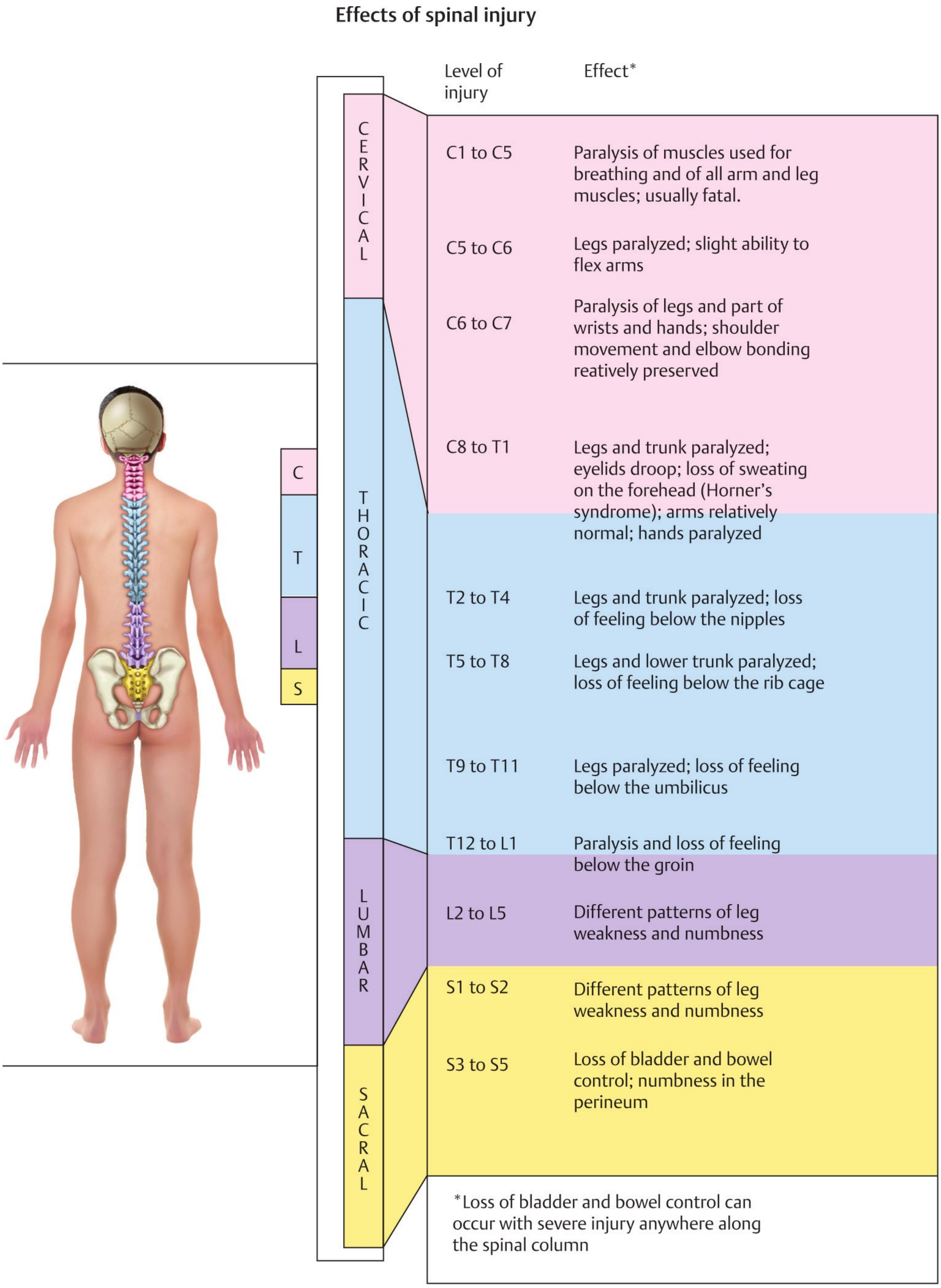

Fig. 9.1 Clinical manifestations of spinal cord injury according to level of injury.

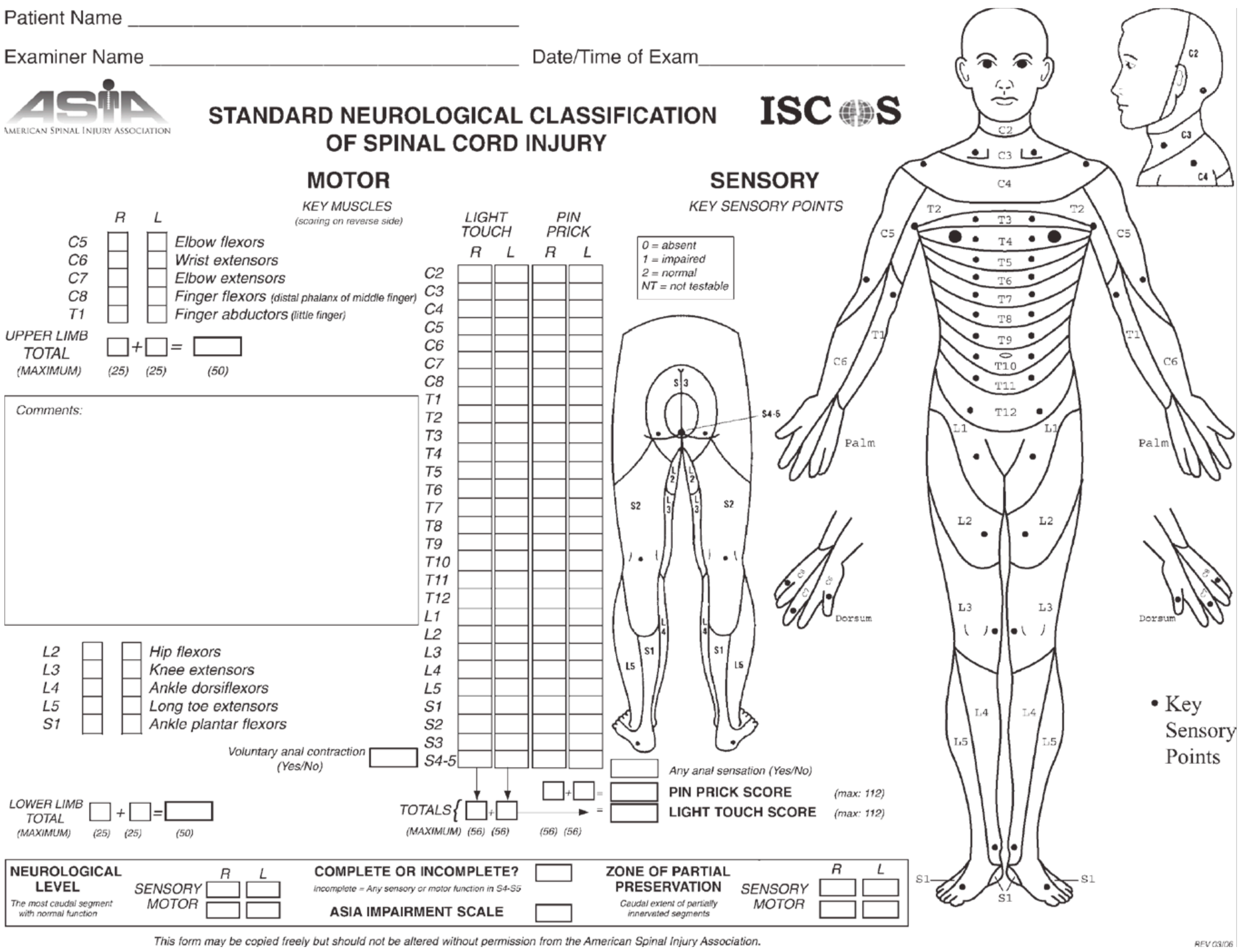

Fig. 9.2 American Spinal Injury Association spinal cord injury assessment form. (From the American Spinal Injury Association. International Standards for Neurological Classification of Spinal Cord Injury. Revised 2002. Chicago, IL: American Spinal Injury Association; 2006. Reproduced with permission.)

b. Always visualize the alignment of the cervicothoracic junction.
c. Computed tomography (CT):
   (1) Used to further delineate bony anatomy.
   (2) Helpful in visualizing the cervicothoracic (C7–T1) junction.
   (3) A CT scan must be obtained if C7–T1 is not visible on lateral radiographs.
      (a) Most trauma centers have now begun routinely obtaining CT scans of the cervical spine as the gold standard for cervical spine clearance, obviating the need for plain radiographs.
d. Magnetic resonance imaging:
   (1) Used in all cases of neurological compromise.
   (2) Useful for visualizing soft tissue anatomy and disruption.

H. Treatment:
1. The severity of the initial impact on the spinal cord is typically reflected by the patient's initial neurological presentation.
2. The extent of the patient's neurological recovery is not solely dependent on the primary injury.
   a. Secondary injury to the spinal cord results from a physiological cascade.
      (1) Initial hemorrhage followed by inflammation, membrane hydrolysis, ischemia, calcium influx, and cellular apoptosis.

Table 9.2 American Spinal Injury Association (ASIA) Classification Scale

1. Determine if patient is in spinal shock.
   – Check bulbocavernosus reflex.
2. Determine neurologic level of injury.
   • Lowest segment with intact sensation and antigravity (3 or more) muscle function strength.
   • In regions where there is no myotome to test, the motor level is presumed to be the same as the sensory level.
3. Determine whether the injury is COMPLETE or INCOMPLETE.
   • COMPLETE define as ASIA A
     – no voluntary anal contraction (sacral sparing) AND
     – 0/5 distal motor AND
     – 0/2 distal sensory scores (no perianal sensation) AND
     – bulbocavernous reflex present (patient not in spinal shock)
   • INCOMPLETE defined as
     – voluntary anal contraction (sacral sparing)
     – sacral sparing critical to determine complete vs. incomplete
     – OR palpable or visible muscle contraction below injury level OR
     – perianal sensation present
4. Determine ASIA Impairment Scale (AIS) Grade:

ASIA Impairment Scale

A	Complete	No motor or sensory function is present in the sacral segments S4–S5.
B	Incomplete	Sensory function preserved but no motor function is preserved below the neurological level and includes segments S4–S5.
C	Incomplete	Motor function is preserved below the neurological level, and more than half of key muscles below the neurological level have a muscle grade less than 3.
D	Incomplete	Motor function is preserved below the neurological level, and at least half of key muscles below the neurological level have a muscle grade of 3 or more.
E	Incomplete	Motor and sensory function are normal.

Source: Kirshblum SC, Burns SP, Biering-Sorensen F, et al. International standards for neurological classification of spinal cord injury (Revised 2011). J Spinal Cord Med 2011;34(6):535–546. Used with permission.

3. Pharmacological intervention:
   a. The most commonly used agent is methylprednisolone. However, the extent of recovery in terms of improved functional ability and the risk to the patient have been extensively debated.
      (1) National Acute Spinal Cord Injury Study (NASCIS) guidelines are most commonly used.
      (2) Methylprednisolone: bolus dose of 30 mg/kg of body weight over 15 minutes, followed by a 45-minute pause, and then a 23-hour continuous infusion of 5.4 mg/kg/h if the patient presents < 3 hours after injury.

(3) If the patient presents between 3 and 8 hours after injury, the steroid infusion is continued for a total of 48 hours.

b. The NASCIS II Trials advocated for the administration of high-dose steroids within 8 hours of injury if the following contraindications are absent:
   (1) Cauda equina syndrome.
   (2) Nerve root injury.
   (3) Life-threatening patient status.
   (4) SCI secondary to gunshot.
   (5) Pregnancy.
   (6) Patient younger than 13 years of age.
   (7) Substance addiction.
   (8) Chronic steroid use.

c. Numerous authors have questioned the validity of the study design and have refuted the findings of the NASCIS trials.

d. In 2013, the Congress of Neurological Surgeons and the American Association of Neurological Surgeons recommended against the use of steroids following an SCI.
   (1) Lack of evidence that demonstrates beneficial outcomes.
   (2) Greater risk of potential side effects (gastrointestinal bleeding, infection, avascular necrosis of the femoral head).

4. Surgical timing:
   a. Data from the Surgical Timing in Acute Spinal Cord Injury Study (STASCIS) trials suggest that early intervention with decompression and stabilization may potentiate better neurological recovery following an SCI.
      (1) Primary injury from physical compression or contusion.
      (2) Secondary injury from the downstream cascade of events that result following the primary insult:
         (a) Neuroprotection involves the prevention of secondary injury with the expeditious decompression of neural elements.
         (b) The potential for neuroprotection varies inversely with time.
      (3) Decompression within 24 hours of injury results in improved neurological outcomes at 6-month follow-up.

Suggested Reading

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10 Cervical Spine Trauma

10.1 General Considerations

I. Introduction.
  A. Fifty thousand cervical spinal column or cord injuries are reported yearly in the United States.
  B. Most spinal column or cord injuries occur in males between the ages of 15 and 24.
  C. Mechanism:
    1. Motor vehicle accidents are the most common cause (40–56%) **(Fig. 10.1)**.
    2. Falls (20–30%).
    3. Gunshots (12–21%).
    4. Sports (6–13%).
  D. The midcervical spine (C4–C6) is the most commonly affected anatomical region.
II. Patient evaluation.
  A. A detailed history, including the mechanism of injury as well as associated injuries, is required.
  B. Early recognition of the injury begins in the field.
    1. A cervical collar and spine board are applied to the patient.
    2. The patient is transferred to an emergency department.
      a. The trauma resuscitation team evaluates airway competency, breathing, and circulation.
    3. Anteroposterior and lateral radiographs of the entire spine can be obtained.
    4. Computed tomography (CT) is the most sensitive and specific imaging study to diagnose cervical spine injuries **(Fig. 10.2)**.
      a. Intravenous contrast is useful to determine vertebral artery injury.
  C. Cervical spine traction:
    1. Indications.
      a. Unstable fractures or fracture dislocations of the cervical spine with or without neurological deficits.
      b. Damage control therapy for cervical spine fractures while the patient is being treated for other injuries.
    2. Contraindications.
      a. Complete permanent quadriplegia.
      b. Unstable fractures with deteriorating neurological signs.
      c. Stable fractures.
      d. A patient who is not awake, alert, and cooperative.
    3. Technique: Gardner-Wells tongs **(Fig. 10.3).**
      a. The skin over the temporal fossa is infiltrated with a local anesthetic agent. The pins are positioned 2 cm above the external auditory canal and below the temporal ridge. The pins are tightened until the spring-

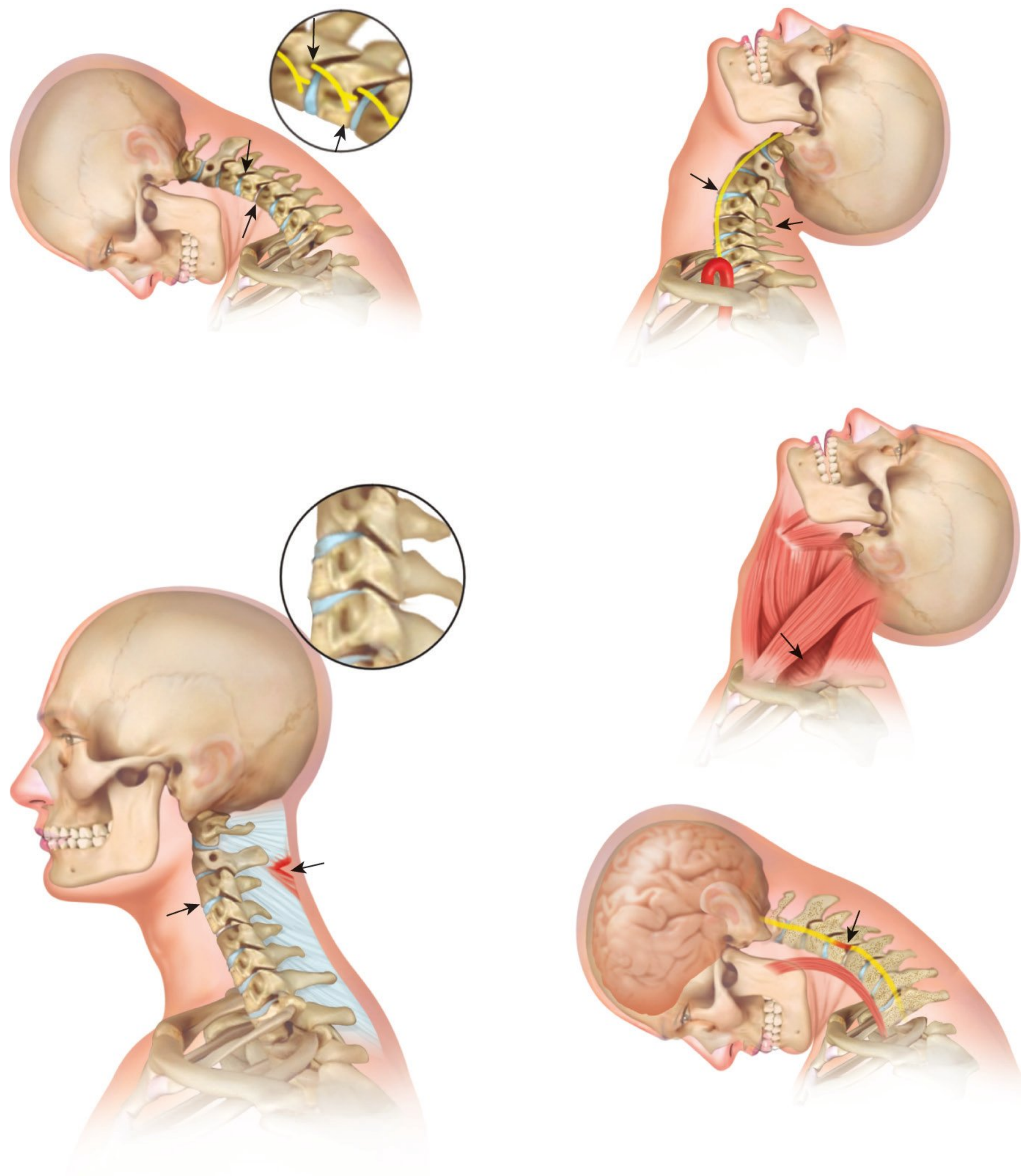

Fig. 10.1 Mechanism of whiplash injuries of the head and neck.

loaded indicator protrudes 1 mm above the surface. Initially, 10 lb of traction-weight is used. Weights are added at 10 lb increments every 20 minutes. Serial neurological exams are performed and radiographs are taken after each weight is placed.

4. Complications.
   a. Failure to reduce: a bilateral, irreducible facet dislocation is unstable and should be treated with urgent open reduction after magnetic resonance imaging (MRI) is performed.
   b. Change in the neurological status: with any change in the neurological status of the patient, the weights should be removed immediately and an MRI scan should be obtained emergently.

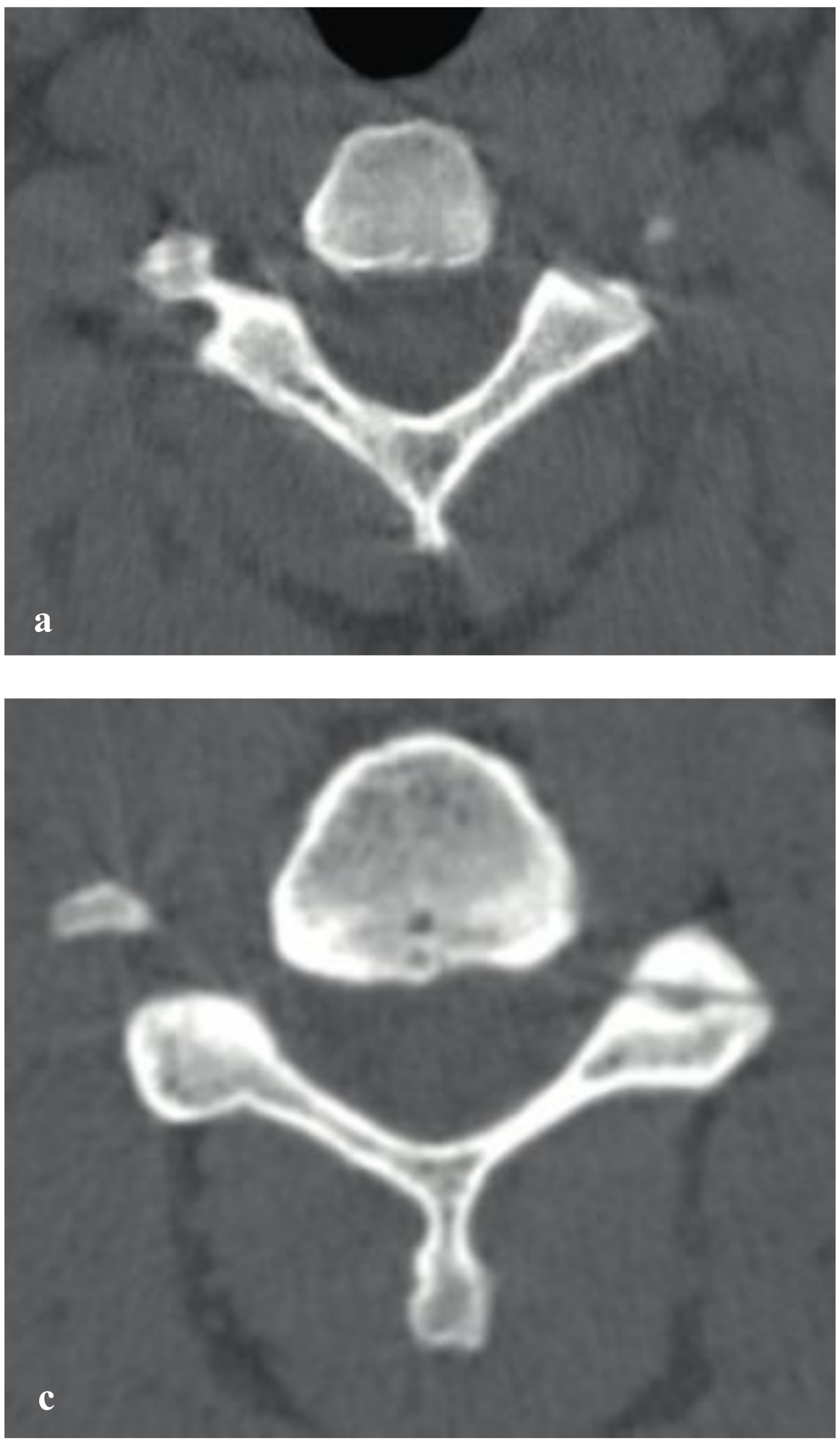


Fig. 10.2 **(a)** Sagittal computed tomographic (CT) images of the cervical spine demonstrating spinal stenosis and spondylosis. **(b)** Axial CT images of the cervical spine demonstrating spinal stenosis and spondylosis at the level of the C3 vertebrae. **(c)** Axial CT images of the cervical spine demonstrating spinal stenosis and spondylosis at the level of the C4 vertebrae.

III. Upper cervical injuries.
  A. Occipital condyle fractures.
    1. Rare injury.
    2. One-third occur in conjunction with atlanto-occipital dislocations.
    3. Diagnosis is most commonly made incidentally with a head CT scan.
      a. Ligamentous injury, intracranial hematoma, and neurological deficit may accompany this injury.
    4. Treatment:
      a. Usually rigid orthosis or halo vest for 3 months.
      b. A flexion–extension film is obtained at 3 months.
      c. Occipital–cervical arthrodesis for resultant instability.
  B. Occiput–C1 dislocation.
    1. Unstable and almost always fatal.
      a. Survivors usually have severe neurological deficits.
    2. Violent, twisting, or flexion-extension force on the head.
    3. Disruption of all ligamentous attachments.
    4. Radiographic diagnosis.
      a. Harris radiographic lines.
    5. Treatment.
      a. Closed reduction.
      b. Occipital-cervical fusion.

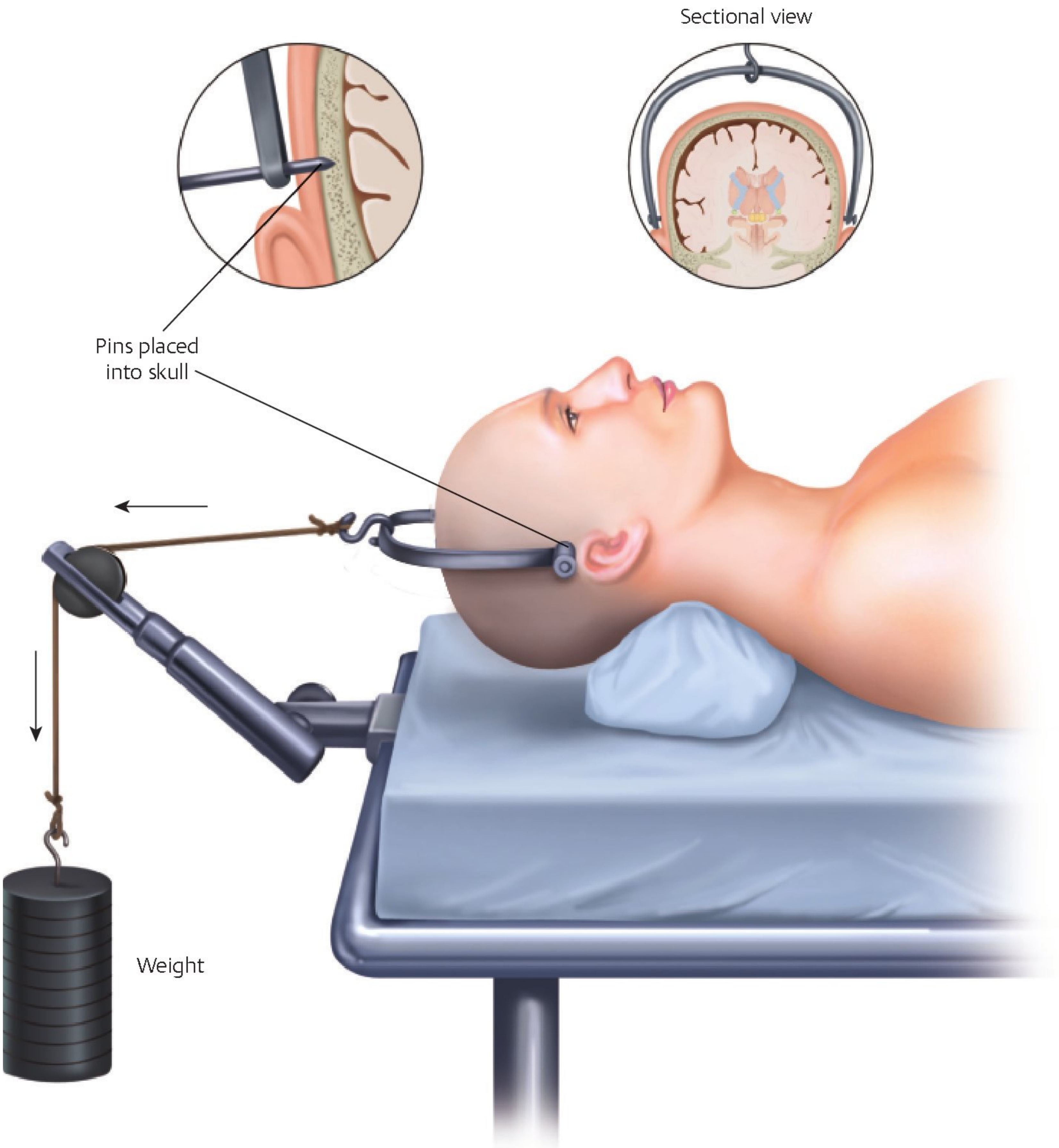

Fig. 10.3 Gardner-Wells tongs in place with traction.

C. C1–C2 subluxation.
   1. More common in children than in adults.
   2. Common complaints.
      a. Neck pain with evidence of torticollis.
      b. Suboccipital pain.
      c. Limited cervical rotation.

D. May be associated with odontoid or atlas fractures.
   1. Rupture of transverse ligament.
      a. Anterior atlantodens interval (ADI).
         (1) Three to 5 mm indicate rupture of the transverse and alar ligaments.
         (2) Seven to 8 mm indicate complete ligamentous disruption.
         (3) Greater than 10 mm causes spinal cord compression.

b. Treatment.
   (1) If instability is 3 to 5 mm, halo or rigid orthosis is used for 2 to 3 months.
   (2) If instability is > 5 mm, then fuse C1–C2.

2. Atlantoaxial rotatory fixation.
   a. The head is tilted toward the side of fixation and the chin and C2 spinous process are pointed toward the opposite direction.

E. Fracture of atlas (C1) **(Fig. 10.4).**
   1. Axial loading disrupts the atlantal ring.
   2. Neurological injury is rare because of the wide spinal canal.
      a. Cranial nerve injuries may be observed.

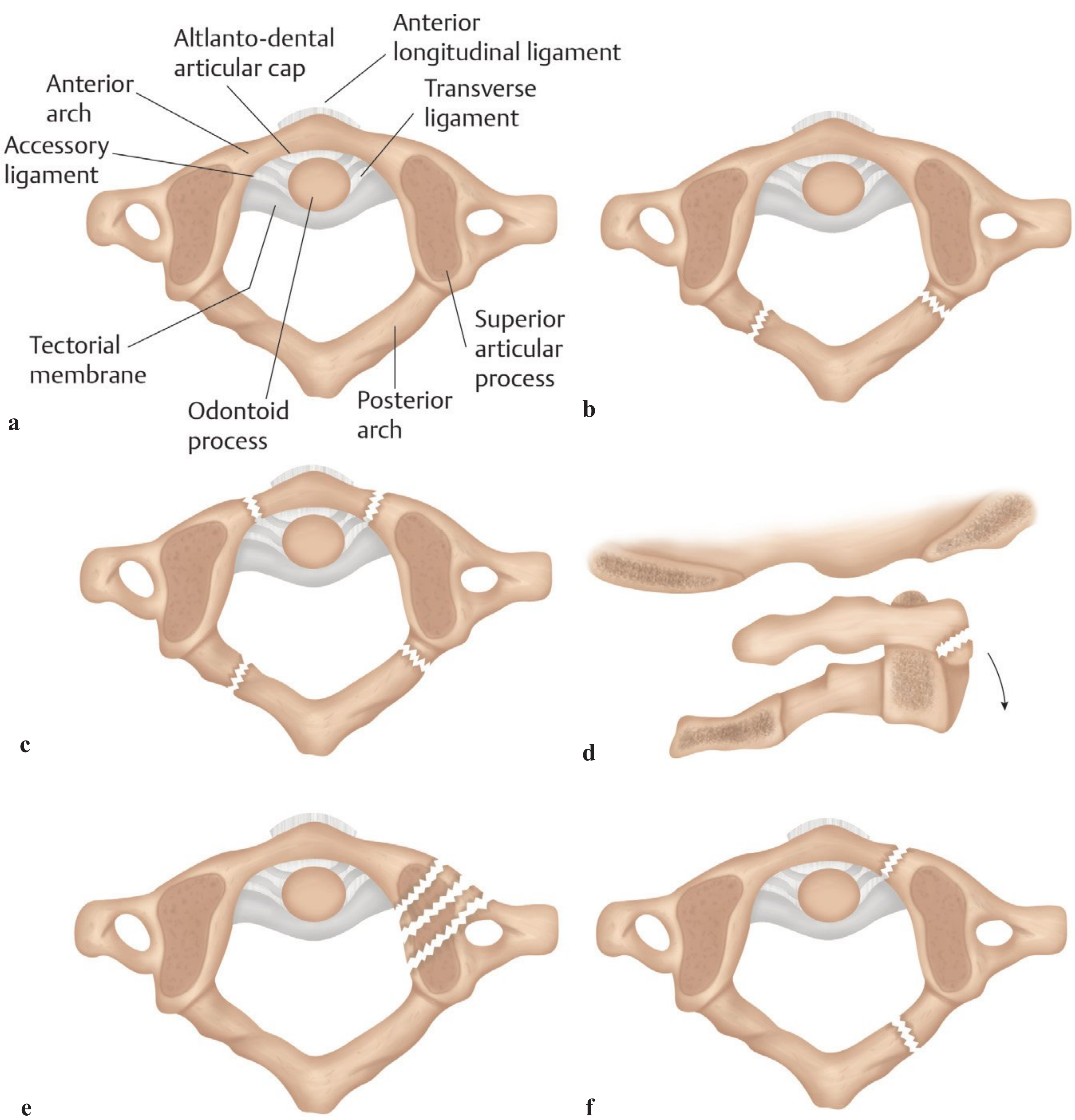

Fig. 10.4 Levine and Edwards classification of atlas fractures. **(a)** Normal anatomy. **(b)** Posterior arch fracture. **(c)** Classic Jefferson's or burst fracture. **(d)** Avulsion fracture of the anterior arch. **(e)** Lateral mass fracture. **(f)** Unilateral ring fracture.

3. An anteroposterior open-mouth odontoid view to assess the lateral masses of C1 relative to the lateral mass of C2:
   a. Greater than 6.9 mm widening of the lateral mass indicates a transverse ligamentous rupture.
      (1) Allow atlas fractures to heal first with halo immobilization for 2 to 3 months.
      (2) C1–C2 fusion may be done if instability is > 5 mm.
4. Treatment:
   a. Cervical orthosis for 3 months if nondisplaced.
   b. Halo vest for 3 months if displaced or delayed union.
   c. Posterior C1–C2 fusion for nonunion.

F. Fractures of the odontoid **(Fig. 10.5).**

1. Type I.
   a. Rare avulsion fractures of the tip.
      (1) Stable and treatment is with a cervical collar.
2. Type II.
   a. Fractures at the base of the odontoid:
      (1) Anterior displacement (flexion injury).
         (a) More common than posterior displacement (extension injury).

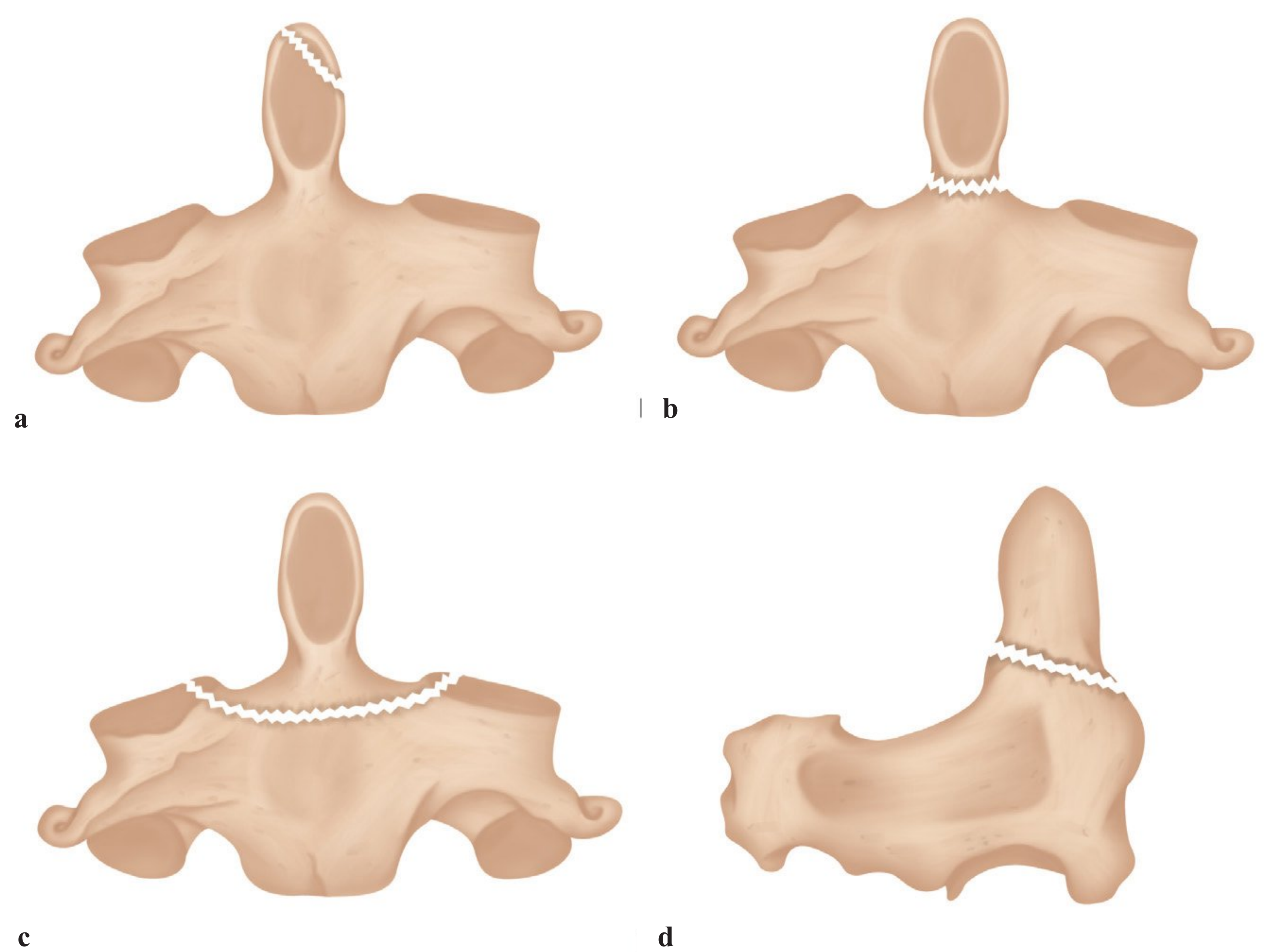

Fig. 10.5 Anderson and D'Alonzo classification of odontoid fractures. **(a)** Odontoid tip avulsion. **(b)** Fracture at the base of the dens. **(c)** Fracture within the body of C2. **(d)** Lateral view of a fracture at the base of the dens.

b. The nonunion rate is 20 to 80%
   (1) Risk factors for nonunion.
      (a) Age over 50 years.
      (b) More than 4 mm displacement.
      (c) Posterior angulation.
c. Treatment:
   (1) Halo traction for reduction of fracture.
      (a) If acceptable reduction is achieved, then a halo jacket is applied for 12 weeks and a cervical collar for 6 weeks.
      (b) Indications for C1–C2 fusion:
         i. Delayed union or nonunion.
         ii. Redisplacement following halo placement.
         iii. Fractures at high risk for nonunion (> 4 mm displacement, older patient).
      (c) Treatment options for odontoid fractures associated with C1 ring fracture:
         i. Consider posterior C1–C2 screw fixation or anterior odontoid screw fixation.
         ii. Halo initially to let C1 heal, then C1–C2 fusion if C2 nonunion develops.

3. Type III.
   a. Fracture through the body.
      (1) Nondisplaced.
         (a) Treat with a cervical orthosis or halo.
      (2) Displaced.
         (a) Halo jacket for 3 months.

G. Traumatic spondylolisthesis of the axis—hangman's fracture **(Fig. 10.6).**
   1. Mechanism.
      a. Acute hyperextension injury.
   2. Types (**Table 10.1).**
   3. Treatment.
      a. Type I.
         (1) Halo jacket for 12 weeks.
      b. Type II.
         (1) Cervical traction to reduce displacement and allow callus formation.
         (2) Halo jacket for 10 to 12 weeks.
      c. Type IIA.
         (1) Reduction in extension followed by halo vest immobilization.
      d. Type III or late instability/nonunion.
         (1) Anterior C2–C3 fusion.
         (2) Posterior screw fixation (C2–C3 plating).

H. Subaxial cervical trauma.
   1. Allen–Ferguson classification of subaxial trauma.
      a. Classification is based on the mechanism of injury.
      b. Provides a biomechanical understanding of the injury pattern.
      c. Classification (Allen) **(Fig. 10.4, Fig. 10.5, Fig. 10.6, Fig. 10.7,** and **Fig. 10.8) (Table 10.2).**

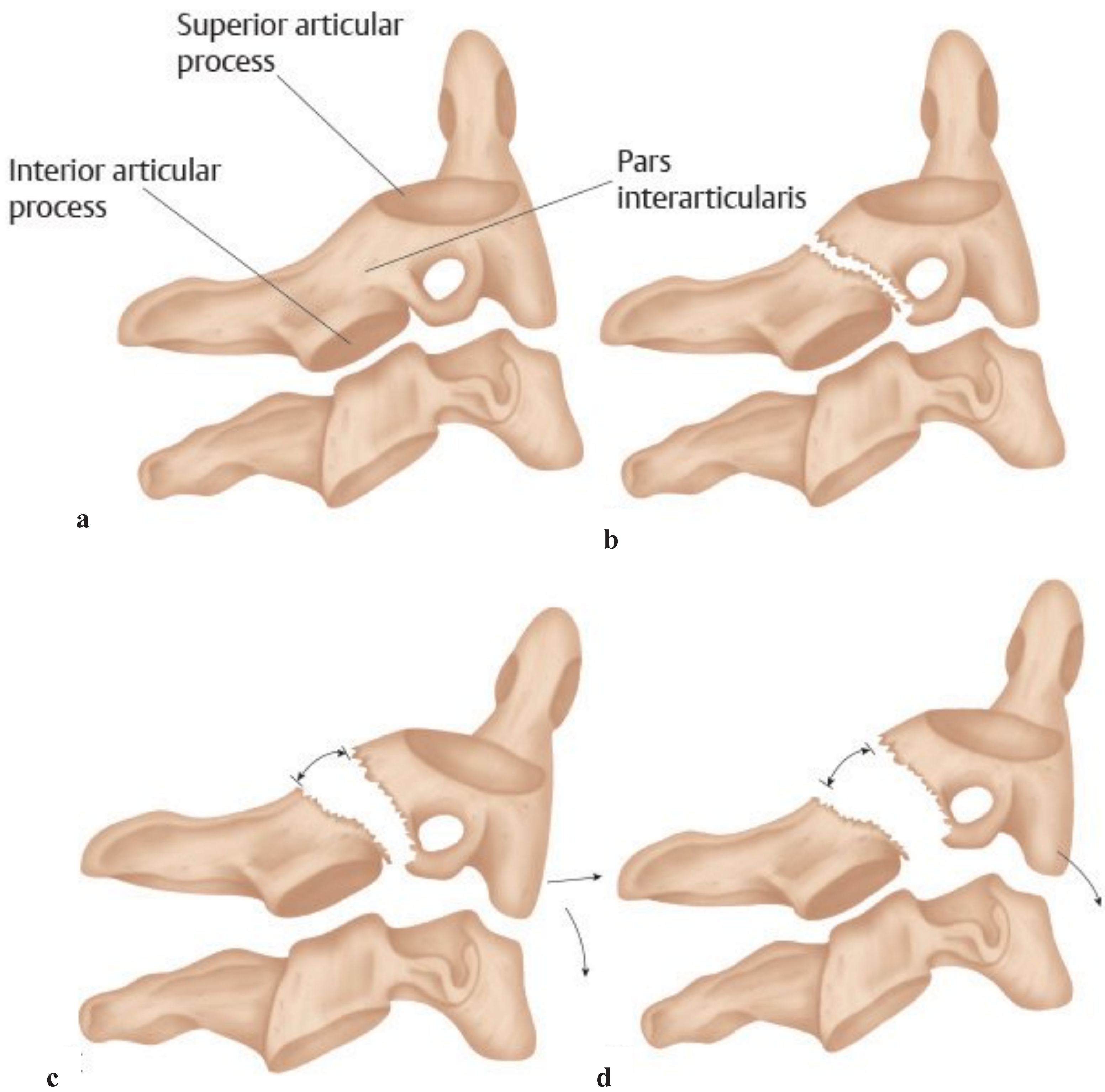

Fig. 10.6 Effendin classification of hangman's fracture. **(a)** Normal anatomy. **(b)** Type I nondisplaced (< 3 mm of displacement). **(c)** Type IIa—no translation, significant angulation, anterior longitudinal ligament intact, posterior longitudinal ligament and C2–C3 disk space disrupted. **(d)** Type III—anterior C2–C3 displacement, angulation, and facet dislocation.

2. Subaxial cervical spine injury classification (SLIC) system for cervical spine injuries **(Table 10.3).**
   a. Classification is based on the morphology of the fracture, the stability of the posterior ligamentous complex, and the patient's neurological status.
   b. A score < 4 can be treated nonoperatively, whereas a score > 4 requires surgical intervention. A score of 4 is surgeon's choice.

IV. Treatment of specific injuries.

A. Unilateral or bilateral facet dislocations.

1. Cervical traction to reduce dislocation as soon as possible followed by cervical arthrodesis.
   a. In an awake, cooperative patient, MRI can be performed after attempted reduction.
      (1) If the patient is obtunded/intoxicated, obtain a prereduction MRI to rule out an associated disk herniation.

Table 10.1 Levine classification of traumatic spondylolisthesis of the axis (hangman's fracture)

Type	Displacement and angulation	Other characteristics
I	Minimal displacement < 3 mm without angulation	– Bilateral pars fracture – C2–C3 ligamentous structures are intact
IA	Minimal displacement with little or no angulation	– Fracture lines extend through foramen transversarum on computed tomography (possible vertebral artery injury)
II	Significant displacement > 3 mm and angulation > 11°	– Most common fracture subtype – Posterior longitudinal ligament is disrupted – Anterior longitudinal ligament is usually intact
IIA	Minimal displacement (< 3 mm) and angulation > 11°	– Additional widening of posterior part of C2–C3 disk space
III	Associated C2–C3 facet dislocation	– Unilateral or bilateral facet dislocation

Steven C. Kirshblum, Stephen P. Burns, Fin Biering-Sorensen, et al. International standards for neurological classification of spinal cord injury (Revised 2011). The Journal of Spinal Cord Medicine, 2011; 34(6): 535-546. Used with permission.

B. Facet dislocations with herniated disk.
   1. Closed reduction may be dangerous, producing further neurological deficits.
      a. Anterior diskectomy and fusion may be performed first.
         (1) Cervical fusion and stabilization may be accomplished by either anterior grafting and plate fixation or anterior grafting and posterior fixation.
         (2) If radiculopathy is present with an associated facet fracture, removal of the fracture fragment is recommended during posterior fusion.
C. Fractures of C3–C7 vertebral bodies.
   1. Wedge compression fractures.
      a. Cervical collar for 6 weeks if posterior elements are intact.
      b. Halo jacket immobilization if there is significant compression or posterior elements are disrupted.
      c. Posterior fusion may be necessary in cases with severe kyphotic angulation or in cases of late instability **(Fig. 10.7** and **Fig. 10.8)**.
   2. Teardrop fractures.
      a. Always unstable due to significant bony comminution and associated disruption of the anterior ligamentous complex.
      b. Posterior ligaments are frequently injured as well.
      c. Treatment: posterior fusion.
   3. Fractures of the spinous process (clay-shoveler's fracture).
      a. Stable flexion injury with avulsion fracture.
      b. Treatment.
         (1) Cervical collar.

Fig. 10.7 Anteroposterior cervical spine radiograph demonstrating a unilateral C5–C6 facet dislocation with the spinous process of C5 rotated toward the right.

Fig. 10.8 Magnetic resonance imaging of a 32-year-old man who sustained a bilateral C5–C6 facet dislocation with significant cord compromise.

Table 10.2 Mechanistic classification (Allen–Ferguson)

Category	Findings
Compression-flexion	Failure of the anterior column via compression Posterior column distraction
Vertical compression	Burst fractures
Distraction-flexion	Facet dislocations
Compression-extension	Posterior column compression Anterior column distraction
Lateral-flexion	Uncommon
Distraction-extension	Associated with disk space widening and/or retrolisthesis

Table 10.3 The subaxial cervical spine injury classification system (SLIC) for cervical spine injuries

Morphology	
No abnormality	0
Compression	1
Burst	+1 = 2
Distraction (e.g., facet dislocation, unstable teardrop or advanced-stage flexion compression injury)	4
Diskoligamentous complex	
Intact	0
Indeterminate (e.g., isolated interspinous widening, magnetic resonance imaging signal change only)	1
Disrupted (e.g., widening of disk space, facet perch or dislocation)	2
Neurological status	
Intact	0
Root injury	1
Complete cord injury	2
Incomplete cord injury	3
Continuous cord compression in setting of neurological deficit (neuro modifier)	+1

4. Soft tissue injury.
   a. Extension-acceleration “whiplash”.
      (1) Involves anterior longitudinal ligament, anterior musculature, and intervertebral disk.
      (2) Symptoms.
         (a) Pain in the neck; referred pain to the head, shoulder, and arm; dysphagia; ocular symptoms; dizziness; and temporal mandibular problems.
         (b) Treatment.
            i. Brace acutely and may need surgery later if cervical spondylosis is problematic.
   b. Flexion-deceleration injury.
      (1) Muscle strain and greater auricular nerve stretch, interspinous ligament, capsular tear, posterior longitudinal ligament, and posterior aspect of the disk.
      (2) Treatment.
         (a) Conservative first and if unstable by White’s criteria and symptomatic, then posterior wiring and fusion.

Suggested Reading

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11 Thoracolumbar Spine Fractures

11.1 General Considerations

I. Introduction.
  A. The thoracolumbar spine is the most common site of spinal injuries.
    1. Transition from less mobile spine segments in the thorax to relatively hypermobile spinal segments in the lumbar spine.
  B. Most injuries occur in males (age 15–29) and are most commonly the result of a motor vehicle accident (50%) or a fall from > 6 feet (25%).
  C. Most injuries occur between T11 and L1 (52%).
    1. L1–L5 (32%).
    2. T1–T10 (16%).
  D. Associated injuries are common and may occur in up to 50% of patients, usually the result of a distractive force.
    1. Intra-abdominal bleeding from liver and splenic injuries.
    2. Arterial or venous vessel disruption.
    3. Pulmonary injuries.
      a. Hemothorax.
      b. Pulmonary contusion.
    4. Noncontiguous spine injuries (5%).

II. Patient evaluation.
  A. General assessment.
    1. Respiratory, cardiothoracic, abdominal, urological evaluation.
    2. Head/cervical spine status.
  B. Neurological assessment.
    1. Frankel scale and American Spinal Injury Association (ASIA) motor index.
    2. Spinal cord, conus, cauda equina, root injuries.
  C. Radiological evaluation.
    1. All patients who have injuries suspicious for spinal trauma should undergo plain radiographic imaging (anteroposterior or lateral) of all vertebral levels.
    2. Plain X-ray is the initial screening modality:
      a. Computed tomography.
        (1) Best imaging modality for evaluation of the middle column, especially with sagittal reconstructions.
      b. Magnetic resonance imaging.
        (1) Most useful in evaluating the spinal cord and soft tissue disruption (disk/ligamentous injury).

III. Classification methods.
  A. Spine stability (Denis classification) **(Fig. 11.1).**
    1. Three columns:
      a. Anterior column.

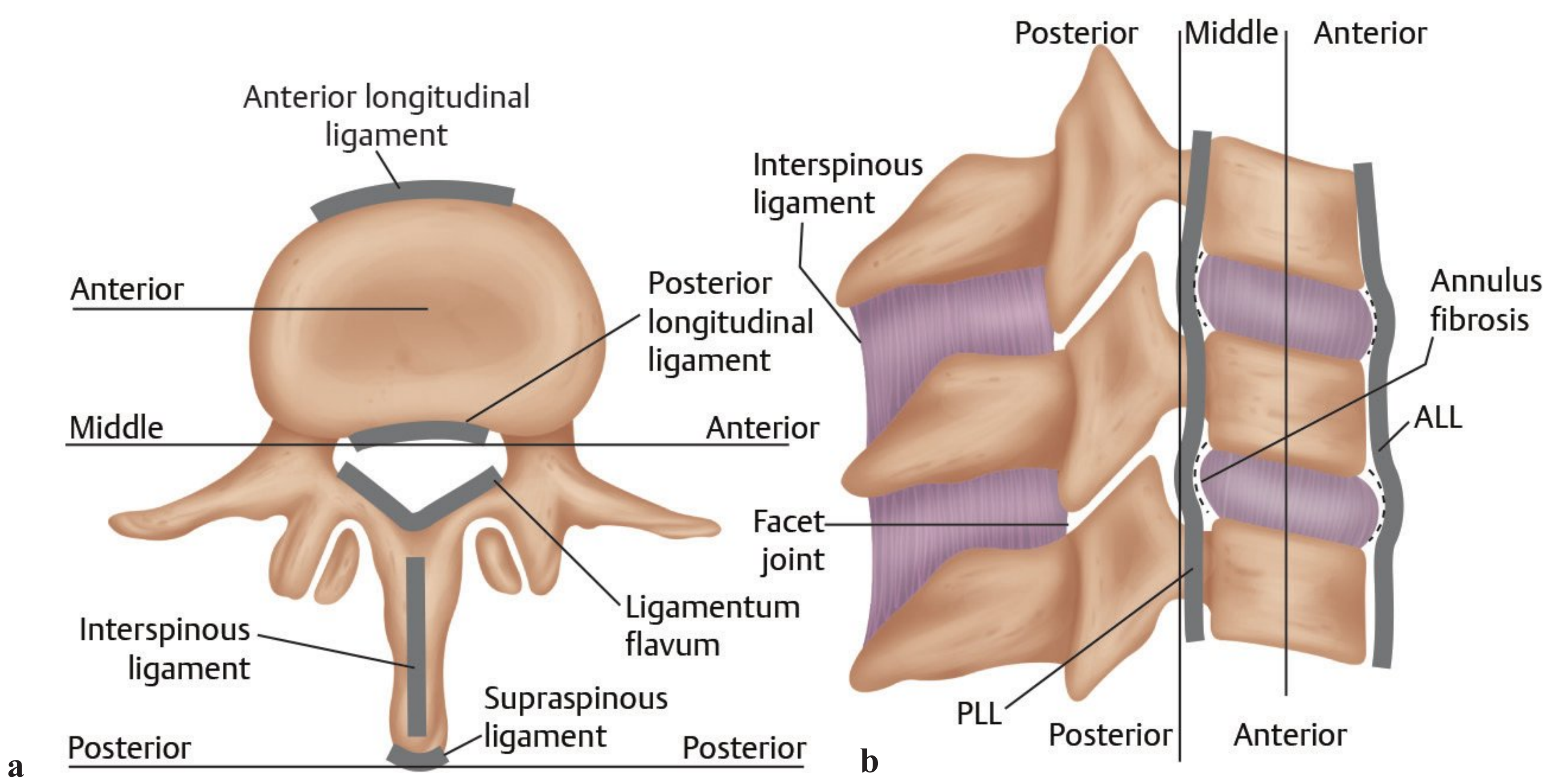

Fig. 11.1 **(a,b)** The three columns of the spine as described by Denis.

(1) Anterior longitudinal ligament, anterior annulus fibrosus, and anterior half of the vertebral body.

b. Middle column.

(1) Posterior longitudinal ligament, posterior annulus fibrosis, posterior half of the vertebral body.

c. Posterior column.

(1) Pedicles, facet joints, lamina, spinous processes, and interspinous and supraspinous ligaments.

2. Clinical application:

a. Instability is defined as a disruption of two or more of the three spinal columns.

b. If the middle column is disrupted, then the spine is unstable except in the following:

(1) Thoracic vertebrae above T8 (stability provided by the ribs).

(2) L4–L5 where the posterior column is intact (significant weight bearing through the posterior column because of lordosis).

(3) Distraction injuries where fractures occur through the cancellous bone.

3. Stability is not "black and white"; it is a gray zone (Denis).

a. Stable fractures.

(1) Transverse process.

(2) Spinous process.

(3) Articular process.

(4) Pars interarticularis.

(5) Compression fractures.

B. Thoracolumbar injury classification and severity (TLICS) score.

1. Operative treatment is based on three factors **(Table 11.1).**

a. Morphology of the fracture.

(1) Compression.

(2) Burst.

Table 11.1 Components of the Thoracolumbar Injury Classification and Severity Score

Morphology of the fracture
1. Compression
2. Burst
3. Translational/rotational
4. Distraction
Posterior ligamentous complex
1. Intact
2. Indeterminate/suspected
3. Injured
Neurological status
1. Intact
2. Root injury
3. Cord
a. Incomplete
b. Complete
4. Cauda equina syndrome

(3) Translational/rotational.
(4) Distraction.

b. Posterior ligamentous complex.
   (1) Intact.
   (2) Indeterminate/suspected.
   (3) Injured.

c. Neurological status.
   (1) Intact.
   (2) Root injury.
   (3) Cord.
      (a) Incomplete.
      (b) Complete.
   (4) Cauda equina syndrome.

IV. Treatment.
  A. General considerations.
    1. Multifactorial.
      a. Neurological status of the patient.
        (1) An anterior approach is preferred in patients who are neurologically compromised and have anterior-based compression (i.e., retropulsed vertebral body fragments).
      b. Posterior ligamentous complex integrity.
        (1) If disrupted, requires reconstruction of the posterior tension band.
      c. Fracture morphology.
    2. Timing of surgery.
      a. Immediate operative intervention theoretically gives the best chance for reduction and neural recovery but is not clinically proven.
      b. Indications for acute surgical intervention:
        (1) Progressive neurological deficit.
        (2) Irreducible dislocations.
        (3) Open or contaminated injuries.
      c. Early surgery (2–3 days):
        (1) Easier reduction and decompression via ligamentotaxis.
        (2) Earlier mobilization of the patient.
        (3) Potential operative complications.
          (a) Transportation issues regarding patients with severe spinal instability.
      d. Late surgery (7–10 days):
        (1) Arguments for delayed intervention include providing a chance for the spinal cord to recover from trauma and edema.
          (a) Skeletal traction and closed reduction and motorized rotating bed for patients with deformity or dislocations.
    3. External immobilization.
      a. Above T5: cervicothoracic–lumbosacral orthosis.
      b. T6–L4: Jewett hyperextension or thoracolumbar–sacral orthosis (TLSO) braces.
      c. L5–S1: pantaloon cast.
  B. Neurological status and stability.
    1. Neurologically intact with a stable spine.
      a. Common in compression fractures, seat belt–type injuries and burst fractures of the lower lumbar spine.
      b. Orthosis or body cast.
    2. Neurologically intact with an unstable spine.
      a. Common in burst fractures and severe compression fractures.
      b. Operative stabilization to prevent neurological loss.
      c. Early rehabilitation is preferred.
    3. Neurologically compromised and spine unstable.
      a. Burst fractures or fracture dislocations of the lower thoracic spine and lumbar spine **(Fig. 11.2).**
      b. Anterior decompression with or without posterior stabilization.

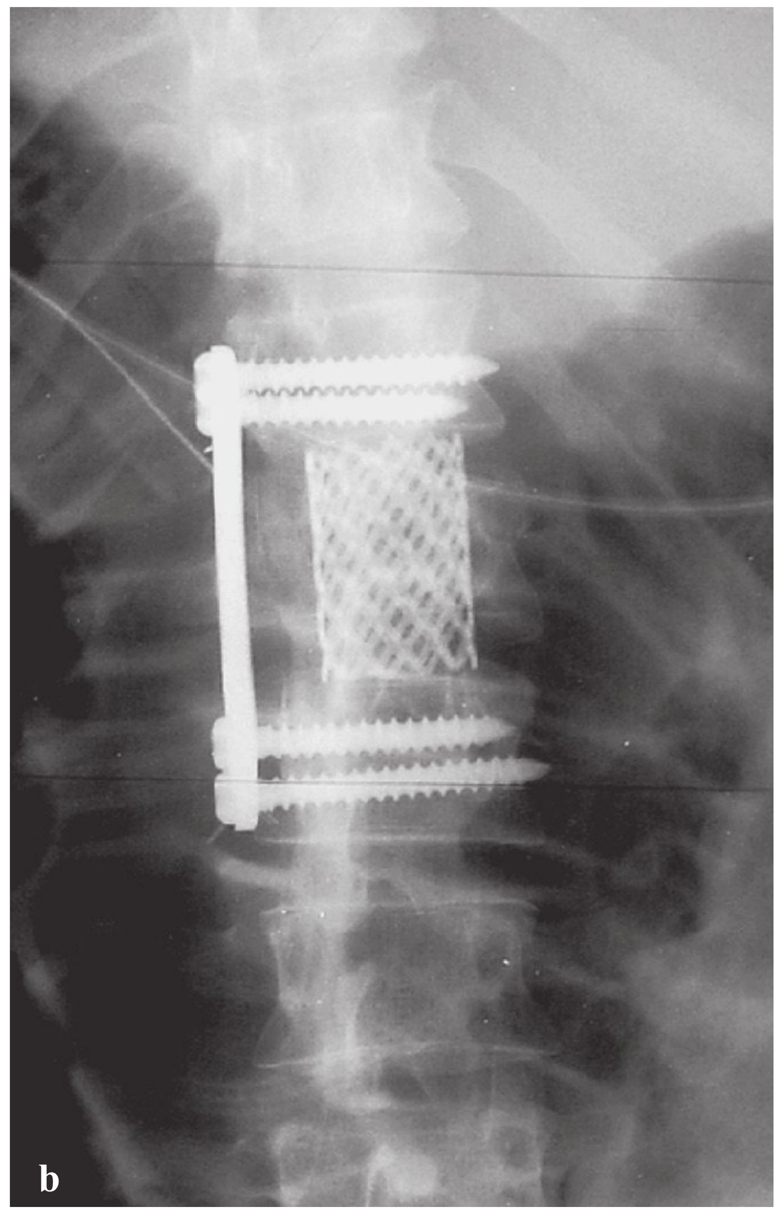

Fig. 11.2 (a) Sagittal magnetic resonance imaging (MRI) of a 34-year-old man who sustained a burst fracture to the T12 vertebral body. Note the retropulsion of the posterior vertebral body with compression of the anterior thecal sac. **(b)** A postoperative anteroposterior radiograph following an anterior L1 corpectomy and fusion using a titanium Harms mesh cage and an anterior plate and screw construct.

C. Specific fracture treatment.
   1. Compression-flexion injuries.
      a. Anterior column injury alone usually does not cause neurological deficits.
         (1) Factors suggestive of instability and disruption of the posterior ligamentous complex.
            (a) Greater than 50% collapse.
            (b) Thirty-degree angulation.
            (c) Thirty-degree kyphosis.
      b. Treatment:
         (1) Conservative if anterior column failure alone.
            (a) Hyperextension orthosis.
         (2) Middle column failure.
            (a) Operative intervention if unstable.
   2. Distraction flexion injuries (seat belt injury) **(Fig. 11.3).**
      a. Bony Chance fracture without subluxation or dislocation.
         (1) Hyperextension body cast.

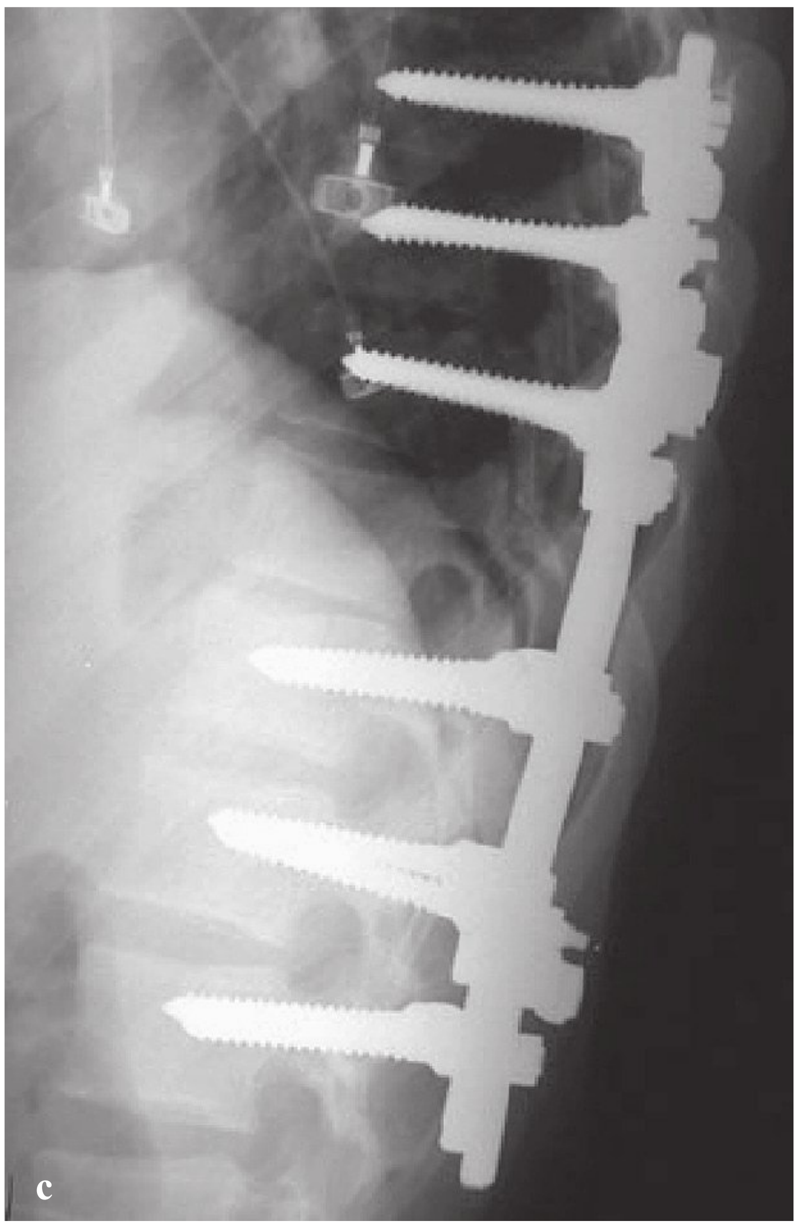

Fig. 11.3 **(a)** A sagittal computed tomographic reconstruction of a fracture-dislocation of the thoracolumbar spine demonstrating marked vertebral body displacement and canal narrowing. **(b)** Sagittal magnetic resonance imaging of the thoracolumbar spine of the same patient demonstrating marked canal narrowing. Note the draping of the spinal cord over the posterosuperior edge of the caudal thoracic vertebrae. **(c)** A postoperative lateral radiograph of the same patient demonstrating reduction of the spinal deformity followed by a fusion and stabilization with segmental pedicle screw anchors spanning three levels above and below the level of injury.

b. Ligamentous flexion-distraction injury.
   (1) Posteriorly instrumented arthrodesis.
   (2) Injury is through soft tissue structures with a decreased likelihood of healing.
3. Torsional flexion injuries (fracture-dislocations) **(Fig. 11.4).**
   a. Frequently cause complete paraplegia.
   b. Surgical posterior stabilization and fusion are necessary for early rehabilitation.
4. Vertical compression injuries (burst fractures).
   a. Neurological involvement is common secondary to a retropulsed posterior vertebral body fragment.
   b. Treatment for patients without neurological compromise:
      (1) Conservative treatment with acute bed rest and TLSO orthosis may be offered for patients who have no neurological deficits with minimal deformity.
      (2) Vertebral body cement augmentation (kyphoplasty):
         (a) Recommended for osteoporosis-related fractures without neurological compromise.
         (b) Efficacy of these procedures remains controversial.
         (c) May help restore vertebral body height and alleviate pain.
         (d) Risks: cement leak, adjacent body fractures.
   c. Surgical options for neurologically compromised patients:
      (1) An anterior approach is preferred in certain situations.
         (a) Large retropulsed anterior fragment with significant neurological deficits.
         (b) Intervention delayed > 2 weeks.
      (2) A posterior approach is preferred if the compressive fragment (i.e., fracture lamina or infolded ligamentum flavum) is located posterolaterally, with minor neurological deficits.
         (a) A posterior approach is required for fracture-dislocations and in situations associated with traumatic dural tears.
         (b) Both anterior and posterior approaches may be needed in three-column injuries.
            i. Particularly in situations with neurological compromise and posterior ligamentous injuries.

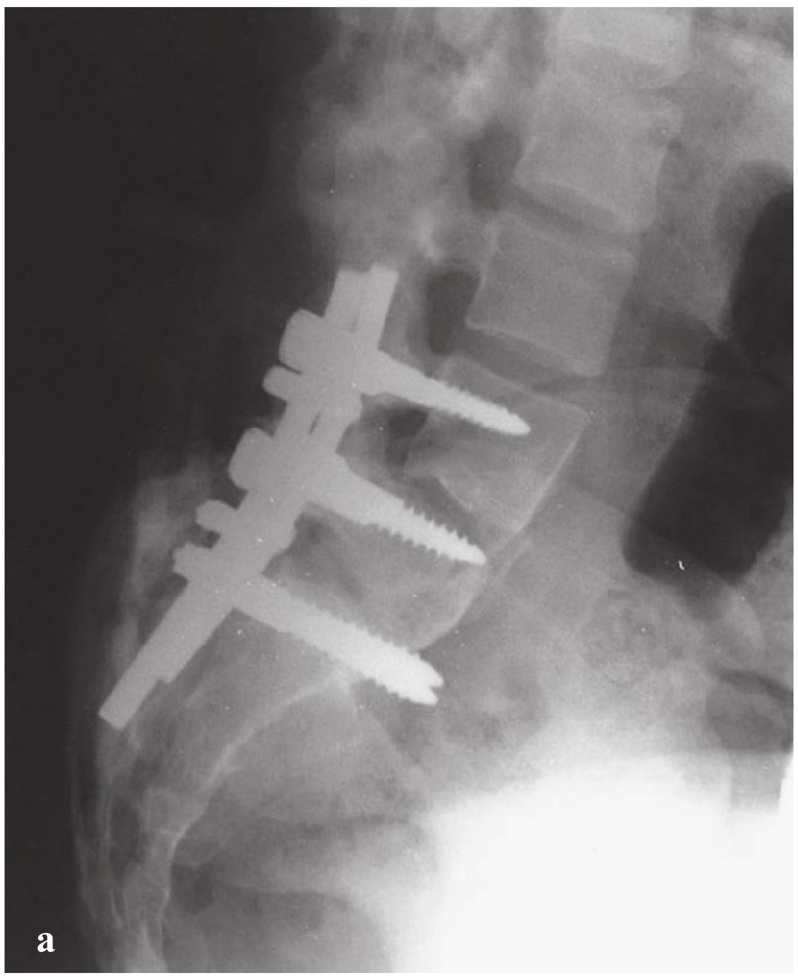

Fig. 11.4 **(a)** Sagittal T2-weighted magnetic resonance imaging demonstrating a complete fracture-dislocations through the L5 vertebral body due to a distraction-extension injury mechanism. **(b)** A lateral postoperative radiograph of the lumbosacral spine following reduction of the fracture-displacement and stabilization with pedicle screw instrumentation from L4 to S1.

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12 Spinal Rehabilitation and Disability Evaluation

12.1 Rehabilitation of Back and Neck Pain

I. General considerations.
   A. It is estimated that 80% of the general population suffers at least one disabling episode of back or neck pain in their lifetime.
   B. The majority of patients recover without sequelae.
   C. Many patients who do not recover have no clear pathophysiological diagnosis as a basis for continued pain.
   D. Advances in medical technology have contributed to increased costs associated with the evaluation and treatment of persistent back or neck pain.
   E. It is well established that if disability involves litigation (worker's compensation or personal injury), the outcome is less predictable and the problem is less likely to resolve quickly.

II. Treatment of acute neck or low back pain.
   A. Evaluate for a neurological deficit that may necessitate urgent surgical intervention.
   B. Diagnostic imaging is rarely necessary for acute episodes when no trauma is involved. (The exception to this is an occupational injury where medicolegal issues may need to be addressed at a later time.)
   C. The initial goal is pain relief, because these patients often experience severe, disabling pain and spasm.
   D. Appropriate medication includes nonsteroidal anti-inflammatory drugs (NSAIDs), short-term narcotic analgesics (2–4 days only), and muscle relaxants if spasm is a significant physical finding.
   E. Nonpharmacological pain-reducing modalities include heat, ice, rest, positioning, relaxation, and massage.
   F. The most important early intervention is patient education.
   G. Patients with low back or neck pain embrace many myths that may interfere with recovery. The extra time spent on initial education will pay off in terms of a quicker recovery.
   H. Essential components of patient education include the following:
      1. Likely causes of current condition based on a discussion of simple anatomy (models or pictures are often helpful).
      2. Natural history of the injury.
      3. Benefits of maintaining activity with a brief discussion of specific aggravating activities.
      4. Instruction on progressive exercise programs emphasizing both endurance (cardiovascular conditioning) and strength (isometrics and core stabilization).
      5. Once the acute episode has subsided, education on health promotion and lifestyle changes (weight loss, maintenance exercise, smoking cessation, and stress management) may help prevent recurrences or decrease their frequency and severity.

6. Formal physical therapy may be instituted initially or reserved for later if necessary, depending on the individual's preinjury physical conditioning, motivation, and physical demands of daily activities, including job requirements.

III. Rehabilitation of chronic injury.

A. General considerations.

1. Controversy exists as to when acute pain becomes chronic.
2. It is generally accepted that there is a lack of progress toward recovery, despite assumed continued tissue healing, and no specific pathophysiological diagnosis.
3. Chronic pain involves psychosocial as well as physical components **(Fig. 12.1)**.
4. The goal is to improve function and maximize quality of life, not necessarily to eliminate the pain.

B. Role of psychiatric disease.

1. The frequency of depression, anxiety, and schizophrenia among surgical candidates for spine surgery is increasing.
2. Depression is more likely to be encountered among female surgical candidates, whereas schizophrenia is more common among male patients.
   a. Dementia has no gender preference among spine surgery candidates.

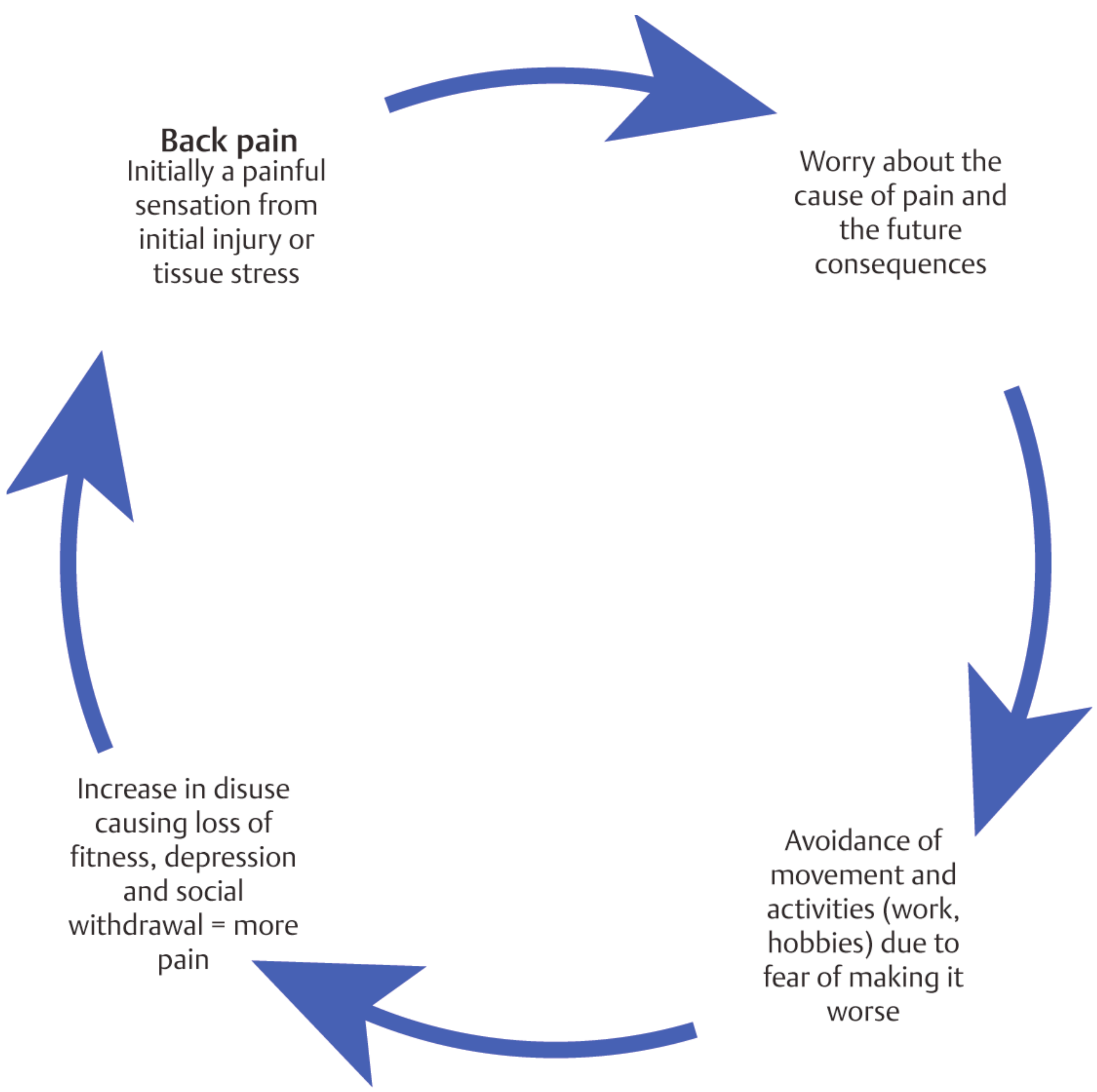

Fig. 12.1 The psychological effects of chronic pain.

