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2.1 Introduction

The lumbar spine exists in a unique anatomical and surgical environment, with biomechanical and load-carrying characteristics distinct from those within the cervicothoracic spine. The ability to approach lumbar elements with a relatively facile lateral trajectory affords new augmentation and fixation opportunities. Though our understanding of lumbar biomechanics in both physiological and pathological settings is well appreciated, the vast majority of investigations of lumbar surgical intervention have evaluated posterior and anterior approaches. The growing popularity of lateral lumbar surgery will undoubtedly lead to additional lumbar biomechanical inquiry. A number of important questions need to

be considered with respect to lateral surgery: defining the benefits of maintenance of posterior elements, the effects of disengaging load-bearing facets, and the outcomes of vertebral interspace height and alignment augmentation. The goal of this chapter is to provide a review of normal lumbar anatomy and biomechanics, as well to review and extrapolate what is already known to altered mechanics in the setting of lumbar pathology and lateral lumbar intervention.

2.2 Lumbar Anatomy

An understanding of lumbar spine anatomy is essential to both appreciate the relevant biomechanics in the physiological and pathologic settings, as well as to understand the effects of lateral surgery. A complex interplay of multiple elements defines the lumbar region, including osseous structures such as the vertebral body and endplate, strong and mobile facet joints, and active and passive soft-tissue components – including muscles, ligaments, and intervertebral discs. Select components contain mechanoreceptors, proprioceptors, and pain receptors that communicate with the central nervous system to maintain stability and avoid injury. The combination of these elements serves four main functions, including bracing viscera and the appendicular skeleton, nerve root and thecal sac protection, extremity control, and mobility. Each lumbar

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element must maintain stability, as well as support mobility and control. Our bipedal evolutionary trajectory has resulted in the development of structures that are capable of impressive force-resistance, as well as efficient and fluid movement. The lumbar spine is itself a widely mobile bridge to the lower extremities [1]. These lumbar structures not only carry a greater load than the cervical and thoracic spines but also lack the stabilizing effect of the ribcage and must support the oversized lever arms required to maintain upright posture [2].

2.2.1 Vertebra

The vertebral body is the basic functional unit of the spine. The five lumbar vertebral bodies are all comparatively more massive than their thoracic and lumbar counterparts, owing to their increased force-carrying requirements. The bodies themselves become progressively larger from cranial to caudal, with each level absorbing the majority of the rostral axial load strain. A honeycombed cancellous architecture, akin to the inner support of an airplane wing, achieves a high strength to weight ratio – approximately four times that of cortical bone. Vertical struts connect from one endplate to the other and absorb the axial loads; outward bowing is contained by orthogonally oriented transverse struts, thereby converting forces from vertical to horizontal [3]. Transverse elements also absorb shear forces. These oblique struts sweep together at the pedicles to resist the localized high tensile forces, then vertically to the facet masses where vertical forces are again deflected, and finally to the lamina and spinous process to resist further tensile and bending forces [1].

The ratio of cortical to cancellous bone is directly related to weight-bearing status, with a more complex cancellous arrangement within the body and facet masses and a greater cortical concentration at the pedicle [2]. Fortunately this arrangement provides greater pedicle screw pull-out resistance, with the notable exception of the sacrum. The pedicles assume a progressive increase in mass from rostral to caudal. The

transverse diameter increases and the pedicle height progressively decreases as the spine is descended. The transverse pedicle angle also increases with more caudal direction, while the opposite is true in the sagittal plane [4, 5].

2.2.2 Intervertebral Discs

Intervertebral discs are specialized structures that have both the tension-resisting properties of a ligament and load-carrying element akin to joint cartilage [3]. Healthy discs provide spine mobility while also absorbing immense axial and shear loads. Each disc has two distinct elements, the nucleus pulposus and annulus fibrosus. The former, a remnant of the primitive notochord, is composed of water (a 90% majority), collagen, and highly hydrophilic proteoglycans. The annulus is a circumferential bundle of laminated collagen sheets, each sheet overlaying the previous in a different orientation and fusing with adjacent layers [1, 6]. The annulus is most robust ventrally and weakest in the posterolateral margins. The disc is bounded cranially and caudally by vertebral endplates, each composed of a thin layer of cortical bone (~1 mm, though thinnest in the center adjacent to the nucleus pulposus) and hyaline cartilage. Healthy endplates are no longer perforated by vascular feeders by the age of skeletal maturity, though they become revascularized in the setting of discal degeneration. The discs, therefore, are the largest structures in the body that rely on diffusion for nutrition and metabolic exchange [7].

