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LEARNING OBJECTIVES

After completion of this monograph the course participant will be able to describe:

1. The osseous anatomy of the lumbopelvic region.
2. The joint anatomy of the lumbopelvic region.
3. The musculoligamentous anatomy of the lumbopelvic region.
4. The neurovascular anatomy of the lumbopelvic region.
5. The biomechanics of the lumbopelvic joints.
6. The biomechanics of the lumbopelvic musculoligamentous structures.
7. The biomechanics of the lumbopelvic neurovascular structures.
8. The biomechanics of lumbar spine cardinal plane and coupled motions.
9. The concept of form and force closure in the sacroiliac joint.
10. The biomechanical influences of the lower limb on the lumbopelvic region.

INTRODUCTION

Low back pain and its related disability are major societal problems. Eighty percent of all people experience low back pain (LBP) at some point in their lives.[1] Back symptoms are the most frequent reason to seek consultation with orthopaedic surgeons or neurosurgeons; they are the second leading reason for physician visits.[2] In the United Kingdom, each year 7% of the adult population will consult with their general practitioner for LBP.[3] In their systematic literature review on the prevalence of LBP in adults, Loney and Stratford[4] found that 19.2+/-9.6 % of people questioned had LBP at the time of survey. The percentage of people with LBP in the year prior to the survey was 32.37+/-23.6%. Based on one US and one Canadian study they estimated mean point prevalence for LBP in North American adults at 5.6%. In other words, 10 of every 178 million North American adults experience LBP in any given day.[4] In the US, the total yearly costs associated with LBP, including both direct health care costs, and indirect costs of work loss and disability payments have been estimated to exceed 50 billion dollars.[2] Three-fourths of patients taken off work due to LBP have sufficiently recovered to return to work within 6 to 8 weeks, but 2 to 3% of patients develop chronic disabling LBP lasting more than one year.[5] Of all worker's compensation claimants, a relatively small percentage of 10 to 25% accounts for the majority (70 to 87%) of medical and disability benefit costs.[5]

Despite this high prevalence of LBP and the astronomical costs associated with this problem a number of myths continues to circulate about LBP. Fritz[6] mentioned 3 common misconceptions. Her *LBP myth #1* is that "most people get better no matter what we do." Practitioners have generally assumed that the natural history of LBP is relatively benign: 80 to 90% of patients with LBP are expected to recover in about 6 weeks, irrespective of the administration or type of treatment.[1] In a prospective trial Skargren et al[7] randomly allocated 323 patients between 18 and 60 years of age to either chiropractic or physical therapy (PT) for treatment of LBP or neck pain. Among other variables, they researched recurrence and seeking of additional health care. Only 19% in the chiropractic group and 18% of patients in the PT group reported no recurrence at a 12 month follow-up; nearly 60% of patients reported 2 or more recurrences. Fifty-nine percent of chiropractic patients and 41% of PT patients sought additional care, but only 36% and 29%, respectively, returned to their initial provider. Croft et al[3] studied 463 adults aged 18 to 75, who consulted with their general practitioner for mechanical LBP. Of these patients, 59% did not consult again with their general practitioner, 32% consulted again within 3 months after the initial visit, and 8% consulted again after the 3 month period. The authors conducted follow-up interviews with 218 patients. Of those interviewed within 1 to 2 weeks after the initial visit, 2% reported having no pain or disability. At 3 months this number increased to 21%. At 12 months only 25% of those interviewed reported no complaints. These studies indicate that LBP may not be as benign and self-limiting as has been assumed, possibly because a large percentage of patients is lost to follow-up as they do not return to their original provider despite continued or recurrent complaints. This is extremely well illustrated by the Croft et al[3] study in which the physician might erroneously assume that after 3 months 92% of patients no longer has LBP, even though follow-up showed continued complaints in 75% of (the admittedly smaller sample of) those interviewed.

LBP myth #2 is that "the situation is improving."[6] Taylor et al[2] reviewed the National Hospital Discharge Survey data for the 1979 to 1990 period to investigate variations in inpatient management of mechanical LBP in patients 20 years or older. They found an increase in low back surgery from 147,500 in 1979 to 279,000 in 1990. Adjusted for population growth this meant an increase from 102 to 158 low back surgeries per 100,000 adults. Non-fusion surgery increased by 47%; surgeries involving fusion increased with 100%. Not only have back surgery rates risen sharply, Fritz[6] noted that chronic LBP disability has also risen dramatically in the past 25 years.

LBP myth #3 is that "the medical community knows how to approach the problem."[6] Controversy clearly exists regarding the most appropriate treatment for LBP. Taylor et al[2] found that in the 1988 to 1990 period annual LBP surgery rates varied widely between the different geographical locations in the US: 113 per 100,000 adults in the western US, 131 in the Northeast, 157 in the Midwest, and 171 in the South. Reported LBP prevalence in these 4 areas was nearly identical. This wide geographical variation despite similar prevalence indicates that the choice for surgical management of LBP is far from uniform.

The extensive personal and societal impact of LBP is obvious. The natural history of LBP may not be as benign as previously assumed. Research appears consistent with the interpretation that 90% of patients will have stopped consulting with their initial or any provider and are thus lost to follow-up rather than having no more pain or disability. A small percentage of patients develop chronic disabling pain and these patients become responsible for the majority of costs. Early identification and appropriate intervention could have a major impact on costs. The medical profession does not appear to have a uniform answer to LBP: increasing surgery rates and wide geographic variation in management strategies need not necessarily be interpreted as a sign of increased treatment success. Mielenz et al[8] investigated 1580 patients with acute LBP initially seen by 208 randomly selected health care providers (orthopaedic surgeons, primary care providers, chiropractors) in North Carolina. A PT saw 199 (12.8%) of the patients, by referral or direct access. The PT patients had higher baseline scores on the Roland-Morris Questionnaire. This instrument describes the impact of LBP on various functional activities; higher scores indicate higher levels of disability. Location of pain also played a role in referral: patients with LBP and pain below the knee in one or both legs were more likely to be referred to PT. This study seems to indicate that the referral of choice for patients with more severe acute LBP is PT rather than medical, surgical, or chiropractic treatment. A natural history less benign than previously assumed, an increase in chronic LBP-related disability, seemingly inadequate surgical treatment options, and PT as the referral of choice for more involved LBP patients makes in-depth knowledge of all aspects of LBP a necessity for every PT involved in treating these patients. In this monograph we will review the anatomy and biomechanics of the lumbopelvic region. Monograph 11.2.4. will deal with changes as a result of aging, pathology affecting the lumbopelvic region, diagnosis, and PT treatment. It will also contain the case studies that will illustrate application of this information to patient management.

ANATOMY

Osseous anatomy

The vertebral column typically consists of between 32 and 34 vertebrae. The lumbopelvic portion consists of 13 to 15 vertebrae. The 5 lumbar vertebrae are considered true or moveable vertebrae, whereas the 5 sacral and 3 to 5 coccygeal vertebrae are known as the false or fixed vertebrae, because they fuse and form the 2 adult bones of sacrum and coccyx.[9]

Lumbar vertebrae

All lumbar vertebrae (Figure 1) consist of a vertebral body and a vertebral arch.[9,10] The vertebral arch is also known as the neural arch, as it encloses the conus medullaris and cauda equina in the vertebral foramen.[10] In the upper lumbar spine this foramen is oval in shape; at the lower levels it is more triangular. Sometimes the vertebral foramen of the lower lumbar vertebrae has a trefoil shape: a triangular shape in which the basal angles are stretched.[11] The laterally located basal angles of the trefoil-shaped vertebral foramen are called the lateral recesses.[11]

The lumbar vertebral bodies are oval or kidney-shaped in the transverse plane with a concave posterior aspect and a larger transverse to sagittal diameter.[12] Vertebral body height is greater anteriorly than posteriorly; this contributes to the sagittal plane lordosis.[12] The lateral and anterior surfaces of the vertebral bodies are slightly concave.[11] On the posterior surface there are 1 or more large foramina called the nutrient foramina: they transmit the nutrient arteries and the basivertebral veins. The anterolateral surfaces have

similar, smaller foramina that transmit the equatorial arteries.[9,11] The top and bottom surfaces of the vertebral body serve as attachment sites for the intervertebral disk (IVD); they are flat or slightly concave[9], covered with smooth hyaline cartilage[10], and perforated by tiny holes.[11] A narrow rim of smooth, less perforated bone marks the perimeter of both the top and bottom surface. This fused ring apophysis represents a secondary ossification center of the vertebral body.[11]

Two parts comprise the vertebral arch: pedicle and lamina. The pedicles are short, thick processes that project dorsally on either side from the cranial aspect of the posterolateral vertebral body. They are somewhat narrower in their middle part forming the concave superior and inferior vertebral notch.[9,10] The intervertebral foramen (IVF) is formed by the deep inferior vertebral notch of a superior vertebra and the much shallower superior vertebral notch of the articulating inferior vertebra.[10] The lamina is the posteroinferiorly directed continuation of the pedicle. Both laminae fuse in the midline and form the base for the spinous process. On either side of the spinous process the laminae form the anterior border of the shallow, longitudinal vertebral grooves that contain the deep back muscles.[9]

Each lumbar vertebra has a multitude of bony processes. On either side a cranially projecting superior articular process and a caudally directed inferior articular process are located at the junction of lamina and pedicle.[9] The articular surfaces or facets of these processes are covered with hyaline cartilage and form the zygapophysial joints (ZJ).[11] The part of the lamina that connects the inferior and superior articular processes is called the interarticular pars. Its location at the junction of the horizontally projecting pedicle and the more vertically oriented lamina subjects it to considerable bending forces thus making it a frequent site for a fatigue fracture.[11]

The spinous process is the dorsal continuation of the fused laminae. In the lumbar spine it is quadrilateral in shape and it is thicker in the lower vertebrae. Like the transverse process it serves as an attachment site for muscles and ligaments. The transverse process projects laterally on either side from the point where the pedicle joins the lamina. The transverse processes of L1 to L3 are horizontal; the L4 to L5 transverse processes incline somewhat dorsally.[9] The length of the transverse processes increases from L1 to L3 and then decreases again, such that the L1 and L4 processes are of equal length. The greater L3 transverse process length may be related to providing the muscles attached to this vertebra an extra mechanical advantage.[11] The L4 and L5 transverse processes originate more ventral on the pedicle than the higher ones: its increased thickness and attachment along the whole length of the pedicle allows for transmission of forces from the strong iliolumbar ligament through the L5 transverse process.[11] The accessory process is located on the dorsal aspect of the base of the transverse process.[9] It varies in size from a small bony bump to a definitive pointed projection of variable length.[11] European anatomy literature considers this accessory process the true transverse process; it considers the transverse processes discussed above as costal processes indicating their origin as rudimentary lumbar ribs.[10,13] The mamillary process is superior and slightly medial to the accessory process. The mamillo-accessory separates it from the accessory process.[11]

Sacrum

The sacrum is a triangular bone wedged between the two innominate bones (Figure 2). Its wider cranial surface, the base, consists of the upper surface of the body of S1, which articulates with the L5-S1 IVD. The ventral border of the body of S1 projects into the pelvis and is called the promontory. Posterior to the body of S1 lies the opening to the sacral canal, the caudal extension of the spinal canal. The superior articular processes face posteromedially and connect to the body of S1 by short pedicles with a superior vertebral notch that forms the inferior border of the lumbosacral IVF. Lateral to the S1 vertebral body the fusion of the transverse and costal processes of the first sacral vertebra forms large triangular surfaces called the wings of the sacrum. They support the psoas muscle and the lumbosacral nerve trunk.[9] Mamillary processes found only on S1 serve as insertion sites to the lumbar multifidus.[11]

The pelvic surface of the sacrum is smooth and concave. Four transverse ridges cross its center indicating the original planes of separation between the 5 sacral vertebrae. The ridges represent the ossified sacral IVDs.[9,10] Four pelvic or anterior sacral foramina are located at either side of the ends of the transverse ridges. The ventral rami of the S1 to S4 nerve roots exit through these foramina.[9] Bars of bone pass superiorly and inferiorly to these foramina and form the sacral transverse processes lateral to the foramina.[11]