3. There is considerable evidence that suggests that psychiatric comorbidities are an independent risk factor for adverse in-hospital events following spine surgery.
    a. Dementia is associated with a higher risk of perioperative mortality.
4. Recent meta-analyses recommend, with weak evidence, that patients with chronic low back pain who have psychiatric comorbidities should be treated with nonoperative management.

C. Functional restoration programs.
1. Repeated measures of factors related to the injury or disability, such as strength, endurance, and coordination.
2. Use functional capacity measures as a basis of monitoring progress and goal attainment.
3. Outcomes vary and may be secondarily measured by subscores of improved quality-of-life scales, symptom interference scales, objective strength or endurance measures, and decreased use of medication.
4. Factors found to contribute to poorer outcomes (in terms of return to work only) include high pretreatment self-reported scores for pain, depression, and disability; relatively short work history prior to injury; previous surgical failure; and job dissatisfaction.
5. Patients with chronic neck pain may benefit from similar programs.
6. Formal functional restoration programs should be reserved for carefully selected patients due to the high cost and the variability of outcomes.

D. Pain centers.
1. Multidisciplinary programs are generally based on a medical model.
2. Use a variety of passive and/or invasive pain-reducing modalities, including heat, cold, ultrasound, massage, transcutaneous electrical nerve stimulator (TENS) unit, acupuncture, and injections.
3. Incorporate concepts of behavior modification, such as biofeedback, stress management, coping strategies, and relaxation techniques.
4. Address occupational issues, such workplace modification.
5. This may be the most appropriate environment for detoxification from narcotics when this is necessary.
6. Treatment of chronic pain with invasive methods, such as implantable morphine pump, spinal cord stimulator, sympathectomy, and rhizotomy, is controversial.

E. Injections for treatment of back and neck pain.
1. Medications, such as local anesthetics and steroids, are injected into various locations within the vertebral column for both diagnostic and therapeutic reasons.
2. Indicated as an adjunct modality for temporary pain relief so that exercises and rehabilitation can ensue.
3. May be used in response to a specific diagnosis established by imaging or based on a clinical diagnosis based on history and physical alone.
4. The placebo effect is always possible and difficult to prove.
5. Types of therapeutic injections:
    a. Local trigger point injection can be used in patients with muscular, tendinous, or myofascial pain with marked point tenderness.

b. An epidural steroid is indicated for persistent radiculopathy despite NSAIDs in patients with a herniated disk or spinal stenosis.
c. A nerve root block is indicated for diagnosis of foraminal nerve root compression and for relief of radicular symptoms due to foraminal stenosis.
d. Facet joint injection is indicated for symptomatic facet joint pain syndrome.
   (1) Most patients should have painful extension and spinal imaging showing facet joint arthritis.
   (2) Facet joint syndrome is difficult to diagnose, and injection is unpredictable for pain relief.
e. Intradiskal steroid injection is controversial and may be indicated for patients with diskogenic back pain.

12.2 Impairment and Disability Evaluation

I. General considerations.
   A. Physicians are called upon to determine physical impairment to satisfy insurers, employers, and government agencies.
   B. Impairment ratings are often required in worker's compensation, personal injury, or applications for Social Security disability.
   C. Definitions:
      1. *Impairment* is a functional or anatomical loss.
         a. It results from a medical condition and can be temporary or permanent.
      2. *Disability* is the extent to which a person can continue to function with the impairment considering the occupational demands, training, education, and other psychosocial factors.
      3. *Whole person* refers to the person prior to the illness or injury.
         a. Describes the person as a sum of all parts (both anatomical and psychological. Impairment is determined by the loss of one part as compared with the whole, thus the term *partial disability*.
         b. The exact implementation of this principle varies state by state.
      4. *Healing period* is defined as the time when progress is being made toward improvement of pain or function and treatment continues.
      5. *Healing plateau* is defined as the time when treatment is for maintenance, and further significant changes in status are not anticipated; this is also known as maximum medical improvement.
II. Determination of disability and impairment.
   A. Four components for determining impairment and disability.
      1. Determine causality:
         a. Requires an opinion about the relationship between the circumstances that caused the impairment and the resultant impairment itself.
      2. Apportionment:
         a. Determine the role of "preexisting conditions," such as degenerative joint disease, in determining impairment from an injury.
         b. The American Medical Association has five types of apportionment:
            (1) An occupational disorder aggravated by a supervening occupational disorder.

(2) An occupational disorder aggravated by a supervening other occupational condition arising out of or in the course of employment by the same employer.

(3) An occupational disorder aggravated by a supervening other occupational condition arising in the course of employment by a different employer.

(4) An occupational disorder aggravated by a preexisting nonoccupational condition.

(5) An occupational disorder aggravating a preexisting nonoccupational condition.

3. Determine end of healing:
   a. Often an arbitrary period based on the individual clinician's practice, patient population, local culture, and experience (Maximum Medical Improvement).
4. Assign impairment rating.
   a. May be temporary or permanent.
   b. Encompasses both residual symptoms as well as permanent restrictions.
   c. Ultimately should be based on an objective assessment of the patient's ability to perform certain functional tasks, such as sitting, lifting, gripping, and pushing.
   d. Formal evaluation methods are evolving.
      (1) None are yet proven to be more objective or reliable than the treating physician's completion of the work capacity evaluation form based on his or her estimation of the patient's current abilities.

III. Spinal impairment rating.

A. There are many rating systems available, including ones developed by the American Medical Association and the American Academy of Orthopaedic Surgeons.

B. Elements for determining impairment include the following:
1. Range of motion.
   a. Measured with goniometer or inclinometer.
2. Neurological impairment.
   a. Includes sensory changes, loss of reflex, and loss of motor function (weakness to paralysis).
3. Specific diagnosis or surgical intervention.
4. Psychosocial impairment.
   a. Includes such items as activities of daily living, social functioning, concentration, and coping.

C. The clinician will benefit from finding one rating system and using it consistently so as to become most proficient.

D. Treating physicians must not view impairment ratings as reflections of treatment failure.

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13 Biochemical Aspects of Intervertebral Disk Degeneration

13.1 General Considerations

I. Intervertebral disk.
  A. Cells.
    1. Notochordal cells:
      a. Present embryologically and disappear by adult life.
    2. Chondrocyte-like cells.
    3. Likely originate from chondrocytes in the cartilaginous end plate.
    4. No significant cell turnover.
    5. These cells undergo apoptosis with aging and disk degeneration.
  B. Gross structures (from peripheral to central) **(Fig. 13.1).**
    1. Outer fibrous annulus fibrosus.
      a. Primarily collagen fibrils that are aligned in oblique layers.
      b. Limited vascular and nerve supply.
      c. Sinuvertebral nerve runs posteriorly.
      d. Sympathetic fibers run anteriorly.
    2. Inner annulus fibrosus.
      a. Fibrocartilaginous tissue.
        (1) Gradually blends with the nucleus pulposus.
        (2) Posterolaterally the annulus is thinner and has more disorganized collagen and a greater proportion of vertical fibers.
          (a) Weakest part of annulus.
          (b) Contributes to a greater proportion of disk herniations.
    3. Transition zone.
      a. Thin zone of fibrous tissue between the inner annulus and the nucleus pulposus.

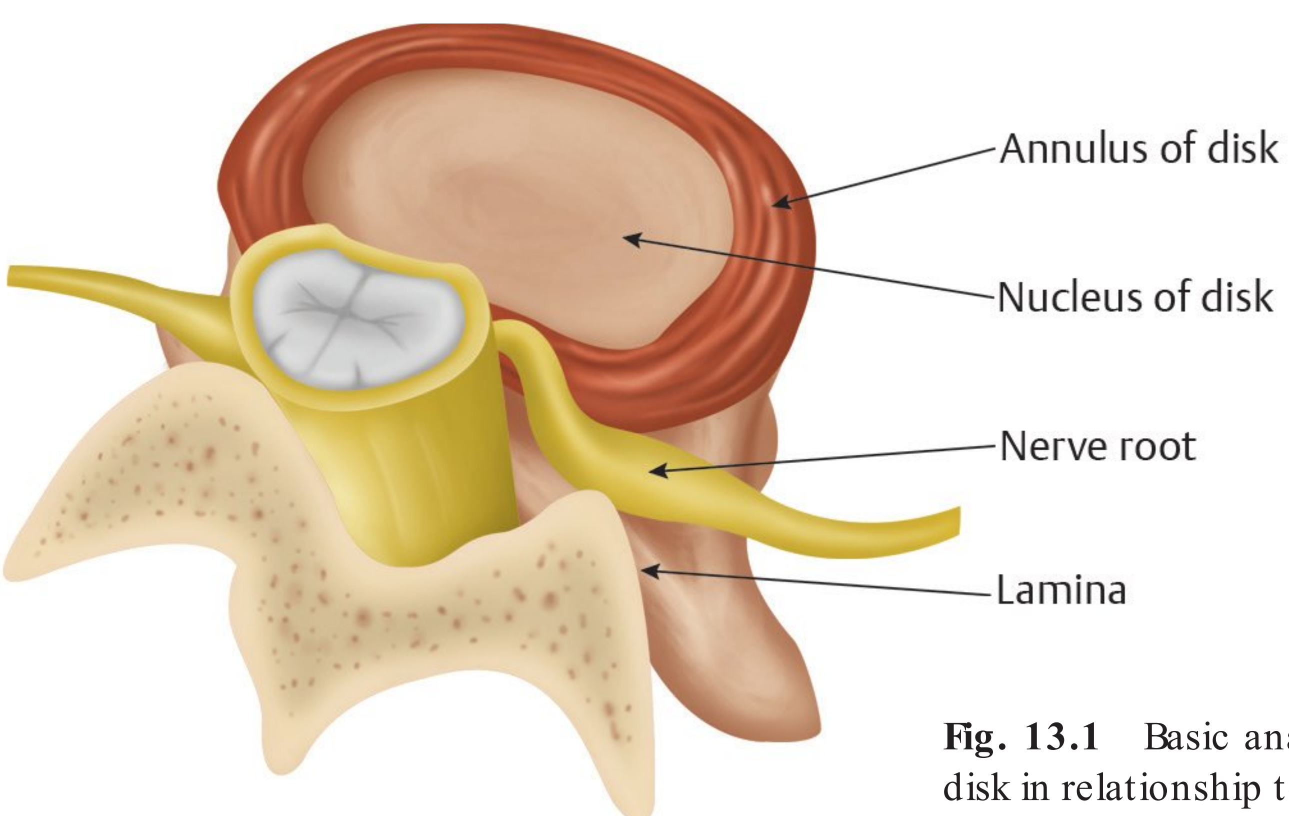

Fig. 13.1 Basic anatomical structures of the disk in relationship to the neural elements.

4. Nucleus pulposus.

C. Matrices **(Table 13.1).**

1. Collagens.

D. Disk degeneration.

1. Collagen synthesis and content increase in the nucleus.
2. Decreased concentration of cross-linking in the annulus.
3. Decreased water content.

E. Proteoglycans (PGs).

1. PG aggregates' constituents.
   a. Central hyaluronan filament.
   b. Link proteins attach multiple glycosaminoglycan molecules.
   c. Large PGs.
      (1) Aggrecan.
         (a) Similar to articular cartilage.
         (b) Half the size of PGs found in cartilage.
         (c) Higher keratin sulfate:chondroitin sulfate ratio.
         (d) Higher molecular weights of keratin sulfate.
         (e) Increased hyaluronan content.
         (f) Important in water retention.
         (g) Provides compressive strength.
   d. Small PGs.
      (1) Biglycan, decorin, lumican, fibromodulin.
      (2) Involved in organization of collagen and fibrillin formation.
      (3) PG content and synthesis vary depending on age, region, and degeneration.
         (a) PG activity in a normal adult annulus is approximately one-third lower than in a young nucleus.
         (b) Synthetic activity is greatest in the inner annulus.

Table 13.1 Components of the intervertebral disk

Annulus (70%)
1. Predominantly type I
2. Type I, II, III, V, VI, IX, XI
3. Predominantly type II
4. Type II, VI, IX, and XI provide tensile strength
Collagen cross-linking by covalent bonds via modification of lysine/hydroxylysine residues
Highest concentration of cross-linking in nucleus
Nucleus (20%)
1. Predominantly type II
2. Type II, VI, IX, and XI

F. Aging and degeneration **(Fig. 13.2).**
   1. The keratin sulfate:chondroitin sulfate ratio increases with age.
   2. Nonaggregating PGs or PGs that cannot bind to hyaluronic acid increase.

G. Homeostasis of intervertebral disk metabolism.
   1. Anabolic enzymes, growth factors, and cytokines.
      a. Growth factors.
         (1) Transforming growth factor-b (TGF-b), b fibroblast growth factor (FGF), insulin-like growth factor-1 (IGF-1), platelet-derived growth factor (PDGF), bone morphogenetic protein-2 (BMP-2), BMP-4, BMP-7.
         (2) IGF-1, epidermal growth factor (EGF), FGF, and TGF-b stimulate matrix synthesis.
         (3) FGF promotes proliferation of chondrocytes in degenerative disks.
         (4) IGF-1 stimulates proteoglycan synthesis in the nucleus.
         (5) BMPs, such as BMP-2, BMP-7, and latent membrane protein (LMP)-1, have been shown to upregulate PG synthesis in vitro and in vivo.
   2. Catabolic enzymes and cytokines.
      a. Enzymatic degradation of the matrix:
         (1) Matrix metalloproteinases (MMPs):
            (a) Collagenase, gelatinase, stromelysin.
         (2) Proinflammatory cytokines and free radicals increase in degenerative disks.
            (a) Nitric oxide, prostaglandin E2 (PGE2), and interleukin (IL)-6 are increased in degenerated disks.
            (b) Phospholipase A2, tumor necrosis factor alpha (TNF-a), and IL-1 are increased in herniated disks and radiculopathy.
      b. Cytokine blockers such as IL-1, TNF blockers, and tissue inhibitors of metalloproteinase can upregulate PG synthesis by blocking catabolic processes.

H. Nutrition by diffusion through the end plates **(Fig. 13.3).**
   1. Blood supply to the end plates and outer annulus decreases with aging.
      a. Lactate concentration increases.
      b. pH decreases.
      c. Cellular metabolism is affected with decreased nutrition **(Fig. 13.4)**.

I. Biologic strategies for intervertebral disk (IVD) degeneration repair or regeneration.
   1. Growth factors.
      a. BMPs.
      b. Blocking cytokine pathways.
   2. Therapeutic gene transfer.
      a. Viral.
      b. Nonviral.
   3. Cell transplantation.
      a. IVD cells.
      b. Chondrocytes.
      c. Mesenchymal stem cells.

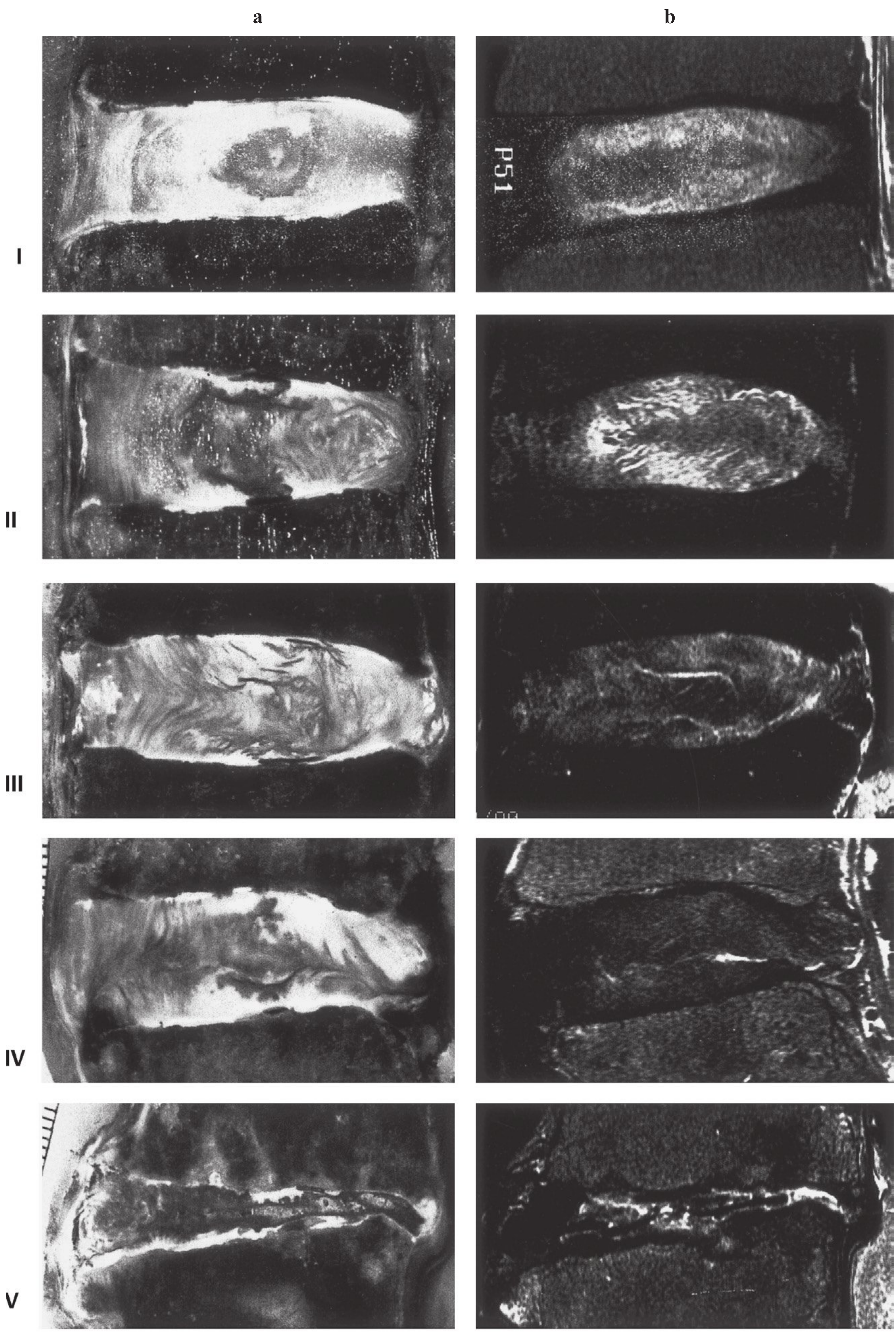

Fig. 13.2 Varying stages of disk degeneration (I–V) categorized via the Thompson grading scale. **(a)** Cadaveric specimens and corresponding **(b)** magnetic resonance images. Grade I represents a healthy disk, and grade V represents a disk with osteophytic end plate changes, loss of disk height, and loss of water content.

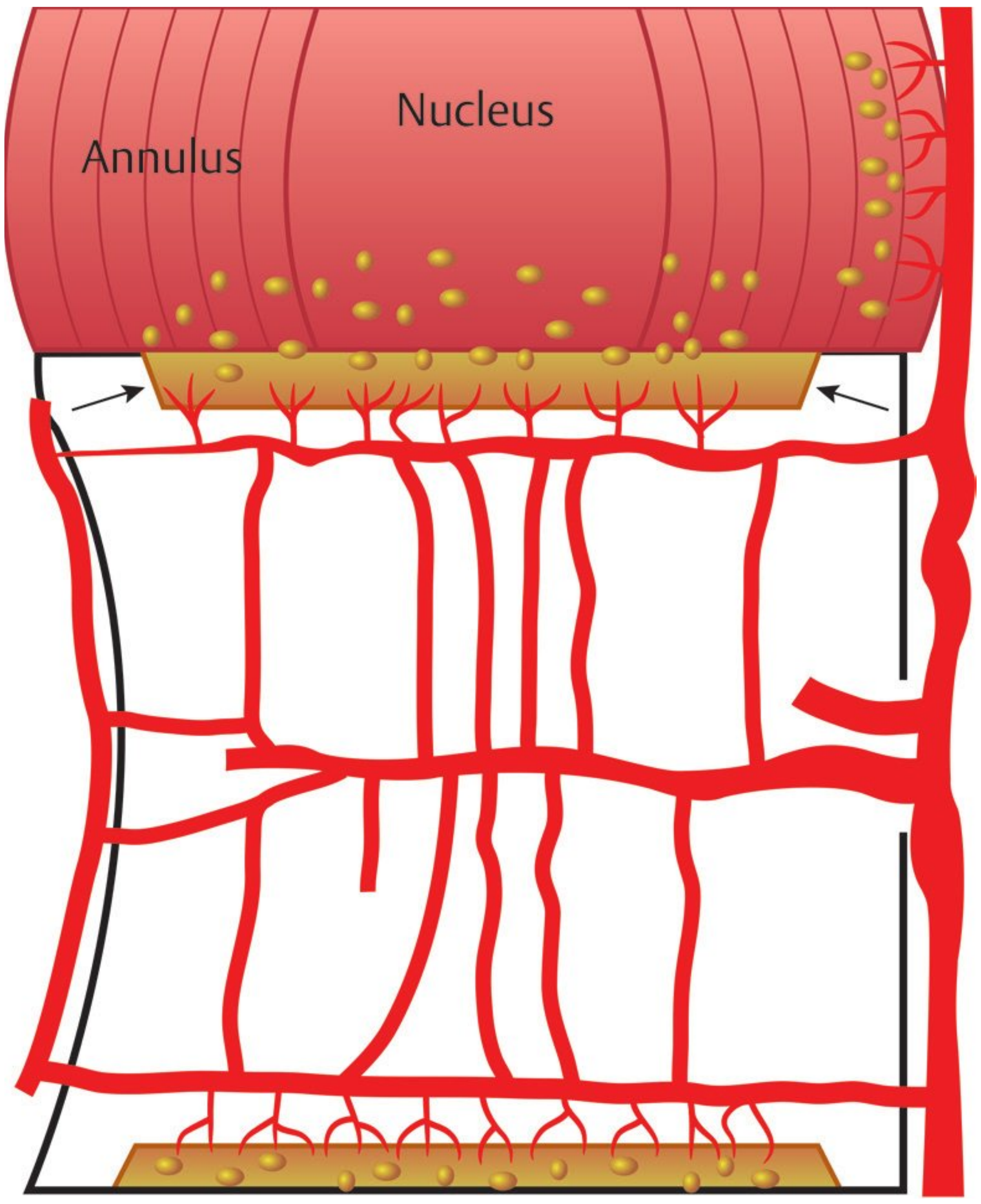

Fig. 13.3 Diagram demonstrating the diffusion of nutrients into the intervertebral disk space.

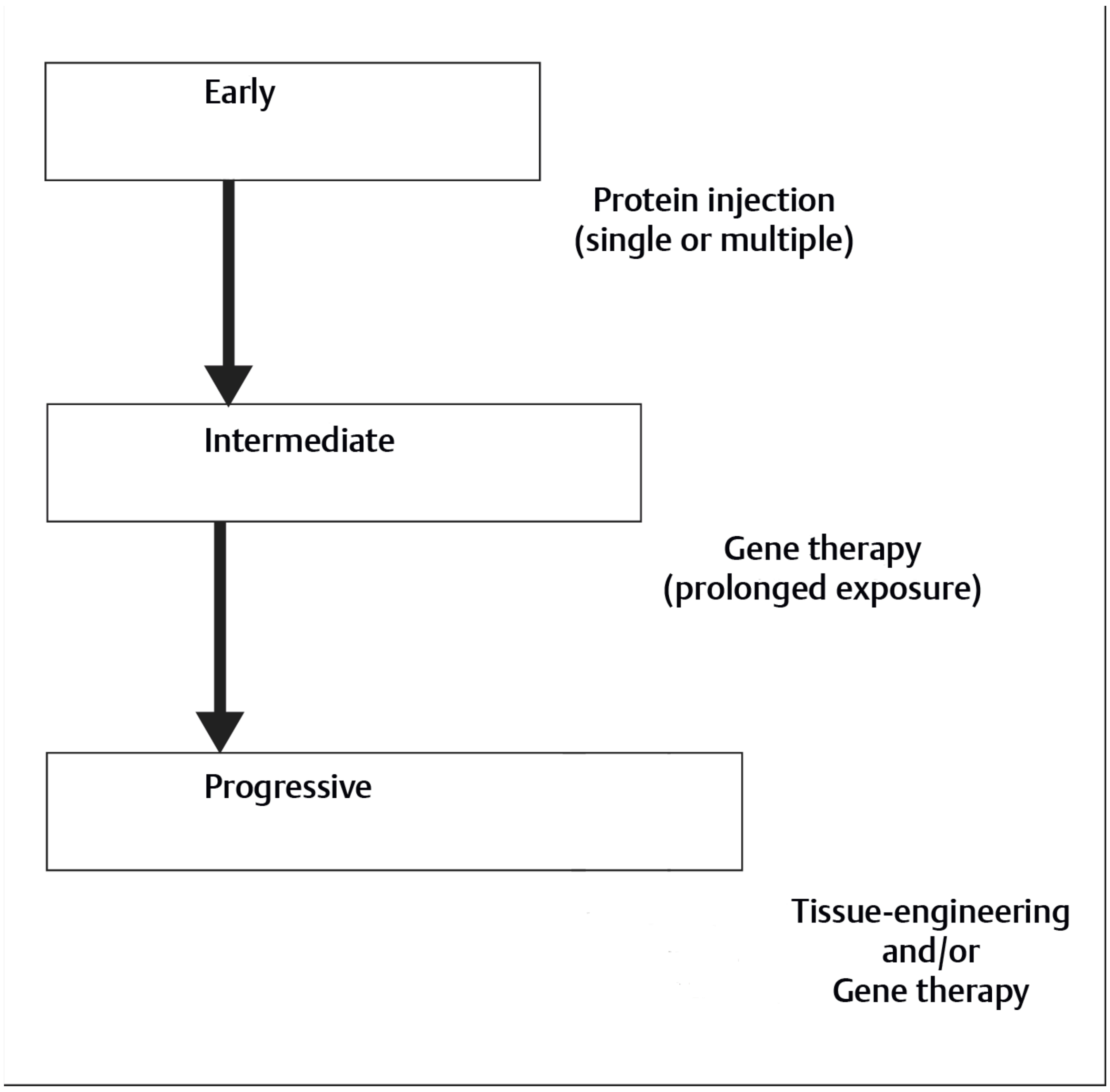

Fig. 13.4 Therapeutic approach for repair of intervertebral disk degeneration.

4. Cells and matrix transplantation.
5. Molecules (peptides or other compounds) that upregulate the anabolic pathway and downregulate inflammatory or pain pathways.

J. Biologic disk regeneration.
1. Increases the synthesis and content of PG and collagen.
2. May improve biomechanical properties of the disk or the motion segment in early stages of disk degeneration.
   a. May not be effective if disk degeneration is severe and the posterior structures are compromised.
   b. Use of growth factor stimulation to restore nucleus height and metabolic function.
3. May not address pain perception.

K. Potential limitations or unanswered questions of growth factor for IVD repair.
1. Duration of therapeutic effect in vivo.
2. Optimal dosage.
3. Optimal delivery system.
   a. Injection.
   b. Prolonged delivery systems.
   c. Carriers.
   d. Multiple proteins.
4. Effect of biomechanical stresses on disk metabolism and influence of growth factors on cells.
5. Efficacy of nonsurgical pain-relieving procedures.
   a. Chemonucleolysis.
      (1) Minimally invasive procedure involving the injection of an enzyme from papayas called chymopapain.
      (2) The enzyme metabolizes disk material that has bulged to reduce nerve root compression.
      (3) In 2003, the Food and Drug Administration discontinued the sale of chymopapain due to growing reports of hemorrhage, pain, paralysis, allergic reactions, fatal anaphylactic shock, and transverse myelitis.

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14 Degenerative Cervical Spine Disorders

14.1 Cervical Degenerative Disease

I. Clinical categories.
  A. Diskogenic axial pain with or without referred pain.
  B. Disk herniation.
    1. Myelopathy.
    2. Radiculopathy.
  C. Cervical spondylosis **(Table 14.1).**
    1. Radiculopathy (foraminal stenosis).
    2. Myelopathy (spinal cord compression).

II. History and examination.
  A. Cervical radiculopathy.
    1. Dermatomal pain distribution **(Fig. 14.1).**
      a. Spurling's sign.
        (1) Pain exacerbated by neck extension and rotation toward the symptomatic side.
      b. Shoulder abduction relief sign.
        (1) Pain ameliorated by shoulder abduction (more often present with soft disk herniations).
    2. Neurological findings (nerve root distribution).
      a. Numbness.
      b. Paresthesias.

Table 14.1 Demographics of cervical spine disease

	Cervical spondylosis	Disk herniation
Age	> 50	< 50
Sex	Male > female	Male = female
Onset	Insidious	Acute
Location of pain	Neck and arm	Arm
Neck stiffness	Yes	No
Weakness	Yes	Yes or no
Myelopathy	More common	Less common
Dermatomal distribution	Multiple	Single

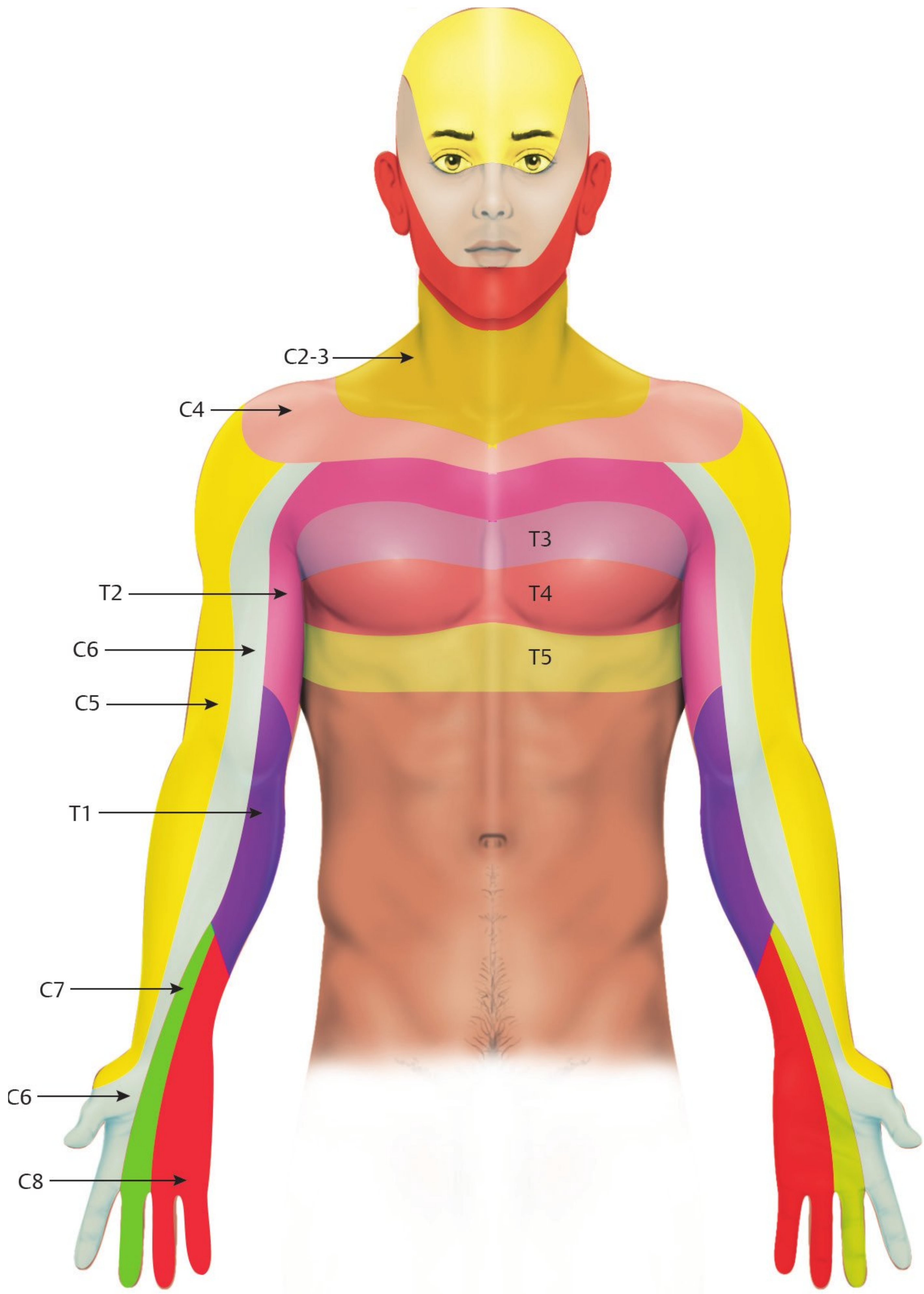

Fig. 14.1 Dermatomal distribution of cervical and thoracic nerve roots.

c. Weakness.
d. Hyporeflexia.

B. Cervical myelopathy.
1. Pain is usually absent.
 a. Discomfort varies from a dull ache to sharp pain.
2. Symptoms:
 a. Wide, ataxic gait pattern.
 b. Poor hand dexterity.
  (1) Buttoning shirt.
  (2) Writing.
  (3) Holding onto a coffee mug.

3. Physical exam findings:
   a. Hyperreflexia.
   b. Positive Hoffman's sign: pressing the dorsal surface of the middle finger elicits a reflex contraction of the thumb and index fingers.
   c. Inverted brachioradialis reflex: brachioradialis tendon tap elicits a reflex flexion of the fingers.
   d. Positive Babinski's sign: lateral stimulation of the plantar surface of the foot elicits toe extension.
   e. Positive Lhermitte's sign: tapping the posterior neck in neck flexion elicits paresthesia down the back and into the extremities.
   f. Myelopathic hand syndrome:
      (1) Thenar atrophy.
      (2) Positive finger escape sign: the patient cannot keep the fourth and fifth digit of the hand in extension.
      (3) Positive grip release test: patient has trouble making a fist and fully extending fingers—normal is 20 times in 10 seconds.
      (4) Dysdiadochokinesia: loss of coordination and dexterity of the hands during rapid movement.

III. Diagnostic imaging **(Fig. 14.2** and **Fig. 14.3).**

A. Plain radiographs.

1. Anteroposterior, lateral, and oblique views.
   a. Overall alignment:
      (1) Patients with spondylosis may have a loss of lordosis or a spondylolisthesis.

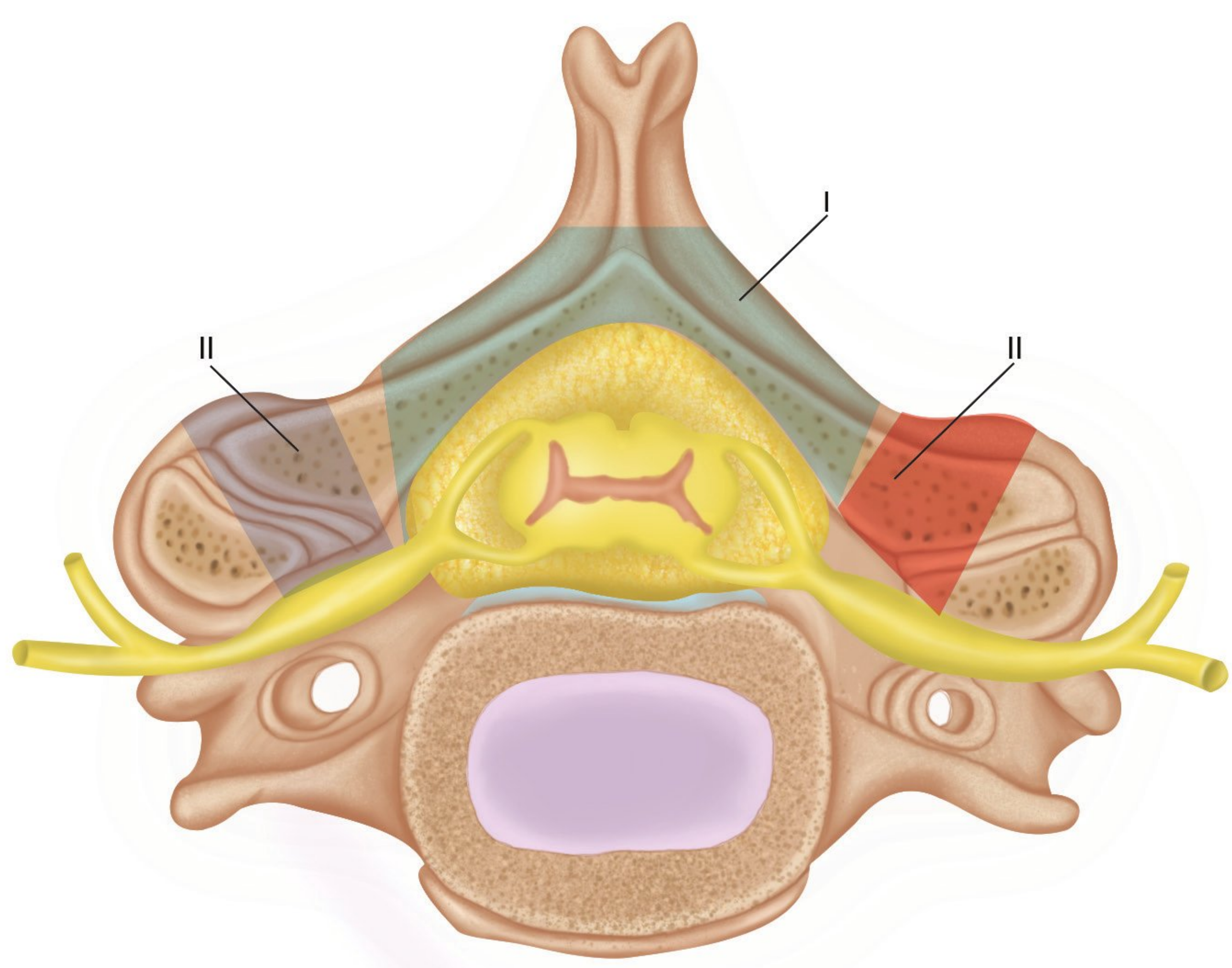

Fig. 14.2 Schematic of an axial cut through the cervical spine. The shaded area in section I represents the lamina that is removed in a laminectomy. Section II represents the bone removed to perform a thorough foraminotomy.

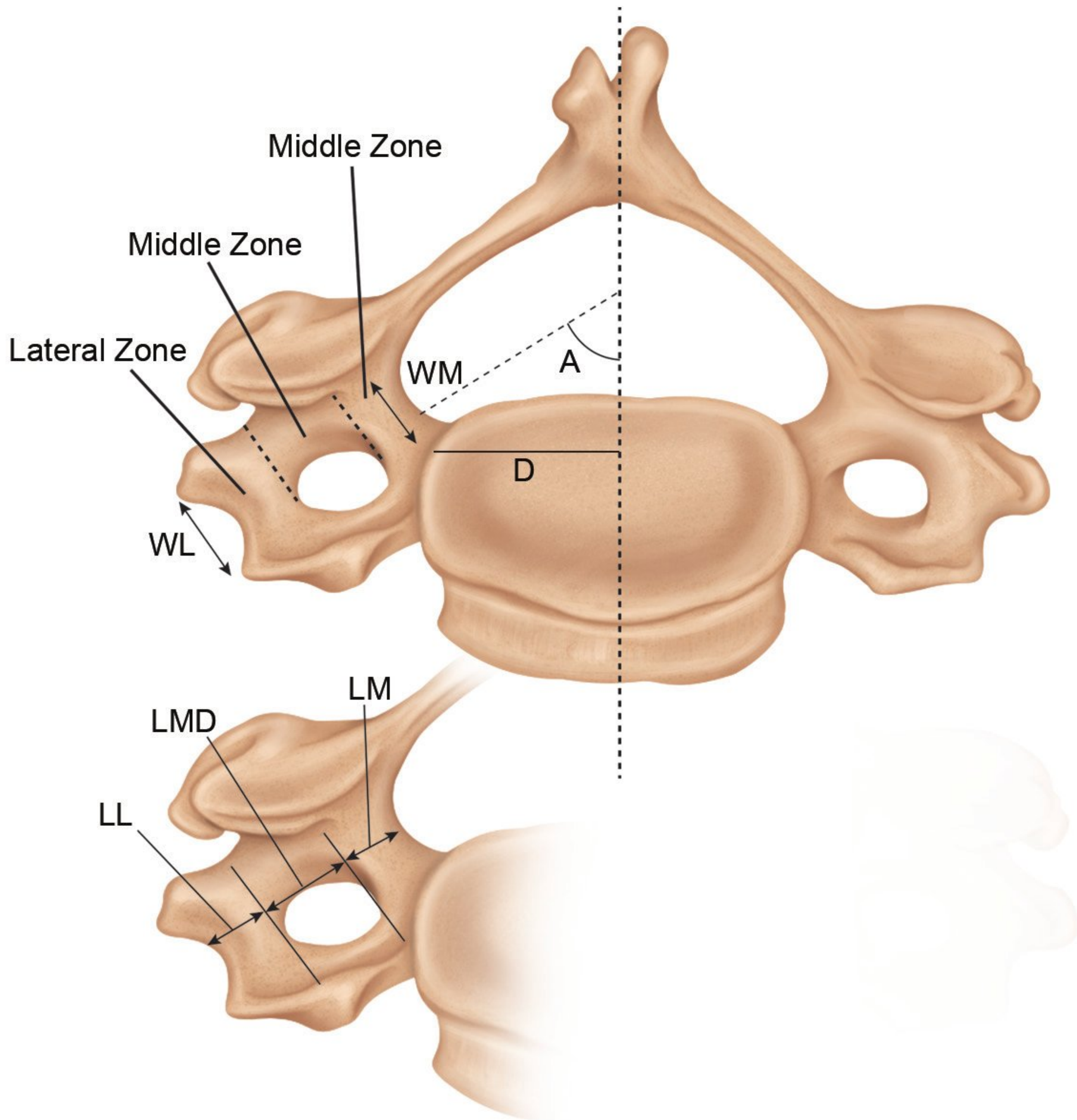

Fig. 14.3 Cross-sectional view of a cervical vertebra. The neuroforamen is divided into three areas: the medial zone, the middle zone, and the lateral zone.

b. Narrowing of the intervertebral disk space.
c. Degenerative changes in the zygapophyseal joints and the presence of osteophytes.
d. Foraminal narrowing is observed on the oblique views **(Fig. 14.4)**.

B. Computed tomography with myelography.
1. Modality of choice for those who cannot undergo magnetic resonance imaging (MRI).
2. Good for postoperative imaging if instrumentation present.
3. Invasive procedure that involves intradural injection of radiopaque dye.

C. MRI **(Fig. 14.5).**
1. Imaging modality of choice for cervical disk disease.
2. Good for evaluating space available for the cord:
   a. Less than 13 mm is relative stenosis.
   b. Less than 10 mm is critical stenosis.
3. Particularly useful to rule out spinal cord lesions, such as syringomyelia, tumors, and myelomalacia.
4. Correlation with clinical symptoms is critical, because the false-positive rate is high.

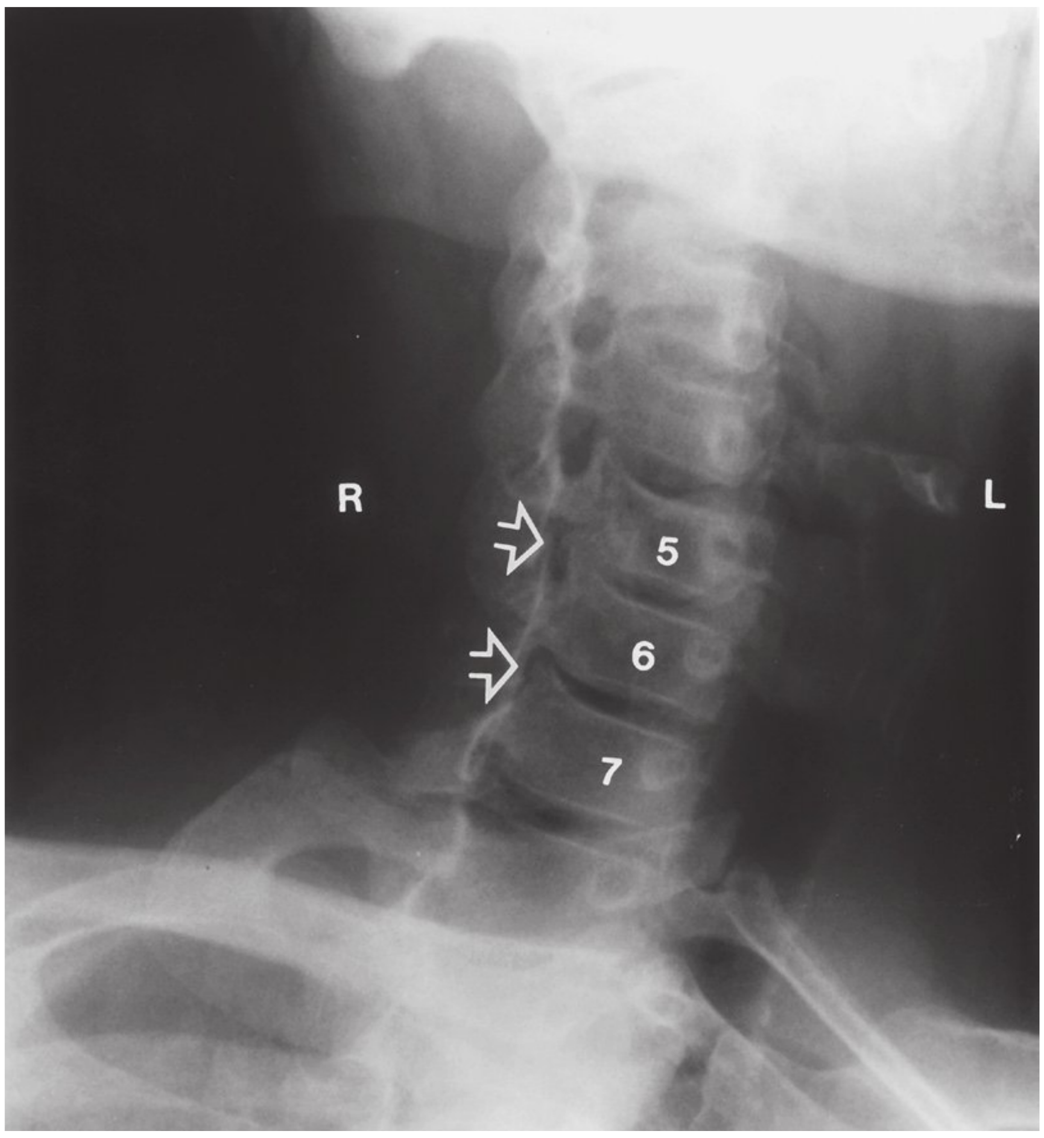

Fig. 14.4 An oblique cervical spine radiograph demonstrating neuroforaminal stenosis secondary to osteophyte formation at the uncinate processes.

IV. Differential diagnosis **(Table 14.2).**

V. Treatment for cervical radiculopathy.

A. Conservative treatment: a 70 to 80%successful outcome is expected with 2 to 3 months of conservative treatment.

1. Acute phase (first 2 weeks).
   a. Nonsteroidal anti-inflammatory medications.
   b. Oral steroids.
   c. Short-term analgesics (limited use of narcotics).
   d. Ice or heat application.
   e. Activity modification.
      (1) Soft collar.
      (2) Home traction.
2. Intermediate healing phase (3–4 weeks).
   a. Stretching and isometric exercises.
   b. Physical therapy:
      (1) Modalities if the patient is not improving.
   c. Epidural steroids may be considered for persistent radicular pain.
3. Rehabilitation phase (> 4 weeks).
   a. Cardiovascular conditioning.
   b. Vigorous strengthening exercise program.

B. Operative indications:

1. Progressive signs of root or cord dysfunction.
2. Failure of conservative treatment in relieving radicular pain or neurological deficits.

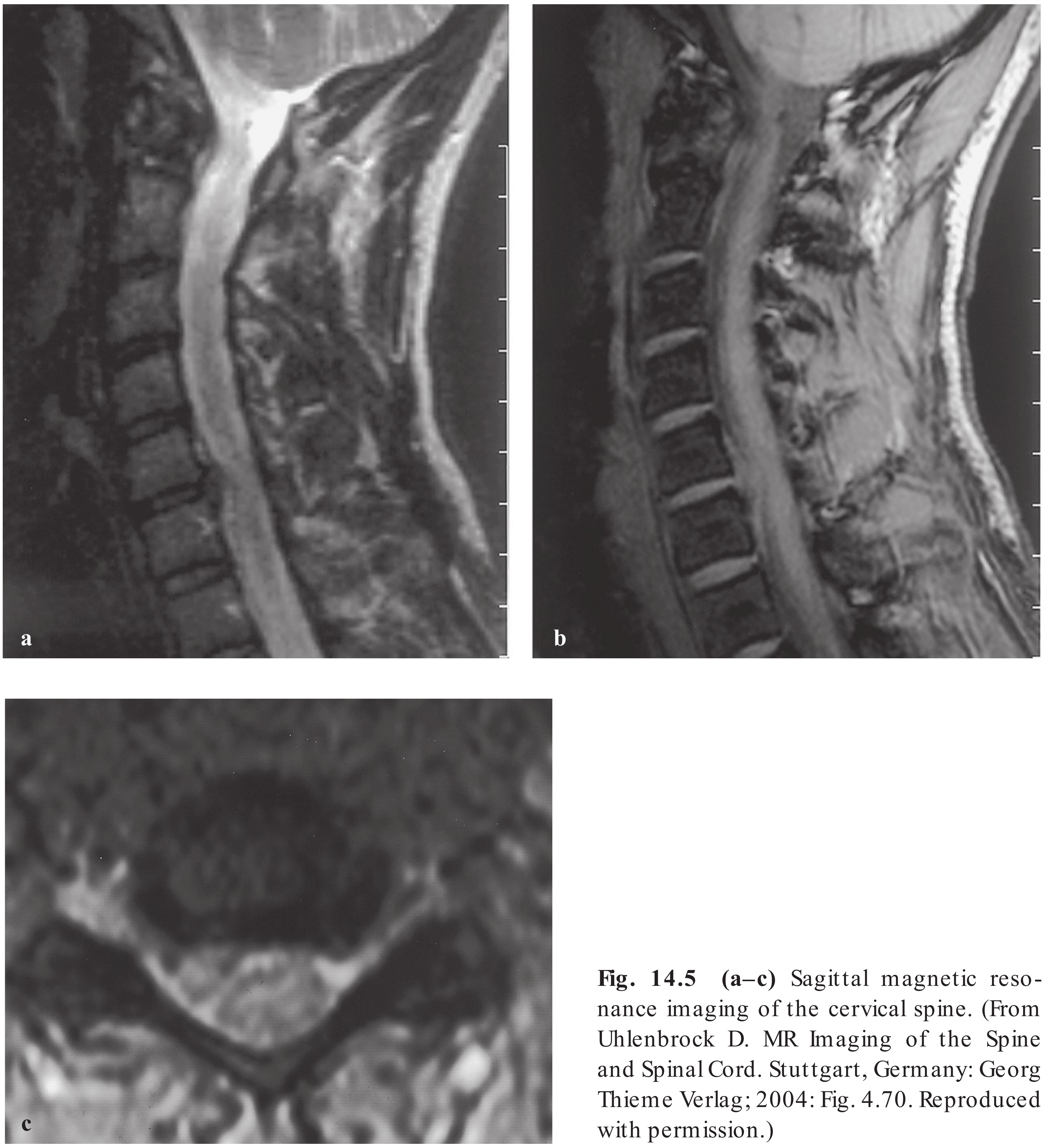

Fig. 14.5 (a–c) Sagittal magnetic resonance imaging of the cervical spine. (From Uhlenbrock D. MR Imaging of the Spine and Spinal Cord. Stuttgart, Germany: Georg Thieme Verlag; 2004: Fig. 4.70. Reproduced with permission.)

3. Axial pain without radiculopathy should be treated conservatively as long as possible, because surgical results are less predictable.

VI. Surgical techniques.

A. Anterior cervical surgery.

1. Indications.

a. Central soft disk herniation.

b. Bilateral radiculopathy at the same level.

c. Unilateral soft disk or foraminal stenosis.

(1) Anterior approach is preferred in patients with significant neck pain in addition to radiculopathy.

d. One- or two-level spondylotic myelopathy.

e. Kyphotic sagittal alignment.

Table 14.2 Differential diagnosis of cervical spine pathology

Pathology	Differential
Trauma	– Cervical sprain – Traumatic neuritis (brachial plexus) – Posttraumatic instability
Tumor	– Superior sulcus (Pancoast) tumor with C8 radiculopathy and Horner's syndrome – Spinal cord tumors – Metastatic disease – Primary bone tumors
Inflammatory conditions	– Rheumatoid arthritis – Ankylosing spondylitis
Infections	– Diskitis – Osteomyelitis – Soft tissue abscess – Shoulder disorders – Rotator cuff tears – Impingement syndrome
Neurological conditions	– Demyelinating disease (Guillain–Barré syndrome) – Amyotrophic lateral sclerosis
Others	– Thoracic outlet syndrome – Reflex sympathetic dystrophy – Angina pectoris – Peripheral nerve entrapments – Multiple sclerosis – Acute brachial neuritis (Parsonage–Turner syndrome)

2. Anterior cervical diskectomy and fusion (ACDF).
   a. Allograft with local autograft may be used for fusion with instrumentation.
      (1) No iliac crest graft site morbidity.
      (2) Recombinant human bone morphogenetic protein-2 (rhBMP-2) was used off label as an allograft to adjunct arthrodesis in the anterior cervical spine.
         (a) In 2007, the Food and Drug Administration (FDA) issued public health notifications regarding the risk of dysphagia and severe, sometimes fatal, soft tissue swelling causing airway compromise with the use of rhBMP-2 in the setting of an ACDF.
         (b) Subsequently, the rate of rhBMP-2 use in the anterior cervical spine has declined substantially.

b. Interbody cage devices:
   (1) Can hold graft materials.
   (2) Provide structural stability.
   (3) Maintain foraminal height.
   (4) Titanium and carbon fiber cages were popularized.
      (a) These metallic cages had a greater modulus of elasticity than bone, resulting in cage subsidence.
      (b) Disk height collapse and kyphotic deformity.
   (5) Polyetheretherketone (PEEK) cages were subsequently introduced **(Fig. 14.6):**
      (a) Radiolucent.
      (b) Nonabsorbable and biocompatible.
      (c) Comparable modus of elasticity to bone.
      (d) Reduced risk of cage subsidence.
   (6) PEEK cages are produced as nonexpandable, expandable, and stackable models.
      (a) Nonexpandable PEEK cages have predefined dimensions, end plate angles, and heights.
         i. Risk for implant displacement.

Fig. 14.6 Illustration of a polyetheretherketone (PEEK) cage. (From Albert TJ, Lee JY, Lim MR. Cervical Spine Surgery Challenges. New York, NY: Thieme Medical Publishers; 2008: Fig. 18.5. Reproduced with permission.)

(b) Expandable PEEK cages contour to the patient's anatomy with minimal intraoperative modifications.
i. Particularly advantageous for corpectomy defects.
c. Use of anterior instrumentation (plating):
(1) Single-level interbody fusion is quite stable, and fusion rates are excellent, with no need for postoperative bracing.
(2) Instrumentation is recommended in the following:
(a) Single-level fusions with allograft.
(b) Avoid postoperative bracing.
(c) Multiple-level interbody fusions.
(d) High-risk patients:
i. Revision fusion.
ii. Smokers.
3. Anterior cervical corpectomy and fusion.
a. Strut allograft and anterior plate instrumentation for stability.
(1) Avoids postoperative halo vest.
b. Expandable cages (PEEK/titanium).
(1) Easier to contour and fit to the corpectomy trough.
B. Cervical disk arthroplasty (CDA).
1. Replacement of an intervertebral disk with an artificial disk device.
2. Purpose was to design motion-sparing devices in an effort to reduce adjacent segment degeneration.
3. Currently five are FDA-approved:
a. Bryan Disc (Medtronic Sofamor Danek).
b. Prestige Disc (Medtronic Sofamor Danek).
c. ProDisc-C (Synthes Spine).
d. Secure C disc (Globus Medical, Inc.).
e. PCM disc (NuVasive, Inc.).
4. Several FDA investigative device exemption trials as well as other prospective studies have demonstrated the long-term noninferiority of CDA when compared with ACDF for the management of degenerative disk disease.
5. The rates of adjacent segment degeneration between CDA and ACDF are still subject to controversy.
a. Similarly, the rates of revision and reoperation have also been scrutinized due to author and industry bias.
C. Posterior cervical surgery.
1. Indications.
a. Unilateral soft disk herniation or foraminal stenosis in patients with radiculopathy and no significant axial symptoms (positive Spurling's sign and no segmental kyphosis).
b. Cervical spondylotic myelopathy (more than three levels of pathology).
c. Ossification of the posterior longitudinal ligament (OPLL).
d. Neutral or lordotic sagittal alignment.
2. Laminoforaminotomy **(Fig. 14.7).**
a. Motion-preserving procedure.
b. Treatment of cervical radiculopathy with minimal axial symptoms.

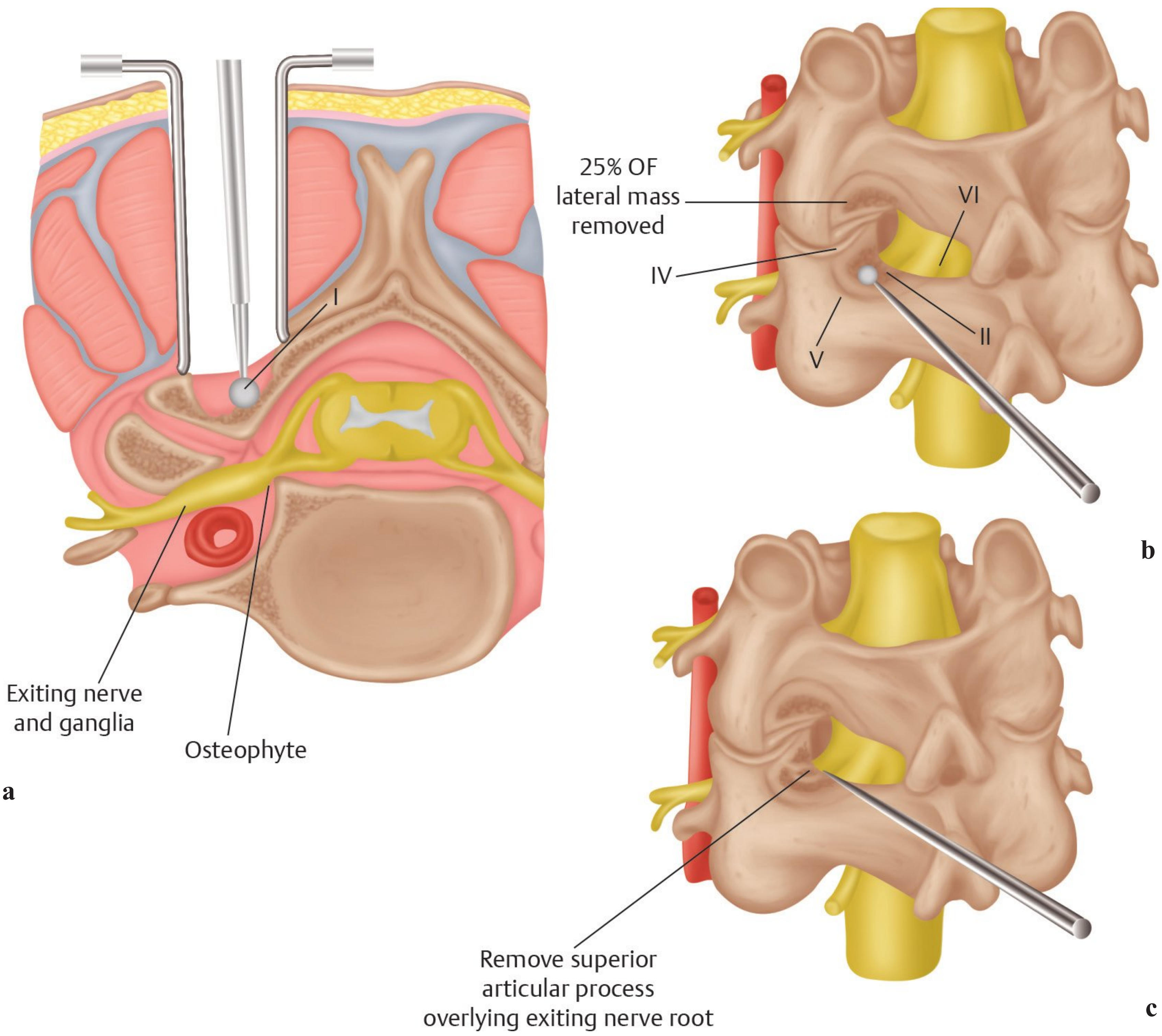

Fig. 14.7 Step-by step methodology for performing a posterior cervical foraminotomy. **(a)** A cutting bur is used to thin the lamina (labeled I) at the junction of the lateral mass–lamina. **(b)** Twenty-five percent of the lateral mass is removed, exposing the lamina (II), superior articular process of the inferior lamina (V), facet joint (IV), and ligamentum flavum (VI). **(c)** A curette is used to remove the superior articular process overlying the nerve root.

3. Laminoplasty **(Fig. 14.8).**
   a. Comparable outcomes and complications when compared with laminectomy and fusion and ACDF.
   b. Motion-preserving procedure.
   c. Same indications as laminectomy and fusion (laminoplasty is preferred in patients with minimal axial neck pain and no significant instability).
   d. Technique with or without instrumentation: instrumentation allows for earlier mobilization and theoretically reduced rates of postoperative axial neck pain.
      (1) French door.
         (a) Midline opening.
         (b) Bilateral hinges.
      (2) Open door (more common).
         (a) Opening side.
         (b) Hinge side.

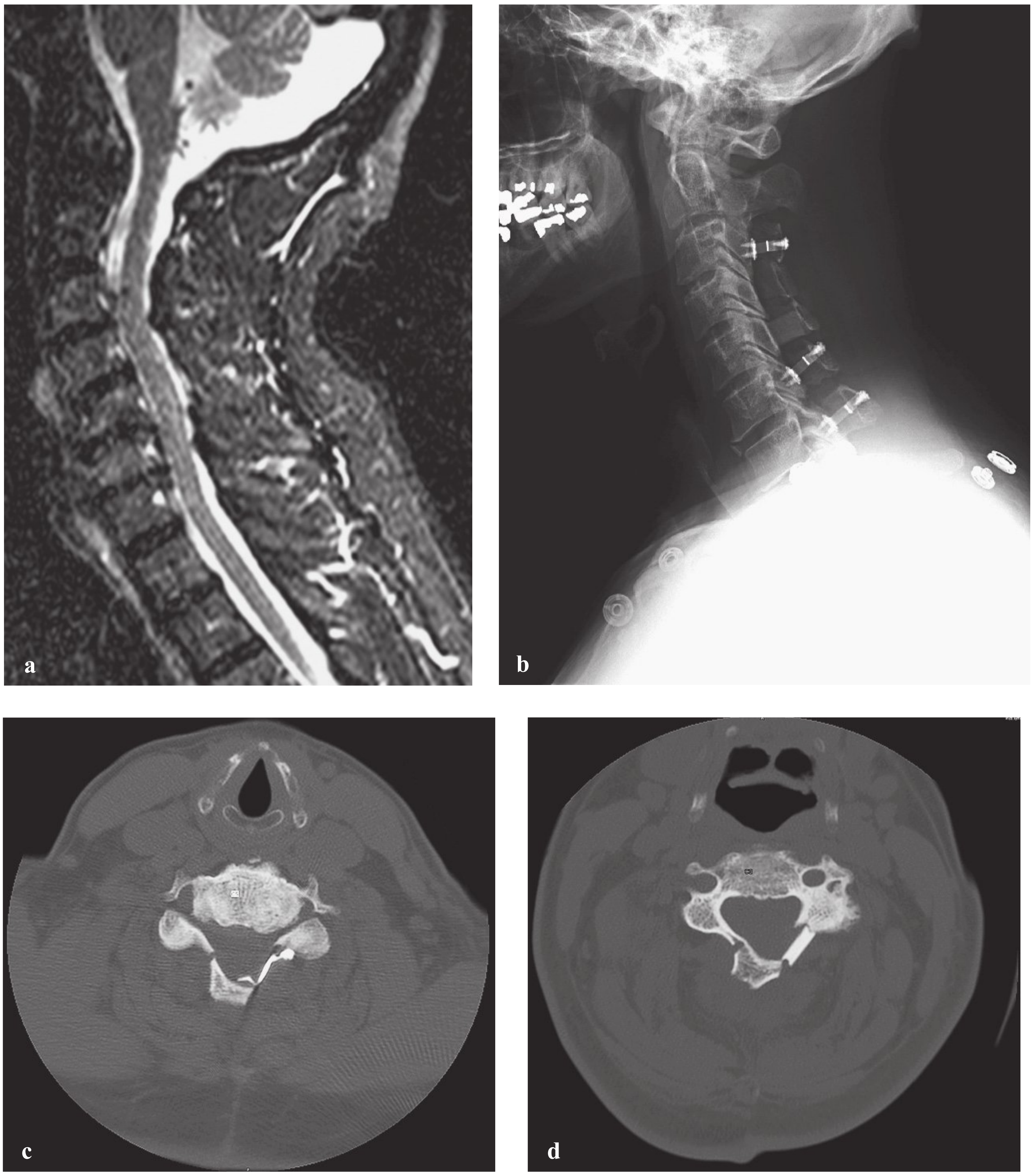

Fig. 14.8 **(a)** Preoperative cervical magnetic resonance imaging demonstrating spinal stenosis extending from C3 to C7. **(b)** A laminoplasty from C3 to C7 was performed (postoperative lateral radiograph demonstrating the placement of titanium cervical plates and allograft). **(c)** Postoperative axial computed tomography demonstrating placement of the titanium plate. **(d)** Postoperative axial computed tomography demonstrating placement of the machined allograft.

4. Laminectomy and fusion with instrumentation (same indications as laminoplasty and preferred for patients with significant neck pain, bilateral foraminal stenosis requiring foraminotomies in addition to laminectomy and instabilities such as spondylolisthesis).
    a. Stabilization is recommended when performing laminectomy to prevent postlaminectomy kyphosis.
        (1) Lateral mass screw fixation.
        (2) Pedicle screw (C2, C7, T1).

(3) C2 Translaminar screw fixation.
  (a) Screws are placed in between the inner and outer tables of the lamina.
  (b) Potential option if the posterior elements of C2 are intact.
  (c) Indications include the following:
    i. Atlantoaxial instability.
    ii. Osteoarthritis.
    iii. Failed C1–C2 arthrodesis.

VII. Complications.
  A. Anterior cervical surgery.
    1. Pseudarthrosis.
    2. Graft dislodgment, resorption, or collapse.
    3. Dysphagia.
    4. Hoarseness.
    5. Vertebral or carotid artery injury.
    6. Dural tears.
    7. Esophageal or tracheal injury.
    8. Nerve injury: C5 nerve root palsy also occurs in anterior surgery; the rates of C5 palsy are slightly decreased relative to posterior cervical surgery.
  B. Posterior cervical surgery.
    1. Neurological deficit.
    2. Axial neck pain.
    3. C5 nerve root palsy.
      a. Believed to occur secondary to posterior cord migration and stretch injury to the C5 nerve root.
      b. C5 palsy occurs with all cervical approaches, including anterior techniques. The rates of C5 palsy are greatest with posterior laminectomy/fusion and laminoplasty. Nevertheless, C5 palsy may occur following anterior cervical fusions.

VIII. Postoperative management.
  A. No rigid collar is needed after instrumented procedures.
    1. The patient may begin range of motion exercises in the immediate postoperative period.
    2. Soft collars may be used for patient comfort.

Suggested Reading

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15 Degenerative Thoracic Spine Conditions

15.1 General Considerations

I. Introduction.
  A. Thoracic pain may be due to several different etiologies **(Table 15.1)**.
    1. The incidence is ~ 15%
    2. It frequently presents in the fourth to sixth decade of life.
    3. Clinical presentation:
      a. May be either radicular or myelopathic.
        (1) The thoracic canal is relatively small.
        (2) Subtle myelopathic symptoms are significant.
        (3) Radiculopathy involves radiating pain around the associated rib.
  B. Diagnosis:
    1. Thoracic disk herniation is frequently seen on magnetic resonance imaging (MRI).
      a. MRI is most useful because of the wide field of view.
        (1) Provides information regarding the status of disk degeneration and spinal canal impingement **(Fig. 15.1).**
        (2) High false-positive rate.
        (3) Also useful in ruling out infections and tumors.
      b. Computed tomography (CT) with myelography:
        (1) More accurate assessment of spinal cord impingement **(Fig. 15.2).**
        (2) Invasive imaging study.
    2. Thoracic stenosis:
      a. Ossification of posterior longitudinal ligament.
        (1) Common in Asian populations.

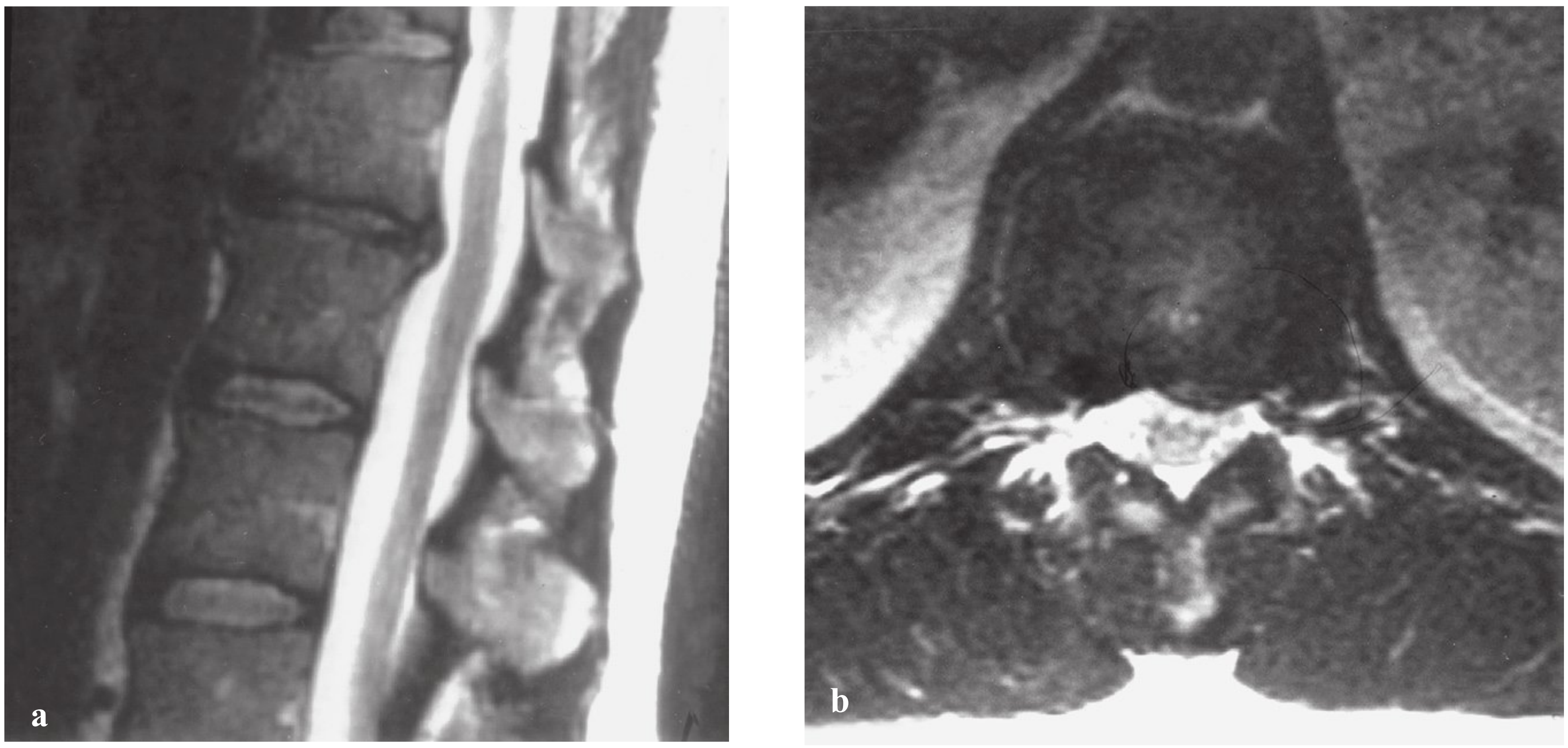

Fig. 15.1 **(a)** T2-weighted sagittal magnetic resonance imaging (MRI) demonstrates a herniated disk at T12–L1. **(b)** A T2-weighted axial MRI demonstrates a left paracentral disk herniation with foraminal impingement.

Table 15.1 Differential diagnosis of thoracic pain

Category	Etiologies
Cardiovascular	Angina pectoris
	Myocardial infarction
	Mitral valve prolapse
	Pericarditis
	Aortic aneurysm
Pulmonary	Pneumonia
	Carcinoma
	Pneumothorax
	Pulmonary embolus
	Pulmonary effusion
Mediastinal	Esophagitis
	Tumors
Intra-abdominal	Hepatitis
	Abscess
	Cholecystitis
Gastrointestinal	Peptic ulcer disease
	Hiatal hernia
	Pancreatitis
Retroperitoneal	Pyelonephritis
	Ureteral stone
	Aneurysm
Neurological	Intraspinal cyst/tumor
	Demyelinating disease
	Transverse myelitis
Infectious	Osteomyelitis
	Diskitis
	Epidural abscess
	Tuberculosis

Category	Etiologies
Traumatic	Compression fractures
	Rib fractures
Neoplastic	Metastatic disease
	Multiple myeloma
	Intradural tumors
Metabolic	Osteoporosis
	Osteomalacia
	Paget's disease
Miscellaneous	Herpes zoster
	Inflammatory disease
	Polymyalgia rheumatica

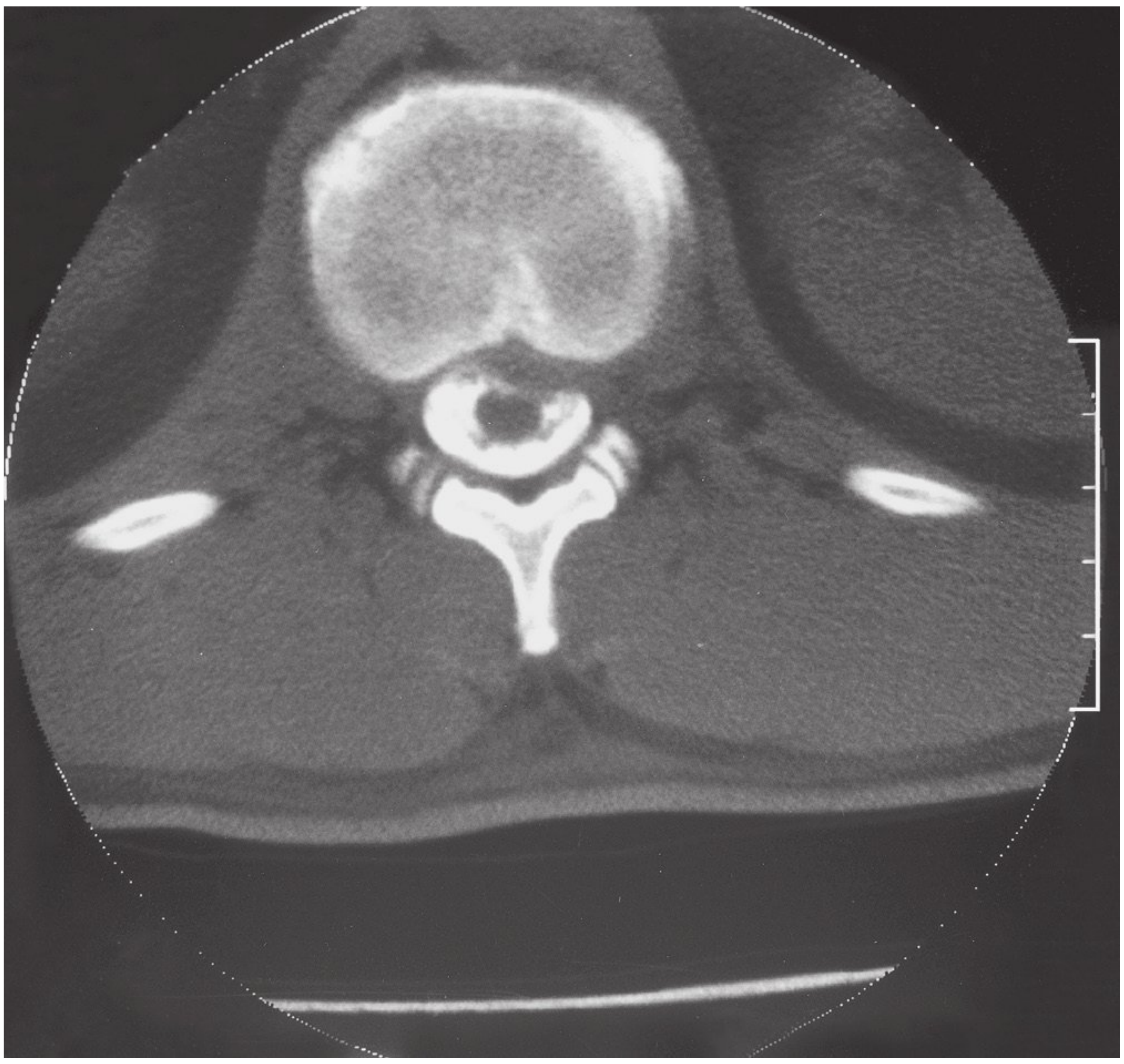

Fig. 15.2 Computed tomographic myelography of the disk herniation in **Fig. 15.1**, demonstrating effacement of the spinal cord.

b. Ossification of ligamentum flavum.
   (1) Posterior spinal cord compression.
   (2) Treated with posterior thoracic decompression.
c. Spondylosis.