Axial loading results in increased intradiscal pressure. These hydrostatic forces are transferred to radial forces that are contained by the high tensile strength annulus as well as ridged and inelastic endplates [1, 2, 7]. Hyperflexion and torsional loading lead to maximal strain at the posterolateral annulus margins, which can result in progressive annular tear and subsequent back pain. Complete failure of the annulus leads to nucleus herniation and nerve root compression [1, 6]. Senescence of the disc complex includes progressive nucleus desiccation and stiffening of the circumferential annulus fibers, the combination

of which leads to altered load sharing and transference of forces to the facets and the disc-plate periphery. Together with decreased bone mineral density and thinning of the cancellous trabecular architecture, weakened vertebral bodies can fail under stress and lead to compression fracture [2, 8].

2.2.3 Facets

The facets are diarthrodial joints that extend bilaterally from the lamina to articulate with corresponding facets of the level above and below. These osseous pillars have opposing cartilage surfaces and a synovial connective tissue lining that provides lubrication to create a low friction environment [9]. A strong ligamentous capsule envelops the joint. The capsule resists deformation, rotation, and translation, and a rich receptor innervation enables robust central nervous system communication to prevent joint overload or injury. The facets not only increase in mass from L1 to L5, but the orientation also progresses toward a more sagittal position with caudal descent (though the L5–S1 facet abruptly assumes a coronal orientation). Together they share in axial load bearing with the intervertebral discs, with 25 % of the load transmitted through the facets in the normal spine and a greater percentage with disc pathology or lumbar extension [1]. The oblique lumbar joint geometry means that they are excellent stops to rotatory, translational, and lateral bending movements and are less effective during flexion [9]. The facets ultimately guide and constrain movement at adjacent segments to prevent motions that could put the disc and neural structures at risk [10].

2.2.4 Muscles and Ligaments

Lumbar muscles are intimately involved with coordinating the impressive degree of physiologic motion of the spine along with constraining motion to qualities and quantities that do not put anatomical structures at risk. Flexors include the psoas muscle, as well as indirectly the rectus abdominus muscle. The latter balances the long

extensors, as well as creating a rigid cylinder around the spine by increasing intra-abdominal pressure and tensing the lumbodorsal fascia [3]. The lumbar extensors consist of the upper erector spinae (longissimus and iliocostalis lumborum) and lumbar erector spinae muscles (multifidus and lumbar portions of the longissimus and iliocostalis). The upper erector spinae muscle arises from the thoracic spine, spans the entire lumbar region, and inserts on the sacrum and posterior superior iliac spine. The lumbar longissimus and iliocostalis muscles have an oblique orientation which results in large posterior translation forces during lifting, counteracting ventral shear forces. The multifidus muscle consists of multiple overlapping fibers, with each fascicle arising from a single tendon connected to a spinous process and inserting on a caudal mammillary process, sacrum, and iliac crest. Multifidus muscle orientation and attachment points permit powerful extension and rotation [11].

A series of vertically oriented ligaments span dorsal and ventral vertebral segments. The posterior longitudinal ligament, supraspinous ligament, interspinous ligament, and ligamentum flavum resist hyperflexion. This latter ligament is infused with a high proportion of elastic fibers that resists buckling during extension. The anterior longitudinal ligament resists hyperextension through a continuous attachment along the ventral vertebral bodies and fewer attachments to the annulus fibrosus [4].

2.3 Lumbar Geometry and Stability

The various anatomical elements of the lumbar spine must interact seamlessly to ensure fluid and efficient motion while protecting adjacent structures from injurious forces and motions. Spinal stability, in essence, is the ability to safely engage in such physiologic movements; conversely, instability is defined by the inability to protect neural and mechanical elements under physiologic loads, leading to neural decline, incapacitating pain, or deformity [12, 13]. A stable lumbar spine is expected to maintain a lordotic posture,

engage in efficient energy expenditure during movement, participate in force transfer to control limb movement, and neutralize noxious forces. A three-component system works to maintain stability: passive tensile forces such as osseous elements, ligaments, facets, and intervertebral discs; active forces are the muscles and tendons that work to maintain position within safe ranges of motion; last is the system of peripheral to central receptors and nerves that coordinate such safe movement and attempt compensation for force stresses [3].

