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# Biomechanics of the Spine

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## Core Messages

- ✓ The main functions of the spine are to protect the spinal cord, to provide mobility to the trunk and to transfer loads from the head and trunk to the pelvis
- ✓ The trabecular bone bears the majority of the vertical compressive loads
- ✓ The vertebral endplate plays an important role in mechanical load transfer and the transport of nutrients
- ✓ Axial disc loads are borne by hydrostatic pressurization of the nucleus pulposus, resisted by circumferential stresses in the anulus fibrosus
- ✓ Approximately 10–20% of the total fluid volume of the disc is exchanged daily
- ✓ Combined axial compression, flexion and lateral bending have been shown to cause disc prolapse
- ✓ The facet joints guide and limit intersegmental motion
- ✓ The ligaments surrounding the spine guide segmental motion and contribute to the intrinsic stability of the spine by limiting excessive motion
- ✓ The spatial distribution of muscles determines their function. Changes to segmental laxity (“neutral zone”) are associated with trauma and degeneration
- ✓ The highest loads on the spine are produced during lifting

## The Human Spine

The human spinal column is a complex structure composed of 24 individual vertebrae plus the sacrum. The principal functions of the spine are to protect the spinal cord, to provide mobility to the trunk and to transfer loads from the head and trunk to the pelvis. By nature of a natural sagittal curvature and the relatively **flexible intervertebral discs** interposed between **semi-rigid vertebrae**, the spinal column is a compliant structure which can filter out shock and vibrations before they reach the brain. The intrinsic, passive stability of the spine is provided by the discs and surrounding ligamentous structures, and supplemented by the actions of the spinal muscles. The **seven intervertebral ligaments** which span each pair of adjacent vertebrae and the two synovial joints on each vertebra (facets or zygapophyseal joints) allow controlled, fully three-dimensional motion.

The spine can be divided into **four distinct regions**: cervical, thoracic, lumbar and sacral. The cervical and lumbar spine are of greatest interest clinically, due to the substantial loading and mobility of these regions and associated high incidence of trauma and degeneration. The thoracic spine forms an integral part of the ribcage and is much less mobile due to the inherent stiffness of this structure. The sacral coccygeal region is formed by nine fused vertebrae, and articulates with the left and right ilia at the sacroiliac joints to form the pelvis.

The main functions are to protect the spinal cord, provide mobility and transfer loads

The spine can be divided into four distinct regions

## The Motion Segment

The functional spinal unit is the smallest spine segment that exhibits the typical mechanical characteristics of the entire spine

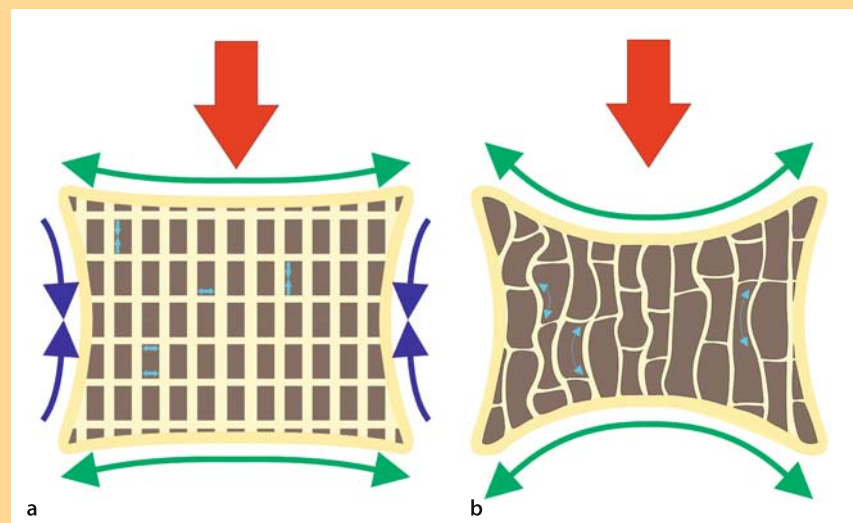
The motion segment, or **functional spinal unit**, comprises two adjacent vertebrae and the intervening soft tissues. With the exception of the C1 and C2 levels, each motion segment consists of an anterior structure, forming the vertebral column, and a complex set of posterior and lateral structures. The C1 (atlas) and C2 (axis) vertebrae, in contrast, have a highly specialized geometry which allows for an extremely wide range of motion at the junction of the head and neck (see Chapter 30). The **neural arch**, consisting of the pedicles and laminae, together with the vertebral body posterior wall form the spinal canal, a structurally significant protective structure around the spinal cord. The transverse and spinous processes provide attachment points for the **skeletal muscles**, while the right and left superior and inferior articular processes of the **facet joints** form natural kinematic constraints for the guidance of spinal intersegmental motion.

### Anterior Structures

#### The Vertebral Body

The trabecular bone bears the majority of the vertical compressive loads

The **principal biomechanical function** of the vertebral body is to support the compressive loads of the spine due to body weight and muscle forces. Correspondingly, vertebral body dimensions increase from the cervical to lumbar region. The architecture of the vertebral body comprises highly porous trabecular bone, but also a fairly dense and solid shell (Fig. 1). The shell is very thin throughout, on average only 0.35–0.5 mm [82]. The **trabecular bone** bears the



**Figure 1. Vertebral body architecture and load transfer**

**a** In the healthy vertebral body, the majority of trabeculae are oriented in the principal direction of compressive loading, with horizontal trabeculae linking and reinforcing the vertical trabecular columns. **b** With advancing osteoporosis, the thickness of individual trabeculae decreases and there is a net loss of horizontal connectivity. The consequences are an increased tendency for individual vertical trabeculae to buckle and collapse under compressive load, as the critical load for buckling of a slender column is proportional to the cross-sectional area of the column and the stiffness of the material and inversely proportional to the square of the unsupported length of the column. Therefore, architectural remodelings which lead to a loss of horizontal connecting trabeculae are perhaps the most critical age-related changes to the vertebral body.

majority of the vertical compressive loads, while the outer shell forms a reinforced structure which additionally resists torsion and shear. Previous analysis of load sharing in the vertebral body has shown that the removal of the **cortex** decreases vertebral strength by only 10% [52]. However, more recent computational analyses have proposed that the cortex and trabecular core share compressive loading in an interdependent manner. The predominant orientation of individual trabeculae is vertical, in line with the principal loading direction, while adjoining horizontal trabeculae stabilize the vertical trabecular columns. Bone loss associated with aging can lead to a loss of these horizontal tie elements, which increases the effective length of the vertical structures and can facilitate the failure of individual trabeculae by buckling.

The **vertebral endplate** forms a structural boundary between the intervertebral disc and the cancellous core of the vertebral body. Comprising a thin layer of semi-porous subchondral bone, approximately 0.5 mm thick, the **principal functions** of the endplate are to prevent extrusion of the disc into the porous vertebral body, and to evenly distribute load to the vertebral body. With its dense cartilage layer, the endplate also serves as a **semi-permeable membrane**, which allows the transfer of water and solutes but prevents the loss of large proteoglycan molecules from the disc. The local material properties of the endplate demonstrate a significant spatial dependence [33]. The vertebral endplate and underlying trabecular bone together form a non-rigid system which demonstrates a significant deflection under compressive loading of up to 0.5 mm [16].

The endplate has been shown to be the weak link in maintaining vertebral body integrity, especially with decreasing bone density, as the heterogeneity of endplate strength is even more pronounced [34]. High compressive loads lead to **endplate failure** due to pressurization of the nucleus pulposus. Nuclear material is often extruded into the adjacent vertebral body following fracture (Schmorl's nodes), thereby establishing a possible source of pain from increased intraosseous pressure [101].

**Vertebral strengths** as measured from in vitro tests on cadaver specimens vary by an order of magnitude (0.8–15.0 kN) [38, 98] due to the natural variation in bone density, bone architecture and vertebral body geometry. A strong correlation has been demonstrated between quantitative volumetric bone density and vertebral strength [17]. Vertebral geometry and structure are equally important factors for the determination of vertebral strength [21]. The increase in vertebral strength caudally is mostly due to the increased vertebral body size, as bone density is fairly constant between individual vertebral levels. The **fatigue life** of vertebrae, the resistance to failure during repetitive loading, depends on the magnitude and duration of compressive loading. Brinckmann et al. [15] have documented in vitro measurements of the fatigue strength of vertebrae which provide valuable information for predicting fracture risks in vivo or specifying safe activity levels (Table 1).

Removal of the cortex decreases vertebral strength by only 10%

The vertebral endplate is important for mechanical load transfer and nutrient transport

The endplate is often the initial site of vertebral body failure

Vertebral body geometry, bone density and architecture determine vertebral strength

Table 1. Fatigue strength of vertebrae

Probability of failure Load	Loading cycles				
	10	100	500	1000	5000
% VCS	10	100	500	1000	5000
30–40%	0%	0%	21%	21%	36%
40–50%	0	38	56	56	67
50–60%	0	45	64	82	91
60–70%	8	62	76	84	92

VCS signifies vertebral compressive strength; 5000 cycles of loading is approximately equivalent to 2 weeks of athletic training

### The Intervertebral Disc

The disc consists of a gel-like nucleus surrounded by a fiber-reinforced anulus

The intervertebral disc is the **largest avascular structure** of the body. The disc transfers and distributes loading through the anterior column and limits motion of the intervertebral joint. The disc must withstand significant compressive loads from body weight and muscle activity, and bending and twisting forces generated over the full range of spinal mobility. The disc is a specialized structure with a heterogenous morphology consisting of an inner, **gelatinous nucleus pulposus** and an outer, **fibrous anulus**. The nucleus pulposus consists of a hydrophilic, **proteoglycan** rich gel in a loosely woven collagen gel. The nucleus is characterized by its ability to bind **water** and swell. The anulus fibrosus is a lamellar structure, consisting of 15–26 distinct concentric fibrocartilage layers with a criss-crossing fiber structure [50]. The **fiber orientation** alternates in successive layers, with fibers oriented at 30° from the mid-disc plane and 120° between adjacent fiber layers. From the outside of the anulus to the inside, the concentration of **Type I collagen** decreases and the concentration of **Type II collagen** increases [27], and consequently there is a regional variation in the mechanical properties of the anulus [12, 83].