II. Treatment of thoracic disk disease.
   A. Conservative modalities.
      1. Nonsteroidal anti-inflammatory drugs.
      2. Exercise program.
      3. Muscle strengthening and cardiovascular fitness.
      4. Physical therapy and modalities as necessary.
      5. Conservative treatment should be tried for at least 6 months in patients without myelopathy.
   B. Surgery.
      1. Indications.
         a. Thoracic disk herniation with myelopathy.
         b. Thoracic radicular pain without myelopathy that becomes unrelenting and resistant to conservative treatment for at least 6 months.
      2. Surgical techniques.
         a. Laminectomy alone may address one- or two-level pathology.
            (1) Instrumented fusion after laminectomy is recommended for multilevel cases or high-risk patients to prevent postoperative kyphosis.
         b. Costotransversectomy:
            (1) An option in posterolateral herniated disk cases.
         c. Most cases require an anterior approach with or without fusion.
            (1) Fusion is advocated in patients with the following:
               (a) Significant back pain.
               (b) Evidence of spinal instability.
               (c) Iatrogenic instability secondary to removal of bone/disk for decompression.
               (d) Kyphotic deformity.
         d. Anterior instrumentation may be used in kyphotic cases.
         e. Thoracoscopic diskectomy is an option that may decrease surgical morbidity.
            (1) Very surgeon dependent.
            (2) Sharp learning curve.
         f. Minimally invasive thoracic approaches have gained popularity.
            (1) Retropleural approach: minimizes the need for postoperative chest tube.

Suggested Reading

Amato V, Giannachi L, Irace C, Corona C. Thoracic spinal stenosis and myelopathy: report of two rare cases and review of the literature. J Neurosurg Sci 2012;56(4):373–378

Hsieh PC, Lee ST, Chen JF. Lower thoracic degenerative spondylolithesis with concomitant lumbar spondylosis. Clin Neurol Neurosurg 2014;118:21–25

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16 Lumbar Disk Disease: Pathogenesis and Treatment Options

16.1 General Considerations

I. Introduction.
   A. Incidence.
      1. Eighty percent of the population will experience back pain.
      2. Two to 3% will experience concomitant lower extremity radiculopathy.
   B. Age:
      1. Mean age of onset is 35 years.
      2. Unusual in patients under 20 years and over 60 years of age.
      3. Herniated disks in children are rare.
         a. Slippage of an entire disk and vertebral end plate or "slipped vertebral apophyses" may mimic a herniated disk.
      4. Herniated disk in the elderly is also uncommon.
         a. May be associated with spinal stenosis.
   C. Gender ratio is approximately equal.
      1. Females typically present a decade later.
   D. Natural history of low back pain and radiculopathy:
      1. Low back pain resolution:
         a. Fifty to 60% recover within 1 week.
         b. Ninety-five percent recover in 3 months.
      2. Radiculopathy resolution:
         a. Fifty percent recover in 1 month.
         b. Seventy-five percent recover in 1 year.
      3. Surgical results are better after 1 year and may persist for up to 4 years (Spine Patient Outcomes Research Trial [SPORT]).
   E. Epidemiological risk factors:
      1. Genetic predisposition.
         a. Currently thought to have the most impact on degenerative disk disease, thanks to contributions by the Twin Spine Study by Battie et al.
      2. Cigarette smoking.
      3. Depression.
      4. Sedentary lifestyle.
      5. Obesity.
      6. Occupational (theoretical).
         a. The Twin Spine Study demonstrated that occupation (repetitive heavy lifting, driving) had little effect on disk degeneration.

II. Pathogenesis.
   A. Intervertebral disk (IVD) degeneration **(Fig. 16.1)**.
      1. Decreased nutrition to the IVD.
         a. End plate sclerosis.
         b. Decreased vascularity.

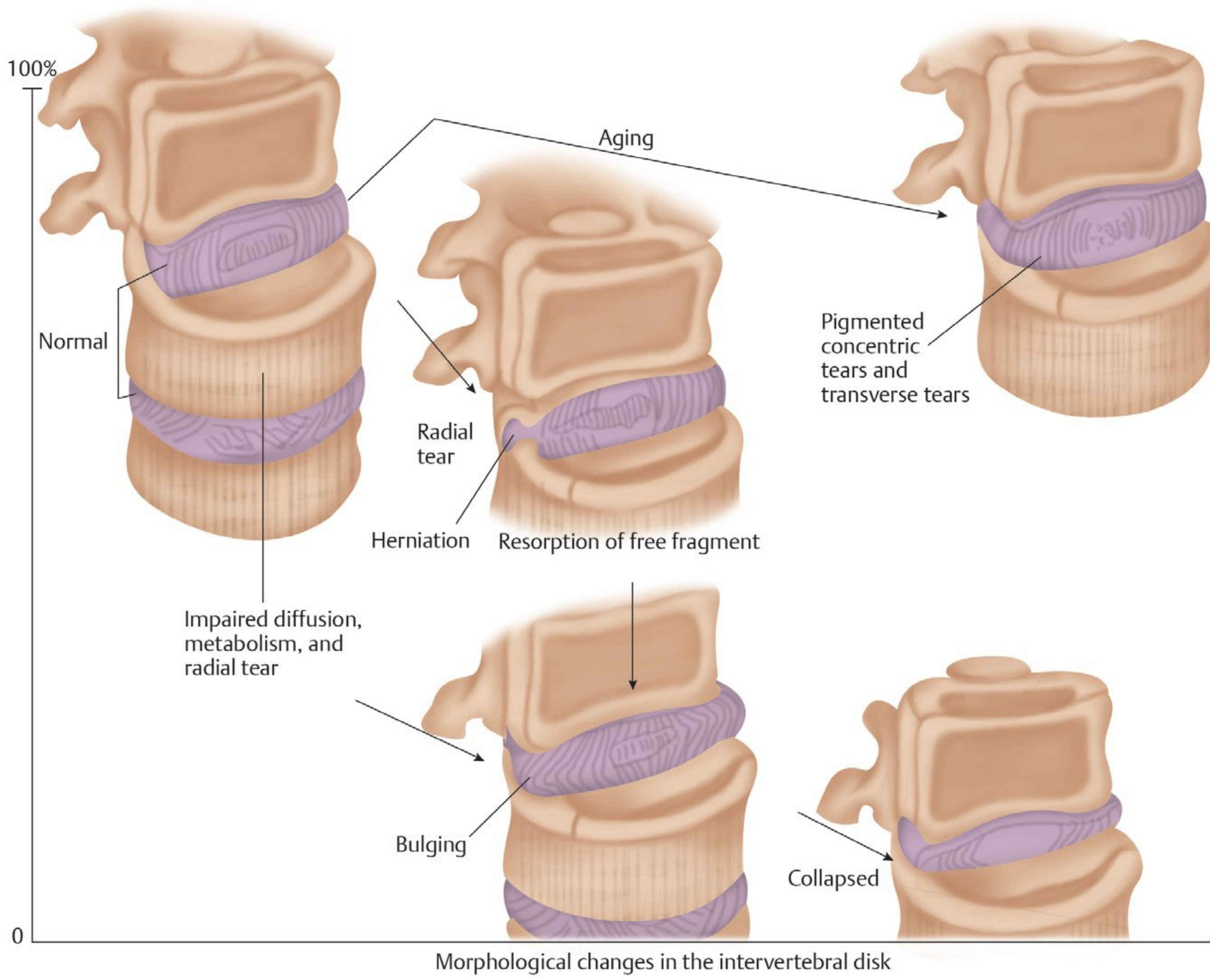


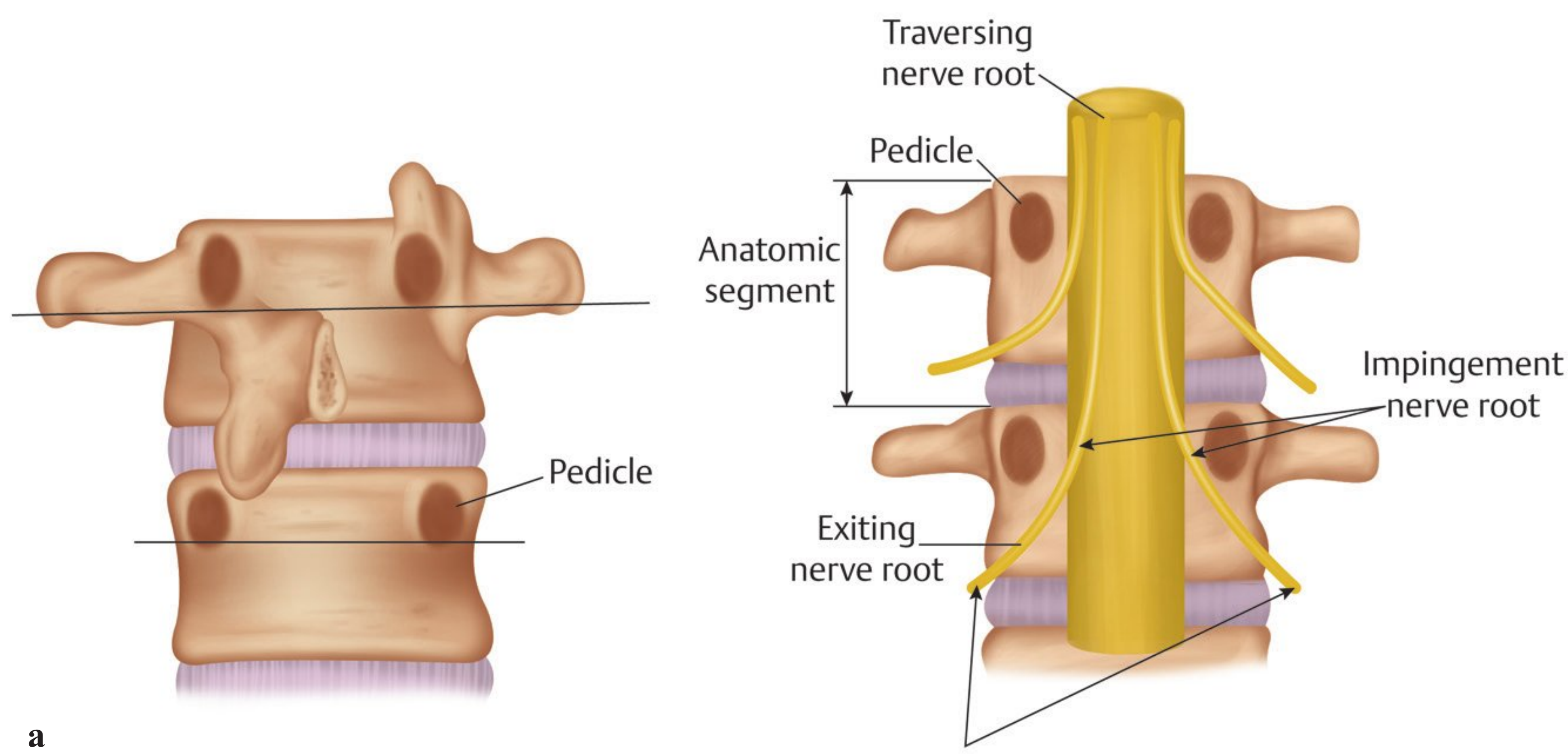
Fig. 16.1 Schematic demonstrating the morphological changes associated with degeneration in the intervertebral disk. Signal intensity (magnetic resonance imaging) decreases as degeneration progresses with loss of disk height.

 (1) O_2 tension decreases.
 (2) Lactate increases.
 (3) pH decreases in the center of the nucleus pulposus.

2. Decreased water content.
   a. Normally 88%; decreases to 60%by the eighth decade.
3. Increased likelihood of annular tears with aging.
   a. Decreased proteoglycan content of the nucleus pulposus leads to greater peripheral annular loading and less central nucleus pulposus resistance.
   b. An annular tear may cause low back pain without radiculopathy.
      (1) This association is still controversial.
   c. Nociceptors are found on the outer annulus and posterior longitudinal ligament.
      (1) The posterolateral corner is most susceptible to a tear from flexion/rotation or torsional stresses.
4. Herniation.
   a. Types of disk herniation **(Table 16.1).**
   b. Locations.
      (1) Most common at L4–L5 and L5–S1, less common at high lumbar and thoracic regions.

Table 16.1 Morphology of disk herniations

Type	Findings
Protrusion	Broad-based disk bulge
Extrusion	Disk herniation through the posterior longitudinal ligament; the fragment is larger than its base but still in continuity
Sequestered	Complete displacement of the disk with no continuity of the fragment and the intact disk



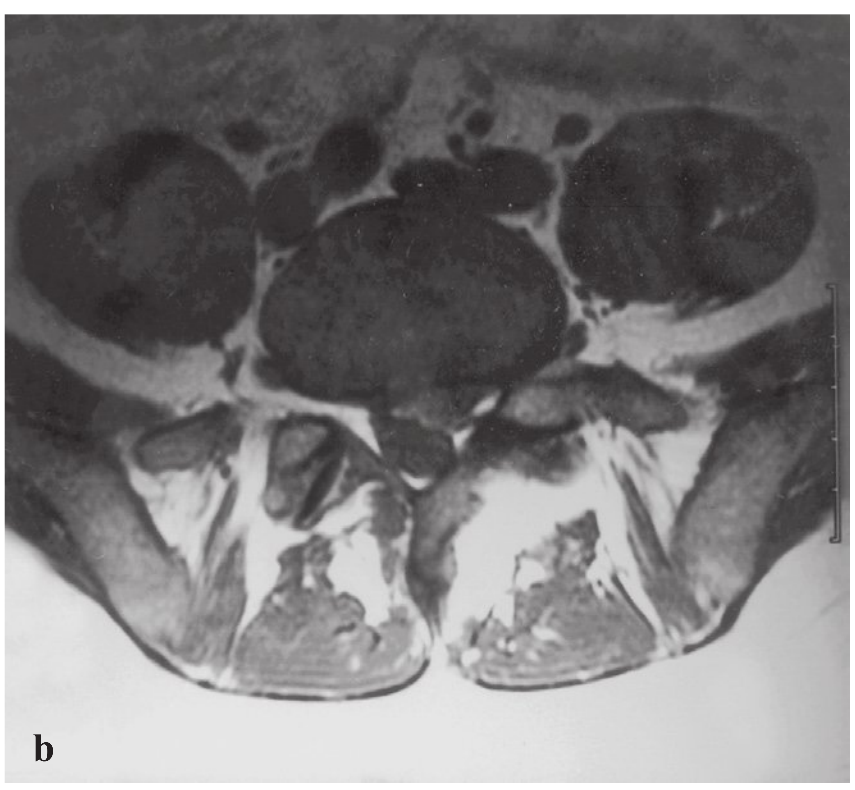


Fig. 16.2 **(a)** Schematic demonstrating the anatomical orientation of the exiting and traversing nerve root in relation to the intervertebral disk. Note that the exiting nerve root can be affected only by a far lateral disk herniation. **(b)** Typically, posterolateral disk herniations affect the traversing nerve root. A magnetic resonance imaging scan shows a posterolateral L5–S1 disk herniation impinging on the left S1 nerve root.

(2) Classically posterolateral with impingement of the traversing nerve below (i.e., L4–L5 herniated nucleus pulposus [HNP] affects the traversing L5 nerve root) **(Fig. 16.2).**

(3) Axillary herniation:

  (a) Usually due to cephalad and medial migration of the disk fragment.

  (b) Medial retraction becomes difficult and dangerous **(Fig. 16.3)**.

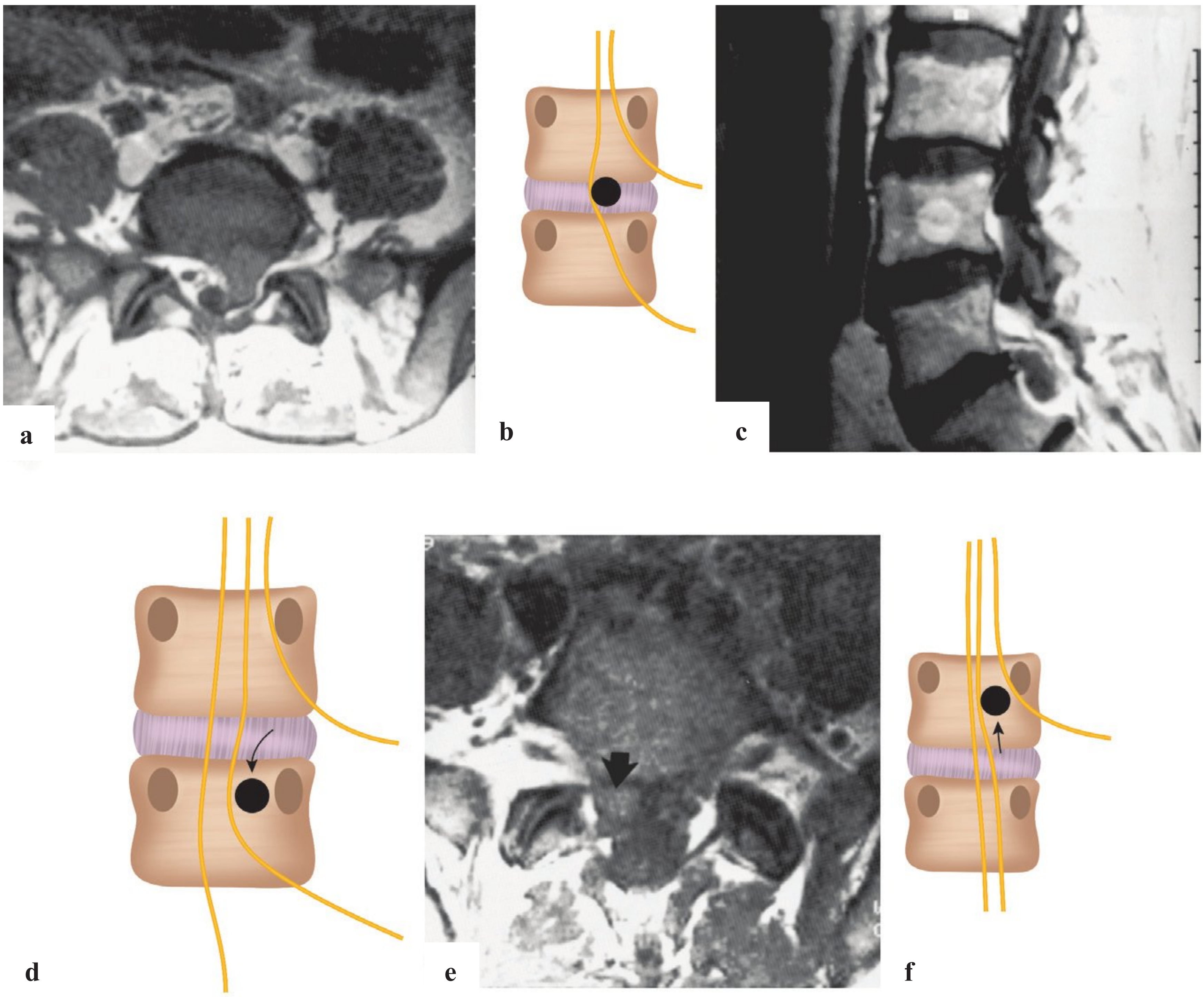


Fig. 16.3 Illustrations and magnetic resonance imaging demonstrating various types of lumbar disk herniations. **(a,b)** Left paracentral L5–S1 disk herniation. **(c,d)** Sequestered disk fragment that migrated inferiorly to the level of the S1 pedicle. **(e,f)** L5–S1 axillary disk herniation.

(4) A small central disk herniation may cause back pain without radiculopathy or cauda equina syndrome.
(5) Extreme lateral or foraminal herniation:
  (a) Impinges upon the exiting nerve root above the disk level.
  (b) More common in older patients.
  (c) Typically found at L3 and L4 **(Fig. 16.4).**
(6) Intradural herniation is extremely rare.

B. Nerve roots.
  1. Anatomy.
    a. Each lumbar nerve root exits below the pedicle and above the disk.
      (1) For example, the L5 nerve exits below the L5 pedicle and above the L5–S1 disk.
    b. The dorsal root ganglion lies in the intervertebral foramen inferior to the pedicle, which may be the main source of pain.
    c. Each nerve root has three branches.
      (1) Ventral ramus for motor function

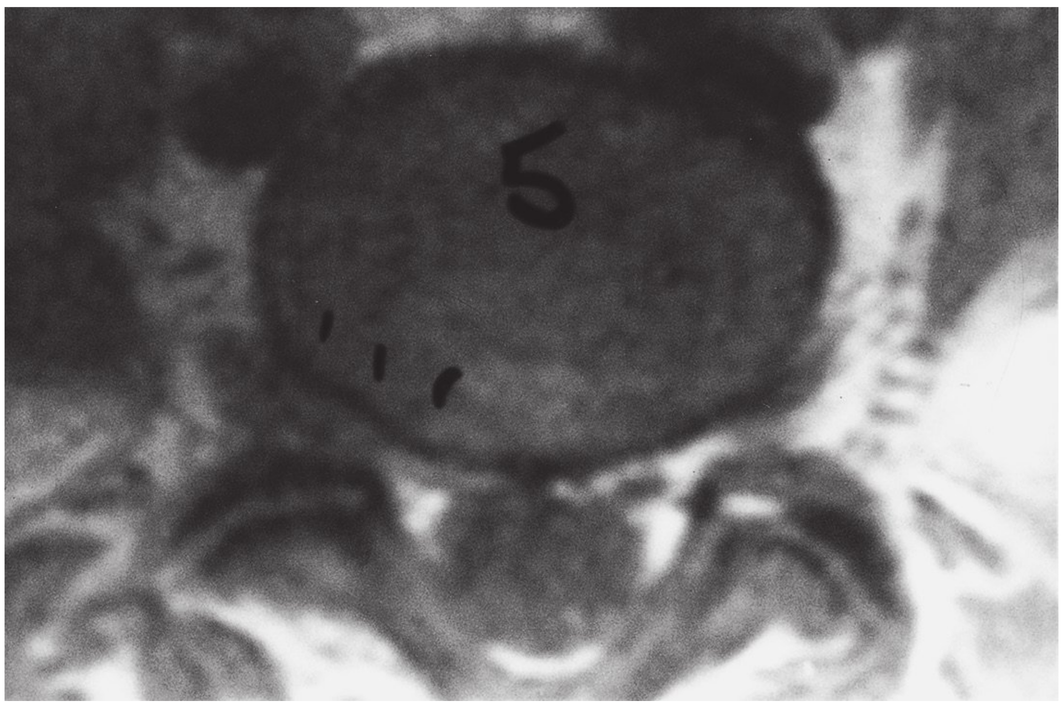


Fig. 16.4 An axial magnetic resonance imaging scan demonstrating a large far lateral disk herniation at L5–S1 causing impingement on the right L5 nerve root.

(2) Sinuvertebral nerve.
  (a) Innervates the outer layer of the posterior annulus fibrosus.
  (b) Anterior part of the annulus fibrosus has sympathetic innervation, and the afferent fibers connect with the sinuvertebral nerves.

(3) Dorsal ramus branch.
  (a) Innervates the facets and posterior muscles.

C. Biochemical pathology of disk herniation.

1. Annulus fibrosus and nucleus pulposus cells produce the extracellular matrix.
   a. Primarily collagen and proteoglycans.
   b. With aging and disk degeneration, the cells die (apoptosis), and the matrix content is decreased.
   c. Both anabolic and catabolic processes maintain the integrity of the matrix.
      (1) Proinflammatory cytokines regulate the catabolic processes.
        (a) Tumor necrosis factor alpha, interleukin-1, phospholipase A2, prostaglandins.
      (2) Growth factors regulate the anabolic processes.
        (a) Bone morphogenetic protein (BMP).
2. Diffusion through the end plates or nutritional pathway is also altered by degenerative changes.
3. An annular tear or HNP is initially associated with significant inflammation.
   a. Cytokine leakage through the annular defect may cause radiculopathy.
   b. Nucleus pulposus fragments elicit inflammatory and neurotoxic changes without significant mechanical compression.
      (1) Rest, anti-inflammatory drugs, or epidural steroids frequently alleviate the symptoms by decreasing the inflammatory reaction.
4. The HNP fragment elicits significant inflammatory reaction and undergoes subsequent resorption.

III. Clinical evaluation.
   A. History.
      1. Sciatica or radiculopathy.
         a. Frequently becomes greater than back pain in large extruded or sequestered disk herniations.
         b. Typically resolves with time in most protruded disk herniations.
         c. Dermatomal distribution.
            (1) Pain is classically worse with sitting, coughing, sneezing, and forward flexion.
            (2) Pain is usually alleviated by lying and rest.
   B. Physical examination.
      1. Observation of the patient's behavior, pain response, spine balance, gait, muscle spasm, muscle atrophy is important.
      2. Bony and soft tissue palpation:
         a. Midline tenderness is frequent at the involved level.
         b. Sciatic notch and tenderness along the course of the sciatic nerve may be present in radiculopathy.
         c. Paraspinal muscle spasm may be palpable.
      3. Range of motion:
         a. Normal range of motion is extremely variable among individuals and even changes from morning to evening.
            (1) Pain reproduction at extremes of motion is a helpful sign.
               (a) Painful lumbar flexion suggests diskogenic etiology.
               (b) Painful lumbar extension suggests facet disease.
         b. Lateral bending may cause ipsilateral lower extremity pain in patients with posterolateral or lateral herniated disks.
            (1) Lateral bending away from the symptomatic limb may aggravate pain in patients with an axillary herniated disk.
         c. Painful dysrhythmic range of motion may indicate mechanical instability, particularly when straightening from forward flexion.
      4. Neurological examination:
         a. Motor, sensory, and reflex deficits may be present along a specific nerve root distribution.
         b. Special tests:
            (1) Straight leg raise.
               (a) Elevation of a painful limb causes radicular limb pain.
                  i. Record the degree of elevation that reproduces pain.
               (b) Dorsiflexion of the foot while raising the leg also stretches the sciatic nerve and causes pain.
            (2) Contralateral leg raise test.
               (a) Positive if elevation of a nonpainful limb causes back and limb pain on the opposite side.
                  i. Usually implies sequestered or large extruded herniated disk.
            (3) Reverse straight leg raise test (femoral nerve stretch test).
               (a) Femoral extension in a prone position stretches the femoral nerve reproducing pain in the L3 or L4 distribution.
      5. Differential diagnosis of low back pain **(Table 16.2):**

Table 16.2 Differential diagnosis of low back pain

Type	Differential
Viscerogenic	Abdominal and renal
Neoplastic	Primary and metastatic bone tumors
Neurogenic	Spinal cord tumors or cysts
Inflammatory diseases	Ankylosing spondylitis, Reiter's syndrome, inflammatory bowel diseases, and psoriatic arthritis (sacroiliitis)
Infectious	Diskitis, osteomyelitis, psoas abscess
Spondylogenic	Myofascial syndromes Iliolumbar syndrome, piriformis syndrome, quadratus lumborum syndrome, and fibrositis (trigger point syndrome) Motion segment disorders Disk disease, facet syndrome, spinal stenosis Bony problems Fractures, including osteoporotic compression fractures Spondylolisthesis Sacral lesions, coccyx pain Psychogenic

6. Distinguishing spondylogenic causes of low back pain:
   a. Diskogenic.
      (1) HNP.
         (a) Leg pain, tension signs, neurological deficits.
   b. Annular tears.
      (1) Back pain and referred pain to the buttock (controversial).
      (2) Midline tenderness, painful forward flexion, back pain increased with straight leg raising test.
   c. Myofascial syndromes.
      (1) Tenderness on the affected muscles rather than in the midline.
      (2) Pain with active contraction and with passive stretch (contralateral bending).
   d. Posterior elements.
      (1) Spondylolysis.
         (a) Painful extension and rotation toward opposite side.
      (2) Facet syndrome.
         (a) Tenderness unilaterally over the joint.
         (b) Painful hyperextension and bending.
      (3) Spinal stenosis.
         (a) Neurogenic claudication.
         (b) Painful extension of the back.

7. Diagnostic studies:
   a. Plain X-rays.
      (1) Detect spondylolysis/spondylolisthesis.
      (2) Disk space narrowing.
      (3) Scoliosis.
      (4) Tumors.
      (5) Infection.
   b. Magnetic resonance imaging (MRI).
      (1) Imaging modality of choice for herniated disks and for spinal stenosis.
      (2) Use of gadolinium contrast increases accuracy when evaluating postoperative disk herniations and may be helpful in evaluating for tumor or infection **(Fig. 16.5)**.
      (3) Gadolinium (on T1-weighted images) enhances in vascular scar tissue and does not enhance in a recurrent disk herniation.
      (4) Computed tomographic (CT) scan or CT myelography:
         (a) If MRI is contraindicated.
         (b) CT may be better in patients with degenerative scoliosis or in patients with metal implants.
   c. Diskography.
      (1) Performed in patients with suspected diskogenic back pain without radiculopathy.
      (2) Reproduction of back pain with injection of dye into the suspected disk and evidence of an annular tear are considered positive findings.
         (a) Positive findings must take into context the patient's symptoms and psychosocial status (e.g., somatization, chronic pain).
         (b) Controversial as the accuracy also depends on the physician performing diskography, and the needle puncture of the disk may accelerate degeneration over time.

Fig. 16.5 Sagittal T1-weighted magnetic resonance imaging demonstrating enhancement of lesions following administration of gadolinium contrast. (From Chen C, Chen WL, Yen H. Candida albicans lumbar spondylodiscitis in an intravenous drug user: a case report. BMC Res Notes 2013;6:529. Reproduced with permission.)

d. Bone scan.
   (1) Perform a bone scan if suspicious for tumor or infection.

IV. Conservative treatment.
   A. Proven methods.
      1. Patient education ("back school").
         a. Decreased mechanical stress and expectation.
      2. Cardiovascular fitness programs.
      3. Smoking cessation.
      4. Maintenance of ideal body weight.
   B. Unproven methods.
      1. Prolonged bed rest, muscle relaxants, traction, bracing, and manipulation
      2. Narcotics and tranquilizers have not been shown to improve outcomes.
         a. They may be used for acute symptoms (1–5 days).
      3. Epidural steroids have not been proven to be helpful.
         a. Use in selected patients with persistent leg pain to help with rehabilitation.

V. Operative management.
   A. Indications.
      1. Failure of conservative treatment for at least 6 weeks.
      2. Progressive neurological deficits.
      3. Presence of neurological findings.
         a. Radicular pain.
         b. Positive tension sign or neurological deficits.
         c. Positive imaging study with clinical correlation.
   B. Surgical techniques **(Fig. 16.6** and **Fig. 16.7) (Table 16.3).**

VI. SPORT.
   A. Patient populations.
      1. Clinical and radiographic diagnosis of HNP.
         a. Persistent symptoms despite 6 weeks of nonoperative treatment.
         b. Randomly assigned to operative and nonoperative cohorts.
   B. Operative versus nonoperative treatment outcomes.
      1. Intent-to-treat analysis: both operative and nonoperative treatment demonstrated similar improvement of symptoms at 1 and 2 years.
         a. Surgical treatment was associated with a faster recovery, better physical function, and overall satisfaction.
      2. As-treated analysis: patients treated surgically demonstrated greater improvement in pain and function after 2 years compared with those treated nonoperatively.
   C. Controversies.
      1. High crossover rate.
         a. Forty-five percent of patients in the nonoperative cohort underwent surgery.
         b. Forty percent of patients in the surgical cohort did not undergo surgery.
      2. Nonoperative management.
         a. Not standardized.
         b. Patients were required to have had a 6-week course of nonoperative management for enrollment, which likely affected the crossover rate.

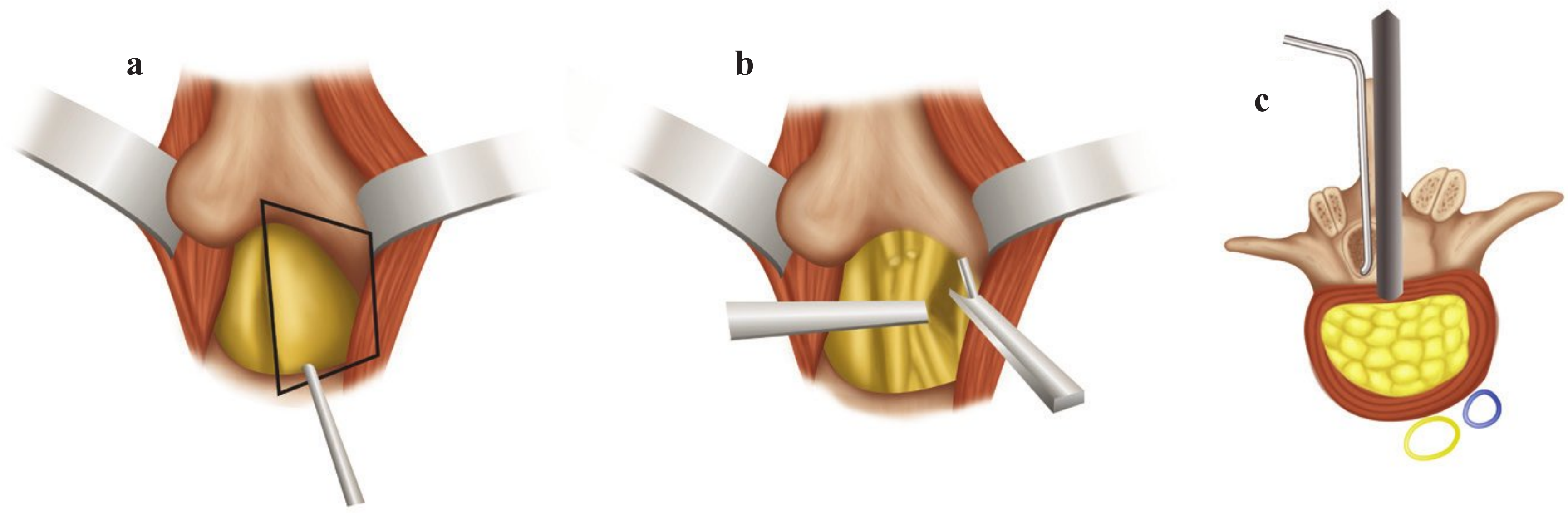
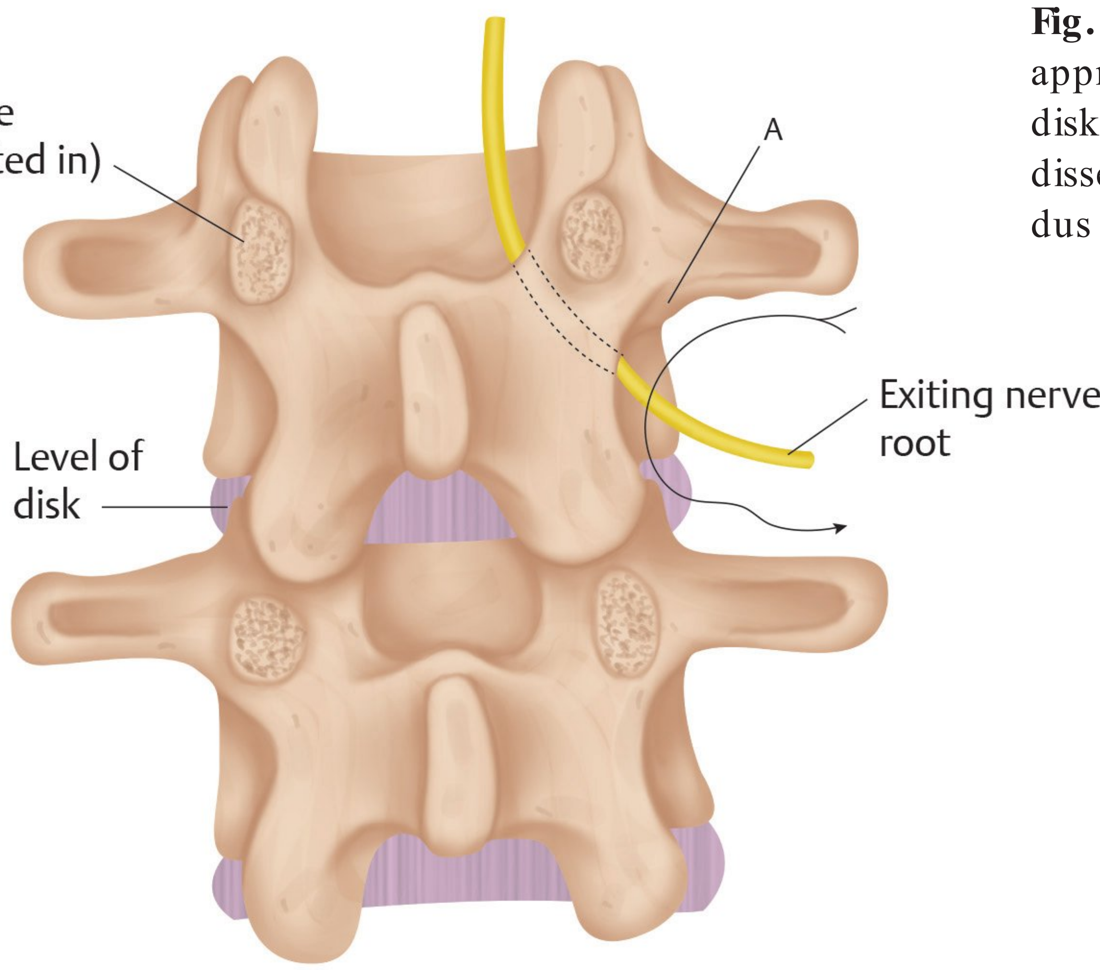


Fig. 16.6 A typical L5–S1 laminotomy and diskectomy. **(a)** The interlaminar exposure starts with removal of the ligamentum flavum. **(b)** Additional removal of laminar bone is performed until the lateral portion of the nerve root is visualized. **(c)** The nerve root is gently retracted medially, and the herniated disk is removed with the aid of a pituitary rongeur.

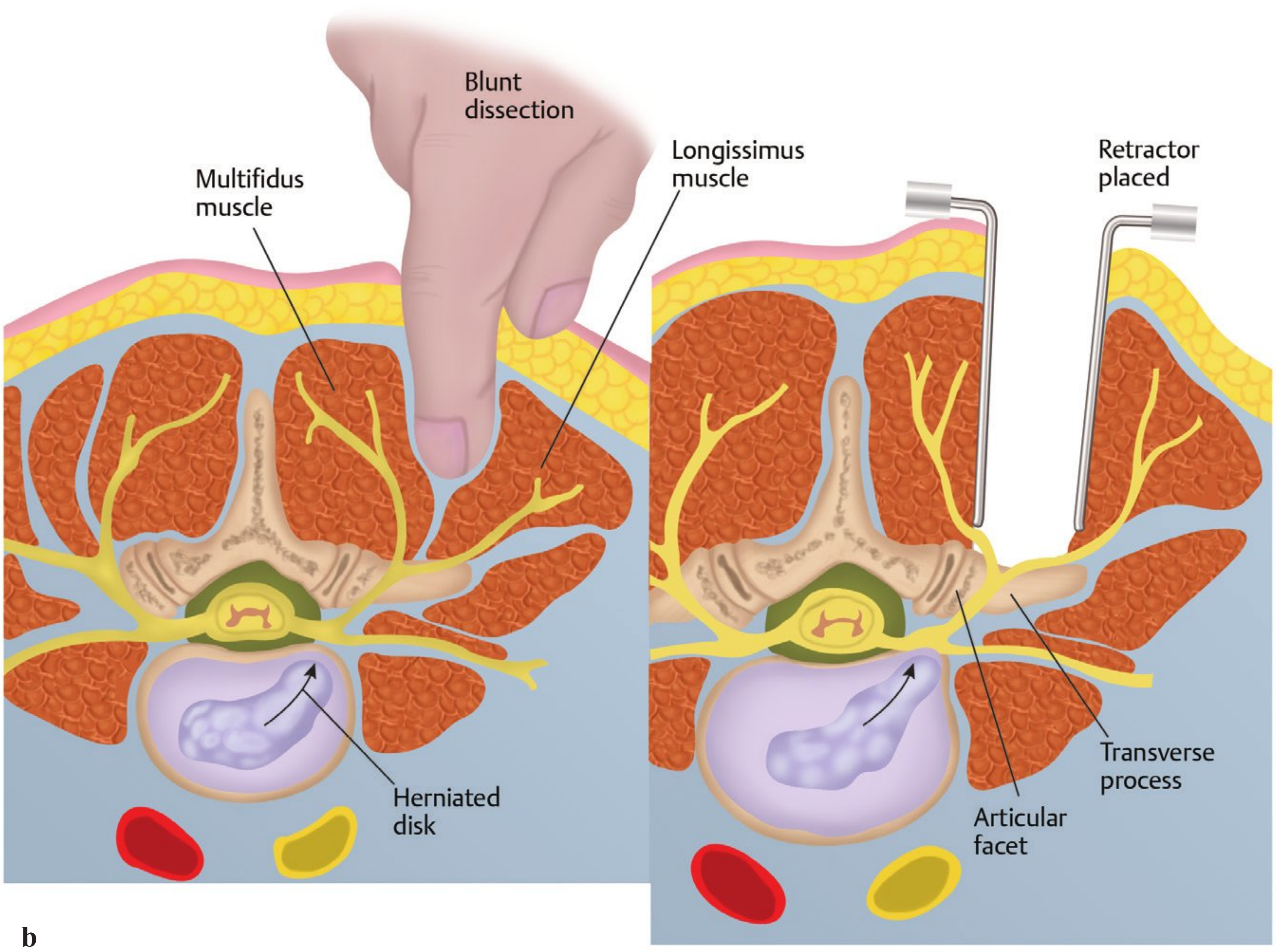
Table 16.3 Various techniques of disk treatment

Surgical technique	Findings
Open diskectomy	– Better visualization of nerve – More muscle dissection – Longer hospital stay – Potential for iatrogenic instability
Microscopically assisted diskectomy	– Limited muscle dissection – Improved visualization and lighting
Percutaneous diskectomy	– Techniques include chemonucleolysis, percutaneous diskectomy, and laser diskotomy – Inferior results when compared with microscopically assisted diskectomy
Intradiskal electrothermal therapy	– Intradiskal procedure for primary diskogenic back pain – Results demonstrate equivocal results when compared with placebo

3. Intent-to-treat versus as-treated analysis.
   a. Intent-to-treat analysis compared patients according to their assigned cohort.
      (1) This demonstrated no difference in primary outcomes between groups in patients with disk herniation; that is, patients assigned to nonoperative treatment were counted as nonoperative patients if they crossed over and had surgical intervention.
   b. As-treated analysis compared patients according to the treatment ultimately received.
      (1) Patients who underwent surgery demonstrated improved and sustained outcomes compared with those treated nonoperatively.



a



b

Fig. 16.7 **(a)** A Wiltse paraspinal approach for excision of a lateral disk herniation. **(b)** Intermuscular dissection is between the multifidus and longissimus.

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17 Surgical Management of Lumbar Degenerative Disk Disease

17.1 General Considerations

I. Introduction.
   A. Lifetime incidence of low back pain is estimated to affect 80% of the general population.
      1. Fourteen percent of new patient visits to physicians are related to low back pain.
      2. Second only to respiratory infections as the most common cause of work absence.
   B. One hundred sixty-five lumbar spine operations per 100,000 individuals occur each year in the United States.
   C. Lumbar fusion for diskogenic pain and lumbar laminectomies for radicular symptoms are the most common spine surgeries performed.
   D. Risk factors for low back pain:
      1. Prior history of low back pain.
      2. Increasing age.
      3. Smoking (nicotine consumption).
      4. Medical comorbidities.
      5. Lower socioeconomic status.
      6. Psychological distress (depression).

II. Etiologies **(Table 17.1).**
   A. Red flags in clinical presentation that require further investigation.
      1. History of significant trauma.
      2. History of previous malignancy.
      3. Age > 50 years.
      4. Systemic symptoms (fever, chills, anorexia, recent weight loss).
      5. Severe progressive neurological deficit.
         a. Especially saddle anesthesia or bowel/bladder dysfunction.
      6. Ongoing infection.
      7. History of immunosuppression.

III. Diagnostic tools.
   A. Establishing a pathoanatomical diagnosis is the key to successful surgical outcomes **(Table 17.2)**.
   B. Arriving at a conclusive diagnosis may be difficult; as many as 85% of patients are categorized as having idiopathic low back pain.
   C. Plain radiographs:
      1. Flexion-extension films.
   D. May demonstrate dynamic instability (spondylolisthesis):
      1. Oblique films.
   E. Helpful in evaluating the integrity of the pars interarticularis in the setting of an isthmic spondylolisthesis:
      1. Lumbar spine films are unnecessary for at least 4 weeks in a patient with new-onset low back pain without any of the previously mentioned red flags.

Table 17.1 Etiologies and differential diagnosis for lower back pain

Type	Differential
Idiopathic or nonspecific (85%)	
Degenerative disk disease	Diskogenic pain Disk herniation Degenerative scoliosis
Developmental	Isthmic spondylolisthesis Idiopathic scoliosis
Congenital	
Traumatic	
Infectious	Osteomyelitis Diskitis
Inflammatory	Ankylosing spondylitis Psoriatic spondylitis Reiter's syndrome
Neoplastic	
Metabolic	Osteoporosis Paget's disease of bone
Referred	Dissecting aortic aneurysm Renal vein thrombosis Renal stones Acute myocardial infarction Pancreatitis Duodenal ulcer Pelvic disease

F. Computed tomography (CT):
   1. Allows excellent visualization of the bony anatomy of the vertebral column.
   2. Not as sensitive as magnetic resonance imaging (MRI) for visualization of soft tissue structures.
   3. CT myelography is an excellent imaging modality for evaluating spinal stenosis, but MRI is used because it is less invasive.

G. MRI:
   1. Excellent axial, coronal, and sagittal visualization of the soft tissues and neural structures both within and surrounding the vertebral column.
   2. Excellent for the evaluation of neural compression within the canal and foramen.
   3. With disk degeneration, T2-weighted MRI demonstrates darkening of the disks due to loss of water, but this finding does not predict the development of back pain in asymptomatic patients.

Table 17.2 Anatomical sources of lumbar spine pain

Intervertebral disks	– Primary pain generator in setting of degenerative disk disease; pain fibers present in the outer third of the annulus fibrosus – Biochemical factors that can mediate painful stimuli: prostaglandins, lactic acid, substance P – During disk degeneration, nerve ingrowth has been observed into deeper aspects of the annulus fibrosus and even into the nucleus
Facet joints	– Extensively innervated with pain fibers. – Synovial folds of the joint lining also possess pain fibers. – Proprioceptive nerve endings also present, which mediate protective muscular reflexes.
Musculoligamentous structures	– Both anterior and posterior longitudinal ligaments (PLL) possess sensory innervation. PLL has been found to have fibers containing substance P. Unencapsulated nerve fibers found in paraspinal musculature respond to metabolites accumulated during prolonged muscle contraction or spasm.
Neural structures	– Pain from mechanical nerve root compression is thought to require the presence of inflammation. – Dorsal root ganglion is sensitive to direct pressure and vibratory forces. – Increase in genetic expression of neuropeptides (substance P) in response to mechanical nerve root compression

H. Single-photon emission CT (SPECT/CT):
   1. Combination of SPECT (high sensitivity and specificity) and CT (high resolution).
   2. Physiological imaging that can detect inflammatory changes in the lumbar spine.
   3. SPECT imaging detects gamma rays from radioisotopes injected into the patient.
   4. Useful in detecting facet joint arthropathy **(Fig. 17.1)**.
   5. Facet injections **(Fig. 17.2):**
      a. Rationalized by the hypothesis that facet arthritis contributes to low back pain.
      b. There are few well-designed studies to evaluate efficacy; thus the use of injections to predict surgical outcomes for patients with low back pain is not supported.

I. Diskography **(Fig. 17.3):**
   1. Performed by the introduction of a needle into the nucleus pulposus and injection of contrast to visualize internal fissures or tears.
   2. Saline may also be injected into the disk to reproduce pain.

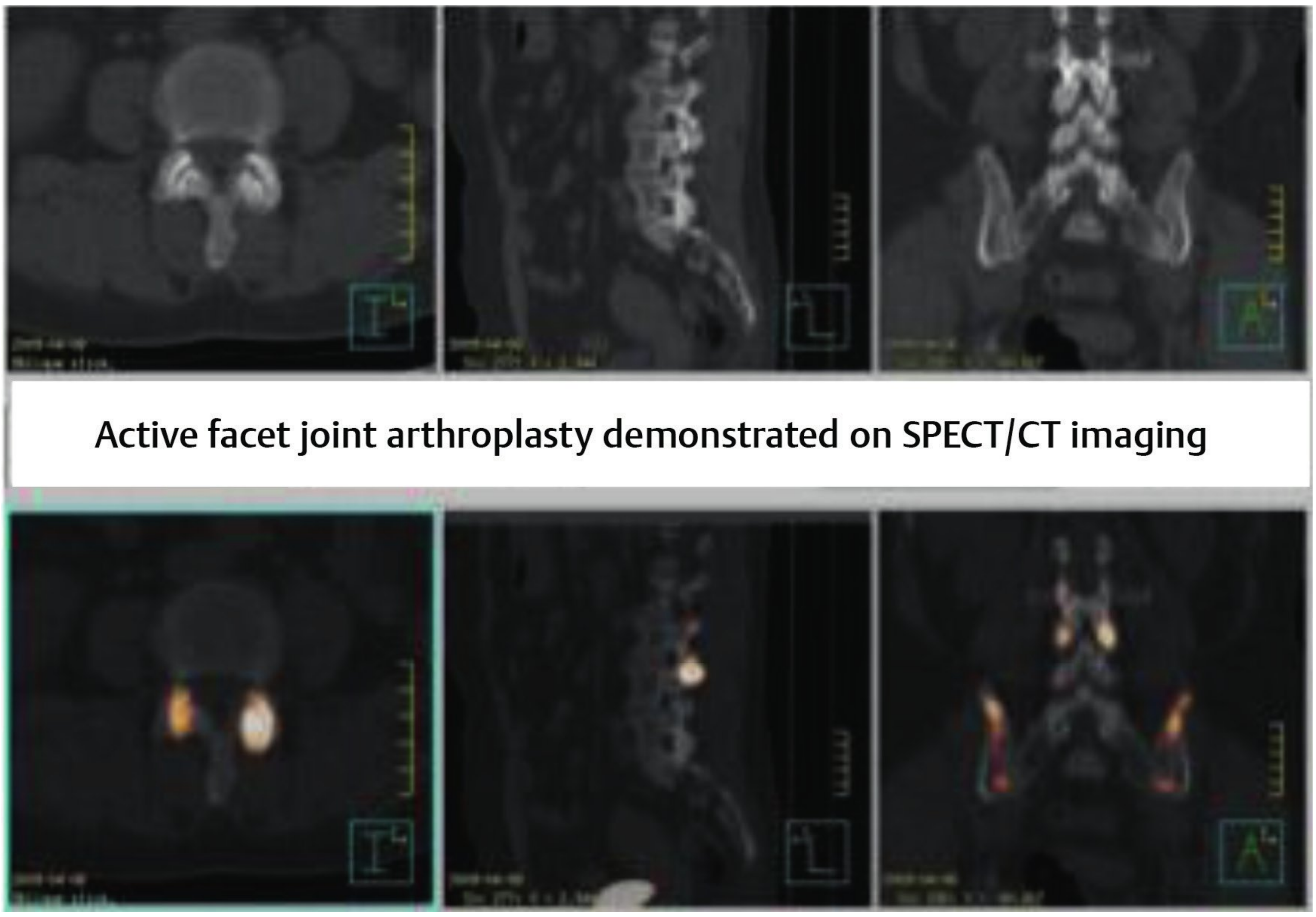

Fig. 17.1 Single-photon emission computed tomography (SPECT/CT).

3. The combination of pain with disk injection and findings of disk degeneration on CT-diskography increases the likelihood that a particular disk is involved with the patient's pain.
   a. Remains controversial, especially without a control.
4. Important findings:
   a. Re-creation of similar pain with injection (concordance).
   b. Pain at low pressurization.
      (1) High pressurization may result in a false positive.
   c. Disk accepts > 2 mL of dye.
5. Highly controversial:
   a. In 2009, Carragee et al reported that diskography was associated with accelerated disk degeneration, disk herniation, and loss of disk height in patients without serious lower back pain.
   b. The validity of this diagnostic intervention has not been proven.
      (1) The lack of a gold standard diagnostic study for diskogenic back pain limits the assessment of the diagnostic validity.
      (2) Carragee et al reported that nearly 50% of patients who had surgery after a positive diskography reported significant pain.
         (a) Thus a positive diskography was not highly predictive in identifying a disk lesion that was responsible for the patient's symptoms.
   c. Diskography is associated with a variable false-positive rate in patients without low back pain.
      (1) Pain-free patients—10%

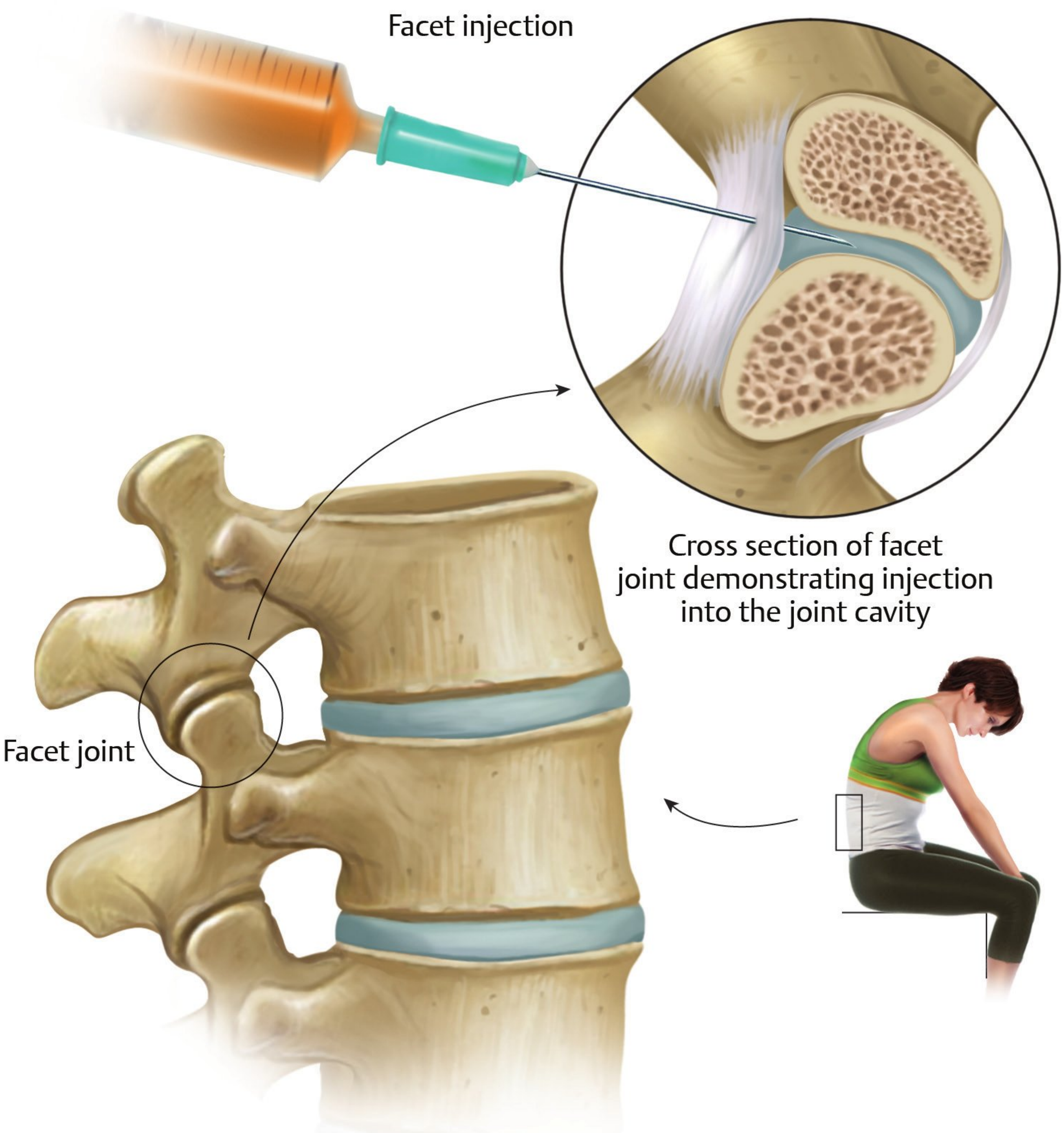

Fig. 17.2 A facet injection is performed in the lumbar spine.

(2) Chronic pain (not in lower back)—40%
(3) Somatization disorder—75%

d. In carefully selected patients with normal psychosomatic states (not involved in litigation), diskography may provide useful diagnostic information in the setting of nonspecific lower back pain.

IV. General surgical indications.
  A. Mechanical instability.
  B. Neurological deficits.
  C. Indications for patients with low back pain without radicular symptoms
    1. Unremitting back pain and disability for more than 1 year.
    2. Failure of physical therapy and nonoperative treatment modalities.
      a. Nonsteroidal anti-inflammatory drugs (NSAIDs), heat, ice, weight loss, activity modification.

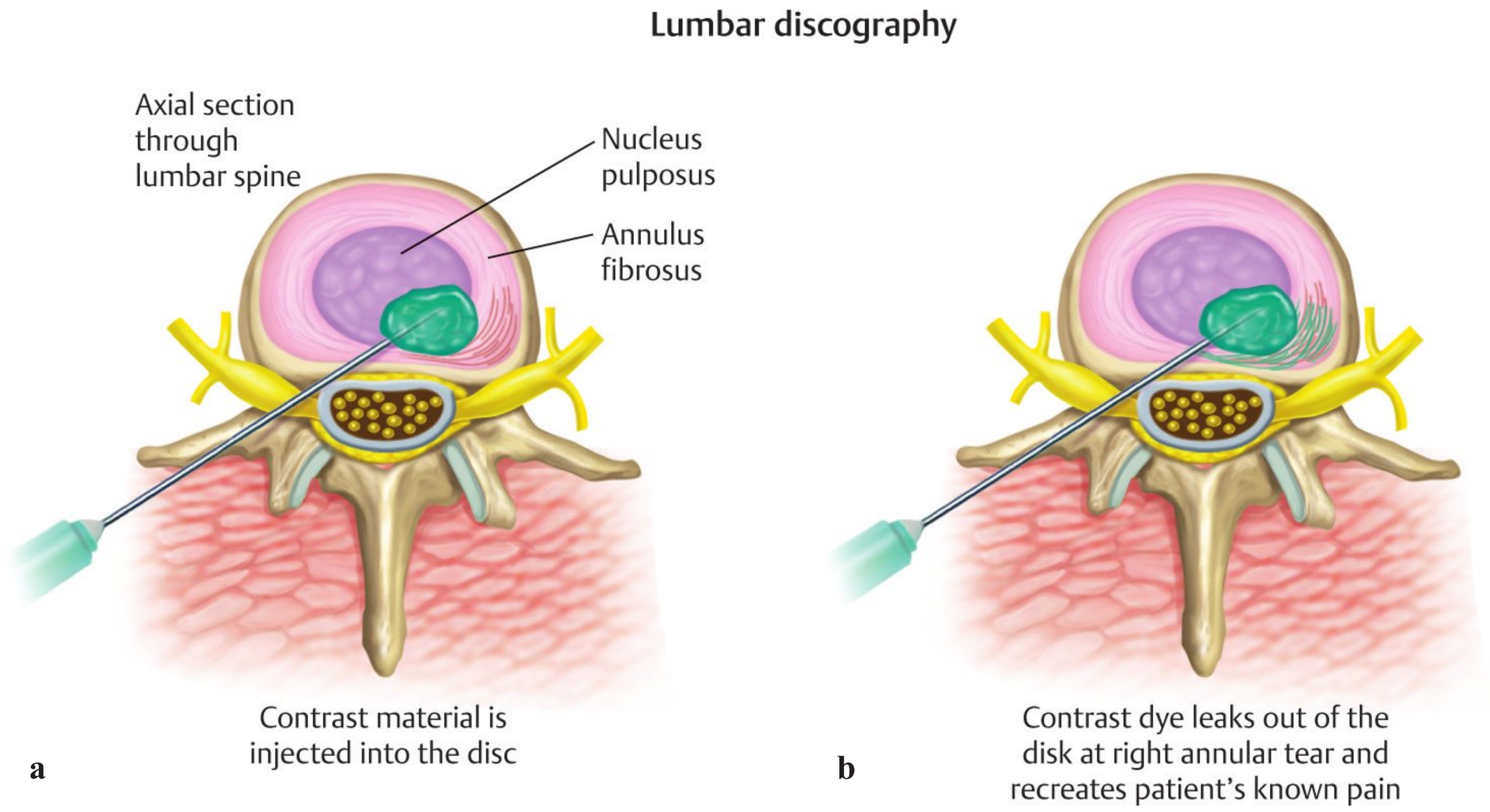

Fig. 17.3 (a,b) Contrast dye injection into the nucleus pulposus.

3. Absence of psychiatric disorders and compensation or ligation issues.
4. Isolated single-level disk degeneration on MRI with concordant pain on diskography or single-level static or dynamic instability.

V. Surgical procedures.
  A. General principles.
    1. Low back pain management largely focuses on spinal fusion.
    2. Decompression is indicated in the setting of leg pain and nerve root compression.
    3. Motion preservation procedure with lumbar total disk arthroplasty.
  B. Spinal fusion principles.
    1. Prevents further segmental motion.
      a. More appropriate for spinal instability.
  C. Keys to attaining a solid arthrodesis.
    1. Meticulous preparation of the graft site (decortication).
    2. Supplementation with appropriate type and amount of bone graft.
    3. Consideration of lumbar spine biomechanics.
      a. Maintaining or restoring the normal lordosis in the sagittal alignment of the lumbar spine.
    4. Optimizing systemic conditions that influence bone healing.
      a. Nicotine, corticosteroids, NSAIDs, nutrition, and infection.
      b. Concern for new or progressive degeneration of adjacent levels to the fused segments mandates that minimum numbers of levels be fused, especially in younger individuals.

VI. Lumbar fusion techniques **(Table 17.3).**
  A. Posterolateral (intertransverse process) fusion (PLF).
    1. Involves either a posterior or a posterolateral muscle-splitting approach.

2. Involves decortication of transverse processes and placement of an autogenous bone graft along the transverse processes.
3. The pseudarthrosis rate without instrumentation is estimated to be between 25 and 45%.
4. Instrumentation lowers the pseudarthrosis rate (15–25%).
5. There is still some preserved motion after this procedure because of the intact disk anteriorly.
   a. If the disk is the main source of pain, patients may demonstrate persistent back pain (controversial).

B. Interbody fusion.
   1. Principles.
      a. Resection of diseased intervertebral disk:
         (1) In appropriately selected patients with diskogenic back pain, removing the intervertebral disk should resolve patient symptoms.
      b. Rigid instrumentation and bone grafts provide initial stability to the anterior column.
         (1) Posterior fixation with pedicle screws provides additional fixation for selected cases.
      c. A solid interbody fusion generally occurs at 6 months to 1 year following surgery.
   2. Techniques.
      a. Posterior lumbar interbody fusion (PLIF) **(Fig. 17.4** and **Fig. 17.5)**.
         (1) Extraction of the disk through a posterior approach via a wide laminectomy with preservation of the facet joint.
         (2) The approach can be widened with the removal of the lower third of the inferior facet and medial two-thirds of the superior facet.
         (3) This is followed by posterior instrumentation.

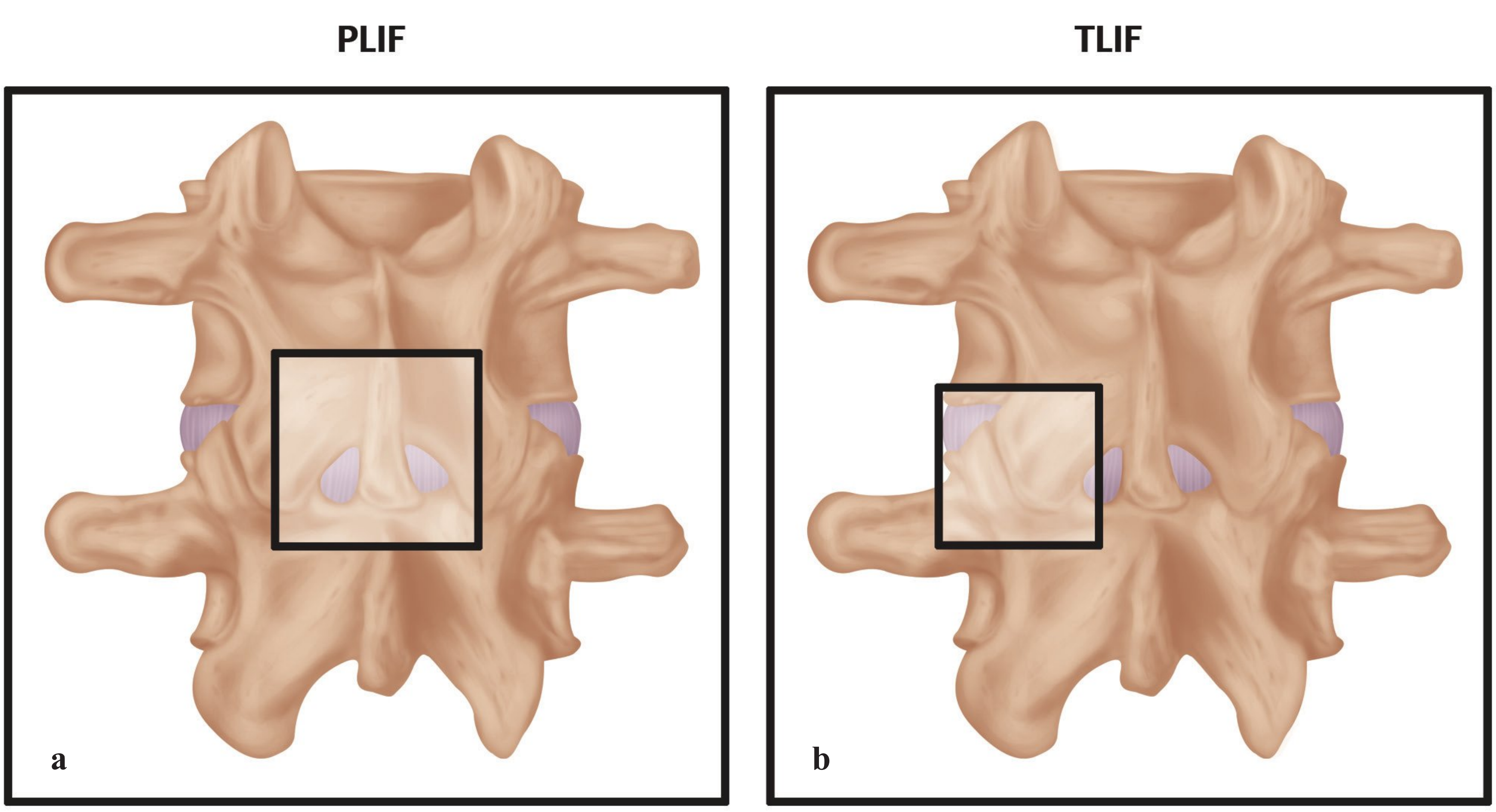

Fig. 17.4 Bony resection required in a **(a)** posterior lumbar interbody fusion and **(b)** transforaminal lumbar interbody fusion.

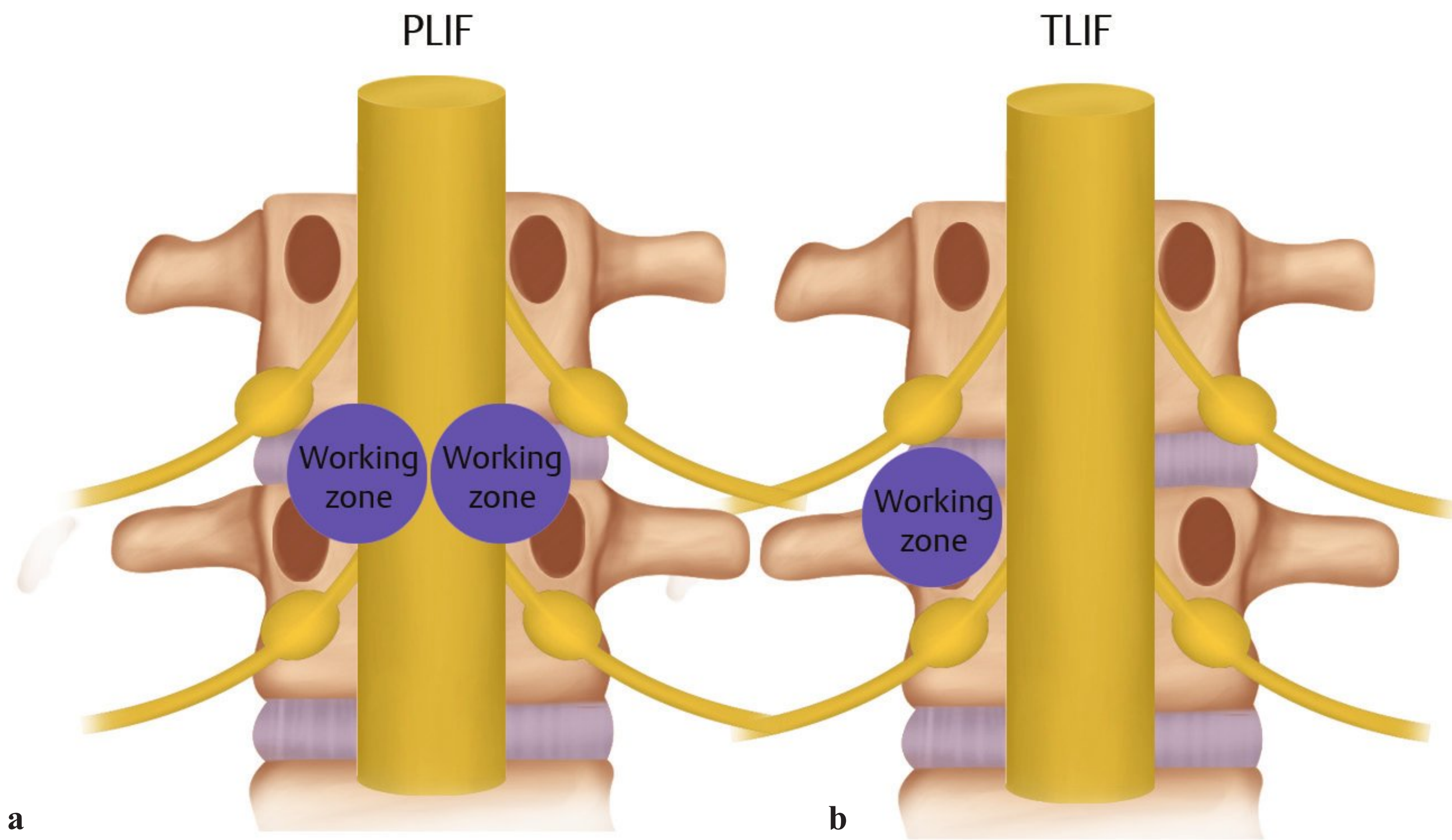

Fig. 17.5 Working zones of the **(a)** posterior lumbar interbody fusion and **(b)** transforaminal lumbar interbody fusion in relation to the neurovascular structures.

b. Transforaminal lumbar interbody fusion (TLIF) **(Fig. 17.6** and **Fig. 17.7).**
   (1) Decreased/minimal manipulation of neural elements when compared with PLIF.
   (2) A partial or complete facetectomy is performed to enable access to the disk space.
   (3) Can be performed bilaterally.
c. Anterior lumbar interbody fusion (ALIF) **(Fig. 17.8** and **Fig. 17.9).**
   (1) Indirect decompression of the exiting roots with the restoration of disk space and foraminal height.
   (2) Limited decompression of posterior elements when compared with PLIF and TLIF procedures.
   (3) Fusion occurs readily with this approach because of compression on the graft.
   (4) Can be used in a revision setting after a previous posterior surgery to avoid dissection of scar tissue.
   (5) Approaches:
      (a) Open left retroperitoneal approach.
      (b) Open transperitoneal approach.
      (c) Laparoscopic approach.
d. Lateral lumbar interbody fusion **(Fig. 17.10).**
   (1) Lateral retroperitoneal approach.
   (2) Considered a variant of ALIF.
   (3) Can be used for thoracic and lumbar interbody fusion.
   (4) Associated with a significantly reduced risk of injury to the great vessels.