2.3.1 The Neutral Zone

Spinal stability is better understood when considering displacement of lumbar elements while subjected to increasing loads. Neutral posture and small but incremental load carrying is characterized by a zone of high laxity (flexibility) with low stiffness. The neutral zone (NZ) is manifest when movements are highly energy efficient, and ligaments, tendons, and muscles are under very little tension [4, 14]. As loads increase the terminus of the NZ is reached and the spine begins to stiffen, and individual spinal elements are placed under tension. Hence, a region of linear increase on the load-displacement curve known as the elastic zone (EZ) is entered. The EZ in a stable spine marks the end of pain and injury-free range of motion. The biphasic lumbar load-displacement curve, composed of the NZ followed by the EZ, allows for the dual and contradictory goals of movement and element protection (Fig. 2.1). The two zones permit energy-conserved motion and increase spinal stiffening near the terminus of physiologic motion; additional forces beyond these zones result in permanent deformation and eventually complete failure. Active spinal forces and central nervous system communication are taxed with maintaining posture within this range of motion [3].

Instability is thus further defined by a decrease in the capacity of spinal stabilizing systems to maintain the neutral zone within physiologic

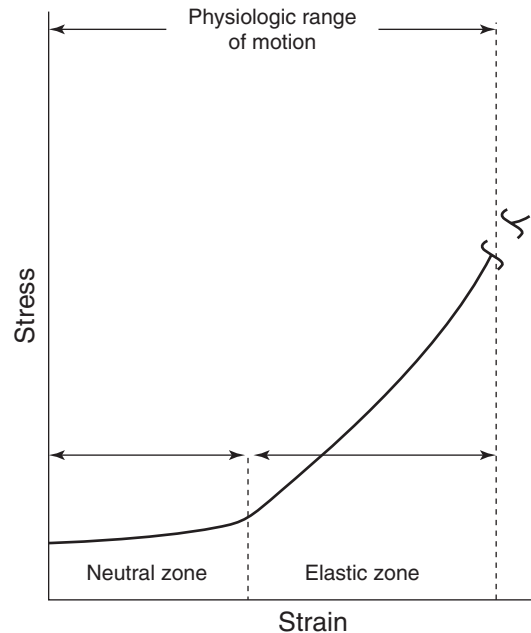


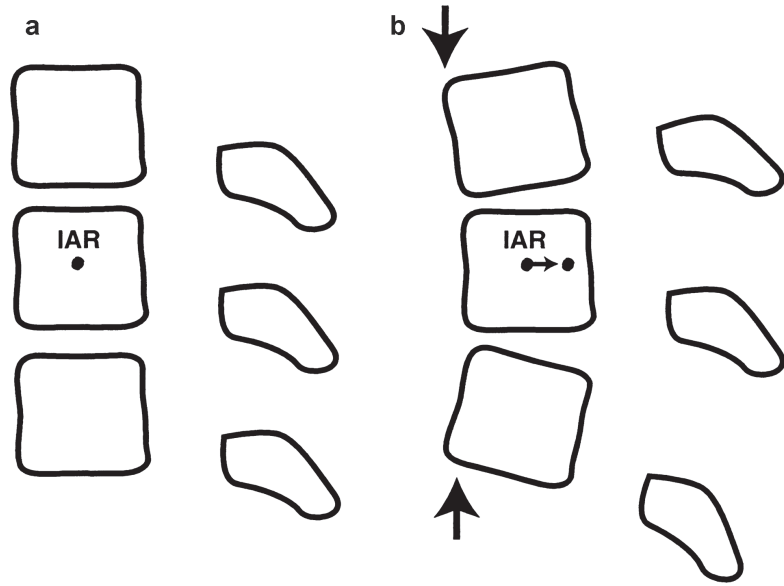
Fig. 2.1 A load-deformation curve illustrating the neutral and elastic zones (deformation or strain on the x-axis, load or stress on the y-axis) (With permission from Thieme Publishing, New York, NY; in *Biomechanics of the Spine* (editor/author: Edward C. Benzel), 2001)