The convex dorsal surface of the sacrum has 3 to 4 midline rudimentary spinous processes. These processes form the median sacral crest. Lateral to this crest the fused laminae form the sacral grooves

providing an attachment site for the deep back muscles. The articular tubercles located at the inferomedial border of the posterior sacral foramina[11] represent the fused sacral ZJs[9,10] and form the intermediate sacral crest. Four foramina on either side transmit the dorsal rami of the sacral nerve roots (Figure 2).[9,10] The sacral transverse processes extend between and lateral to the posterior sacral foramina, enclosing them. The lateral posterior edges of these fused transverse processes are known as the transverse tubercles. These tubercles form the lateral sacral crest.[11]

The caudal part of the sacrum is the apex. The terminal end of the sacrum has a flattened surface where it articulates with the sacrococcygeal disk.[11] The laminae of S5 fail to meet in the midline: the resulting opening is called the sacral hiatus. This hiatus is the caudal aperture of the sacral canal.[9] Depending on the number of non-fused laminae this hiatus can display individual differences in shape and size.[10] At the caudal end of the intermediate sacral crest the inferior articular processes of the fifth sacral vertebra or sacral cornua extend downward for articulation with the cornua of the coccyx.[9]

Fusion of the costal and transverse processes of the sacral vertebrae forms the lateral mass of the sacrum.[9,11] Its lateral surface is narrow at the level of the bottom 2 and wider at the the level of the top 3 vertebrae.[11] Caudally it ends in a projection, the inferior lateral angle. There is a notch medial to this angle; the transverse process of the first coccygeal vertebra converts this into a foramen for transmitting the S5 ventral ramus.[9] The superior part of the lateral sacrum has a cartilaginous ear-shaped or auricular joint surface. This auricular surface articulates with a similarly named joint surface on the ilium.[10] Dorsal to the auricular surface is a rough area, the sacral tuberosity. This area corresponds to a rough surface on the ilium, the iliac tuberosity.[10] These areas serve as attachment sites for the interosseus sacral ligament; the sacral tuberosity has 3 deep, uneven impressions for its insertion.[9,13]

Some describe the sacrum as a double wedge tapering both from superior to inferior and from anterior to posterior.[14] Others disagree with the anteroposterior narrowing.[15,16] Snijders et al[17] described the lateral aspect of the sacrum as undulated and resembling a propeller (Figure 2); we will discuss later the effect of this shape on sacroiliac joint (SIJ) biomechanics.

Coccyx

The coccyx is a triangular bone usually formed by 4, but sometimes by as little as 3, or as many as 6 segments.[9,10] We can identify rudimentary vertebral bodies, and articular and transverse processes in the top 3 coccygeal vertebrae. All coccygeal vertebrae lack pedicles, laminae, and spinous processes.[9] The lowest 3 segments are usually fused[9], but occassionally synovial intercoccygeal joints are present.[18] The top coccygeal vertebra frequently exists as a separate bone.[9] The dorsal coccygeal surface has a row of rudimentary articular tubercles on either side. At the level of the first coccygeal vertebra these tubercles are the coccygeal cornua. They articulate with the sacral cornua and complete the IVF for the dorsal ramus of the S5 nerve root. Similar to the sacrum, the pelvic surface of the coccyx has transverse ridges indicating the separate coccygeal vertebrae. The apex of the coccyx is the most caudal part of the vertebral colum. Sometimes it is bifid or deflected to one side. The coccyx is an attachment site for muscles of the pelvic floor and gluteus maximus, sacrococcygeal, sacrotuberous, and sacrospinous ligaments, and is in contact with the rectum.[9]

Anatomy of joints

Lumbar intervertebral disk

The lumbar IVD connects the body of one vertebra to that of adjacent superior and inferior vertebrae. The joint thus formed is classified as a symphysis or amphiarthrosis.[9,19] Bogduk[11] referred to this joint as the interbody joint. We will discuss the 3 components of the IVD: anulus fibrosus (AF), nucleus pulposus (NP), and vertebral endplates.[11]

The anulus consists of 60 to 75% water[11,20]. Collagen fibers make up 50 to 70% of its dry weight.[11,20,21] Type I collagen constitutes 80% of the collagen in the outer AF.[22] Type II collagen is absent in the outer AF[21], but is found increasingly in the deeper lamellae[22]. The collagen fibers of the anulus are arranged in concentric rings around the nucleus. These concentric sheaths are called lamellae. Authors disagree on the number of lamellae: observations differ between 10 and 21.[23-25] This number depends on the location in the disk: the number of layers is maximal in the lateral aspects of the disk, less anteriorly and least posteriorly.[25] Lamellae are thicker towards the center and in the anterior and lateral disk; posteriorly they are thinner and packed together more tightly causing the AF to be thinner posteriorly.[11] The posterior disk is concave which

offsets the thinner posterior AF: cross-sectional anular area is greater than if the disk were posteriorly convex. This increases the resistance of the posterior AF to trunk flexion.[11] Within each lamella collagen fibers are arranged parallel to each other. Generally, all fibers in one lamella are oriented at an angle of 65-70 degrees from the vertical axis of the spine.[11,26] Fibers in adjacent lamellae are oriented in opposite directions: fiber direction in one lamella may be 65 degrees in relation to the vertical axis of the spine, whereas in the adjacent deep and superficial lamellae the fibers will run at an angle of -65 degrees.[20] Lamellae do not necessarily completely encircle the NP. An incomplete lamella is one that ceases to pass around the circumference of the disk; around its terminal edge lamellae superficial and deep to it either approximate or fuse.[11] In any given quadrant 40% of lamellae are incomplete.[11] Layer interruption is greatest posterolaterally[25]: in this area some 50% of the lamellae are incomplete.[11] Intramolecular and intermolecular crosslinks stabilize the collagen network and increase its tensile properties. Crosslink content is higher in the inner than the outer region of the AF.[27] Elastic fibers concentrated near attachment sites of the AF with the endplate make up 10% of the dry weight of the AF; they course circularly, obliquely, and vertically within the lamellae.[11]

Chondroitin-6-sulphate, chondroitin-4-sulphate, keratan-sulphate and hyaluronic acid are the glycosaminoglycans (GAGs) found in the IVD.[11] GAGs are polyanionic: they have a high concentration of fixed negative charges. Their sulphate and carboxyl groups generate a substantial swelling pressure by way of charge-to-charge repulsion and electrostatic interaction with water molecules.[20,27] Proteoglycans (PGs) consist of a core protein to which the polyanionic GAGs are attached.[28] PGs make up 20% of the dry weight of the anulus. Of the PGs in the AF, 50 to 60% are in aggregated form, i.e. joined by a link protein to a hyaluronic acid chain.[11] This aggregation immobilizes the GAGs in the collagen network and may partly protect them against proteolytic degradation.[28] PGs serve multiple functions. They stabilize the collagen fibers by way of electrostatic and covalent bonds.[11] The high swelling pressure of their constituent GAGs keeps the collagen network extended and restricts fluid loss from the disk allowing it to perform its mechanical function.[28] Transport of nutrients and oxygen through the matrix depends on composition and organization of the macromolecular framework and matrix water content, both largely determined by PG concentration.[22] PGs prevent or modulate diffusion of large, possibly harmful solutes.[20,29] The water electrostatically bound to the PGs is not distributed uniformly over the AF: PG concentration decreases from the inner to the outer AF and so does water content.[11] Water content is also higher in the dorsal and dorsolateral and lower in the ventral AF.[20,30]

The disk is essentially avascular.[11] The exception is the outer AF which is well nourished by branches of the metaphysial arteries.[11] This may explain the presence of aerobically functioning fibroblasts in the peripheral AF; chondrocytes found in the more central AF are more capable of anaerobic metabolism. However, nutrient supply via this route decreases within a few millimeters into the AF.[28] Osmotic pressure is the suction pressure with which concentrated solutions draw water through semipermeable membranes. Colloid osmotic pressure is the osmotic pressure of polymolecular solutions. We can consider the limiting layers of the AF and vertebral endplates as semipermeable membranes.[30] Swelling pressure of polyanionic GAGs and colloid osmotic pressure as a result of molecules produced during synthesis by the cells cause nutrients and oxygen essential for cell function to travel from bloodvessels at the margin of the disk through the matrix.[29] Oxygen concentrations in the center of the disk are approximately 2 to 5% of those found peripherally.[11,29] Metabolism in the more central AF and NP is mainly anaerobic resulting in lactic acid production.[29] The negatively charged matrix and high lactic acid concentrations cause an acidic pH in the disc[29]. Optimal PG and protein synthesis depend on maintaining the pH within a narrow range.[29] Cell density in the adult disk is highest in the outer lamellae; yet, metabolic activity is highest in the anaerobic chondrocytes of the midanular region.[28] Inability to maintain normal disk homeostasis as a result of changes in the vascular and osmotic processes within the disk will affect pH and therefore cell function. The resulting decreased production of collagen and PGs will negatively affect mechanical characteristics of the disk. Altered pH levels play an important role in the process of disk degradation[11]; we will discuss this in monograph 11.2.4. As discussed earlier PGs also prevent large serum proteins such as immunoglobulins (IGs) from entering the disk. IGs have been implicated in auto-immune reactions leading to degradation of disk tissue.[29] PGs also prevent ingrowth of nerves and blood vessels into the disk; these structures could be the cause of diskogenic pain in degenerated disks. A pH of 6.5 has been measured in degenerated human IVDs. Smoking is strongly associated with disc degeneration and has been shown to decrease intradiskal pH levels.[29]

The center portion of the disk is made up by the nucleus. There is no clear boundary between the NP and the AF, but rather a gradual merging between the outer NP and the central AF. The nucleus consists of 70 to 90% water. PGs make up 65% of the dry weight of the NP.[11] Keratan sulphate concentration is greater in the nucleus than in the AF.[31,32] Only 25% of the nuclear PGs are aggregated. The majority of the PG molecules lack a functional binding site allowing them to bind to hyaluronic acid. Therefore, they remain non-aggregated.[11] Collagen fibers make up 15 to 20% of the dry weight of the NP.[11,24] The NP contains only type II collagen.[11,21] Covalent bonds are abundant possibly playing a role in fiber stabilization and in connecting the fibers to the gel matrix.[21] The remainder of the NP consists of elastic fibers, non-collagenous proteins, and chondrocytes, the latter predominantly located near the vertebral endplates.[11]

The vertebral endplate covers the area of the vertebral body encircled by the ring apophysis. It is 0.6 to1 mm thick.[11,33] The endplate aids in the diffusion of nutrients, prevents the loss of smaller PGs, and acts as a physical barrier preventing the NP from bulging into the marrow of the vertebral body.[33] Near the vertebral body it consists of hyaline cartilage; towards the NP it is made up of fibrocartilage.[11] Collagen accounts for 50 to 70% of the dry weight of the endplates.[21] The collagen fibers of the disk enter the endplate thus enclosing the NP.[11,33] This parallel and horizontal arrangement of the collagen fibers gives the endplate a role in resisting nuclear axial pressure.[33] In younger disks the outer lamellae insert into the vertebral endplate which at that time covers the entire superior and inferior surface of the vertebral bodies. The ossifying ring apophysis absorbs these collagen fibers into the bone.[11] Over 10% of the surface of the endplate subchondral bone is deficient; here the marrow cavity abuts or penetrates into the endplate facilitating diffusion[11]. In other parts of the endplate small capillaries contact the uncalcified cartilage of the endplates.[33] PG and water content increase in the endplate towards the disk; collagen content increases towards the bone.[33] The endplates are functionally part of the disk. Not only are the endplates only weakly attached to the vertebral bodies and strongly attached to the disk, but at the chemical level the endplate resembles the rest of the disk and does therefore not form an additional barrier to diffusion, at least for small nutrients as sulphate, glucose, amino acids , and oxygen.[11,33]

Matrix metalloproteinases (MMPs) are proteolytic enzymes found in the disk. MMP-1 (collagenase) chemically breaks down type II collagen. MMP-2 (gelatinase) further breaks down the fragments of type II collagen produced by collagenase. MMP-3 (stromelysin) breaks down type II, IX, and XI collagen, fibronectin, and aggregated molecules. These proteolytic enzymes are secreted initially as inactive forms that can be subsequently activated by agents such as plasmin or inhibited by proteins known as tissue inhibitors of metalloproteinases. An imbalance between activation and inhibition may play a role in matrix and disk degradation.[11] We will discuss this in monograph 11.2.4.