Axial disc loads are borne by hydrostatic pressurization of the nucleus pulposus, resisted by circumferential stresses in the anulus fibrosus

The intervertebral disc is loaded in a complex combination of compression, bending, and torsion. Bending and torsion loads are resisted by the strong, oriented fiber bundles of the anulus. In the healthy disc, axial loads are borne by hydrostatic pressurization of the nucleus pulposus, resisted by circumferential stresses in the anulus fibrosus [62], analogous to the function of a pneumatic tyre (Fig. 2). Pressure within the nucleus is approximately 1.5 times the externally applied load per unit disc area. As the nucleus is incompressible, the **disc bulges** under load – approximately 1 mm for physiological loads [85] – and considerable tensile stresses are generated in the anulus. The stress in the anulus fibers is approximately 4–5 times the applied stress in the nucleus [31, 61, 62]. Anulus fibers elongate by up to 9% during torsional loading, still well below the ultimate elongation at failure of over 25% [84].

Approximately 10–20% of the disc's total fluid volume is exchanged daily, resembling a "pumping effect"

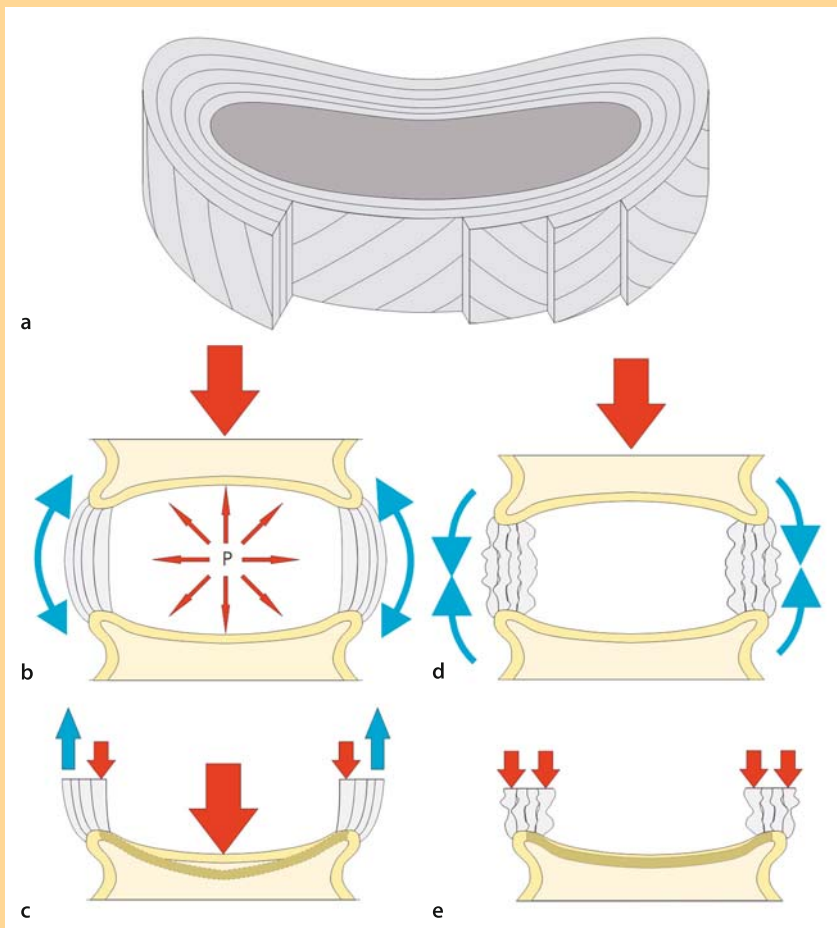
Compressive forces and pretension in the longitudinal ligaments and anulus are balanced by an **osmotic swelling pressure** in the nucleus pulposus, which is proportional to the concentration of the **hydrophilic proteoglycans** [93]. Proteoglycan content and disc hydration decreases with age due to degenerative processes. The intrinsic swelling pressure of the unloaded disc is approximately 10 N/cm<sup>2</sup>, or 0.1 MPa [61]. As the applied force increases above this base level, disc hydration decreases as water is expressed from the disc [3, 49] and consequently the net concentration of proteoglycans increases. The rate of fluid expression is slow, due to the low intrinsic permeability of the disc [39]. A net daily fluid loss of approximately 10–20% has been observed in vivo and in vitro [49, 55]. Fluid lost during daily loading is regained overnight during rest, and it has been postulated that this **diurnal fluid exchange** is critical for disc nutrition [30].

Disc degeneration substantially alters load transfer

Disc degeneration have a profound effect on the mechanism of load transfer through the disc. With degeneration, dehydration of the disc leads to a lower elasticity and viscoelasticity. Loads are less evenly distributed, and the capacity of the disc to store and dissipate energy decreases. Using the technique of "**stress profilometry**", it has been shown that age-related changes to the disc composition result in a shift of load from the nucleus to the anulus [5, 6, 56]. Therefore, structural changes in the anulus and endplate with degeneration may lead to a transfer of load from the nucleus to the **posterior anulus**, which may cause pain and also lead to annular rupture.

Degeneration exposes the posterior anulus to a high failure risk

The mechanical response of the disc to complex loading has been well described. The response of the disc to compressive loading is characterized by



**Figure 2. Load transfer in normal and degenerated discs**

**a** The intervertebral disc consists of a gel-like nucleus surrounded by a fibrous annulus consisting of multiple concentric lamellae. **b** In the healthy disc (*left*), compressive loads create a hydrostatic pressure within the fluid nucleus, which is resisted by tensile stresses in the outer annulus. **c** Loads are transferred through the central portion of the vertebral endplate, causing substantial deflection of the endplate (up to 0.5 mm). **d, e** In the degenerated disc, the nucleus is dehydrated and compressive loads are transferred by compressive stresses in the annulus. This may lead to an inward bulge of the inner annulus, buckling of the lamellae and cleft formation. Endplate loading is reduced, as stresses are transferred through the stronger and stiffer outer endplate region.

flexibility at low loads and increasing stiffness at high loads [98]. Likewise, a highly non-linear response of disc to torsion has been demonstrated [28]. Very little torque is required for the first 0–3° of rotation, between 3° and 12° rotation there is a linear relationship between torque and rotation and failure of the annulus fibers occurs at a rotation of more than 20° rotation. Measurements of **internal disc displacements** during loading [80, 90] have shown a characteristic motion of the nucleus away from the direction of applied bending load (e.g. a posterior shift of the nucleus during flexion).

Nucleus pressurization and displacement results in heterogeneous disc bulging. Posterior disc bulging is greatest during extension and least during flexion, which has implications for the most common disc injury, disc protrusion and prolapse. **Extrusion of nuclear material** through the annulus usually occurs in the **posterolateral direction** and can cause compression of the dura and/or nerve

The nucleus shifts depending on the loading direction

Nucleus extrusion usually occurs posterolaterally

Combined axial compression, flexion and lateral bending have been shown to cause disc prolapse

roots. It has been postulated that this is due to fatigue failure of inner annulus fibers [2, 4], as fissures in the annulus allow the expression of nuclear material under pressure. While pure compressive loading does not cause herniation, even at high loads and with deliberate annulus injury [95], combined axial compression, flexion and lateral bending have been shown to cause prolapse [1], loading conditions which result in a 50% increase in posterior annulus deformation and a considerable increase in nuclear pressure.

### Posterior Elements

The facet joints guide and limit intersegmental motion

The posterior elements guide the motion of the spinal segments and limit the extent of torsion and anterior-posterior shear. The transverse and spinous processes are the important attachment points for the ligaments and muscles which initiate spine motion and which are exceptionally important for stability [47]. The **orientation of the facet joints** is of key importance for guiding spinal kinematics. The three-dimensional orientation of the facets changes along the spine from cervical to sacral [70] (Table 2). **Facet asymmetry** is observed in approximately 25% of the population [98] with an average asymmetry, or facet tropism, of 10° (maximum 42°). With tropism, compression and shear loading can lead to an induced rotation towards the more oblique facet [22].

Deformity of the facets or fracture of the pars interarticularis compromises segmental shear resistance

**Load sharing in the facet joints** can be measured directly [25, 46] or calculated with mechanical models [57, 81, 100]. In hyperextension, approximately 30% of the load is transmitted through the facets. In an upright standing position, 10–20% of the compressive load is carried by the facets. The facet joints resist more than 50% of the anterior shear load in a forward flexed position, up to 2000 N without failure [23]. If this capacity to resist shear is compromised (e.g. by genetic malformation of the facets, stress fractures of the pars interarticularis, facet tropism) an anterior slip of one vertebra relative to the adjacent vertebra can occur. **Isthmic spondylolisthesis** is most prevalent at L5–S1 and degenerative spondylolisthesis of L4–L5 has been associated with the predominantly sagittal orientation of the facets [36]. During torsion, the contralateral facet is heavily loaded. Facet joint pressure is also influenced by disc height: a 1-mm decrease in disc height results in a 36% increase in facet pressure; a 4-mm decrease in disc height a 61% increase in facet joint pressure [24]. Due to the innervation of the facet capsules, there is therefore the potential for disc degeneration to cause facet joint pain.

Table 2. Facet joint orientation and functional significance

Spine region	Facet orientation	Consequence
C1–C2	Parallel to transverse	Substantial rotation
Cervical	45° to transverse Parallel to frontal	Flexion, extension and rotation Substantial motion coupling
Thoracic	60° to transverse 20° to frontal	Lateral bending, rotation Limited flexion and extension
Lumbar	45° to frontal Parallel to sagittal	Flexion, extension and lateral bending Negligible rotation
Lumbosacral	Oblique	Substantial rotation

Data derived from [70]



## Ligaments of the Spine

The ligaments surrounding the spine guide segmental motion and contribute to the intrinsic stability of the spine by limiting excessive motion. There are two primary ligament systems in the spine, the intrasegmental and intersegmental systems. The **intrasegmental system** holds individual vertebrae together, and consists of the ligamentum flavum, facet capsule, and interspinous and intertransverse ligaments. The **intersegmental system** holds many vertebrae together and includes the anterior and posterior longitudinal ligaments, and the supraspinous ligaments. All ligaments except the ligamentum flavum have a high collagen content. The **ligamentum flavum**, connecting two adjacent neural arches, has a high elastin content, is always under tension and pre-stresses the disc even in the neutral position [26].