ranges, that is, within quantitative and qualitative ranges of motion that do not cause neurologic dysfunction, pain, or deformity. The neutral zone is naturally floppy. Rigidity for the maintenance of the upright posture is sustained by active muscle contractions. The neutral zone is therefore dynamic, with both physiologic and pathologic behaviors augmenting its size. Stretching or ligamentous, disc, or other passive system injuries, including destabilizing surgery, expand the zone. The lumbar musculature stiffens the spine to prevent injury. Core strengthening (including the strengthening of the lumbar musculature) shrinks the neutral zone, as does surgical fixation [12, 14].

2.3.2 Bending Moments and Load Carrying

The maintenance of stability requires resistance to deforming forces. These can be divided into

Fig. 2.2 Translation of the IAR from preload (a) to a more dorsal position (b) after application of a ventral bending moment (With permission from Thieme Publishing, New York, NY; in *Biomechanics of the Spine* (editor/author: Edward C. Benzel), 2001)



component force vectors that have a well-defined direction in space. To determine the effect of a vector on an individual vertebral body functional unit, the point at which the vertebral body pivots at any given time (IAR, instantaneous axis of rotation) and the orthogonal distance from the IAR to the force vector (the moment arm) are defined. When a spine segment moves, the IAR typically passes through or close by the vertebral body. The IAR is dynamic, moving dorsally with flexion in the sagittal plane and more ventral with extension (Fig. 2.2). Its position can be predicted with flexion-extension radiographs. There are twelve potential movements around each IAR, encompassing both translation and rotation with respect to the $x/y/z$ axes [3, 12]. To determine the amount of force at a spinal level, one must calculate the force that a vector generates on the IAR by means of torque on the “imaginary lever” of the moment arm. The product of the moment arm length (D) and the vector force (F) applied to the moment arm is the bending moment (M) (Fig. 2.3).

$$D * F = M [12]$$

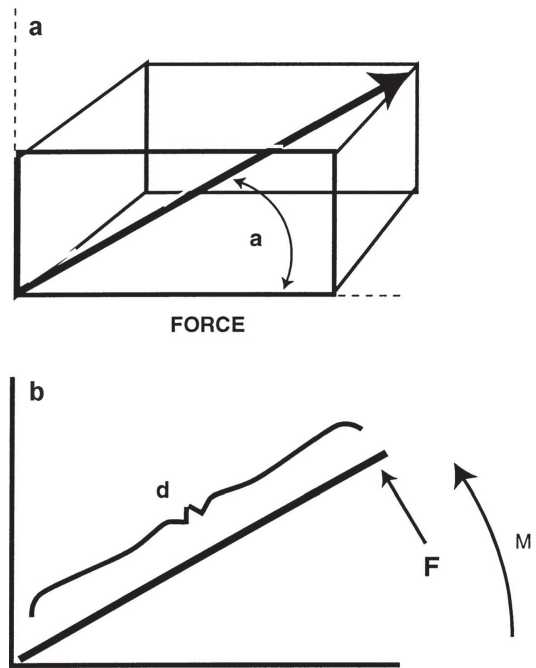
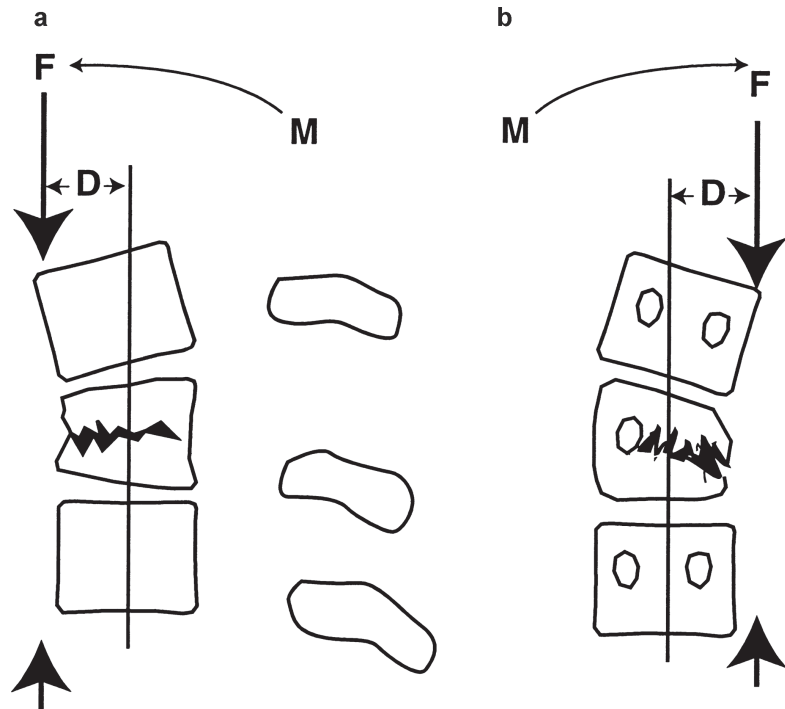