Nerves penetrate the IVD and sometimes extend as far as the middle third of the AF; however, the innervation is usually limited to the outer third of the anulus.[23] The sources of the nerve endings in the disk are two extensive microscopic nerve plexuses that accompany and innervate the anterior and posterior longitudinal ligaments.[11,23] The anterior plexus derives from the 2 anterolaterally located sympathetic trunks and branches from the grey rami communicantes. The sinuvertebral nerves, the grey rami communicantes, and the ventral rami of the spinal nerve contribute to the posterior plexus.[11,23] The grey rami communicantes form a less pronounced lateral plexus that connects the anterior and posterior plexuses.[11] Paris and Nyberg[34] reported that branches from the mixed spinal nerve found were associated with the posterolateral aspect of the disk; these probably are also just contributors to the posterior plexus. The disk contains both free nerve endings and encapsulated receptors. We find some of the free nerve endings in the vicinity of blood vessels; therefore, these probably have a vasomotor or vasosensory function.[11] The majority of free nerve endings is not associated with blood vessels and has neurotransmitters typical of sensory neurons: they probably serve a sensory function.[23] Encapsulated receptors in the disk resemble Ruffini end organs, Ruffini corpuscles, and Pacini corpuscles.[11,35] Ruffini end organs are similar to Golgi tendon organs: they are high-threshold slowly adapting mechanoreceptors that are only active at the extremes of range of motion.[35,36] Ruffini corpuscles are slowly adapting nerve end organs sensitive to static stretch providing awareness of joint position and movement.[37] Pacinian corpuscles are the least common in the disk.[35] These fast adaptive corpuscles have a role in sensing fast motion and acceleration.[37] Encapsulated receptors were only found in the outer 2 or 3 lamellae of the AF and seemed more prevalent in the disks of patients with LBP[35] prompting Roberts et al[35] to postulate a possible role of sensitized disk mechanoreceptors in the production of LBP and the commonly associated increased paraspinal muscle tone. Altered thresholds to depolarization or disruption of mechanoreceptors may play a role in coordinative impairments in LBP.

Zygapophysial joint

The zygapophysial joints (ZJ), formed by articulation of the superior and inferior articular facets, are paired posterior synovial or diarthrodial joints. Together with the IVD they link two adjacent vertebrae and along with the IVD they form the basic functional unit of the spine: the motion segment.[38] The joints are also commonly referred to as the intervertebral, facet or zygapophyseal joints. The articular facets are ovoid in shape.[11] Panjabi et al[39] extensively studied facet geometry. Please consult their article for facet measurements. Their findings did not support the statement by Bogduk[11] that the superior facets are smaller than their articulating inferior counterparts.

Fetal and infant ZJs are oriented in the frontal plane. By posterior growth from their lateral margins they change during early childhood and become curved or biplanar joints with posterior parts that are more or less parallel to the sagittal plane.[40] In horizontal plane transsection the ZJs vary from a planar shape to a pronounced J- or C-shaped curve.[11] The larger concave superior facet faces posteromedially; it "embraces" the smaller convex anterolaterally facing inferior facet.[40] Left-right asymmetries in the amount of ZJ curvature are frequent.[38]

The ZJ orientation is the angle made by a plane passing through the anteromedial and posterolateral-most ends of the joint cavity with respect to the (mid)sagittal plane.[11] This measure does not account for the facet shape discussed above. Bogduk[11] reported a joint angle for most L1-L2 joints of approximately 15^0, 30^0 for L2-L3, and 45^0 for L3 to S1. He also reported L4 to S1 angles between 45 and 90^0. Haeg and Wallner[41] reported joint angles in non-herniated motion segments of $50+/-11^0$ at L4-L5 and $50+/-9^0$ at L5-S1. Ahmed et al[42] reported ZJ angles ranging from 5 to 55^o (median 29^0) for the L2-L3 joint and a range of $10-85^0$ (median 51^o) for the L4-L5 ZJ. Facet angle asymmetry or articular tropism is common and has been implicated in the etiology of disc herniation.[10,38,41,42] We will discuss this in the section on biomechanics.

As all diarthrodial joints the ZJ articular facets are covered with hyaline cartilage. The cartilage is thickest in the center of the joint with a height of 2 to 4 mm.[11,38] The joint capsule consists of an outer fibrous layer and an inner synovial lining. This synovial membrane attaches at the margins of the articular cartilage and reflects to cover the various intra-articular structures.[11] Around the dorsal, inferior and superior margins of the joint the fibrous capsule is formed by collagen fibers passing more or less transversely from the one to the other articular process.The inferior and superior portions of the capsule attach further away from the cartilage-bone interface and thus create subcapsular pockets over the inferior and superior edges of both superior and inferior articular processes. In the normal joint these recesses are filled with fat. A tiny foramen in the superior and inferior part of the capsule allows for passage of this intracapsular fat into the extracapsular space during movement. On the ventral aspect of the joint the fibrous capsule is totally replaced by the flaval ligament which attaches close to the osteochondrous junction. The fibrous capsule is made up of 2 layers: the outer layer consists of densely packed collagen fibers and the inner layer contains irregularly arranged elastic fibers.[11] Posteriorly, the joint capsule is thick; the fibrous capsule is reinforced by a fascicle from the multifidus muscle passing inferolaterally from a spinous process and inserting into the joint capsule and the adjacent mamilary process.[11,40] This aspect of the joint separates when this fascicle is sectioned; it functions to actively maintain congruous joint contact in the posterior part of the joint.[40] The superior and inferior parts of the fibrous capsule are loose and abundant: superiorly the capsule balloons up towards the base of the next transverse process, inferiorly it extends back over the posterior aspect of the lamina.[11]

Bogduk[11] distinguished 2 types of intra-articular ZJ structures. Fat is one of these structures. The other type of intra-articular inclusions are the meniscoid structures of which there are 3 types. The *connective tissue rim* is the simplest and smallest of the meniscoid inclusions: it is little more than a wedge-shaped thickening of the internal surface of the capsule which at the dorsal and ventral aspects of the joint fills the space left by the curved margins of the articular cartilages. The connective tissue rims may serve to transmit some load on impaction of the joint surfaces as they increase the surface of the contact area. The second type, the *adipose tissue pad*, is located mainly at the superoventral and inferodorsal poles of the joint. In the lumbar spine it projects about 2 mm into the joint and consists of a fold of synovium enclosing some fat and blood vessels. At its base the synovial membrane reflects onto the joint capsule and is continuous with the synovium of the rest of the joint. Similarly, the fat within this structure is continuous with the other fat in the joint. The third type of inclusion, the *fibro-adipose meniscoid*, is the largest of the intra-articular meniscoids: it projects up to 5 mm into the joint from the inner surface of the superior and inferior capsules and is made

up of a leaf-like fold of synovium which encloses fat, collagen, and some blood vessels. The fat is located mainly in the base of the meniscoid where it is continuous with the other fat in the joint and communicates with the extracapsular fat through the foramina in the superior and inferior capsule. The apex of the meniscoid consists mainly of densely packed collagen. Adipose tissue pads and fibro-adipose meniscoids may have a protective function.[11] During flexion the inferior articular facets slide up 5 to 8 mm along the superior facets exposing the upper portion of the inferior facets and the lower portion of the superior facets. The inclusions cover these otherwise exposed surfaces and maintain a film of synovial fluid between themselves and the joint surface thus ensuring lubrication of joint surfaces upon returning to neutral[11]. Bogduk[11] hypothesized a role for the fibro-adipose meniscoid in the clinical syndrome of "acute locked back": upon flexion the meniscoid may be pulled out of the joint space and may fail to re-enter upon attempted extension impacting the edge of the cartilage, buckling, and acting as a space-occupying lesion stimulating nociceptors in the distended joint capsule. Bogduk[11] and Taylor and Twomey[40] mentioned a fourth type of meniscoid, likely the result of degenerative or age-related processes; we will discuss this in monograph 11.2.4.

The ZJs receive their main innervation from the medial branches of the dorsal ramus of the spinal nerves. The L1 to L4 medial branches of the dorsal ramus run across the top of their respective transverse processes and pierce the dorsal leaf of the intertransverse ligament, then course along the junction between the transverse and superior articular process and through the mamillo-accessory notch covered by the mamillo-accessory ligament. The L5 medial branch crosses the ala of the sacrum rather than the transverse process, but after that has a similar course. After crossing the lamina the medial branches subdivide into branches supplying the segmental multifidus muscle, interspinous muscle and ligament, and ZJ. Each medial branch supplies the joint at the same level and the level above and below by an ascending and a descending articular branch which split off just after the mamillo-accessory ligament.[11,43] The ventral part of the ZJ and the flaval ligament are innervated by a direct dorsal branch of the sinuvertebral nerve.[36] Encapsulated, unencapsulated, as well as free nerve endings give the fibrous capsule nociceptive and proprioceptive potential.[11] Substance P is a neurotransmitter commonly found in nociceptive fibers.[43] Beaman et al[43] found fibers containing this neurotransmitter in erosion channels and the marrow spaces of the subchondral bone in degenerated ZJs; this may implicate these joints in the etiology of LBP.[43] Giles and Taylor[44] found nerve fibers in the synovial intra-articular inclusions. Based on location and size they divided the structures in vasomotor and nociceptive nerves thus further supporting the possible role of the intra-articular inclusions in the production of LBP.

Sacroiliac joint

The sacroiliac joint (SIJ) is formed by articulation of the sacral and ilial auricular surfaces.[10] The average square area of the joint is 1.5 cm^2 at birth, 7 cm^2 at age 7, and 17.5 cm^2 in the adult[45]. The joint surfaces have been described as L-shaped with a shorter, superior, vertically oriented part extending across the lateral surface of S1 and a longer, inferior, more horizontally aligned part extending across S2 and across a variable distance on the lateral aspect of S3.[11,46] The auricular surfaces vary widely in size and shape between individuals and even from left to right in the same individual.[45,47] Schunke[47] noted that the shorter superior arm is occassionally absent.

The iliac cartilage resembles fibrocartilage: chondrocytes are clumped together and interspersed between bundles of collagen fibrils. These fibrils are oriented parallel to the joint surface, even in the deeper layers of the iliac cartilage. Iliac cartilage is rough and has a bluish color due to the closely underlying trabecular bone. Histologic analysis, however, shows the collagen to be type II collagen typical of hyaline cartilage rather than the expected type I collagen found in fibrocartilage.[45] The sacral cartilage is typical hyaline cartilage with a smooth and creamy-white appearance.[45,48] It is consistently reported to be 1.5 to 3 times thicker than the iliac cartilage; this may explain earlier wear on the iliac cartilage.[48] Sacral adult cartilage is thicker in the anterior than in the posterior and superior aspect of the joint surface, measuring between 1 to 3 mm in depth; the iliac cartilage averages 1 mm in depth.[45,49]

At birth the auricular surfaces are flat and oriented parallel to the long axis of the lumbar spine.[15,45] A gradual change in joint contour and orientation occurs with age presumably as a result of the mechanical forces of growth and bipedal gait.[45] Authors disagree on the age at which these joint surface irregularities first appear reporting irregularities already in fetuses[50], in a 12 year old boy[51], or no irregularities in a 24 year old man[52]. Schunke[47] described the frequent presence of a longitudinal iliac ridge fitting into a sacral groove, but also stated that the reverse situation or any combination of grooves, ridges, eminences, and

depressions may be found in the adult sacroiliac joint. Kapandji[19] mentioned a central circular iliac ridge with on either side a groove with the opposite topography of a central circular groove with 2 accompanying ridges on the sacral surface. The center of the circular ridges and grooves is in the area of the iliac tuberosity, suggested by Farabeuf as the location for an axis of rotation of the SIJ.[19] Weisl[50] specifically stated he found no arcuate sacral gutter and corresponding iliac elevation as decribed by other authors. During dissection Bowen and Cassidy[49] found macroscopic irregularities starting to develop during the teenage years; they described the convex iliac ridge with corresponding sacral groove present by the beginning of the third decade. Brunner et al[52] found close congruity of opposing joint surfaces in 14 SIJs. Female joints had circular iliac elevations with corresponding sacral depressions around an axis located in the iliac tuberosities. Older male surfaces had interlocking irregularities; the surfaces of one younger man were almost level permitting motion in all directions. Vleeming et al[51] found complementary ridges in 47 cadaveric SIJs with joint shape irregularities more pronounced in men than women.