The properties of lumbar ligaments have been most extensively studied (Table 3). **Tensile properties** have been reported for the ligamentum flavum [26], anterior longitudinal and posterior longitudinal [88], inter- and supraspinous [97] and intertransverse ligaments [20]. The response to tensile loading is typically non-linear, with an initial low stiffness **neutral zone**, an **elastic zone** with a linear relationship between load and displacement, followed by a plastic zone where permanent non-recoverable deformation of the ligament occurs. The neutral zone plus the elastic zone represent the physiological range of deformation. **Physiological strain levels** in ligaments have been determined by conducting in vitro tests on cadaveric specimens, using motion extents determined from radiographic in vivo measurements of spinal motion [69]:

- flexion: supraspinous, 30%; interspinous, 27%; posterior longitudinal, 13%
- extension: anterior longitudinal, 13%
- rotation: capsular ligaments, 17%

The **functional role** of individual ligaments and the relative contribution of each to overall segmental stability can be determined in vitro by repetitive loading and sequential sectioning of individual anatomical structures [71]. During flexion, the ligamentum flavum, capsular ligaments and interspinous ligaments are highly strained. During extension, the anterior longitudinal ligament is loaded. During side bending, the contralateral transverse ligaments, the ligamentum flavum and the capsular ligaments are tensioned, whereas rotation is resisted by the capsular ligaments [69]. A larger relative distance between individual ligaments and the rotation center of the intervertebral joint corresponds with a greater stabilizing potential.

The ligaments guide segmental motion and contribute to the intrinsic stability by limiting excessive motion

Ligament response to load is non-linear: initially flexible neutral zone and subsequent stiffening

The ligaments resist various spinal movements

Table 3. Typical values for lumbar ligament strength and stiffness

Ligament	Failure load (N)	Failure strain (% elongation)
Anterior longitudinal	450	26%
Posterior longitudinal	324	26%
Ligamentum flavum	285	26%
Interspinous	125	13%
Supraspinous	150	32%

Data derived from [20, 98]

## Motion Segment Stiffness

Degenerations and injury alter spinal stiffness

In vitro testing of cadaveric specimens has been performed to determine the intrinsic functional stiffness of spinal motion segments. In general, the **functional stiffness** is adapted to the loading which each spine segment experiences. Degeneration and/or injury can have a significant influence on stiffness. Typical stiffness values are as follows [11, 54, 58, 68, 79]:

- cervical spine: lateral shear 33 N/mm, compression 1 317 N/mm
- thoracic spine: lateral shear 100 N/mm, anterior posterior shear 900 N/mm, compression 1 250 N/mm
- lumbar spine: shear 100–200 N/mm; compression 600–700 N/mm
- sacroiliac joint: shear, 100–300 N/mm

Posterior elements contribute significantly to overall segmental stiffness

**Muscle forces** can significantly alter the mechanical response of the spine. Compressive preload leads to a significant stiffening of the spinal motion segment [40].

At the sacroiliac joint, coordinated activity of the pelvic, trunk and hip muscles creates a medially oriented force which locks the articular surfaces of the sacroiliac joints and the pubic symphysis, stiffening the pelvis [96]. The posterior elements contribute significantly to the overall stiffness of the motion segment. **Removal of posterior elements** in sequential testing in vitro produced a 1.7 times increase in shear translation, a 2.1 times increase in bending displacement and a 2.7 times increase in torsion [54].

Trunk muscles stabilize the spine and redistribute loads

The spine is an elastic column, with enhanced stability due to the complex curvature of the spine (kyphosis and lordosis), the support of the longitudinal ligaments, the elasticity of the ligamentum flavum, and most importantly the active muscle forces. While cadaver spines have been shown to buckle with the application of very low vertical loads (20–40 N) [35], the **extrinsic support** provided by trunk muscles stabilizes and redistributes loading on the spine and allows the spine to withstand loads of several times body weight.

## Muscles

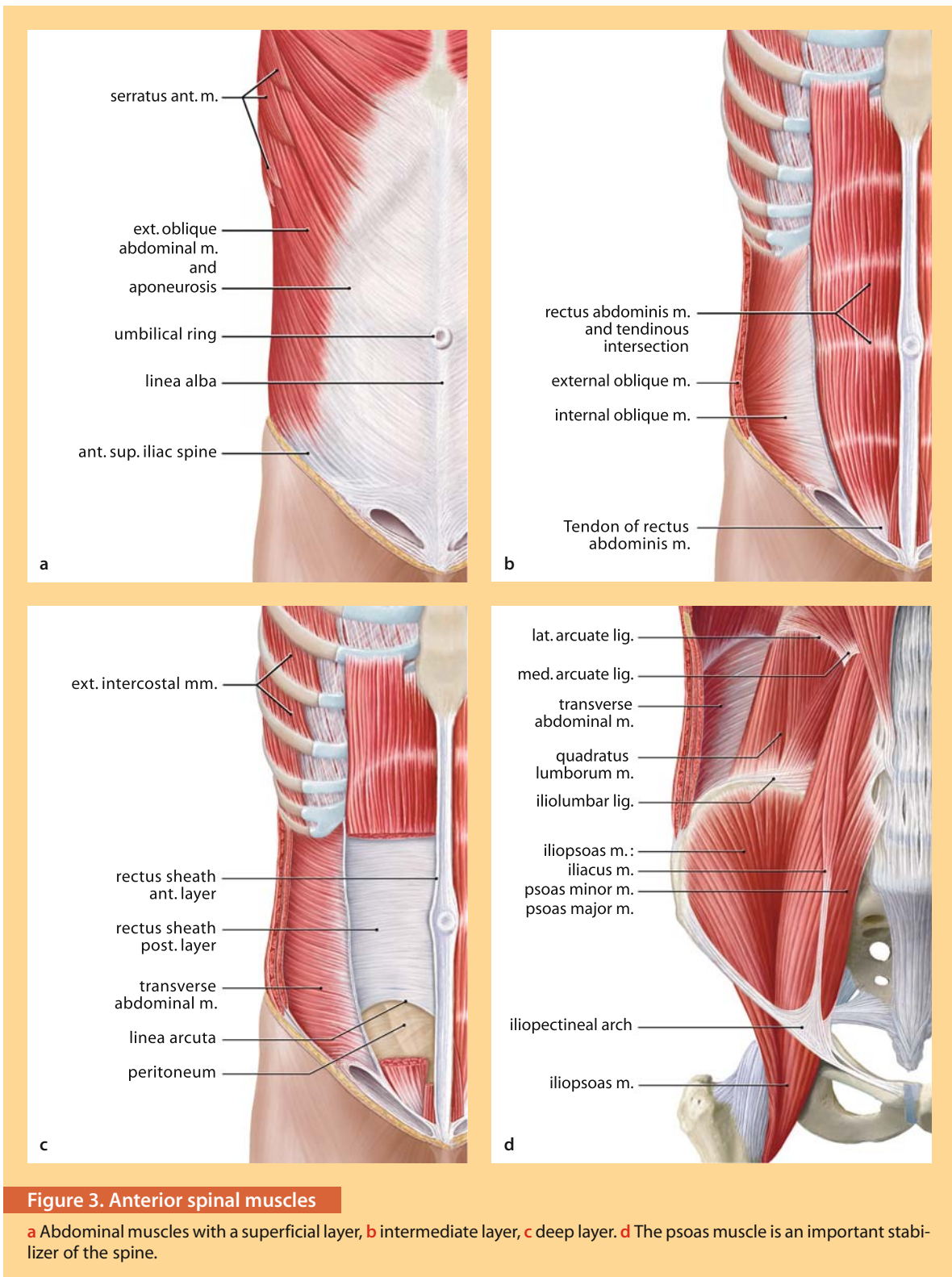
The spatial distribution of muscles determines their function

The spatial distribution of muscles generally determines their function. The trunk musculature can be divided functionally into extensors and flexors. The **main flexors** are the abdominal muscles (rectus abdominis, internal and external oblique, and transverse abdominal muscle) and the psoas muscles (**Fig. 3**).