Fig. 2.3 (a) Force vector defined in three-dimensional space; (b) a bending moment (M) is created when a force (F) is applied at a distance (d) from a fulcrum (the IAR) (With permission from Thieme Publishing, New York, NY; in *Biomechanics of the Spine* (editor/author: Edward C. Benzel), 2001)

Fig. 2.4 The maximum bending moment (M , a product of force (F) and moment arm length (D)) is at the center of the circle created by the radius of its arc. (a) Lateral view of vertebral body failure from an excessive bending moment; (b) anteroposterior view (With permission from Thieme Publishing, New York, NY; in *Biomechanics of the Spine* (editor/author: Edward C. Benzel), 2001)



The bending moment results in rotation about the IAR. Counteracting forces positioned to produce bending moments equal in quantity though opposite in direction result in zero net motion, in keeping with Newton's second law of motion. The bending moments produced by forward flexion of the thoracic spine are counteracted by extension bending moments in the lumbar spine.

2.3.3 Lumbar Lordosis

The lordotic curve of the lumbar spine is an evolutionary adaptation to facilitate stability during upright posture and bipedal gait. Humans are born with a pan-kyphosis and assume cervical and lumbar lordosis with ambulation. The curves combine to center the trunk over the femoral heads and increase the resistance to vertical loads by deforming in ordered directions that are maintained by the active and passive spinal elements [3]. The exaggerated lordosis of the lower lumbar spine places the IAR in-line with major force vectors, resulting in only small bending moments. This protective factor results in a lower rate of

fracture with supranormal forces; fractures that do occur here result in more pure axial loads and a higher relative rate of burst fractures. In contrast, the straightening and slight kyphosis of the thoracolumbar junction shifts the IAR dorsally and increases the subsequent flexion bending moment, imparting significant stress on the discs and anterior vertebral bodies. Supranormal vectors, as occurs during trauma or repetitive flexion, predispose to compression fracture and disc degeneration [12] (Fig. 2.4). Lumbar hyperflexion during lifting also shifts the IAR dorsally, drastically increasing the flexion bending moment. Together with offloading the facets onto already stressed discs and vertebral bodies, such posture considerably increases ventral shear forces and the potential to damage spinal elements at lower compressive loads [11].

Maintaining lumbar lordosis, or augmenting lordosis in the setting of a flat back, decreases the moment arm and ventral stress. Interbody lordotic cages or posterior osteotomies can achieve this effect surgically [15]. Furthermore, a greater flexion moment arm must be counteracted by the active and passive elements of lumbar extension

