Biomechanics of the Spine

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3.1 Introduction

The purpose of the current chapter is to review the normal mechanics of the spine, i.e., the structure and function of the various components such as discs, vertebral bodies, and spinous ligaments, and to delineate how the normal behavior of these structures is altered by age and various clinical interventions. A growing segment of spine radiology involves not just imaging but musculoskeletal intervention as well. Radiologists now are performing tasks that previously were in the realm of neurosurgery and orthopaedics. Because procedures such as thermal ablation of the disc, vertebroplasty, and kyphoplasty not only address pain relief but also may alter the mechanical behavior of the disc and/or vertebral body, it is important to have a fundamental understanding of the biomechanics of the spine. Knowledge of the normal biomechanics can help the clinician understand the effect a given intervention may have.

3.2 Spine

3.2.1 Structure

The spine is a complex structure that can be divided into five regions: the cervical, thoracic, lumbar, and sacral spines; and the coccyx. Of primary interest are the unfused vertebrae of the cervical through the lumbar regions, although recently the fused vertebrae of the sacrum, especially with regard to insufficiency fractures, have been the subject of increasing clinical interest.

The cervical region has seven vertebrae (C1–C7), which form a lordotic curve. The upper two vertebra are different in design compared with the other un-

KEY POINTS

- Main spinal motions, minor spinal motions:
  - C0–C1: flexion/extension
  - C1–C2: rotation
  - C2–C7: flexion/extension rotation lateral bending
  - T1–T12: flexion/extension rotation lateral bending
  - L1–L5: flexion/extension lateral bending
- Cervical and lumbar lordosis and thoracic kyphosis make the spine function as a spring reducing impact forces
- The spine transmits loads from the upper body through the pelvis into the lower extremities. The anterior center of gravity creates anterior bending and axial compression
- Bone mineral density (BMD) determines compressive strength:
  - Compressive strength ≈ BMD^2
  - BMD < average - 1 SD = osteopenic
  - BMD < average - 2.5 SD = osteoporotic
- Vertebrae:
  - Bear the compressive load
  - Facet joints limit axial rotation
- Disc:
  - Nucleus: hydrophilic mucopolysaccharides – 70 to 90% water
  - Annulus fibrosus: multiple-angled collagen layers
  - Bears the same compressive load as the vertebrae
  - Shock absorber, resists torsion, tensile and shear loads
  - Traumatic disc herniations require not only compressive force but also flexion and lateral bending, and typically occur in the lower lumbar region
- Spinal ligaments:
  - Guide motion and restrict excessive motion
  - Mix of collagen and elastin fibers
  - Ligamentum flavum more elastin = more stretch (up to 100% strain before failure compared with 10 to 15% for most other ligaments)

fused vertebra of the spine. The specialized design permits the unique function of these levels. The atlas (C1) mates with the occiput of the skull, and the space between them allows for flexion and extension but almost no rotation (White and Panjabi 1990). The axis (C2) is below the atlas and, as its name implies, most of the rotation that occurs in the spine occurs through the C1–C2 junction. There is essentially no lateral bending, but some flexion and extension. The remainder of the cervical spine (C3–C7), is fairly flexible, allowing motion in flexion/extension, lateral bending, and rotation (White and Panjabi 1990).

In contrast, the thoracic spine has 12 vertebrae (T1–T12). By virtue of its interaction with the rib cage, the thoracic spine does not allow much flexion/extension or lateral bending, but it does allow for some axial rotation. The curvature of the thoracic spine is naturally kyphotic.

In the lumbar spine, which consists of five vertebrae (L1–L5), the dominant motion is flexion. There is some extension and lateral bending, but almost no rotation. The curvature of the lumbar spine is normally lordotic.

Regardless of the level and type of motion, motion of the spine occurs in the spaces between the vertebrae, i.e., in the disc and at the facet joints.