The trunk musculature can be divided functionally into extensors and flexors

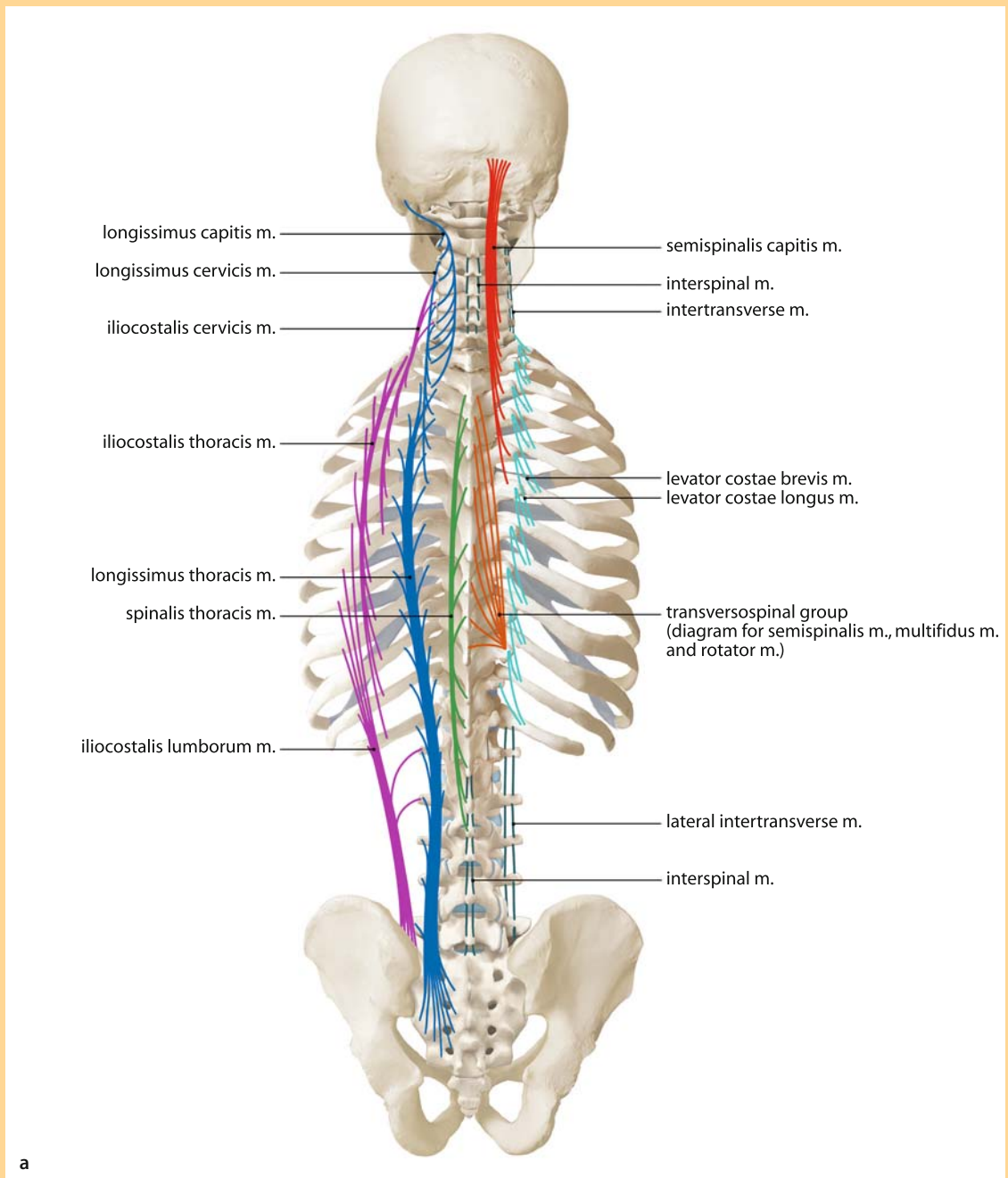
The **main extensors** are the sacrospinalis group, transversospinal group, and short back muscle group (**Fig. 4**). Symmetric contraction of extensor muscles produces extension of the spine, while asymmetric contraction induces lateral bending or twisting [8]. The most **superficial layer** of trunk muscles on the posterior and lateral walls are broad, connecting to the shoulder blades, head and upper extremities (rhomboids, latissimus dorsi, pectoralis, trapezius) (**Fig. 5**). Some lower trunk muscles connect to a strong superficial fascial sheet, the **lum-bodorsal fascia**, which is a tensile-bearing structure attached to the upper borders of the pelvis (e.g. transversus abdominis) [13]. The iliopsoas muscle originates on the anterior aspect of the lumbar spine and passes over the hip joint to the inside of the femur. Vertebral muscle is composed of 50–60% **type I muscle fibers**, the so-called “**slow twitch**”, fatigue-resistant muscle fibers found in most postural muscles [9].





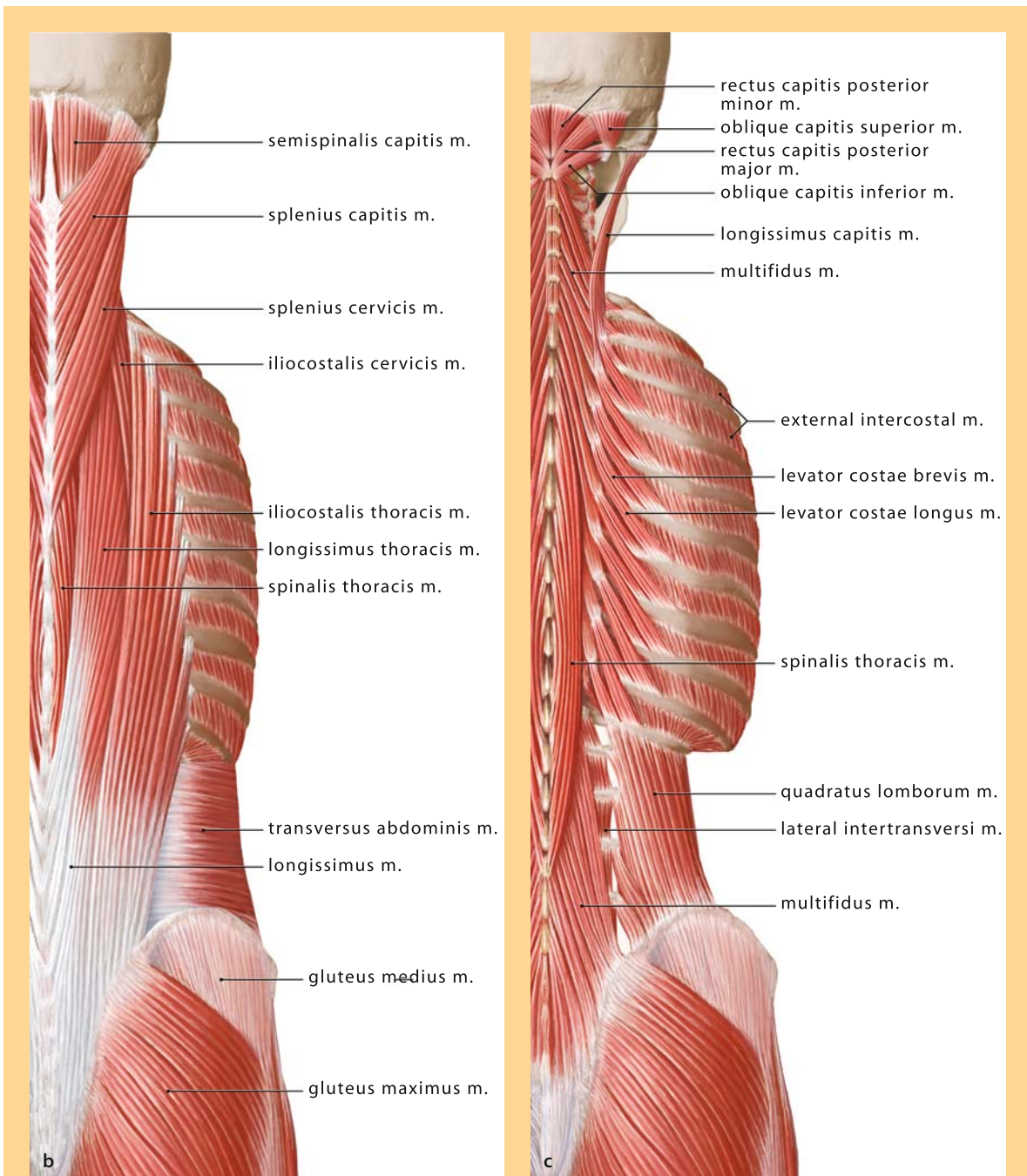
**Figure 3. Anterior spinal muscles**

**a** Abdominal muscles with a superficial layer, **b** intermediate layer, **c** deep layer. **d** The psoas muscle is an important stabilizer of the spine.



**Figure 4. Deep muscles of the back**

**a** The deep muscles of the back can be separated into the sacrospinalis (erector spinae) group (left side), the transversospinal group (right side), and the short back muscles group. The sacrospinalis group consists of the iliocostalis muscles, longissimus muscles and spinalis muscles. The transversospinal group consists of semispinalis muscles, multifidus muscles and the rotator muscles. The short back muscle group consists of the intertransverse and interspinal muscles.



**Figure 4. (Cont.)**

**b, c** The spatial distribution of the deep spinal muscles determines their function. **c** The suboccipital muscles consist of rectus capitis posterior major muscle, rectus capitis posterior minor muscle, oblique capitis superior muscles, and oblique capitis inferior muscle.



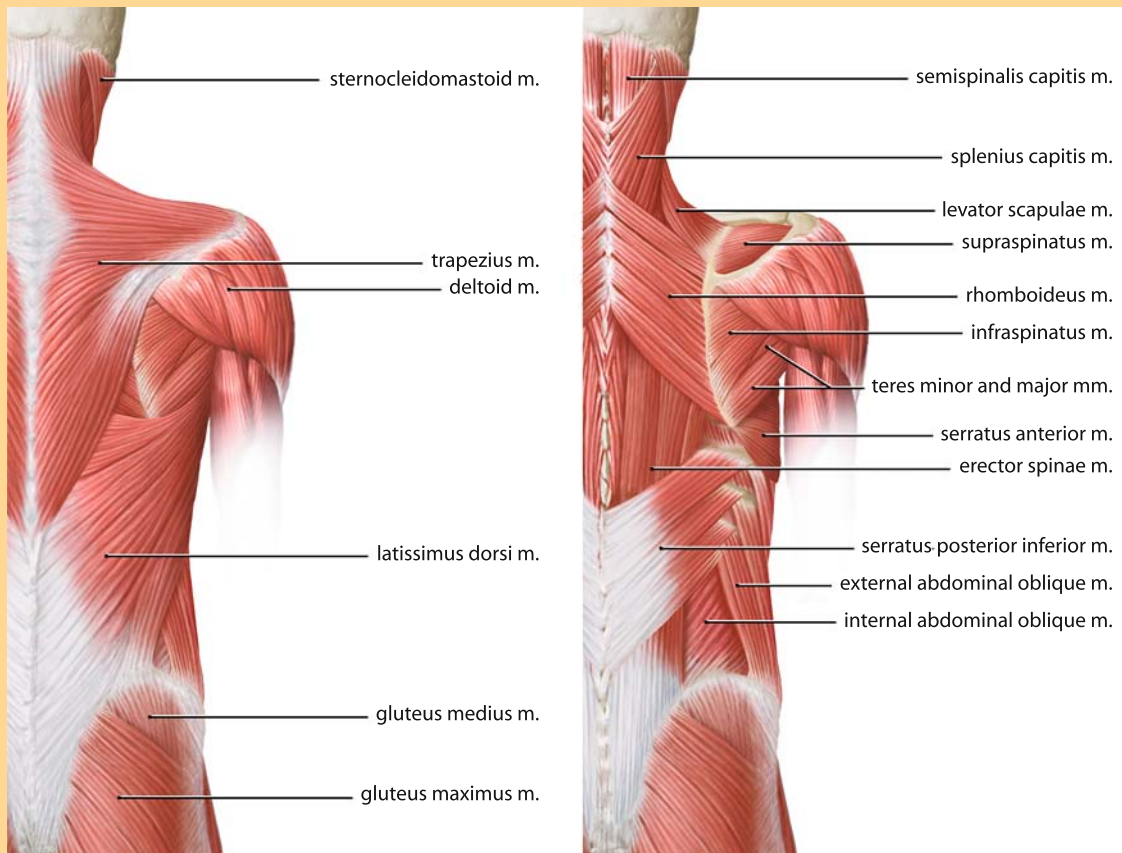


Figure 5. Superficial muscles of the back

The geometric relationship between the muscle line of action and the intervertebral center of rotation determines the functional potential