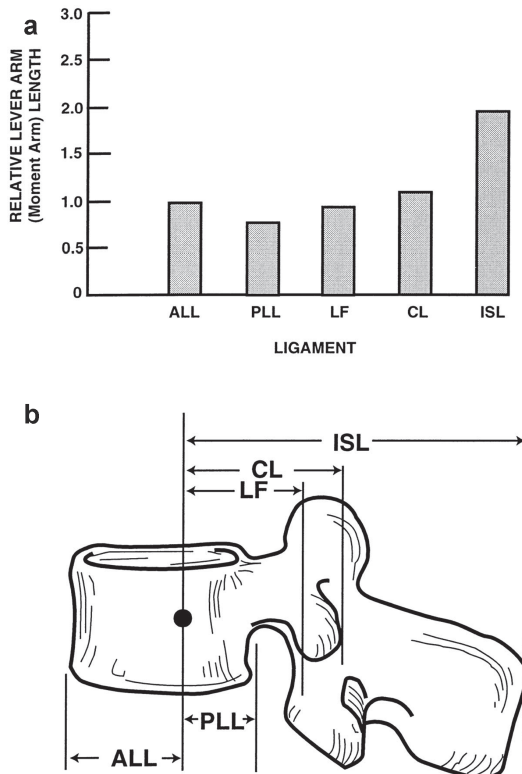


Fig. 2.5 The relative lever arm lengths of lumbar ligaments. (a) The moment arms of lumbar ligaments (yielding flexion or extension). (b) Ligament distance from the IAR. (Dot IAR, ALL anterior longitudinal ligament, PLL posterior longitudinal ligament, LF ligamentum flavum, CL capsular ligament, ISL interspinous ligament) (With permission from Thieme Publishing, New York, NY; in *Biomechanics of the Spine* (editor/author: Edward C. Benzel), 2001)

to maintain sagittal balance within the physiologic neutral zone. Posterior ligaments vary in their capacity to resist flexion based on their intrinsic strength and their moment arm length from the IAR. The supraspinous and interspinous ligaments have superior mechanical advantage and a proportionally high bending moment compared to the PLL, and thus their disruption can lead to progressive kyphosis. Capsular ligaments have superior strength and a moderate-length moment arm [12, 16] (Fig. 2.5).

Lumbar muscular extensors are most efficient while in lordosis. Lumbar flexion reduces the lever arm length of the erector spinae and the muscles must operate at a mechanical disadvantage

when resisting the forward flexion of the trunk. Sustained and more forceful contractions lead to inefficient energy expenditure and back pain, fatigue, and poor posture. Multifidus loses optimal fascicle orientation with lumbar flexion, impairing its ability to resist flexion, translation, and shear forces [11].

2.4 Biomechanics of Lumbar Pathology

Common lumbar pathology, including stenosis, degenerative disc disease, and spondylolisthesis, results in anatomic changes that alter normal biomechanics. Furthermore, traditional surgical interventions designed to treat these conditions can themselves disrupt mechanics and lead to progressive lumbar instability. Lumbar stenosis is a progressive degenerative process defined by canal, lateral recess, and foraminal narrowing. It is frequently a result of ligamentous and osseous hypertrophy in response to pathologic motion within degenerative lumbar functional units [17]. Direct decompression of effected neural elements is the treatment of choice, including laminectomy, laminotomy, and foraminotomy.

Degenerative disc disease (DDD) is one of the most common causes of low back pain. Several interrelated degenerative changes can lead to derangement of normal biomechanics, including annular tears, endplate fissuring and ossification, and loss of disc height through decreased water content and proteoglycan degradation. These degenerative changes increase the likelihood of nucleus extrusion, decreased weight bearing by the nucleus pulposus, and shifting of force carrying onto adjacent articular surfaces and facet joints [7]. Therefore, facet joint degeneration is almost always associated with or preceded by degeneration of the intervertebral disc [9]. Furthermore, decreased hydrostatic pressure diminishes tension along the longitudinal ligaments and inner annulus, compromising the ability of the disc to resist shear forces and forward displacement [18]. The lumbar discs and vertebral bodies are wedge shaped, and decreased disc height together with osseous compression in

weakened cancellous bone yields a progressive loss of lordosis. As described above, active and passive lumbar extensors function most efficiently in physiologic lordosis. DDD not only results in pain at the overloaded facets and deranged endplate but also from progressive kyphosis and the biomechanical disadvantage of overburdened lumbar extensor musculature. Surgical restoration of disc height and reestablishment of a lordotic curve addresses these pain generators.