3.2.2 Function

The spine, which transmits loads from the upper body through the pelvis into the lower extremities, is conceptually divided into three columns: anterior, medial, and posterior columns (Denis 1983). Because the center of gravity of the human body is located anterior to the spinal column, the center of gravity creates a combined load resulting in axial compression and an anterior bending moment (Fig. 3.1). When the spine is in flexion, the instantaneous axis of rotation is generally in the vertebral bodies near the superior endplate (Panjabi et al. 1984). The instantaneous axis of rotation may be considered a fulcrum and, as such, tensile forces must be active posterior to the fulcrum to balance the anterior bending moment caused by the body's center of gravity. The balancing forces are provided

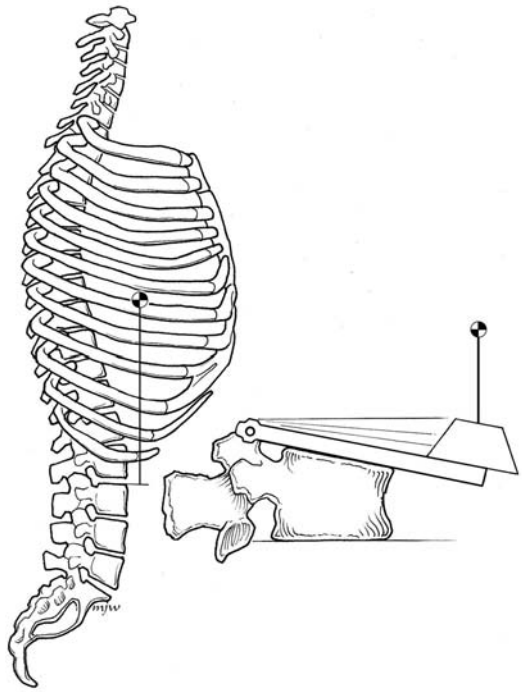

Fig. 3.1. The body's center of mass is located anterior to the spinal column and causes an anterior bending moment on the vertebral bodies. The anterior bending moment creates stresses on the anterior cortex of the vertebral body greater than would be expected from body weight alone

by the paraspinous muscles, posterior ligaments, and the posterior portion of the annulus fibrosis. During anterior flexion (e.g., bending over to tie a pair of shoes), the body's center of gravity moves anteriorly, increasing the bending moment on the spine and the compressive stresses on the anterior column. Bending over to pick up a load not only moves the center of gravity anteriorly, but it also increases the magnitude of the anteriorly located load, which, when combined with the increased moment arm, dramatically increases the compressive stresses on the anterior column. It is this excessive compressive stress that often results in vertebral compression fractures in osteoporotic patients. By definition, vertebral compression fractures exhibit disruption of the anterior column (Denis 1983).

3.2.3 Mechanical Behavior

Compressive vertebral strength is related roughly to the square of the vertebral bone mineral density (BMD) (Lang et al. 1988). When a patient's BMD is 1 standard deviation below the average for the sex-, height-, weight-, and race-matched young (20–30 years old) population, the patient is considered to be osteopenic. A patient with a BMD more than 2.5 standard deviations below that standard is considered osteoporotic (WHO Study Group 1994). In patients with osteoporosis, vertebral BMD might be half of what it was in their youth, which means their vertebral compressive strength may be as low as a one fourth of what it was when they were young. It is not surprising, then, that each year in the United States, more than 700,000 vertebral compression fractures are reported (Riggs and Melton 1995), 300,000–400,000 of which result in hospital admissions.

The lordotic and kyphotic curves of the spine function as a spring, allowing the spine to flex and thereby reduce impact magnitude and increase impulse time compared with what would be the case if the spine were a perfectly straight post. The viscoelastic nature of the spinous ligaments and intervertebral discs increases the impulse time even more, thereby reducing axial impacts.

3.3 Vertebrae

3.3.1 Structure

Except for the first and second cervical vertebrae, all vertebrae share the same basic structure. The roughly cylindrical anterior portion has a thin, hard, cortical shell filled with cancellous bone. The posterior portion, or neural arch, is composed of the pedicles and lamina. This bony ring protects the spinal canal and serves as the foundation for the articular, transverse, and spinous processes. The latter two processes serve as attachment sites of the muscles of the spine. The former process serves as the support for the inferior and superior facet joints.