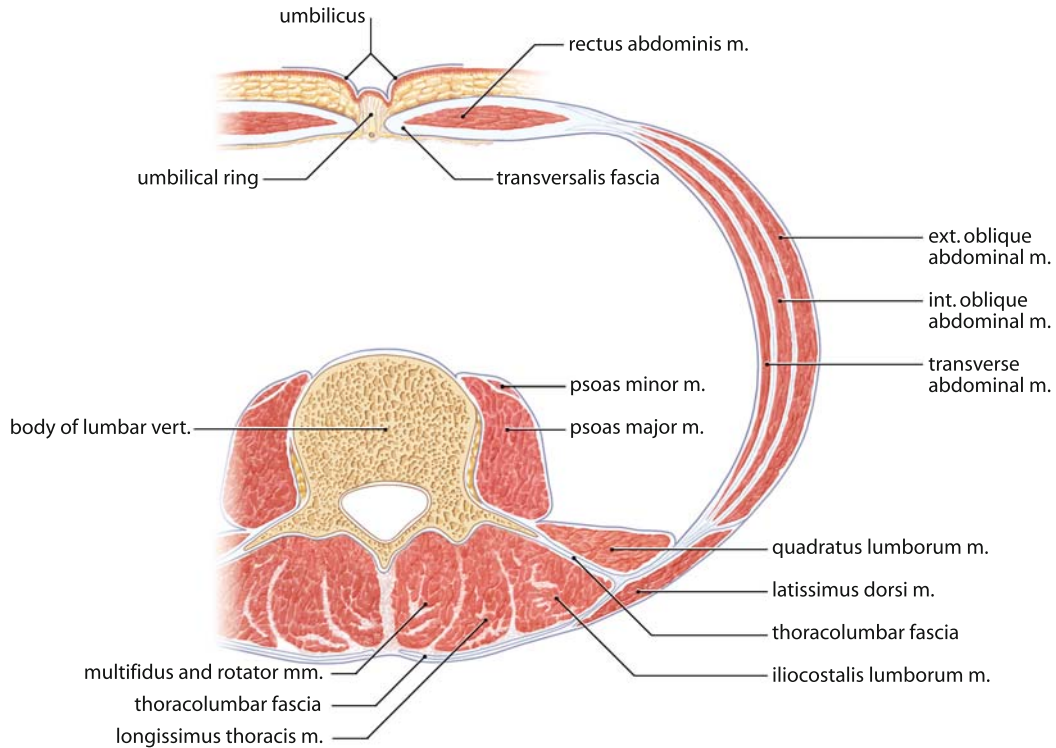
**Spinal muscle activity** can be determined by direct electromyographic measurement or by using mathematical models of the spine, which include a detailed description of the origin and insertion points of muscles, muscle cross sections, muscle fiber length and muscle type. Of particular importance is the geometric relationship of the muscle line of action to the rotation center of the joint in consideration (the moment arm: larger moment arm → greater potential to produce torque). **Moment arms** for cervical and lumbar spine muscles have been determined from MR and CT images [53, 64, 89, 91]. Detailed descriptions of the anatomy of spinal muscles have been published, which include the variation in moment arm length resulting from changing posture [14, 48, 65, 92]. Owing to the large number of muscles, the inherent redundancy, and the possibility for muscular co-contraction, the calculation of muscle activity with mathematical models often requires the use of additional formulae which consider optimal muscle stress levels or maximum contraction forces to obtain a unique solution.

### Spinal Stability Through Muscular Activity

Spine stability is enhanced by the activity of the transverse abdominis, multifidus and psoas muscles

The **muscular system** can also be divided into three functional groups [10]:

- local stabilizers
- global stabilizers
- global mobilizers



**Figure 6. Interplay of anterior and posterior spinal muscles**

The transverse abdominis, the deep lumbar multifidus and the psoas are among the local stabilizing muscles best suited to control the neutral zone in the lumbar spine. The transverse abdominis attaches directly to the lumbar spine and stiffens the spine by creating an extensor moment on the lumbar spine and by creating pressure on the anterior aspect of the spine (intra-abdominal pressure), resisting collapse of the natural curvature of the spine. The multifidus attaches directly to each segment of the lumbar spine and intrinsically stiffens the intervertebral joint by direct contraction. The psoas' prime fiber orientation on the anterior aspect of the vertebrae facilitates spinal stabilization.

**Local stabilizers** (Fig. 6) attach directly to the lumbar spine, usually spanning single spinal segments, and control the neutral position of the intervertebral joint. Examples of local stabilizers are the transverse abdominis, the deep lumbar multifidus and the psoas. **Local stabilizers** operate at low loads and do not induce motion, but rather serve to stiffen the spinal segment and control motion. A dysfunction of the local stabilizer can result in poor segmental control and pain due to abnormal motion. The **global muscle** system comprises the larger torque-producing muscles which contract concentrically or eccentrically to produce and control movement. Contraction of these muscles can also enhance spinal rigidity. Examples of global muscles are the oblique abdominis, rectus abdominis and erector spinae (spinalis, longissimus and iliocostalis). Although global muscles are traditionally targeted for treating patients with low back pain, there is compelling evidence that retraining of the local stability system may be most beneficial. **Clinical instability** has been defined as a significant decrease in the ability to maintain the intervertebral neutral zone within physiological limits [67], and the muscles best suited to control the neutral zone in the lumbar spine are the transverse abdominis, the deep lumbar multifidus and the psoas [41]. The transverse abdominis attaches directly to the lumbar spine via the lumbodorsal fascia and

Training of local stabilizers improves spinal stability

The psoas is an important spine stabilizer

stiffens the spine by inducing an extensor moment on the lumbar spine and by creating pressure on the anterior aspect of the spine (intra-abdominal pressure), resisting collapse of the natural curvature of the spine. The **multifidus** attaches directly to each segment of the lumbar spine and intrinsically stiffens the intervertebral joint by direct contraction. The **psoas** has been described functionally as a hip flexor. However, the presence of multiple fascicles of the psoas attaching to the individual lumbar vertebrae, and the predominant fiber orientation on the anterior aspect of the vertebrae, facilitate its function as a spine stabilizer [74].

### Muscle Activity During Flexion and Extension

Flexion is achieved through the forward weight shift of the upper body and controlled by compensatory activity of the extensor muscles

Due to the nearly oblique configuration of thoracic facets and the intrinsic stiffness of the ribcage, the majority of spine flexion and extension occurs in the lumbar spine, augmented by pelvic tilt [19, 29]. **Flexion** is initiated by the abdominal muscles and the vertebral portion of the psoas. Additional flexion is achieved through the weight shift of the upper body, which induces an increasing forward bending moment, and is controlled by compensatory activity of the extensor muscles. Posterior hip muscles control the forward tilting of the pelvis. In full flexion, it has been proposed that the forward bending moment is counteracted passively by the elasticity of the muscles and posterior ligaments of the spine, which are initially slack but progressively tightened as the spine flexes [29]. However, more recent studies with measurements of muscle activity have shown that deep lateral lumbar erector spinae muscles are still active in full flexion [7], perhaps for stabilization. During **hyperextension** from upright, extensor muscles are active to initiate the motion, but as extension progresses, the shifting body weight is sufficient to produce a backward bending moment which is modulated by increasing activity of the abdominal muscles.

### Muscle Activity During Lateral Flexion and Rotation

**Lateral flexion** of the trunk can occur in the lumbar and thoracic spine. The spinotransversal and transversospinal systems of the erector spinae muscles and the abdominal muscles are active during lateral bending. Ipsilateral contractions initiate the motion and contralateral contractions control the progression of bending [8]. During **axial rotation**, the back and abdominal muscles are active, and both ipsilateral and contralateral contractions contribute to the motion. High degrees of coactivation have been measured during axial rotation, perhaps due to the suboptimal muscle lines of action for this motion [44].

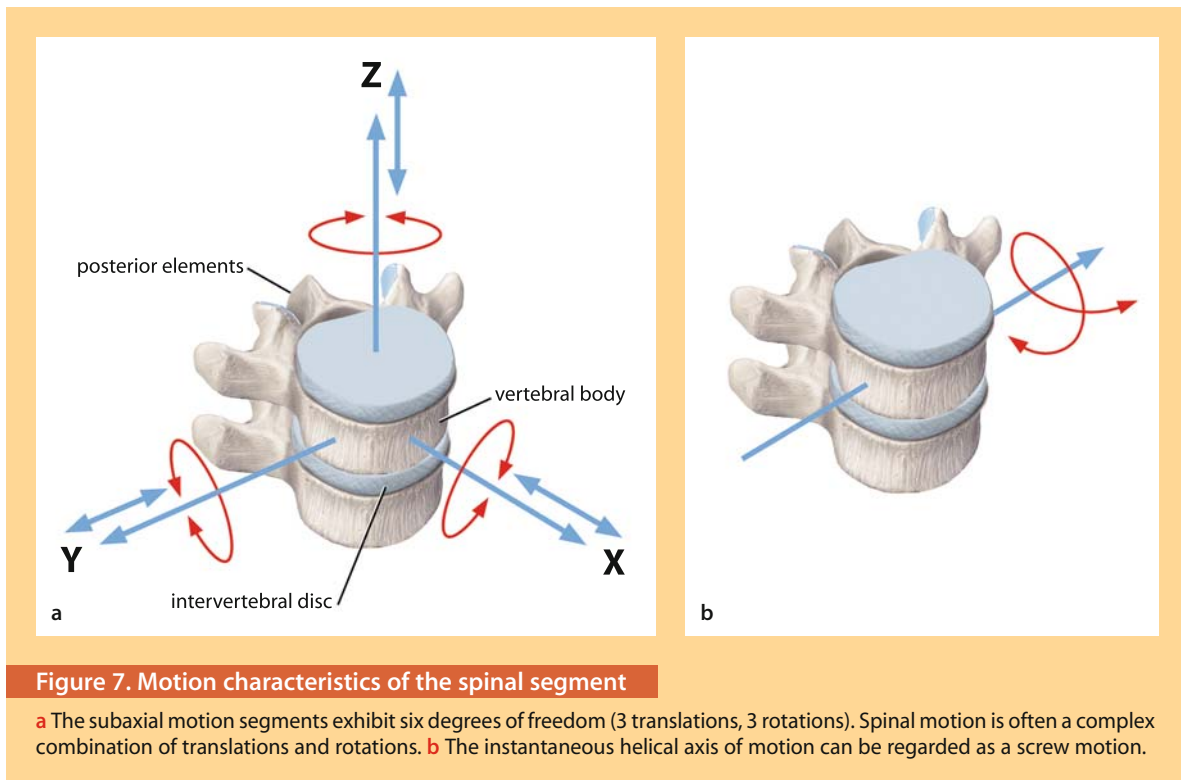
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## Spine Kinematics