Spondylolisthesis describes the translational motion of one vertebral body on another. Classification includes dysplasia from a congenitally incomplete neural arch, traumatic, pathologic, isthmic, and degenerative. Pathologic and traumatic etiologies imply a localized trauma or generalized osseous condition that permits forward slip. Isthmic spondylolisthesis is the result of pars interarticularis incompetence (spondylolysis), while a degenerative etiology is associated with facet joint spondylosis and DDD [19]. The latter is associated with an increased female prevalence and most commonly occurs at the L4–5 level [6]. From a biomechanical perspective, forward flexion increases shear stress on the ventral disc, which is counteracted by extensor muscles and ligaments. The application of these force vectors places a maximum amount of stress along the pedicle and pars, ventral disc, and facet; eventual failure of one or all of these structures results in translation of one vertebral functional unit over another. Progression of spondylolisthesis is associated with disc degeneration and is highly correlated with loss of disc height [18]. Spondylolisthesis is clinically manifest most commonly by mechanical back pain, and displacement of the neural arch can lead to compression of nerve roots and the thecal sac. Surgical intervention targets decompression and fixation/fusion to arrest and possibly correct listhesis.

2.4.1 Lumbar Surgery

Many biomechanical considerations should be appreciated during a posterior approach to the lumbar spine for decompression and posterolateral fixation and fusion [20]. Extensive soft-tissue

dissection during exposure risks muscle denervation and disruption of the active stabilizing systems. Resection of the interspinous ligaments weakens the passive extensor stabilizers and diminishes their large bending moments that counteract flexion vectors. Facet capsules, associated with strong ligamentous complexes with moderate moment arms, resist flexion and are easily injured during exposure and decompression. The facet joints themselves are important for guiding and constraining motion as well as for axial load bearing. Excessive facet joint resection places increased loads on adjoining joints and discs and results in accelerated degeneration. Aggressive resection of the pars interarticularis disassociates functional units and increases shear stress on ventral structures, leading to iatrogenic spondylolisthesis (Fig. 2.6).

Because anterior column degeneration is a primary cause for spondylolisthesis, surgical management is aimed at restoration of anterior column support. Insertion of interbody spacers allows tensile strain restoration, disc height augmentation, correction of anterior column alignment, and subluxation reduction [21]. Indirect decompression of the neural foramen may also occur [22]. If posterior elements are also affected, as with isthmic spondylolysis, anterior interbody instrumentation must be followed by posterior instrumentation.

Traditional treatment options for lumbar interbody fusion include anterior lumbar interbody fusion (ALIF), transforaminal lumbar interbody fusion (TLIF), and posterior lumbar interbody fusion (PLIF). PLIF and TLIF use a posterior approach and therefore the biomechanical considerations of a posterior approach must be considered. Facet joint resection mandates posterolateral fixation and fusion to ensure stability. The ALIF approach spares the posterior elements, though sacrifices the anterior longitudinal ligament (ALL) when creating a ventral annulotomy. The ALL is a strong tension band in extension [4]. After disruption of this ligament, the PLL and dorsal elements alone must recreate this tension band effect, although placement of an anterior plate restores stability in extension [23]. Stand-alone interbody spacers risk over