3.3.2 Function

The primary mechanical function of the vertebrae is to support the axial compression of the body weight. The vertebral body bears most of the compressive

load, but the facets also are involved in resisting axial load. Some researchers report that the facet bears between 3 and 25% of the load (Lorenz et al. 1983; Yang and King 1984), and that if the facet joint is arthritic, it may bear 47% or more (Yang and King 1984). Although the facet joints bear some axial load, they serve to limit relative axial rotation between vertebrae. In fact, intervertebral discs can withstand 22° or more of axial rotation before they fail (Farfan et al. 1970), but the facets limit axial rotation to about 5° to prevent such disc injury (Gregersen and Lucas 1967). The neural arch also protects the spinal cord from injury. The interior of the vertebral body serves to support the endplates by means of the cancellous framework, but it also functions as a vascular space filled with marrow, fat, and blood. Part of the nutrition of the disc is supplied through the endplates.

3.3.3 Mechanical Behavior

Vertebral bodies increase in compressive strength (and size) from C1 to L5 (Brinckmann et al. 1989; Burklein et al. 2001; Moro et al. 1995), probably in response to the higher mechanical demands on the vertebral bodies secondary to the increasing body weight they bear from the superior to the inferior spine. The strength of a given vertebral level is a function primarily of its bone density. In theory, bone strength is a function of the square of the density, but studies have found a wide range of powers (1.2–27; Lotz et al. 1990). Vertebral bodies are strongest along the axis of the spine. In normal vertebral bodies, most of the compressive strength comes from the trabecular bone beneath the endplates. The trabeculae are arranged predominantly in a vertical fashion (as support columns) with some horizontal cross-bracing (Fig. 3.2). The compressive strength of the trabecular structure in the medial–lateral and anterior–posterior directions is approximately half that in the axial direction (Galante et al. 1970). Trabecular compressive strength is greatest in the center of the vertebral body, where it is most needed to resist endplate bending (Keller et al. 1989). The remaining compressive strength of the vertebral body comes from the cortical shell (Rockoff et al. 1969).

3.3.4 Effect of Aging

As the vertebral bodies age, the cortical shell bears a greater share of the load (Rockoff et al. 1969), perhaps as a consequence of the general decrease of cancellous bone associated with osteoporosis, i.e., the cortical shell may bear a greater percentage of the load because there is simply less cancellous bone with which to share the load. Cancellous bone density may also be off-loaded as the disc dehydrates and its health degenerates. Axial load tends to be transmitted through the nucleus pulposus in healthy discs, causing the endplates to deflect. In vertebral bodies with healthy discs that develop compression fractures, the predominant mode is endplate fracture (Rolander and Blair 1975) When the disc degenerates, the load is transmitted through the annulus into the cortical shell, bypassing the cancellous bone. In this instance, the fracture mode is predominantly that of cortical shell

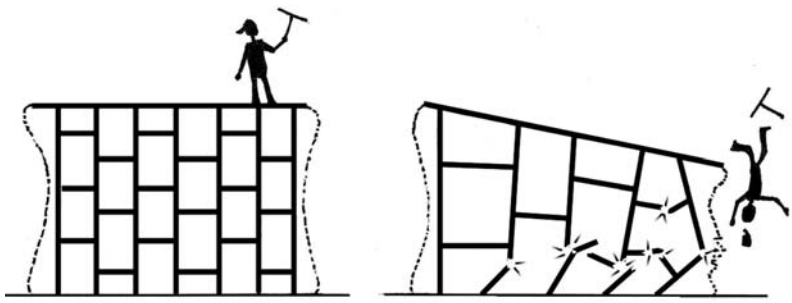

Fig. 3.2. The cancellous interior of each vertebral body functions as a scaffold supporting the endplates (*left*). As the spine becomes osteoporotic, the support columns become fewer and thinner, and there are fewer cross-braces. These changes conspire to weaken the scaffold, placing it at risk for collapse (*right*)

fractures. Because the load apparently is shunted toward the cortex, the lack of mechanical stimulation may encourage the cancellous bone underneath the endplate to resorb.

Bone density generally decreases as a function of aging. Because of the power-law relationship between bone density and strength, if bone density of a vertebral body decreases to half of its young healthy norm, then strength might be a quarter of what it once was in youth.

The bone density of osteoporotic vertebral bodies is at least 2.5 standard deviations below that of their young, sex-, race-, and weight-matched counterparts, as defined by the World Health Organization (Kanis and WHO Study Group 1994; WHO Study Group 1994). In addition to this diminished bone density, the organization of the remaining cancellous bone is altered as the horizontal crossbraces are resorbed (Bell et al. 1967), resulting in long rather than short columns. The load needed to cause a column to buckle is an inverse function of the square of the column length (Fig. 3.2); therefore, if the column's effective length doubles, the load needed to buckle it decreases by a factor of 4.