As a result of these and other age-related changes discussed in monograph 11.2.4. and the unique nature of the joint cartilage the SIJ has been classified erroneously as an amphiarthrosis or synarthrosis.[45] An amphiarthrosis is a joint in which 2 hyaline cartilage articular surfaces are joined by fibrocartilage; in a synarthrosis articular surfaces are joined by fibrous tissue[45]. However, the SIJ fulfills all criteria for a diarthrosis or synovial joint[45,49]:

- A joint cavity is present containing synovial fluid.
- The adjacent bones have a ligamentous connection.
- The joint has an outer fibrous capsule with an inner synovial lining.
- The joint consists of cartilage covered surfaces allowing for motion.

The anterior border of the auricular surface coincides with the anterior border of the bony sacrum.[11] The ventral sacroiliac ligaments are merely a thickening of the anterior joint capsule.[46] Paris[53] reported research describing an anterior capsule synovial recess which may be mistaken for an anterior capsular tear with joint infiltration. The posterior capsule is described as rudimentary, absent[45] or as blending with the interosseus ligament.[49] The interosseus ligament thus forms the posterior border of the joint. Lavignolle et al[54] described the SIJ as a diarthro-amphiarthrosis: they considered the syndesmosis formed posterosuperiorly by the interosseus ligament between ilium and sacrum a functional complement to the diarthrodial anteroinferior auricular joint described above.

Schunke[47] found supernumerary articular facets dorsal to the auricular surfaces in 16 of 47 pelves, varying in diameter from 2 mm to over 1 cm. Microscopic evaluation of one such joint demonstrated surfaces composed of fibrocartilage. Accessory SIJs may be the result of weight-bearing stresses.[45] Their prevalence has been estimated between 8 and 35.8%.[45] Prevalence increases with age; they are rarely found before the fourth decade of life.[45,48] Accesory joints can be single, double, or even triple on one side[47]; they can occur unilaterally or bilaterally and sometimes they are true synovial joints.

Authors disagree on the innervation of the SIJ; also, none of them report direct observations. Greenman[55] reported innervation primarily from the S1 spinal nerve. Winkel[56] reported dorsal innervation by the dorsal rami of the S1 and S2 and ventral innervation by the ventral rami of (L3)L4 to S1 (S2) and the superior gluteal nerve. Alderink[46] reported the L5 to S2 posterior rami for posterior and the L3 to S1 ventral rami and branches of the superior gluteal nerve (L5-S2) for anterior SIJ innervation. Free, encapsulated, and complex unencapsulated nerve endings in the SIJ capsule may allow for a nociceptive and proprioceptive function.[45] There have been no reported studies on the innervation of accessory SIJs.[48] It is obvious a consensus on SIJ innervation is lacking. The multisegmental innervation mentioned has the theoretical potential for widespread referral.

Sacrococcygeal and intercoccygeal joints

The sacrococcygeal joint is usually an amphiarthrosis.[19,57] The convex joint surfaces on the apex of the sacrum and the concave opposing surface on the base of the coccyx are oval with a larger mediolateral diameter.[9,19,57] The sacrococcygeal joint may consist of an IVD, a synovial joint, or an IVD with a cleft parallel to the vertebral endplates.[18] In young people fibrocartilaginous disks are interposed between the coccygeal vertebrae.[9] Intercoccygeal joints may be true synovial joints.[18] Ossification of the intercoccygeal joints is common in men; in women this does not usually occur until later in life.[9] Sometimes the entire coccyx is ossified, in other patients this ossification is limited to the sacrococcygeal joint.[18]

Pubic symphysis

The pubic symphysis is also an amphiarthrosis.[19,57] Oval hyaline cartilage covered joint surfaces on both pubic bones are connected by a fibrocartilaginous disk.[9] Fibers of this disk run in a partly transverse and partly spiraling orientation. Located in the middle of the disk is a posterosuperior sagittal plane cleft which is not covered by a synovial membrane. Gender differences are present in the pubic symphysis. In men the interpubic disk is relatively higher, thinner and more fibrous with a smaller cleft than in women.[10] Abramson[58] found an average radiographic width of the joint cavity of 4.4 mm in men. Width in women seemed dependent on the amount of children they had had: the width in those without children measured on average 4.09 mm, in those with one child 4.15 mm, and in those with multiple children 4.15 mm.

Ligamentous anatomy

Lumbar spine

The anterior longitudinal ligament (ALL) extends along the anterior surfaces of the vertebral bodies. Caudally, it is continuous with the anteromedial aspect of the SIJ capsule.[59] It is composed of collagenous fibers with very few elastic fibers.[60] The ALL has multiple layers.[9] The shortest, deepest fibers attach to the margins of adjacent vertebral bodies. Several layers of increasingly longer fibers overlying the deep unisegmental fibers span 2 to up to 5 segments. The main attachment sites are the bone and periosteum of the lower and upper margins of the vertebral bodies. Secondary attachments are the concave anterior surfaces of the vertebral bodies: the main portion of the ligament bridges this concavity, but some fibers from its deep surface blend with the periosteum of the anterior vertebral body. Loose areolar tissue, blood vessels and nerves fill the rest of this space. The ligament only forms loose attachments to the anular fibers of the IVD; the space between these 2 structures likewise is filled with loose areolar tissue.[11] The crura of the diaphragm attach to the anterior vertebral bodies of L1 to L3. Bogduk[11] noted that what used to be interpreted as fibers of the ALL may in fact be the tendinous fibers of the diaphragm found to extend caudally along the anterior spine below L3.

The posterior longitudinal ligament (PLL) extends along the posterior aspect of the vertebral bodies inside the vertebral canal. It also is mainly collagenous with very few elastic fibers.[60] The ligament broadens as it passes over the IVDs and narrows to pass around the bases of the pedicles.[9,59] This gives the ligament a serrated, saw-toothed or hourglass appearance.[11] In contrast to its anterior counterpart its attachments are strongest to the IVD.[59] It does not attach to the concave posterior surface of the vertebral bodies: the basivertebral veins occupy this space.[9] As in the ALL its superficial fibers are longer and span 3 to 5 segments.[9,11] The deeper layer extends between adjacent vertebrae.[9] Bogduk[11] stated that the deepest fibers span 2 disks.

The flaval ligament is unique in that it consists of 80% elastic and 20% collagen fibers compared to the mainly collagenous composition of the other ligaments.[11,59] These elastic fibers are responsible for the yellow color for which the ligament is named: flavum is Latin for yellow.[59] The ligament contains 2 morphologically different types of elastic fibers[11,61]: the regular fibers in the central part of the ligament and eulanin fibers, a combination of elastin and tubular microfibrils, at the sites of bony attachment. The collagen fibers in this ligament do not organize into the normal kind of crimped fibers we find in other longitudinal ligaments. Instead, they are dispersed in the elastic tissue. Their orientation only becomes more aligned as the ligament stretches causing increased stiffness.[60] The fibers of the medial portion of the flaval ligament run longitudinally between the ventral surface of the lamina of a superior vertebra to the dorsal and superior margin of the lamina of an inferior vertebra.[9] The ligament becomes more fibrocartilaginous at its attachment sites to the bone with an increased density of chondroblasts.[62] The lateral portion of the ligament passes in front of the ZJs and forms their anterior joint capsule.[11] Elastic fiber content decreases as the ligaments connect in the midline and become the interspinous ligament.[59]

The interspinous ligament (ISL) connects adjacent spinous processes from their base to their apex. It is continuous with the flaval ligaments ventrally and the supraspinous ligaments dorsally.[9] Histologically, the ISL mainly consists of collagen fibers, but the density of elastic fibers increases ventrally near its connection to the flaval ligament.[11,61] It is more fibrocartilaginous near its attachment to the bone with an increased density of chondrocytes.[59,61] There have been multiple conflicting descriptions of its orientation.[59] We can best describe its orientation as a fan: the narrow or proximal end of the fan blends with the flaval ligament and the broad end extends posteriorly towards the tips of the spinous processes. In the center of the ligament

the fibers are oriented parallel to the spinous processes; distally the fibers fan posterocranially and posterocaudally attaching to these processes.[59]

Gray's Anatomy[9] described the supraspinous ligament (SSL) as a strong fibrous cord connecting the apices of the spinous processes. Willard[59] described it as a part of the interspinous-supraspinous-thoracolumbar ligamentous complex allowing the thoracolumbar fascia (TLF) to influence vertebral motion. The SSL is well-developed in the upper lumbar region, but its lower limit varies. It terminates at L3 in 22%, at L4 in 73%, and bridges L4-5 in only 5% of the population; at L5-S1 it is frequently absent.[11] Bogduk[11] described the SSL as consisting of 3 layers: a superficial subcutaneous layer consisting of longitudinal collagenous fibers spanning 3 to 4 spinous processes, a middle layer of about 1 mm thick consisting of interconnecting tendinous fibers of the dorsal layer of the TLF and the aponeurosis of the longissimus thoracis muscle, and a deep layer of strong tendinous fibers derived from the longissimus thoracis. The deep layer is reinforced further by tendinous fibers from the multifidus muscle. Hukins et al[60] found no single continuous longitudinal collagenous structure and describe tendons of the erector spinae blending into the SSL. Bogduk[11] stated that this parallel orientation of tendinous fibers with their attachment to the spinous processes creates only the semblance of a supraspinous ligament. Only its superficial layer, which is displaced from the spinous processes, lacks continuity with muscle. Therefore, we cannot consider it a true ligament, but rather a condensation of the membranous layer of the superficial fascia anchoring the midline of the skin to the TLF.[11]

The iliolumbar ligament does not directly stabilize the sacrum, but plays a role in both SIJ and lumbosacral mechanics.[46] Therefore, we may consider it an accessory sacroiliac ligament. DonTigny[63] hypothesized SIJ dysfunction to influence iliolumbar ligament tension destabilizing the lower lumbar segments with a resulting higher predisposition for disk herniations. Descriptions of this ligament vary. Gray's Anatomy[9] described the ligament as attaching to the lateral, anterior, and inferior aspect of the transverse process of L5 and from there coursing laterally, attaching by 2 main bands to the pelvis. The more inferior band is called the lumbosacral ligament. This ligament attaches to the base of the sacrum and blends with the anterior SIJ ligaments. The more superior band inserts into the iliac crest immediately anterior to the SIJ. Bogduk[11] described 5 parts to this ligament. Hanson and Sonesson[64] found only an anterior and a posterior part on dissection, both originating on the L5 transverse process and inserting into the iliac tuberosity. They found no evidence of attachment to L4 in any specimen. Uhthoff[65] did report an origin from the L4 transverse process in 2 of 12 fetuses. There has been some discussion as to development of the iliolumbar ligament. Luk et al[66] found that the iliolumbar ligament consisted completely of muscle fibers derived from the quadratus lumborum up until the first decade of life. In the second to fourth decade the ligament "ligamentized" and lost its muscle fiber content, until in the fifth decade no muscle fibers could be found. Macintosh and Bogduk[67] suggested that the iliolumbar ligament represents the "ligamentized" L5 fascicle of the iliocostalis lumborum. In contrast, Uhthoff[65] found well-formed iliolumbar ligaments with an anterior and posterior band in fetuses over 11 weeks of gestational age. Hanson and Sonesson[64] also noted no evidence of metaplasia. The quadratus lumborum muscle has an attachment site on part of the iliolumbar ligament.[11] Lee[68] hypothesized that this muscle may contribute to lumbosacral stability by increasing tension in the iliolumbar ligament.