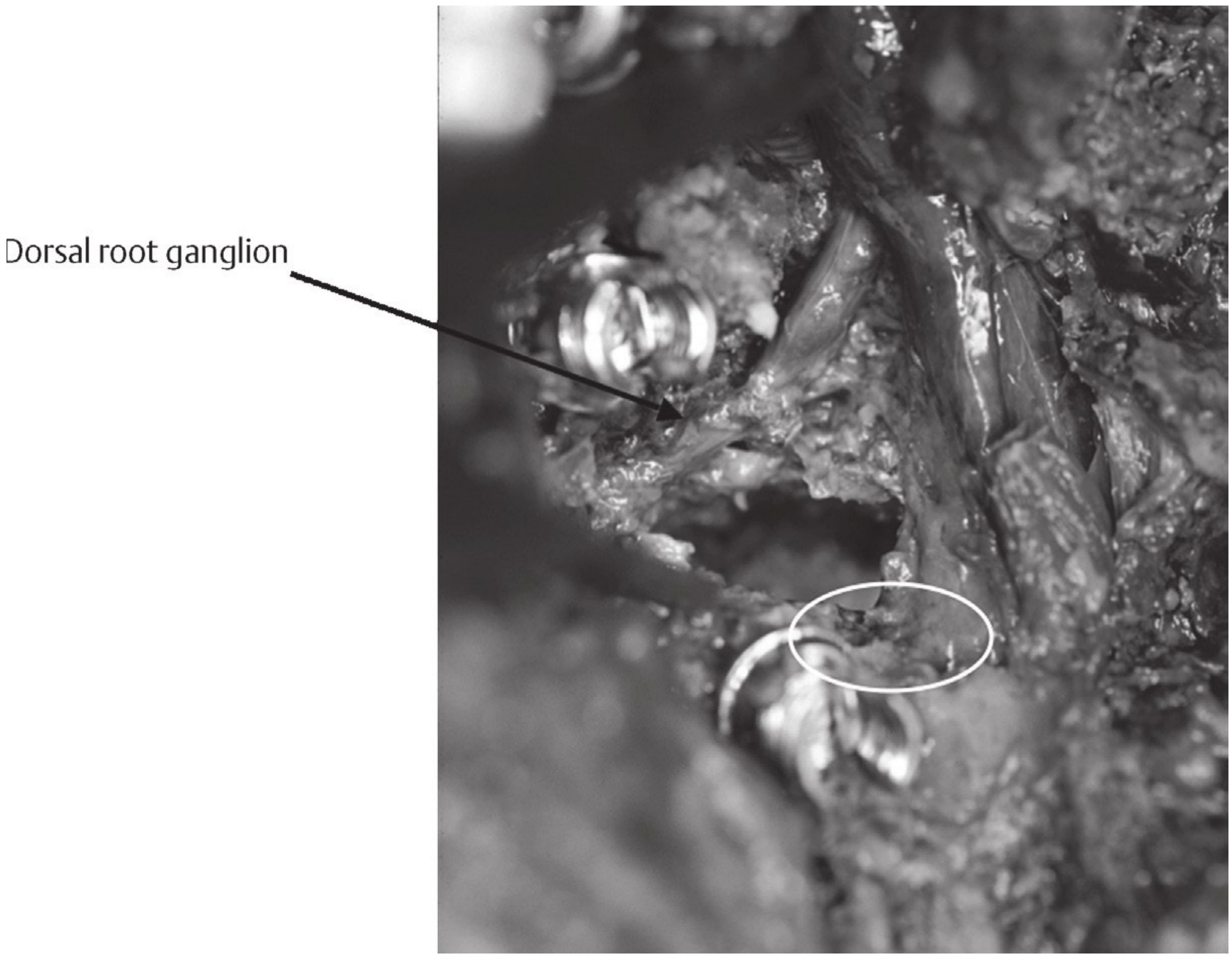

Fig. 17.6 Intraoperative photograph of the L4–L5 interspace demonstrating the dorsal root ganglion of the L4 nerve root (exiting).

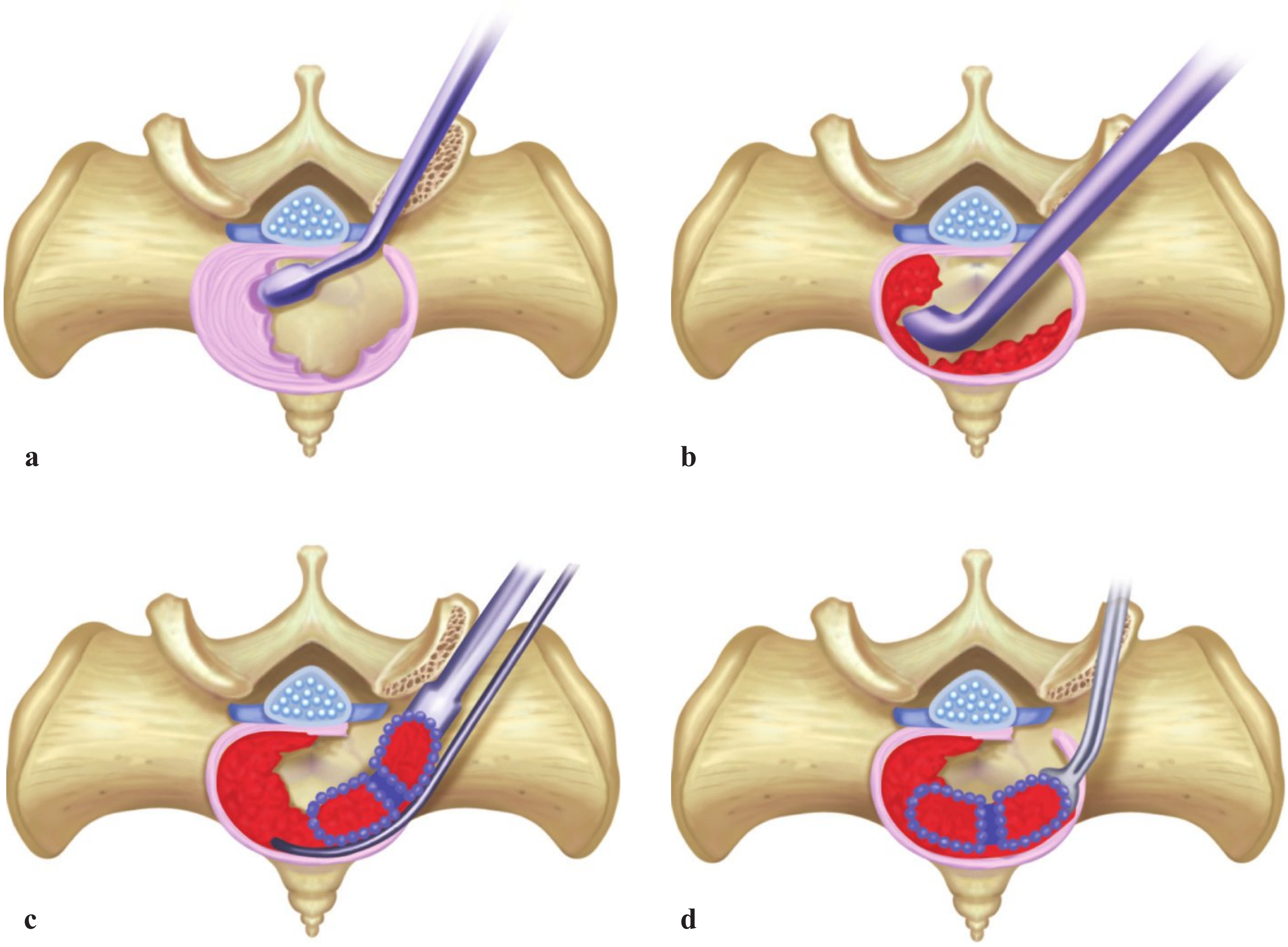

Fig. 17.7 **(a–d)** Removal of the disk material and placement of the interbody cage with bone graft material.

Fig. 17.8 Postoperative lateral radiograph of an anterior LT Cage (Medtronic) at L5–S1.

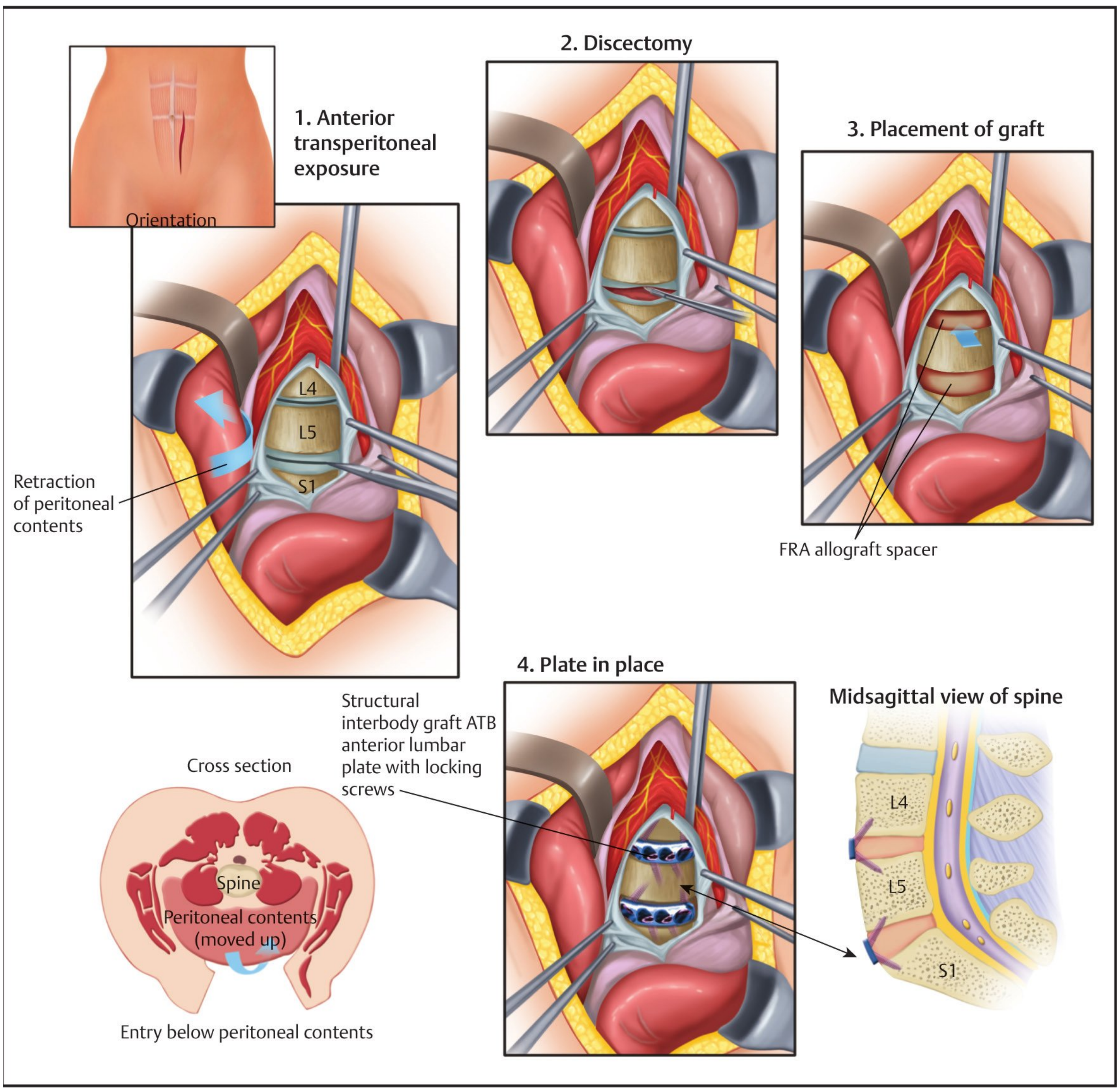

Fig. 17.9 Anterior lumbar interbody fusion procedure.

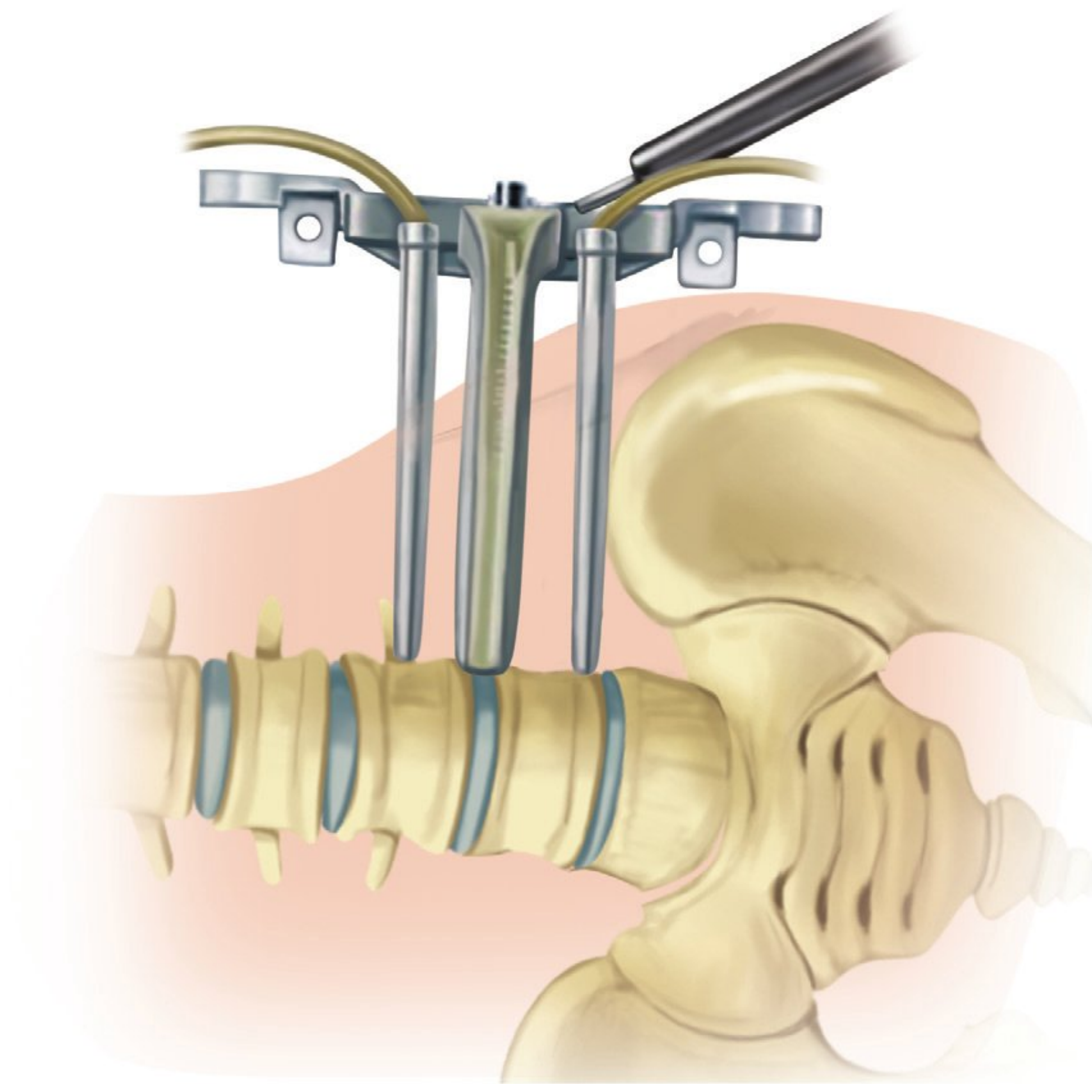

Fig. 17.10 Exposure for the lateral lumbar interbody fusion.

(5) The disk space is reached through a transpsoas approach.
 (a) Risk of injury to the lumbar plexus.
 (b) The genitofemoral nerve is most commonly injured (postoperative groin and thigh paresthesia).
 (c) Intraoperative neuromonitoring is critical during passage through the psoas.

e. Axial lumbar interbody fusion (AxiaLIF).
 (1) Minimally invasive technique that uses the presacral space to create a surgical working plane.
 (2) Can only address L4 to S1 disk pathology.
 (3) Risk of rectal injury, sacral fracture, and pelvic hematoma.
 (4) Special implants provide axial compression between vertebral bodies.
 (5) Limited evidence in the published literature has demonstrated variable outcomes and fusion rates.

VII. Motion-preserving procedures (total disk arthroplasty).
 A. Principles.
 B. Biomechanical studies have demonstrated that motion-preservation devices are associated with lower stresses on adjacent segments when compared with traditional fusion techniques in an effort to reduce adjacent segment degeneration.
 C. Indications:
  1. Indicated for one- or two-level diskogenic back pain without radiculopathy.

D. Outcomes compared with lumbar arthrodesis:
   1. Mid- and long-term data are now available to compare lumbar total disk replacement (TDR) with fusion procedures.
      a. TDR has demonstrated comparable safety and efficacy to lumbar fusion procedures at 7 years.
      b. Early experience with TDR has demonstrated satisfactory clinical outcomes with acceptable complication (14.4%) and reoperation (7.2%) rates.
      c. Superiority of TDR over lumbar fusion has not been proven with regard to adjacent-level degeneration.
      d. Studies have reported revision rates between 7.7 and 32.1% at 5 years (up to 39.3% at 10 years), depending upon the device.

VIII. Conclusions.

A. Low back pain is a multifactorial issue.
B. Careful patient selection is the key to successful clinical outcomes in patients with nonspecific low back pain.
   1. Pathological findings in imaging studies must be carefully assessed and correlated with the patient's history and physical exam findings.
   2. Psychosomatic evaluation may play an important role in selecting patients who undergo surgery for lower back pain.
C. Nonspecific low back pain remains a controversial area with limited understanding of the pathoanatomy and benefits of the available treatment options.
D. Numerous surgical techniques exist for the attainment of lumbar arthrodesis.
E. Motion preserving devices (TDR) have been demonstrated to be a safe and effective alternative to lumbar fusion surgery. However, the theoretical advantage of reducing adjacent-segment degeneration remains unproven.

Suggested Reading

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18 Lumbar Spinal Stenosis

18.1 General Considerations

I. Introduction.

A. Definition: narrowing of the spinal canal (central stenosis), lateral recess (lateral recess stenosis), or foramen (foraminal stenosis) with neural impingement that produces symptoms of neurogenic claudication or radiculopathy.

B. Degenerative spinal stenosis evidenced on imaging studies is significant only if clinically symptomatic.

C. More common after the fifth decade.

D. Men more affected than women.

E. Associated with disk degeneration.

II. Classification.

A. Congenital: usually developmental and primarily central canal stenosis **(Fig. 18.1).**

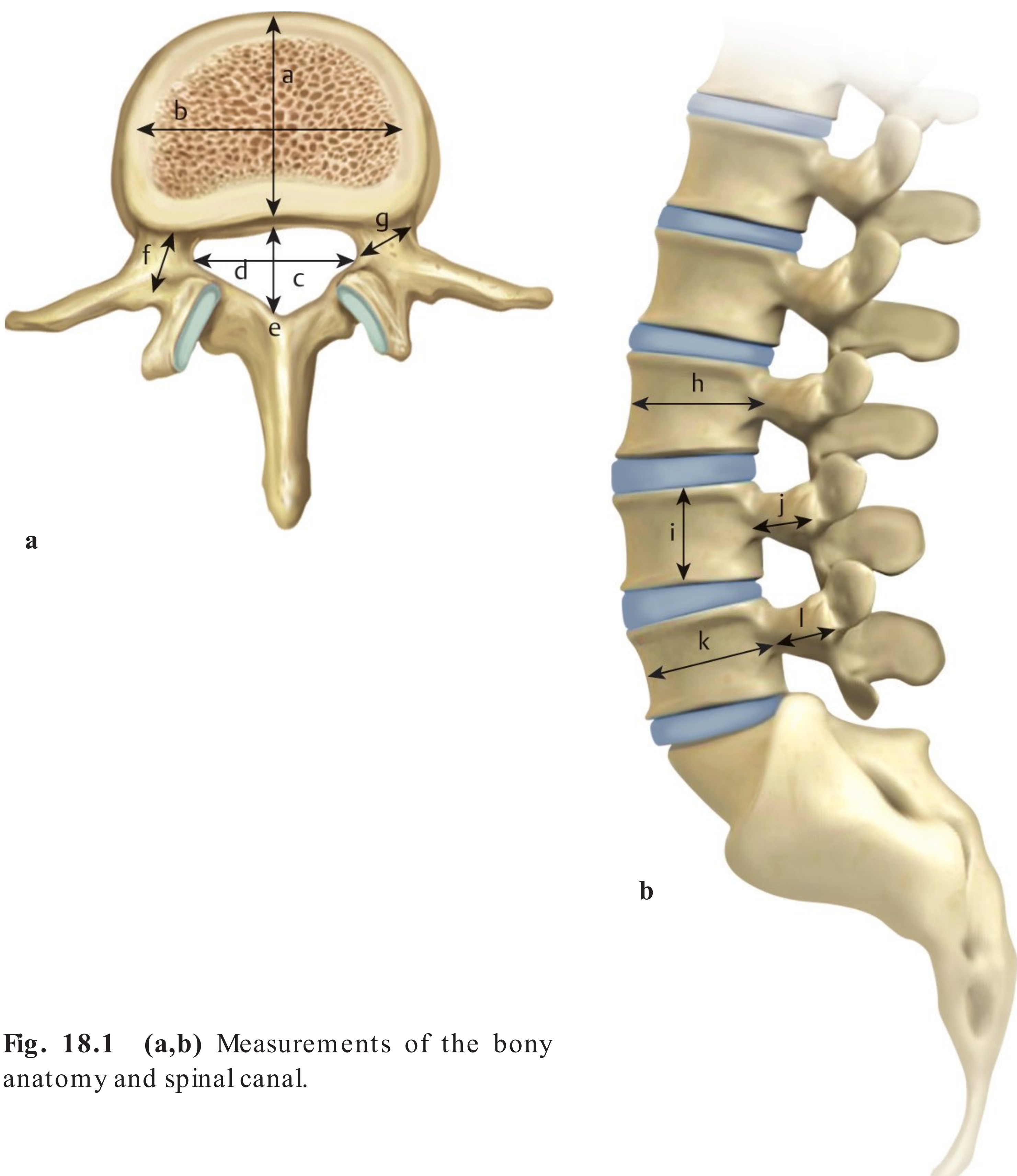


Fig. 18.1 (a,b) Measurements of the bony anatomy and spinal canal.

1. Characteristics.
   a. Earlier clinical presentation (fourth and fifth decade).
   b. Multilevel involvement.
   c. Fewer degenerative changes on imaging.
   d. Subtle anatomical changes that may compress the thecal sac.
2. Radiographic findings.
   a. Smaller cross-sectional spinal canal area.
   b. Shorter anterior-posterior (AP) pedicle length.
   c. The midline, axial AP canal diameter, medial–lateral vertebral body width, and sagittal AP canal diameters are smaller.
   d. Smaller AP pedicle length to vertebral body ratio.
   e. No difference in the AP vertebral body diameter, vertebral body height, canal width, or pedicle width.

B. Acquired (most common in the sixth decade).
1. Degenerative stenosis.
   a. Central stenosis.
      (1) Enlargement of the inferior articular process, ligamentum flavum, and intervertebral disk protrusion or herniation.
   b. Lateral stenosis.
      (1) Enlargement of the superior articular process and ligamentum flavum.
   c. Foraminal stenosis.
      (1) Narrowing of the foramen secondary to far lateral disk herniation and pars hypertrophy in isthmic spondylolisthesis.
2. Degenerative spondylolisthesis.
   a. For example, at the L4–L5 level, the L5 nerve is entrapped between the inferior articular process of L4 and the posterior aspect of the body of L5.
3. Combined.
   a. Disk herniation, superimposed on a degenerative or congenitally stenotic canal.
4. Iatrogenic.
   a. Postlaminectomy, postfusion, post–disk surgery.
5. Posttraumatic.
   a. Secondary to retropulsion of bone in a burst fracture and fracture dislocation.
6. Miscellaneous.
   a. Paget's disease, fluorosis.
   b. Dwarfism (achondroplastic).

III. Pathogenesis.

A. Variations of the spinal canal may predispose to spinal stenosis.
1. Three types of spinal canal.
   a. Round canal.
   b. Oval canal.
   c. Trefoil canal (15%).
      (1) Napoleon hat shape.
      (2) A trefoil canal predisposes to lateral recess stenosis.

B. Disk degeneration:
   1. Aging versus degeneration.
   2. Changes in the collagen, proteoglycans, and water content.
C. Facet joint involvement:
   1. Follows disk degeneration.
   2. Joint cartilage loss, hypertrophy, osteophytes, and subluxation.
D. Three-joint complex:
   1. Two posterior facet joints and the disk are all involved in the pathogenesis.
   2. Degenerative changes of the three-joint complex secondary to repeated rotational and compression injuries.
   3. Intervertebral disks develop circumferential and radial tears with a loss of disk height.
   4. Posterior joints undergo synovitis, cartilage destruction, and osteophyte formation.
      a. Results in capsular laxity, ligamentum flavum hypertrophy or buckling, and joint instability or subluxation.
   5. Instability:
      a. Degenerative spondylolisthesis.
      b. Retrolisthesis.
      c. Degenerative scoliosis.
      d. Rotatory subluxation.
E. L4 or L5 nerves are more typically affected:
   1. Greater compressive and shear stresses.
   2. Pedicles of the lower lumbar spine have convex inferior borders as compared with the concave inferior border in the upper lumbar spine.
   3. Disk degeneration is most common at L4–L5 and L5–S1.
F. Neural compression:
   1. Anatomical site of compression:
      a. Cauda equina and thecal sac (central canal).
      b. Traversing nerve root (lateral recess).
      c. Dorsal root ganglion (intervertebral foramen).
      d. Spinal nerve (extraforaminal).
   2. The cauda equina is compressed centrally from the anteroposterior direction at the intervertebral disk level..
      a. Bulging disk anteriorly.
      b. Ligamentum flavum and facet joints posteriorly.
   3. The nerve root can be compressed at multiple anatomical locations **(Fig. 18.2)**.
      a. Entrance zone.
         (1) Posterolateral herniated disk.
         (2) Hypertrophic superior articular process.
      b. Middle zone.
         (1) Pars interarticularis (spondylolysis).
   4. Exit zone (foramen) **(Fig. 18.3)**:
      a. Anatomy.
         (1) Bounded by vertebral body and disk anteriorly, pedicles superiorly and inferiorly, and pars, ligamentum flavum, and tip of superior articular process posteriorly.

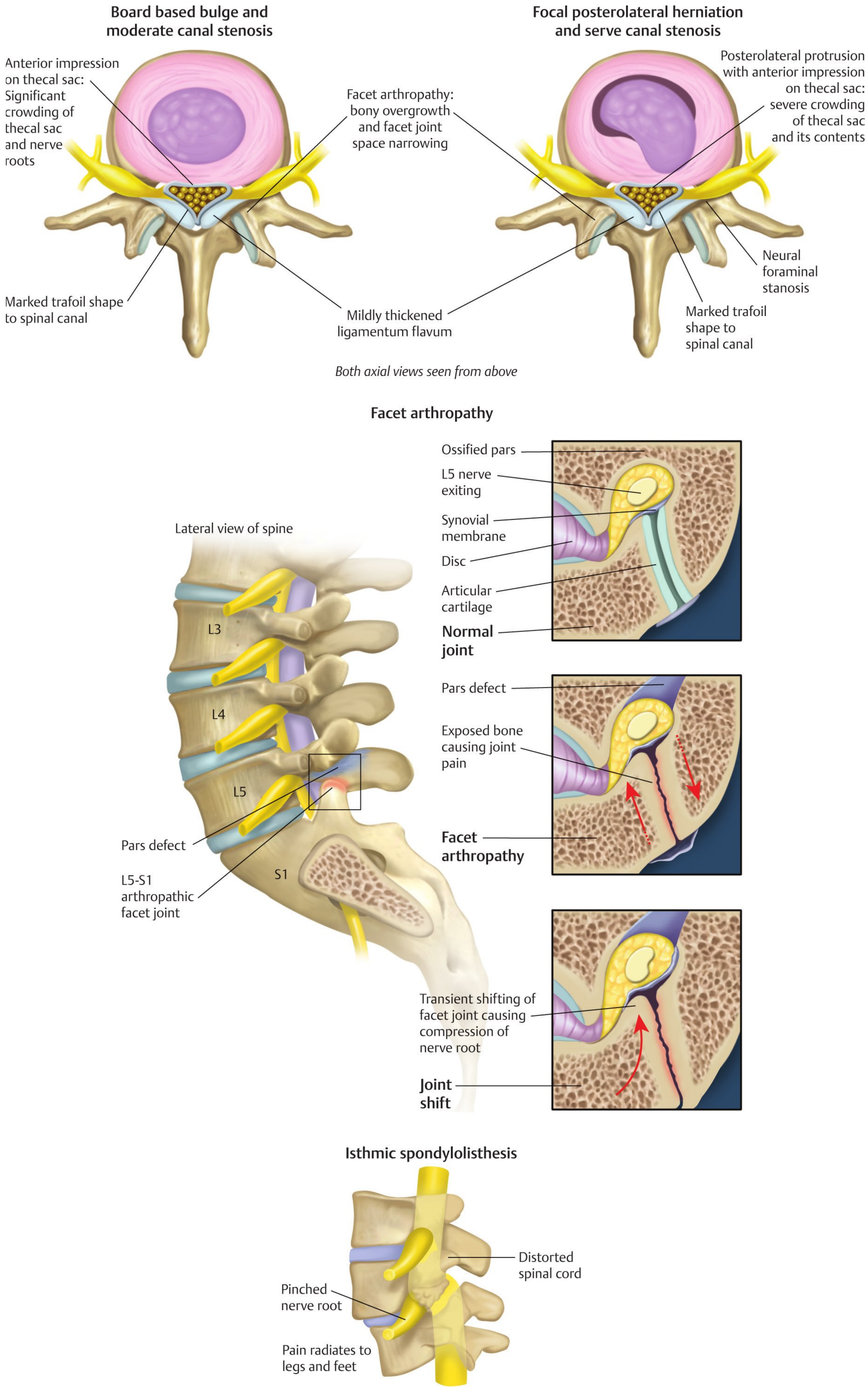


Fig. 18.2 Various anatomical locations for nerve root compression.

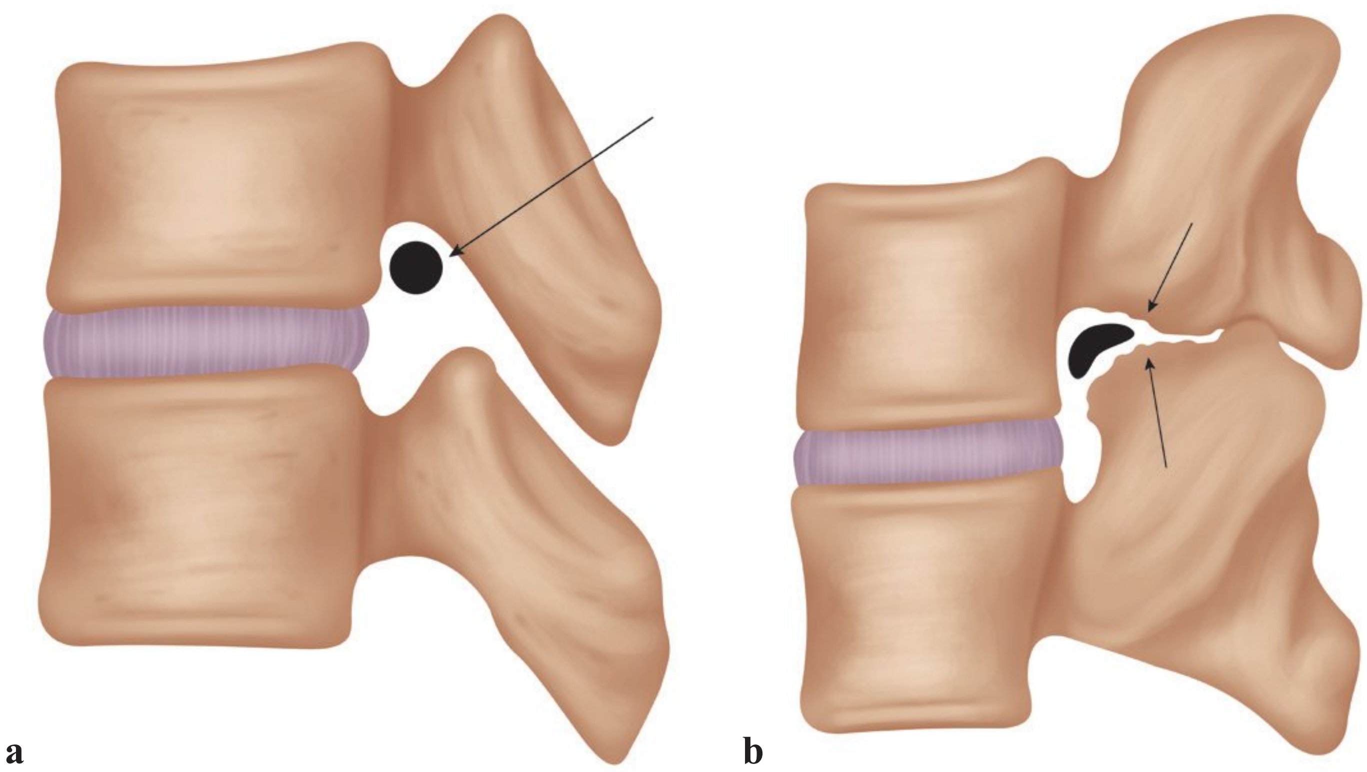


Fig. 18.3 **(a)** Anatomical location of the nerve root within the neuroforamen. **(b)** Neuroforaminal stenosis with nerve root impingement secondary to facet hypertrophy and osteophytic compression secondary to the superior articular process.

(2) Laterally herniated disk or annulus.
(3) Superior facet subluxation may compress the nerve against the pedicle, body, or bulging annulus.

b. Extraforaminal compression.
(1) Extreme lateral or extraforaminal herniated disk.
(2) Also known as far-out syndrome.
(a) The exiting nerve root is compressed between the transverse processes of L5 and the sacral ala in spondylolisthesis (L5–S1).
(3) Transverse process fracture or bone graft anterior to the transverse processes.

5. Dimensions of stenosis **(Fig. 18.4):**
a. Central.
(1) Absolute stenosis: midsagittal lumbar diameter < 10 mm.
(2) Relative stenosis: 10 to 13.5 mm.
b. Lateral recess.
(1) Less than 3 to 4 mm.
c. Foramen.
(1) Foraminal height < 15 mm.
(2) Posterior disk height < 3 mm (80%likelihood of nerve root compression).

6. Pathophysiology of radiculopathy:
a. Combination of compression and inflammation.
(1) Compression alone may not cause pain.
(2) Inflammatory mediators:
(a) Phospholipase A2.
(b) Neuropeptides.
b. Dynamic instability.
(1) Spinal canal and foramen.

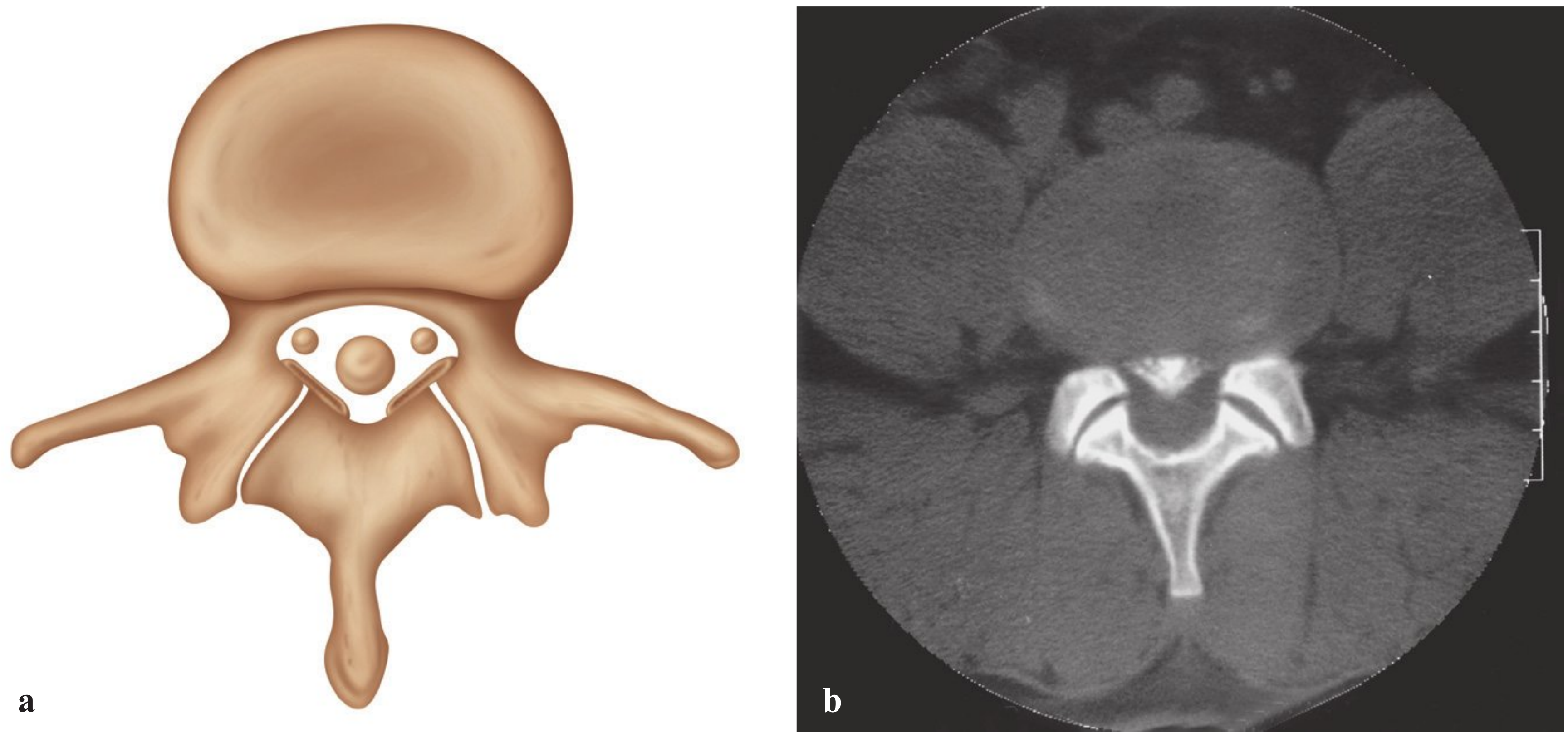


Fig. 18.4 **(a)** Cross-sectional view of the normal anatomy of the neural structures within the spinal canal. **(b)** Computed tomographic myelography demonstrating central stenosis with significant ligamentum flavum hypertrophy.

c. Venous stasis.
d. Arterial ischemia.
e. Nutritional deficit.
(1) Abnormal cerebrospinal flow.
f. Percent constriction of cauda equina.
(1) Twenty-five percent: no deficits.
(2) Fifty percent or greater: motor deficits and complete loss of somatosensory evoked potentials (SSEPs).

IV. Clinical findings.
A. Pain.
1. Variable:
a. Monoradiculopathy.
b. Bilateral neurogenic claudication.
c. Atypical leg pain.
d. Cauda equina symptoms.
2. Typically in the lower back, buttock, and lower extremities.
3. Pain is worse with standing and walking.
4. Relieved by rest, flexed posture, and sitting.
5. The history is the key in making the diagnosis of spinal stenosis.
B. Claudication-like symptoms in 50%
1. One must rule out vascular claudication.
2. In vascular claudication:
a. Relief after rest is more prompt.
b. Flexion of the spine does not relieve symptoms.
(1) For example, bicycling and walking uphill may not cause neurogenic claudication because the spine is flexed.
3. Vascular and neurogenic claudication may coexist.

C. Physical examination.
   1. Paucity of objective findings.
   2. The sciatic tension sign is often negative.
   3. Neurological deficits may or may not be present.
   4. The most important sign is painful and limited extension.
   5. Thorough abdominal and vascular examination should be done routinely.
      a. Vascular changes may demonstrate lower extremity ulcer formation, hair loss, edema, and skin mottling.
D. Diagnostic tools.
   1. Plain radiographs.
      a. Disk space narrowing or degenerative disk disease.
      b. End plate osteophytes and sclerosis.
      c. Facet enlargement or osteophyte formation.
      d. Narrowed neuroforaminal canal.
      e. Loss of lumbar lordosis.
   2. Magnetic resonance imaging **(Fig. 18.5).**
      a. Best modality for evaluating lumbar spinal stenosis.
      b. Excellent for soft tissue details, but bony margins are better demonstrated by CT scans.
E. Congenital stenosis.
   1. Lumbar spinal stenosis typically affects individuals over 60 years of age, but younger patients with congenital stenosis present in the fourth and fifth decades.
   2. The presenting symptoms are somewhat different in that many suffer with low back pain (LBP) with or without neurogenic claudication.

Fig. 18.5 Magnetic resonance imaging demonstrating spinal stenosis secondary to a herniated lumbar disk. (From Imhof H, ed. Spinal Imaging [Direct Diagnosis in Radiology]. Stuttgart, Germany: Georg Thieme Verlag; 2008: Fig. 3.35. Reproduced with permission.)

3. LBP is typically worse with standing and walking as compared with diskogenic LBP, which is typically worse with sitting.
4. The spinal canal is narrow at multiple levels with short pedicles. The sagittal canal to vertebra ratio is < 0.4 (Singh et al).
5. The patients with congenital stenosis presenting with "neurogenic LBP" are frequently misdiagnosed as diskogenic LBP and are given inappropriate treatment.
6. Treatment is initially conservative as for other stenotic patients, but if there is no response, multilevel laminectomy is recommended rather than fusion.

V. Differential diagnoses.
   A. Trauma (sprains, strains, compression fractures).
   B. Infections (vertebral osteomyelitis).
   C. Inflammatory disorders.
   D. Congenital defects (achondroplasia).
   E. Metabolic (osteoporosis, Paget's disease).
   F. Degenerative (disk herniation, facet syndrome).
   G. Neoplasms (intraspinal, bone tumors, and metastasis).
   H. Neurological disorders (peripheral neuropathies).
   I. Circulatory (abdominal aortic aneurysm, vascular claudication).
   J. Myofascial syndromes.
   K. Psychoneurotic problems.

VI. Treatment.
   A. Conservative.
      1. Nonsteroidal anti-inflammatory drugs.
      2. Lumbosacral corsets.
      3. Flexion exercises.
      4. Epidural or foraminal injection.
   B. Surgery.
      1. Indications.
         a. Cauda equina syndrome.
         b. Progressive motor weakness.
         c. Limb pain that is unresponsive to conservative treatment and if symptoms significantly affect quality of life.
      2. Surgical techniques **(Fig. 18.6).**
         a. Decompression: the key is adequate decompression while preserving stability of the motion segment by undercutting the facet joint and preserving the pars interarticularis. For multilevel decompression, care must be taken to preserve the pars interarticularis in the upper lumbar spine, because the pars is more medial. Therefore, laminectomy is more lateral at the facets, which is the location for lateral recess stenosis, and more medial or narrower at the pars.
            (1) Central stenosis:
               (a) Laminectomy **(Fig. 18.7).**
            (2) Lateral recess:
               (a) Remove an overgrown superior facet by undercutting.
            (3) Foraminal decompression: foraminal decompression requires more facet removal, but every effort must be made to preserve the facet joint

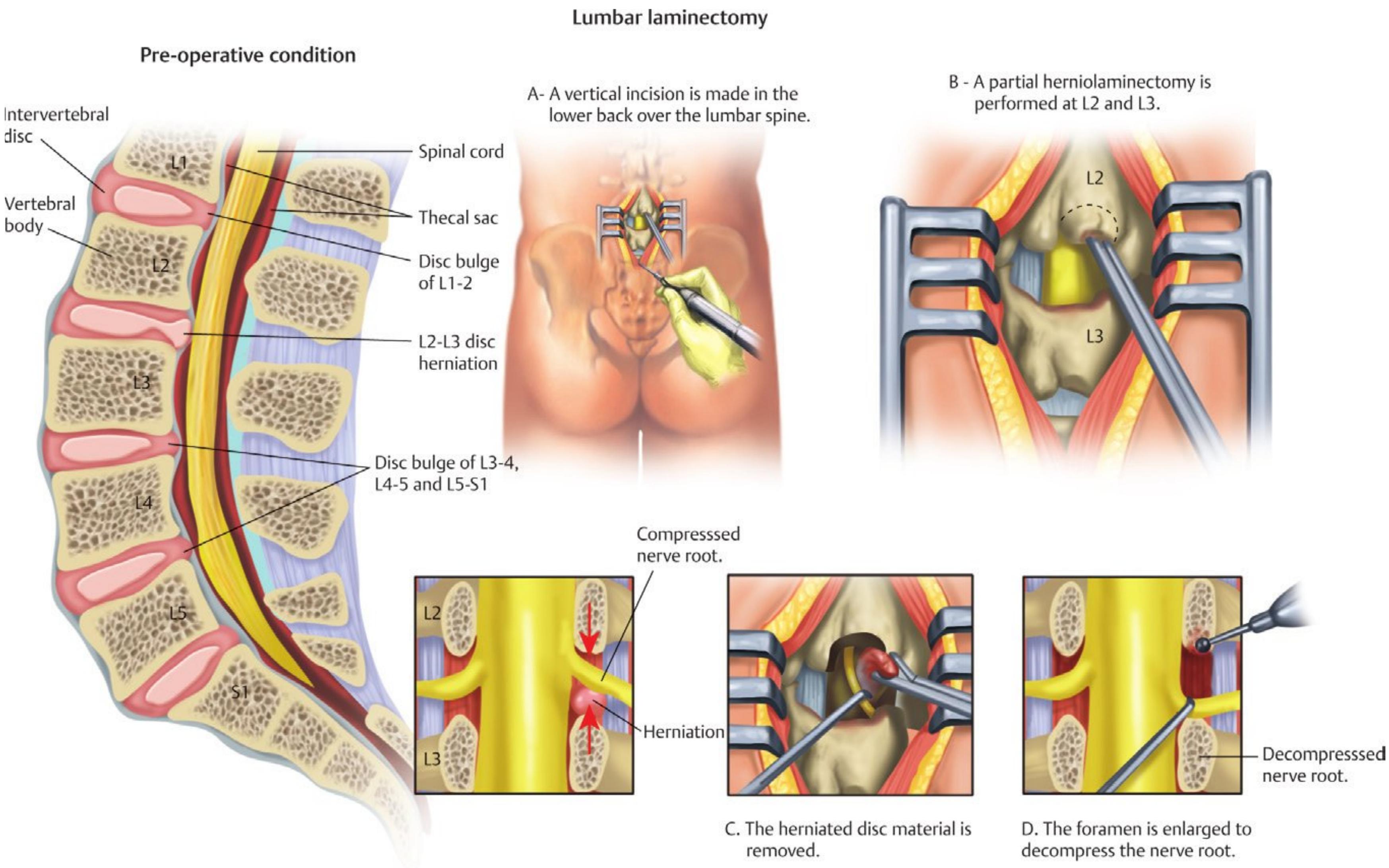


Fig. 18.6 Steps for a lumbar laminectomy.

as much as possible by undercutting and using special instruments, such as a curved foraminotomy rongeur or rasp **(Fig. 18.8)**.

(a) If the nerve root is tight after laminectomy and facet undercutting, additional sites may be responsible for nerve compression.

i. Superior facet against posterior vertebral body.

ii. Superior facet against pedicle.

iii. Superior facet or pedicle against bulging lateral annulus.

iv. Inferior facet and vertebral body (degenerative spondylolisthesis).

v. Transverse processes of L5 and sacral ala (“far-out syndrome”).

b. Lumbar fusion:

(1) Approaches.

(a) Posterior: posterolateral fusion, posterior lumbar interbody fusion, and transforaminal lumbar interbody fusion.

(b) Anterior: anterior lumbar interbody fusion, extreme lateral lumbar interbody fusion.

(2) Fusion is recommended when there is stenosis in conjunction with the following conditions:

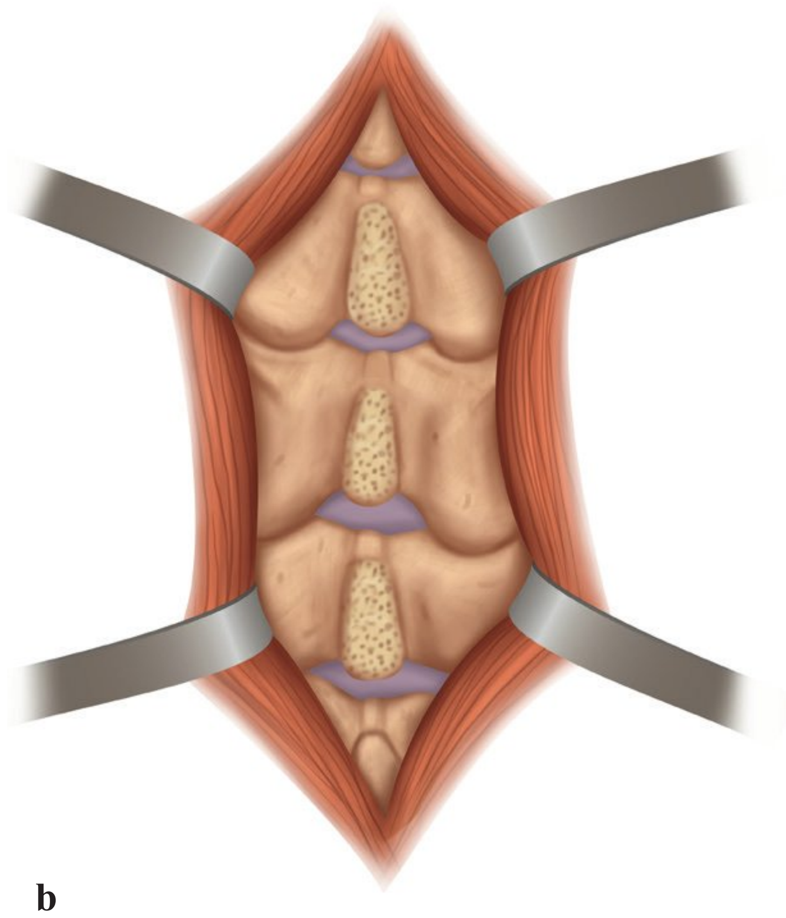
(a) Unstable degenerative scoliosis or kyphosis.

i. Progressive curves.

ii. Curves > 20°.

iii. Loss of sagittal balance and lumbar lordosis.


a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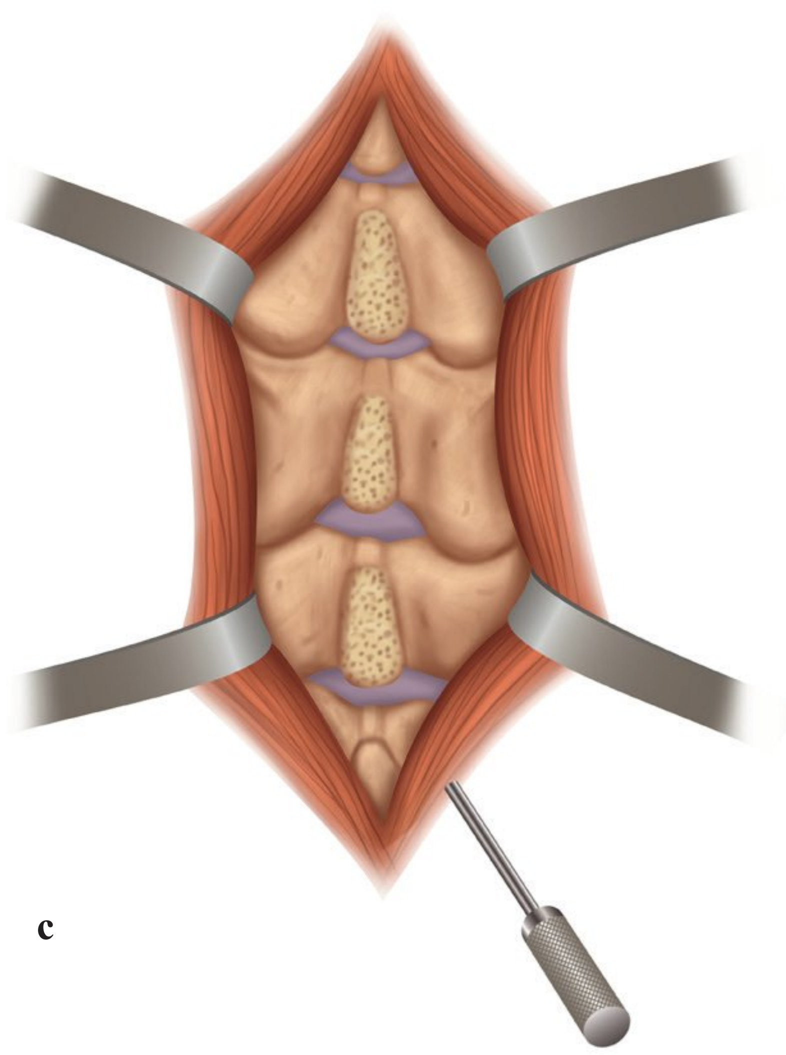
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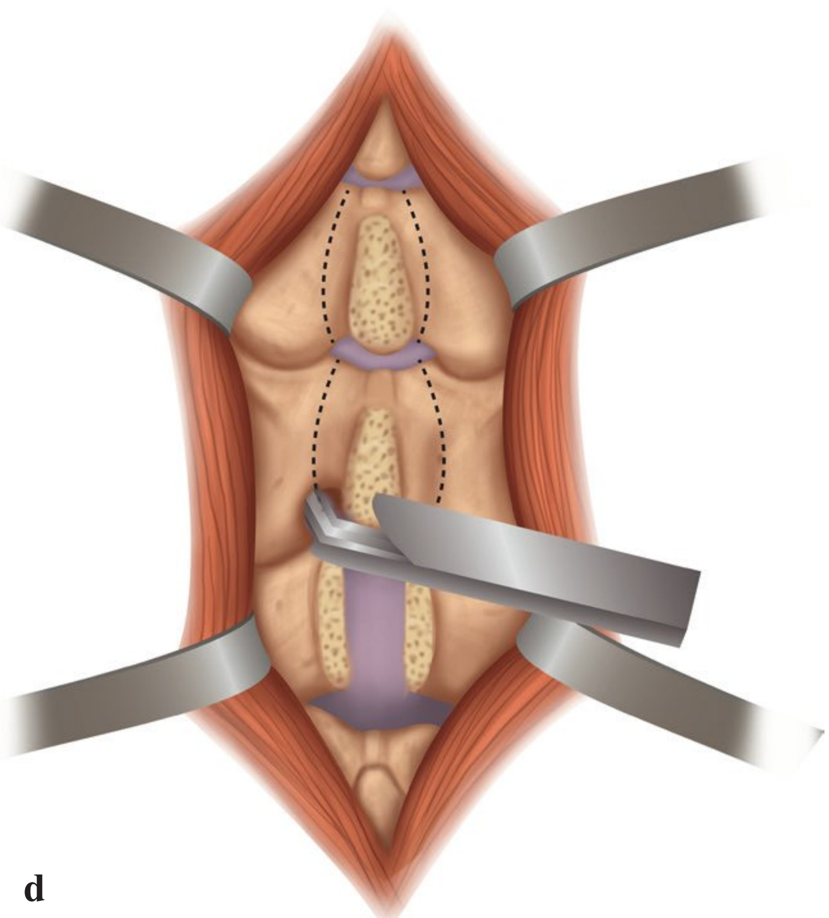
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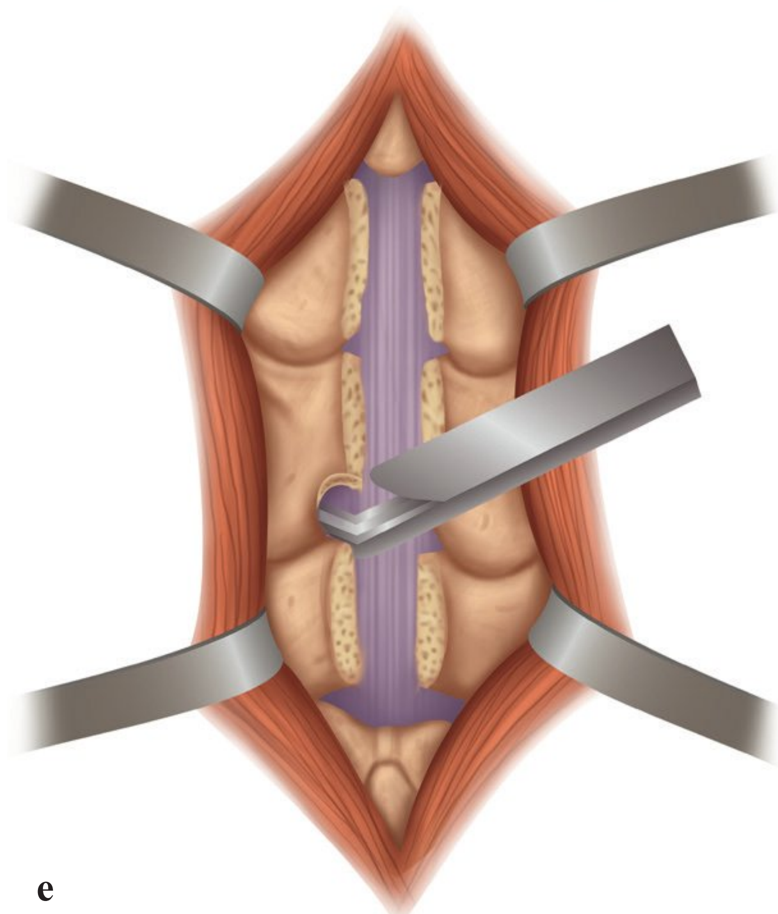
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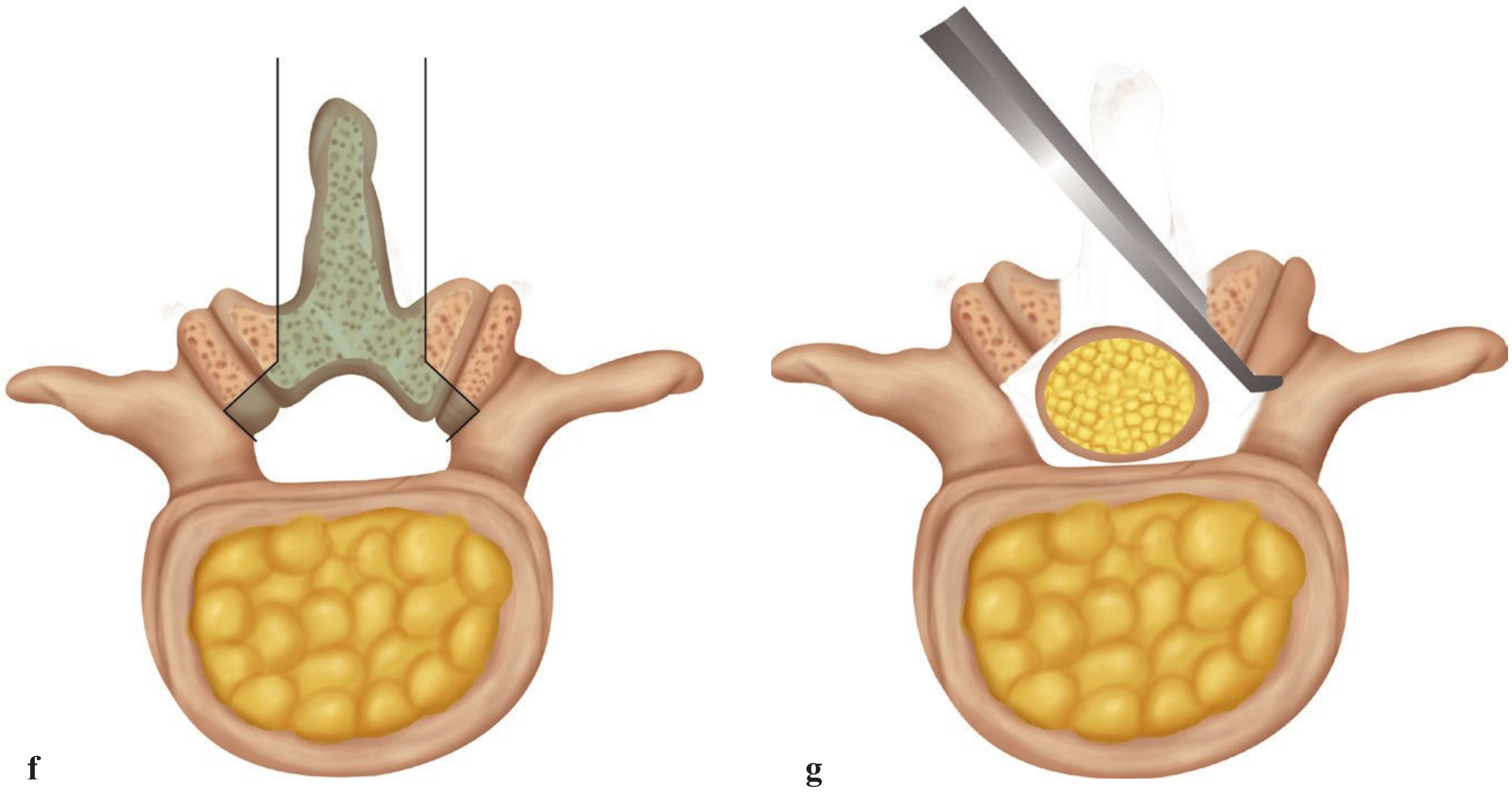

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Fig. 18.7 **(a)** Standard midline incision made for a lumbar laminectomy. **(b)** The spinous processes have been removed. The shaded area depicts the lamina needed to be removed to accomplish a central decompression. **(c)** A curette is used to remove the ligamentum flavum from the undersurface of the lamina to gain access to the spinal canal. **(d)** A Kerrison rongeur is used to remove the lamina in a piecemeal fashion. **(e)** The Kerrison rongeur is then used to undercut and remove bone and the ligamentum flavum within the lateral recess. **(f)** A Kerrison rongeur is gently placed into the neuroforamen to undercut the superior-articular process to create space for the exiting nerve root. **(g)** The shaded areas represent the area of bone and soft tissue needed to be removed to ensure a thorough central and lateral recess decompression.

iv. Lateral listhesis.
v. Flexible curves.
vi. Patients with radicular symptoms on the concave side of the curve.

(b) Degenerative spondylolisthesis.
(c) Iatrogenic instability.
  i. Greater than 50%facet resection bilaterally.
  ii. Removal of one complete facet complex.
(d) Recurrent same-level or adjacent-level decompressions.

(3) Motion preservation.
(4) Interspinous spacers.
  (a) Approved by the Food and Drug Administration.
  (b) The mechanism of action is local distraction and kyphosis that theoretically reduces soft tissue infolding into the central canal (ligamentum flavum).
  (c) Indications:
    i. Neurogenic claudication pain that is relieved when the spine is flexed.
    ii. Spondylolisthesis up to grade 1.5 **(Table 18.1).**

Table 18.1 Comparison of vascular and neurogenic claudication

Findings	Vascular	Neurogenic
Claudication distance	Fixed	Variable
Relief after cessation of activity	Immediate	Delayed
Relief of pain	Standing	Flexion or sitting
Uphill walking	Pain	No pain
Bicycling	Pain	No pain
Location and radiation	Distal to proximal	Proximal to distal
Atrophy	Rare	Occasional
Back pain	Uncommon	Common
Skin	Loss of hair	Normal

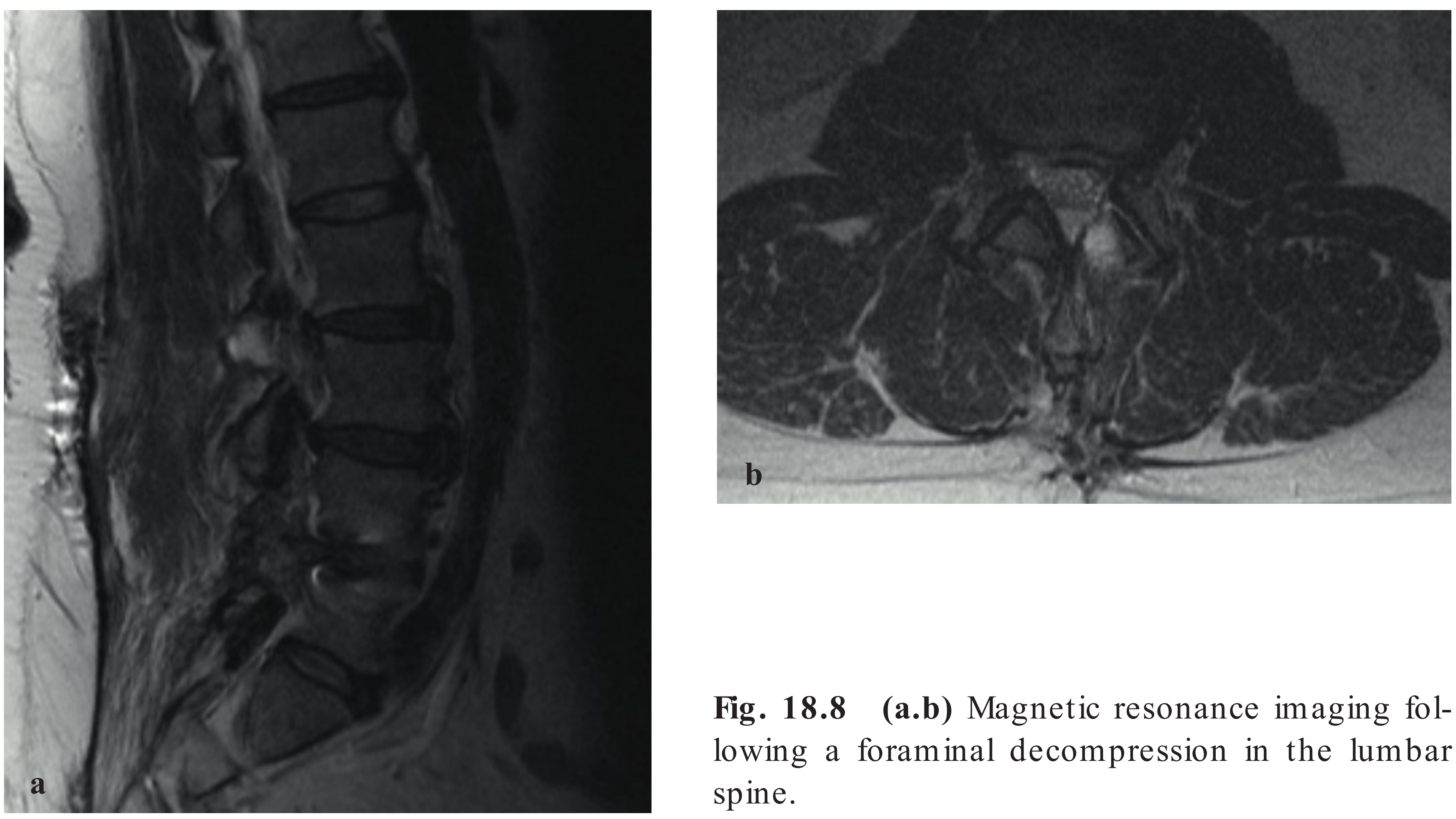

Fig. 18.8 **(a.b)** Magnetic resonance imaging following a foraminal decompression in the lumbar spine.

C. Spine patient outcomes research trials (SPORT).
   1. Patient populations.
      a. Spinal stenosis without spondylolisthesis.
      b. Minimum 12 weeks of symptoms.
      c. Randomly assigned to operative and nonoperative cohorts.
   2. Operative versus nonoperative outcomes in patients with spinal stenosis.
      a. The majority of patients (89%) underwent decompression surgery without fusion.

b. As-treated analysis demonstrated that patients treated surgically reported greater improvement in pain and function through 2 years compared with those treated nonoperatively.

3. Controversies.
   a. High crossover rate:
      (1) Forty-nine percent of patients in the nonoperative cohort underwent surgery.
      (2) Thirty-two percent of patients in the surgical cohort did not undergo surgery.
   b. Nonoperative management was not standardized.
      (1) Included physical therapy, chiropractic, injections.
   c. Intent-to-treat versus as-treated analysis:
      (1) The intent-to-treat analysis compared patients according to their assigned cohort.
         (a) This demonstrated no difference in primary outcomes between groups in patients with disk herniation.
         (b) In this analysis, if a patient was assigned to the nonoperative treatment arm, but went on to have surgery, that patient was counted as a nonoperative patient.
      (2) The as-treated analysis compared patients according to the treatment ultimately received regardless of their assigned treatment arm.
      (3) Patients who underwent surgery demonstrated improved and sustained outcomes compared with those treated nonoperatively at 4 years.

Suggested Reading

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Pearson A, Lurie J, Tosteson T, Zhao W, Abdu W, Weinstein JN. Who should have surgery for spinal stenosis? Treatment effect predictors in SPORT. Spine 2012;37(21):1791–1802

Skidmore G, Ackerman SJ, Bergin C, et al. Cost-effectiveness of the X-STOP® interspinous spacer for lumbar spinal stenosis. Spine 2011;36(5):E345–E356

Weinstein JN, Tosteson TD, Lurie JD, et al; SPORT Investigators. Surgical versus nonsurgical therapy for lumbar spinal stenosis. N Engl J Med 2008;358(8):794–810

Weinstein JN, Tosteson TD, Lurie JD, et al. Surgical versus nonoperative treatment for lumbar spinal stenosis four-year results of the Spine Patient Outcomes Research Trial. Spine 2010;35(14):1329–1338

19 Lumbar Spondylolisthesis

19.1 General Considerations

I. Introduction.
  A. Definitions.
    1. *Spondylolisthesis*—displacement of one vertebra on another.
    2. *Spondylolysis*—lytic defect in the pars interarticularis.
      a. Bone between the superior and inferior articular processes.

II. General considerations.
  A. Hereditary factors.
    1. The familial tendency is stronger in dysplastic type (94%) compared with isthmic type (32%).
    2. More common in white males (6.4%), less common in black females (1.1%).
      a. Higher incidence among Eskimo population (up to 45%).
    3. Association with spina bifida of the sacrum and dysplastic changes of the bony elements in high dysplastic types.
  B. Epidemiology.
    1. More common in men than in women.
    2. Higher incidence among football players, female gymnasts, and soldiers carrying heavy backpacks.
    3. Lower incidence among nonambulatory patients.

III. Biomechanics.
  A. Abrupt change in stiffness across the lumbosacral junction.
    1. The pars interarticularis is strong but susceptible to fatigue fractures.
      a. Especially with extension injuries.
    2. Shear stress at the pars can be increased by flexion contractures of the hip and secondary hyperlordosis.
      a. Pincerlike effect on L5 pars from the superior articular process of S1 and inferior articular process of L4.

IV. Classifications.
  A. Modified Wiltse **(Table 19.1).**
  B. Spinal deformity study group L5–S1 classification **(Fig. 19.1).**
    1. Based on the sacropelvic morphology, slip grade, and spinal balance.
    2. Type 1 and 2 spondylolisthesis carry a lower risk of progression compared with type 3.
    3. Reduction is likely warranted for types 5 and 6.
  C. Classification of spondylolisthesis (Marchetti–Bartolozzi).
    1. Developmental.
      a. Deficient "bony hook," anatomical abnormalities of the L5 pedicle, pars, inferior facets.
        (1) High dysplasia.
          (a) Severe bony anomalies with significant kyphosis.

Table 19.1 Wiltse spondylolisthesis classification

Type	Name	Description	Affected level
I	Congenital/dysplastic	Dysplasia of the sacrum, fifth lumbar arch, facets, or both	L5–S1
II	Isthmic/spondylolytic	Pars interarticularis defect	L5–S1
III	Degenerative	Degeneration of the facets and disk	L4–L5 (90%) L3–L4 or L5–S1 (10%)
IV	Traumatic	Fracture of the neural arch excluding the pars interarticularis	L5–S1
V	Pathological	Pathological lesion or generalized metabolic disturbance	Any level
VI	Iatrogenic	Iatrogenic disruption of the facet, ligament, disk, or bone	Any level

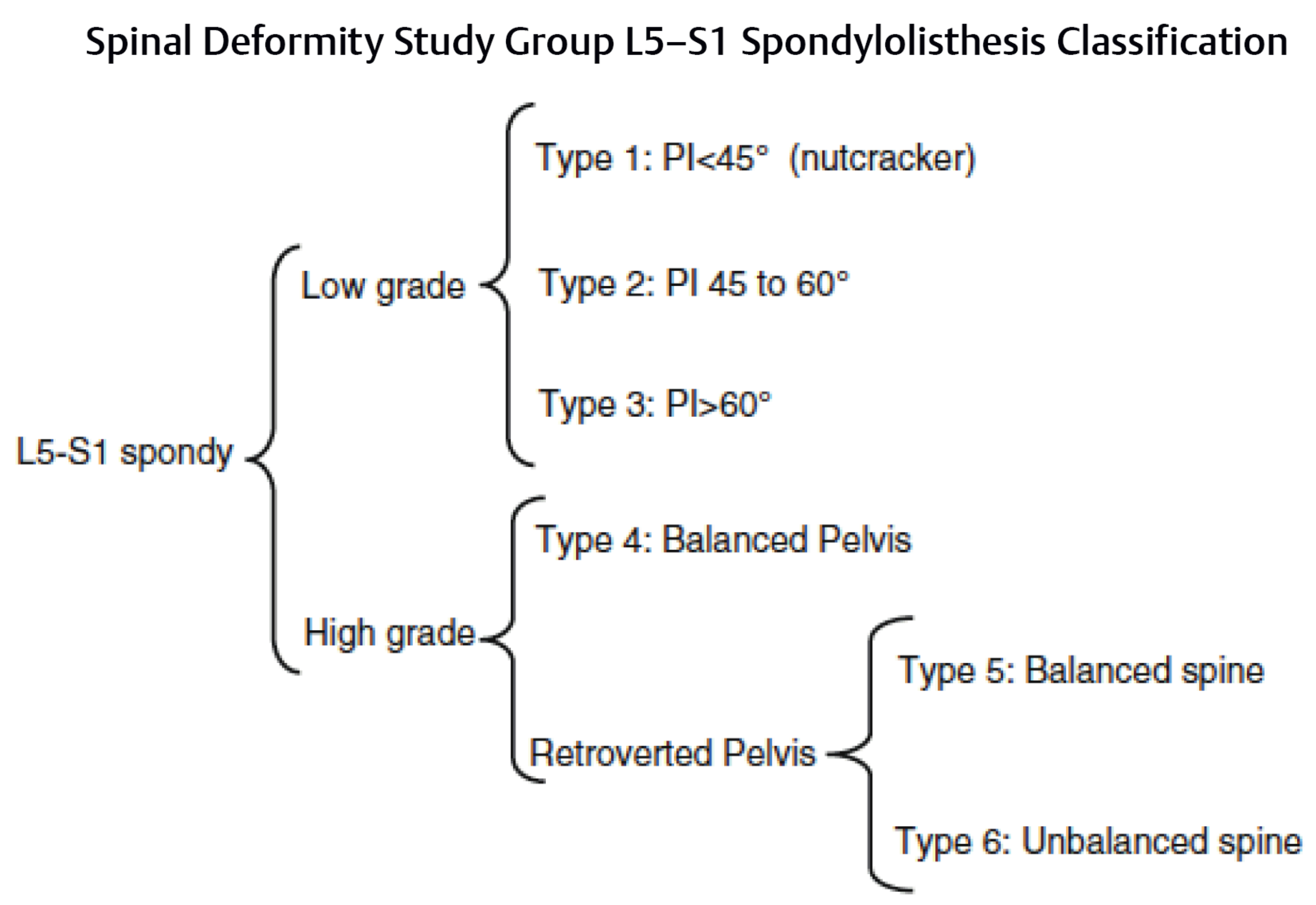

Fig. 19.1 Spinal deformity study group L5–S1 classification for spondylolisthesis.

(b) Common during 7 to 20 years of age.
(c) Compensatory lumbar hyperlordosis.
(2) Low dysplasia.
(a) Slower progression.
(b) Frequently asymptomatic.
(c) Disk degeneration aggravates the motion segment instability.
(3) Acquired.
(a) Traumatic (acute vs. chronic stress fractures).
(b) Postsurgical.
(c) Pathological.
(d) Degenerative.
(e) Specific entities (modified Wiltse classification).

2. Congenital or dysplastic (14%).
a. Epidemiology.
(1) Displacement occurs early.
(a) Typically during the adolescent growth spurt.
(b) Two:one female:male ratio.
(c) Genetic component.
i. Increased risk among all affected first-degree relatives.
b. Etiology.
(1) Congenital or dysplastic abnormality of the L5–S1 facet joint:
(a) Prevents proper articulation.
(b) Displacement is early but limited based on the intact posterior neural arch.
i. Increased rate of neurological symptoms (25–35%).
(2) The pars interarticularis is intact but poorly developed or elongated.
c. Clinical findings.
(1) Pain radiating into lower extremities.
(a) Little or no back pain.
(2) Cauda equina.
d. Treatment.
(3) Most congenital spondylolisthesis patients with progression of the slip require decompression and arthrodesis.

3. Isthmic spondylolisthesis.
a. Epidemiology.
(1) Most common spondylolytic disorder among children and young adults.
(a) Common from 7 to 20 years.
(b) Onset usually coincides with adolescent spurt, and progression occurs between 10 and 15 years of age.
(2) Most common at L5 over S1 vertebrae (95%).
(3) Most often asymptomatic.
(a) Low back pain and radiculopathy (L5 nerve root) may develop.
b. Clinical findings.
(1) Restricted forward flexion of the hips and back.
(2) Tight hamstrings.
(3) Flat buttock (vertical sacrum).