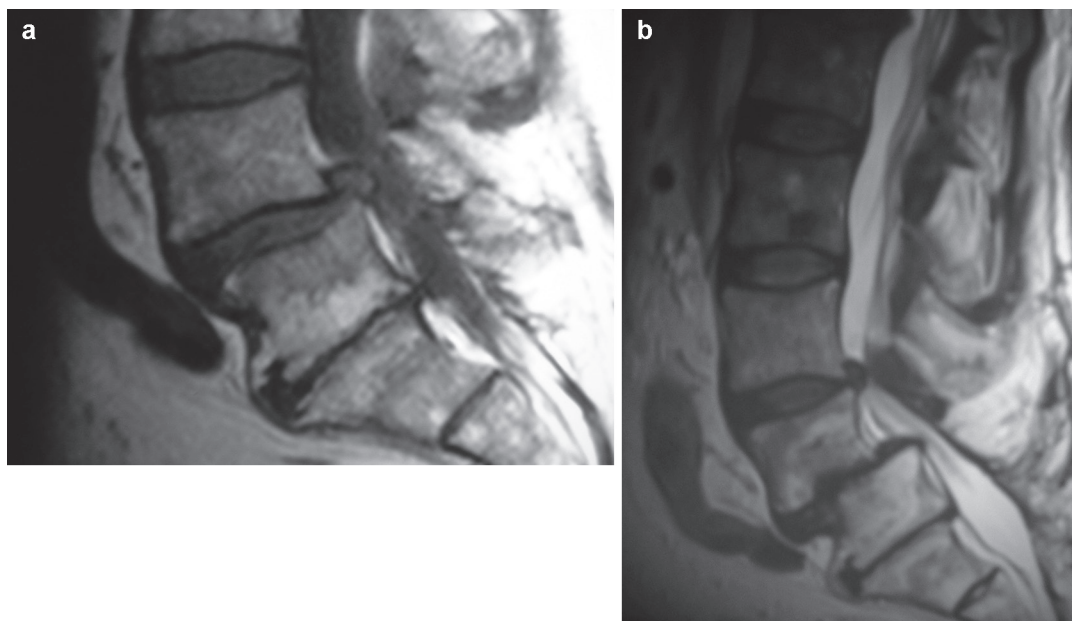


Fig. 2.6 Iatrogenic progressive L4/5 spondylolisthesis after lumbar laminectomy. (a) T1-weighted MRI prior to laminectomy; (b) T2-weighted MRI post-laminectomy

distraction of the facet joints, leading to shifting of load sharing ventrally and impaired resistance to forward flexion.

2.5 Lateral Lumbar Fixation

A lateral lumbar approach avoids many of the destructive changes that accompany anterior or posterior approaches [24] and may offer further biomechanical advantage by nature of the interbody graft and fusion. All three major tensile ligaments, including the ALL, PLL, and interspinous ligament complex, are left intact during dissection and discectomy, maintaining inherent stability. Denervation of the erector spinae muscle is circumvented, though the transpsoas technique does place temporary traction on a major lumbar flexor, the psoas muscle. The facet joint and capsule are not disturbed, thus avoiding iatrogenic instability from posterior bony element resection.

Lumbar spine biomechanics are augmented in several ways by the lateral interbody method. Placement of a large intervertebral graft with accompanying disc space distraction permits

indirect decompression of the foramen and spinal canal via ligamentotaxis and unbuckling of the PLL and ligamentum flavum. Restoration of anterior column height and tensioning of the ligaments may reduce spondylolisthesis and restore physiologic lordosis [18, 21]. Correction of sagittal kyphotic deformity places each element of the vertebral functional unit into the most efficient orientation for maintenance of stability within the neutral zone; it also may reduce the incidence of adjacent level disc degeneration [11, 15, 25, 26]. Compared to other techniques, a larger interbody cage can be used that covers the entire transverse dimension of the endplate and creates a larger area for fusion [27]. Larger implants that cover greater endplate surface area may also reduce the rate of cage subsidence and the risk of ventral loss of height [28].

Restoration of disc height and fusion with a large interbody implant may be sufficient for correction of degenerative spondylolisthesis [21]. Conversely, an incompetent posterior ligamentous or osseous complex (i.e., spondylolysis) must be supplemented with dorsal fixation in order to avoid progressive instability [18, 20]. Cage shape, bone density, and simultaneous use

of dorsal instrumentation determine compressive strength of cages used in interbody techniques [20]. Cylindrical cages provide less implant-endplate contact area than rectangular grafts and therefore resist motion to a lesser extent than rectangular grafts [27, 29]. Since larger grafts create a larger surface area for fusion and provide more stability, rectangular grafts are preferred.

As mentioned earlier, excessive disc space distraction with an oversized interbody implant risks facet joint distraction and a diminished ability to resist flexion. Care must be taken to avoid this potentially destabilizing maneuver, and the goals of indirect foraminal decompression and disc height restoration must be balanced with the integrity of the facet joints. Supplemental posterior fixation should be considered to ensure a high rate of fusion [30, 31].

Conclusions

The lumbar spine relies on a complex interplay among ligamentous, osseous, and muscular structures to coordinate physiologic motion while maintaining stability. While many degenerative conditions alter normal anatomy and disrupt spinal biomechanics, surgical interventions can themselves accelerate instability during dissection and through aggressive decompression. Lateral lumbar exposures circumvent many of the lumbar stabilizing elements, thus avoiding much of the iatrogenic biomechanical morbidity of posterior and anterior approaches. Moreover, lateral reconstruction may offer further advantage through robust disc height restoration, reestablishment of lordosis through ventral lengthening, and a large surface area for fusion across the endplates.

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