Bogduk[11] described 3 "false" ligaments in the lumbar spine. The intertransverse ligaments extend between transverse processes. Their collagen fibers are not as densely packed, nor are they as regular as in other ligaments. Their appearance is more like that of a membranous part of the fascial system separating paravertebral compartments. According to Bogduk[11] they do not have a mechanical function. The intertransverse ligaments are absent at the lumbosacral junction; the iliolumbar ligament replaces them there. Transforaminal ligaments, reportedly present in 47% of the population, are most likely fascial thickenings of the intertransverse ligament. They may decrease the available IVF diameter. The mamillo-accessory ligament may be a semispinalis tendon rather than a true ligament, connecting the tips of the ipsilateral accessory and mamillary processes of 1 lumbar vertebra.[11]

Longitudinal ligament innervation is covered in the review of the innervation of the IVD. A direct dorsal branch of the sinuvertebral nerve and the medial branch of a dorsal ramus both innervate the flaval ligament.[36] The ISL and SSL are also innervated by this medial branch. Yahia et al[36] and Jiang et al[37] described capsulated and free nerve endings in ISL, SSL, and flaval ligament, hypothesizing a proprioceptive role for these structures.[37]

Sacroiliac joint

The interosseus sacroiliac ligaments, also known as the short axial ligaments[46], are the strongest ligamentous connections between sacrum and ilium.[9,45] The ligaments consist of series of short, strong fibers running between the sacral and iliac tuberosities. The more superficial fibers may blend with the fibers of the short posterior sacroiliac (SI) ligament, but frequently the dorsal rami of the sacral spinal nerves separate the interosseus ligaments from the overlying dorsal ligaments.[46]

We earlier discussed the ventral SI ligaments. The dorsal SI ligaments consist of a deeper part, the short posterior SI ligament, and a more superficial portion called the long posterior SI ligament. The short posterior SI ligament runs nearly horizontally from the first and second transverse tubercles on the dorsal surface of the sacrum to the iliac tuberosity. The more superficial long posterior SI ligament partly covers the short portion and runs longitudinally from the second, third, and fourth transverse tubercles to the posterior superior iliac spine (PSIS).[9,46,69] Laterally, the dorsal SI ligaments are continuous with the sacrotuberous ligament; medially they connect to the posterior layer of the TLF.[46]

The sacrotuberous ligament is a broad, flat, fan-shaped ligament derived from the posteroinferior aspect of the SIJ capsule.[59] It resists sacral nutation.[46,59] Sacral nutation, also known as sacral flexion, is defined as the motion in which the sacral promontory moves anteroinferiorly and the coccyx and apex of the sacrum move posterosuperiorly. The multiplanar SIJ orientation causes the iliac crests to approximate and the iliac tuberosities to separate during nutation. With sacral counternutation or extension the opposite movements occur.[19,46] We can divide the sacrotuberous ligament into several large fibrous bands. The lateral band connects the posterior inferior iliac spine (PIIS) to the ischial tuberosity. The medial band runs between the lateral part of the coccyx and the ischial tuberosity. The thinner superior band attaches the PSIS to the lateral upper part of the coccyx. Several central bands have their origin on the lateral band and insert onto the lowest transverse tubercle of the sacrum.[59] Gray's Anatomy[9] also mentioned an attachment site on the fourth transverse tubercle. Some of the fibers continue on in an anterior direction after insertion into the ischial tuberosity along the inferior border of the ramus of the ischium as the falciform process. The obturator fascia attaches to this process[9,10] and thus establishes an anatomical connection between the dorsal SI stabilizing system and the pelvic floor, which we will discuss later. The tendons of the deepest laminae of the multifidus muscle often extend into the superior surface of the sacrotuberous ligament.[59] In a dissection study of 12 pelves Vleeming et al[70] found the dorsal fascia of the piriformis muscle to be continuous with the sacrotuberous ligament in all cases. Also in all cases the gluteus maximus muscle took part of its origin from the ligament. Gray's Anatomy[9] described the sacrotuberous ligament as continuous with the tendon of the long head of the biceps. Vleeming et al[70] dissected 22 pelves and found unilateral fusion of the long head of the biceps tendon with the sacrotuberous ligament in 5 pelves, bilateral fusion in 6 pelves. Bilateral fusion was more common in female specimens. In 2 pelves Vleeming et al[70] noted no connection between the tendon of the long head of the biceps femoris and the ischial tuberosity. Van Wingerden et al[71] also described hardly any connection between the ischial tuberosity and the biceps femoris tendon-lateral part of the sacrotuberous ligament-combination in 6 of 10 dissections. Instead, they found a sliding mechanism, the ischial bursa, between the ischial tuberosity and the tendon-ligament complex. In the other preparations they found the biceps femoris tendon and the sacrotuberous ligament fixed to the ischial tuberosity. In all specimens they found the medial part of the sarotuberous ligament connected to the ischial tuberosity.

The sacrospinous ligament derives from the anteroinferior SIJ capsule.[59] It is a thin, triangular ligament with its base on the lateral margins of the lower part of the sacrum and upper part of the coccyx and the inferior aspect of the SIJ capsule.[9,10,59] It inserts onto the ischial spine transforming the greater and lesser sciatic notch into the greater and lesser sciatic foramen, respectively.[9,10] Posteriorly the sacrospinous ligament partly connects to and is covered by the sacrotuberous ligament.[9] The anterior surface of the sacrospinous ligament serves as an attachment site for the coccygeus muscle.

We discussed the innervation of the SIJ ligaments in our review of innervation of the SIJ. Alderink[46] mentioned a plexus formed by the L5 through S2 posterior rami located between the interosseus ligament and the posterior SI ligaments as the source of innervation of the SI ligaments. Bernard and Cassidy[45] mentioned the presence of unmyelinated free nerve endings in the SI ligaments able to transmit nociceptive and thermal stimuli.

Sacroccocygeal and intercoccygeal joints

The ventral sacrococcygeal ligament is a continuation of the ALL.[9,19] The dorsal sacrococcygeal ligament originates at the sacral hiatus and inserts into the dorsal surface of the coccyx. It consists of a longer superficial part with an origin on the median sacral crest and a deeper short portion which is a continuation of the PLL.[9] The lateral sacrococcygeal ligaments connect the inferior lateral angle of the sacrum to the transverse processes on either side of the first coccygeal vertebra.[9] Kapandji[19] mentioned 3 lateral sacroccocygeal ligaments on either side. The intercornual ligaments connect the sacral cornua to the coccygeal cornua.[9] The sacrococcygeal disk is sometimes called the interosseus ligament.[19,57]

Pubic symphysis

The strong anterior pubic ligament reinforces the anterior aspect of the joint with obliquely and transversely oriented fibers.[57] Extensions of the aponeuroses of the transverse abdominis, rectus abdominis, pyramidalis, internal abdominal oblique, adductor longus, and gracilis muscles further reinforce and blend with this ligament.[19,57] The weaker posterior pubic ligament is a fibrous extension of the periosteum of the pubic bone.[57] The superior pubic ligament is a thick fibrous band that serves as the origin for the rectus abdominis muscle and extends as far lateral as the pubic tubercle.[9,57] The arcuate or inferior pubic ligament reinforces the inferior aspect of the pubic symphysis and blends superiorly with the interpubic disk.[9]

Muscular anatomy

Extensive description of the lumbopelvic muscles and related structures can be found in many sources.[9-11, 68, 72] We will discuss only those structures that play an important role in segmental stabilization of the lumbopelvic region.

Lumbar back muscles

The lumbar back muscles are located behind the plane of the lumbar transverse processes; they are innervated the dorsal rami of the lumbar spinal nerves.[11] Bogduk[11] distinguished 3 groups:

- short intersegmental muscles (interspinales and intertransversarii mediales),
- polysegmental muscles attaching to the lumbar vertebrae (multifidus, longissimus thoracis pars lumborum, and iliocostalis lumborum pars lumborum), and
- long polysegmental muscles crossing the lumbar spine, but not attaching to the lumbar vertebrae (longissimus thoracis pars thoracis and iliocostalis lumborum pars thoracis).

Short intersegmental muscles

The lumbar interspinales are paired muscles located on either side of the ISLs. They connect adjacent lumbar spinous processes. The intertransversarii mediales originate from the mamillary process, accessory process, and mamillo-accessory ligament and insert into the superior aspect of the subjacent mamillary process. Both muscles are quite small and have a considerable mechanical disadvantage because of the location of their attachments. The density of muscle spindles in these monosegmental spinal muscles is between 2 and 6 times that of the longer polysegmental muscles. The medial branch of a dorsal ramus supplies only the segmental muscles that arise from the vertebra with the same segmental number as the nerve.[11] Because of the high muscle spindle density and the unisegmental innervation Bogduk[11] hypothesized a proprioceptive rather than mechanical function for these muscles.

Polysegmental muscles

The multifidus is the largest and most medial of the lumbar back muscles (Figure 3). The shortest fascicles originate at the caudal aspect of the dorsal surface of the lamina and insert into the mamillary process of the vertebra 2 levels inferiorly. Bogduk[11] called these fascicles the laminar fibers. They resemble the lumbar rotatores not mentioned by Bogduk[11], but described in Gray's Anatomy[9] as unisegmental muscles between caudal lamina and a subjacent mamillary process. The L5 laminar fibers have no mamillary process in which to insert. Instead, they insert into an area just superior to the first dorsal sacral foramen. The longer fascicles are arranged in 5 overlapping groups and form the major part of the bulk of the multifidus. They originate from the base and caudolateral aspect of the lumbar spinous processes and by way of a common tendon from the caudal tip of the spinous process. The fascicle from the base of the L1 spinous process inserts into the L4 mamillary process. The fascicles from the common tendon diverge to insert into the mamillary processes of L5 and S1 and the PSIS. The fascicle from the base of the L2 spinous process inserts into the L5 mamillary process and the common tendon fascicles insert into the S1 mamillary process,

the PSIS and an area on the iliac crest just anterior and inferior to the PSIS. The fascicle from the base of the spinous process of L3 inserts into the mamillary process of the sacrum and the fascicles from the common tendon insert into an area extending caudally from the PSIS to the lateral edge of S3. The L4 fascicles insert onto the sacrum medial to the insertion of the L3 fascicles, but lateral to the dorsal sacral foramina, whereas the L5 fascicles insert onto the sacrum medial to these foramina.[11] We discussed the insertion of the deep fibers of the multifidus muscle into the posterior ZJ capsule adjacent to the mamillary processes[11,40], as well as the insertion of the deepest lamina of the multifidus in the sacrotuberous ligament[59]. This latter insertion allows the multifidus to exert tension on the SIJ both directly through its insertions into the sacrum and indirectly through its connection to the sacrotuberous ligament.

The lumbar erector spinae group is located lateral to the multifidus muscle. It consists of 3 muscles[9,67]: the iliocostalis lumborum (pars lumborum) makes up its most lateral portion, the longissimus thoracis (pars lumborum) is located more medially and the spinalis thoracis is the most medial portion. The spinalis thoracis inserts with 3 or 4 tendons into the upper 2 lumbar and lowest 2 thoracic vertebrae.[9] Being mainly aponeurotic in the lumbar region this muscle plays a minor role in the morphology of the erector spinae.[67] The fascicles from the longissimus thoracis pars lumborum originate from the lumbar accessory processes and the medial 3 quarters of the adjacent transverse processes (Figure 4). The L5 fascicle is the deepest and shortest and inserts onto the ventromedial surface of the ilium and the ventral SI ligament. The L1 through L4 fascicles insert onto the PSIS. The fascicles with a more cranial origin cover the fascicles with a more caudal origin, such that their tendons are stacked, thus forming the composite structure of the *lumbar intermuscular aponeurosis* (LIA). This aponeurosis covers the lateral aspect of the lumbar portion of the longissimus thoracis and separates it from the lumbar portion of the iliocostalis lumborum. The lumbar portion of the iliocostalis lumborum consists of 4 large fascicles arising from the lateral one quarter of the L1 to L4 transverse processes and the adjacent middle layer of the TLF (Figure 5). As with the longissimus thoracis (pars lumborum) the more cranial fascicles cover the more caudal fascicles. The L4 fascicle is the deepest and inserts into the ventral margin of the iliac crest along a line extending 5 cm laterally from the lateral aspect of the PSIS. The L3 to L1 fascicles attach progressively more medially and caudally into the lateral aspect of the PSIS covering the lower, shorter fascicles.[11,67]

The dorsal ramus of a spinal nerve divides into a medial, intermediate, and lateral branch.[11] The medial branch innervates the segmental multifidus muscle. Each medial branch supplies only that muscle that arise from the vertebra with the same segmental number as the nerve. The intermediate branches innervate the longissimus by forming a multisegmental plexus within the muscle. The exception to this polysegmental innervation are the fibers of the longissimus which arise from the L5 transverse process; they are innervated exclusively by the intermediate branch of the L5 dorsal ramus. The lateral branch supplies the iliocostalis muscle.[11,72]

Long polysegmental muscles

The *erector spinae aponeurosis* (ESA) covers the lumbar erector spinae. This thick aponeurotic structure is formed almost exclusively by the caudal tendons of the thoracic component of the erector spinae group; it receives only a small contribution from the underlying lumbar multifidus. The thoracic portion of the longissimus thoracis originates from the T1(T2) to T12 transverse processes and adjacent ribs and inserts into the lumbar and sacral spinous processes below L2 and into the sacrum along a line between the S3 spinous process and the caudal aspect of the PSIS. Its caudal tendons form the medial portion of the ESA. The laterally located T12 caudal tendon merges with the posterior aspect of the LIA. The iliocostalis lumborum (pars thoracis) has its origin on the lower 7 or 8 ribs and inserts into the PSIS and dorsal edge of the iliac crest. Its caudal tendons form the lateral part of the ESA.[11,67] The ESA contains no caudal tendons from the lumbar erector spinae and is thus independent from these muscles. In the lower lumbar region it is freely mobile over the dorsal aspect of the fibers of the longissimus thoracis (pars lumborum).[67] Bogduk[11] stated that the ESA moves freely over the surface of all lumbar muscle fibers.