(4) Lumbosacral kyphosis.
(5) Compensatory lordosis.
(6) Anterior protrusion of the pelvis.
(7) Pelvic waddle gait.

c. Roentgenographic findings **(Fig. 19.2).**
(1) Defect at the pars interarticularis:
(a) Seen at the neck of the "Scottie dog" projection on oblique view.
(2) Trapezoidal L5 vertebral body:
(a) Rounded sacral dome.
i. On an anteroposterior (AP) view this appears as the reverse "Napoleon's hat" sign.
(3) Computed tomographic scan demonstrates the pars defects and stenosis.
(a) Single-photon emission computed tomography (SPECT) can detect metabolic activity in the region of the pars interarticularis defect.
(4) Magnetic resonance imaging (MRI) is the study of choice for assessing spinal stenosis.
(a) May demonstrate "wide canal sign."
i. Suggestive of a bilateral pars defect.
(5) Radiographic measurements:
(a) Meyerding classification.
i. Grade I: 0 to 25%slip.
ii. Grade II: 26 to 50%slip.
iii. Grade III: 51 to 75%slip.
iv. Grade IV: 76 to 100%slip.
v. Grade V: spondyloptosis or > 100%slip.

Fig. 19.2 An L4–L5 spondylolisthesis with neuroforaminal impingement of the L4 nerve root within the foramen.

(b) Slip angle **(Fig. 19.3).**
  i. The angle of kyphosis is measured as the angle between the superior end plate of L5 and a line perpendicular to the posterior border of the sacrum.
  ii. Most sensitive indicator of potential instability.
  iii. Correction of the slip angle is the most important goal of surgical reduction.
  iv. Correction of the slip is not important in achieving clinical success.
  v. In high-grade spondylolisthesis, an interbody may help achieve reduction.

(c) Lumbar index.
  i. Measurement of the wedging of the anterior L5 vertebral body
    - Ratio of the posterior and anterior height of the slipped vertebra.

d. Treatment
  (1) Activity modification (nonoperative).
    (a) Back and abdominal strengthening exercise.
    (b) Hamstring stretching.
    (c) Brace if persistent pain despite activity modification.
    (d) A positive bone scan or SPECT scan implies the potential for osseous healing via immobilization.

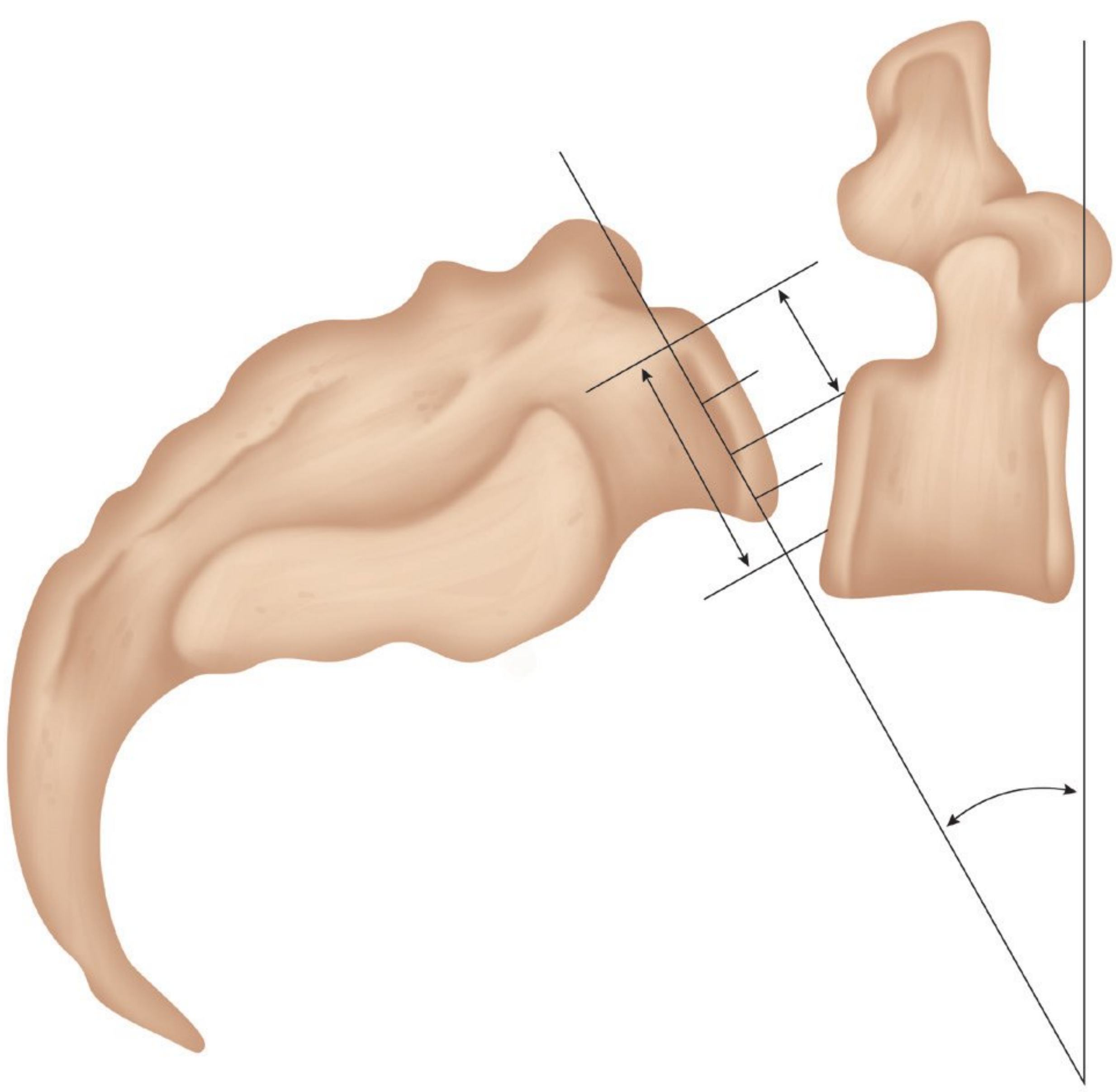

Fig. 19.3 Measurement of slip angle.

(2) Operative.
  (a) Goals of surgery.
    i. Pain reduction.
    ii. Prevention of further slippage.
    iii. Restoration of normal posture.
    iv. Prevention of neurological deficits.
  (b) Surgical techniques.
    i. Direct pars repair.
    ii. Posterolateral fusion with or without decompression.
      • Possible slip reduction.
      • Possible instrumentation.
    iii. Possible interbody fusion (anterior lumbar interbody fusion, posterior lumbar interbody fusion, transforaminal lumbar interbody fusion) **(Fig. 19.4).**
  (c) Pseudarthrosis.
    i. Fusion rate decreased in smokers (57%) versus nonsmokers (95%).
    ii. Common in in situ fusions without instrumentation.
      • Increased stress across fusion mass.
      • Difficulty in exposing L5 transverse process.
  (d) Slip progression.
    i. Occurs in 33% of cases regardless of the presence of a solid fusion (uninstrumented).
    ii. Increased risk of progression.
      • High-grade slips.
      • Gill laminectomy.
      • No postoperative immobilization.
  (e) High-grade slip reduction.
    i. May cause L5 nerve root neurapraxia.
    ii. Full correction is not needed.
    iii. Correction of the kyphosis is most important.
    iv. Reduction improves the fusion rate.

D. Degenerative spondylolisthesis.
  1. Epidemiology.
    a. Most often occurs at the L4–L5 level.
    b. Five times more common in women.
    c. Symptoms usually appear after age 40.
  2. Clinical findings.
    a. Low back pain with bilateral lower extremity radiation.
      (1) Fifty percent of patients have radiculopathy, most commonly in the L5 nerve root distribution.
    b. Stiffness is not a common finding.
      (1) Most patients are hyperflexible.
    c. Associated complaints of stenotic symptoms.
      (1) Proximal muscle weakness.

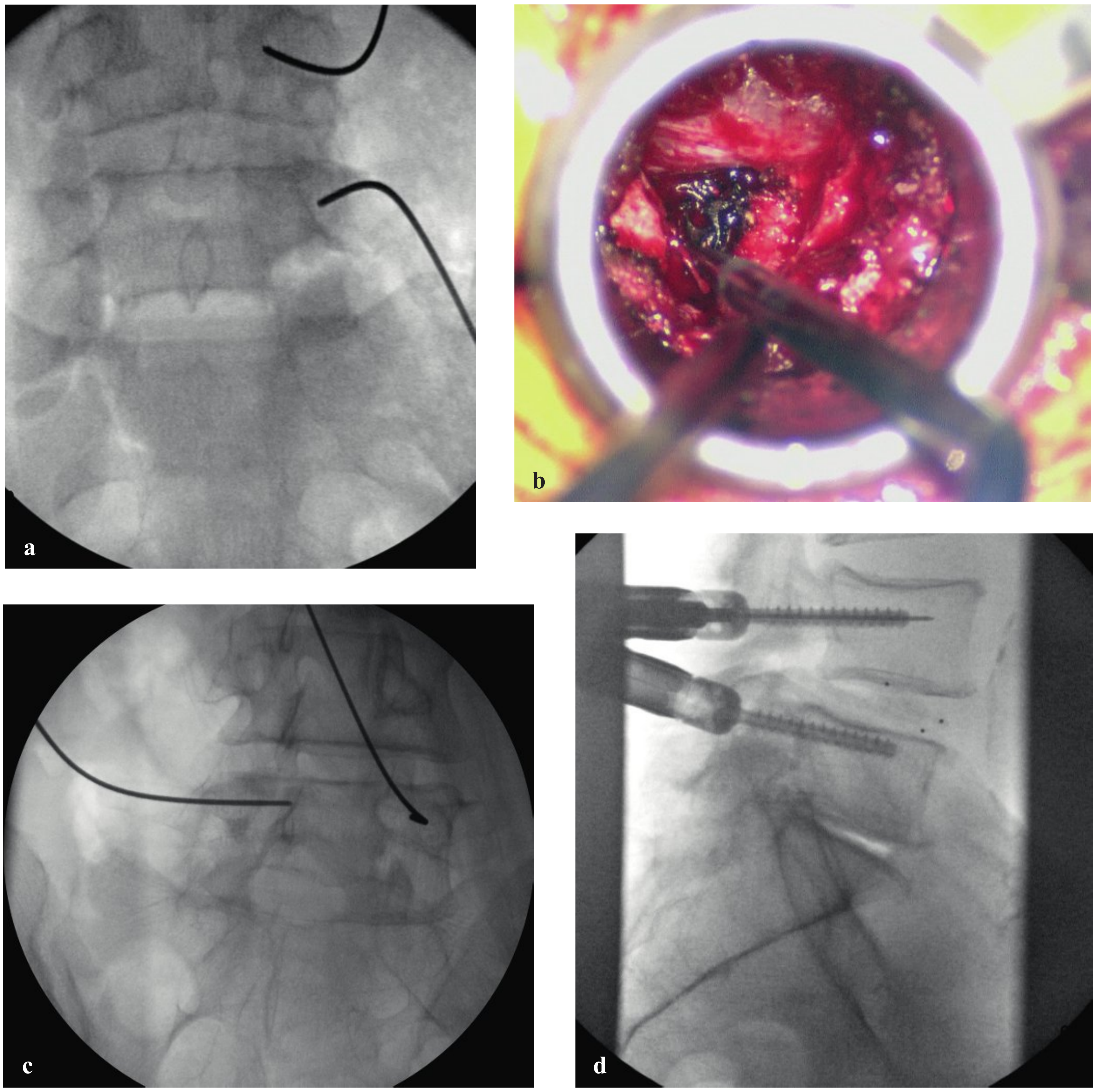

Fig. 19.4 Images demonstrating the minimally invasive transforaminal lumbar interbody fusion and percutaneous pedicel screw placement. **(a)** Intraoperative anteroposterior (AP) fluoroscopic image demonstrating placement of the guidewires along the medial wall of the pedicle. **(b)** Intraoperative image demonstrating removal of the ligamentum flavum and coagulation of the epidural veins overlying the disk space via the tubular retractor system. **(c)** Intraoperative "bulls-eye" view fluoroscopic image demonstrating the advancement of guidewires for percutaneous pedicle screw placement. **(d)** Intraoperative lateral fluoroscopic image demonstrating pedicle screw placement in the L4 and L5 pedicles. **(e)** Postoperative lateral radiograph demonstrating pedicle screw placement in the L4 and L5 pedicles with posterior instrumentation.

        (2) Neurogenic claudication.
            (a) Shopping cart sign.
                i. Relief with forward flexion.
3. Radiographic findings.
    a. Plain radiographs.
        (1) A standing lateral radiograph is more sensitive than a non-weight-bearing view.
        (2) Flexion–extension views:
            (a) Greater than 4 mm of motion is considered indicative of dynamic instability.
            (b) Greater than 10° of motion.
    b. CT myelogram.
        (1) Determines amount of spinal stenosis.
        (2) Evaluates degree of osteopenia.
        (3) Detailed view of facet hypertrophy.
        (4) The traversing nerve root is compressed by the superior articular process of the inferior vertebrae.
    c. MRI.
        (1) Gold standard for evaluation of disk, ligaments, and neural structures.
        (2) Provides information regarding neurological compression.
        (3) Delineates synovial cysts and hypertrophic ligamentum flavum.
4. Treatment.
    a. Nonoperative.
        (1) Short-term bed rest (1–2 days).
        (2) Nonsteroidal anti-inflammatory drugs.
        (3) Oral steroids.
            (a) Best reserved for acute exacerbations of leg pain.
        (4) Physical therapy.
            (a) Range of motion.
            (b) Aerobic conditioning.
    b. Operative **(Fig. 19.5)**.
        (1) Indications.
            (a) Persistent or recurrent severe leg pain.
            (b) Progressive neurological deficit.
        (2) Treatment options **(Table 19.2)**.
5. Spine patient outcomes research trials (SPORT).
    a. Patient populations.
        (1) Patients with degenerative spondylolisthesis and spinal stenosis.
        (2) Persistent symptoms (e.g., radicular leg pain, neurological claudication) for 12 weeks.
        (3) Assigned randomly to operative and nonoperative cohorts.
    b. Operative versus nonoperative treatment outcomes.
        (1) Surgery consisted of standard lumbar decompression with or without single-level fusion.
            (a) Iliac crest bone graft with or without posterior pedicle screw fixation.

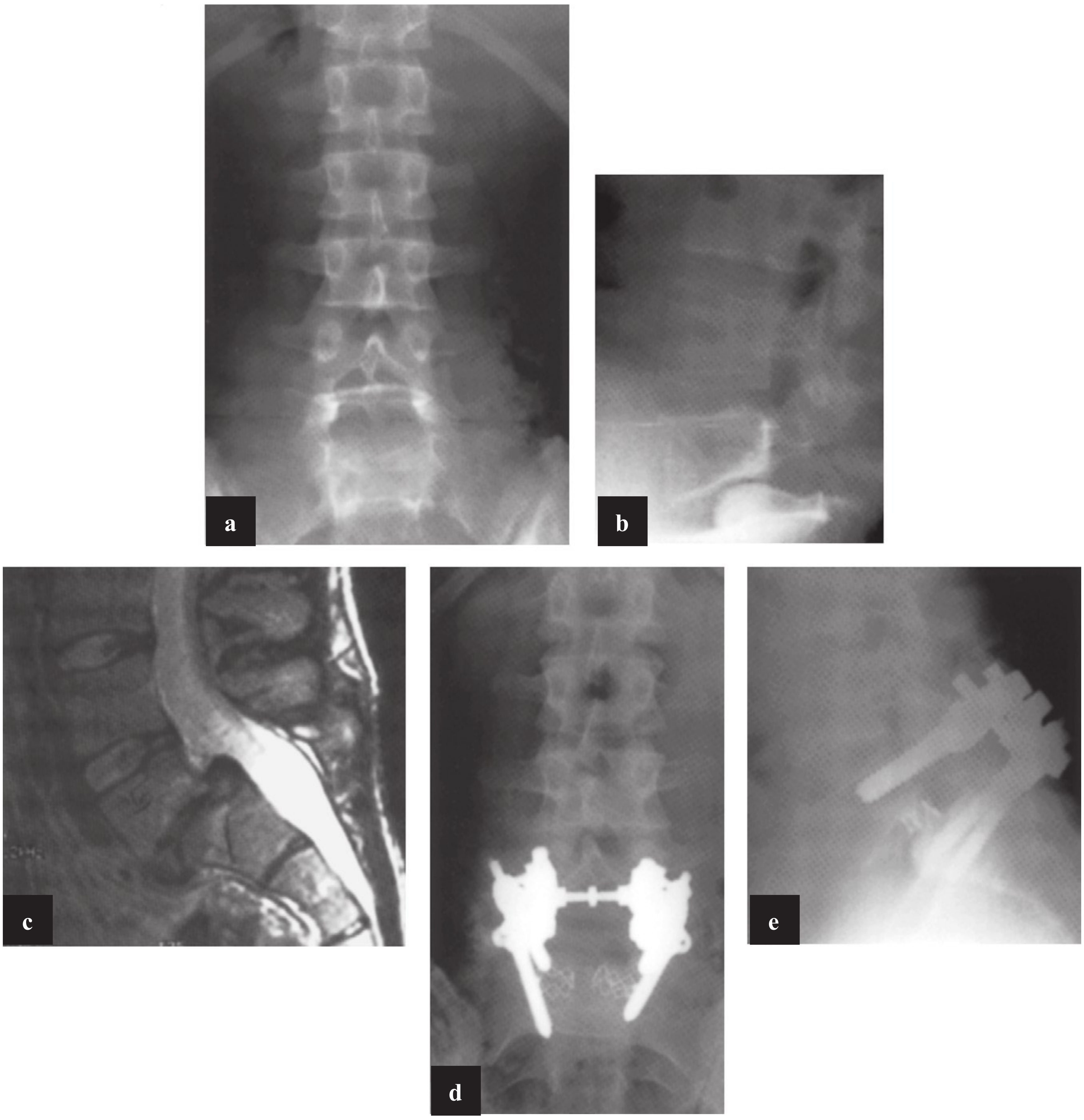

Fig. 19.5 Images of an 18-year-old man with a high-grade L5–S1 isthmic spondylolisthesis and bilateral L5 radiculopathy. **(a)** Anteroposterior radiograph showing that the L5 transverse processes overlie the sacrum due to the severe slip. **(b)** Lateral radiograph showing the grade 3 slip. **(c)** Magnetic resonance imaging scan shows typical changes at L5–S1 rounding of the sacral dome. **(d,e)** Postoperative radiographs demonstrating the placement of interbody cages to increase the fusion rate with supplementary pedicle screw fixation.

(2) The surgical cohort demonstrated greater improvements in pain and function after 2 years compared with those treated nonoperatively in the as-treated analysis.

c. Controversies.

(1) High crossover rate:

(a) Forty-nine percent of patients in the nonoperative cohort underwent surgery.

(b) Thirty-six percent of patients in the surgical cohort did not undergo surgery.

Table 19.2 Surgical options for adult spondylolisthesis

Procedure	Advantages	Disadvantages	Complications
Laminectomy	– Rapid pain relief – Avoids morbidity of a fusion	– Does not address instability	– Slip progression (25–50%)
Laminectomy with posterolateral fusion	– Decreased slip progression if fusion obtained	– Possible failure of fusion	– Increased rate of pseudarthrosis as compared with noninterbody techniques
Instrumented fusion with interbody graft	– Increased fusion rates – Partial reduction of deformity – Allows for more aggressive decompression	– Improves slip angle – Longer operative times	– Instrument placement – Increased infection rate – Implant migration or failure

(2) Nonoperative management was not standardized.
(3) Surgical procedures were not standardized.
  (a) Decompression alone.
  (b) Decompression and fusion.
    i. Autograft with iliac crest bone graft (ICBG).
    ii. Fusion with or without posterior instrumentation.
    iii. Bone morphogenetic protein was not used.
(4) Intent-to-treat versus as-treated analysis:
  (a) Intent-to-treat analysis compared patients according to their assigned cohort.
    i. This demonstrated no difference in primary outcomes between groups in patients with spondylolisthesis.
    ii. Patients assigned to nonoperative treatment were counted as nonoperative patients even if they crossed over and had surgical intervention.
  (b) As-treated analysis compared patients according to the treatment ultimately received.
  (c) Patients who underwent surgery demonstrated improved and sustained outcomes compared with those treated nonoperatively for 4 years.

E. Traumatic spondylolisthesis.
  1. Extremely rare injuries.
  2. A posterior fracture may be part of a larger injury.
    a. Be suspicious for a fracture or dislocation of the spine.

F. Pathological spondylolisthesis.
  1. Generalized bone disease.
    a. Osteoporosis and osteomalacia.
      (1) Instability results from contiguous stress fractures healing in an elongated pattern.
    b. Paget's disease and osteogenesis imperfecta.
    c. Primary or secondary neoplasm.

Suggested Reading

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Weinstein JN, Lurie JD, Tosteson TD, et al. 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 2009;91(6):1295–1304

20 Adult Spinal Deformity

20.1 General Considerations

I. Introduction.
  A. Adult scoliosis is more rigid than adolescent spinal deformity (ASD) and is more likely to be symptomatic.
  B. The curve may progress, especially if the curve is > 50° **(Fig. 20.1)**.
    1. The curve may progress 1 to 2° per year.

II. Risk factors for lumbar curve progression:
  A. Lateral and rotatory listhesis.
  B. Large apical rotation.

III. Spinal stenosis, disk disease, and osteopenia are associated pathologies.
  A. Asymmetrical loss of disk height and vertebra may contribute to the increase in Cobb angle.

IV. Adult scoliosis is more likely to be symptomatic, with pain and disability.

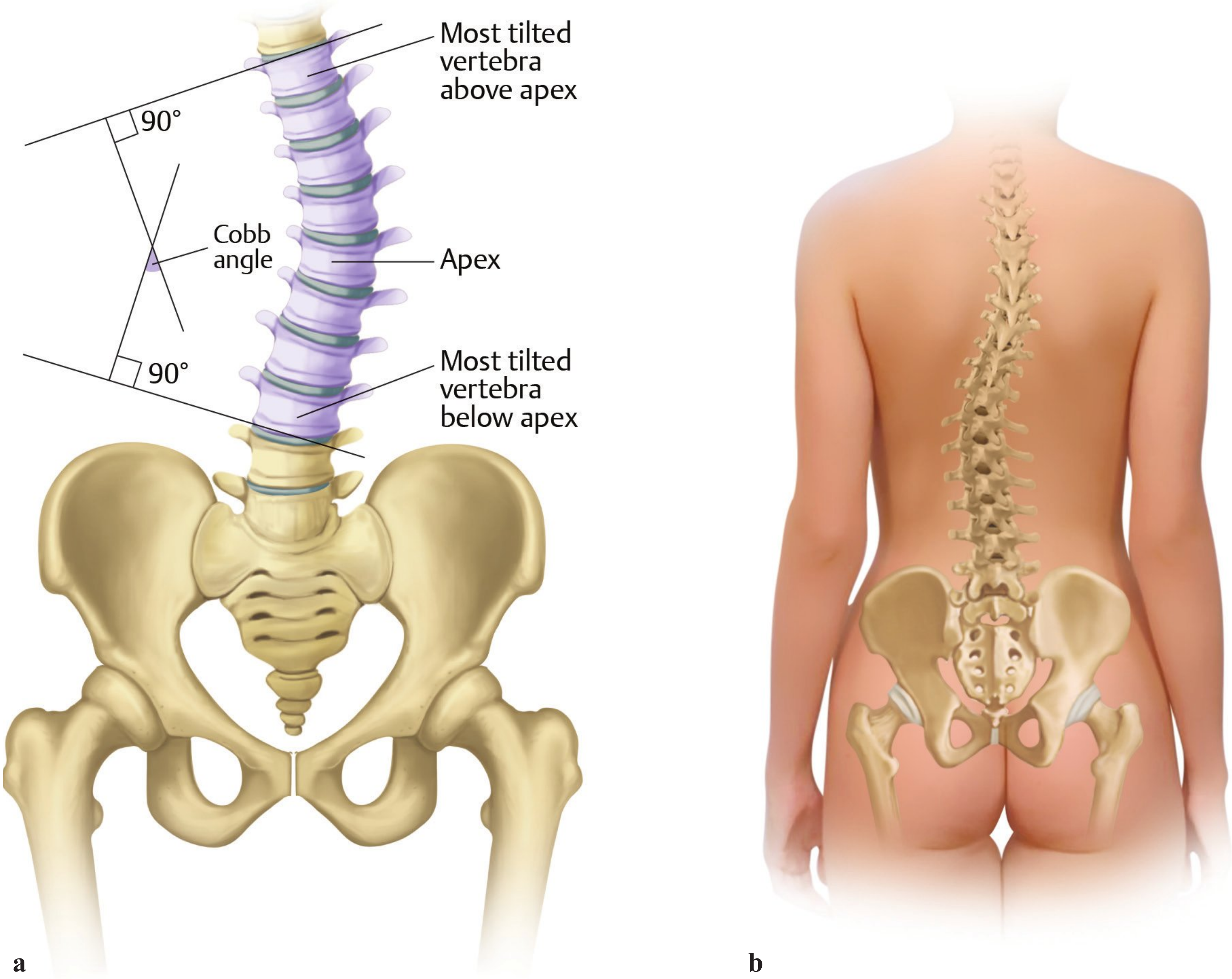

Fig. 20.1 **(a,b)** Cobb angle measurement.

A. Pain often originates on the convexity of the curve due to muscle fatigue. This is followed by facet degeneration on the concave side.
B. There is increased incidence of low back pain if the lumbar curve is > 45°.
C. It is necessary to rule out other sources of pain, such as abdominal aneurysm, renal stones, and tumors, as well as disk disease and spinal stenosis.
D. Pain, in the absence of a progressive curve, is rarely an indication for surgery.
E. Sciatica may result from nerve root compression in the concavity of the curve.

V. Respiratory compromise may occur, resulting in dyspnea, pulmonary hypertension, and cor pulmonale.
VI. Adults often have other medical comorbidities that make the surgery more risky.
VII. Sacropelvic radiographic parameters **(Table 20.1) (Fig. 20.2).**
VIII. Classification:

A. Scoliosis Research Society (SRS)—Schwab Adult Spinal Deformity Classification (Schwab et al).
   1. Uses radiographic parameters and patient-reported functional assessment scores.
      a. Radiographic parameters were correlated with functional outcomes.
   2. Curve type involves the assessment of coronal deformity with added sagittal deformity modifiers **(Fig. 20.3).**
      a. Curve Type T: thoracic major curve > 30° (apical T9 or higher).
      b. Curve Type L: thoracolumbar or lumbar major curve > 30° (apical T10 or lower).

Table 20.1 Sacropelvic radiographic parameters

Radiographic parameter	Measurement	Note
Pelvic incidence (PI = PT + SS)	Angle between the straight line from the femoral head to the midpoint of the sacral plate and a perpendicular line to the sacral plate	Will not change position after skeletal maturity (morphological parameter)
Pelvic tilt (PT)	Angle between the straight line from the femoral head to the midpoint of the sacral plate and a vertical line from the femoral head	Changes with position and increases with age due to compensatory forces (positional parameter[a])
Sacral slope (SS)	Angle between the sacral plate and the horizontal axis	Changes with position (positional parameter[a])
Sagittal vertical axis	A plumb line is drawn in the sagittal axis from the C7 vertebral body, and the distance from the posterosuperior sacral end plate to the plumb line is measured	Measurement of global alignment. If plumb line is posterior to the S1 vertebral body, it is negative (normal). If anterior, it is referred to as positive.

[a]If one positional parameter changes, it affects all other parameters.

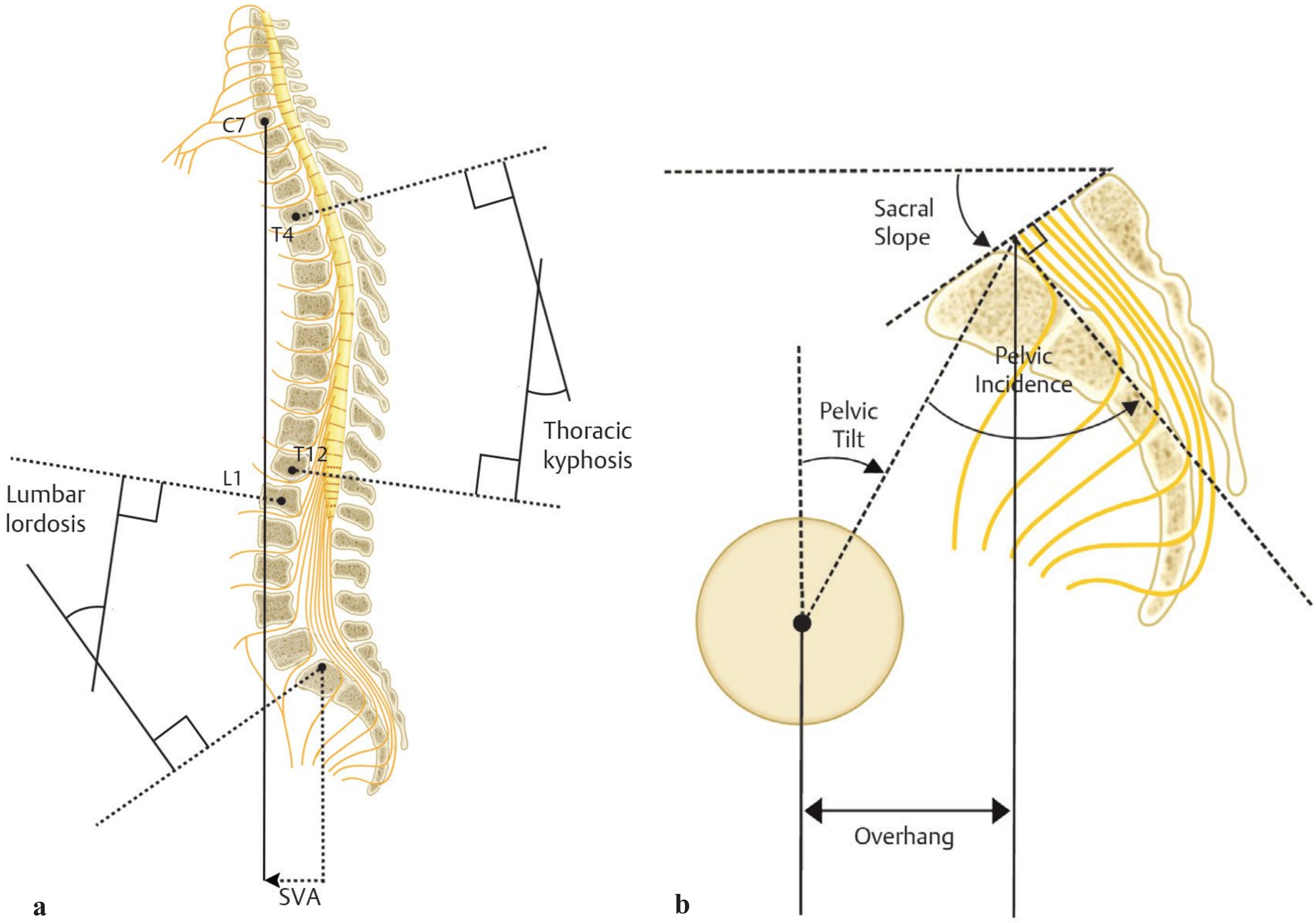

Fig. 20.2 **(a,b)** Sacropelvic radiographic parameters.

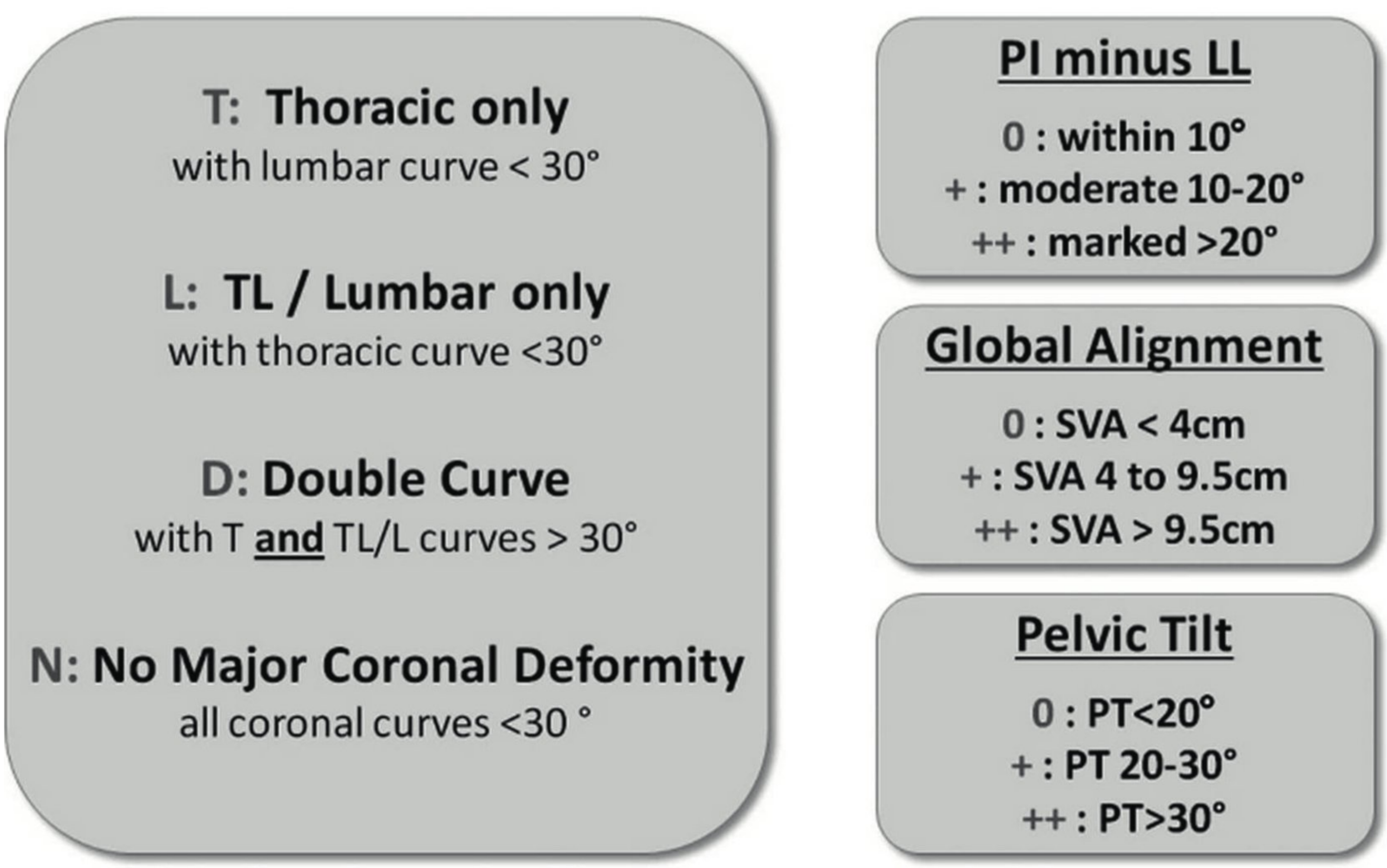

Fig. 20.3 The SRS–Schwab adult spinal deformity classification: assessment and clinical correlations based on a prospective operative and nonoperative cohort. (Neurosurgery 2013;73(4):559–568.)

c. Curve Type D: double major curves > 30°.
d. Curve Type N: no major coronal deformity.
3. Sagittal modifiers (three types).
a. All are correlated with pain and disability and are important for preoperative planning and determination of operative management.
b. Pelvic incidence (PI) – LL.
c. Pelvic tilt (PT) (pelvic retroversion).
d. Sagittal vertical axis (SVA).

IX. Evaluation:
A. Careful history and examination, including previous evaluations for scoliosis:
1. Assessment of kyphosis, lordosis, rib hump, and curve flexibility.
2. Neurological examination.
B. X-rays: 36 in standing anteroposterior and lateral to measure magnitude of the curvatures and obtain radiographic parameters as already defined:
1. It is important to make sure that the patient is not flexing the knees, which will cause underestimation of sagittal imbalance.
2. Patients with spinal stenosis and deformity may flex forward to relieve spinal stenosis symptoms. It is important to assess both structural flat-back and functional or flexible kyphosis to avoid overestimating sagittal imbalance.
C. Radicular pain or symptoms of spinal stenosis warrant magnetic resonance imaging to assess for neural compression.

X. Treatment:
A. Goals of treatment include improvement of functional improvement and pain and restoration of coronal and sagittal alignment.
B. Conservative treatment is indicated for nonprogressive curves causing localized back pain. The treatment plan incorporates the general principles of treating back pain, including a brief period of rest, nonsteroidal anti-inflammatory drugs, stretching, an exercise program, and nerve blocks.
C. Radiographic parameters, as determined by the SRS—Schwab Classification, should guide surgical planning.
1. Goal parameters include the following:
a. LL = PI ± 9.
b. PT < 20°.
c. SVA < 50 mm.

XI. Techniques:
A. Operative management is likely warranted with symptomatic patients and should be guided by the target radiographic parameters as already stated.
B. Relatively flexible thoracic curves or balanced double major curves require posterior fusion and instrumentation.
C. Rigid and severe unbalanced thoracic curves (> 80° curves) may require anterior release and fusion followed by posterior fusion and instrumentation.
D. Relatively flexible thoracolumbar or lumbar curves can be treated by anterior fusion with instrumentation (if there is no kyphosis and the curve is limited to T10 to L4).
E. Severe and rigid thoracolumbar or lumbar curves > 75°, redundant statement, and associated kyphosis may require anterior release and fusion followed by posterior fusion and instrumentation.

F. Degenerative scoliosis with radiculopathy requires posterior laminectomy and fusion with transpedicular instrumentation of the lumbar spine with or without anterior fusion.

XII. Complications:

A. Higher than adolescent spine, especially pulmonary problems.

B. Pseudarthrosis is less for combined anterior and posterior approach than for posterior fusion alone.

C. Flat-back syndrome (loss of lumbar lordosis) may result if the posterior surgical techniques produce a distractive force or if the anterior technique produces significant compression force along the lumbar spine. Segmental instrumentation and preservation of the lumbar lordosis and sagittal balance are critical.

D. Proximal junctional kyphosis (PJK) **(Fig. 20.4)**:

1. Postoperative adjacent-segment pathology that is defined as a kyphosis of > 10° of the cephalad vertebrae above a previously instrumented segment:
   a. If severe enough, may warrant a revision procedure.

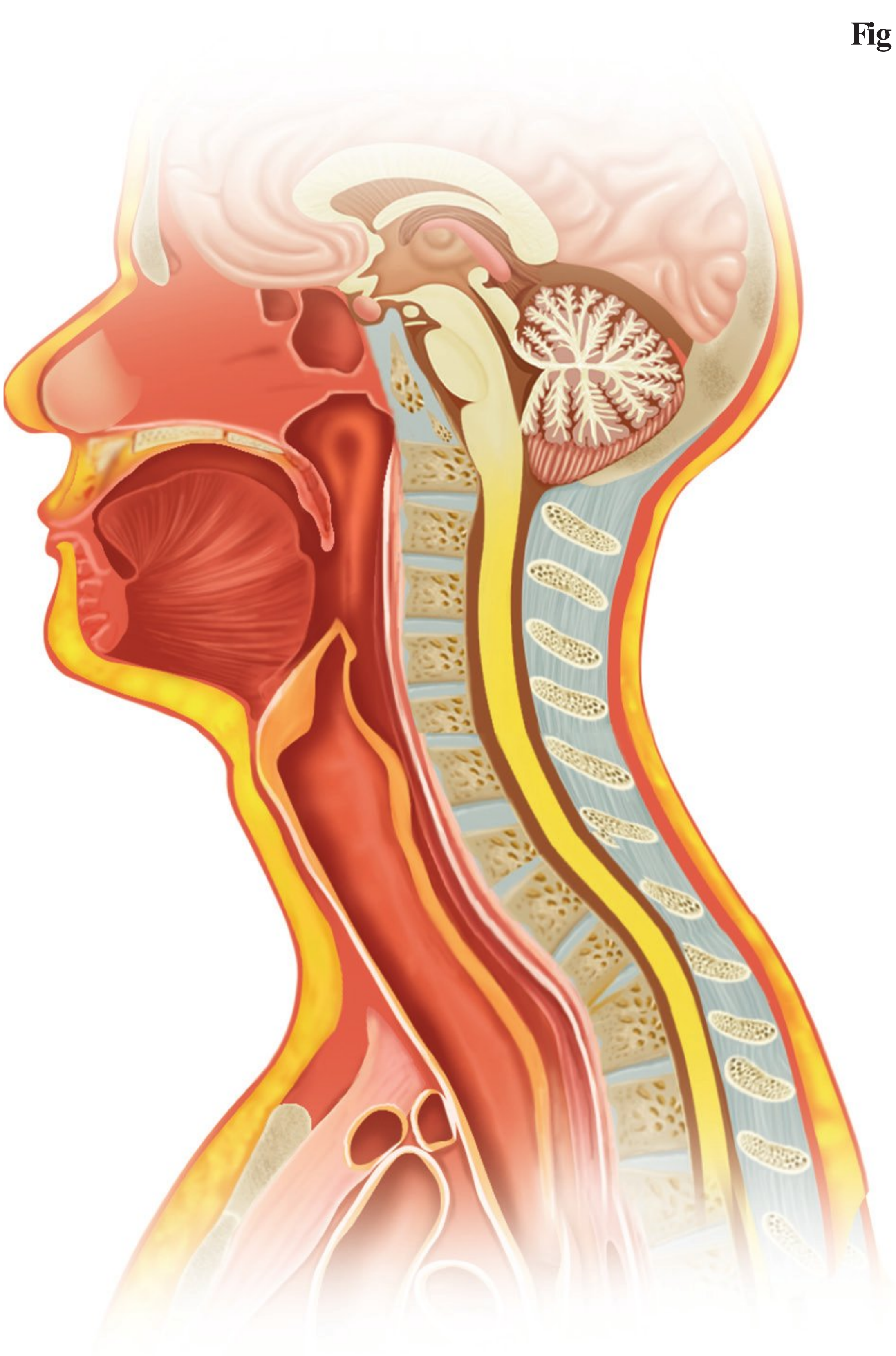

Fig. 20.4 Proximal junctional kyphosis.

2. Risk factors:
   a. Increased age.
   b. Fusion to sacrum.
   c. Circumferential fusion.
   d. Thoracoplasty.
   e. Upper instrumented vertebrae at T1–T3.
3. Prevalence of 17 to 39%, mostly at 2 years.
4. There is moderate evidence suggesting that PJK does have deleterious effects on functional outcomes.

E. Infection:
1. Incidence of 0.5 to 8%.
2. More common with posterior surgery.

F. Neurological complications:
1. Incidence of 1 to 5%.
2. Most common with combined posterior and anterior surgery.

G. Pulmonary embolism:
1. Incidence of 1 to 20%.

Suggested Reading

Kim HJ, Lenke LG, Shaffrey CI, Van Alstyne EM, Skelly AC. Proximal junctional kyphosis as a distinct form of adjacent segment pathology after spinal deformity surgery: a systematic review. Spine 2012;37(22, Suppl):S144–S164

Klineberg E, Gupta M, McCarthy I, Hostin R. Detection of pseudarthrosis in adult spinal deformity: The use of health-related quality-of-life outcomes to predict pseudarthrosis. J Spinal Disord Tech 2013

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21 Pediatric Spinal Deformity

21.1 General Considerations

I. Classification **(Table 21.1).**

21.2 Idiopathic Adolescent Scoliosis

I. Etiology.
   A. Neuromuscular.
      1. Changes in the muscle fiber types and muscle spindles have been demonstrated.
      2. Increased calmodulin level, which is responsible for the regulation of muscle contraction, and decreased melatonin levels (calmodulin antagonist) have been demonstrated in patients with idiopathic adolescent scoliosis (IAS).
   B. Hormonal.
   C. Connective tissue.
      1. Elastic and collagen fibers are the primary elements that support the spine.
      2. Abnormalities of the collagen/proteoglycan in the intervertebral disks have also been reported.
   D. Genetic: 5:1 female preponderance (> 10° curves), familial (20 times greater likelihood in families) with monozygotic twin concordance rate of 73%, genetic (sex-linked trait with incomplete penetrance and variable expressivity).
   E. Melatonin or serotonin abnormalities.
II. Anatomical characteristics.
   A. Lateral curvature or deformity in the coronal plane.
   B. Decreased thoracic kyphosis or thoracic hypokyphosis (deformity in the sagittal plane):
      1. Earlier accelerated spinal growth as compared with normal individuals may be related.
   C. Vertebral rotation: the spinous process rotates toward the concavity (deformity in the axial plane) and causes rib hump.
   D. Patterns of thoracic scoliosis:
      1. King classification **(Table 21.2)**: helps determine fusion levels for surgery (not all curves fit into this classification).
      2. Lenke classification: newer, more extensive, classifies the curves based on curve type, coronal lumbar modifier, and thoracic sagittal profile **(Fig. 21.1).**
         a. Four series of plain film spine radiographs are used: upright posteroanterior, lateral, supine right-bending, and supine left-bending.
         b. Curves can be classified into six types:
            (1) Type 1: Main thoracic (MT).
            (2) Type 2: Double thoracic (DT).
            (3) Type 3: Double major (DM).
            (4) Type 4: Triple major (TM).

Table 21.1 Classification of pediatric spinal deformity

Classif cation	Type	Subtypes
Nonstructural	Postural Sciatic Inflammatory Compensatory	
Structural	Idiopathic	Infantile (< 3 years) Juvenile (3–10 years) Adolescent (10 years through maturity)
	Neuromuscular Neuropathic Myopathic	Cerebral palsy Syringomyelia Poliomyelitis Spinal muscular atrophy Friedreich's ataxia Arthrogryposis Muscular dystrophy Myotonia dystrophica
	Congenital	Diastematomyelia, spina bifida, hemivertebra, wedge vertebra, unsegmented bar with contralateral hemivertebra, block vertebra
	Neurofibromatosis	
	Mesenchymal disorders	Marfan's syndrome Ehlers–Danlos syndrome
	Rheumatoid disease	
	Trauma	
	Extraspinal contracture	Burns Thoracic surgery
	Osteochondral dystrophies	
	Infection	
	Metabolic disorders	
	Related to lumbosacral joint	
	Tumors	

Table 21.2 King classification system

Type	King classif cation	Notes
Double major right thoracic and left lumbar	I	Lumbar curve is larger than the thoracic curve
Right thoracic and compensatory left lumbar	II	Thoracic curve is larger than the lumbar curve
Right thoracic	III	Left lumbar curve does not cross the midline
Right thoracolumbar	IV	
Double thoracic	V	

Curve Type

Type	Proximal Thoracic	Main Thoracic	Thoracolumbar / Lumbar	Curve Type
1	Nonstructural	Structural (major)	Nonstructural	Main thoracic (MT)
2	Nonstructural	Structural (major)	Nonstructural	Double thoracic (DT)
3	Nonstructural	Structural (major)	Structural	Double major (DM)
4	Structural	Structural (major)	Structural	Triole major (TM)
5	Nonstructural	Nonstructural	Structural (major)	Thoracolumbar / lumbar (TL/L)
6	Nonstructural	Structural	Structural (major)	Thoracolumbar / lumbar structural MT

Structural Criteria

Proximal thoracic: - Side bending cobb ≥ 25°
- T2 – T5 kyphosis ≥ + 20°

Main thoracic : Side bending cobb ≥ 25°

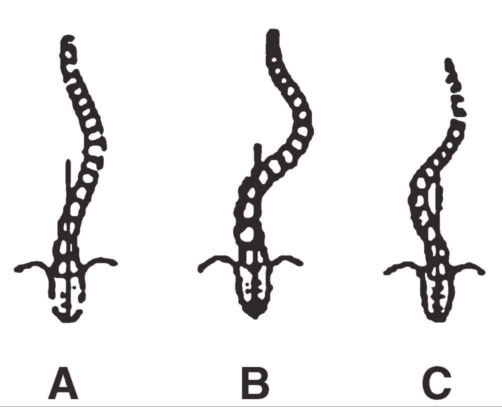
Thoracolumbar / lumbar : - Side bending cobb ≥ 25°
- T10 – L2 kyphosis ≥ + 20°

Location of Apex (SRS definition)

Curve	Apex
Thoracic	T2 – T11–12 DISC
Thoracolumbar	T2 – L1
Lumbar	L1–2 DISC – L4

Modifiers

Lumbar Spino Modifier	CSVL to Lumbar Apox
A	CSVL between pedicles
B	CSVL touches apical body(ies)
C	CSVL completely medical



Thoracic Sagittal Profile T5 - T12		
–	(hypo)	< 10°
N	(normal)	10°-40°
+	(hyper)	> 40°

Curve type (1-6) + Lumbar spine modifier (A, B, or C) + Thoracic sagittal modifier (–, N, or +)
Classification (e.g. 1B+): ________

Fig. 21.1 Lenke classification. (From Lenke LG, Betz RR, Harms J, et al. Adolescent idiopathic scoliosis: a new classification to determine extent of spinal arthrodesis. J Bone Joint Surg Am 2001;83-A:1169–1181. Reproduced with permission.)

(5) Type 5: Thoracolumbar/lumbar (TL/L).

(6) Type 6: Thoracolumbar/lumbar–main thoracic (TL/L-MT).

c. The major curve is the largest curve.

d. Minor curves are then evaluated for structural criteria.

(1) Structural curve: coronal plane rigidity > 25° upon side-bending or kyphosis > 20° on sagittal radiographs.

e. Spinal arthrodesis should include only the major curve and the structural minor curves.

f. A lumbar coronal modifier is determined.

(1) A central sacral vertical line (CSVL) is drawn vertically from the midpoint of S1.

(a) Modifier A: the CSVL traverses between the pedicles of the apical vertebrae.

(b) Modifier B: the CSVL lies between the medial border of the concave pedicle and the lateral edge of the apical vertebral body.

(c) Modifier C: the CSVL is not adjacent to the lateral border of the apical vertebral body.

g. The last component involves assessment of the sagittal thoracic alignment.

(1) The Cobb angle from T5 to T12 is measured.

(2) If the Cobb angle is +10 to +40°, a normal modifier is assigned.

(3) A minus sign indicates a Cobb angle < 10° (hypokyphotic curve).

(4) A plus sign indicates a Cobb angle > 40° (hyperkyphotic).

h. Forty-two different types of curves are possible because type 5 and 6 curves are associated with a lumbar coronal modifier of "C."

3. Isolated lumbar or thoracolumbar curves.

III. Natural history and prognosis.

A. Prevalence: 25/1,000 (2.5%) exhibit > 10° curves and (0.4%) 4/1,000 exhibit > 20° curves **(Table 21.3)**.

IV. Diagnosis.

A. Screening: generally occurs in school children aged 10 to 14:

1. Leads to a substantial number of referrals.

2. One-third of all referrals have scoliosis to some degree.

3. Genetic testing is available to determine the risk of curve progression in patients with AIS:

a. ScoliScore (Transgenomic, Inc.).

(1) Thought to determine the risk of curve progression past 40°.

(2) Should be used as an adjunct to clinical examination and radiographic findings.

(3) There is mixed evidence regarding the efficacy of this genetic test.

B. History: age, gender, onset of menarche, pain, family history.

1. Pain occurs in up to 30% of patients with AIS.

2. Peak growth occurs in girls at age 11 to 12 and in boys at age 13 to 14.

C. Physical examination:

1. Observation.

a. Asymmetry of shoulder level, breasts, waist, or pelvis.

b. Protruding scapula or ribs.

c. Loss of thoracic lordosis.

Table 21.3 Factors contributing to curve progression

Curve magnitude	The greater the angulation and rotation, the greater the tendency for progression. For example, a 20° curve has a 20% likelihood of progression, and a 40° curve has a 60% likelihood of progression.
Age	Younger age is a more important prognostic factor than gender or family history. Ninety percent of spinal growth has occurred at puberty; however, this age has the highest risk of progression.
Risser score	A score of 1 or less has a higher likelihood of progression.
Curve size	Shorter curves progress more
Location	The lower the curve is in the spinal column, the greater the likelihood of progression (thoracic < lumbar).
Flexibility	Stiffer curves in immature individuals and more flexible curves in mature individual are more likely to progress.
Gender	Girls are more commonly affected, especially for larger curves.
Family history	
Slender spine	

d. Adams forward bend test.
   (1) The patient bends at the waist to 90°.
   (2) During the process, assess for asymmetry in bending and rotational deformity of thoracic and lumbar curves.

2. Measurements.
   a. A scoliometer is used to measure the rib hump (rotational deformity on forward bending).
   b. A plumb line dropped from C7 indicates coronal balance relative to the gluteal cleft.
   c. Leg length discrepancy.
3. Neurological examination.
   a. Deep tendon reflexes.
   b. Abdominal reflexes.
      (1) Check for symmetrical umbilical movement upon lateral to medial light stroke on the abdomen.
      (2) Asymmetrical movement correlates with neural axis pathology.

D. X-ray examination:
   1. The Cobb angle **(Fig. 21.2)** is used to determine the magnitude of the curve. The upper and lower end vertebrae of each curve are identified. A line is drawn at the upper end of the cranial end vertebra along the end plate or by marking the upper or lower margin of the pedicles. A line is then drawn

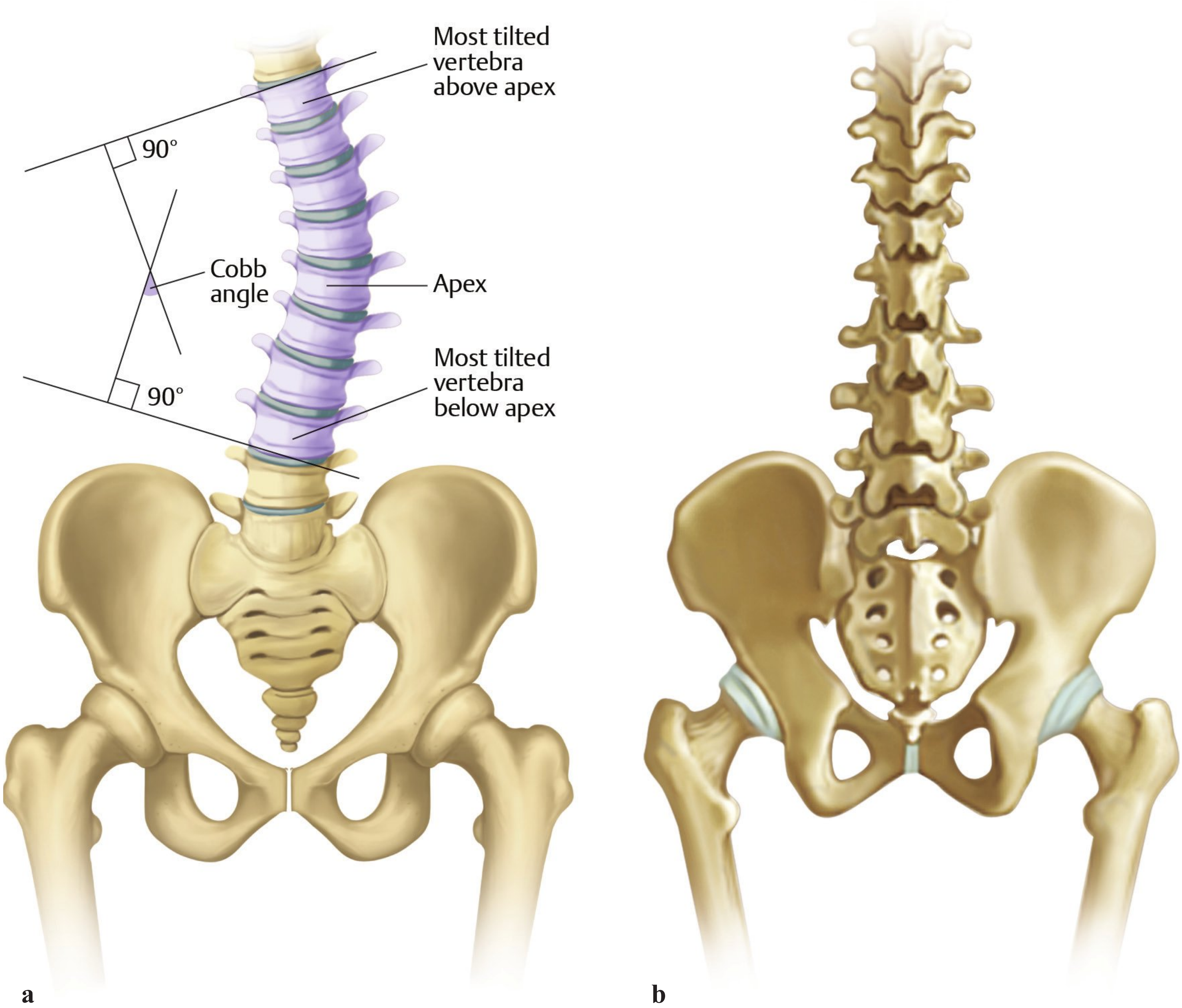


Fig. 21.2 (a,b) Cobb angle analysis for manual adolescent idiopathic scoliosis measurements.

at the lower end of the caudal vertebra of the curve, using the end plate or pedicles. A right angle to this line is then drawn. The angle to be measured is the angle formed by the two lines at the end vertebrae.

2. Bone age determination can be useful to help determine the risk of spinal curve progression.
   a. Risser sign **(Fig. 21.3)**: ossification of the iliac epiphysis progresses from the anterior iliac spine posteriorly. A Risser 0 has the least ossification and greatest risk of progression, whereas a Risser 5 indicates the epiphysis has fused with the iliac crest, and the risk of progression is minimal.
   b. A ring apophysis fusion indicates cessation of all vertebral body growth potential.
   c. Left wrist and hand: the X-ray is compared with standards in the Greulich and Pyle atlas.

E. Pulmonary function test: > 70° curves have decreased vital capacity, particularly with hypokyphosis.

F. Indications for magnetic resonance imaging (MRI):
   1. Neurological abnormalities.

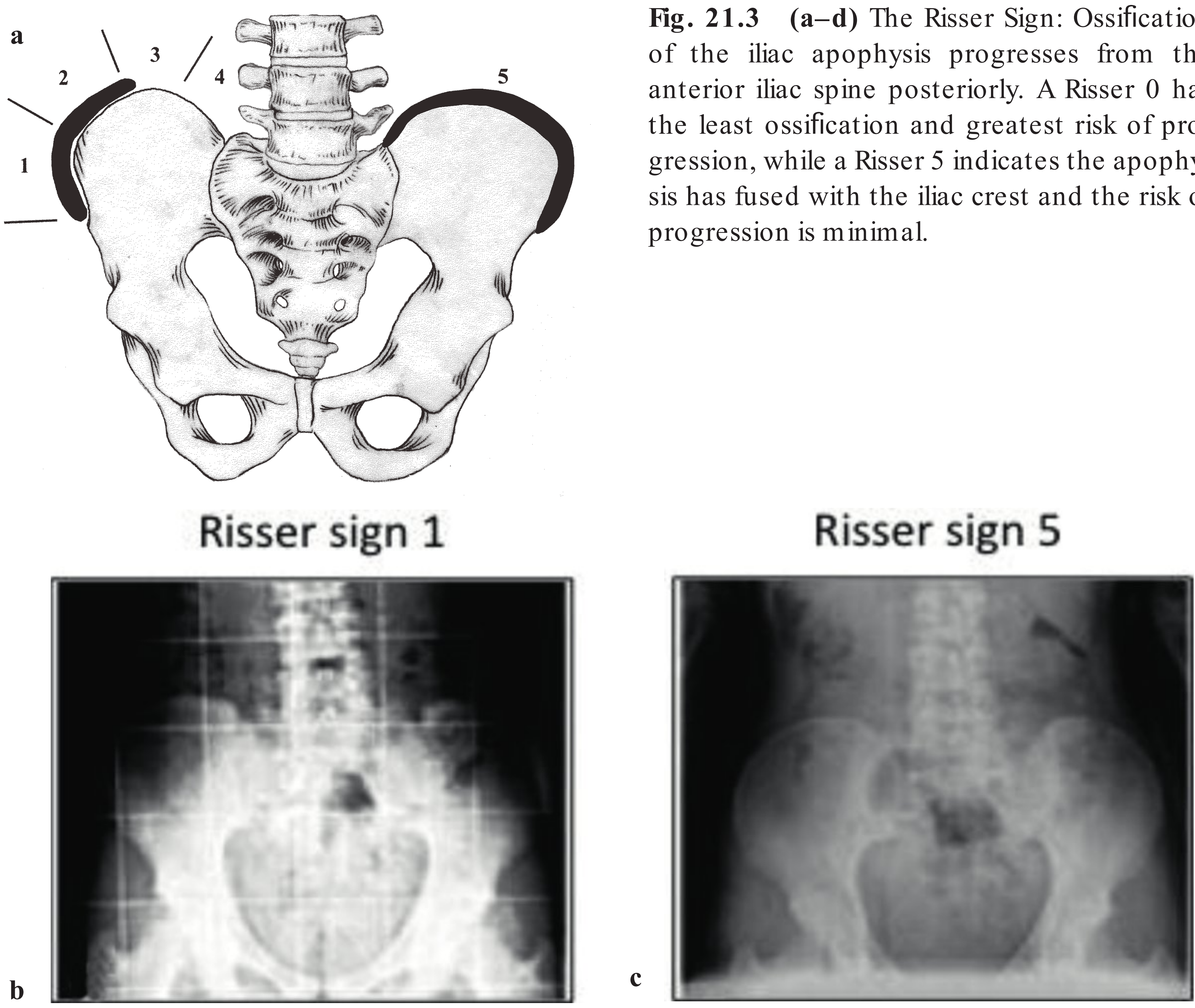
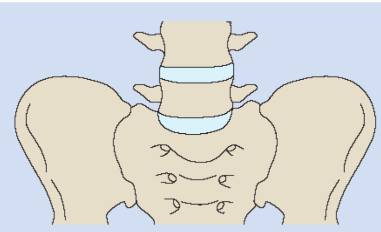
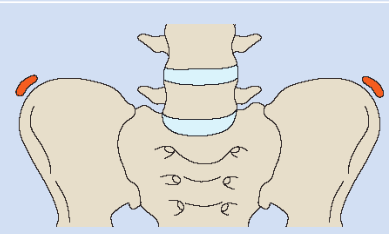
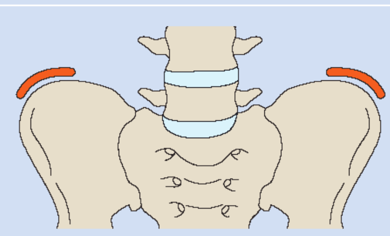
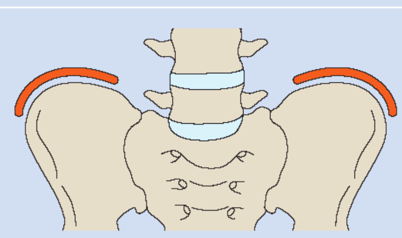
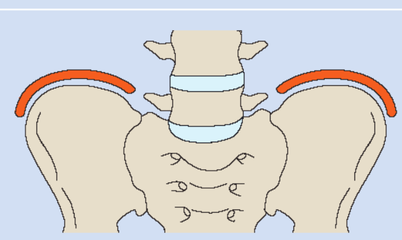
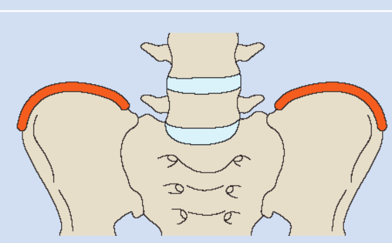


Fig. 21.3 (a–d) The Risser Sign: Ossification of the iliac apophysis progresses from the anterior iliac spine posteriorly. A Risser 0 has the least ossification and greatest risk of progression, while a Risser 5 indicates the apophysis has fused with the iliac crest and the risk of progression is minimal.

Stage	Description	
0	Bony iliac apophysis not yet visible on radiographs	
1	Initial (< 25%) ossification of the iliac apophysis	
2	From 25% to 50% ossification of the iliac apophysis	
3	From 50% to 75% ossification of the iliac apophysis	
4	More than 75% ossification of the iliac apophysis	
5	Iliac apophysis fuses to the iliac crest	

d

2. Congenital vertebral abnormalities.
3. Juvenile and infantile onset.
4. Rapid progression.
5. Cutaneous manifestations of dysraphism.

V. Management.
  A. Goals of treatment.
    1. Prevent progression and maintain balance.
    2. Maintain respiratory function.
    3. Reduce pain and preserve neurological status.
    4. Cosmesis.
  B. Nonoperative treatment.
    1. For most patients with scoliosis, progression may not be severe enough to warrant treatment.
    2. Observation is indicated for curves < 25° in immature patients and > 50° in mature patients.
      a. Obtain X-ray 3 months after the first visit and then every 6 to 9 months for curves < 20° and every 4 to 6 months for curves > 20°.
      b. A significant change is defined as progression of > 10° in curves < 20° and > 5° in curves > 20°.
    3. Exercise is indicated only as an adjunct treatment, especially for patients with obesity, back pain, lumbar hyperlordosis, flexible kyphosis, and trunk and extremity muscle tightness.
    4. Orthosis: curve > 30 to 45° (first visit) and > 25° with documented progression in immature patients (Risser 3 or less):
      a. Not for cervicothoracic curves and hypokyphotic thoracic curves.
      b. The goal is to prevent progression: ~ 85% of compliant patients demonstrate progression cessation and improve (~ 50%correction), but most patients tend to return within 5° of the original curve after the brace treatment is ceased.
      c. Protocol: the patient must wear the brace 23 hours a day until 2 years after menarche or Risser 4 and be weaned off in 1 year (part-time wear is also reported).
      d. Orthosis types:
        (1) Thoracolumbar sacral orthosis (Boston overlap).
          (a) Appropriate up to T8 apex.
          (b) All curve types.
          (c) Medium compliance.
        (2) Bending brace (Charleston).
          (a) Thoracolumbar and lumbar curves (25–35°).
          (b) Best compliance.
        (3) Mehta cast.
          (a) Treatment option for very young children.
        (4) Cervico Thoracolumbar Sacral Orthosis (CTLSO) (GB Orthopaedics).
          (a) Thoracic curves with apex above T7.
          (b) Low compliance.
    5. Electrical stimulation treatment has been abandoned.

C. Operative treatment.
   1. Indications for surgery.
      a. Progressive curves > 40 to 45° in growing children **(Fig. 21.4).**
      b. Failure of bracing.
      c. Progressive curves beyond 50° in adults.

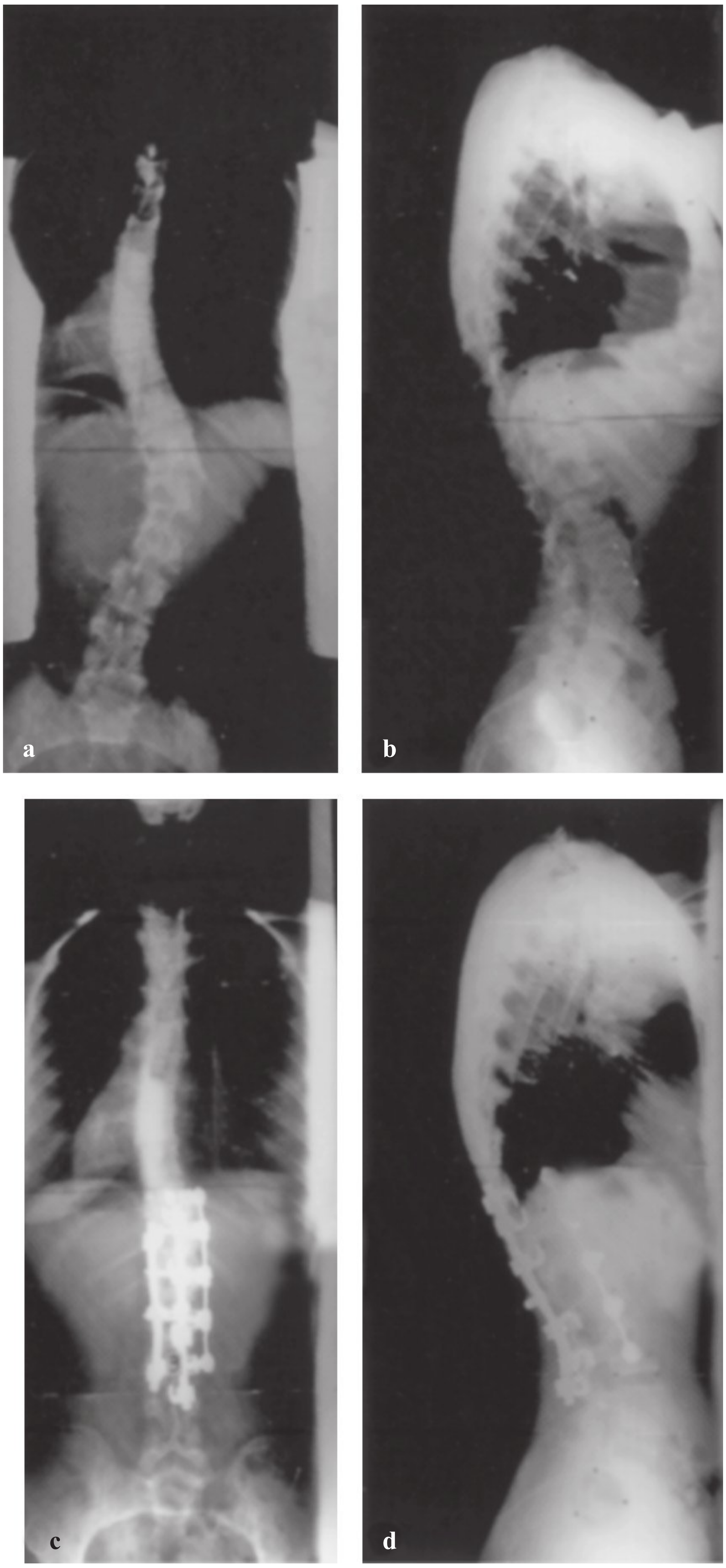


Fig. 21.4 A 17-year-old girl with adolescent idiopathic scoliosis. **(a)** Anteroposterior radiograph demonstrating a 49° right thoracolumbar curve with maintenance of the overall coronal balance. **(b)** Lateral radiograph demonstrates relatively preserved sagittal alignment. Postoperative **(c)** anteroposterior and **(d)** lateral radiographs with an anteroposterior release and fusion.

2. Goals of surgery.
   a. Spinal and pelvic balance is more important than curve correction.
   b. Prevent respiratory compromise.
   c. Prevent back pain.
   d. Cosmesis.
3. Operative management based on the Lenke classification.
   a. Type 1: posterior fusion and instrumentation (PFI) is favored.
      (1) Lower extent vertebrae (LEV) is controversial.
   b. Type 2: require PFI.
      (1) The proximal fusion level (T2 or T3) is determined by the size of the proximal thoracic curve and shoulder alignment.
      (2) LEV is controversial.
   c. Type 3: PFI is warranted.
   d. Type 4: Rare and warrants fusion of the proximal thoracic curve, main thoracic curve, and thoracolumbar/lumbar curves.
      (1) May require an anterior release for rigid curves.
   e. Type 5: Only require fusion of the thoracolumbar/lumbar curve given that it is structural (anterior or posterior).
   f. Type 6: PFI of the major thoracolumbar/lumbar and the minor thoracic curve.

VI. Instrumentation.
   A. Contemporary systems.
      1. Vertical Expandable Prosthetic Titanium Rib (VEPTR) (Synthes Spine) **(Fig. 21.5).**
         a. Received humanitarian device exemption status to treat chest wall and spinal deformity associated with thoracic insufficiency syndrome.
         b. Maximizes lung volume by expanding the rib cage and enabling growth and correction of the spinal deformity.
         c. Long-term outcomes with VEPTR are pending, because sagittal deformity remains unaddressed.
      2. Growing rod instrumentation.
         a. Used in adolescent children to enable normal growth of the spine while addressing spinal deformity correction.
         b. Requires lengthening at periodic intervals.

VII. Surgical technique.
   A. Intraoperative cell saver system.
   B. Intraoperative neuromonitoring and wake-up test or motor evoked potentials.
   C. Fusion technique:
      1. Subperiosteal dissection out to the tips of the transverse processes.
      2. Decortication, facet cartilage excision.
      3. Iliac crest autograft or rib graft from thoracoplasty.
   D. Instrumentation techniques: most deformity corrections are now performed using the pedicle screw–based systems.

VIII. Postsurgical care, outcomes, and complications.
   A. Bracing after surgery is not required.
   B. Patients are slowly advanced in their activities until full recovery in 6 to 12 months.

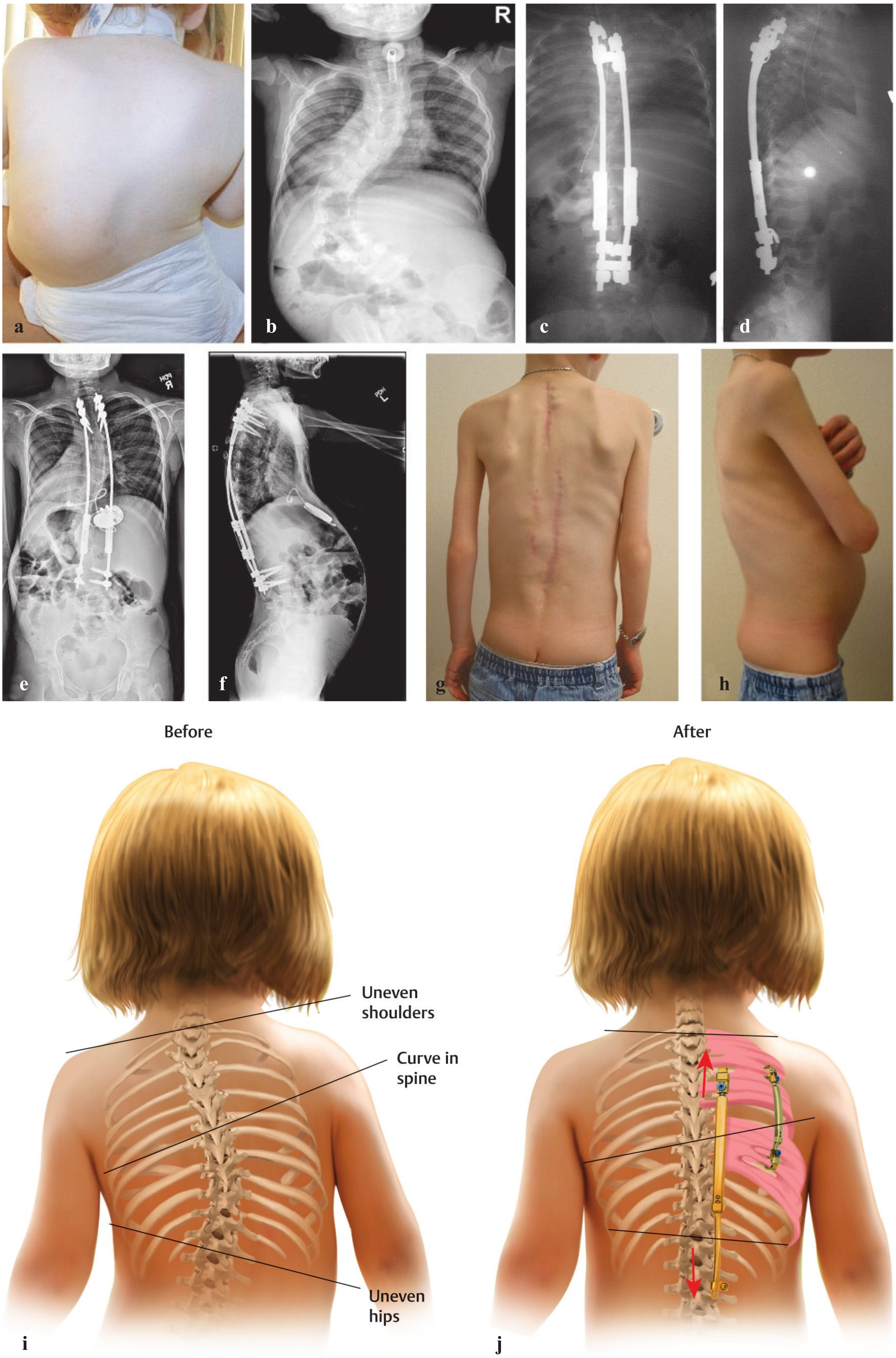


Fig. 21.5 **(a–j)** Vertical expandable prosthetic titanium rib in the pediatric spinal deformity patient.