The sum of limited motion at each segment creates considerable spinal mobility in all planes

The spine provides mobility to the trunk. Only limited movements are possible between adjacent vertebrae, but the sum of these movements amounts to considerable spinal mobility in all anatomical planes. The range of motion differs at various levels of the spine and depends on the structural properties of the disc and ligaments and the orientation of the facet joints. Motion at the intervertebral joint has **six degrees of freedom**: rotation about and translation along the inferior-superior, medial-lateral and anterior-posterior axis (**Fig. 7a**). Spinal motion is often a complex, combined motion of simultaneous flexion or extension, side bending and rotation.





**Figure 7. Motion characteristics of the spinal segment**

**a** The subaxial motion segments exhibit six degrees of freedom (3 translations, 3 rotations). Spinal motion is often a complex combination of translations and rotations. **b** The instantaneous helical axis of motion can be regarded as a screw motion.

## Range of Motion

Spinal kinematics and spinal range of motion can be determined *in vivo* using, e.g. surface markers, goniometers, pantographs, or computerized digitizers. While these methods are adequate for postural measurements, they lack the accuracy required for intersegmental motion measurement [51, 76]. More reliable *in vivo* radiographic and *in vitro* cadaveric measurements have been performed to determine the average range of motion for various levels of the spine [43, 72, 73]. **Intersegmental range of motion** is site specific, determined by local anatomical geometry and functional demands (Fig. 8).

Intersegmental motion is site specific

## Mechanical Response of the Spinal Motion Segment

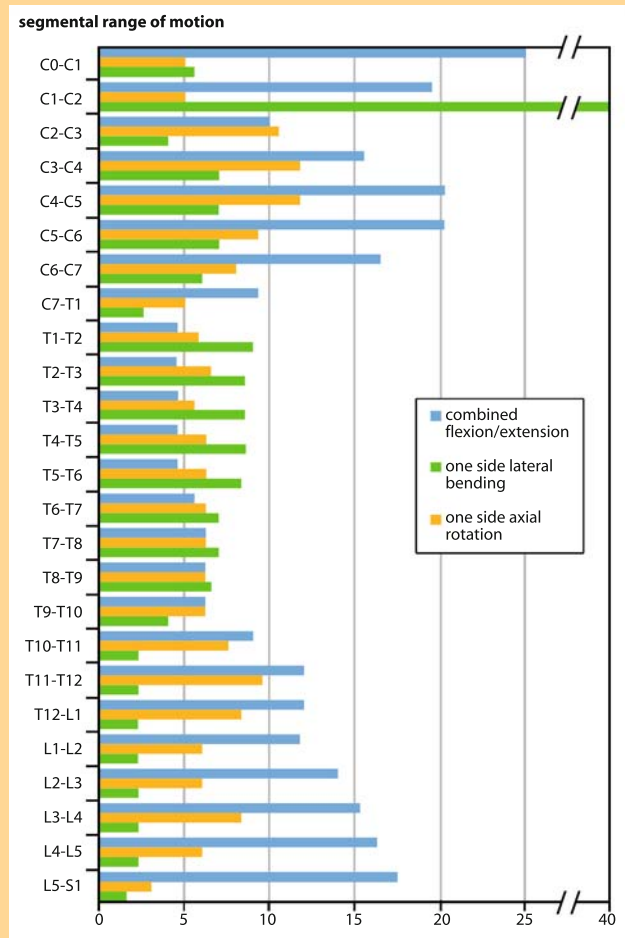
A common method for measuring and expressing the complex structural properties and motion of the spinal segment is through three-dimensional flexibility testing. **Flexibility** is the ability of a structure to deform under the application of a load. The mechanical response of the spine is typically determined by applying pure bending moments, with or without the addition of an axial compressive preload, in each of the three physiological directions of flexion-extension, lateral bending and axial rotation, and recording the overall principal and coupled motion of the specimen. Measuring the flexibility of individual functional spinal units or multisegment spine segments, i.e. the total motion achieved for a given load, is somewhat analogous to the clinical concepts of range of motion and spinal instability. The **load-displacement curve** of the spine is generally non-linear. For small loads, displacements are relatively large due to ligament and intervertebral disc laxity about the neutral position of the spine. At higher loads, the resistance to deformation increases substantially. The overall motion in the low load region of the response curve has been termed the neutral zone and is a quantitative measure of joint laxity around the neutral position. The displacement

For small loads displacements are relatively large due to ligament and disc laxity about the neutral position

The load-displacement curve of the spine is non-linear

**Figure 8. Average segmental range of spinal motion**

Intersegmental range of motion is site specific, determined by local anatomical geometry and functional demands. The extensive mobility of the cervical spine in all anatomical directions is apparent. The specific geometry of the C1–C2 joint can be recognized by the substantial rotation at this level. Motion in the thoracic spine is limited by the stiffening effect of the ribcage. In the lumbar spine, substantial flexion–extension motion is possible, but rotation is limited by the geometry of the facet joints. Summarized from [98].



Changes to the neutral zone are associated with trauma and degeneration and resemble clinical instability

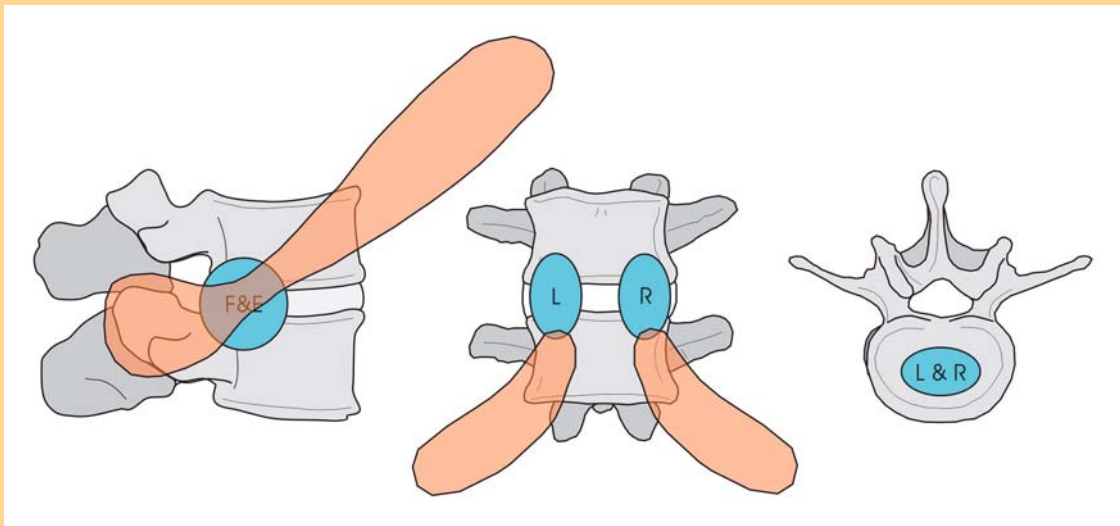
beyond the **neutral zone** and up to the maximum physiological limit has been termed the **elastic zone**. The sum of the neutral zone and elastic zone provides the total physiological range of motion of the spine. Flexibility coefficients for the spine reported in the literature are generally calculated from the elastic zone of the response curve (**Table 4**).

The neutral zone is a parameter that correlates well with other signs indicative of **instability of the spine**. The extent of the neutral zone increases following disc degeneration [98], surgical injury (e.g. facetectomy), high speed trauma [66] and repetitive cyclic loading [45]. Together, the neutral zone and total range of motion provide a quantitative measure of normal segmental motion, hypermobility due to injury or degeneration, or the relative merits of stabilizing implants or interventions.

**Table 4. Typical average flexibility coefficients of the functional spinal unit**

Region	Flexion	Extension	Lateral bending	Rotation
Cervical	2.33°/Nm	1.37	1.47	0.86
Thoracic	0.45	0.36	0.36	0.40
Lumbar	0.74	0.48	0.57	0.20
Lumbosacral	1.00	0.78	0.13	0.55

Data derived from in vitro testing [11, 54, 58, 68, 79, 86, 87]



**Figure 9. Typical instant center of lumbar rotation**

For planar motion, there is a unique instant center of rotation which fully describes the motion between two adjacent vertebrae. For the healthy spine segment, the center of rotation generally lies within the intervertebral disc. With degeneration, segmental instability can result in a significant alteration of the motion patterns of the spine. Changes to the instant center of rotation may have consequences for the loading of peripheral structures of the spine. As determined from *in vitro* and *in vivo* spinal motion analysis studies [41, 69, 70, 98].