Thoracolumbar fascia

The thoracolumbar fascia (TLF) consists of 3 layers. The thin anterior layer is in fact the ventral fascia of the quadratus lumborum. Medially it attaches to the ventral aspect of the lumbar transverse processes. Here the anterior layer blends with the intertransverse ligaments. Lateral to the quadratus lumborum, the anterior layer blends with the other layers of the TLF. The middle layer attaches medially to the tips of the transverse processes and is directly continuous with the intertransverse ligaments. Laterally it serves as the origin for

the aponeurosis of the transversus abdominis muscle. The posterior layer of the TLF has its origin on the tips of the lumbar spinous processes. It covers the lumbar back muscles posteriorly and blends with the middle layer of the TLF lateral to the lateral margin of the iliocostalis lumborum. At the site of this union the 2 fascial layers form a dense raphe known as the *lateral raphe*.[11,73] The posterior layer of the TLF covers the back muscles from the sacral region, through the thoracic region, as far cranially as the fascia nuchae.[73] It consists of a deep and a superficial lamina. At the L4 to L5 level and dorsal to the sacrum strong connections exist between both laminae. The transversus abdominis and internal oblique muscles are attached indirectly to the posterior layer of the TLF by way of their insertion into the lateral raphe.

Superficial lamina posterior layer TLF

The superficial lamina of the posterior layer is continuous with the the latissimus dorsi and gluteus maximus aponeuroses (Figure 6). A few fibers of the superficial lamina are also continuous with the external oblique and trapezius muscles. Fibers in the superficial lamina are oriented from craniolateral to caudomedial.Cranial to L4 the fibers of the superficial lamina attach to the SSLs and spinous processes. Caudal to L4 or L5 fibers of this lamina attach only loosely or not at all to the midline structures, but cross the midline to attach to the contralateral side. Here they insert into the sacrum, PSIS, and iliac crest. This crossing-over usually occurs at the level of L4, sometimes as high as L2 or L3. At sacral levels the superficial lamina is continuous with the fascia of the gluteus maximus. Fibers here run from craniomedial to caudolateral. Most fibers attach to the median sacral crest, but again some fibers cross the midline to attach to the contralateral PSIS, iliac crest, lateral raphe and fascia of the latissimus dorsi. This crossing-over occurs at the L4 or L5 level, and in some specimens as far caudally as S1 or S2.[73,74] We will discuss the importance of these contralateral insertions of the superficial lamina later.

Deep lamina posterior layer TLF

At the lower lumbar and sacral level fiber direction in the deep lamina is from craniomedial to caudolateral (Figure 7). In the lower thoracic region the fibers of the deep lamina fuse with the fascia of the serratus posterior inferior muscle. In the upper lumbar region the lamina is freely mobile over the lumbar back muscles. Fibers in the lumbar region attach to the interspinous ligaments and insert into the lateral raphe and the iliac crest. In the pelvic region the fibers attach to the PSIS, iliac crest, long posterior SI ligament and the erector spinae fascia in the depression between PSIS and PIIS and median sacral crest. Fibers of the deep lamina are continuous with the sacrotuberous ligament. As described earlier strong connections exist between both laminae of the posterior layer of the TLF at lower lumbar and sacral levels. Between L5 and S1 in some specimens the fibers of the deep lamina crossed the midline.[73,74]

Abdominal muscles

Transverse abdominis muscle

The transverse abdominis muscle originates from the lateral one-third of the inguinal ligament, the anterior two-thirds of the inner lip of the iliac crest, the lateral raphe of the TLF, and the internal aspects of the lower 6 costal cartilages where it interdigitates with the diaphragm. Running transversely around the trunk its upper and middle fibers blend with the fascial envelope of the rectus abdominis muscle finally attaching into the midline linea alba. The lower fibers blend with the insertion of the internal oblique muscle on the pubic crest.[68] The attachment to the entire lateral raphe allows the transversus abdominis to exert tension on both the middle and posterior layers of the TLF in the middle and lower regions of the lumbar spine.[72]

Internal oblique muscle

The internal oblique muscle forms the middle layer of the lateral abdominal wall lying between transversus abdominis and the external oblique muscles.[68,72] It inserts to the lateral two-thirds of the inguinal ligament, the anterior two-thirds of the iliac crest, and the lateral raphe of the TLF. This attachment to the lateral raphe is 2 to 3 cm wide; the internal oblique connects to fibers of the deep lamina arising from the L3 to L5 spinous processes. In some people this attachment to the TLF is absent; this affects the potential role of this muscle in lumbar stabilization.[72] Its posterior iliac fibers run superiorly to insert to the inferior border of the lowest 3 to 4 ribs. Fibers from the internal oblique muscle arising from the inguinal ligament pass inferomedially to the pubic crest inserting in a joint tendon with the transversus abdominis as described above. The intermediate fibers end in a bilaminar aponeurosis. The upper fibers attach to the outer surface of the seventh to ninth costal cartilages. The lower fibers of this aponeurosis pass horizontally, parallel to the

fibers of the transversus abdominis.[72] The relative position of the internal oblique to external oblique and rectus abdominis muscles is described in detail in other texts.[9,10,72]

Quadratus lumborum muscle

The anterior and middle layers of the TLF enclose the quadratus lumborum muscle. Its lateral portion does not attach to the lumbar vertebrae; it connects the lateral ilium to the twelfth rib.[72] The medial portion of this muscle inserts into the apices of the L1 through L4 transverse processes[9] giving this portion a possible role in lumbar stability.[72]

Pyramidalis muscle

The pyramidalis muscle has its origin in the linea alba midway between the umbilicus and the os pubis and inserts to the the pubic bone and symphysis.[68] Lee[68] suggested this muscle may cause superoinferior asymmetry of the symphysis. Its innervation by the ventral ramus T12 establishes an anatomical connection between thoracolumbar and lumbopelvic dysfunction. Lee[68] suggested that treatment of the T12-L1 spinal segment may aid in restoring normal symphysial function.

Hip region muscles

Piriformis muscle

The piriformis muscle originates from the ventral surface of the sacrum between the first to fourth anterior sacral foramen, the ventral surface of the sacrotuberous ligament and the margin of the greater sciatic foramen. The muscle and tendon travel through the greater sciatic foramen to insert into the greater trochanter. As one of the few muscles to cross the SIJ joint line it is likely have a role in SIJ stabilization.[68] Lee[68] hypothesized that increased tension may restrict movement and/or produce pain. Alderink[46] mentioned a role for unilateral piriformis in causing sacral movements around an oblique axis. Because it is occassionaly pierced by the sciatic nerve[9] a so-called piriformis syndrome has been implicated as a cause for sciatic distribution symptoms.

Gluteus maximus muscle

The gluteus maximus muscle originates on the posterior gluteal line of the ilium, the iliac bone and iliac crest superior and dorsal to this line, the posterior aspect of the sacrum, the side of the coccyx, the ESA, the sacrotuberous ligament, and the gluteal aponeurosis covering the gluteus medius muscle.[9] Its aponeurosis is continuous with the superficial lamina of the posterior layer of the TLF.[73,74] The muscle inserts into the iliotibial band; deeper fibers of the lower portion attach to the gluteal tuberosity.[9] It should be obvious that decreased strength in the gluteus maximus, as can occur in an S1 and S2 nerve root compression[57,75], will decrease SIJ and possibly lumbar stability.

Psoas major muscle

The psoas major takes its origin from the ventromedial three-quarters of the L1 through L5 transverse processes, the anterior aspect of the T12 through L5 vertebral bodies, and the IVDs between these vertebral bodies.[11] Gray's Anatomy[9] noted an attachment to tendinous arches covering the lateral aspect of the vertebral bodies. These arches consist of the medial, deep fascia of the muscle and allow passage to the lumbar arteries and veins.[11] The muscle attaches through a common tendon with the iliacus muscle to the lesser trochanter of the femur.[9] Fiber orientation allows for only very small moments with regards to sagittal plane motion of the lumbar spine; the psoas major muscle is however capable of exerting massive compression loads on the lumbar spine.[11] The potential of this muscle for affecting lumbar biomechanics and nutritional status of lumbar spine tissues should be obvious.

Pelvic floor muscles

The pelvic floor plays an important role in generating and maintaining intra-abdominal pressure (IAP).[72] An SIJ dysfunction may alter tension in the myofascial structures of the pelvic floor.[63] This may affect sacrococcygeal range of motion. The pelvic floor is formed by the levator ani complex.[68] This complex consists of 4 different muscles. The puborectalis and pubococcygeus muscles have their origin anteriorly at the body of the pubic bone and the ventral part of the obturator fascia. We discussed the connection between this fascia and the sacrotuberous ligament. The puborectalis muscle courses in a posterior direction passing lateral to the urethra, (vagina,) and rectum uniting with its contralateral counterpart to form a muscular sling around the anorectal flexure. The muscle has no posterior osseous attachment. The pubococcygeus muscle passes caudal to the puborectalis attaching to a midline raphe posterior to the rectum; this raphe connects it to the last 2 coccygeal segments. The iliococcygeus and ischiococcygeus

(coccygeus) muscles originate from the the medial aspect of the ischial spine and sacrospinous ligament and the dorsal part of the obturator fascia. They attach to the anterior aspect of the apex of the sacrum.[68] The pelvic floor muscles are innervated by the pudendal plexus which contains fibers from S4 and sometimes also S3 and S5.[9] This explains pelvic floor symptoms with compression of the lumbosacral roots in cauda equina syndrome.

Neural anatomy

The vertebral foramina of the successive lumbar vertebrae form the lumbar spinal canal[10]. This canal contains the spinal cord which most commonly tapers to a point called the conus medullaris opposite the level of the L1-L2 IVD.[11,76] Sometimes, however, the cord ends as high as T12-L1 or as low as L2-L3.[11] The lumbar, sacral, and coccygeal roots descend further within the dural sac and form the cauda equina.[11] The dural sac extends caudally to the S1-S2 level.[77]

The spinal cord is covered by the 3 meningeal layers: the pia, arachnoid, and dura mater.[76] The pia and arachnoid are also known as the leptomeninges. Both are made of a lattice of collagen fibers allowing for stretch and compression without kinking. The pia is closest to the spinal cord separating the cord from the subarachnoid space. The arachnoid consists of multiple layers and contains the cerebrospinal fluid (CSF) in the subarachnoid space. This CSF primarily has a nutritional role, but may also have a protective function by acting as a hydraulic cushion surrounding the cord and the nerve roots.[76] The subdural space is a potential space: it contains a small amount of serous fluid probably allowing for sliding of the arachnoid on the dura mater. The dura primarily consists of longitudinally aligned collagen fibers with some elastic fibers. The epidural space also is a potential space separating the dural sac from its osseo-ligamentous boundaries.[11] It contains fat, epidural attachments of the dural sac, blood vessels, and areolar connective tissue.[11,76,77] Wiltse et al[78] described a fibrovascular peridural membrane surrounding the dural sac. The mechanical strength of this periosteum homologue supposedly is one-fourth that of the dura. The peridural membrane spans the width of the vertebral body deep to the PLL and attaches to the ventral surface of the deep layer of this ligament.[11,78] It lines the entire vertebral canal replacing the periosteum. The peridural membrane only crosses the level of the disk laterally at the entrance to the lateral recess; from here it extends a circumneural sheath which surrounds the dural sleeve of the nerve roots and spinal nerve up to 10 cm beyond the IVF.[78] In addition to adding to the mechanical strength of the neural system the peridural membrane may cover and contain hematomas mimicking a sequestrated nuclear fragment. Because treatment for a submembranous hematoma (which will resolve with time) will be quite different from treatment of a disc sequestration, appropriate imaging techniques are indicated when the diagnosis is uncertain.[78]

There are three leptomeningeal attachments that maintain the position of the spinal cord within the dural sac.[77] The *internal filum terminale* is a prolongation of the pia mater that passes intrathecally from the base of the cord to the apex of the dural sac. It protects the neuraxis from longitudinal stress by appropriately elongating and shortening in response to spinal movements.[77] The *denticulate ligaments* run from pia mater to dura and suspend the cord centrally in the dural sac.[76] In the lumbar (and thoracic) region there is a distinct *intermediate leptomeningeal layer* between the pia and arachnoid allowing intermeningeal sliding.[76,77] Dorsally this layer is better developed than ventrally forming a trabeculated septum.