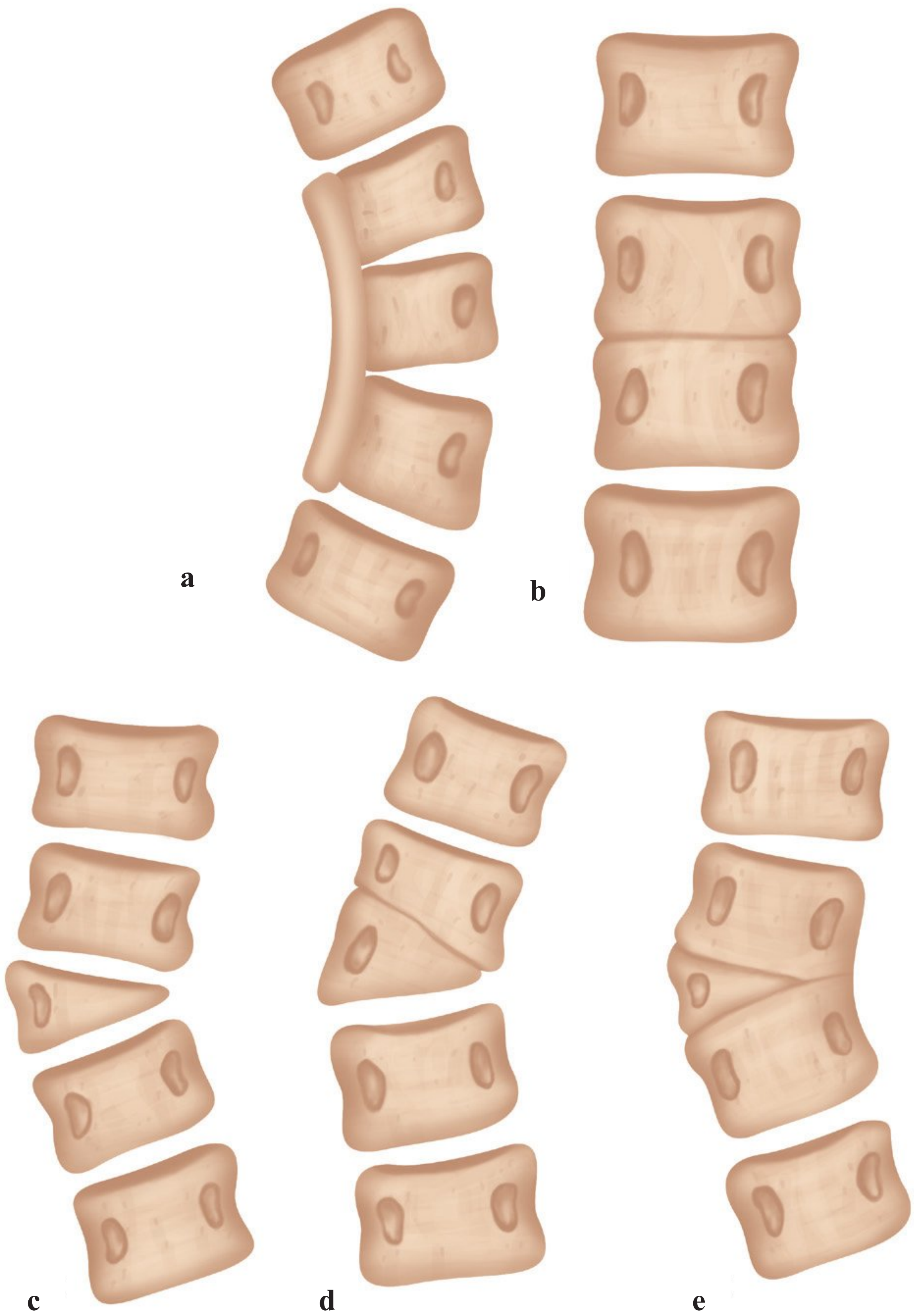


Fig. 21.6 Types of congenital scoliosis. **(a)** Unilateral unsegmented bar. **(b)** Block vertebra. **(c)** Fully segmented hemivertebra. **(d)** Semisegmented hemivertebra. **(e)** Nonsegmented hemivertebra.

C. Depending on instrumentation used, correction percentage varies from 50 to 75%
D. Fusion below L3 increases the incidence of lower back pain.
E. Posterior spinal instrumentation has a reoperation rate of 5 to 19%
F. Other complications:
   1. Delayed infection.
      a. Incidence of 1 to 7%
      b. Requires hardware removal and antibiotics.
   2. Late-onset surgical pain.
      a. Incidence of 5%
      b. Requires hardware removal.
   3. Pseudarthrosis.
      a. Incidence of 3%
      b. Compression instrumentation or bone graft needed for treatment.

21.3 Idiopathic Infantile and Juvenile Scoliosis

I. Idiopathic infantile scoliosis.
  A. Usually detected at 2 to 3 months of age.
  B. Greater incidence in boys than girls.
  C. More common in England.
  D. Ninety percent of cases involve the left thoracic.
  E. Prognosis:
    1. Sixty to 70% of cases resolve spontaneously.
  F. Two types of progressive curves:
    1. Benign curves are typically characterized by > 1-year onset, double curves, and greater flexibility.
    2. Malignant curves are characterized by > 1-year onset, thoracic location, and rigid curves.
  G. Good prognosis if Mehta angle (rib–vertebral angle) is < 20° and the convex rib does not overlap the vertebral body on the posteroanterior radiograph (phase I). In phase II, there is an overlap between the rib and vertebral body, and the prognosis is worse.
  H. The treatment consists of bracing for curves > 30°. If the curve is progressive, surgery is recommended. Options include subcutaneous rod or telescoping rod without fusion or combined anterior and posterior fusion.

II. Idiopathic juvenile scoliosis.
  A. The right thoracic pattern is the most common.
  B. Variable progression: one-third are observed, one-third are braced, and one-third require surgery.
  C. Brace if > 30°.
  D. Surgery if progressive curve > 45° despite brace treatment, especially during puberty.

21.4 Other Type of Scoliosis

I. Congenital scoliosis **(Fig. 21.6).**
  A. Failure of segmentation or formation or both.
  B. Associated anomalies: genitourinary (renal agenesis, ureteral obstruction).
  C. Paralytic scoliosis.
  D. Neurofibromatosis.

II. Neuromuscular scoliosis **(Fig. 21.7).**
  A. General considerations.
    1. Bracing does not prevent the natural progression of the scoliosis.
    2. Differences in surgical approach include the length of fusions and operating on smaller curves.
    3. Hooks and screws are used more frequently.
    4. Segmental Luque wires are used.
    5. Increased rates of complications.
  B. Cerebral palsy.
    1. Scoliosis affected by the imbalance of the paraspinal muscles.

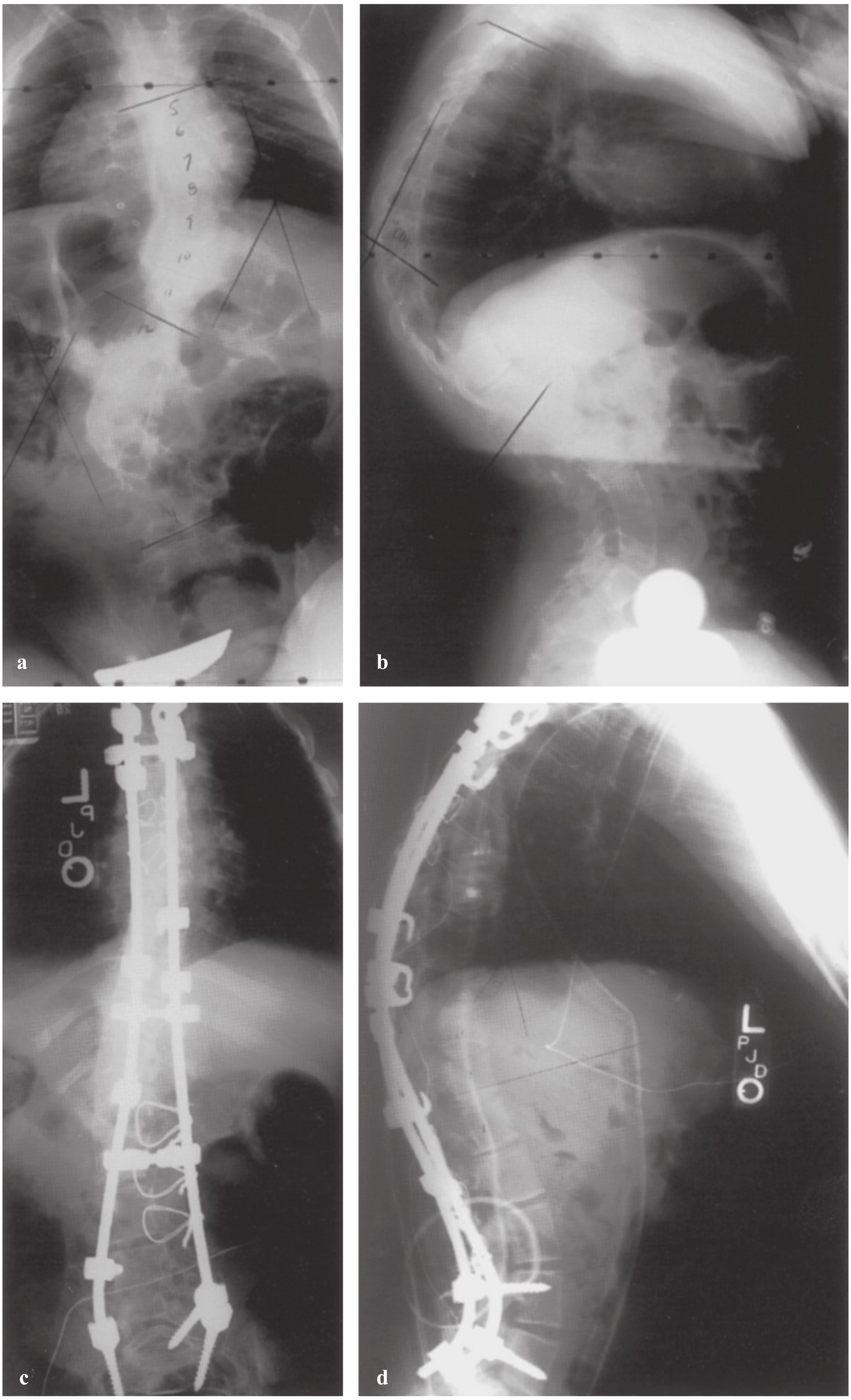


Fig. 21.7 A 13-year-old boy with neuromuscular scoliosis. **(a)** Anteroposterior and **(b)** lateral radiographs. There appears to be lumbar hyperlordosis and a compensatory thoracic kyphosis. **(c,d)** Postoperative radiographs demonstrate a combination of pedicle screw, hook, and sublaminar wiring fixation.

2. Surgery.
    a. Curves > 50°.
    b. Levels to be repaired:
        (1) Ambulatory patient.
            (a) Proximal to distal stable vertebra.
        (2) Nonambulatory patients.
            (a) T2 to pelvis.
    c. Usually posterior approach but anterior approach may need to be performed for curves > 100°.

C. Myelomeningocele.
1. Secondary to birth defect: exposure of the meninges and spinal cord may result in bowel, bladder, motor, and sensory deficits.
2. Incidence of 1 in 1,000: correlated with folate deficiency in pregnancy.
3. Fifteen percent of patients with this deficit are allergic to latex.
4. MRI is used for diagnosis due to the neurological complications.
5. Surgery:
    a. Indicated for patients who have difficulty with sitting or are at risk for developing pressure sores.
    b. A combined posterior and anterior approach is used.

D. Spinal muscular atrophy.
1. Progressive weakness due to loss of anterior horn cell neurons.
2. Three types.
    a. Type I (Werdnig–Hoffmann disease).
        (1) Onset in neonatal period and death by age 2.
    b. Type II.
        (1) Onset at ~ 5 to 6 months of age.
    c. Type III.
        (1) Onset before age 3 and progressive weakness with loss of ambulatory strength by age 15.
        (2) Surgery.
            (a) Indicated in progressive scoliosis.
            (b) The approach should be anterior and posterior for a young patient with large curves and only posterior for the older patient with smaller curves.

E. Duchenne's muscular dystrophy.
1. X-linked recessive disorder.
2. Spinal deformity develops secondary to muscle imbalance only after loss of ambulation.
3. Increased incidence of malignant hypertension with general anesthesia.
4. Preoperative pulmonary function and cardiac contractility assessment are necessary.
5. Surgery:
    a. Indicated in progressive scoliosis > 25 to 30%
    b. A T2 to sacrum posterior approach is used.

21.5 Kyphotic Deformities

I. General considerations of kyphotic deformities.
  A. Anatomical variation.
    1. Cervical lordosis, thoracic kyphosis, and lumbosacral lordosis
    2. The sagittal plumb line (odontoid) should normally cross C7–T1, T12–L1, and the posterior aspect of the sacrum (S1).
    3. Normal thoracic kyphosis:
      a. Twenty to 45°, mean = 34°.
    4. Normal lumbar lordosis:
      a. Forty to 60°.
      b. Two-thirds of lordosis occur between L4–L5 and L5–S1.
  B. Biomechanics.
    1. Anterior column failure results in compression, and posterior column failure results in tension.
    2. Posterior structures:
      a. The lamina and ligamentum flavum are relatively stronger in resisting tension than facets, capsules, and interspinous ligaments.
    3. Deformity increases the moment arm, which can result in further decompensation.
    4. Eccentric loading can affect cartilaginous growth.
      a. Compression decreases growth anteriorly.
      b. Tension increases growth posteriorly, resulting in increased kyphosis.
  C. Classification **(Table 21.4).**
  D. Specific kyphotic deformities.
    1. Postural kyphosis.
      a. Most common in adolescents and young adults.
      b. Round back deformity.
      c. Modest kyphosis (40–60°).
      d. Smooth and flexible.
      e. No radiographic changes.
      f. No specific treatment—conservative management.
    2. Congenital kyphosis.
      a. May be single or multilevel.
        (1) Type I.
          (a) Failure of formation (hemivertebra).
          (b) Worse prognosis for progression and paraplegia.
          (c) Upper spinal levels have a worse prognosis than lower levels.
        (2) Type II.
          (a) Failure of segmentation (bar).
        (3) Type III.
          (a) Combination of I and II.
        (4) Treatment.
          (a) Nonoperative treatment is ineffective.

Table 21.4 Classification of kyphotic deformities

Type	Notes
Postural	
Congenital	Defect of formation Defect of segmentation Mixed
Neuromuscular **(Fig. 21.8)**	
Scheuermann's disease **(Fig. 21.9)**	
Myelomeningocele	Developmental (late paralytic) Congenital (present at birth)
Traumatic	Due to bone, ligament, and/or cord injury
Postsurgical	Postlaminectomy Following excision of a vertebral body
Postirradiation	
Metabolic	Osteoporosis – Senile – Juvenile Osteomalacia Osteogenesis imperfecta
Skeletal dysplasia	Achondroplasia Mucopolysaccharidoses Neurofibromatosis
Collagen disease	Marie–Strümpell disease
Tumor	Benign Malignant – Primary – Metastatic
Inflammatory and infectious	

(b) Surgery:
   i. Type I lesions.
      - Posterior in situ fusion if < 50° at 1 to 5 years.
      - Anterior and posterior fusion if > 50° and older children.
         - Better correction and maintenance.
         - Lower rate of pseudarthrosis.

- Anterior decompression.
  - Release of all the tethering structures:
    - Anterior longitudinal ligament.
    - Intervertebral disk and end plate.
    - Posterior longitudinal ligament.
  - Intraoperative distraction and correction of the deformity:
    - Rib, fibula, or iliac crest strut grafts.
  - Simultaneous or second-stage posterior fusion with compression instrumentation.
  - Postoperative bracing is recommended.

ii. Type II lesions.

- Posterior fusion only if kyphosis is < 55°.
  - For severe deformity, an anterior osteotomy and correction and fusion may be attempted followed by a posterior fusion.
- Skeletal traction is contraindicated as it may cause paraplegia.

3. Scheuermann's disease (juvenile kyphosis).

a. Scheuermann first described radiological manifestation of this disease in 1920.

(1) The incidence is 0.4 to 8.3% of the population, but only 1% seek medical attention.

(2) Pathogenesis is unknown.

(a) Familial tendency with no genetic link.

(b) Collagen weakness and stunted ossification of the vertebral end plate are characteristic.

(c) Osteopenia.

(d) Nutritional deficiencies.

(e) Mechanical alterations and muscle weakness have been theorized.

i. No scientific evidence exists.

ii. Growth centers adjacent to the vertebral end plate (not ring apophyses):

- Anterior cartilaginous columns experience stunted growth with axial loading.
- Posterior physis hypertrophy due to tensile forces.

iii. With kyphotic deformity, spinal flexors become stronger than extensors because of the moment arm.

(3) Pathoanatomy:

(a) Thickened and contracted anterior longitudinal ligament.

(b) Wedging of the anterior vertebral bodies.

(c) Nucleus pulposus.

i. Protrusion anteriorly and into the bony spongiosa (Schmorl's nodes).

(4) Clinical findings:

(a) Onset is commonly between 12 and 14 years of age.

(b) Equal male:female ratio.

(c) Deformity is the most common presenting complaint.
(d) Pain occurs in ~50% among those who seek medical attention.
i. Increased rate of symptomatology if the lumbar spine is involved
• Some patients develop lumbar spondylolysis later.

(5) Physical examination:
(a) Increase in thoracic kyphosis (rigid).
(b) Compensatory lumbar and cervical lordosis:
i. Round shoulders.
ii. Forward tilting of the head.
(c) Muscle tightness and contractures are seen, typically in the hamstrings.
(d) Thirty percent have an associated mild scoliosis.

(6) Radiographic findings:
(a) Early:
i. Disordered endochondral ossification.
ii. Irregular end plates.
iii. Narrowing of the intervertebral disk space.
iv. Schmorl's nodes.
(b) Intermediate:
i. Vertebral wedging.
ii. Increasing kyphosis > 45°.
• More than 5° of anterior wedging in three or more vertebrae at the apex of the kyphosis (Sorenson's criteria).
(c) Late:
i. Degenerative changes.
• Osteophytes.
• Facet hypertrophy.
(d) Standing lateral and supine hyperextension views are used to assess the rigidity of the curve.

(7) Treatment:
(a) Observation is indicated for mild deformity with minimal symptoms.
(b) Bracing indications:
i. Vertebral wedging > 5°.
ii. Kyphotic curves between 45 and 65° and 1 to 2 years of growth remaining:
• Milwaukee brace for apex above T9.
• Thoracolumbar Sacral Orthosis (TLSO) for the apex below T9 and thoracolumbar curves.
iii. Curve correction and wedging improvement of ~40% can be expected after 6 to 12 months.
iv. The brace should be weaned with skeletal maturity, but loss of correction is expected after 10 years.
(c) Exercise:
i. Stressing pelvic tilt, abdominal strengthening, spinal flexibility, and thoracic spine extension exercises is an important part of the treatment plan.

(8) Surgery:
  (a) Indications.
    i. Severe deformity after growth completion with unrelenting pain.
      • Typically > 75° and > 10° wedging in three or more contiguous vertebral bodies.
    ii. Resistance to bracing after 6 months.
    iii. Neurological signs or symptoms.
  (b) Techniques.
    i. Posterior-only instrumented fusion.
      • Curves < 75° and bending correction to < 50°.
      • Posterior instrumentation should extend the entire kyphotic region, and distally it should include one lordotic vertebra (usually L1 or L2). The posterior sacral vertical line is a vertical line that crosses the posterior superior corner of the sacrum. The vertebra that bisects this line is sometimes considered as the end vertebra for fusion to prevent junctional deformity.
    ii. Anterior fusion (transthoracic approach) followed by posterior fusion and instrumentation.
      • Curves > 75° with minimal bending correction (> 50°).
  (c) Postoperative protocol.
    i. TLSO for 6 to 9 months until solid fusion.
  (d) Complications.
    i. Pseudarthrosis and instrumentation failure (greater in posterior fusion alone).
    ii. Loss of correction.
    iii. Infection.
    iv. Pulmonary complications.
    v. Neurological deficits.

4. Neuromuscular kyphosis **(Fig. 21.8).**
  a. Associated conditions:
    (1) Poliomyelitis.
    (2) Anterior horn cell diseases (spinal muscular atrophy).
    (3) Cerebral palsy.
    (4) Charcot–Marie–Tooth disease.
    (5) Muscular dystrophy.
    (6) Friedreich's ataxia.
  b. Lack of the extensor muscle strength contributes to development of a kyphotic deformity.
  c. The natural history is progressive even after skeletal maturity.
  d. Treatment:
    (1) Bracing until the patient is ~ 11 to 12 years old to maximize truncal height.
    (2) Posterior fusion with instrumentation in milder and flexible curves.
      (a) Compression instrumentation is preferred over Luque rods with sublaminar wires for correction of kyphosis.

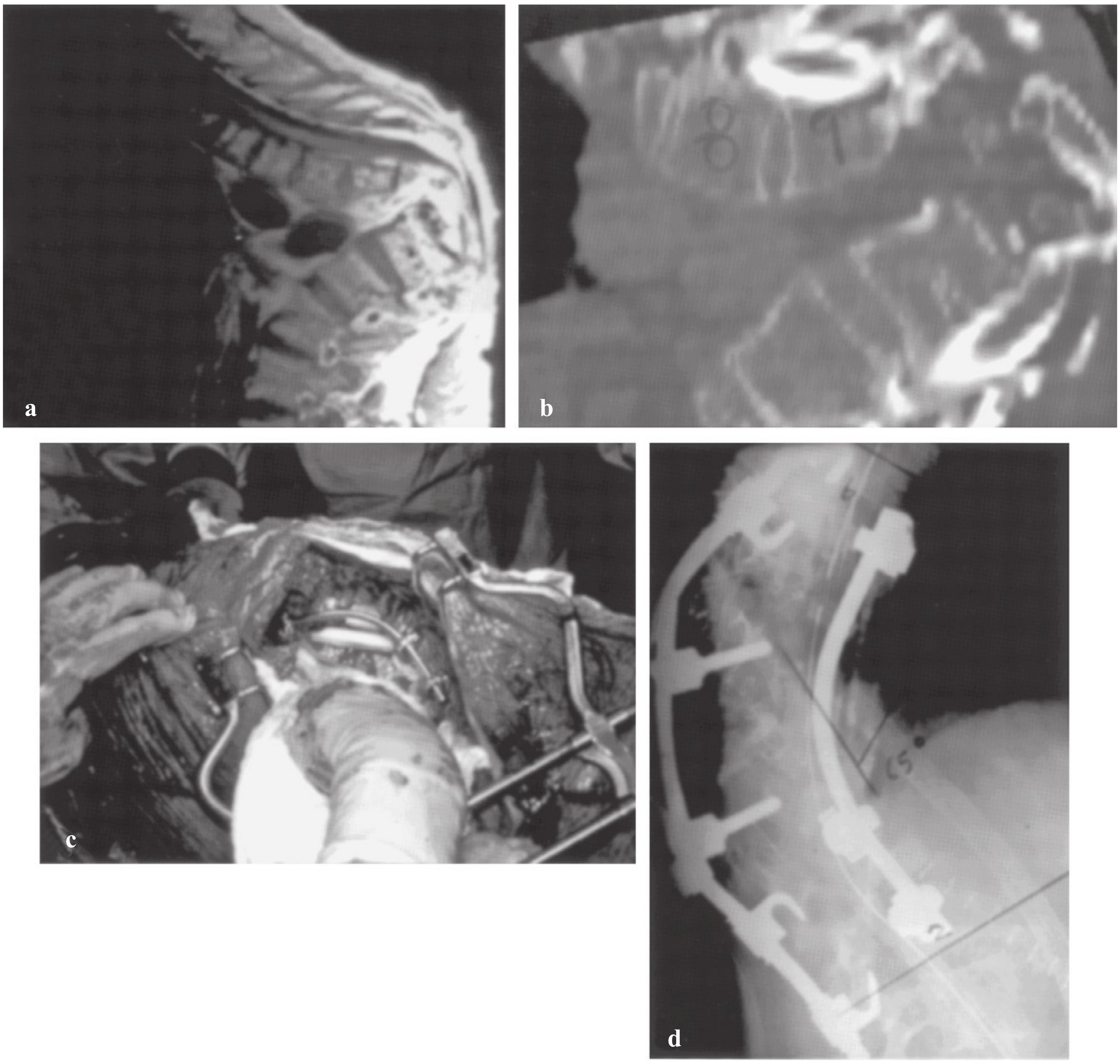


Fig. 21.8 Images of a 44-year-old man with neurofibromatosis who presented with severe kyphoscoliosis and paraplegia. A simultaneous anterior and posterior approach was performed on this patient to combine anterior and posterior procedures. The steps of the procedures included a posterior osteotomy, anterior vertebrectomy, spinal cord decompression, anterior fusion with strut grafting, posterior compression instrumentation, and anterior instrumentation, in this order. **(a)** Preoperative magnetic resonance image showing 170° kyphosis with cord compression. **(b)** Computed tomographic (CT) myelogram demonstrating severe kyphotic deformity with signal cutoff distal to the T9 vertebra. **(c)** Intraoperative photograph showing anterior vertebrectomy, fibular strut graft, and anterior instrumentation. **(d)** Postoperative radiograph showing 65° of kyphosis.

(b) Combined anterior and posterior fusion with instrumentation in severe fixed deformities.

5. Myelomeningocele.
   a. Congenital.
      (1) It is generally not recommended to correct deformity at birth because of the lack of bone stock and associated problems.
   b. At 3 to 5 years of age, posterior resection followed by correction of kyphosis.
      (1) Posterior instrumentation should be applied two to three vertebrae proximal to the apex.
      (2) Followed by 6 to 9 months of bracing.

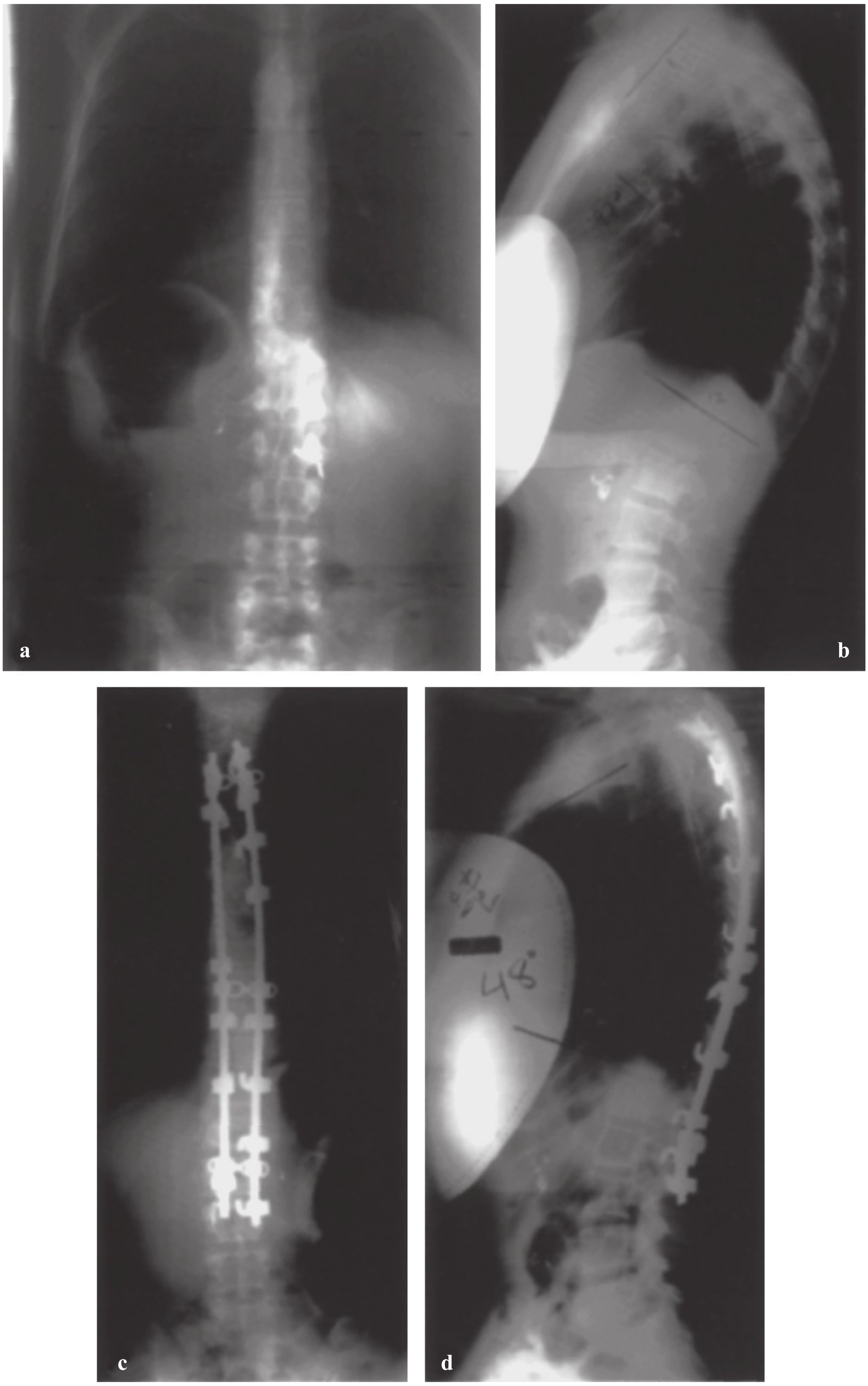


Fig. 21.9 Preoperative radiographs of an 18-year-old woman with Scheuermann's kyphosis. **(a)** Anteroposterior radiograph demonstrating no coronal plane deformities. **(b)** Lateral radiograph measuring 80° of kyphosis from T1 to T12. **(c,d)** Because of the patient's relative flexibility, an all posterior approach was performed, restoring her sagittal balance.

6. Developmental or paralytic kyphosis.
   a. The deformity is progressive because the extensors of the spine (sacrospinalis and quadratus lumborum muscles) rotate anteriorly to increase flexion force.
   b. Treatment:
      (1) Bracing is indicated for young patients with mild deformity.
      (2) Posterior fusion involving long fusion to the sacrum.
      (3) Anterior release and fusion followed by posterior fusion with compression instrumentations.
7. Posttraumatic kyphosis.
   a. Acute or late secondary to severe compression fractures, burst fractures, or fracture-dislocations.
   b. More common for unstable fractures after conservative treatment.
   c. Symptoms include deformity, pain, and neurological deficit.
   d. Treatment:
      (1) Observation and conservative treatment if the deformity causes mild pain that is controllable.
      (2) Surgery is most commonly done by a combined anterior and posterior fusion.
         (a) Anterior decompression with anterior instrumentation alone may be done if adequate correction has been achieved and the instrumentation is stable.
         (b) Posterior-only transpedicular osteotomy may be done if there is no neurological compression.
8. Postsurgical kyphosis.
   a. Usually observed after laminectomy for spinal cord tumors and syringomyelia.
   b. Fusion is recommended at the time of surgery when extensive laminectomy has been performed.
   c. Severe deformity is approached with a combined anterior and posterior fusion.
9. Infectious kyphosis **(Fig. 21.10).**
   a. Infection may be caused by tuberculosis and pyogenic osteomyelitis.
   b. The thoracolumbar junction is most commonly affected.
   c. Treatment:
      (1) Antibiotic treatment includes long-term intravenous antibiotics and bracing.
      (2) Anterior debridement and fusion indications:
         (a) Unresponsive to medical treatment.
         (b) Multiple-level involvement.
         (c) Spinal cord compression.
         (d) Presence of an abscess.
      (3) Progressive kyphosis needs anterior and posterior procedures.
10. Inflammatory kyphosis (ankylosing spondylitis).
   a. Characterized by loss of a lumbar lordosis and increased cervical and thoracic kyphosis.
   b. Primary location of disabling deformity should be assessed.

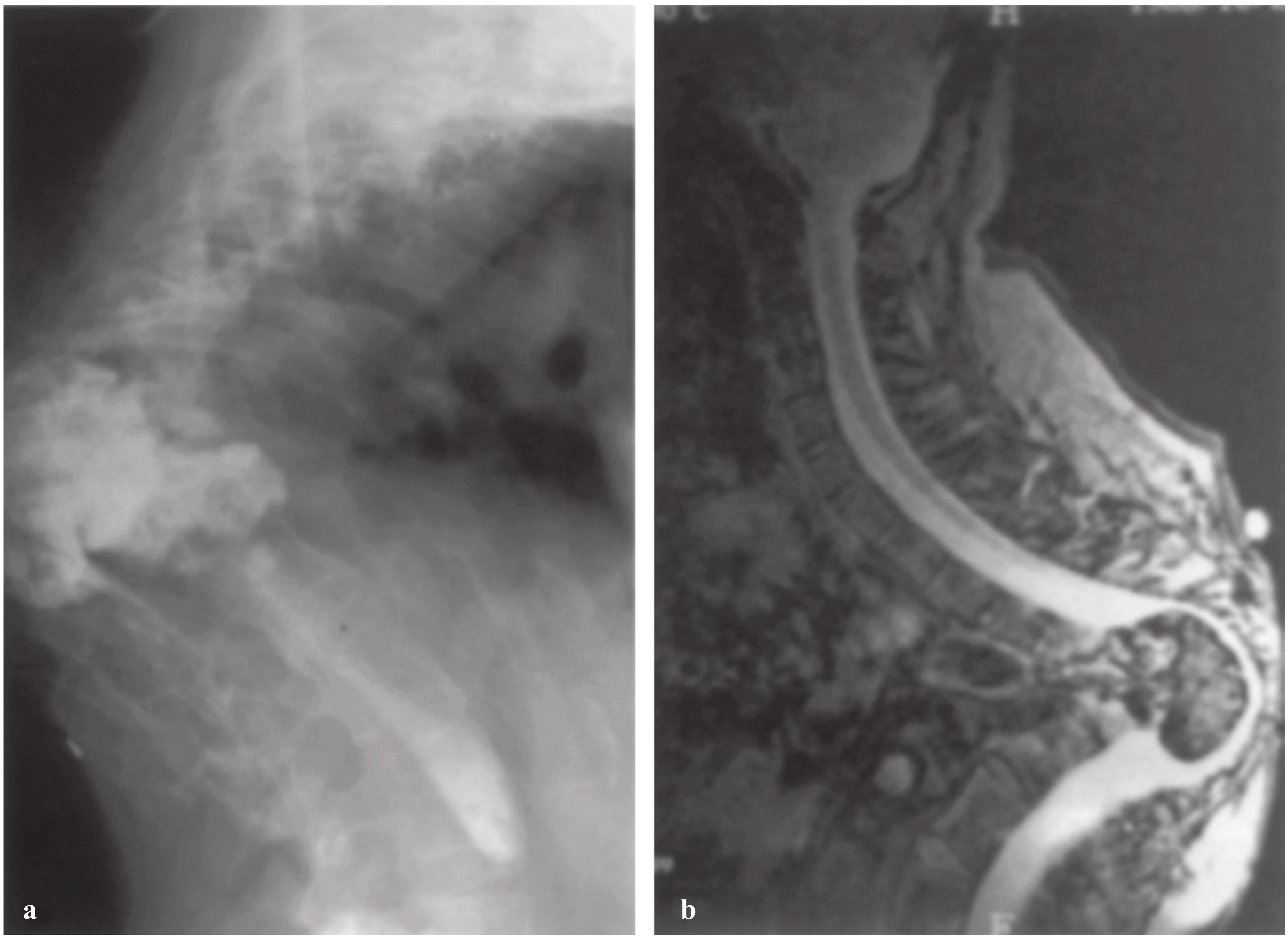


Fig. 21.10 A 65-year-old man with old tuberculosis who had a posterior fusion 40 years prior. Despite severe kyphosis, this patient was ambulatory with minimal symptoms. **(a)** Lateral radiograph showing severe angular kyphosis. **(b)** Sagittal T2-weighted magnetic resonance image (MRI) showing an old granuloma and spinal cord compression at the apex of the kyphotic deformity.

c. Hip flexion contracture should be corrected first.
   (1) May avoid the need for spinal surgery.
d. Lumbar osteotomy is indicated for a significant loss of lumbar lordosis.
   (1) Types of osteotomy.
      (a) Smith–Petersen/Ponte opening wedge extension osteotomy.
         i. Typically performed at L2–L3 and L3–L4 junction followed by instrumentation and fusion.
         ii. The angle of correction corresponds to the spine flexion deformity on standing.
         iii. The apex of the osteotomy should be anterior to the neural tube and at the junction of the posterior longitudinal ligament and intervertebral disk.
      (b) Pedicle subtraction osteotomy.
         i. Does not lengthen the anterior column.
         ii. A closing wedge osteotomy shortens the vertebral column and is safer neurologically.
         iii. Posterior resection of the lamina, pars, and pedicles is performed followed by a vertebral wedge resection.

11. Flat back syndrome.
    a. Etiologies.
        (1) Distraction of the lumbar spine (Harrington instrumentation).
        (2) Lumbar fusion with loss of lordosis.
    b. Clinical findings.
        (1) Sagittally imbalanced posture.
        (2) Back pain with referred pain down to legs.
        (3) Transition syndrome above and below kyphosis.
        (4) Compensatory hip and knee flexion contractures.
    c. Treatment.
        (1) Conservative treatment with nonsteroidal anti-inflammatory drugs, physical therapy, range of motion exercises, pain management.
        (2) Surgery.
            (a) Lumbar osteotomy to balance the sagittal contour of the spine.
                i. Pedicle subtraction osteotomy.
                ii. Smith–Petersen osteotomy.

Suggested Reading

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22 Pediatric Cervical Spine Disorders

22.1 General Considerations

I. Developmental anatomy.
  A. Atlas.
    1. Neurocentral synchondrosis forms at 6 to 24 months.
      a. Fusion begins at 4 to 6 years.
      b. Posterior synchondrosis fuses at 5 years.
  B. Dens.
    1. Two primary ossification centers coalesce at 1 to 3 months of age.
    2. Separated from the vertebral body by a dentocentral synchondrosis that fuses at 6 to 8 years.
  C. Normal variants.
    1. Posterior bifid C1 arch.
    2. Bipartite superior articular surface of the atlas.
    3. Pseudonotch of the atlas.
    4. Absence or partial absence of the posterior arch of the atlas.
    5. Posteriorly displaced spinolaminar line of the axis.
    6. Posteriorly angulated dens.
    7. Pseudosubluxation of the axis ($<$ 10 years old).

II. Physical examination.
  A. Limited range of motion.
  B. Torticollis.
  C. Facial asymmetry.
  D. Associated abnormalities.
    1. Scoliosis, renal, cardiac, or other head and neck anomalies.

III. Diagnostic evaluation **(Fig. 22.1** and **Fig. 22.2).**
  A. A flexion-extension view is very important to assess stability.
  B. Critical measurements on flexed lateral view:
    1. Atlantoaxial interval: 4.5 mm (children), compared with 3 mm (adults).
      a. From the posterior aspect of the anterior C1 arch to the odontoid process.
    2. Space available for spinal cord (SAC): 13 mm.
      a. Determines the minimal amount of space required within the spinal canal to accommodate the spinal cord.

IV. Specific disorders.
  A. Basilar invagination **(Fig. 22.3).**
    1. Deformity of the bones at the base of the skull at the margin of the foramen magnum.
      a. The odontoid is migrated cephalad.

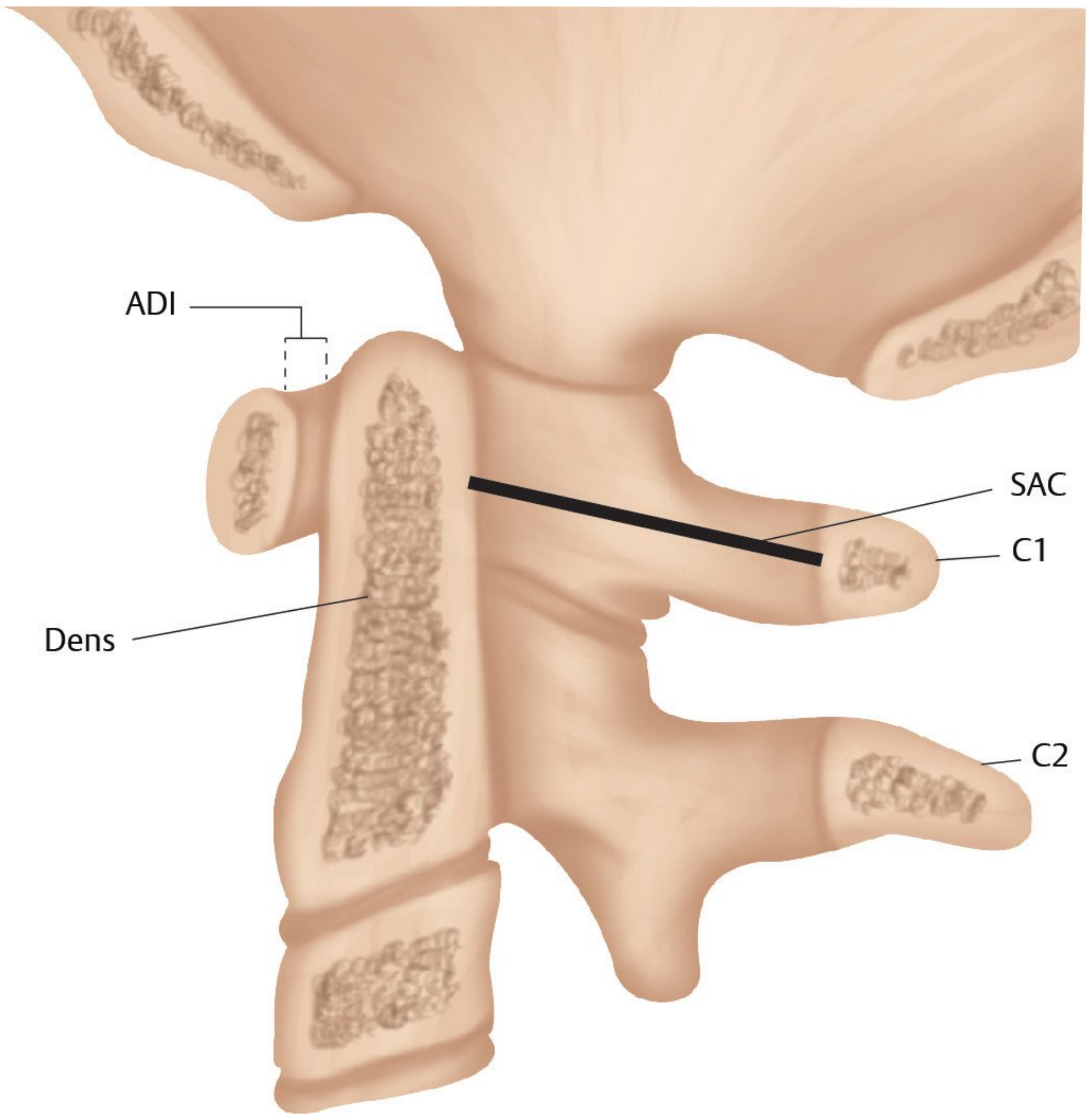

Fig. 22.1 Lateral illustration of the normal relationship of the C1–C2 articulation demonstrating the atlantodens interval (ADI) and the space available for cord (SAC).

2. Types.
   a. Primary.
      (1) Congenital.
      (2) Associated with other findings.
         (a) Atlanto-occipital fusion.
         (b) Hypoplasia of atlas.
         (c) Bifid posterior arch of the atlas.
         (d) Odontoid abnormalities.
         (e) Klippel–Feil syndrome.
   b. Secondary.
      (1) Developmental condition with softening of the base of the skull.
      (2) Associated with other clinical disorders.
         (a) Osteomalacia.
         (b) Rickets.
         (c) Paget's disease.
         (d) Osteogenesis imperfecta.
         (e) Renal osteodystrophy.
         (f) Rheumatoid arthritis.
         (g) Neurofibromatosis.
         (h) Ankylosing spondylitis.
         (i) Achondroplasia.

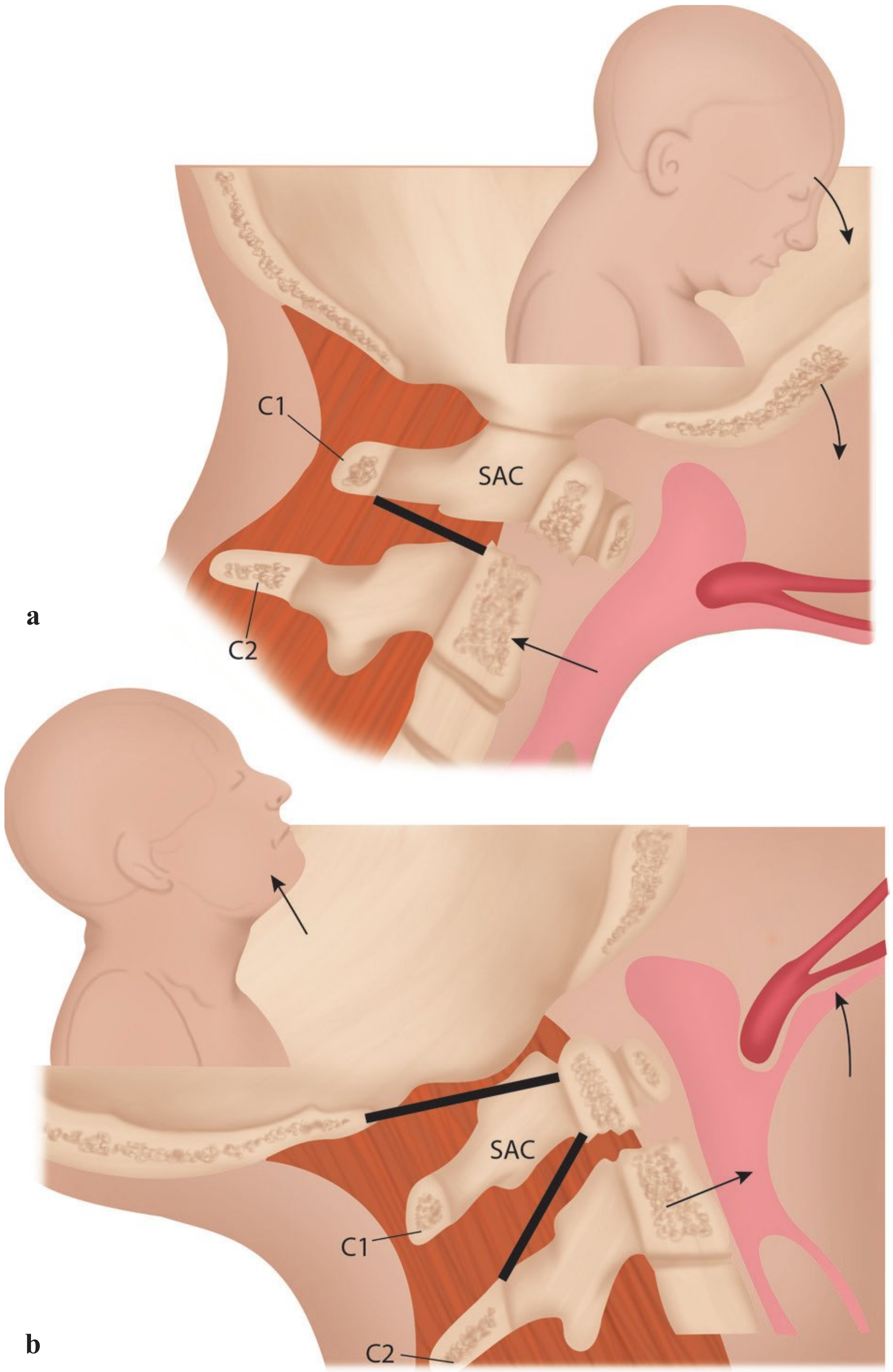

Fig. 22.2 **(a)** In flexion, the space available for cord (SAC) may decrease between the posterior aspect of the dens and the anterior aspect of the C1 posterior ring. **(b)** In extension, the SAC may decrease between the posterior aspect of the dens and the anterior aspect of the C2 lamina or the foramen magnum.

3. Clinical findings.
   a. Commonly become symptomatic in second and third decades.
      (1) Short neck.
      (2) Asymmetric face.
      (3) Torticollis.
      (4) Weakness/paresthesias.
      (5) Cranial nerve palsies.
      (6) Cerebellar signs (unsteady gait and nystagmus).
      (7) Pain (head and neck).

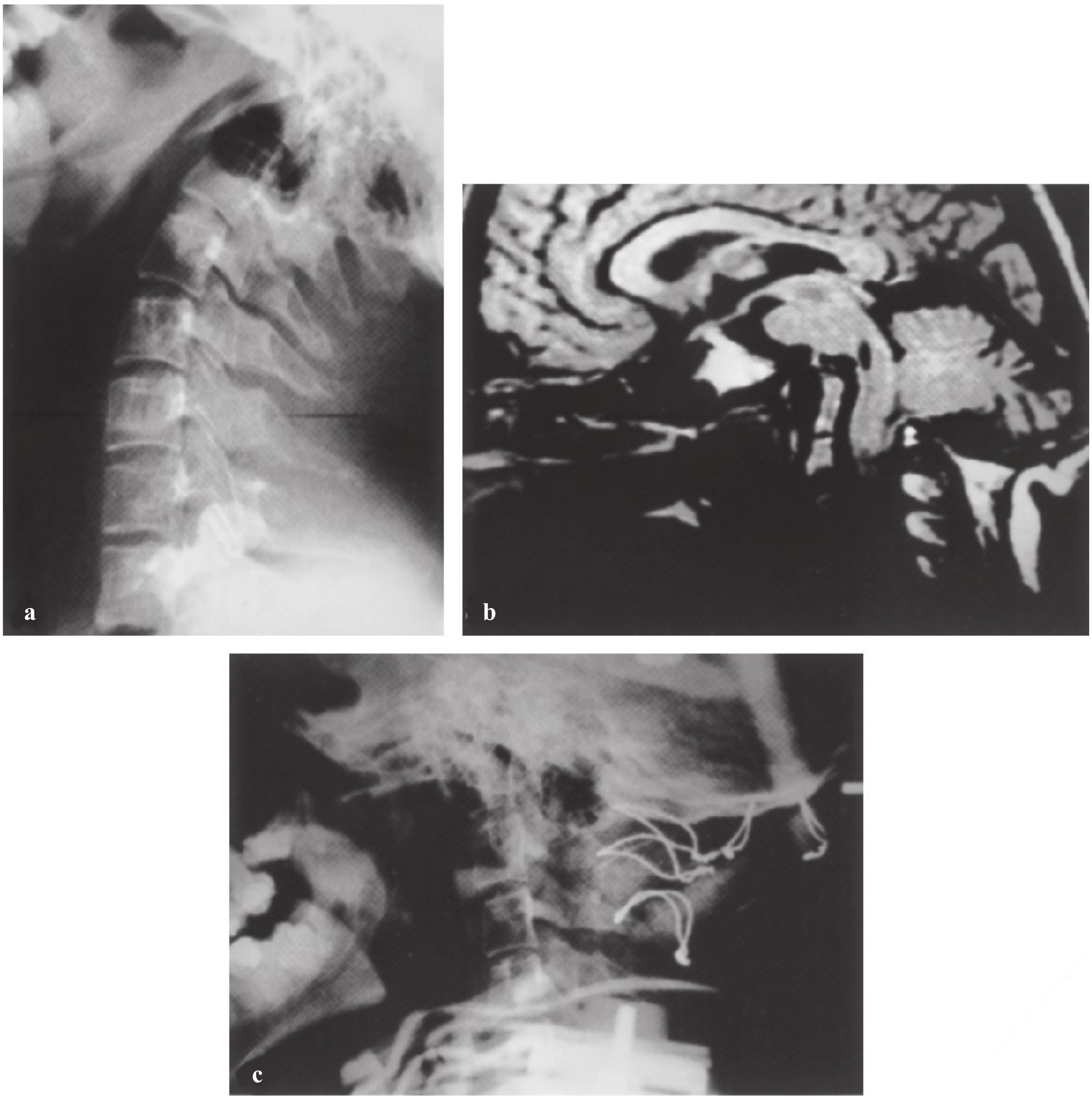

Fig. 22.3 An 18-year-old man with osteogenesis imperfecta and basilar invagination presented with unrelenting headache, neck pain, and myelopathy. **(a)** Lateral radiograph demonstrating basilar invagination with the odontoid protruding into the foramen magnum. **(b)** Magnetic resonance imaging scan showing the odontoid indenting the brain stem. **(c)** Postoperative lateral radiograph showing a triple wire technique and posterior occipital–cervical fusion.

(8) Syncope and dizziness (vertebral artery compression).
(9) Seizures/hydrocephalus (cerebrospinal obstruction).

4. Treatment.
   a. Posterior impingement.
      (1) Suboccipital craniectomy and decompression of the posterior ring of C1 with posterior stabilization.
   b. Anterior impingement.
      (1) Mobile odontoid:
         (a) Occipitocervical fusion in extension is recommended.

(2) If the odontoid cannot be reduced, anterior excision of the odontoid and posterior stabilization are recommended.

B. Klippel–Feil syndrome **(Fig. 22.4).**

1. Congenital fusion of cervical vertebrae.
2. Failure of normal segmentation of the cervical spine during the third to eighth week.

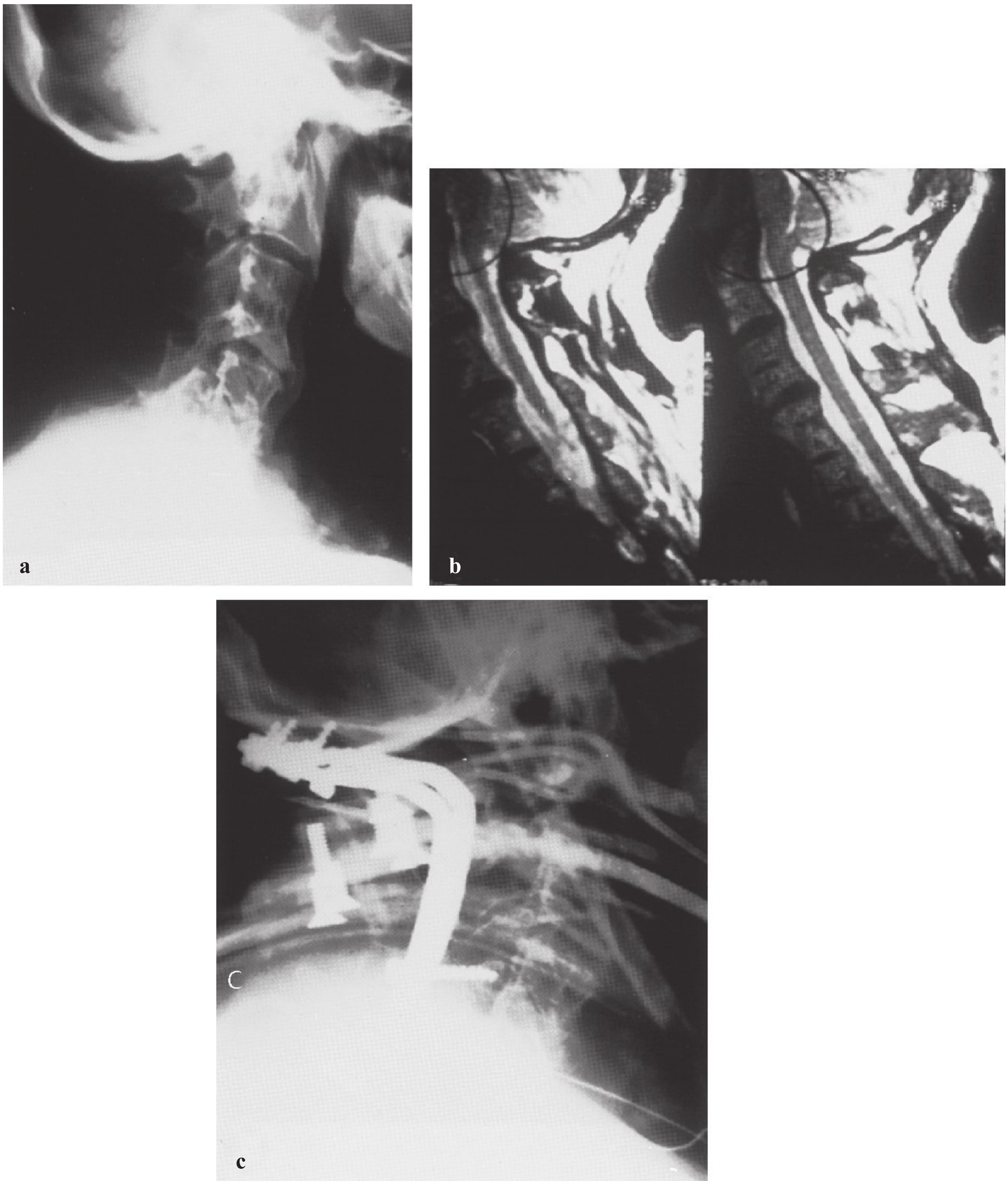

Fig. 22.4 A 55-year-old man with Klippel–Feil syndrome and basilar invagination. The patient presented with severe neck pain and headache. **(a)** Lateral radiograph showing congenital fusion between C3–C4 and C5–C6. **(b)** Magnetic resonance imaging also shows the congenitally fused vertebrae with disk degeneration at C4–C5. Basilar invagination is noted as well. **(c)** Postoperative radiograph showing an occiput–C6 fusion with plate–screw fixation.

3. Associated anomalies.
   a. Genitourinary (35%).
   b. Central nervous system.
   c. Cardiopulmonary.
   d. Sprengel's deformities (40%).
   e. Upper extremity anomalies.
   f. Scoliosis (60%).
4. Clinical findings.
   a. Low posterior neck line.
   b. Short neck.
   c. Limited neck motion.
      (1) The majority of patients have a normal appearance with mild restriction of motion.
5. Radiographic findings.
   a. Vertebral synostoses.
   b. Flattening and widening of the vertebral bodies.
   c. Absent disk spaces or hypoplasia.
6. Treatment.
   a. The majority of patients are asymptomatic.
      (1) Symptoms may appear later in life.
   b. Conservative treatment is indicated for most patients.
      (1) Anti-inflammatory medications.
      (2) Exercise program.
   c. Fusion may be indicated in select patients with instability and spinal cord impingement.

C. Anomalies of the odontoid.
1. Etiology.
   a. Trauma.
      (1) Salter I fracture with a nonunion **(Fig. 22.5).**
   b. Congenital.
      (1) Failure to fuse (normally fuses at 3–6 years).
2. Clinical findings.
   a. Neck pain.
   b. Torticollis.
   c. Neurological symptoms.
3. Treatment.
   a. Conservative if stable.
   b. Surgical intervention.
      (1) Indications for surgical stabilization.
         (a) More than 7 to 10 mm of instability even without symptoms.
         (b) Atlantodens interval (ADI) > 4.5 mm with flexion-extension films and SAC < 13 mm.
      (2) Technique.
         (a) C1–C2 fusion with wire fixation:
            i. The vertebral artery is proportionally closer to the midline in the child.

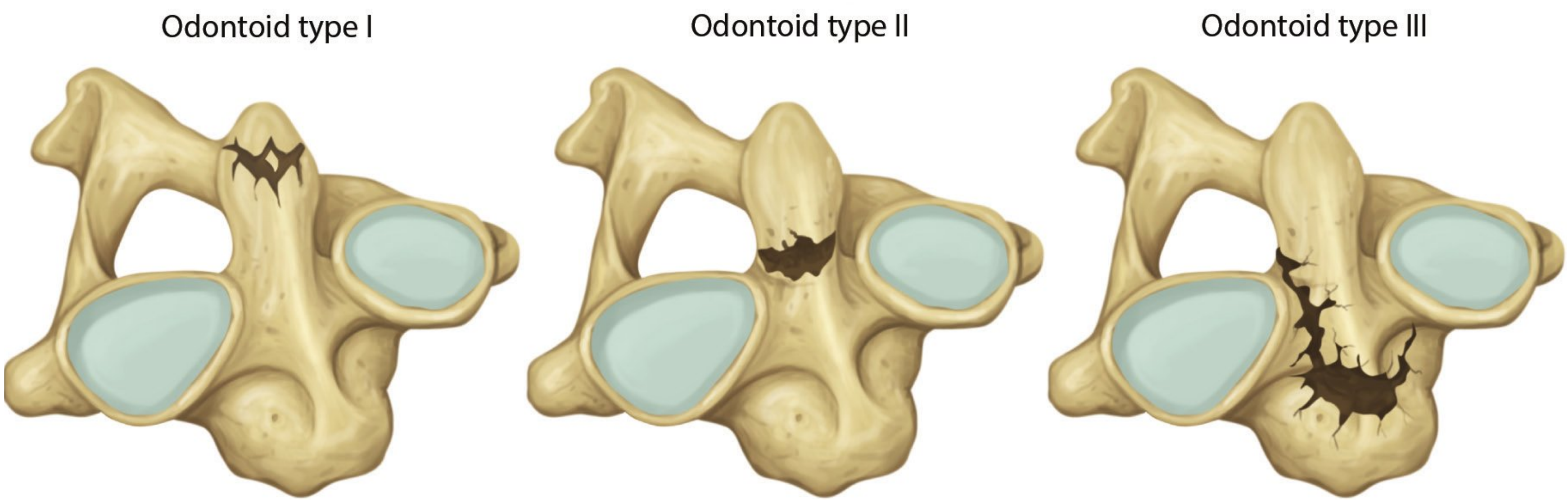

Fig. 22.5 Illustration of the three types of odontoid fractures.

(b) Preoperative traction and reduction may be necessary.
(c) Postoperative halo vest stabilization.
(d) C1–C2 fusion with C1–C2 transarticular screw fixation:
  i. More rigid and avoids halo vest postoperatively.
(e) Occiput–C2 fusion is necessary if the C1 ring is deficient.

D. Congenital atlanto-occipital fusion.
  1. Failure of segmentation.
  2. Most commonly recognized anomaly of the craniovertebral junction.
  3. Prone to C1–C2 instability if associated with C2–C3 fusion or anomalies of the odontoid (70%).
  4. Associated with dwarfism, funnel chest, pes cavus, syndactyly, cleft palate, and genitourinary anomalies.
  5. Clinical findings.
    a. Short neck, restricted neck motion, and torticollis.
    b. Fifty percent have relative basilar impressions secondary to diminished vertebral height of the atlas ring.
    c. Neurological involvement, especially if the odontoid is above the foramen magnum level.
  6. Radiographic findings.
    a. Flexion-extension view.
      (1) ADI > 3 to 4 mm.
      (2) SAC < 13 mm.
  7. Treatment.
    a. Conservative treatment.
      (1) Cervical collar.
      (2) Traction.
    b. Surgery.
      (1) Occiput–C1–C2 fusion after traction and reduction if C1–C2 instability is present.
      (2) Posterior decompression and fusion if there is posterior compression of the spinal cord.

E. Torticollis (wry neck).
   1. Typically discovered in the first 6 to 8 weeks of life.
   2. Ischemia and contracture of the sternocleidomastoid muscle may be pathogenic.
      a. Venous occlusion and fibrous replacement of tissue secondary to intrauterine position.
   3. Clinical findings:
      a. Twenty percent associated incidence of congenital hip dislocation.
      b. Eighty-five percent of cases involve the right side.
      c. The head is tilted toward the involved side, and the chin is rotated to the opposite side.
      d. Soft, nontender enlargement beneath the skin resolves in 6 to 12 weeks.
      e. Contracture of the muscle follows with decreased range of motion of the neck.
      f. Facial asymmetry and mild dorsal compensatory scoliosis.
   4. Differential diagnosis:
      a. Congenital cervical spine anomalies.
      b. Extraocular muscle imbalance.
   5. Treatment:
      a. Stretching exercise, positioning, and brace.
         (1) Eighty-five to 90%response within 1 year.
      b. Surgical indications.
         (1) After 1 year if persistent facial asymmetry.
         (2) Head tilting.
         (3) Decreased range of motion.
      c. Surgical options.
         (1) Unipolar/bipolar release.
            (a) Bipolar release with Z-lengthening gives the best results.
               i. Be careful of the posterior auricular nerve and spinal accessory nerve.

F. Atlantoaxial instabilities.
   1. Etiologies.
      a. Inflammation.
         (1) Pharyngeal infection (Grisel's syndrome).
         (2) Juvenile rheumatoid arthritis.
      b. Down's syndrome.
         (1) Twenty-five percent incidence.
         (2) Boys older than 10 years are at greater risk for myelopathy following rupture of transverse ligament.
      c. Dysplasia.
         (1) Achondroplasia.
         (2) Diastrophic dysplasia.
         (3) Spondyloepiphyseal dysplasia.
         (4) Morquio's syndrome.
         (5) Larsen's syndrome.
      d. Congenital anomalies.

e. Spontaneous rotatory subluxation of C1–C2.
   (1) Fielding classification.
      (a) Type I.
         i. Simple shift without displacement.
      (b) Type II.
         i. Less than 5 mm of C1–C2 displacement.
      (c) Type III.
         i. Greater than 5 mm of C1–C2 displacement.

2. Treatment.
   a. Conservative.
      (1) Mild rotatory deformity.
         (a) Collar.
         (b) Analgesics.
   b. Surgical.
      (1) C1–C2 fusion if neurological symptoms present.
      (2) SAC < 13 mm.

G. Traumatic cervical injuries.

1. Cervical spine fractures.
   a. Vertebral fractures account for 2 to 3% of all childhood injuries.
   b. Fifteen percent of all spinal cord injuries occur in children.
   c. Under 10 years, bony injuries are less common.
   d. Patient positioning may be a problem in children under 5 years.
      (1) The head is larger than the trunk.
         (a) The back board may displace the fracture.
         (b) Keep the head lower than the chest.
2. Radiographic evaluation.
   a. Interpretation of cervical radiographs is more difficult.
      (1) Incomplete ossification.
      (2) Normal anatomical variants.
         (a) Pseudosubluxation of C2–C3.
   b. Spinal cord injury without radiographic abnormality (SCIWORA):
      (1) Common in children under 10 years.
      (2) Magnetic resonance imaging (MRI) is helpful in identifying the location and extent of injury.
3. Specific injuries.
   a. Occipitoatlantal instability.
      (1) Most injuries are fatal.
      (2) Radiographs:
         (a) More than 1 mm increase in distance between odontoid tip and basion.
         (b) Power's ratio (distance from the basion to the posterior arch of C1:distance from the opisthion to the anterior arch of C1).
            i. Greater than 1 is consistent with instability.
      (3) Treatment:
         (a) Occiput–C1 fusion and halo brace.

b. Jefferson fracture **(Fig. 22.6).**
   (1) Axial loading injury mechanism.
      (a) Usually have an associated head injury.
      (b) More commonly recognized due to the increased use of computed tomography (CT).
   (2) Radiographic finding.
      (a) Widening between odontoid and lateral mass of C1.
      (b) Overhang of the lateral mass of C1 may be normal in a child due to differential ossification.
      (c) CT is the best for delineating fractures and helps to differentiate the following:
         i. Neurocentric synchondrosis (fused by 6 years).
         ii. Posterior synchondrosis (fused at 5 years).
         iii. Irregular ossification.
            • Especially the anterior arch may have multiple ossification centers.
   (3) Treatment.
      (a) Minerva orthosis or halo brace, depending on displacement or rupture of the transverse ligament.
c. Odontoid fractures.
   (1) Usually occur in children < 4 years old because synchondrosis fuses by 6 years.
   (2) Radiographs.
      (a) Angulation of odontoid with displacement (ADI > 4.5 mm).
      (b) Flexion-extension radiographs.
         i. May demonstrate instability and displacement of the fracture.
         ii. Great caution should be taken during the flexion and extension examination.

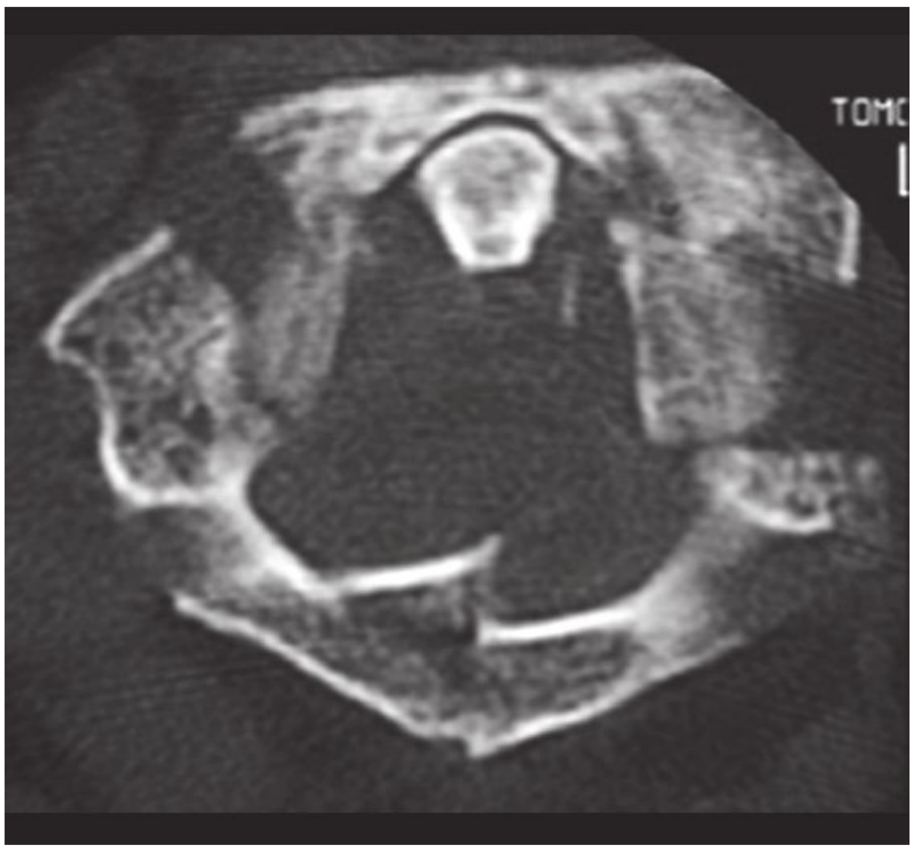

Fig. 22.6 Axial computed tomographic image of a Jefferson fracture. (From Imhof H, ed. Spinal Imaging [Direct Diagnosis in Radiology]. Stuttgart, Germany: Georg Thieme Verlag; 2008: Fig. 2.16b. Reproduced with permission.)

(3) Treatment.
   (a) Reduction by posterior translation and mild extension.
   (b) Minerva or halo vest.
      i. Nonunion (rare).
         • Loss of continuity between the odontoid and body of C2 (os odontoideum).
      ii. Malunion (common).

d. Hangman's fracture—bilateral fracture of the C2 pedicles (traumatic spondylolisthesis of C2 on C3) **(Fig. 22.7)**.
   (1) Mechanism of injury:
      (a) Extension or distraction.
      (b) Commonly associated with facial abrasions or fractures.
   (2) The majority of patients remain neurologically intact.
   (3) Radiographs:
      (a) Fractures of the pedicles and displacement or angulation may be significant.
   (4) Treatment:
      (a) Closed reduction.
         i. Posterior translation and slight extension.
      (b) Minerva orthosis or halo vest.

e. Lower cervical spine injuries **(Fig. 22.8)**.
   (1) Bony injuries are less common in children under 10 years.
   (2) Cervical dislocations should be reduced as soon as possible.
   (3) Stabilization is performed using posterior spinous process wires and fusion using iliac crest bone graft.

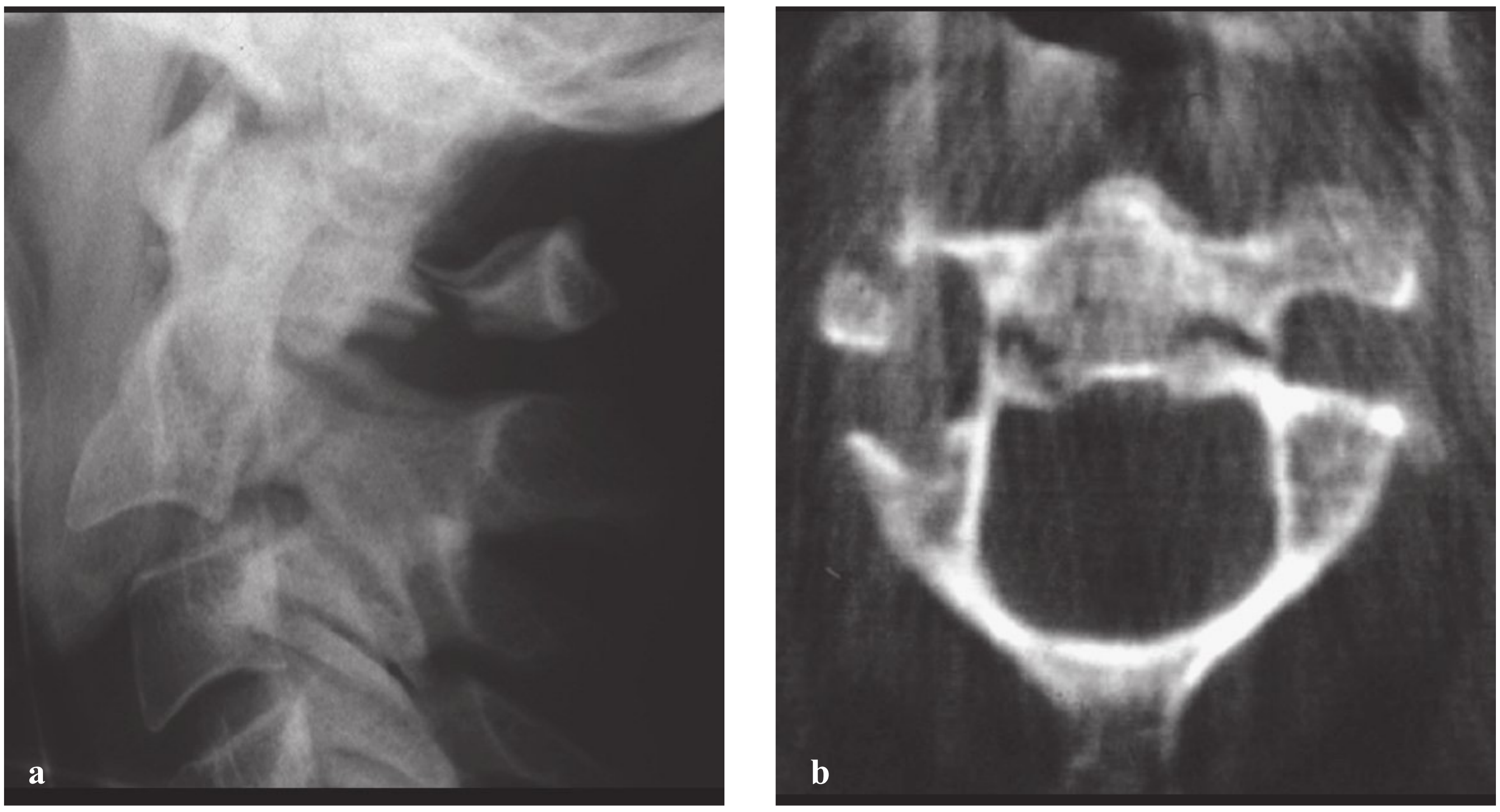

Fig. 22.7 Lateral radiograph of a hangman's fracture (bilateral C2 pedicle fracture). (From Imhof H, ed. Spinal Imaging [Direct Diagnosis in Radiology]. Stuttgart, Germany: Georg Thieme Verlag; 2008: Fig. 2.18. Reproduced with permission.)