3.4 Disc

3.4.1 Structure

The disc comprises two major parts: the hydrated gel center (or nucleus pulposus) and the surrounding collagen-rich annulus fibrosus. The nucleus is composed of hydrophilic mucopolysaccharides. Approximately 70–90% of the nucleus is water (Panagiotacopulos et al. 1987). The annulus fibrosus, which wraps around the nucleus, is composed of several layers of fibrous tissue. The orientation of the collagen fibers in each layer (or lamina) is approximately 30° relative to the endplate (Fig. 3.3) (Inoue 1981).

3.4.2 Function

The disc serves many functions. It bears the compressive load of the body's weight above it as well as the resultant compressive load of active muscle contraction during activities of daily living. The disc is a hydrated viscoelastic structure and, as such, dampens the axial loads transmitted through the spine. It serves as a shock absorber between vertebral levels and resists tensile and shear loads that result from spine flexion/extension, lateral bending, and twisting.

3.4.3 Mechanical Behavior

The orientation of the fibers alternates from 30 to –30° with each subsequent layer (Inoue 1981). Presumably, the fiber orientation is an evolutionary optimization that allows the disc to resist torsion, shear, and tension. When the spine is subjected to a compressive load, the nucleus pulposus, which is essentially incompressible because of its high water content, forces the annulus fibrosus laterally. This lateral expansion places the collagen fibers of the annulus in tension to resist the compressive loading to which the composite structure of the disc is subjected. One might expect that severe compression would cause the collagen fibers to rupture and allow the nucleus to prolapse; however, herniation of the nucleus pulposus reportedly does not occur even under high compressive loads alone (Markolf and Morris 1974; Virgin 1951). Traumatic disc herniation requires a combined compressive load with flexion and lateral bending (Adams and Hutton 1982). In that study, discs prone to such injury typically were in the lower lumbar region, from the 40- to 49-year-old subgroup of donors, and had some apparent disc degeneration.

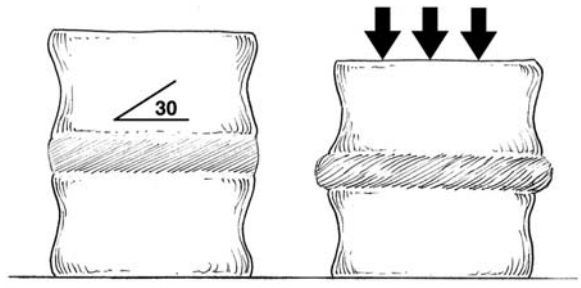

Fig. 3.3. The annulus fibrosis is composed of layers of collagen fibers. The collagen fibers are oriented at either 30 or –30° relative to the endplate. The orientation alternates with each successive layer (*left*). When a load is placed on the vertebral body, the hydrated nucleus pulposus becomes pressurized and pushes laterally against the annulus (*right*)

Discs, like other collagenous soft tissue structures of the body, are viscoelastic, i.e., the material behavior of the structure depends not only on the stress or strain that is applied, but also on the time period over which it is applied. When a disc is subjected to a compressive load, the disc will compress instantly, but it also will continue to compress until it reaches some equilibrium level. This compressive deformation as a function of time is called creep. The amount of creep deformation is a function of the load magnitude, the time period over which the load is applied, and the degree of degeneration of the disc. Degenerated discs creep more and creep more quickly than do healthy hydrated discs (Kazarian 1975), suggesting that degenerated discs lose some of their viscoelasticity and, therefore, some of their shock absorption characteristics. Viscoelasticity imparts a damage tolerance to tissues. If tissues are stressed or strained at high rates, their apparent modulus and failure strength increase and their ability to absorb energy increases.