Epidural attachments tether the dural sac to the spinal canal. Cranially the dural sac attaches to the foramen magnum and caudally to the posterior aspect of the coccyx by way of the extrathecal continuation of the internal filum terminale called the *external filum terminale.*[76,77] The filum is more elastic than the cord thus protecting it from being overstretched.[76] *Hofmann's ligaments* are condensations of the epidural fascia that connect the dural sac to the PLL and the proximal nerve root sleeve to this ligament and the periosteum of the inferior pedicle.[11,77] These ligaments are also referred to as the *dural* or *meningovertebral ligaments.*[11] Posteriorly a dorsomedian septum or plica connects the flaval ligament to the posterior dura limiting anterior motion of the cord.[76]

Nerve rootlets extend caudally from the conus medullaris.[76] Two to 12 of these rootlets merge and form one nerve root.[11] Close to the cord glial cells surround the axons of the rootlets, resembling the configuration in the rest of the central nervous system.[79] Several millimeters distal to the cord this central glial segment gives way to the non-glial segment: here the axons are structured more like their counterparts in the peripheral nervous system. Even though there are some islets of glial cels left, the axons are mainly surrounded by Schwann cells.[79] Schwann cells produce the myelin sheaths in myelinated fibers.[76]

Endoneurium surrounds the axons and the Schwann cells in the rootlets and roots. This endoneurium consists of closely packed, mainly longitudinally oriented collagenous tissue containing fibroblasts, capillaries, and mastocytes.[76] The amount of collagen in the root endoneurium is approximately 6 times higher than in the cord, but about 5 times less than in the peripheral nerves.[79] As the rootlets exit the cord, their endoneurium is covered by a thin layer of connective tissue, the root sheath. This root sheath is an analogue of the pia mater.[79-81] A ventral and a dorsal nerve root penetrate the dural sac in an inferolateral direction just above the level of their IVF taking with them an extension of the arachnoid and dura mater called the dural sleeve. Nerve roots located within the common dural sac are referred to as intrathecal nerve roots; after penetrating the dural sac they are called extrathecal.[79] The nerve root sheath and the subarachnoid space also extend along the roots.[11] Nerve root sleeves are located posterior to their respective vertebral bodies. However, successively lower nerve roots originate increasingly higher behind the bodies with finally the L5 nerve root sleeve arising at the level of the L4-L5 IVD[11] making it vulnerable to compression both by a central L4-L5, and a lateral L5-S1 disc herniation. A similar relation exists between the S1 nerve root and the central aspect of the L5-S1 disc. An enlargement on the dorsal root just prior to its joining the ventral root to become the spinal nerve is the dorsal root ganglion (DRG). This DRG is typically located in the IVF and contains the cell bodies of the primary afferent neurons.[11] The exact location of the lateral-most extension of the subarachnoid space is unknown.[81] Bogduk[11] stated it extend as far as the spinal nerve implying it also encloses the DRG. Yoshizawa et al[81] and Olmarker and Rydevik[79] stated that the subarachnoid space likely ends proximal to the DRG. A multi-layered connective tissue sheath similar to the perineurium of peripheral nerves and a loose layer resembling the epineurium enclose the DRG.[79] Rydevik et al[82] described this tight capsule of the DRG as a diffusion barrier. The CSF in the subarachnoid space provides for about half of the nerve roots' metabolic needs[76]; the role it plays in supplying the neurons in the DRG is questionable. Just lateral to the IVF the spinal nerve branches into the ventral and dorsal ramus.[11] The epidural tissues and the dura merge with the epineurium and the external perineurium; the endoneurium is a continuation of the root sheath. The outer layers of the perineurium are continuous with the dura and arachnoid; its inner layers are continuous with the pial layer.[76] The endoneurium of the DRG, spinal nerve and rami likely lacks lymphatic drainage; this makes it vulnerable to alteration in pressure as could occur with edema interfering with nerve conduction and axoplasmic flow.[76] Due to its structure and location the DRG appears extremely vulnerable to this type of injury. We will discuss the role of edema in radiculopathy in the biomechanics section.

The dorsal ramus divides into medial, intermediate, and lateral branches.[11] We discussed the role of these branches in ZJ and deep back muscle innervation. With differing regularity the L1 through L3 lateral branches emerge lateral to the iliocostalis muscle and become cutaneous nerves: the L1 lateral branch becomes cutaneous in 60% of the population, L1 and L2 both become cutaneous in 27%, but only in 13% do all 3 levels furnish cutaneous branches.[11] These (cutaneous) superior clunial nerves[9] establish a connection between pain in the area of the buttocks as far down as the greater trochanter and a dysfunction of the thoracolumbar and upper lumbar spine. The lumbosacral ventral rami form the lumbar and lumbosacral plexus and supply the peripheral nerves to innervate the lumbopelvic region and lower extremities.[11]

Preganglionic sympathetic fibers for the lower limb arise from the lateral horn of the T10 through L2 spinal segments.[76] These efferent fibers branch off from the T10 through L2 ventral rami in the white rami communicantes and join the ganglia of the sympathetic trunk.[11] A continuation of the epineurium of the ventral ramus encapsulates these ganglia. In the lumbopelvic region the ganglia are located anterolateral to the lumbar vertebral bodies, anterior to the sacrum, and they finally join anterior to the coccyx.[76] The sympathetic fibers descend in the sympathetic trunk and join the appropriate ventral ramus by way of the grey rami communicantes.[11] The sympathetic system establishes a connection between the visceral and musculoskeletal structures innervated by the T10 through L2 segments and the lumbopelvic region and lower limbs: nociceptive input from the thoracolumbar visceral and somatic structures by way of viscerosympathetic and somatosympathetic reflexes may increase muscle tone, decrease depolarization threshold of sensory receptors, and impair circulation and local homeostasis in the tissues of the lumbopelvic region and lower limbs.[57] Table 1 summarizes the anatomical connections between the thoracolumbar and lumbopelvic regions.

Distal to the DRG a union of a somatic root from the ventral ramus and an autonomic root from the grey ramus communicans (or a sympathetic ganglion) forms the sinuvertebral or recurrent meningeal nerve.[76] European literature describes this nerve as the recurrent nerve of Von Luschka.[79] The sinuvertebral nerve re-enters the IVF just below the upper pedicle in the form of a single trunk, in the shape of a number of nerve

filaments, or as a trunk accompanied by additional filaments.[11] We discussed earlier the role of this nerve in the innervation of the flaval ligament and the ventral part of the ZJ, as well as its contribution to the posterior plexus innervating the PLL and the outer AF. Bogduk[11] described the role of the recurrent nerve in innervating both the IVD at its level of entry into the vertebral canal by way of a descending branch and the disk of the level above through an ascending branch. The lumbar sinuvertebral nerve also supplies the periosteum and blood vessels in the spinal canal and the dura mater.[11,76] Bogduk[11] descibed descending meningeal branches of the sinuvertebral nerve extending over up to 2 segments and ascending branches going up 1 segment in the dura. Other authors mentioned the sinuvertebral nerve crossing over the midline to also innervate the contralateral side of the dura[76] and the meningeal branches extending caudally and cranially for as many as 4 segments with an axial spread of one sinuvertebral nerve of up to 8 segments.[76,77] This would give the dura the potential for widespread and extrasegmental pain referral in case of nociceptive stimulation as might occur with disk herniation.[77] The ventral dura receives an additional nerve supply from the perivascular nerve plexus of the radicular ramus of a segmental artery.[76] Innervation of the dura is richer in the superficial than in the deeper layers and denser ventrally than dorsally. In fact, towards the midline the posterior dura may be totally insensitive. The innervation of the dural sleeve is similar to that of the dura. The sinuvertebral nerves pass through the dural ligaments and may supply these with innervation as well.[76]

Vascular anatomy

The lumbar spine derives its blood supply from the *lumbar arteries*. The upper 4 arteries branch off from the *aorta*; the fifth arises from the *median sacral artery*. The lumbar arteries run posteriorly in the concave lateral aspect of the vertebral body covered by the fascial arches of the psoas muscle. At the level of the IVF a lumbar artery divides into multiple branches. Lateral branches supply the abdominal wall. Posterior branches accompany the dorsal ramus to supply the ipsilateral paraspinals, ZJ, lamina and spinous process. Three distinct medially directed branches originate opposite the IVF: the *anterior* and *posterior spinal canal* and the *radicular branch*. After entering the IVF the spinal canal branches bifurcate into ascending and descending branches; these anastomose with branches from adjacent segments and form an anterior arterial arcade over the back of the vertebral bodies, and a posterior arcade over the laminae and flaval ligaments. Secondary branches from the posterior arcade supply the epidural tissues and dura, and pierce the bone to supply the interior laminae and spinous processes.[11]

The radicular branch enters the spinal nerve as it exits the IVF and divides into *ventral and dorsal distal radicular arteries* that accompany the ventral and dorsal root, respectively.[11] The dorsal distal radicular artery forms a vascular plexus surrounding and supplying the DRG.[11,79] Yoshizawa et al[81] describe an independent branch of the segmental lumbar artery entering the foramen to supply the DRG. The distal radicular arteries anastomose with their proximal counterparts, the *ventral and dorsal proximal radicular arteries*. These proximal radicular arteries are supplied by longitudinal systems derived from the *anterior spinal artery* and *dorsal spinal arteries*. The proximal arteries are ensheathed in their own pial layer and penetrate the root several millimeters from the conus medullaris. Up until that point ventral and dorsal rootlets are supplied directly by branches from the longitudinal systems. The proximal radicular arteries supply the proximal one-third of the roots; the distal radicular arteries provide for the distal two-thirds. However, the principal source of root nutrition is the surrounding CSF: radicular arteries supply only 35% of the glucose absorbed by the nerve root.[11]

The lumbar artery gives off 10 to 20 ascending and descending branches along the lateral aspect of the vertebral body. These *primary periosteal arteries* supply the periosteum and the peripheral bone of the body. Similar periosteal branches from the anterior arterial arcade in the spinal canal supply the posterior body. At the upper and lower parts of the vertebral bodies the primary periosteal arteries form an anastomotic ring, the *metaphysial anastomosis*. This ring runs around the anterior and lateral aspects of the top and bottom of the body. Penetrating branches from this anastomosis called the *metaphysial arteries* supply the peripheral body. Branches of the *nutrient arteries*, derived from the anterior spinal canal arterial system, and the *equatorial arteries*, derived from the lumbar arteries, supply the central core of the body. We discussed the entry of these vessels into the bodies in the section on osseous anatomy. The rich arterial supply may play a role in explaining the preference of metastases for the vertebral body. We will discuss this in monograph 11.2.4. Branches of the metaphysial and nutrient arteries form dense capillary plexuses deep to and in the base of the endplate cartilage. These endplate plexuses and the small branches of the metaphysial arteries

anastomosing over the outer AF surface are the only entry points for nutrients and oxygen into the interior of the IVD.[11]

Venous return occurs through the *lumbar* and *ascending lumbar veins*. The lumbar veins accompany the lumbar arteries and drain into the *inferior vena cava*. The ascending lumbar vein is a longitudinal vessel ventral to the base of the transverse processes; it communicates with the segmental lumbar veins. The ascending lumbar veins drain in the *common iliac*, *azygos* and *hemiazygos veins*. Three distinct venous plexuses cover the lumbar spine. The *anterior external vertebral venous plexus* covers the anterolateral aspect of the vertebral bodies; it is formed by interconnection of the lumbar veins. The other 2 plexuses are located inside the spinal canal: the *anterior internal vertebral venous plexus* covers the posterior bodies and the *posterior internal vertebral venous plexus* is located on the inner surface of the neural arches. The internal vertebral venous plexuses communicate with the ascending lumbar veins at the level of each IVF.[11] The veins in the internal plexuses are valveless and under little pressure.[76] Increased IAP may prevent drainage from the internal plexuses into the ascending lumbar veins and redirect blood flow within the spinal canal with the internal plexuses draining into the thoracic and sacral vessels.[11] The plexuses take up much of the remaining non-neural spinal canal space.[76] Sudden rushes of blood into the internal plexuses as can occur during coughing, sneezing and straining may increase the diameter of these vessels decreasing the available space for a space-occupying lesion such as a herniated IVD or a stenotic lesion resulting in nociceptive stimulation. The Valsalva maneuver for diagnosing increased "intrathecal" pressure[75] is based on this mechanism. Engorgement of the internal venous plexuses can also occur due to increased cardiopulmonary resistance in the caval venous return system. Patients with progressively decreasing right ventricular compliance in combination with spinal stenosis may eventually have neurogenic pain even in previously painfree static or recumbent positions.[83] Parke[83] mentioned cases in which caval abnormalities with resultant epidural hypertension caused symptoms of cord or radicular compression even despite the absence of stenotic compression. Veins from the posterior bony elements and paraspinals drain into the lumbar or ascending lumbar veins. Drainage from the nerve roots occurs through a proximal radicular system towards the cord and a distal radicular system that drains into the lumbar and ascending lumbar veins.[11]