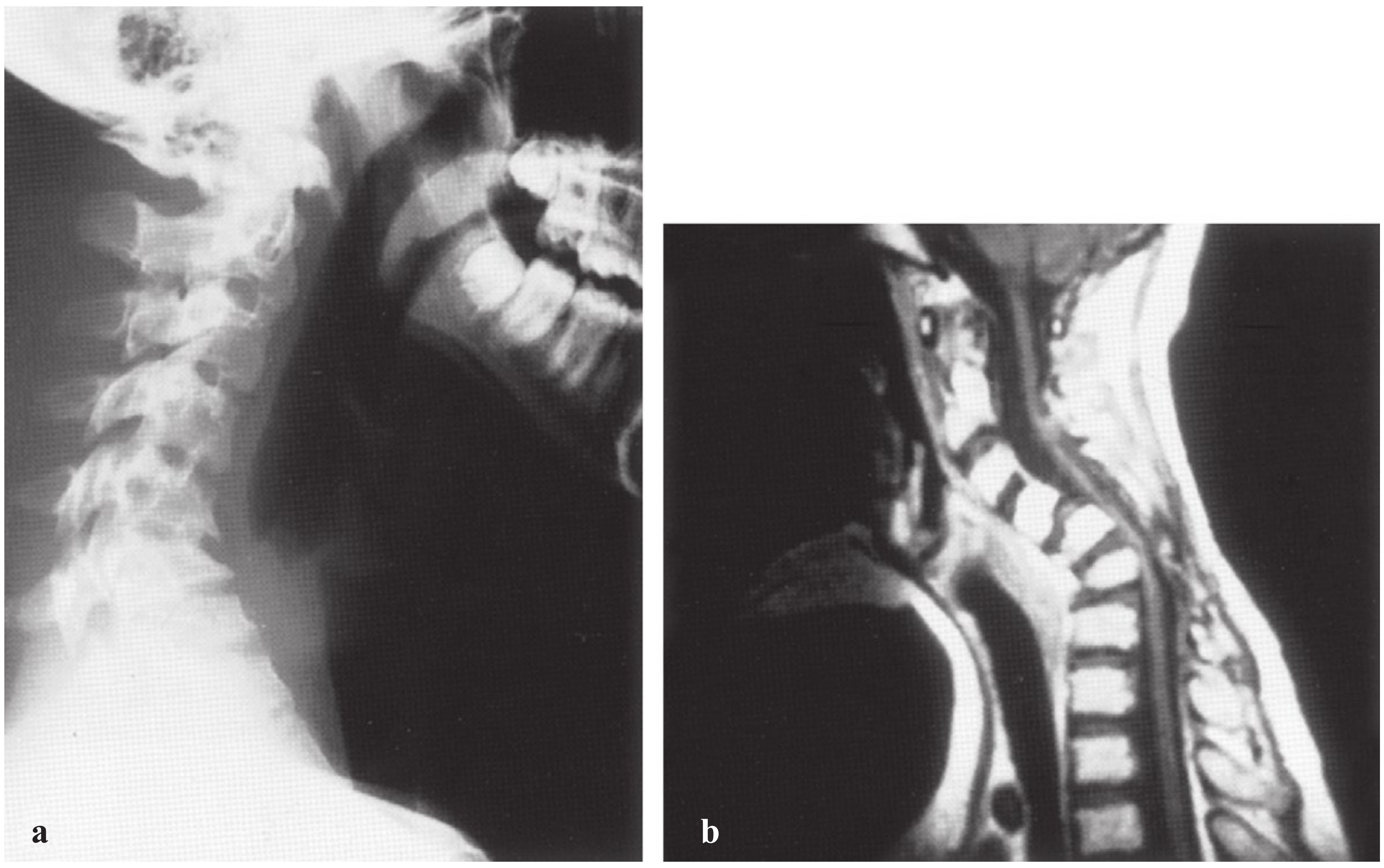

Fig. 22.8 Cervical kyphosis. **(a)** Lateral radiograph of a 17-year-old patient with cerebral palsy who presented with severe thoracic lordosis and cervical kyphosis with progressive myelopathy. This patient underwent anterior vertebrectomy and fusion and subsequent surgical procedures for correction of his thoracic lordosis. **(b)** Magnetic resonance imaging of a 14-year-old boy with postlaminectomy kyphosis and myelopathy. Anterior corpectomy and fusion were required for correction.

Suggested Reading

Brockmeyer DL, Ragel BT, Kestle JR. The pediatric cervical spine instability study. A pilot study assessing the prognostic value of four imaging modalities in clearing the cervical spine for children with severe traumatic injuries. Childs Nerv Syst 2012;28(5):699–705

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Martus JE, Griffith TE, Dear JC, Rathjen KE. Pediatric cervical kyphosis: a comparison of arthrodesis techniques. Spine 2011;36(17):E1145–E1153

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23 Spinal Tumors

23.1 General Considerations

I. Evaluation.
  A. History:
    1. Pain (localized vs. radicular) is the most common chief complaint (85% of patients).
      a. Other common presenting symptoms include motor weakness (41%) and a palpable mass (16%).
    2. Pain secondary to a spinal tumor is typically localized, progressive, unrelenting, nonmechanical, and worse at night.
      a. Patients do not have relief with rest.
    3. Systemic signs and symptoms:
      a. Fevers/chills.
      b. Lethargy.
      c. Unexplained weight loss.
    4. Neurological symptoms may be present, such as weakness, sensory changes, or bowel and bladder changes.
    5. Age may help to narrow the differential diagnosis.
      a. In older patients, metastasis and multiple myeloma are more common.
    6. A history of a primary tumor elsewhere in the body raises the concern of metastases.
      a. Metastatic tumors are more common than primary tumors in the spine.

Table 23.1 Common metastatic spine tumors

Primary tumor	Risk factors
Breast cancer	First-degree relative
	History of increased estrogen exposure (early menarche, late menopause, nulliparity, prolonged hormone replacement therapy)
	Radiation exposure
Prostate cancer	Increased age (> 45 years)
	Bladder outlet obstruction
Thyroid cancer	Iodine excess/deficiency
	Radiation exposure
Lung cancer	History of smoking
Renal cell carcinoma	Tobacco use

   b. The spine is the most common site of bone metastases.
   c. See **Table 23.1** for risk factors.

B. Physical examination of the spine should include palpation, range of motion, and neurological examination.
   1. Neurological examination.
      a. Detailed motor examination.
      b. Sensory examination.
         (1) Light touch.
         (2) Pinprick.
         (3) Vibration.
         (4) Assessment of long tract findings.
            (a) Reflexes.
   2. Physical examination of potential metastatic foci **(Table 23.2).**

C. Diagnostic studies:
   1. Helpful in differentiating tumor from infection:
      a. White blood cell count (WBC), erythrocyte sedimentation rate, and C-reactive protein should all be elevated with infection and normal or slightly elevated with tumor.
         (1) Exception is lymphoma, which is associated with an elevated WBC.
   2. Multiple myeloma is associated with protein spikes on serum or urine analysis.
   3. Thyroid-stimulating hormone and free T4 levels are useful in identifying thyroid disease.
   4. Prostate-specific antigen (PSA) is useful for prostate cancer.
   5. Calcium and phosphate are commonly associated electrolyte abnormalities that may need to be corrected.
   6. Radiological evaluation **(Table 23.3)**:
      a. Osteolytic lesions: breast (85%), renal cell, thyroid, and lung.
      b. Osteoblastic lesions: breast (15%) and prostate **(Fig. 23.1).**

Table 23.2 Classic physical exam findings in metastatic spine tumors

Primary tumor	**Physical exam f ndings**
Breast cancer	Hard, fixed, nontender breast mass
	Nipple retraction
	Skin erythema or edema
Prostate cancer	Large, hard, nodular prostate on digital rectal examination
Thyroid cancer	Painless, palpable thyroid
Lung cancer	Baseline change in cough
	Hemoptysis
Renal cell carcinoma	Classic triad of hematuria, flank pain, and abdominal mass
	Tobacco use

Table 23.3 Diagnostic imaging of spine tumors

Imaging study	Advantages	Disadvantages
Plain radiography	Simple screening method Helpful in diagnosis (benign vs. malignant, osteolytic vs. osteoblastic)	Low sensitivity (> 50% of cancellous bone loss is needed for radiographic identification of bone destruction) Winking owl sign
Bone scan	Most sensitive tool for metastases (osteoblastic lesions)	Low specificity (cannot differentiate fracture, infection, and neoplasm)
Computed tomography	Best for evaluating bone destruction Important for preoperative planning	Ineffective as a screening tool
Magnetic resonance imaging	High sensitivity, especially when used with gadolinium Provides information about soft tissue component Helpful in evaluating spinal cord compression	Extent of cord compression does not consistently correlate with symptoms or outcome
Myelography	Good visualization of epidural metastasis and cord compression	Invasive
Angiography	Selective embolization of the neoplasm may decrease bleeding during surgery	Invasive

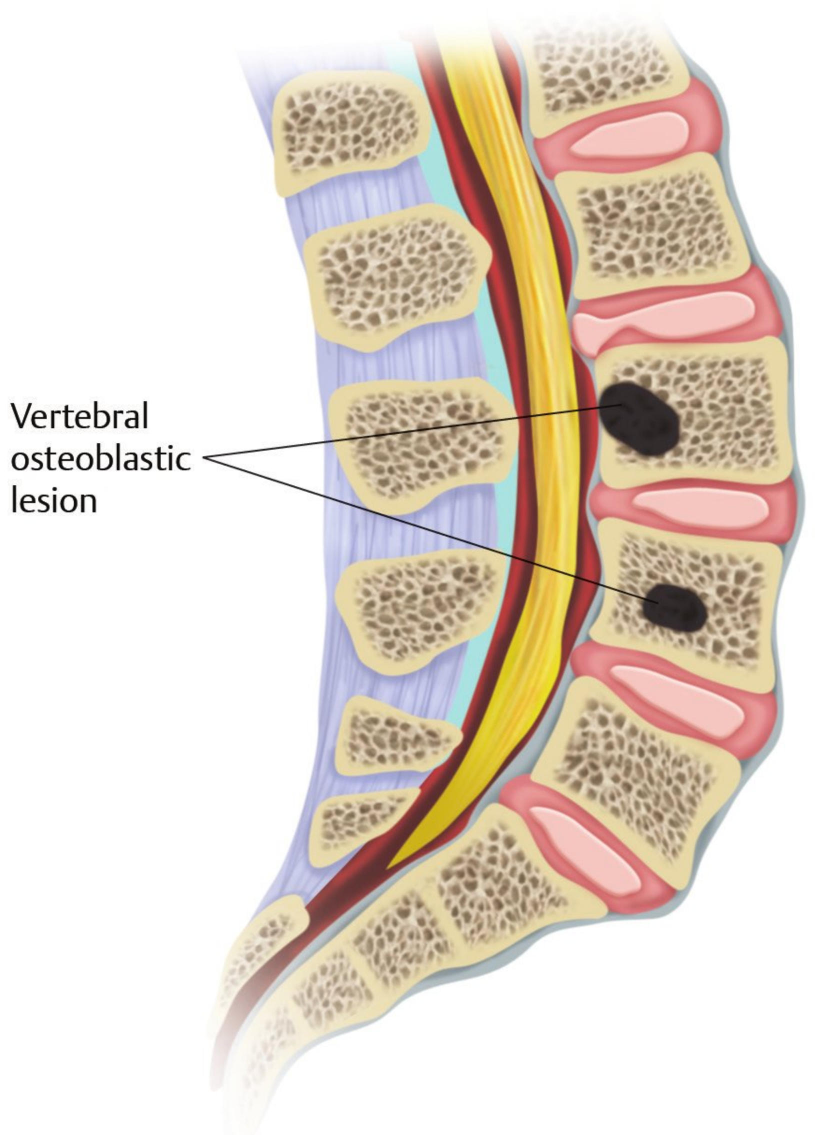

Fig. 23.1 Osteoblastic lesions in the L3 and L4 vertebral bodies.

Table 23.4 Magnetic resonance imaging findings in spinal infection versus tumors versus compression fractures

Diagnosis	T1	T2	Differentiating f ndings
Vertebral osteomyelitis	– Decreased signal within disk and end plates – Loss of end plate definition	– Increased signal within disk and end plates – Loss of end plate definition	– Disk/end plate involvement > vertebral body – Hyperintense abscesses on T2 – Tuberculous spondylitis does not involve contiguous levels – Soft tissue mass is poorly defined
Osteoporotic compression fracture	– Decreased signal in the involved body – Incomplete marrow replacement	– Increased signal in the body – Incomplete marrow replacement	– Returns to isointensity on T1 and T2 – Marrow preservation in the posterior third of the body
Neoplastic disease	– Decreased signal – Defined area of infiltrative edema – Pedicle involvement	– Increased signal – Defined area of infiltrative edema – Pedicle involvement	– No disk or cartilaginous involvement – Noncontiguous involvement is frequent – No restoration of normal signal intensity as in fracture – Soft tissue masses are eccentric, large, and well defined

7. Magnetic resonance imaging (MRI) differentiation of infection, fracture, and tumor **(Table 23.4).**

D. Primary tumor types:
   1. See **Table 23.5, Table 23.6, Table 23.7,** and **Table 23.8.**

E. Staging:
   1. Weinstein–Boriani–Biagini system **(Fig. 23.2) (Table 23.9).**
      a. Three-dimensional description of tumor invasion.

F. Treatment:
   1. Goals:
      a. Establishment of a definitive diagnosis.
      b. Maintenance of neurological function.
      c. Restoration of spinal stability.
      d. Pain relief.
      e. Control of local tumor and prevention of metastases.

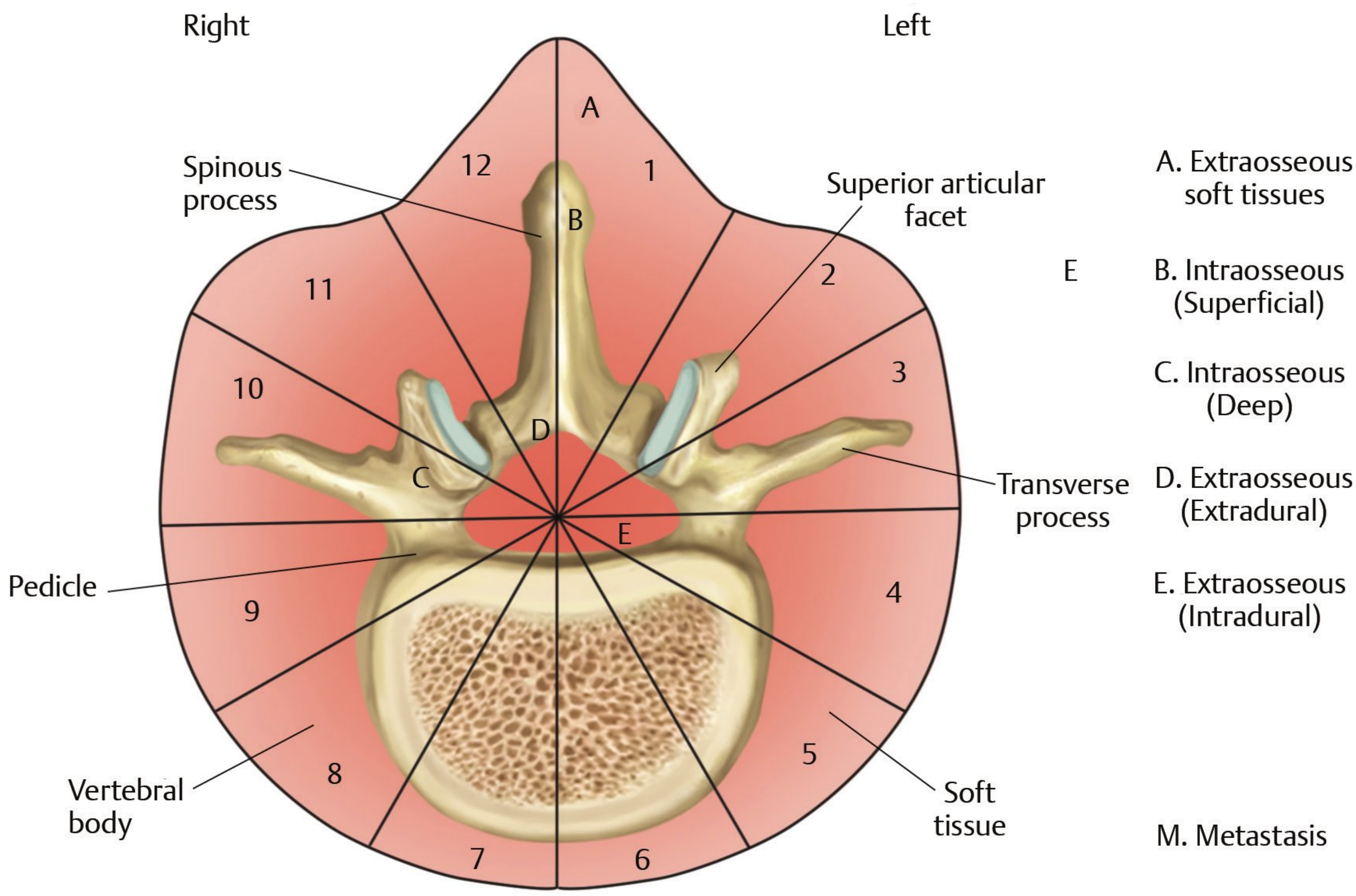

Fig. 23.2 The Weinstein–Boriani–Biagini system for spinal tumor staging.

Table 23.9 Weinstein–Boriani–Biagini staging system

Type	Notes
Anatomical zones	Twelve pie-like zones starting at the spinous process and rotating clockwise
Involvement of different vertebral layers	Extraosseous soft tissue Intraosseous (superficial) Intraosseous (deep) Extraosseous (extradural) Extraosseous (intradural)
Specification of the spinal segment(s) involved	

2. Treatment is dictated by diagnosis, location of tumor, and general health of the patient.
3. Radiation versus surgery:
   a. In 2005, Patchell et al reported the results of a multicenter, randomized, controlled trial that compared the outcomes associated with surgery plus radiation versus radiation alone for patients with nerve compression from metastatic cancer to the spine.

(1) Advantages of surgery plus postoperative radiation versus radiation alone.
   (a) A greater number of patients were able to walk after treatment (84% vs 57%).
   (b) Longer maintenance of continence.
   (c) Greater muscle strength, functional ability, and increased survival.
   (d) Decreased requirement of corticosteroid and opioid medication after treatment.
   (e) The study was terminated early due to the significant advantage of the surgical treatment over radiation alone.

4. Radiation therapy is recommended for the following patients:
   a. Cord compression caused by a soft tissue tumor or without compromise of the surrounding bony architecture.
   b. Radioresponsive tumors:
      (1) Hematopoietic.
      (2) Prostate.
      (3) Breast.
   c. Decompression with concomitant radiation therapy is associated with superior outcomes when compared with radiotherapy alone for patients with metastatic cancer causing spinal cord compression.
   d. Spinal radiation prior to surgical intervention is associated with greater rates of wound complications (dehiscence, infection, revision) and adverse surgical outcomes.
5. Surgery:
   a. Indications.
      (1) Diagnostic evaluation.
      (2) Curative excision (benign tumors and certain malignant tumors).
      (3) Spinal instability or deformity secondary to neoplastic bone destruction.
      (4) Neurological deterioration.
      (5) Failure of previous radiation therapy.
      (6) Radiation-resistant tumors.
      (7) Unremitting pain.
   b. Surgical stratification.
      (1) Diagnosis of tumor.
         (a) Benign versus malignant **(Fig. 23.3** and **Fig. 23.4).**
         (b) Primary versus metastatic.
      (2) Stage.
         (a) Degree of spinal involvement.
         (b) Potential metastatic spread.
      (3) Neurological status.
         (a) Primary indicator of postsurgical outcome.
            i. Rapid progression of symptoms (< 1 week) is a poor prognostic indicator.
            ii. Patients with severe deficits (inability to walk, loss of bowel/bladder function) are less likely to recover.

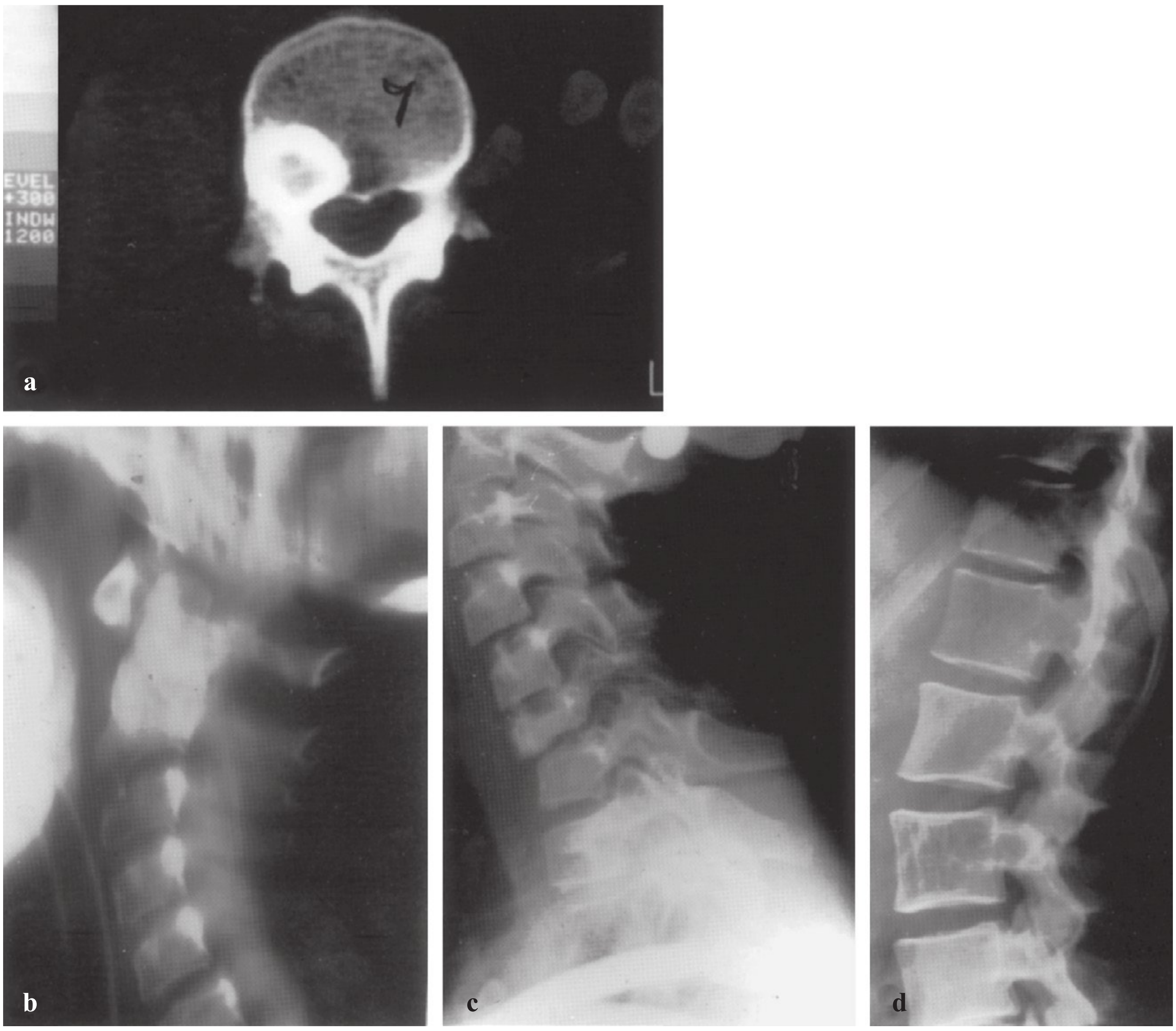

Fig. 23.3 Examples of primary benign tumors of the spine. **(a)** Computed tomography (CT) scan demonstrating an osteoid osteoma with a central nidus and sclerotic rim at the posterior part of the vertebral body. **(b)** Lateral radiograph demonstrating an osteoblastoma of C2 with an expansile sclerotic bone. **(c)** Lateral radiographs demonstrating vertebra plana at C6 due to eosinophilic granuloma. **(d)** Lateral lumbar radiograph showing a hemangioma with osteopenia and vertical striations of the vertebral body.

(4) Prognosis.
(5) Structural stability **(Fig. 23.5).**
(6) Pain status.

c. Surgical approach.

(1) Excise the entire lesion if possible.
  (a) Total en bloc spondylectomy **(Fig. 23.6** and **Fig. 23.7).**
    i. Accomplished through a posterior approach.
    ii. Particularly useful if excising the lesion is curative.
      • Chondrosarcoma.
(2) Approach anteriorly or posteriorly or both depending on the location of the tumor.
  (a) A decompressive laminectomy does *not* address anterior pathology and predisposes patients to postoperative instability.

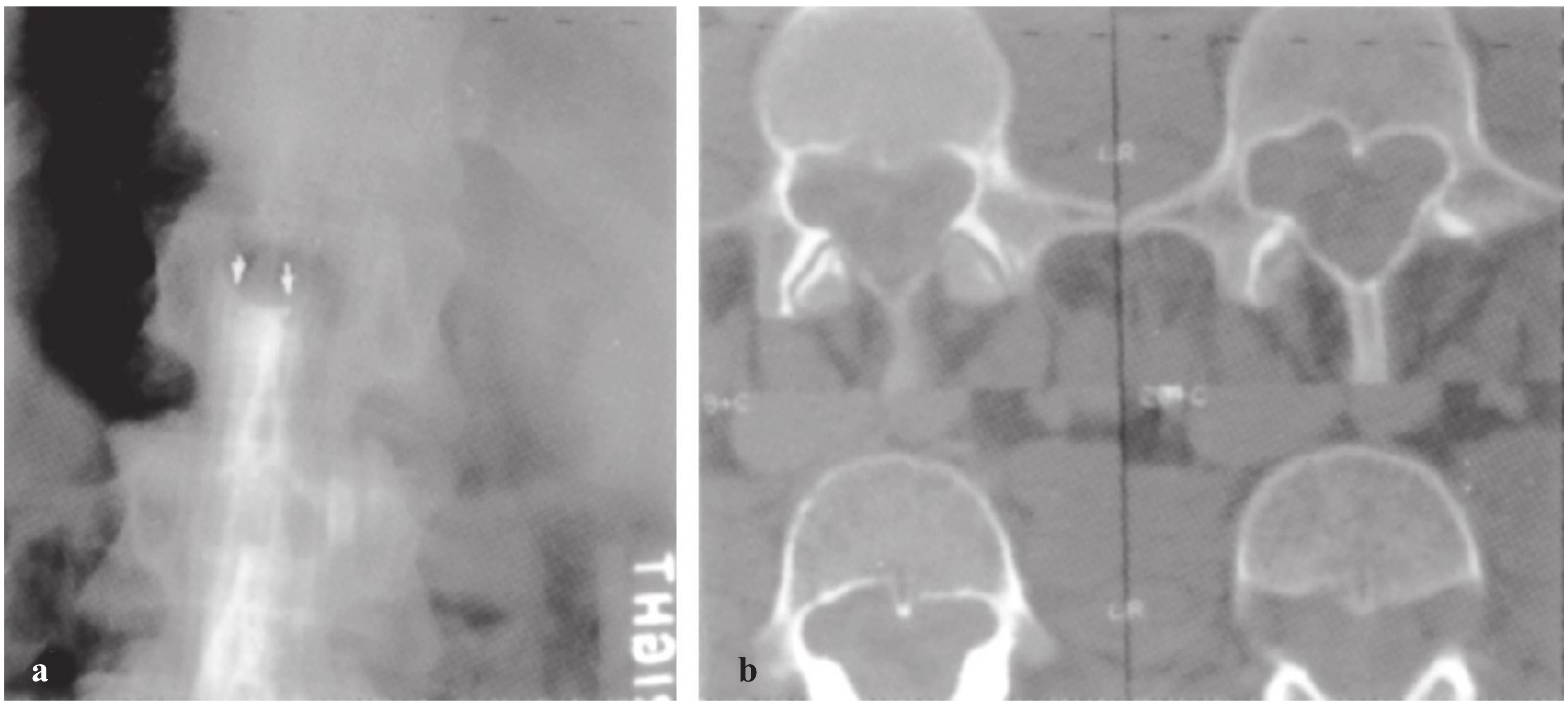

Fig. 23.4 Intradural neurofibroma. **(a)** Anteroposterior myelography showing a myelographic block at L1 due to a neurofibroma. The myelographic block is smooth and meniscal in shape (arrows) due to the intradural adhesions, whereas extradural lesions produce a ragged margin. **(b)** Computed tomographic scans showing erosion of the vertebral body and pedicle due to expansion of intradural neurofibromas.

(3) Metastatic tumors are usually approached anteriorly if the spinal cord compression is anterior.
   (a) Reconstruction can be performed with autograft, allograft, methyl methacrylate cement, or synthetic materials.
      i. Autograft/allograft allows potential biologic incorporation.
      ii. Methyl methacrylate offers instantaneous stability but may fail in patients whose expected life span is prolonged (> 1 year).
      iii. Patients who receive postoperative irradiation have decreased chances of achieving biological fusion.

d. Stereotactic radiosurgery (SRS).
   (1) Delivers very high doses of radiation to an ultraspecified locus of tissue in an effort to minimize damage to the surrounding structures.
   (2) Coupled with robotic navigation to guide the trajectory of the radiation beam in six dimensions.
   (3) Advancements in SRS, including intensity-modulated radiotherapy, have further amplified the precision and accuracy of radiotherapy to limit damage to the spinal cord.
      (a) This high level of precision allows for multiple treatments, if necessary.
   (4) Can be used in conjunction with surgical decompression.

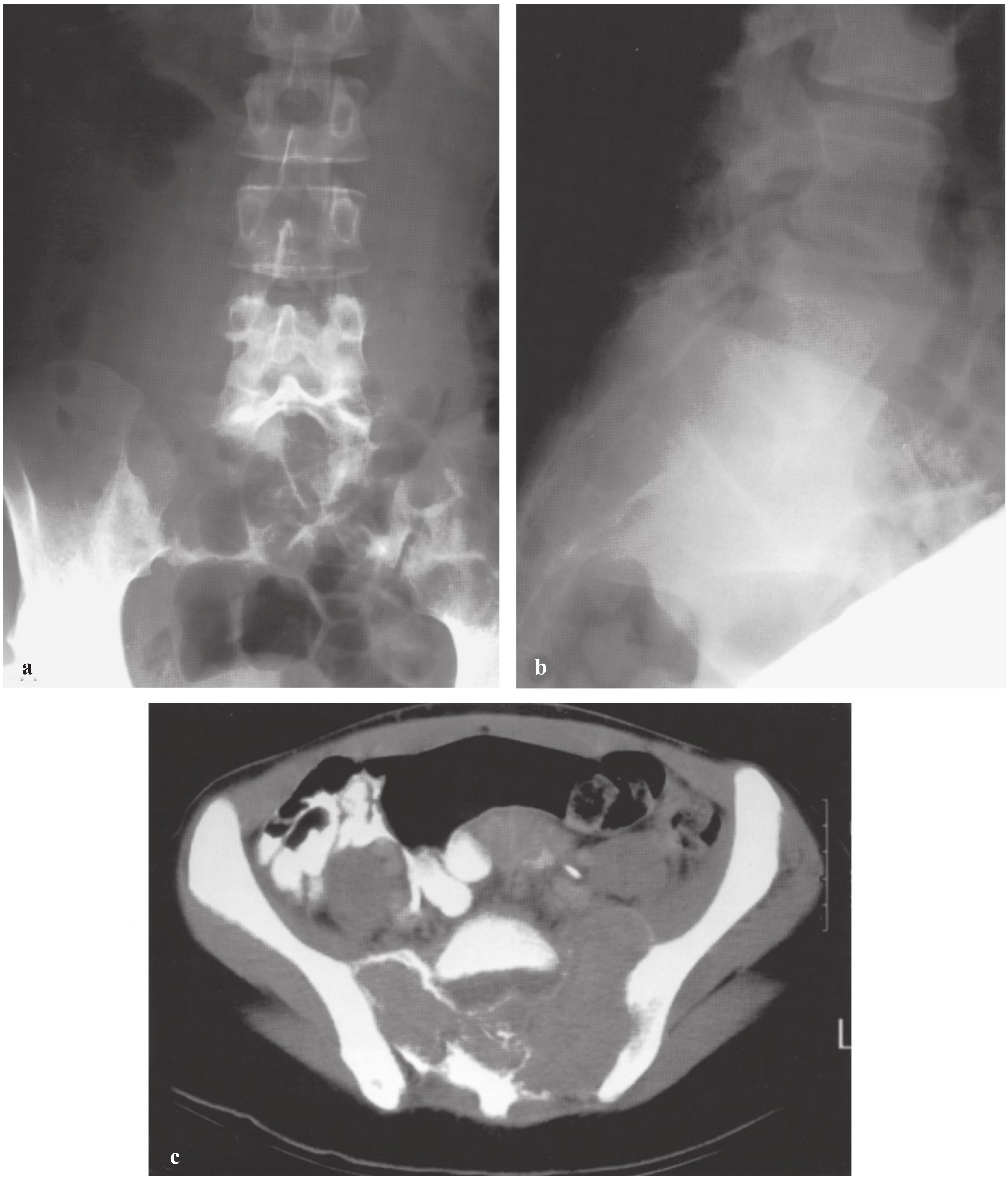

Fig. 23.5 Images of a 17-year-old girl with giant cell tumor involving the sacrum. **(a)** Anteroposterior view of the spine and pelvis showing a destructive lesion involving the sacrum and left sacroiliac joint. **(b)** Lateral radiograph showing a destructive lesion at S1–S2. Note that the sacrum is not well demarcated. **(c)** Computed tomographic scan showing the extent of the tumor with involvement of the left sacroiliac joint.

Fig. 23.5 (Continued) Images of a 17-year-old girl with giant cell tumor involving the sacrum. **(d)** T2-weighted sagittal magnetic resonance image showing a large tumor extension into the pelvis anteriorly and into the spinal canal posteriorly. **(e,f)** A posterior approach was used to perform a laminectomy of L5 and the sacrum with excision of the tumor. Reconstruction was performed with a transiliac fibular graft, lumbar pedicle screws, bilateral iliac screws, and rod fixation. The patient was mobilized immediately, and healing was evident without recurrence at follow-up.

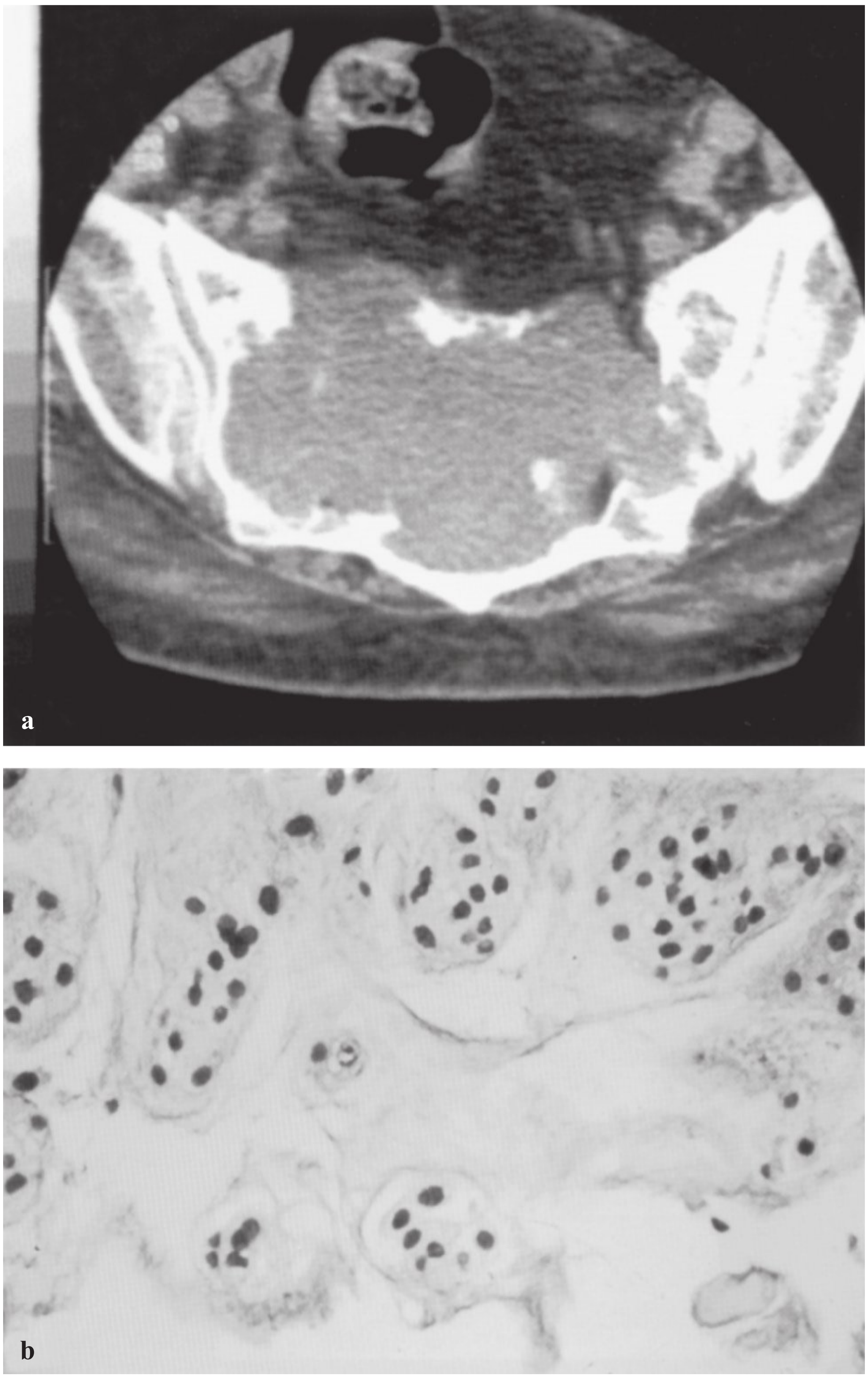

Fig. 23.6 (**a**) Axial computed tomographic image of a chondrosarcoma in the sacrum. (**b**) Microscopic image of chondrosarcoma cells.

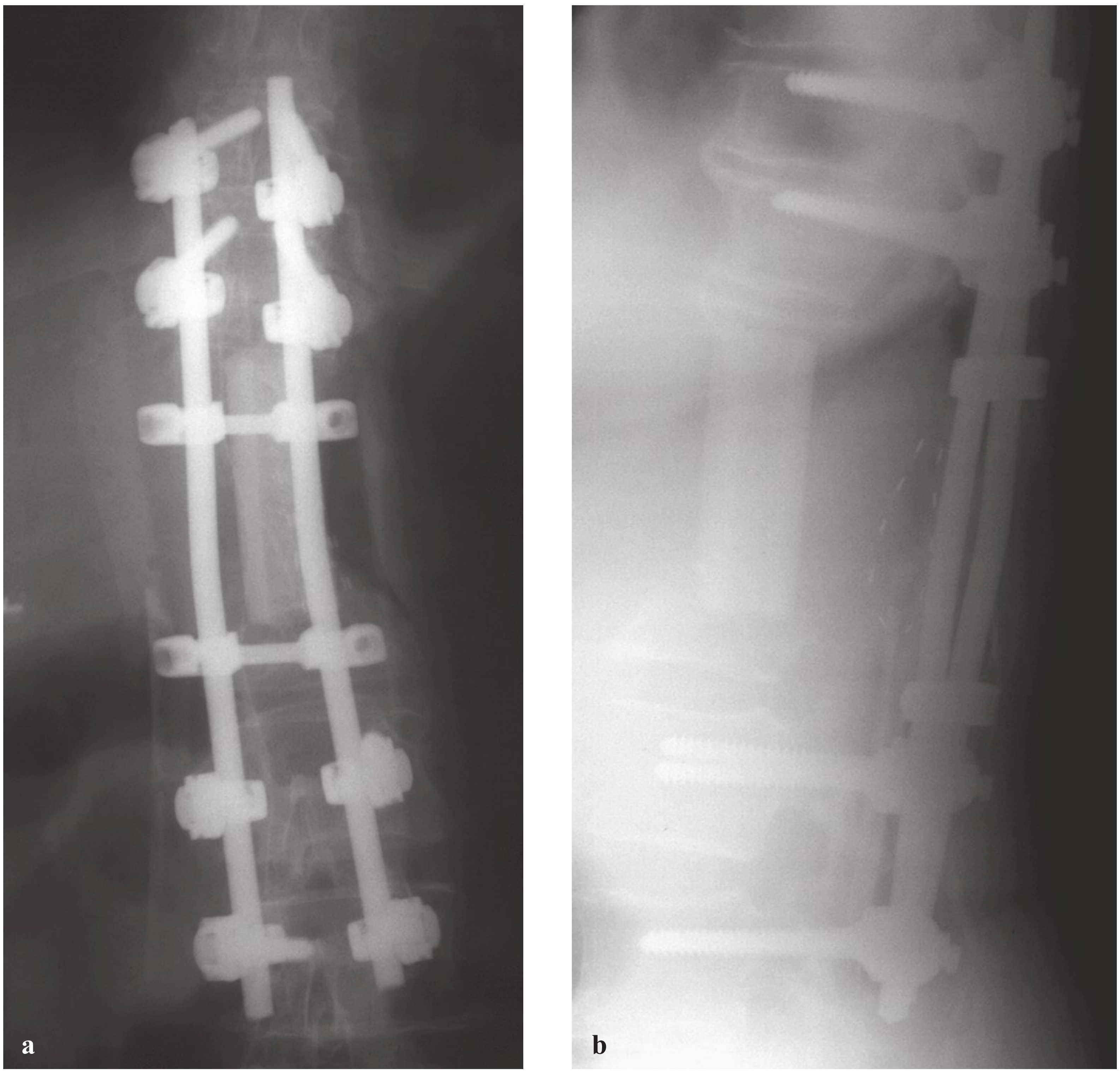

Fig. 23.7 **(a,b)** Anteroposterior and lateral radiographs following total spondylectomy for a T11 chordoma with spinal instrumentation and graft in position. (From Dickman CA, Fehlings MG, Gokaslan ZL. Spinal Cord and Spinal Column Tumors. New York, NY: Thieme Medical Publishers; 2006: Fig. 34.14. Reproduced with permission.)

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24 Spinal Infections

24.1 General Considerations

I. Vertebral osteomyelitis.
  A. Incidence/risk factors.
    1. Approximately 2 to 7% of all osteomyelitis (1–2% in children).
    2. Lumbar > thoracic > cervical.
    3. Males > females (2:1).
    4. More common after the fifth decade of life (> 50% of cases).
    5. Risk factors include diabetes, malnutrition, perioperative hyperglycemia, obesity, smoking, immunocompromise (steroids, HIV/AIDS), previous surgery.
  B. Etiology.
    1. Hematogenous spread is the most common route for vertebral osteomyelitis.
      a. Urinary tract is the most common source (e.g., urinary tract infections, transient bacteremia from genitourinary procedures).
      b. Soft tissue infections.
      c. Respiratory infections.
    2. Unidentified source.
    3. Direct inoculation (e.g., penetrating trauma, invasive spinal procedure).
    4. Causative bacteria (in order of frequency):
      a. Gram-positive aerobic cocci (> 80%).
        (1) *Staphylococcus aureus* (> 50%).
          (a) Methicillin-resistant *S. aureus* (7%).
        (2) *Streptococcus* (10–20%).
        (3) Coagulase-negative *Staphylococcus* (10%).
        (4) *Propionibacterium acnes* (delayed infection).
      b. Gram-negative aerobic cocci (15–20%).
        (1) Most common origin is from the urinary tract (*Escherichia coli, Pseudomonas aeruginosa, Proteus*).
      c. Gastrointestinal tract organisms.
        (1) *Salmonella* (rare).
          (a) More common in patients with sickle cell.
      d. Granulomatous infections (far less common).
        (1) *Mycobacterium tuberculosis*, fungi, spirochetes.
        (2) More common in the thoracic region.
  C. Pathology.
    1. Inoculation.
      a. Hematogenous spread to the vertebral metaphysis most likely occurs via rich arterial anastomosis (nutrient artery) **(Fig. 24.1)**.
        (1) Batson's valveless venous plexus is not considered to play a significant role in bacterial hematogenous seeding.

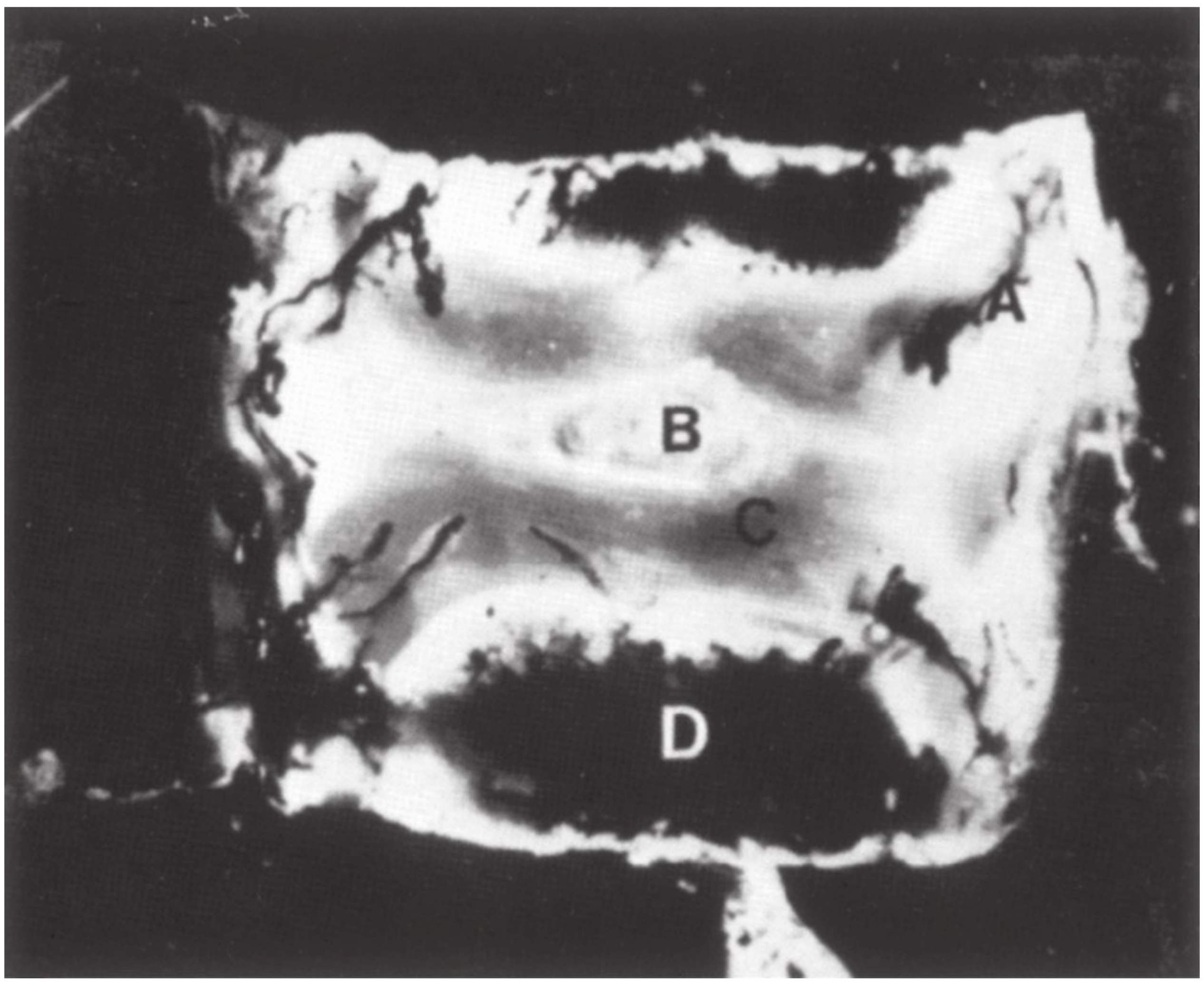

Fig. 24.1 Sagittally sectioned human fetal specimen (26 weeks gestation), injected, cleared, and transilluminated, showing cartilage canals and absence of vessels in nucleus pulposus. (A) Cartilage canal; (B) nucleus pulposus; (C) hyaline cartilage; (D) ossified vertebral body.

  (2) Vertebral metaphysis is a low-flow environment that may allow for the direct spread of bacteria into and across the intervertebral disk.

2. Spread to the intervertebral disks.
   a. Bone/disk destruction **(Fig. 24.2).**
      (1) Bacteria produce enzymes that digest disk tissue.
      (2) Bone resorption by osteoclasts is activated by various inflammatory mediators.
3. Soft tissue extension.
   a. Psoas abscess.
   b. Paraspinal muscle abscess.
   c. Epidural abscess.
      (1) May result in neurological compromise secondary to direct compression of the spinal cord and nerve roots.

D. Clinical findings.
1. Delay in diagnosis is common.
2. Back or neck pain is the most common presenting complaint (90%).
   a. Symptoms are typically present for more than 3 months in 50% of patients.
   b. Acute presentation with septicemia and toxemia is extremely rare.
3. Localized pain and tenderness with a decreased range of motion are the most consistent findings.
4. History of fever > 100°F (with or without chills) is present in over 50% of patients.
5. In children, a limp and refusal to walk are characteristically present.

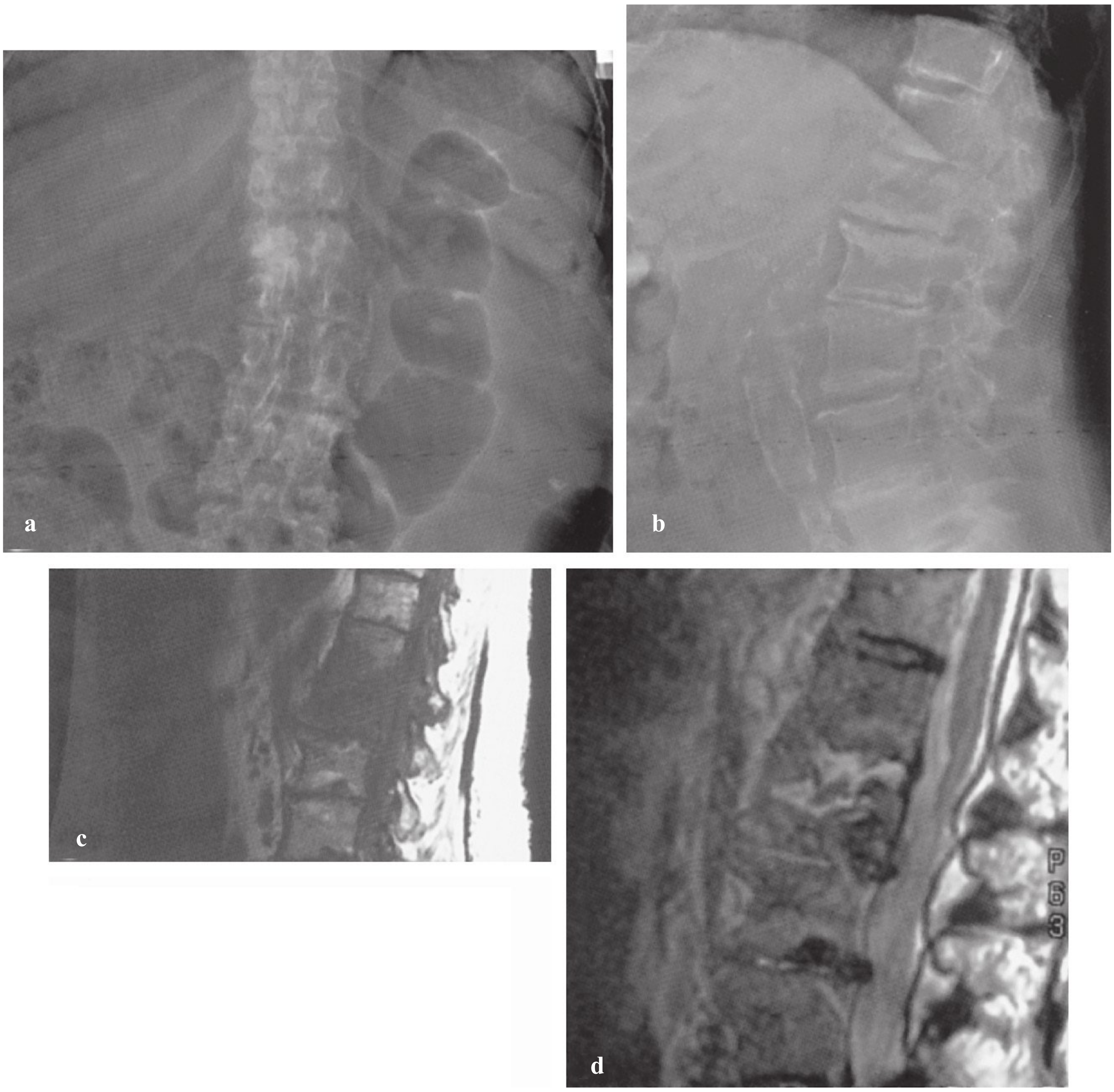

Fig. 24.2 A 76-year-old woman with rheumatoid arthritis and a T12–L1 diskitis/L1 osteomyelitis. Results of three needle biopsies were negative. **(a,b)** Anteroposterior and lateral radiographs demonstrated a diskitis at T12–L1 with destruction of the L1 vertebral body. **(c)** T1-weighted sagittal magnetic resonance imaging (MRI) sequence shows decreased signal throughout and across the T12–L1 disk space. The end plates are blurred and indistinct. **(d)** T2-weighted sagittal MRI sequence shows high signal within the T12–L1 disk and the L1 vertebral body.

E. Laboratory findings **(Table 24.1).**

F. Radiographic imaging studies **(Table 24.2).**

G. Treatment.

1. Goals.
   a. Establish a tissue diagnosis and identify the organism.
   b. Eradicate the infection.
   c. Provide long-term pain relief.
   d. Prevent or relieve any neurological deficits.
   e. Restore spinal stability/alignment.

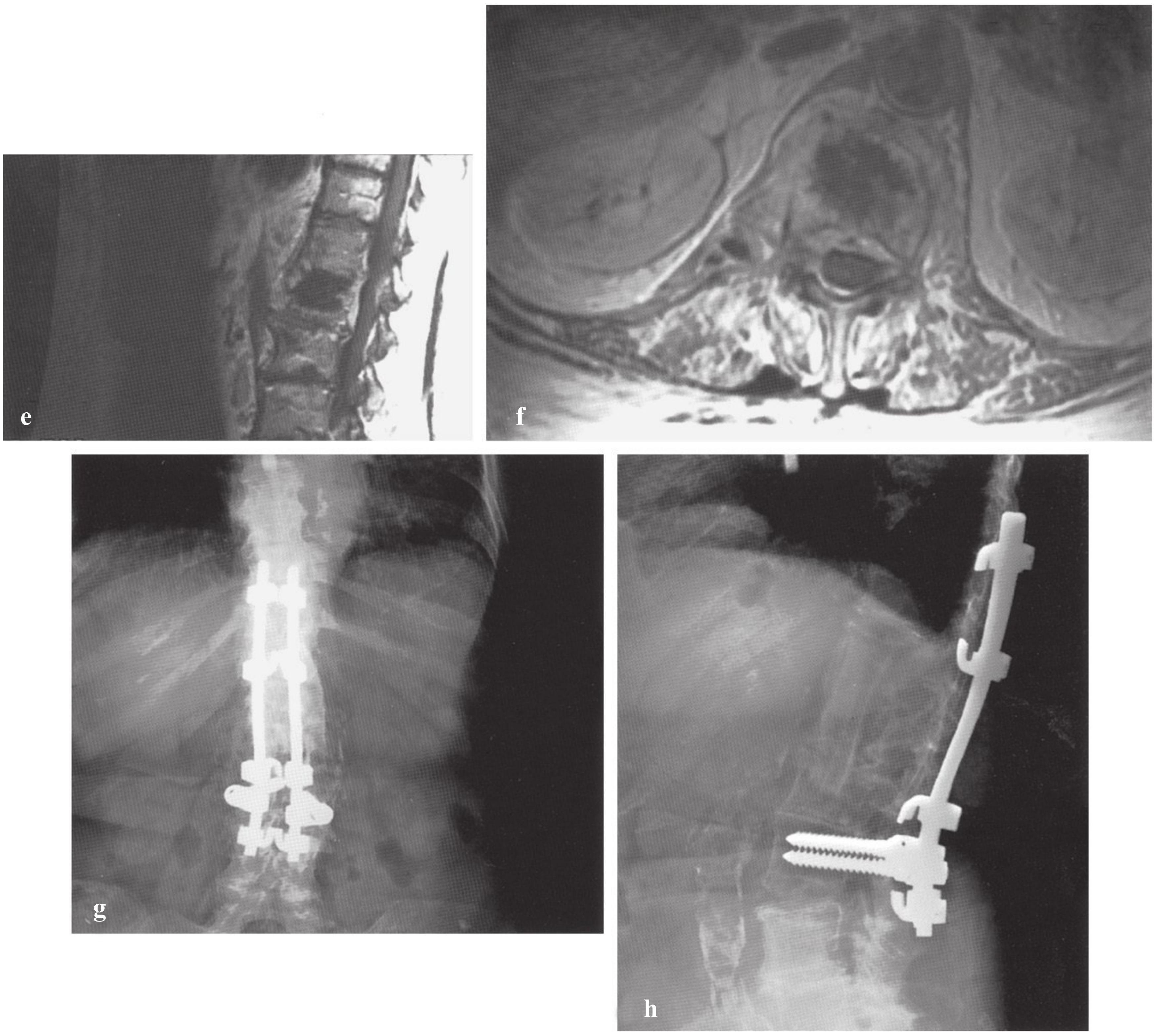

Fig. 24.2 (Continued) A 76-year-old woman with rheumatoid arthritis and a T12–L1 diskitis/L1 osteomyelitis. Results of three needle biopsies were negative. **(e)** T1-weighted sagittal MRI sequence with gadolinium shows enhancement of the T12–L1 disk space and L1 vertebral body. There is a slight amount of enhancing tissue in the anterior epidural space without compression of the conus medullaris or cauda equina. **(f)** T1-weighted axial MRI sequence with gadolinium shows enhancement of the T12–L1 disk space. **(g,h)** Anteroposterior and lateral radiographs taken 6 months after surgery demonstrating incorporation of the bone graft anteriorly with solid fixation posteriorly.

2. Principles.
   a. Medically optimize the patient.
      (1) Nutritional supplementation.
      (2) Correct any laboratory abnormalities.
   b. Treat extraspinal sources of infection.
      (1) Urinary tract.
      (2) Cardiovascular (infected thrombus).
      (3) Gastrointestinal.
   c. Broad-spectrum antibiotics should be started, and then antibiotic therapy should be specific to the organism identified.

Table 24.1 Laboratory markers in spinal infections

Test	Findings
ESR	Elevated at presentation in more than 80% of cases
	ESR normalizes in two-thirds of patients adequately treated
WBC	> 10,000/mm^3 in more than 50% of cases
	WBC count has a low sensitivity for diagnosis
CRP	More sensitive and specific than ESR for monitoring postoperative spine infections
Blood cultures	Most useful in children with vertebral pyogenic osteomyelitis
	Only positive in ~ 35% of patients
	Reliable in detecting the offending organism
Needle biopsy	False-negative examinations are common when patient is on antibiotics
Open biopsy	Indicated if needle biopsy is negative, nondiagnostic, or both despite high clinical suspicion
	Lower false-negative rate than closed biopsy

Abbreviations: CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; WBC, white blood cell count.

d. Erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) levels are useful to obtain prior to therapy.
   (1) May be followed as an indication of treatment efficacy.

3. Operative treatment.
   a. Indications.
      (1) Cases that have failed nonoperative management.
      (2) Progressive neurological deficit.
         (a) Due to direct compression from the infection.
         (b) Due to progressive deformity or instability.
      (3) Abscess or granuloma formation.
         (a) Antibiotics are ineffective.
      (4) Intractable pain not responsive to conservative measures.
   b. Technique **(Fig. 24.3).**
      (1) Anterior approach is the most useful for vertebral body debridement (corpectomy).
         (a) Laminectomy alone for decompression is contraindicated because of the potential for spinal destabilization.

Table 24.2 Diagnostic imaging in spinal infections

Imaging study	Findings
Plain radiographs	– Findings lag behind clinical presentation (at least 2 weeks from the onset of infection) – Disk space narrowing with erosive changes (75%) – Osteolysis, diffuse osteopenia, focal defect – 50% trabecular bone destruction before radiographic evidence is noted – Osteosclerosis (11%) – Chronic cases may reveal spontaneous bone fusion (50%)
Nuclear imaging	– Effective as an initial screening tool – Earlier detection and localization when compared with plain films – Combination of gallium (inflammatory) and technetium (bone) scans provides > 90% accuracy in diagnosis – Indium-111-labeled leukocyte (white blood cell) scans are not sensitive in the spine – High false-negative rate may be related to leukopenia
Computed tomography	– Best modality for identifying bone destruction
Magnetic resonance imaging	– Imaging modality of choice for spine infections – T1-weighted images—decreased signal around adjacent – T2-weighted images—high signal intensity in bodies near adjacent end plates and disk space end plates and disk space – Loss of end plate definition – Involved portions of disks and vertebral bodies enhance with gadolinium – Allows for visualization of soft tissue involvement (paraspinal, psoas abscess) – Best imaging modality to differentiate infection versus tumor

(2) Autogenous bone graft is the gold standard for reconstruction (iliac crest, rib, or fibula).
   (a) However, autograft-filled titanium cages and cortical strut allograft have demonstrated good clinical results.
   (b) Titanium alloys have demonstrated lower bacterial adhesion than stainless steel alloys and are now commonly used in the setting of anterior corpectomies.
   (c) More recently, polyetheretherketone (PEEK) interbody devices (expandable) have been successfully used in the setting of a corpectomy for a spinal infection.

(3) Thoracic and lumbar vertebral osteomyelitis may be treated by a single posterior approach (debridement and fixation) using an interbody technique.

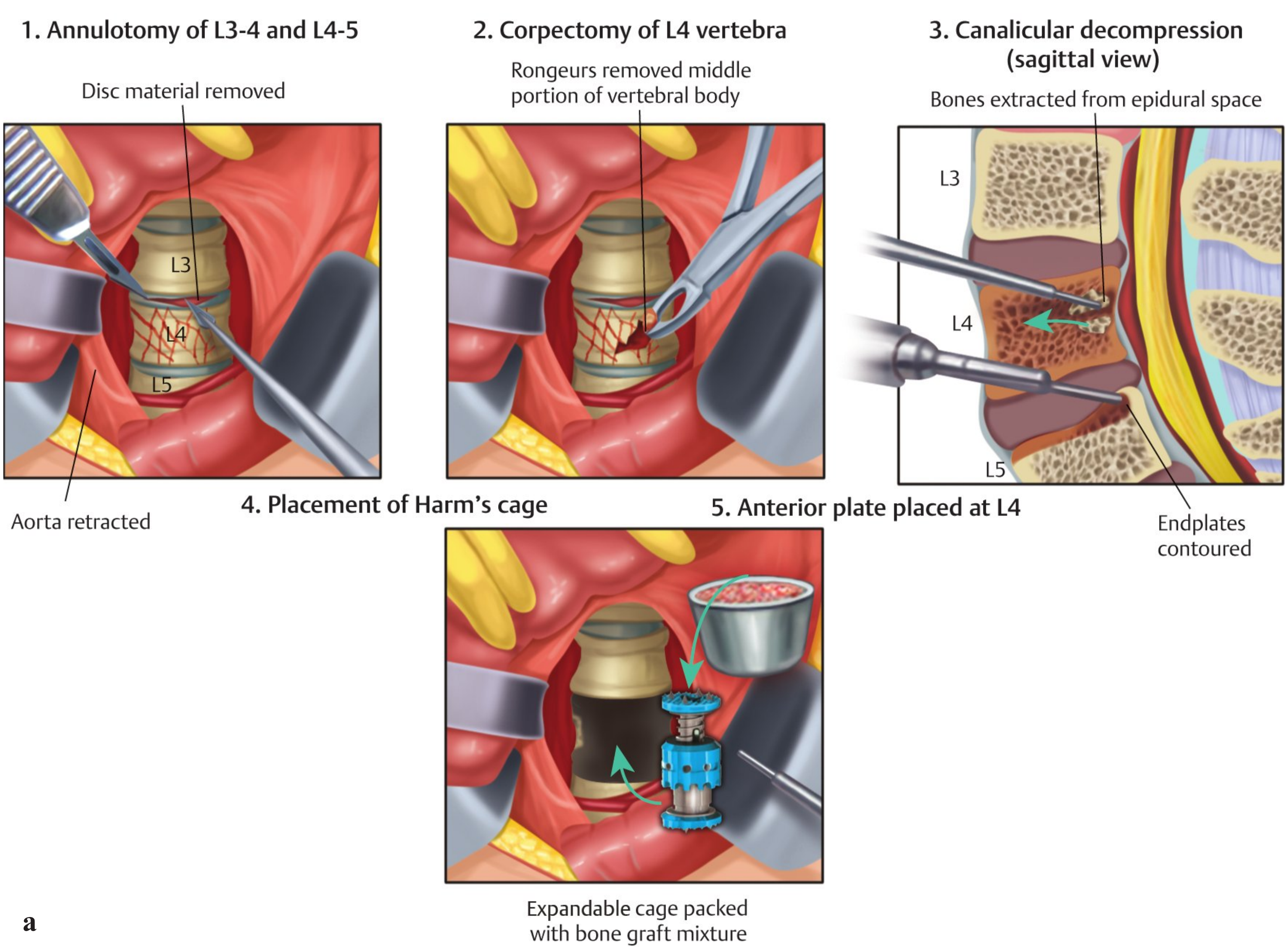

Fig. 24.3 **(a)** A corpectomy procedure.

II. Epidural abscess.
 A. Etiology.
  1. Associated with vertebral pyogenic osteomyelitis in 28% of cases.
  2. *S. aureus* most common causative organism (~ 60%).
  3. Regional or location frequencies:
   a. Thoracic (50%).
    (1) Neurological deficits are more common.
   b. Lumbar (35%).
   c. Cervical (14%).
  4. Most cases are in adults (rare in children).
   a. Postoperative (16%).
 B. Clinical presentation.
  1. Highly variable, leading to misdiagnosis and delayed treatment in > 50% of patients.
  2. Localized spine tenderness is more common.
  3. Nuchal rigidity and other meningeal signs are possible.
  4. With or without neurological deficit.
 C. Diagnosis.
  1. ESR is elevated in > 98% of cases.
  2. White blood cell count (WBC) is unreliable.

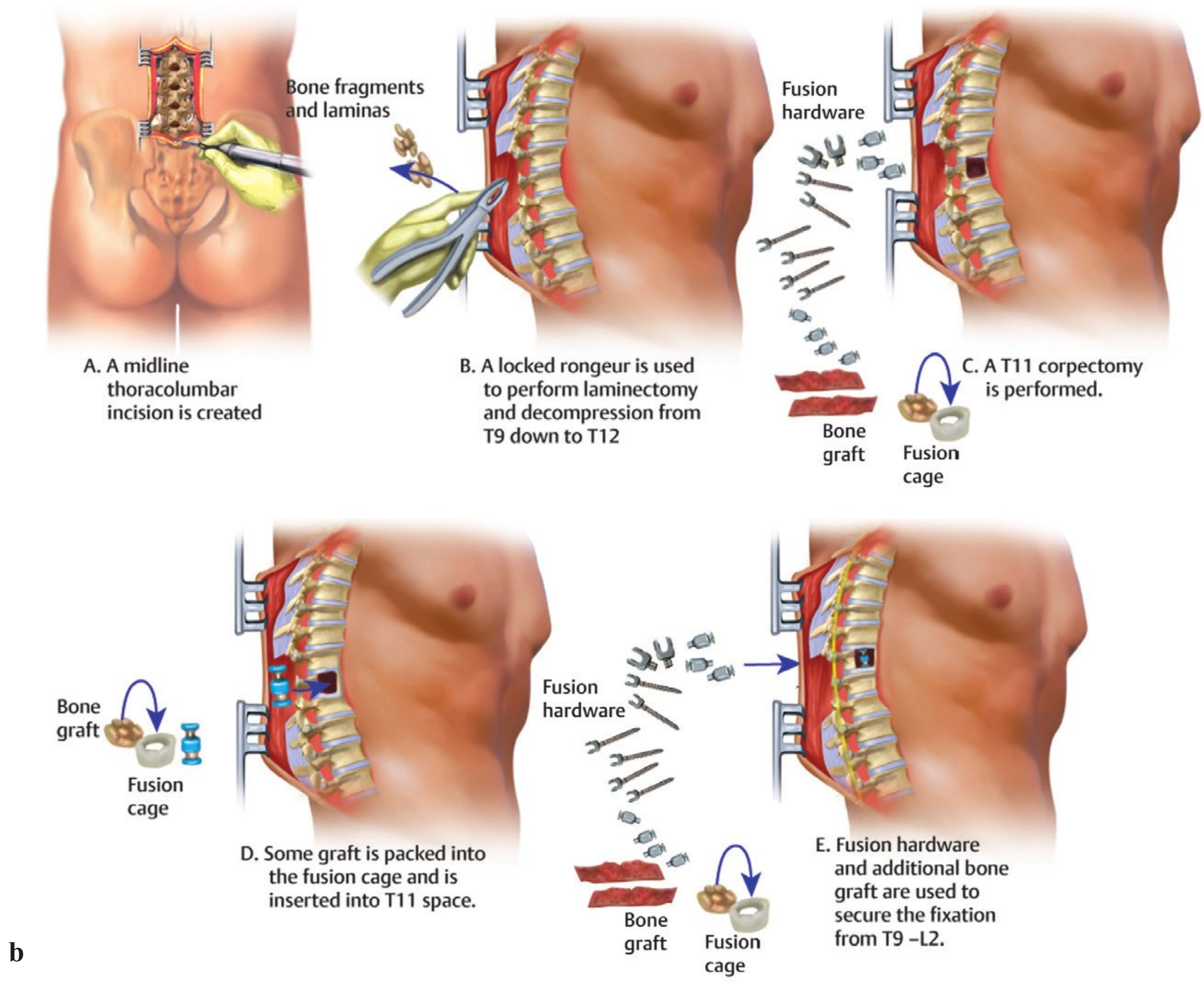

Fig. 24.3 (Continued) **(b)** Incorporation of an expandable cage.

3. Magnetic resonance imaging (MRI) is the imaging modality of choice **(Fig. 24.4** and **Fig. 24.5)**.
   a. Intense focal signal on T2.
   b. Epidural metastasis and subdural abscess should be considered in the differential.

D. Treatment.
   1. Epidural abscess requires urgent surgical attention.
   2. Epidural abscess in the presence of a worsening neurological deficit is a surgical emergency.
      a. Exceptions.
         (1) Nonoperative treatment consisting of antibiotic therapy with close monitoring may be considered if surgery would endanger the patient's life.

III. Disk space infections.
   A. Epidemiology/etiology.
      1. May occur as a result of direct inoculation.
         a. Surgical procedures.
            (1) Diskogram.
            (2) Diskectomy.
            (3) Intradiskal electrothermal therapy (IDET).

Fig. 24.4 Sagittal T2-weighted magnetic resonance image demonstrating increased signal intensity of the intervertebral disks and vertebral bodies between L2–L5 suggestive of vertebral osteomyelitis with disk involvement. (From Imhof H, ed. Spinal Imaging (Direct Diagnosis in Radiology). Stuttgart, Germany: Georg Thieme Verlag; 2008: Fig. 4.46. Reproduced with permission.)

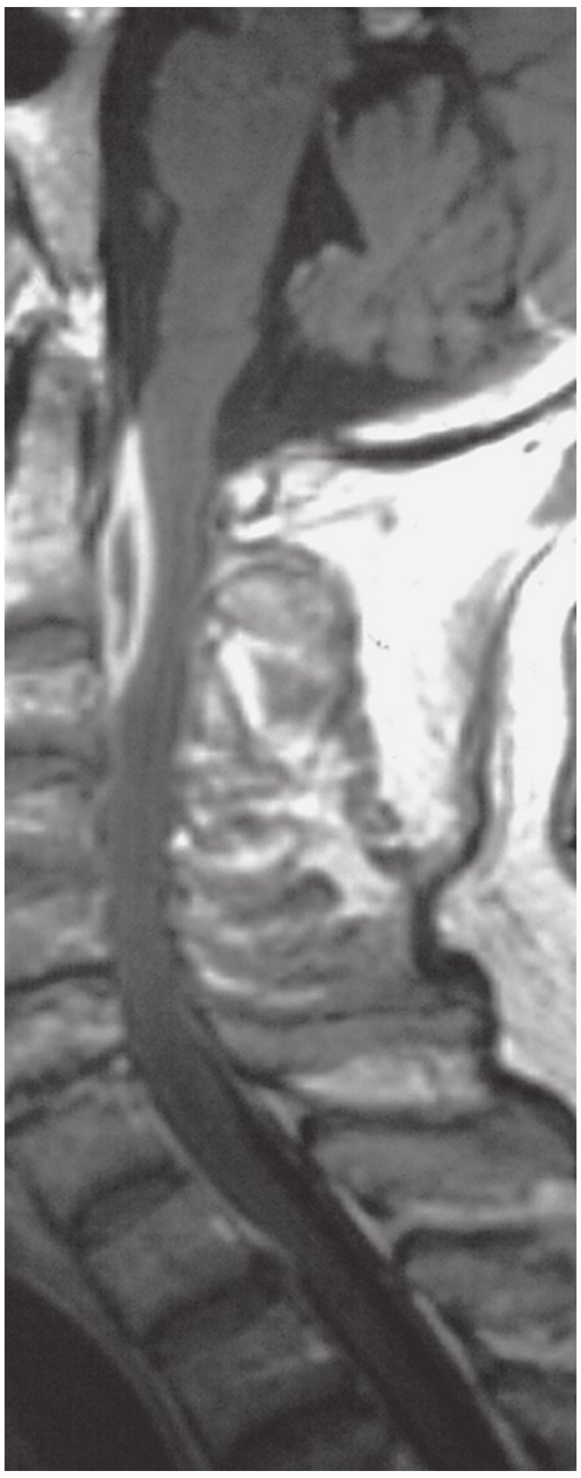

Fig. 24.5 Sagittal T2-weighted magnetic resonance imaging of a subdural abscess versus epidural metastases in the cervical spine. (From Uhlenbrock D. MR Imaging of the Spine and Spinal Cord. Stuttgart, Germany: Georg Thieme Verlag; 2004: Fig. 6.50. Reproduced with permission.)

2. Hematogenous spread:
    a. This is the route most commonly encountered in the pediatric population.
        (1) Blood supply from the disk is from the surface of the adjacent vertebral bodies.

3. The lumbar spine is most commonly involved.

B. Clinical findings.

1. The typical patient is between 2 and 7 years of age.
   a. Patients may not complain of back pain.
   b. Limping, refusal to walk, or hip pain may be presenting symptoms.
2. ESR and WBC are elevated.
3. MRI or bone scan is positive early in the disease **(Fig. 24.6)**.
   a. Plain radiographs may demonstrate narrowing of the intervertebral space, sclerosis, or bony erosion.

C. Treatment.

1. Surgery is rarely indicated or needed.
2. Immobilization with a brace.
3. Antibiotic therapy.
4. Biopsy is indicated if antibiotics are not effective.

IV. Tuberculosis of the spine.

A. Epidemiology/etiology.

1. Most common granulomatous infection in the world.
2. Hematogenous spread is the most common source (pulmonary or gastrointestinal infections).