Quantitative measurements of the extent of motion only partially describe spinal kinematics. A common simplification for the analysis of spinal kinematics is to consider the motion only in a single principal plane (e.g. flexion-extension). For planar motion, there is a unique **instant center of rotation** which fully describes the motion between two adjacent vertebrae (Fig. 9). The instant center of rotation generally lies within the disc space for healthy spines, but with disc degeneration the center of rotation pathway can be significantly altered [32]. With improvement in dynamic, *in vivo* methods for measuring spinal kinematics, a detailed analysis of the instant center of rotation and its variations may provide a tool for diagnosing particular pathological conditions of the spine. Furthermore, a complete knowledge of the normal motion characteristics of a spine segment is of crucial importance for the design of next-generation functional spinal implants such as disc prostheses. A more complete three-dimensional description of the relative motion between two vertebrae is offered by the **helical axis of motion** (Fig. 7b). Any discrete motion in three-dimensional space can be expressed as a simple screw motion; the motion consists of a rotation about and a translation along a single unique axis in space. Although more complex, the helical axis of motion allows a three-dimensional visualization of the unique motion coupling in spinal kinematics [42].

There is a unique center of rotation for every intersegmental motion

### Clinical Instability

Clinical instability has been defined as an abnormal response of the spine to applied loads and is often characterized by excessive motion of spinal segments. The biomechanical definition of spinal instability has been further refined to encompass changes to the neutral zone, implying that motion extremes alone are not indicative of pathology. The abnormal response of the spine generally reflects incompetence of the passive and active structures (e.g. ligaments, muscles) that hold the spine in a stable position.

Spinal instability is not well defined

Definition of spinal instability remains a matter of debate

There is no reliable imaging based definition of spinal instability

Instability cannot be defined by imaging studies

The diagnosis of **spinal stability** remains an important yet controversial task for the practitioner, as many treatment decisions are based on this assessment. However, an objective and clinically relevant definition of spine instability remains elusive due to the multi-faceted nature and etiology of instability.

Classification systems have been proposed which are designed to categorize instability of the cervical, thoracic and lumbar spine resulting from traumatic injuries [98], but these do not take into account other causes of instability such as idiopathic disc and facet degeneration. **Clinical instability** as a definition can be applied equally well to soft-tissue pathologies which impart a laxity to the spine.

Diagnosis of spinal instability is routinely based on established imaging methods. Plain radiography is perhaps the most commonly used diagnostic tool but this has often questionable value and provides only indirect evidence of spinal instability. In many cases instability is only recognizable using functional radiography (flexion/extension) but this technique has limited reproducibility. Functional computed tomography offers a higher sensitivity than radiography for identifying abnormal motion potentially causing or aggravating a neurological deficit. MR imaging facilitates the identification of soft tissue abnormalities associated with instability. Nevertheless, there is no single imaging modality which discriminates with sufficient certainty “normal” and “abnormal” motion, therefore raising questions about the value of imaging-based methods for the diagnosis of instability.

Investigation using multiple **imaging techniques** likely provides the most objective assessment of instability. However, a significant barrier to reliable diagnosis is the non-specific nature of back pain and the uncertain relationship between instability and pain. Most researchers therefore define instability by clinical terms, rather than mechanical [75]. In the absence of a universally accepted definition of spinal instability we concur with the working definition of White and Panjabi [98] (**Table 5**):

**Table 5.** Definition of spinal instability

Clinical instability is the loss of the ability of the spine under physiologic loads to maintain its pattern of displacement so that there is no initial or additional neurologic deficit, no major deformity, and no incapacitating pain.

## Kinetics (Spinal Loading)

Spinal loads are generated by a combination of body weight, muscle activity, pre-tension in ligaments and external forces

Loads on the spine are generated by a combination of body weight, muscle activity, pre-tension in ligaments and external forces. Simplified calculations of spinal loading are possible using force diagrams (“free-body diagram”) for coplanar forces. **Direct measurements of spinal loading** are not possible, but can be inferred from, e.g. measurements of internal disc pressure [61] or forces acting on internal spinal fixation hardware [78]. Alternatively, the electromyographic activity of trunk muscles can be measured and correlated with calculated values for muscle contraction forces. This muscle activity data can then be included in mathematical models to estimate total spinal loading for a variety of physical activities.

## Static Loading

Posture influences the loading of the spine

Posture influences the loading of the spine. In addition to the weight of the trunk, the spine is further compressed by the active postural muscles during standing. The **center of gravity line** of the body generally falls ahead of the lumbar spine,

Table 6. Typical spinal loads

Activity	Load on L3 disc (N)
Supine, awake	250
Supine, traction	0
Supine, arm exercises	500
Upright sitting without support	700
Sitting with lumbar support, 110° incline	400
Standing at ease	500
Coughing	600
Forward bend 20°	600
Forward bend 40°	1 000
Forward bend 20° with 20 kg	1 200
Forward bend, 20° and rotated 20° with 10 kg	2 100
Sit up exercises	1 200
Lifting 10 kg, back straight, knees bent	1 700
Lifting 10 kg, back bent	1 900
Holding 5 kg, arms extended	1 900

Data derived from in vivo pressure measurements from over 100 subjects [63]

which creates a net forward bending moment. This moment must be counteracted by elastic ligament forces muscle activity in the erector muscles. Abdominal muscles and the psoas are active due to the natural postural sway during standing [59]. Pelvic tilt can alter spine loading. A backward tilt of the pelvis decreases the sacral angle and flattens the lumbar spine, the thoracic spine extends slightly to compensate changes to the body's center of gravity and muscle exertion is consequently decreased. Conversely, a forward tilt of pelvis increases the sacral angle, accentuating lumbar lordosis and thoracic kyphosis, and increasing muscle forces.

The loads on the anterior column during a variety of static postures have been derived from in vivo **disc pressure measurements** [60]. Employing a mathematical relationship between applied spinal compressive loading and disc pressure established in carefully controlled in vitro experiments, Nachemson et al. [63] have published extensive data on spinal loading (Table 6). In subsequent experiments, Wilke et al. [99] have provided additional data demonstrating similar disc pressures for lying prone and lying on the side, and, paradoxically, lower disc pressures for slouched sitting compared to sitting upright. Incidentally, this study also confirmed the intrinsic disc swelling and uptake of fluid overnight during rest.

In vivo spinal loading during daily activities can be derived from disc pressure measurements

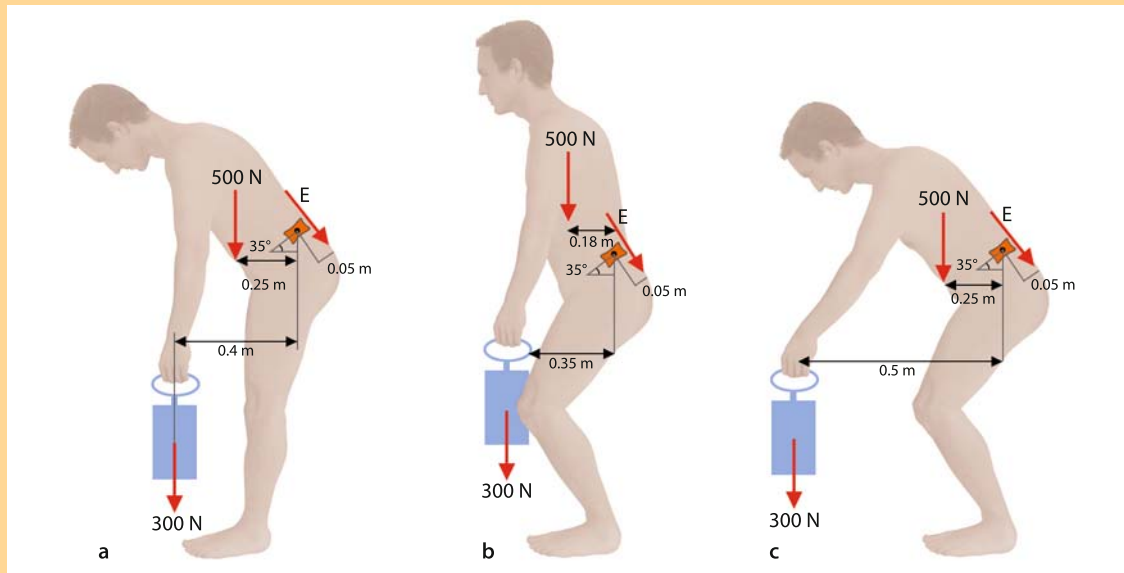
## Loads During Lifting

The highest loads on the spine are produced during **lifting**. Consequently this is the subject of considerable research in the fields of biomechanics and ergonomics. Loads during lifting can be extremely high and may approach the failure load of single vertebrae (5 000–8 000 N).

The highest loads on the spine are produced during lifting

As previously mentioned, the **vertebral endplate** is the weak link and often will fail before the intervertebral disc is compromised. Microdamage near the endplate due to repeated application of high loads [37] is a possible consequence of heavy lifting, and a decreased capacity for vertebral loading has been observed following this initial yielding of the vertebral body [77]. **Lifting forces** are directly influenced by the weight of the object being lifted, the size of object, spinal posture, lifting speed, and lifting technique, although no significant differences have been shown between spine compression and shear forces for stoop or squat lifting techniques [94] (Fig. 10). It is possible that other mechanisms to reduce the load on the spine, such as intra-abdominal pressure or muscular co-contraction, may somewhat compensate for poor lifting technique.

Lifting forces are directly influenced by the weight of the object, spinal posture, lifting speed and lifting technique



**Figure 10. Influence of lifting technique on spinal forces**

**a–c** Three different methods of lifting an object are shown in the diagrams, and the forces a lumbar disc experiences in each case are calculated. The disc is subject to three forces, as depicted in the diagrams: the force exerted by the upper body weight, the force exerted by the weight of the object and the force produced by the erector spinae muscles. The upper body weight and the weight of the object act in front of the disc and therefore create forward bending moments about the disc. To counteract these bending moments, the erector spinae muscles contract to create a balancing extension moment about the disc. Bending moments are a product of the force being applied and the distance at which the force is applied. Consequently, an increase in the distance between the object being lifted and the spine increases the forward bending moment, and furthermore the limited distance between the disc and the line of action of the erector spinae muscles necessitates a correspondingly high force in the muscles to produce the necessary balancing extension moment. Three examples are shown below for possible lifting postures, with a calculation of the net bending moments induced by the weight of the torso and the object being lifted, the required muscle force to counterbalance this and the resulting load which the disc experiences. **b** Lifting with a straight back and bringing the object closer to the body centerline has obvious benefits for minimizing spinal loading. **c** On the other hand, reaching too far for the object can induce substantially higher spinal loading.