3.5 Spinal Ligaments

3.5.1 Structure

The ligaments of the spine are composed of collagen and elastin fibers enmeshed in a hydrated gel. The relative amounts of collagen to elastin fibers dictates the mechanical response of a given ligament. For example, the ligamentum flavum, which runs from the lamina of one vertebral body to the lamina of the adjacent vertebral body, has the highest elastin content of all spinal ligaments. The high elastin-to-collagen content allows the ligament to stretch when the spine is in full flexion. Most ligaments fail at approximately 10–15% strain, but some, such as the ligamentum flavum and supraspinous ligament in the lumbar spine, reportedly withstand strains as high as 100% until failure (Fig. 3.4) (Pintar et al. 1992).

3.5.2 Function

Spinal ligaments, like other ligaments of the body, are tasked with connecting one bone to another – in this case, one vertebral body to another. They guide joint motion and permit flexibility of the spine without allowing excessive motion that would place the spinal cord at risk for injury. By tethering the vertebral bodies together, the spinal ligaments relieve the muscles about the spine of some of the burden of maintaining stability.

3.5.3 Mechanical Behavior

The ligaments exhibit nonlinear viscoelastic behavior. Because the dry weight of ligaments primarily is composed of collagen fibers, ligaments are designed to resist tension. They have no inherent resistance to compression, and they buckle easily. The low modulus response of ligaments in the "toe" region, the initial part of the stress-strain curve, allows the spine to be flexible without the need for spinous muscle to expend much energy overcoming ligament resistance. As the spine is placed in more extreme positions, the ligaments are stretched. As the ligament as a whole is stretched, the collagen fibers, which are generally wavy in the relaxed state, become straightened. Sequential straightening of the fibers increases the apparent modulus of the ligament. In this manner, the ligaments' resistance to stretching dramatically increases. If spinal motion continues, the ligaments are stretched further into the linear region of the stress-vs-strain curve. In this region, practically all of the collagen fibers have been straightened in an

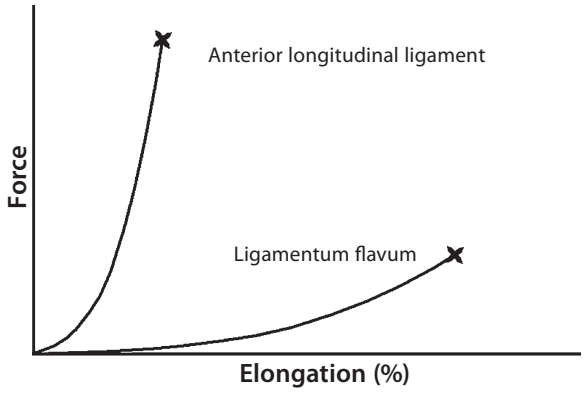

Fig. 3.4. The structure and function of ligaments are determined by the job each ligament needs to perform. Ligaments that need to be relatively strong and stiff, such as the anterior longitudinal ligament, have a high collagen to elastin ratio, whereas ligaments that need to be flexible, such as the ligamentum flavum, contain large percentages of elastin

attempt to prevent any further motion. If additional motion occurs, collagen fiber will rupture, resulting in a loss of stiffness, damage to the ligament, and eventual failure. Because ligaments are viscoelastic, the apparent strength and stiffness of a ligament is increased dramatically if the stretching rate is rapid (Fig. 3.5).

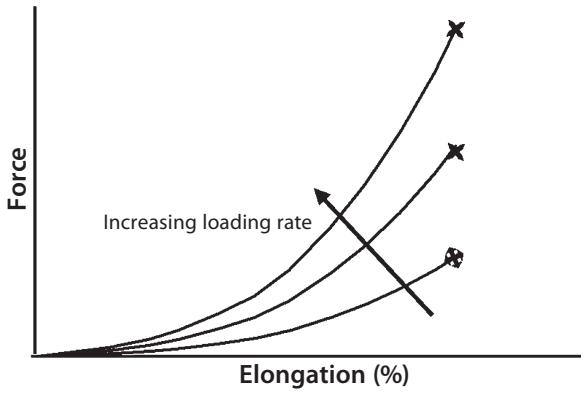

Fig. 3.5. Ligaments are viscoelastic and, as such, increase their apparent stiffness and strength when the rate at which they are loaded increases. They also are able to absorb more energy. These characteristics prevent damage during impact loading, such as typically happens with traumatic events

References

Adams MA, Hutton WC (1982) Prolapsed intervertebral disc. A hyperflexion injury. 1981 Volvo Award in Basic Science. Spine 7:184–191