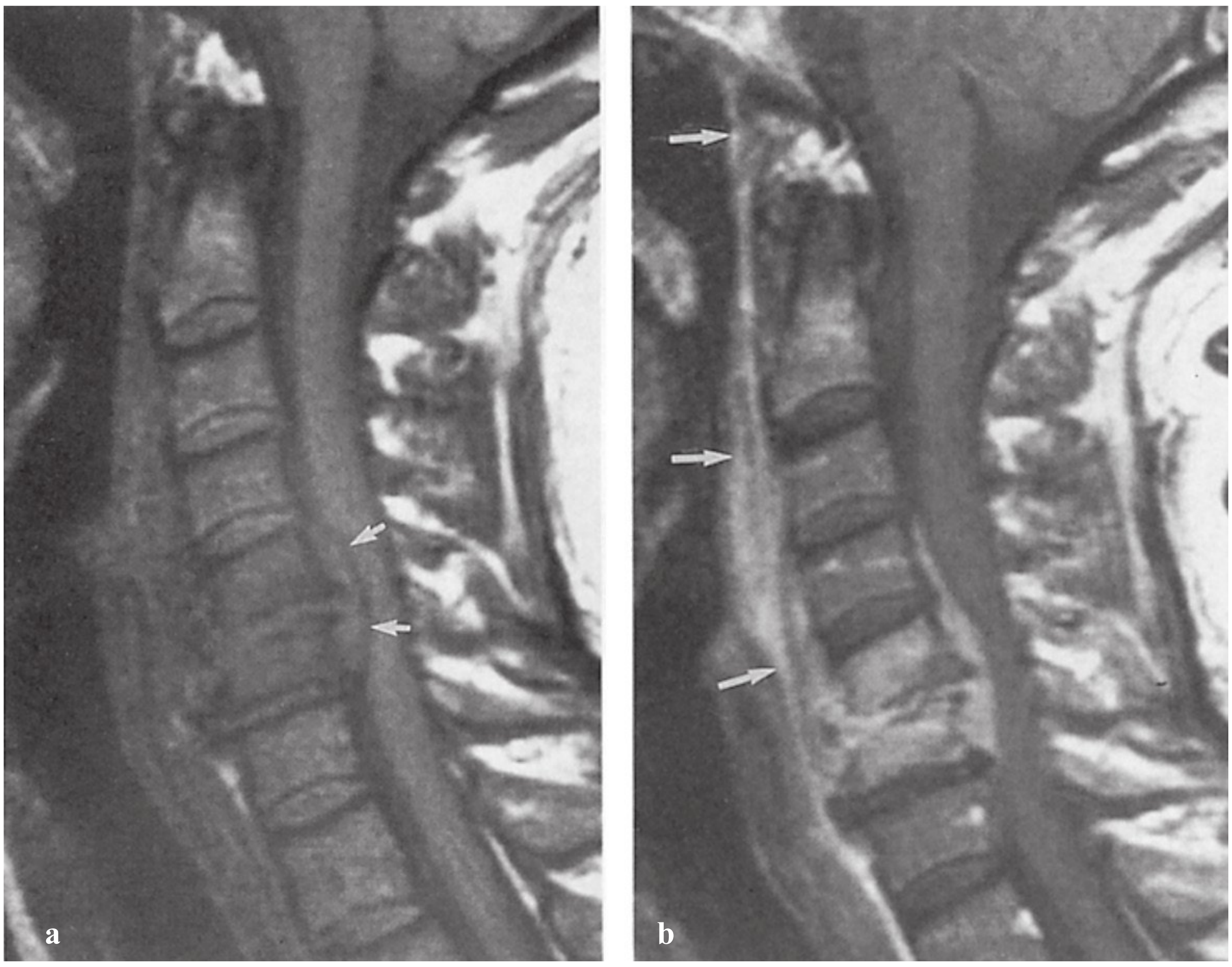

Fig. 24.6 (a,b) Sagittal T1-weighted magnetic resonance imaging demonstrating decreased signal intensity, epidural collection, and irregularity of the cervical spine suggestive of vertebral osteomyelitis. (From Uhlenbrock D. MR Imaging of the Spine and Spinal Cord. Stuttgart, Germany: Georg Thieme Verlag; 2004: Fig. 6.48. Reproduced with permission.)

3. The spine is the most common source of skeletal involvement.
   a. Most cases involve the anterior spine.
   b. Involvement of adjacent levels from expansion through the disk space.
   c. Fifty percent of infections are localized and can be categorized **(Fig. 24.7)**.
      (1) Peridiskal (most common): starts in the metaphysis and spreads under the anterior longitudinal ligament.

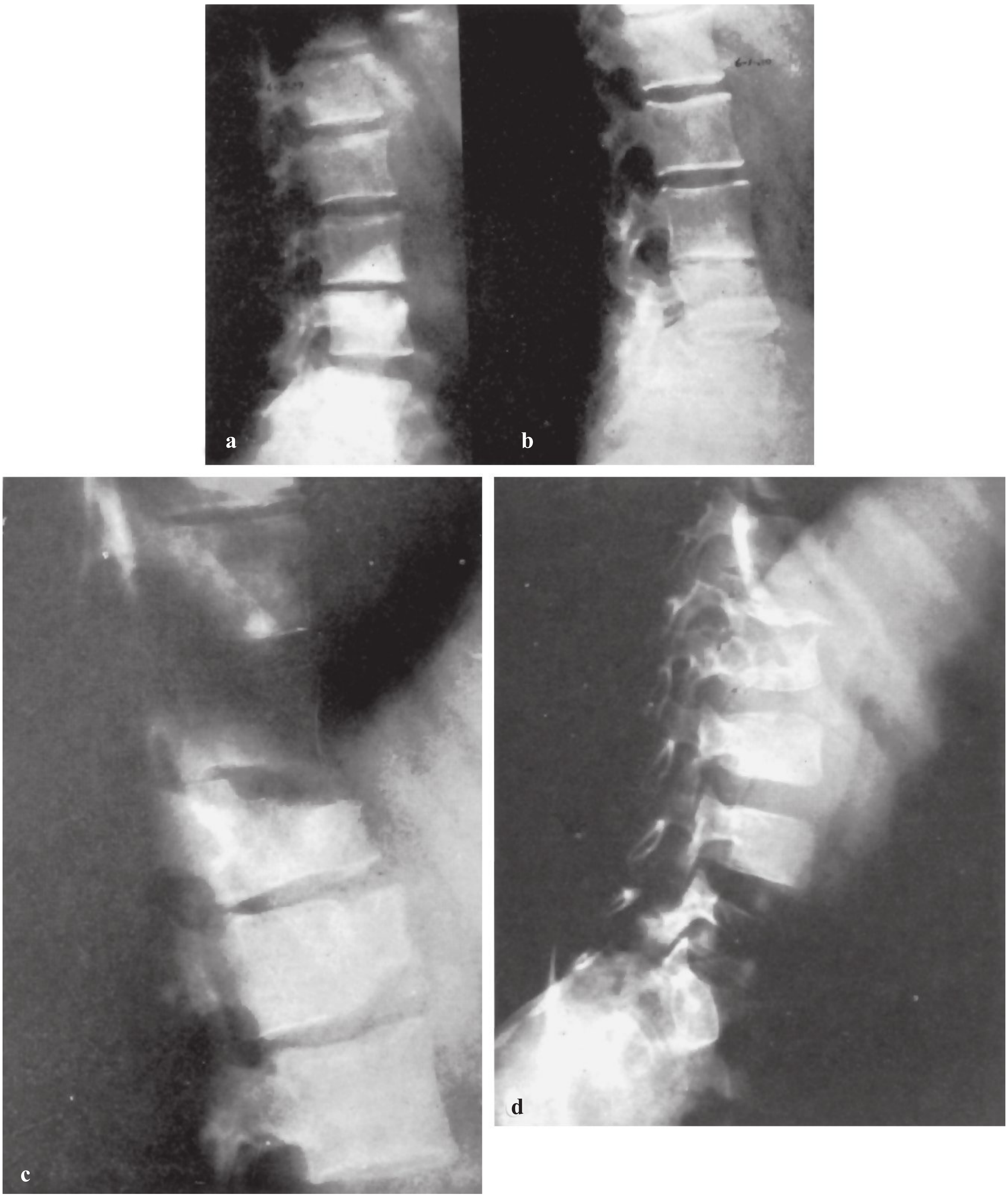

Fig. 24.7 Radiographic features of the three types of tuberculous spondylitis. **(a,b)** Peridiskal involvement is characterized by disk-space narrowing followed by variable bone destruction. The radiograph on the left is early in the disease. The radiograph on the right is after resolution of the disease with minor deformity. **(c)** Anterior multilevel disease is distinguished by scalloped erosions of the anterior aspect of several adjacent vertebrae (T11, T12, L1). **(d)** Central involvement resembles a tumor with central body rarefaction and bone destruction followed by collapse (L1 and L2).

(2) Central (rare): starts within a single vertebral body.
(3) Anterior (rare): starts under the anterior longitudinal ligament.

B. Clinical presentation/diagnosis.
1. Pain is present with evidence of systemic illness.
a. Fever, malaise, and weight loss.
2. Local tenderness, muscle spasm, and limited range of motion.
3. Tissue biopsy can be difficult because of the long incubation period for mycobacterium.
a. Fifty percent false-negative rate.
4. Differential diagnosis:
a. Neoplasms.
b. Sarcoidosis.
c. Charcot spine.

C. Radiological evaluation.
1. MRI is the modality of choice.
a. Unique characteristics of tuberculosis versus pyogenic infections.
(1) Disk space is often spared.
(2) Involvement of anterior bodies over contiguous segments.
(3) Paraspinal abscesses and granulomas are distinguished with the use of gadolinium.

D. Treatment.
1. Conservative management.
a. Antituberculous drugs constitute first-line treatment.
b. Nine to 12 months of therapy.
c. Successful therapy is associated with a fall in the serial ESR values, a decrease in pain, and weight gain.
2. Surgical management.
a. Indications:
(1) Failure to respond to antituberculosis therapy.
(2) Neurological compromise or evidence of progressive spinal instability.
b. The Hong Kong procedure **(Fig. 24.8)**:
(1) Anterior approach for anterior pathology.
(2) Radical debridement and removal of all necrotic tissue.
(3) Strut graft/fusion using autograft or allograft restoring anterior column support.
(4) Posteriorly supplemented instrumentation in more than two levels anteriorly.
c. Laminectomy alone is contraindicated.

V. Postoperative infections.
A. Risk factors **(Table 24.3).**
B. Prevention.
1. Optimization of patient risk factors prior to surgery.
2. Strict sterile technique.
3. Dilute iodine in irrigation solution or dilute iodine wound soak prior to closure.

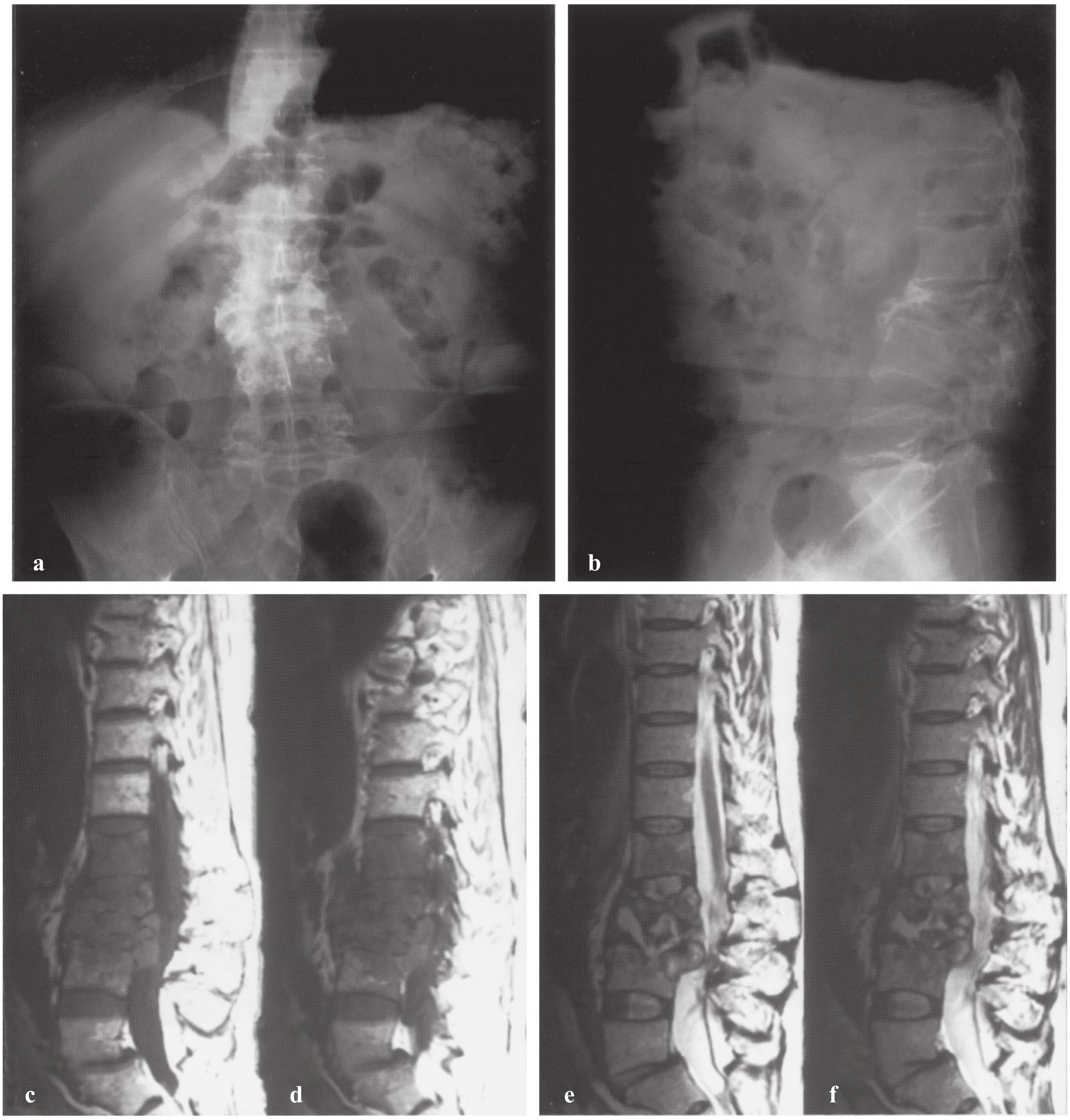

Fig. 24.8 Tuberculosis of the lumbar spine. A 52-year-old man with disseminated tuberculosis was treated with 1 year of therapy with three antituberculous drugs. He presented with progressive low back pain and neurogenic claudication. **(a)** Anteroposterior and **(b)** lateral lumbar spine radiographs show collapse of L2 and L3 with kyphotic deformity. **(c,d)** T1-weighted image sagittal magnetic resonance imaging (MRI) sequence demonstrates decreased signal in the bodies of L1 to L4, kyphotic deformity, and epidural mass composed of necrotic bone, disk, and purulent debris. **(e,f)** T2-weighted sagittal MRI sequence demonstrates areas of high signal intensity within the L2 and L3 vertebral bodies and in the anterior paraspinal region.

4. Vancomycin mixed bone graft or pulse lavage.
   a. Most recent meta-analyses demonstrate favorable outcomes.
   b. Results may depend on the incidence of postoperative spinal infections in the local population.
      (1) Vancomycin mixed bone graft is likely more efficacious if the number to treat is greater.

Table 24.3 Risk factors for postoperative infections

Diabetes mellitus
Chronic corticosteroid use
Chemotherapy
Revision surgery
Prolonged operative time (> 4 h)
Morbid obesity
Preoperative/postoperative infectious condition
Tooth abscess
Urinary tract infection
Pneumonia
Open sores
Prolonged drainage from the surgical wound

C. Presentation.
   1. Elevated ESR and WBC with concomitant fever and wound drainage are suspicious signs.

D. Diagnosis.
   1. MRI with gadolinium enhancement is the best modality to detect an SSI.
   2. Vertebral and soft tissue changes must be differentiated between normal postoperative changes and vertebral osteomyelitis.
      a. Both states are associated with type 1 end plate changes characterized by adjacent marrow edema and hypointense signal on T1 imaging.
      b. Gadolinium contrast demonstrates areas of enhancement in the disk space.
      c. Infection is associated with circumferential disk enhancement, whereas linear areas of enhancement are more consistent with normal changes.

E. Early versus late.
   1. Early infections are typically recognized by systemic complaints.
      a. Fevers, chills, local wound erythema, drainage, increased back pain
   2. Late infections:
      a. More common, particularly in the presence of instrumentation.
         (1) Difficult clinical diagnosis that should be considered if significant risk factors are apparent.

F. Superficial versus deep.
   1. Difficult to differentiate by physical examination.
      a. Irrigation and debridement should include opening the deep fascial layer to evaluate for the presence of an occult deep wound infection.

G. Management.
   1. The type of postoperative infection dictates management:
      a. Superficial SSIs respond to a course of intravenous antibiotics and/or bedside drainage.
      b. Medical therapy alone is unsuccessful with subfascial infections due to poor antibiotic penetration.
         (1) May warrant multiple episodes of extensive debridement of infected and necrotic tissue.
   2. Instrumentation should be retained to maintain the stability of the spinal column; however, loosened implants should be removed, and the patient should be monitored closely for pseudarthrosis.
   3. Negative-pressure wound therapy:
      a. Vacuum-sealed suction device that encloses the surgical wound.
      b. Enhances would healing and closure due to the following mechanisms:
         (1) Removal of interstitial fluid that reduces interstitial fluid pressure and enhances blood perfusion.
         (2) Negative pressure causes mechanical stimulation that facilitates cell growth, increases blood flow, and reduces bacterial load.

Suggested Reading

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25 Rheumatoid Arthritis

25.1 General Considerations

I. Epidemiology.
   A. Most common during the fifth and sixth decade.
   B. Female > male (3:1).
   C. Twenty-five to 80% of patients with rheumatoid arthritis (RA) will have cervical spine involvement.
      1. The thoracic and lumbar spine are rarely affected.
II. Pathogenesis.
   A. Complex interaction between genes and environment.
      1. Genetic component.
         a. Increased susceptibility mediated by HLA-DR antigens.
         b. Twelve to 15% concordance rate of RA between identical twins.
      2. Environmental components.
         a. Smoking (strongest evidence).
            (1) Increases susceptibility 20 to 40 times.
            (2) Activates inflammatory cascade resulting in complement attachment to the tissues.
         b. Other environmental components.
            (1) Infections (no causal effect has been proven).
               (a) Mycoplasma.
               (b) *Proteus mirabilis.*
               (c) Epstein–Barr virus (EBV).
               (d) Retrovirus.
            (2) Occupation exposure.
               (a) Silica.
            (3) Alcohol intake.
   B. Cellular interaction with synovial tissue.
      1. T lymphocytes.
         a. Constitute > 50% of cells in most RA synovium.
         b. Activate B cells to increase antibody production.
         c. A defect in programmed cell death (apoptosis) results in lymphoproliferation.
         d. Antigens that can induce a T cell–mediated adaptive immune response:
            (1) Type II collagen.
            (2) Immunoglobulin G (IgG).
            (3) Citrullinated proteins.
            (4) Glycoproteins (secreted by synovial cells and chondrocytes).
      2. Angiogenesis and cell migration.
         a. New synovial blood vessels are formed.
            (1) Increases fluid transudations.

(2) Promotes transmigration of lymphocytes into the synovium and polymorphonuclear leukocytes into the synovial fluid.

3. Tumor necrosis factor (TNF).
   a. Activates production of adhesion cells in newly formed endothelial cells.
   b. Helps recruit inflammatory cells into the synovium.
   c. Inhibits apoptosis of inflammatory cells.
   d. Induces the production of cytokines to propagate the inflammatory cascade.
4. Cellular components in synovium and synovial fluid.
   a. Synovium.
      (1) T lymphocytes (CD4).
      (2) Fibroblast-like synoviocytes.
         (a) Mediate initial destruction of cartilage at the cartilage–pannus junction (periphery of joints).
   b. Synovial fluid.
      (1) T lymphocytes (CD8).
      (2) Neutrophils.

III. Clinical findings.
   A. Insidious onset.
   B. Constitutional symptoms.
   C. Symmetrical polyarthritis.
      1. Morning stiffness.
      2. Joint pain and swelling.
      3. Wrist and finger involvement (does not affect distal interphalangeal [DIP] joint).
      4. Axial spine is involved in 20 to 50% of the cases.
   D. Extra-articular involvement.
      1. Subcutaneous nodules.
      2. Pleuropericarditis.
      3. Episcleritis.
   E. Variable clinical course.
      1. Spontaneous exacerbations and decrease in symptoms.
      2. Structural damage is cumulative and irreversible.

IV. Laboratory findings.
   A. Synovial fluid aspirate (rarely obtained in practice).
      1. Inflammatory effusion.
      2. Leukocyte count 1,500 to 25,000/mm^3.
      3. Predominance of polymorphonuclear (PMN) cells.
   B. Serological tests.
      1. Not used for screening purposes.
      2. Rheumatoid factor (RF) (75–85%).
      3. Anticitrullinated peptide antibodies (ACPA) (> 95%).
      4. Positive serological findings can be present as early as 10 years prior to any symptoms.
      5. Both RF and ACPA are associated with a higher risk of developing erosive joint damage and functional impairment.

C. Other hematologic findings.
  1. Anemia of chronic disease.
  2. Elevated ESR and CRP.

V. Radiographic studies.
  A. All RA patients should have cervical spine examination.
    1. Plain film radiographs.
      a. Helps determine atlantoaxial instability.
      b. Used to predict risk of paralysis.
    2. Magnetic resonance imaging (MRI).
      a. Useful in visualizing spinal cord compression due to odontoid pannus (space available for the cord [SAC]).
    3. Computed tomographic (CT) scan.
      a. Provides excellent bony detail.
      b. Excellent ability to detect spinal cord compression from synovial pannus if performed with intrathecal contrast.
      c. Reserved for patients with contraindication to MRI (e.g., pacemakers).

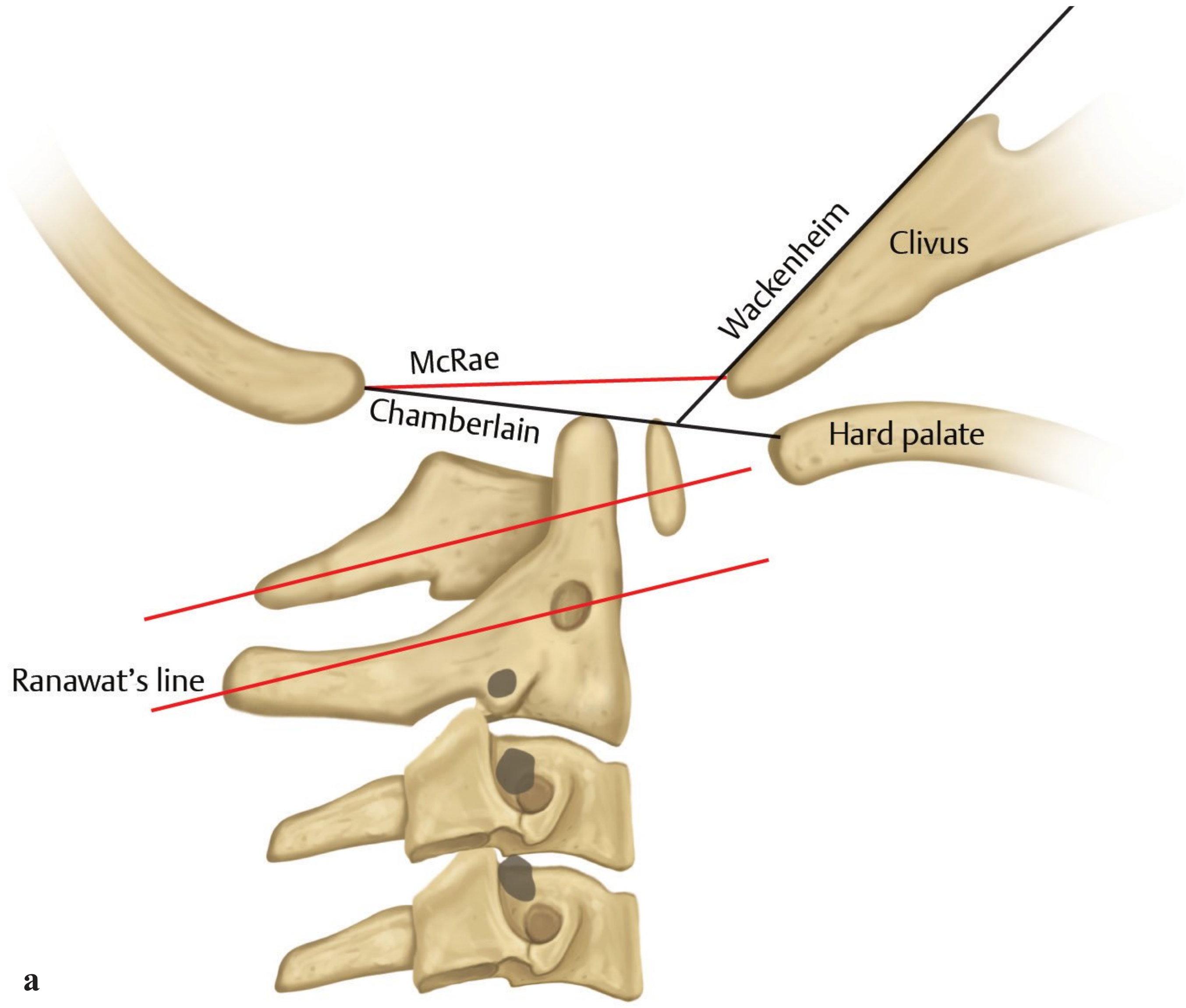
VI. Cervical spine deformity.
  A. Instability depends on the severity of the disease process.
  B. Subluxation appears 1 decade after the disease onset.
  C. Radiographic progression of subluxation has been observed in 35 to 80% of patients.
    1. Seven to 34% will develop a neurological deficit.
    2. Recent evidence has demonstrated that aggressive early medical treatment with disease-modifying antirheumatic drugs (DMARDs) can significantly decrease the risk of cervical spine atlantoaxial disorders.
  D. Atlantoaxial instability (or subluxation) **(Fig. 25.1)**:
    1. Most common cervical spine abnormality in RA (49%).
    2. Results from erosive synovitis in the following joints:
      a. Atlantoaxial.
      b. Atlanto-odontoid.
      c. Atlanto-occipital.
    3. Radiographic findings.
      a. Anterior atlantodens interval (AADI):
        (1) Distance between the posterior margin of C1 to the anterior surface of the odontoid:
          (a) The normal distance is 3 mm in adults and 4.5 mm in children.
        (2) AADI is an unreliable predictor of paralysis because of poor correlation between the AADI and the degree of cord compression as shown by magnetic resonance imaging (MRI).
          (a) AADI $\geq$ 8 mm has a positive predictive value (PPV) of 61% and a negative predictive value (NPV) of 56%
      b. The posterior atlantodens interval (PADI) has been demonstrated as a better predictor of paralysis.
        (1) The interval between the posterior dens and the anterior margin of the lamina at C1 is measured in a lateral plain film radiograph.
          (a) An interval $\leq$ 14 mm has a PPV of 69% and an NPV of 94% with regard to predicting paralysis.
          (b) Preferred screening test.

Fig. 25.1 Sagittal T2-weighted magnetic resonance imaging taken of the patient demonstrating that the spinal cord is decompressed in extension.

(2) Limitations:
  (a) Retro-odontoid synovial pannus may occupy as much as 3 mm of space.
  (b) May not represent the true SAC.
  (c) PADI < 14 mm on lateral plain film radiograph warrants MRI.
    i. PADI ≥ 13 mm measured on MRI represents spinal cord compression.

4. Clinical symptoms.
   a. Neck pain.
   b. Headache.
   c. Vertigo.
   d. Myelopathy.
      (1) Paresthesias.
      (2) Abnormal gait.
      (3) Bowel/bladder difficulties.
      (4) Difficulty with fine motor control.

E. Atlantoaxial impaction or basilar invagination **(Fig. 25.2)**:
   1. Second most common cervical spine abnormality in RA (38%).
   2. Characteristics:
      a. Superior migration of the odontoid (SMO).
      b. Vertical subluxation of the axis.
      c. Pseudobasilar invagination.
   3. Synovitis and cartilage destruction of the occipitoatlantal and atlantoaxial joints.



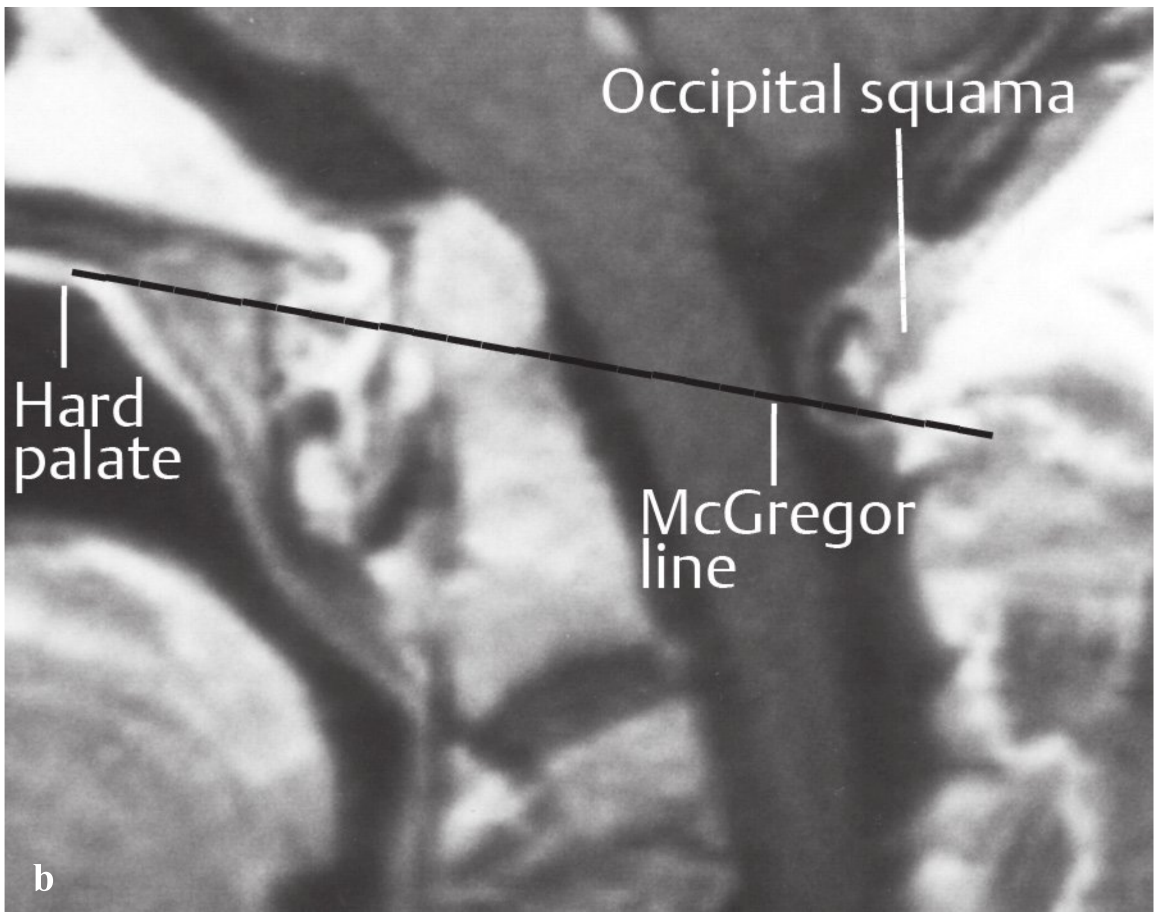


Fig. 25.2 **(a)** Illustration and **(b)** radiograph demonstrating the measurements of the skull, including McRae's line, Chamberlain's line, Wackenheim's line, and Ranawat's line. (Fig. 25.2b from Bohndorf K, Imhof H, Pope TH Jr. Musculoskeletal Imaging: A Concise Multimodality Approach. Stuttgart, Germany: Georg Thieme Verlag; 2001: Fig. 9.105. Reproduced with permission.)

4. Symptoms include occipital headache, myelopathy, or brain stem compression signs.
5. Radiographic measurements **(Table 25.1) (Fig. 25.3)**:
   a. Used to identify the degree of odontoid encroachment on the spinal cord.
   b. Most are difficult to reproduce.

Table 25.1 Radiographic lines to measure superior migration of the odontoid

Name	Measurements	Characteristic	Results
McGregor line	Line connecting posterior margin of the hard palate to the most caudal point of the occiput	Most consistent reference	Vertical settling is defined as migration of odontoid > 4.5 mm
Redlund–Johnell line	Distance between the midpoint of the inferior margin of the body of the axis to the McGregor line	Measures the occiput to C2 complex	Increased risk of neurological injury with values < 34 mm for men and < 29 mm for women
Ranawat index	Distance between the center of the pedicle of the axis and the transverse axis of the atlas	Evaluates the C1–C2 segment	< 13 mm is diagnostic of vertical settling
McRae line	Connects the anterior and the posterior margins of the foramen magnum		The tip of the odontoid should lie 1 cm below this line

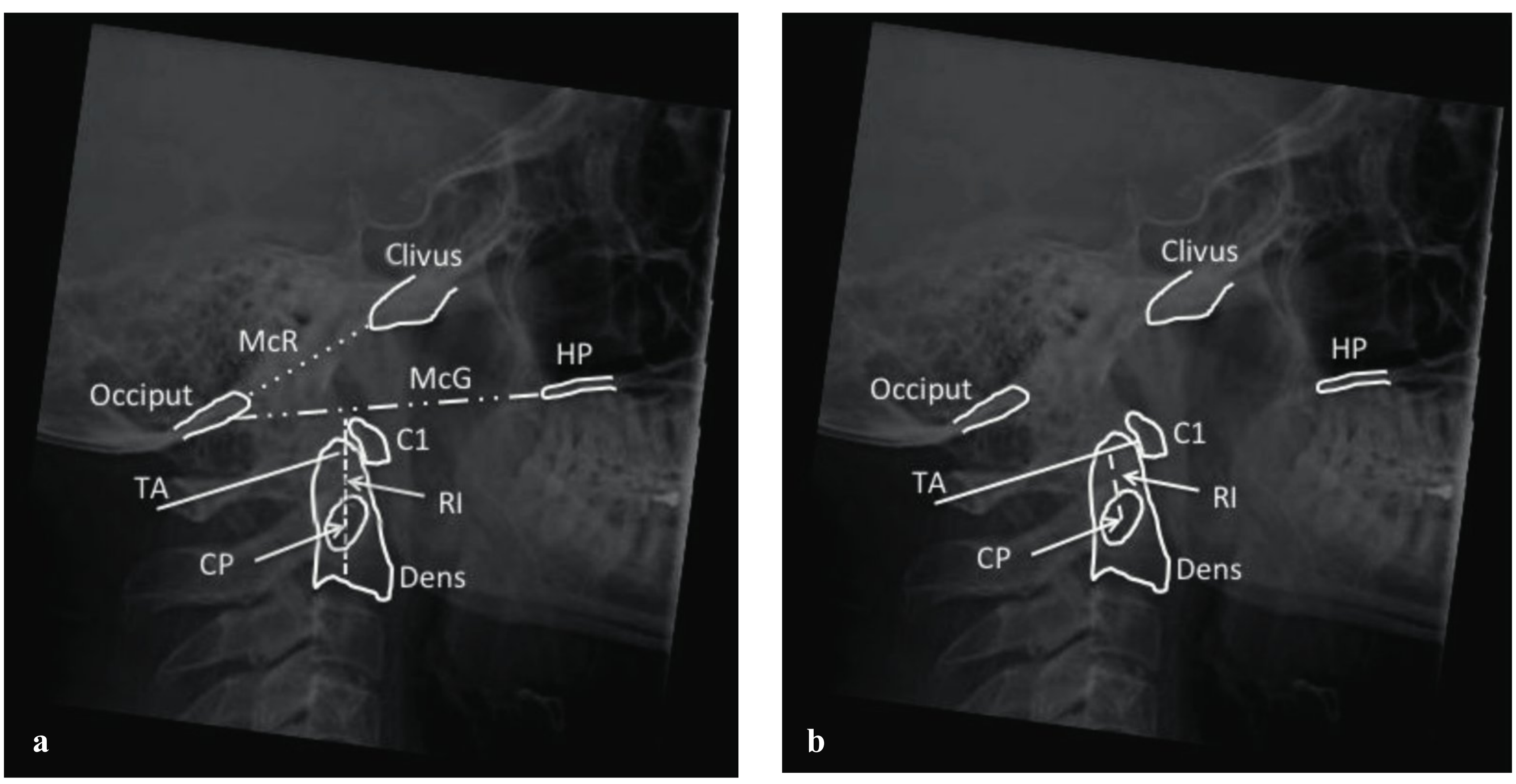


Fig. 25.3 Lateral plain film radiograph demonstrating the anatomical landmarks used to draw the **(a)** McGregor line (McG), Redlund–Johnell line (RJ), McRae line (McR), and **(b)** the Ranawat index. HP, hard palate; TA, transverse axis of atlas; CP, center of pedicle of axis.

6. MRI measurements:
   a. Any degree of atlantoaxial impaction on plain film radiograph warrants MRI.
   b. Cervicomedullary angle:
      (1) Effective indicator of cord distortion.
      (2) The angle formed by lines drawn parallel to the anterior border of the medulla and upper cervical spinal cord.
      (3) Angles < 135° have been associated with myelopathy.

F. Subaxial subluxation **(Fig. 25.4)**:
   1. Least common deformity in RA (10–20%).

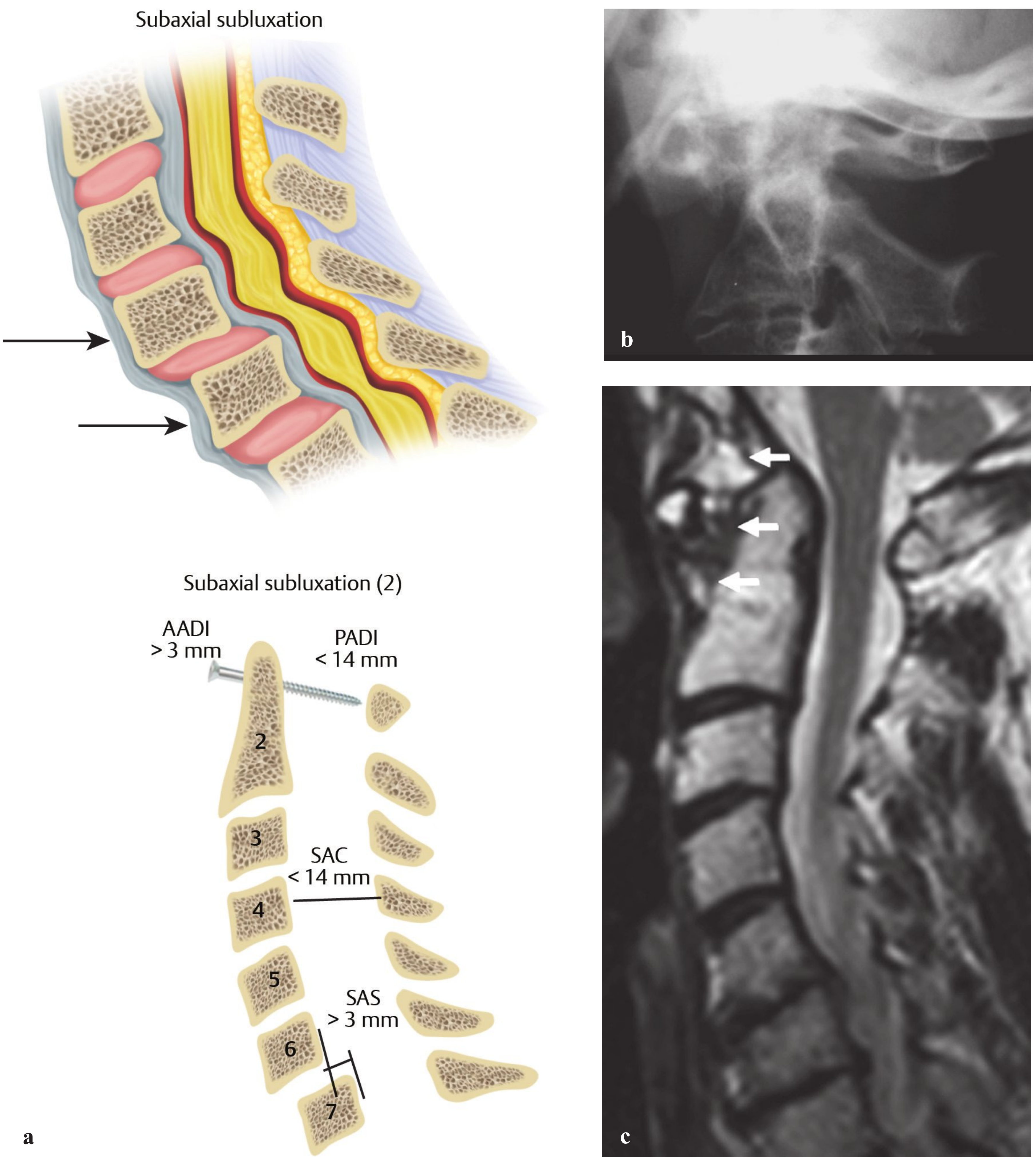


Fig. 25.4 **(a)** Illustration and **(b,c)** lateral radiographs demonstrating subaxial subluxation of the cervical spine. (Fig. 25.4b,c reprinted from Imhof H, ed. Spinal Imaging [Direct Diagnosis in Radiology]. Stuttgart, Germany: Georg Thieme Verlag; 2008: Figs. 4.2, 4.3. Reproduced with permission.)

2. Synovitis of facet joints, intervertebral disks (spondylodiskitis), and interspinous ligament.
3. Osteophytes are rarely observed.
4. Multilevel involvement:
   a. Particularly common at C2–C3 and C3–C4 region.
   b. Degenerative involvement typically occurs at C5–C6.
5. End plate erosions are present in 12 to 15% of patients.
6. On lateral plain film radiographs a canal diameter < 14 mm represents a higher risk of neurological involvement and warrants MRI.
7. RA patients with a previous upper cervical fusion have an increased risk of developing subaxial subluxation.

VII. Treatment considerations.

A. Avoid the development of an irreversible neurological deficit.

B. Prevent sudden death from unrecognized neural compression (~ 10%).

C. Early aggressive medical management:

1. DMARDs: mechanisms of action in RA are unclear.
   a. Methotrexate (MTX).
      (1) Inhibits the purine metabolisms, resulting in accumulation of adenosine (strong anti-inflammatory effects).
      (2) Inhibits T cell activation and expression of adhesion molecules.
         (a) Decreases production of T cell–mediated inflammatory cytokines.
      (3) Also inhibits folic acid metabolism.
         (a) Patients should consume folic acid supplement during treatment.
   b. Sulfasalazine.
      (1) Associated with increased production of adenosine (similar to MTX).
      (2) Free radical scavenger.
      (3) Inhibits TNF by inducing macrophage apoptosis.
      (4) Appears to suppress B cell function, but not T cells.
   c. Hydroxychloroquine (HCQ).
      (1) Decreases T cell activation by interfering with antigen-presenting cells.
         (a) As a lipophilic weak base, it trespasses the cell membrane and accumulates in lysosomes, increasing the intracellular pH.
            i. In macrophages and dendritic cells, the increase of intracellular pH inhibits antigen coupling with antigen-presenting peptides.
         (b) HCQ blocks toll-like receptors (TDR) that are responsible for activating dendritic antigen-presenting cells.
2. Soft cervical collars:
   a. Relieve symptoms but do not prevent disease progression.

D. Predictors of postoperative neurological recovery:

1. Ranawat classification **(Table 25.2).**
   a. More severe preoperative neurological deficits are associated with a poorer neurological recovery.
2. Location of disease.
   a. Proximal location of the pathology worsens the prognosis.

Table 25.2 Ranawat classification of rheumatoid myelopathy

Class	Clinical characteristics
I	No neural deficit
II	Subjective weakness with hyperreflexia and dysesthesia
IIIA	Objective weakness and long-tract signs; ambulatory patient
IIIB	Objective weakness and long-tract signs; nonambulatory patient

3. PADI.
   a. Preoperative.
      (1) A PADI < 10 mm is associated with poor prognosis.
      (2) In patients with isolated atlantoaxial subluxation with PADI > 10 mm predicted improvement was at least one Ranawat class.
      (3) In combined atlantoaxial subluxation and impaction recovery was associated with a PADI ≥ 13 mm.
      (4) All patients with a PADI ≥ 14 mm demonstrate significant motor recovery.
4. Factors that do not predict neurological recovery.
   a. Age.
   b. Gender.
   c. Duration of paralysis.
   d. Preoperative AADI.

VIII. Indications for surgical stabilization.
  A. Spinal instability with accompanying:
    1. Intractable pain.
    2. Neurological defect.
  B. Radiographic parameters (regardless of neurological involvement).
    1. Atlantoaxial subluxation with PADI ≤ 14 mm.
    2. Superior odontoid migration ≥ 5 mm.
    3. Subaxial subluxation with sagittal canal diameter ≤ 14 mm.
    4. Cervicomedullary angle < 135°.

IX. Surgical stabilization.
  A. General considerations.
    1. Preoperative halo traction can provide pain relief, correct deformity, and arrest or reverse neurological deterioration.
    2. Awake fiberoptic intubation without neck extension is indicated.
  B. Specific conditions.
    1. Atlantoaxial subluxation **(Fig. 25.5).**
      a. Posterior atlantoaxial fusion.
      b. C1–C2 transarticular screw fixation (Magerl).
        (1) May not be technically possible depending on the deformity and course of the vertebral artery.
      c. C1–C2 lateral mass/pedicle screw fixation (Harms construct).

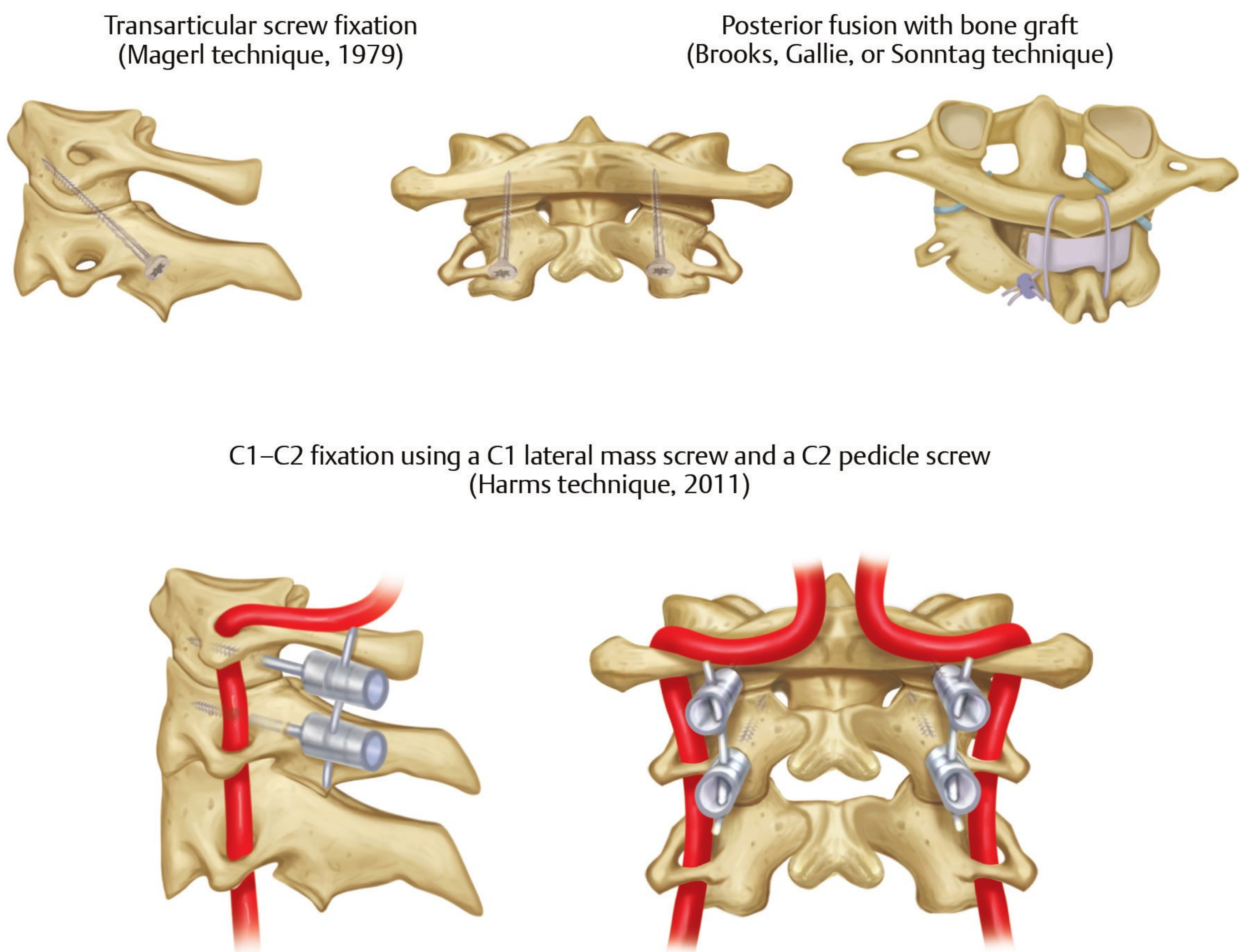


Fig. 25.5 Techniques for posterior cervical screw fixation, including the Magerl, Sonntag, and Harms techniques.

2. Superior migration of the odontoid **(Fig. 25.6a).**
   a. Posterior occipitocervical fusion.
   b. Anterior decompression via transoral resection of the odontoid is indicated when there is evidence of significant anterior pannus or marked vertical translocation of the odontoid (> 5 mm).
3. Subaxial subluxation.
   a. Posterior cervical fusion with lateral instrumentation.
      (1) Rarely, when notable subluxation is present and cannot be reduced, anterior decompression with corpectomy and reconstruction with strut bone grafting may be indicated **(Fig. 25.6b,c)**.

X. Postoperative outcomes.
   A. Recent improvement in outcomes.
      1. Earlier diagnosis.
      2. Decrease in use of corticosteroids.
      3. Improvement with combination DMARDs treatment.
      4. Better instrumentation.
   B. Occipitocervical fusion for atlantoaxial impaction (Casey et al).
      1. Better outcomes compared with those without occipital fusion.
      2. Forty-five percent demonstrated neurological improvement.

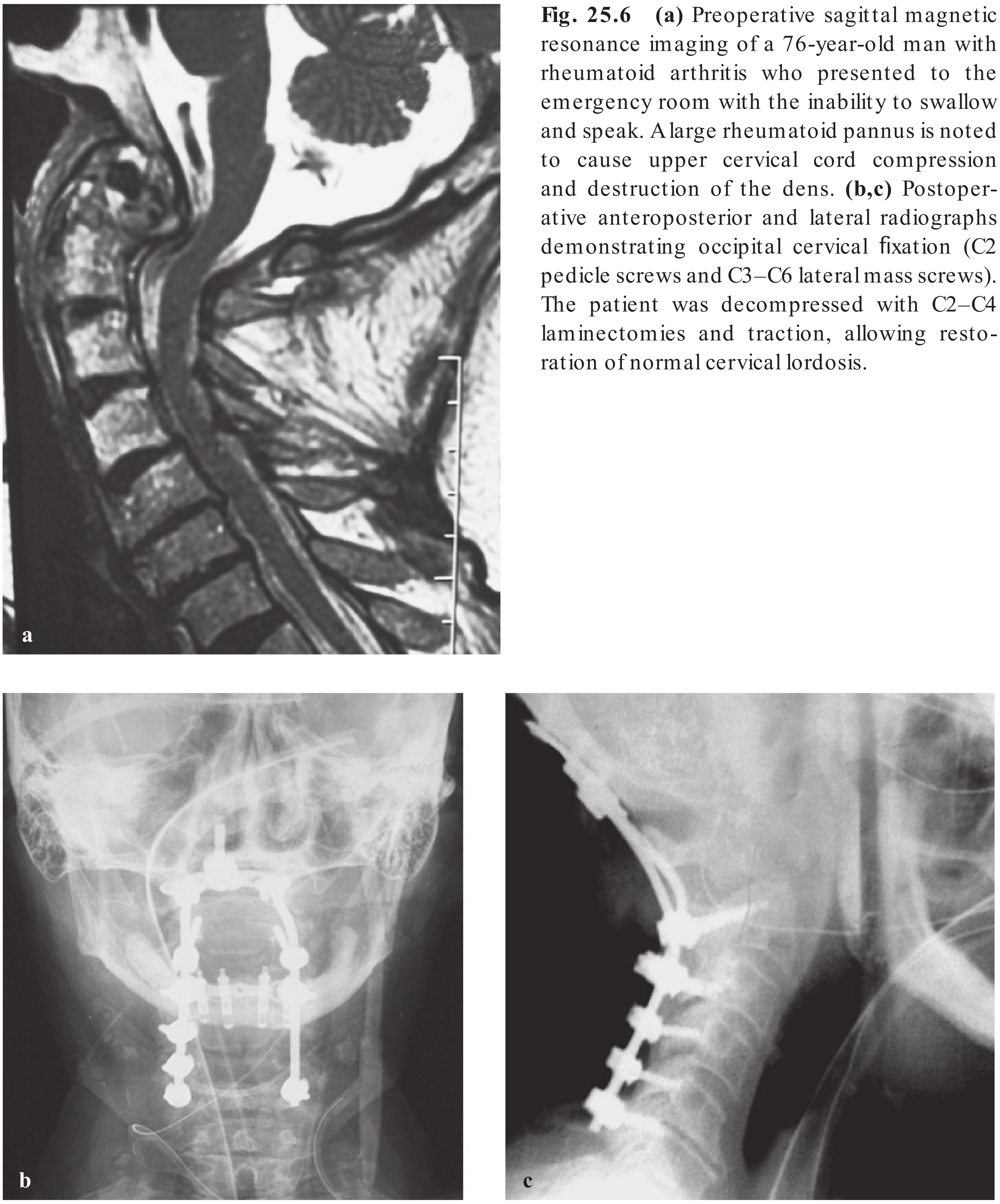


Fig. 25.6 **(a)** Preoperative sagittal magnetic resonance imaging of a 76-year-old man with rheumatoid arthritis who presented to the emergency room with the inability to swallow and speak. A large rheumatoid pannus is noted to cause upper cervical cord compression and destruction of the dens. **(b,c)** Postoperative anteroposterior and lateral radiographs demonstrating occipital cervical fixation (C2 pedicle screws and C3–C6 lateral mass screws). The patient was decompressed with C2–C4 laminectomies and traction, allowing restoration of normal cervical lordosis.

3. Ninety-seven percent demonstrated pain relief.
4. Progression of subaxial instability below the level of fusion was the main cause of failure (reoperation).
5. Perioperative mortality was 10%

C. Anterior and/or posterior decompression and fusion for subaxial subluxation (Olerud et al).

1. Neck pain was typically relieved.
2. Myelopathy was associated with worsened outcomes.
3. Surgery is recommended before patients develop myelopathy.

D. General complications.
  1. High infection rate (25%).
  2. Pseudarthrosis (poor bone quality).
  3. Adjacent-level instability.

Suggested Reading

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26 Seronegative Spondyloarthropathies

26.1 General Considerations

I. Introduction.
   A. Group of inflammatory disorders affecting various joints and periarticular structures.
   B. Extraskeletal manifestations:
      1. Gastrointestinal (GI).
      2. Skin.
      3. Ocular.
      4. Cardiac.
      5. Respiratory.
   C. The majority of cases are HLA-B27 (+) and rheumatoid factor (RF) (−).
   D. Most laboratory findings are nonspecific.
II. Ankylosing spondylitis **(Fig. 26.1).**
   A. Epidemiology.
      1. Mainly predominant during second and third decades of life.

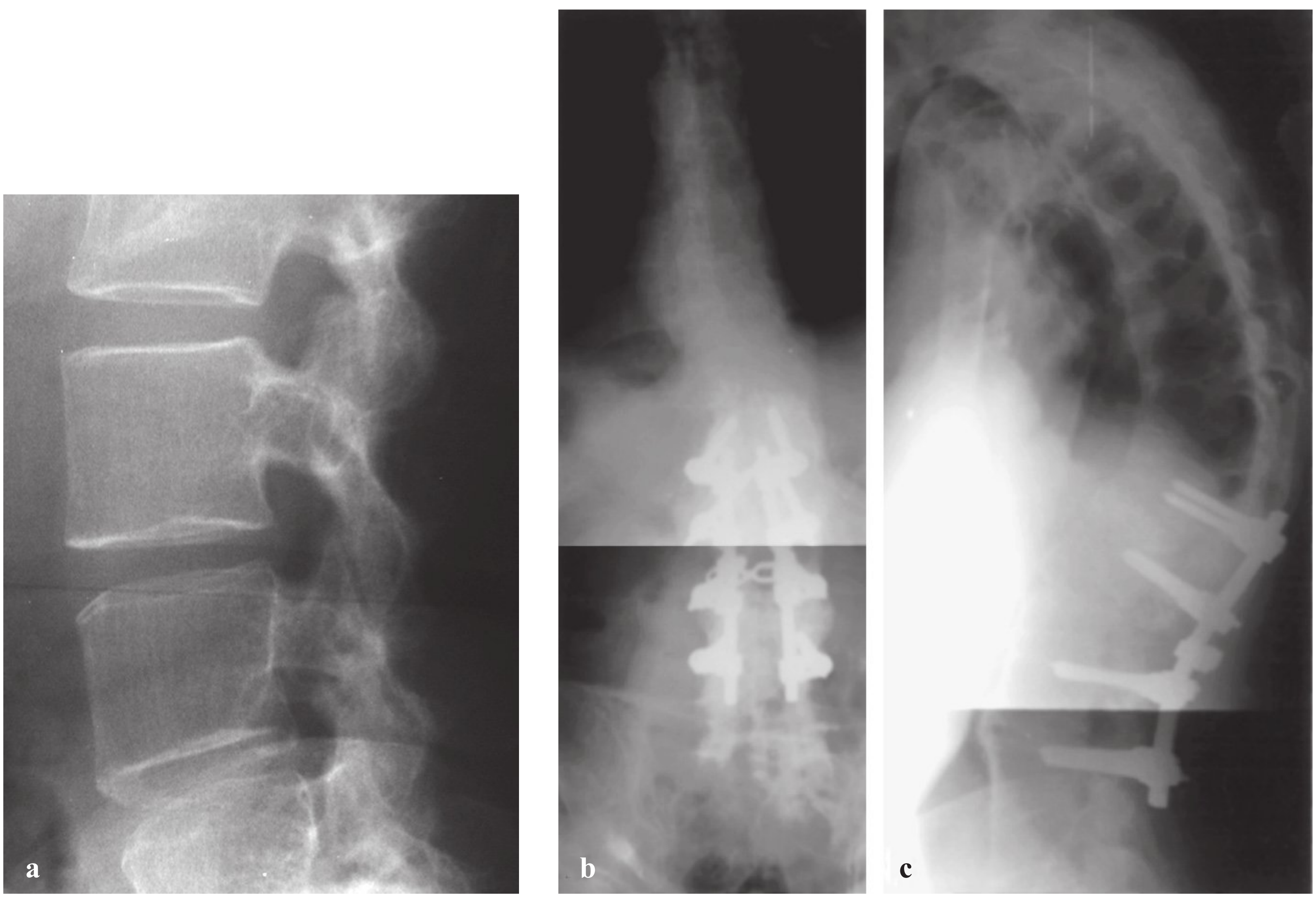

Fig. 26.1 **(a)** Lateral radiograph of a patient with ankylosing spondylitis with severe sagittal plane imbalance. **(b)** Anteroposterior radiograph following an L2 pedicle subtraction osteotomy. **(c)** Lateral radiograph shows the osteotomy site at L2 with correction of the positive sagittal balance. (Fig. 26.1a from Imhof H, ed. Spinal Imaging (Direct Diagnosis in Radiology). Stuttgart, Germany: Georg Thieme Verlag; 2008: Fig. 4.18. Reproduced with permission.)

2. Males > females.
    a. Men typically present a more severe disease expression.
    b. Prevalence is 1 in 1,000.
    c. Mostly Caucasian (HLA-B27).

B. Pathogenesis.
1. HLA-B27 positive in 88 to 96% of patients (nonspecific).
    a. Eight percent of general population.
2. Synovitis from lymphocyte and plasma cell infiltrate.
3. Chronic inflammatory changes (cartilage destruction and bony erosion) to the axial skeleton.
    a. Bilateral sacroiliitis is the most common and earliest sign of disease (pathognomonic).
    b. Enthesitis at tendon bony insertions.

C. Clinical findings.
1. Insidious onset:
    a. Symptoms are generally present for 3 months.
2. Symptoms include low back pain and stiffness.
    a. Worsened in the morning and improve with activity.
3. Fifteen to 25% have peripheral joint arthritis.
4. Forward-flexed posture:
    a. Decreased lumbar lordosis.
    b. Increased thoracic kyphosis.
5. Physical findings:
    a. Limited range of motion of the lumbar spine (Schober's test).
    b. Decreased chest expansion (< 7–8 cm).
    c. Positive sacroiliac stress maneuver (Patrick's test).
    d. Rigid kyphotic spine.
        (1) Most effective measure of spinal deformity is the chin–brow to vertical angle.
        (2) Occiput to wall test (cervical spine involvement).
    e. Compensatory hip flexion contractures.
    f. Tenderness over the ischial tuberosity, greater trochanter, anterior-superior iliac spine (ASIS), and iliac crest (enthesitis).
6. Extraskeletal manifestations:
    a. General complaints
        (1) Fatigue.
        (2) Weight loss.
        (3) Low-grade fever.
    b. Gastrointestinal.
        (1) Inflammatory bowel disease.
    c. Cardiac.
        (1) Cardiac conduction defects.
        (2) Aortitis.
    d. Acute iritis (most common extraskeletal complaint).
        (1) Pain.
        (2) Photophobia.

(3) Blurred vision.

e. Pulmonary fibrosis (cause of death in 10% of patients).

(1) Dyspnea and cough.

f. Amyloidosis.

D. Laboratory findings.

1. HLA-B27 (~90%): often not ordered due to high cost.
2. Elevated erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP).
3. Anemia of chronic disease (normochromic/normocytic).
4. RF (−).
5. Antinuclear antibody (ANA) (−).

E. Radiographic findings **(Fig. 26.2** and **Fig. 26.3) (Table 26.1).**

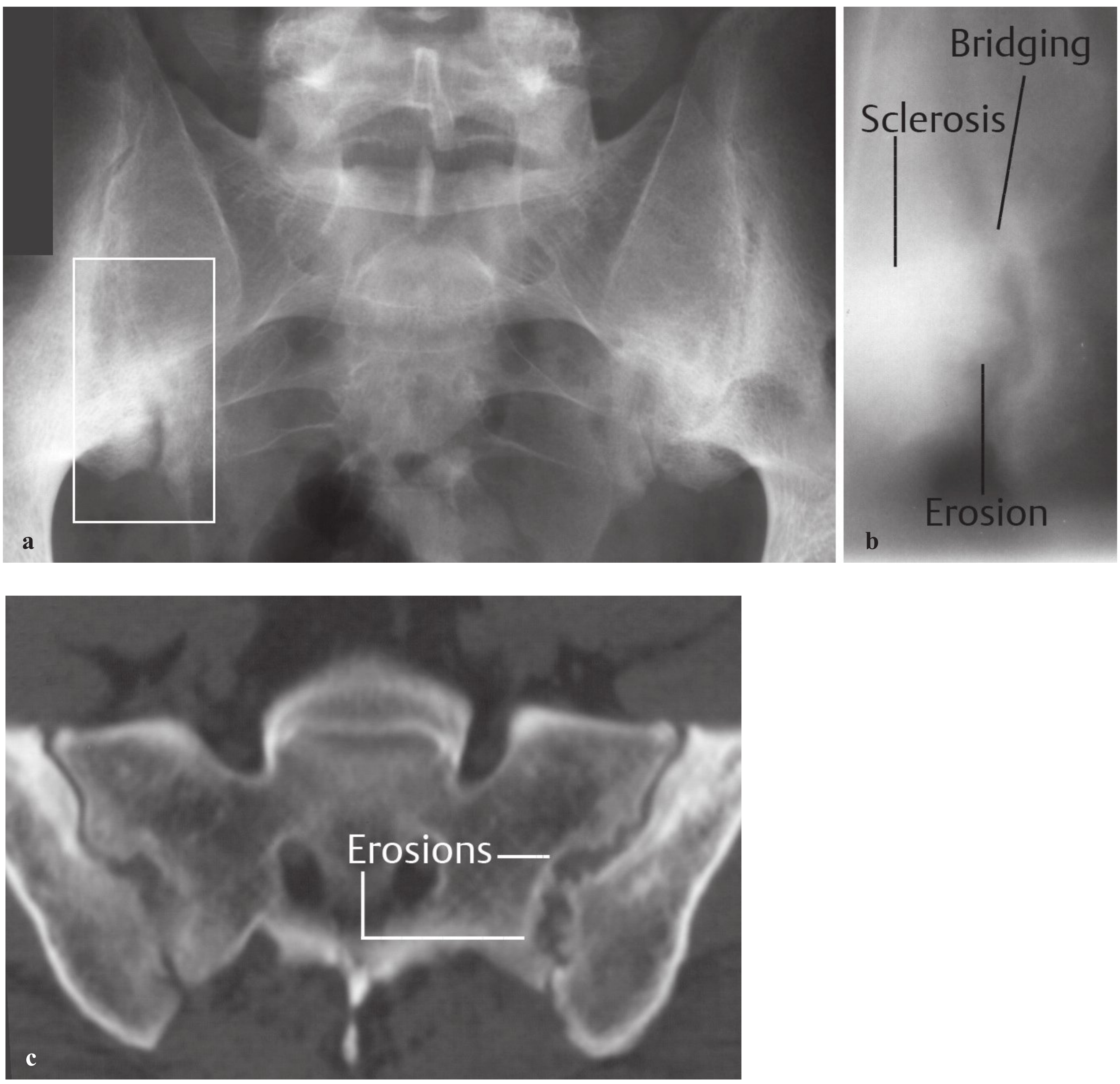

Fig. 26.2 (a,b) Radiograph demonstrating sclerosis and erosion at the sacroiliac joint. **(c)** Axial computed tomographic image demonstrating erosions at the sacroiliac joint. (From Bohndorf K, Imhof H, Pope TH Jr. Musculoskeletal Imaging: A Concise Multimodality Approach. Stuttgart, Germany: Georg Thieme Verlag; 2001: Figs. 9.112, 9.113. Reproduced with permission.)

Fig. 26.3 Anteroposterior radiograph demonstrating bamboo spine in a patient with ankylosing spondylitis. (From Imhof H, ed. Spinal Imaging [Direct Diagnosis in Radiology]. Stuttgart, Germany: Georg Thieme Verlag; 2008: Fig. 4.27. Reproduced with permission.)

F. Treatment.
  1. Nonoperative.
    a. Physical therapy.
      (1) Extension exercises.
      (2) Swimming.
    b. Respiratory therapy.
      (1) Breathing exercises.
      (2) Cessation of smoking.
    c. Medications.
      (1) Nonsteroidal anti-inflammatory drugs (NSAIDs).
        (a) Symptomatic pain relief.

Table 26.1 Radiographic findings

Type	Notes
Sacroiliitis erosion **(Fig. 26.2)**	– Reactive bone and fusion of the joint (on the lower portion of iliac side first) – Bilateral and symmetric – Magnetic resonance imaging is more sensitive than plain film radiographs to detect inflammatory changes in the sacroiliac joint
Spine	– Syndesmophyte formation from the margins of the vertebral bodies ("bamboo spine") **(Fig. 26.3)** – Vertical paravertebral ossification – Erosion of the vertebrae ("squaring of the vertebral body") – Osteoporosis, disk and apophyseal joint narrowing
Occult fractures	– Occur with minimal trauma – Lumbar spine and lower cervical spine are the most common sites of fracture – Plain radiographs can be difficult to interpret – Computed tomography can be hard to interpret because of the difficulty in obtaining true axial cuts – Magnetic resonance imaging is the most reliable test for occult fractures and hematoma evaluation

(2) Corticosteroids (short courses with tapering dose).
 (a) Systemic.
 (b) Topical drops (uveitis).
(3) Disease-modifying medications.
 (a) Sulfasalazine (most beneficial).
 (b) Methotrexate.
(4) Tumor necrosis factor alpha (TNF-α) inhibitors.
 (a) Adalimumab and etanercept.
 (b) Never first-line treatment.
(5) Bisphosphonates.

2. Surgical management.
   a. Indications.
      (1) Flexion deformity associated with pain and neurological compromise.
      (2) Loss of horizontal gaze (e.g., chin on chest deformity).
      (3) Unstable spine fractures.
   b. Spinal deformity in ankylosing spondylitis.
      (1) Loss of lumbar lordosis and thoracic kyphosis.
      (2) Primary location of disabling deformity should be assessed.

(3) Osteotomies:
   (a) Cervical.
      i. Osteotomy at C7–T1 junction with laminectomy from C6 to T2 is considered for primary cervical kyphosis.
   (b) Thoracic.
      i. In severe kyphotic thoracic deformity, multiple posterior thoracic resection osteotomies can be done after anterior osteotomies.
      ii. Costotransverse osteotomies.
      iii. Thoracic kyphosis can often be addressed via a lumbar osteotomy.
   (c) Lumbar.
      i. Osteotomies are performed typically between L2 and L4.
         - Pedicle subtraction (closing wedge) osteotomy (CWO).
         - Modified Smith–Petersen (opening wedge) osteotomy (OWO) **(Fig. 26.4).**
      ii. OWO versus CWO:
         - No difference in postoperative pain scores.
         - Similar radiographic outcomes.
            ▫ Sagittal vertical axis.
            ▫ Lumbar lordosis.
            ▫ Global kyphosis.
         - Complications.
            ▫ Greater blood loss with CWO.
            ▫ No significant differences in other complications (e.g., durotomies, ileus, infections, neurological injuries).

III. Reactive arthritis.
   A. Epidemiology.
      1. Males > females.
      2. More common in Caucasians.
      3. Associated organisms.
         a. *Chlamydia*.
         b. *Campylobacter*.
         c. *Yersinia*.
         d. *Shigella*.
         e. *Salmonella*.
   B. Clinical presentation.
      1. Classic clinical triad.
         a. Urethritis.
            (1) Balanitis circinata (painless ulcer in the glans penis).
         b. Conjunctivitis.
            (1) Iritis.
            (2) Uveitis.
            (3) Episcleritis.
            (4) Corneal ulceration.

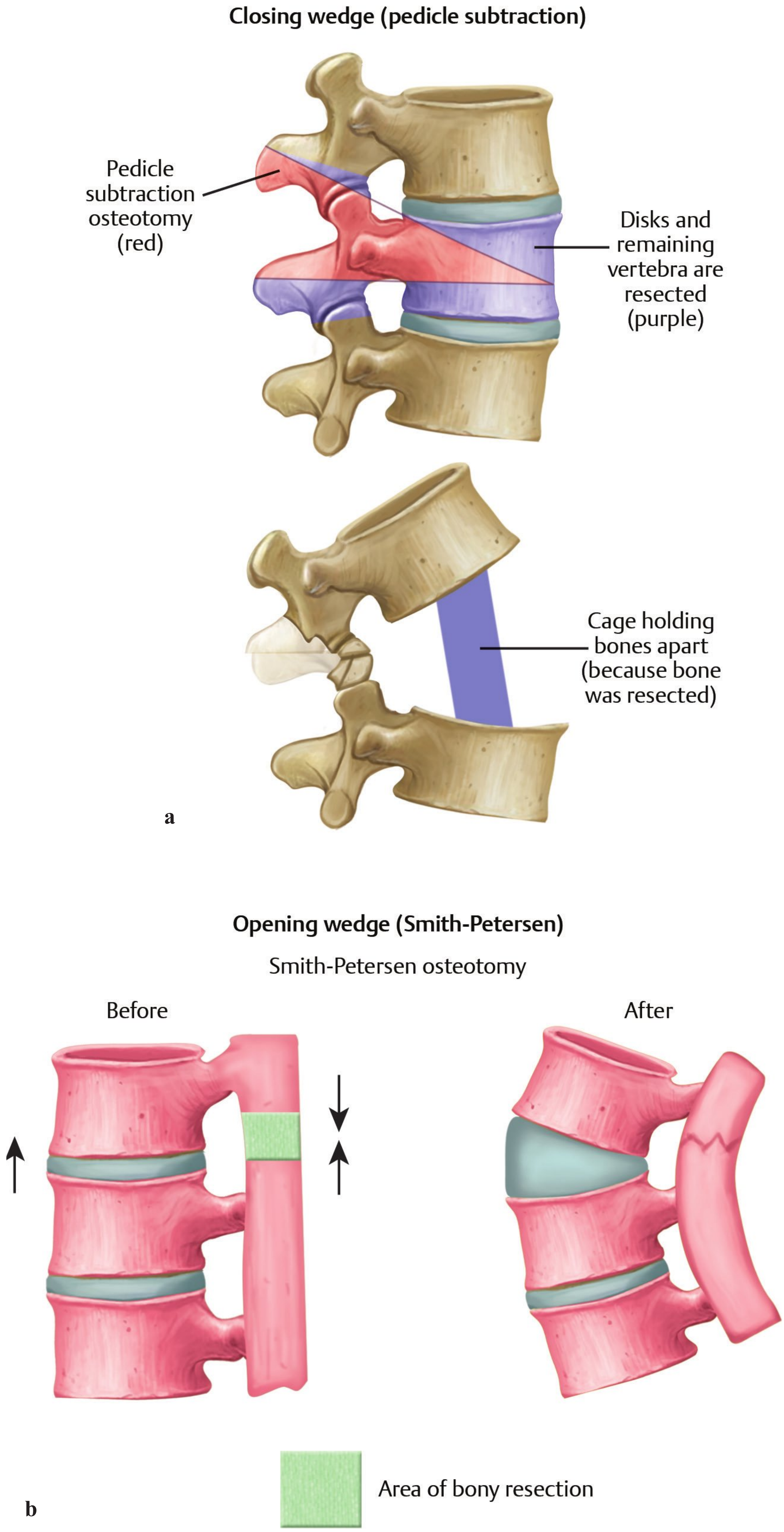

Fig. 26.4 **(a)** The closing wedge pedicle subtraction and **(b)** the opening wedge Smith–Petersen osteotomy procedures.

c. Polyarthritis.
(1) Asymmetric.
(2) Appears 2 to 4 weeks after infectious event.
(3) Lower extremities > upper extremities.
(a) Rare hip involvement.
(4) Enthesitis (inflammation of the connective tissue between a tendon or ligament and bone).
(a) Achilles tendon.
(5) Lower back pain.
(a) Sacroiliitis (3–10% progress to ankylosing spondylitis).

C. Laboratory findings.
1. Elevated ESR.
2. RF (−) and ANA (−).
3. Chronic anemia.
4. HLA-B27 (+) (> 90%).

D. Radiographic findings.
1. Periostitis in the heel and toes.
2. Sacroiliitis (unilateral).
3. Nonmarginal asymmetric syndesmophytes (large and bulky).

IV. Psoriatic arthritis.

A. Epidemiology.
1. Affects 5 to 7% of patients with psoriasis.
2. Affects 0.1% of the general population.
3. Male = female.
4. Onset ranges between the third and sixth decade.
5. More common in Caucasians.

B. Clinical findings.
1. Arthritis (precedes skin lesions in 15% of cases).
a. Spine stiffness > 30 minutes.
b. Oligoarticular and monoarticular.
(1) Asymmetric.
(2) Distal interphalangeal (DIP) involvement.
c. Enthesitis.
d. Sacroiliitis.
2. Skin lesions.
a. Balanitis circinata.
b. Oral ulcers.
c. Keratoderma blennorrhagica.
3. Key features.
a. Nail pitting.
b. Sausage-shaped digits.

C. Laboratory findings.
1. Elevated ESR and CRP.
2. HLA-B27 (+) (20%).

D. Radiographic findings.
   1. Asymmetric sacroiliitis (autofusion).
   2. Bony erosion.
   3. Syndesmophyte formation (bamboo spine).
E. Treatment.
   1. Physical therapy.
      a. Range of motion exercises.
   2. Medications.
      a. NSAIDs.
         (1) Aspirin, indomethacin, or naproxen.
      b. Second-line.
         (1) TNF-α inhibitors (infliximab).
         (2) Methotrexate.

V. Enteropathic arthritis (inflammatory bowel disease).
A. Epidemiology.
   1. Males > females.
   2. Associated with patients with Crohn's disease and ulcerative colitis (10–20%).
B. Clinical findings.
   1. Arthritis.
      a. Asymmetric.
      b. Monoarticular or polyarticular.
         (1) Large joints.
            (a) Peripheral arthritis improves with gastrointestinal improvement.
         (2) Spine (sacroiliitis).
            (a) Bilateral (similar to AS).
            (b) Independent of bowel disease.
   2. Extra-articular symptoms.
      a. Erythema nodosa (Crohn's).
      b. Pyoderma gangrenosum (UC).
      c. Oral ulcers.
      d. Uveitis.
C. Laboratory findings.
   1. Chronic anemia.
   2. Elevated ESR and CRP.
   3. RF (−) and ANA (−).
   4. Antineutrophil cytoplasmic antibodies (ANCA) (+) (60%).
   5. Five percent HLA-B27 (+).

Suggested Reading

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Note: Page numbers followed by f and t indicate figures and tables, respectively.

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