**a:**  
 Total forward bending moment  
 = 245 Nm  
 Force produced by erector spinae  
 muscles = 4900 N  
 Total reaction force on disc = 5574 N

**b:**  
 Total forward bending moment  
 = 195 Nm  
 Force produced by erector spinae  
 muscles = 3900 N  
 Total reaction force on disc = 4578 N

**c:**  
 Total forward bending moment  
 = 275 Nm  
 Force produced by erector spinae  
 muscles = 5500 N  
 Total reaction force on disc = 6172 N

### Dynamic Loading

Motion increases muscle activity and spinal loads considerably in comparison to static and quasistatic postures. Inertial forces generated during the acceleration and deceleration of the trunk and extremities can add substantially to the overall load transferred along the spinal column. For example, the loads on the lumbar spine are approximately 0.2–2.5 times body weight during walking [18]. With a higher walking cadence, loading increases. Posture during motion also influences spinal loading. The greater the degree of forward flexion of the trunk during walking, the larger the muscle forces which are required to maintain the position of the trunk and consequently compressive forces at the individual discs increase.



Table 7. Glossary of biomechanical terms

<b>Force:</b>	A directed interaction between two objects that tends to change the physical state of both (i.e. acceleration or internal stresses). Force has both direction and magnitude.
<b>Moment:</b>	A turning force produced by a linear force acting at a distance from a given rotation axis. The concept of the moment arm, this characteristic distance, is key to the operation of the lever and most other simple machines capable of generating a mechanical advantage.
<b>Stress:</b>	The internal distribution and intensity of forces within a body that balance and react to the externally applied loads. Stress is expressed in force per unit area and is calculated on the basis of the original dimensions of the cross section of the specimen.
<b>Deformation:</b>	The change in shape or form in a material caused by stress or force.
<b>Strain:</b>	Deformation of a physical body under the action of applied forces. Strain is expressed as a change in size and/or shape relative to the original undeformed state.
<b>Stiffness:</b>	The resistance of an elastic body to deflection by an applied force. A stiff material is difficult to stretch or bend.
<b>Young's modulus:</b>	Young's modulus, or the tensile elastic modulus, is a parameter that reflects the resistance of a material to elongation. The higher the Young's modulus, the larger the force needed to deform the material.
<b>Elasticity:</b>	The theory of elasticity describes how a solid object moves and deforms in response to external stress. Elasticity expresses the tendency of a body to return to its original shape after it has been stretched or compressed.

## Recapitulation

**Human spine.** The main functions of the spine are to **protect** the spinal cord, to **provide mobility** to the trunk and to **transfer loads** from the head and trunk to the pelvis. The spine can be divided into **four distinct functional regions**: cervical, thoracic, lumbar and sacral. The cervical and lumbar regions are of greatest interest clinically, due to the substantial loading and mobility of these regions and the associated high incidence of trauma and degeneration.

**Motion segment.** The motion segment, or **functional spinal unit**, comprises two adjacent vertebrae and the intervening soft tissues. Each motion segment consists of an anterior structure, forming the **vertebral column**, and a complex set of posterior and lateral structures. The anterior column supports compressive spinal loads, while the **posterior elements** control spinal motion, protect the spinal cord and provide attachment points for muscles and ligaments.

**Vertebral body.** The principal biomechanical function of the vertebral body is to support the **compressive loads** of the spine due to body weight and muscle forces. The vertebral body comprises a highly porous trabecular core and a dense, solid shell. The trabecular bone bears the majority of the vertical compressive loads, while the outer shell forms a reinforced structure which additionally resists torsion and shear. The **vertebral endplate**

plays an **important role in load transfer** and is often the initial site of vertebral body failure. A strong correlation has been demonstrated between quantitative volumetric bone density and vertebral strength. Vertebral geometry and structure are equally important factors for the determination of vertebral strength.

**Intervertebral disc.** The intervertebral disc is the **largest avascular structure** of the body. The disc consists of a **gel-like nucleus** surrounded by a strong, **fiber-reinforced anulus**. Axial disc loads are borne by hydrostatic pressurization of the nucleus pulposus, resisted by circumferential stresses in the anulus fibrosus. Interstitial fluid is expressed from the disc during loading. Approximately 10–20% of the total fluid volume of the disc is exchanged daily. **Disc degeneration** substantially **alters** the mechanism of **load transfer**. Combined axial compression, flexion and lateral bending have been shown to cause disc prolapse.

**Posterior elements.** The facet joints guide and **limit intersegmental motion**. Deformity of the facets or fracture of the pars interarticularis may compromise **segmental shear resistance** and can lead to spondylolisthesis.

**Spinal ligaments.** The ligaments surrounding the spine **guide segmental motion** and contribute to

the intrinsic stability of the spine by limiting excessive motion. **Ligament response** to load is non-linear, with an initially flexible neutral zone and a subsequent stiffening under increasing load. Physiological strain levels in the ligaments approach 30% total elongation.

**Muscles.** The spatial distribution of muscles determines their function. The trunk musculature can be divided functionally into **extensors** and **flexors**, or **local stabilizers** and **global mobilizers**. The geometric relationship between the muscle line of action and the intervertebral center of rotation determines the functional potential of a muscle.

**Spine kinematics.** Spinal motion is often a complex, combined motion of simultaneous flexion/extension, side bending and rotation. The sum of limited motion at each motion segment creates considerable spinal mobility in all planes.

**Motion segment mechanical response.** The functional stiffness of the motion segment is adapted to the loading which each spine segment experiences. Compressive spine loads (i.e. muscle loads)

stiffen the spine segment. **Posterior elements contribute** significantly to overall **segmental stiffness**. The extrinsic support provided by trunk muscles stabilizes and redistributes loading on the spine and allows the spine to withstand loads of several times body weight without buckling. For small loads, displacements are relatively large due to ligament and disc laxity about the neutral position (neutral zone). At higher loads, resistance increases substantially. Changes to the neutral zone are associated with trauma and degeneration (i.e. "clinical instability"). There is a unique **center of rotation** for each intersegmental motion.

**Spinal loading.** Spinal loads are generated by a combination of body weight, muscle activity, pre-tension in ligaments and external forces. In vivo spinal loading during daily activities can be derived from disc pressure measurements. The **highest loads** on the spine are produced **during lifting**. Lifting forces are directly influenced by the weight of the object, spinal posture, lifting speed and lifting technique. Inertial effects during dynamic activities substantially increase spinal loading.

### Key Articles

**Nachemson A, Morris JM (1964) In vivo measurements of intradiscal pressure: discometry, a method for the determination of pressure in the lower lumbar discs. J Bone Joint Surg Am 46:1077 – 1092**

A report on the first series of in vivo disc pressure measurements conducted in 19 patients. This study provided new insight into the loading of the spinal column during daily activities. Study subjects covered a variety of gender, body types, and medical conditions. All subjects had normal discs, as determined from discogram. All subjects experienced back pain; some had already undergone fusion. A good correlation was shown between the body weight of segments above disc and the calculated load on disc. A qualitative relationship was found between the posture and disc loading (e.g. lowest for lying prone, higher for standing and highest for sitting slouched). Loads of 100–175 kg were reported for lower lumbar discs when seated. Standing loads ranged from 90 to 120 kg. This study laid the groundwork for a broad range of future studies on disc mechanics, spinal loading, and ergonomics.

**White AA, Panjabi MM (1990) Clinical biomechanics of the spine, 2nd edn. Philadelphia: J.B. Lippincott Company**

In an extensive research career, Prof. Manohar M. Panjabi has contributed several landmark publications on the topic of spinal biomechanics. This volume, co-authored with Prof. Augustus A. White, must be considered the most important single-source reference on the topic. Combining orthopedic surgery with biomechanical engineering, this reference and teaching text reviews and analyzes the clinical and scientific data on the mechanics of the human spine. The text covers all aspects of the physical and functional properties of the spine, kinematics and kinetics, scoliosis, trauma, clinical instability, the mechanics of pain, functional bracing and surgical management of the spine. Although our knowledge of the latter topic has progressed since the publication of this volume, the book as a whole remains timeless.

**Panjabi MM (1992) The stabilizing system of the spine. Part I: Function, dysfunction, adaptation and enhancement. J Spinal Disord 5:383–389**

**Panjabi MM (1992) The stabilizing system of the spine. Part II: Neutral zone and instability hypothesis. J Spinal Disord 5:390–396**

The first paper presents the conceptual basis for the assertion that the spinal stabilizing system consists of three subsystems. Passive stability is provided by the vertebrae, discs and ligaments. Active stability is provided by the muscles and tendons surrounding the spinal column. The nerves and central nervous system provide the necessary control and feedback systems to provide stability. Dysfunction of any of these three systems can lead to immediate or long term response which compromise stability and may cause pain. The second paper describes the neutral zone of intervertebral motion, around which little resistance is offered by the passive stabilizing components of the spine. Panjabi presents evidence for the correlation between the neutral zone with other parameters indicative of spinal instability. The clinical importance of the neutral zone is outlined, as are the influence of injury and pathology on the neutral zone and the compensatory mechanisms which are employed to maintain the neutral zone within certain physiological thresholds. Together, these two papers present a thorough definition of the concept of clinical instability and provide the context for interpreting the effectiveness of current spinal stabilization methods.

**Pope MH, Frymoyer JW, Krag MH (1992) Diagnosing instability. Clin Orthop Relat Res 279:60–67**

This review paper summarizes the problems associated with diagnosing clinical instability. The various definitions of instability are reviewed and preference is given to the definition of instability as a loss of stiffness. The authors emphasize that roentgenographic changes, particularly those associated with degeneration, have no relationship to instability. Various imaging methods are compared and contrasted, including multiple roentgenographic images and stereoroentgenography. Further kinematic measurement techniques employing kinematic frames attached directly to external fixation techniques are cited as promising for the fidelity of the data they may provide. The limitations of a purely mechanical definition of clinical instability are discussed.

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