Bell GH, Dunbar O, Beck JS et al. (1967) Variations in strength of vertebrae with age and their relation to osteoporosis. Calcif Tissue Res 1:75–86

Brinckmann P, Biggemann M, Hilweg D (1989) Prediction of the compressive strength of human lumbar vertebrae. Spine 14:606–610

Burklein D, Lochmuller EM, Kuhn V et al. (2001) Correlation of thoracic and lumbar vertebral failure loads with in situ vs ex situ dual energy X-ray absorptiometry. J Biomech 35:579–587

Denis F (1983) The three column spine and its significance in the classification of acute thoracolumbar spinal injuries. Spine 8:817–831

Farfan HF, Cossette JW, Robertson GH et al. (1970) The effects of torsion on the lumbar intervertebral joints: the role of torsion in the production of disc degeneration. J Bone Joint Surg 52A:468–497

Galante J, Rostoker W, Ray RD (1970) Physical properties of trabecular bone. Calcif Tissue Res 5:236–246

Gregersen GG, Lucas DB (1967) An in vivo study of the axial rotation of the human thoracolumbar spine. J Bone Joint Surg 49A:247–262

Inoue H (1981) Three-dimensional architecture of lumbar intervertebral discs. Spine 6:139–146

Kanis JA, WHO Study Group (1994) Assessment of fracture risk and its application to screening for postmenopausal osteoporosis: synopsis of a WHO report. Osteoporos Int 4:368–381

Kazarian LE (1975) Creep characteristics of the human spinal column. Orthop Clin North Am 6:3–18

Keller TS, Hansson TH, Abram AC et al. (1989) Regional variations in the compressive properties of lumbar vertebral trabeculae. Effects of disc degeneration. Spine 14:1012–1019

Lang SM, Moyle DD, Berg EW et al. (1988) Correlation of mechanical properties of vertebral trabecular bone with equivalent mineral density as measured by computed tomography. J Bone Joint Surg 70A:1531–1538

Lorenz M, Patwardhan A, Vanderby R Jr (1983) Load-bearing characteristics of lumbar facets in normal and surgically altered spinal segments. Spine 8:122–130

Lotz JC, Gerhart TN, Hayes WC (1990) Mechanical properties of trabecular bone from the proximal femur: a quantitative CT study. J Comput Assist Tomogr 14:107–114

Markolf KL, Morris JM (1974) The structural components of the intervertebral disc. A study of their contributions to the ability of the disc to withstand compressive forces. J Bone Joint Surg 56A:675–687

Moro M, Hecker AT, Bouxsein ML et al. (1995) Failure load of thoracic vertebrae correlates with lumbar bone mineral density measured by DXA. Calcif Tissue Int 56:206–209

Panagiotacopulos ND, Pope MH, Bloch R et al. (1987) Water content in human intervertebral discs. Part II. Viscoelastic behavior. Spine 12:918–924

Panjabi MM, Krag MH, Dimnet JC et al. (1984) Thoracic spine centers of rotation in the sagittal plane. J Orthop Res 1:387–394

Pintar FA, Yoganandan N, Myers T et al. (1992) Biomechanical properties of human lumbar spine ligaments. J Biomech 25:1351–1356

Riggs BL, Melton LJ III (1995) The worldwide problem of osteoporosis: insights afforded by epidemiology. Bone 17:505S–511S

Rockoff SD, Sweet E, Bleustein J (1969) The relative contribution of trabecular and cortical bone to the strength of human lumbar vertebrae. Calcif Tissue Res 3:163–175

Rolander SD, Blair WE (1975) Deformation and fracture of the lumbar vertebral end plate. Orthop Clin North Am 6:75–81

Virgin WJ (1951) Experimental investigations into the physical properties of the intervertebral disc. J Bone Joint Surg 33B:607–611

White AA III, Panjabi MM (1990) Kinematics of the spine. In: Clinical biomechanics of the spine, 2nd edn. Lippincott, Philadelphia, pp 85–125

WHO Study Group (1994) Assessment of fracture risk and its application to screening for postmenopausal osteoporosis. Report of a WHO Study Group. World Health Organ Tech Rep Ser 843:1–129

Yang KH, King AI (1984) Mechanism of facet load transmission as a hypothesis for low-back pain. Spine 9:557–565