A *subchondral postcapillary venous network* drains the endplate by way of short vertical veins into the *horizontal subarticular collecting vein system*. Peripheral elements of this collecting vein system drain into the anterior external and anterior internal vertebral venous plexuses. All other regions of the vertebral body drain into horizontal veins running through the middle of the vertebral body. These *basivertebral veins* mainly drain into the anterior internal vertebral venous plexus after exiting the body through the posterior nutrient foramen, and secondarily into the anterior external vertebral venous plexus.[11]

The vascular system also serves a mechanical role. Blood contained within the marrow spaces and intra-osseous veins increases resistance of the vertebral body to compression. Bulging of the endplate with compression extrudes blood from the vertebra. This process requires energy and thus buffers the body from compressive loads.[11]

The SIJ derives its anterior arterial supply from an anastomosis between the *median sacral artery* and the *lateral sacral branches of the internal iliac artery*. These vessels enter the anterior sacral foramina and anastomose with the posterior arterial supply provided by the *gluteal arteries*. Venous drainage occurs by way of tributaries of the *median and lateral sacral veins.*[45]

BIOMECHANICS

We will first discuss the biomechanical behavior of the individual anatomical components of the lumbopelvic region. For this I have chosen to use the term *isolated biomechanics*. In the second part of the section on lumbopelvic biomechanics we combine the biomechanical behavior of these components to describe what I have termed *integrated biomechanics*.

Isolated biomechanics

Intervertebral disk

White and Panjabi[84] defined degrees of freedom as the number of independent coordinates, in a coordinate system, needed to completely specify the position of an object in space. They stated a vertebra has 6 degrees of freedom. This means it is capable of rotations and translations in 3 different, orthogonal planes. The rotations correspond to the clinically defined motions of flexion-extension, sidebending, and

(axial) rotation. The 3 translations occuring between vertebrae are anteroposterior glide, mediolateral glide, and distraction-compression. In vivo the interbody joint or IVD is of course incapable of independent motion: it is restrained by the posterior elements of the vertebral arch and its ligaments, the ZJs, and the paravertebral muscles.[11] Reviewing isolated disk biomechanics may, therefore, seem futile, yet it is a necessary prerequisite for understanding the integrated biomechanics of the intact vertebral motion segment.

The disk has 3 functions: it stabilizes the spine by anchoring the vertebral bodies to each other, it allows movement between vertebrae, and it absorbs and distributes loads applied to the spine.[22] The disk is able to perform these 3 functions as a result of the interaction between PGs, water, and collagen in the NP, AF, and endplates.

The AF is able to withstand compression even without a nucleus present[11]: lamellae are held together by the interactions with PGs and will resist buckling thereby sustaining axially applied weight. Prolonged weightbearing, however, will expell water from the AF and deform the AF by buckling of the collagen lamellae. The NP acts to reinforce the AF during compression. The fluid nature of the NP allows it to deform under pressure, but similar to a fluid the volume of the NP has to remain constant. When the NP is subjected to pressure it will deform transmitting the applied pressure in all directions. Axial compression will force the NP to expand radially or in an outward direction against the lamellae of the AF. The pressure applied by the NP will stretch the collagen-PG complex in the lamellae. Tensile properties of the collagen and the forces involved in the PG-collagen interaction will resist this deformation and will oppose the radial pressure of the NP. An equilibrium will be attained in which radial pressure of the NP equals tensile or “hoop stresses” in the anulus. Axial compression will also cause the NP to apply pressure to both endplates. Endplates and vertebral bodies resist deformation: this direct transmission of forces through the NP from one endplate and vertebral body to the next further unloads the AF. After removal of compression the elastic energy stored in the collagen of the anular lamellae will help with reversing the deformation to the disk.[11]

Tensile properties are not uniform throughout the entire AF. Failure stress (maximum stress tolerated at failure) is significantly higher in the outer as compared to the inner AF; it is about 3 times lower in the posterolateral than in the anterior AF.[27,85] The same applies to strain energy density (energy required per unit of tissue to deform the test specimen).[85] The tensile modulus is a measure of the stiffness of the tissue. Skaggs et al[27] found that this modulus was higher in the outer than in the inner AF, and higher in the anterior than the posterolateral AF. Tensile behavior of the anulus is not linearly related to collagen content; differences in type I and II collagen content or ultrastructural organization may play a role[27,85]. We discussed the increase in crosslink content towards the inner AF; this probably has a role other than contribution to tensile stiffness. It may provide a fine collagen meshwork in the inner AF helping to maintain the higher PG content in the NP.[27] This non-uniform distribution of tensile properties may serve an important function. Higher tensile values in the outer than the inner regions will allow for generating a more uniform hoop stress in the whole AF resulting in diminished injury potential. Table 2 summarizes the reasons why the posterolateral AF is predisposed to failure as often seen clinically.[27]

Excessive axial compression might seem a likely mechanism of injury for producing an anular lesion and subsequent disk herniation. Brinckmann and Horst[86] removed the vertebral arch and subjected human motion segments to gradually increased axial loading. The axial load resulted in a uniform increase of radial bulge around the disk circumference. Axial overload caused fracture of the trabecular structure of the vertebral bodies resulting in an increased concavity of the endplates without macroscopic damage to these structures. The endplate bows inward because the cortical bone of the vertebral body strongly supports the peripheral endplate; weaker trabecular bone supports the central portion.[11] The outer AF remained intact with axial overload.[86] Holmes et al[87] subjected 17 lumbar motion segments with their posterior elements removed to stepwise increased axial overload. Of these segments, 9 showed evidence of endplate failure. Rigidity of non-failing was significantly higher than that of failing segments. Bone mineral content correlated with rigidity.

We might expect AF disruption to predispose the disk to herniation under compression, but even with the dorsolateral AF weakened by a radial incision through the anular fibers leaving only a 1 mm thick external layer, axial loading only caused a 0.5 mm bulge localized close to the incision. This magnitude of bulge occurs frequently in normal disks with physiologic loads. In no case did axial loading cause rupture of the peripheral AF, even under axial overload causing vertebral body fracture.[88] Loss of disk tissue, whether as a result of a discectomy or due to an endplate fracture with central protrusion of nuclear material, has a more

profound effect on mechanical behavior of the disk under compression. Brinckmann and Grootenboer[89] performed a partial diskectomy on 15 cadaveric lumbar disks and found that loss of disk tissue caused loss of disk height, increase in radial bulge, and decrease in intradiskal pressure. Reduced disk height will increase the compressive load on the posterior elements of the spine, especially in lordotic postures.[90] An increased bulge may compress neural tissue. High intradiskal pressure is a prerequisite for the disk to meet its physiologic requirements: high tensile stress in the AF is necessary to contain and control the NP and this tensile stress is provided by the intradiskal pressure.[89]

Distraction is a motion which rarely occurs in everyday functioning, but PTs use it frequently as a therapeutic intervention in LBP. True perpendicular distraction separates the attachments of all collagen fibers over an equal distance and, therefore every fiber is strained and acts to resist the separation of the vertebral bodies. Distraction and compression are translations along the long axis of the spine. During mediolateral and anteroposterior translatioal movements or gliding conditions are different. All points on the inferior aspect of a superior vertebra move an equal distance parallel to the upper surface of the inferior vertebra. A sagittal plane glide separates origin and insertion of half of the collagen fibers in both lateral aspects of the disk: strain on these fibers will limit translation. The other half of the lateral fibers, as a result of the opposite direction of fibers in successive lamellae, will have origin and insertion approximated and will, therefore, not resist the gliding movement. The sagittal plane glide elongates fibers in the anterior and posterior AF, but their resistance to movement will be less as the separation of origin and insertion is not in the principal direction of fiber orientation.[11] The situation in a frontal plane translation will be similar with half of the fibers in the anterior and posterior AF resisting movement and the both parts of the lateral AF contributing to a lesser extent in limiting gliding movement. Shear stiffness of the lumbar disk during anteroposterior or lateral translations is about 260 N/mm.[84] This high value indicates that a large force is required to cause transverse plane displacement in a normal motion segment. Therefore, the AF rarely fails due to pure shear loading.[84]

Bending movements such as frontal plane (sidebending) and sagittal plane rotation (flexion and extension), involve lowering one end of the vertebral body and raising the opposite end. This causes compression of the NP on the side of approximation and a decrease in nuclear pressure on the side of separation.[84] The AF will buckle and bulge on the side of compression and stretch on the side of separation. Resting tension in the ligaments and muscular contraction forces will increase nuclear compression during bending movements. The NP will attempt to escape compression and exert force on the AF at the side of separation. Increased tension in that portion of the AF will resist further movement.[11] McKenzie[91] has based a treatment approach for diskogenic LBP on the assumption that nuclear material moves in response of sagittal, frontal, and even transverse plane sustained or repeated rotation. Clinically, patient history frequently implicates bending movements as the mechanism of injury for disk lesions. However, bending cadaveric motion segments 6 to 8^0 in the frontal, sagittal, or other vertical planes does not result in failure. Failure only occurs after removal of the posterior elements and 15^0 of forward flexion, but not by anular failure, but rather by bony avulsion of the AF insertion off the vertebra.[84]

Transverse plane (axial) rotation will cause all points on the inferior surface of the vertebra to move circumferentially in the direction of rotation. Because of the alternating direction of collagen fibers in succesive lamellae axial rotation only separates origin and insertion of those fibers oriented in the direction of the movement. It approximates origin and insertion of the other fibers. In axial rotation only 50% of the collagen fibers in the AF are in a position to restrict motion.[11] Torsional loading of intact lumbar motion segments around a fixed axis through the posterior disk caused AF failure at an average of 16^0. Degenerated disks may be more prone to failure due to rotation: average rotation at failure was 14.5^0 in degenerated disks and average failure torque was 25% higher in non-degenerated versus degenerated disks.[84] In vivo, the ZJs do not allow for this magnitude of rotation. Axial rotation alone is unlikely to cause disk prolapse.[92]

Physical properties of a structure documenting its time-dependent behavior are known as viscoelastic properties.[84] Axial compression causes an increase in radial anular bulging.[86] Creep as a result of prolonged compressive loading might further increase circumferential bulging and decrease disk height. Increased bulging and decreased disk height may play a role in transient radicular compression as a result of prolonged upright posture. Botsford et al[93] used MRI to compare the effects of 6 hours of supine lying to the effects of 7 hours of sitting and standing in 8 non-symptomatic men between 21 and 27 years old. They found a mean decrease in disk volume of 16.2% and a mean height decrease of 11.1% between L3 and S1 with upright posture. Anteroposterior disk diameter decreased significantly with 6%. They concluded that fluid loss

caused the bulk of the height loss with minimal, if any, radial bulging of the disk. Fluid loss increases electrolyte concentration in the disk; the osmotic pressure gradient thus produced allows reimbibition after load removal.[11] Obviously, in healthy IVDs loss of disk height may, but increase in radial bulging will not, contribute to nerve root compression with prolonged upright posture. This study[93] does not allow conclusions on the mechanical behavior of degenerated disks under similar conditions.

In summary, the disk is well designed to handle mechanical stresses during movement. A healthy NP is paramount to maintaining normal IVD biomechanics. Loss of nuclear volume as a result of therapeutic procedures (diskectomy, chemonucleolysis) or pathological processes (herniation) predisposes the IVD to failure. Tensile properties and structural characteristics seem to predispose the posterolateral AF to failure (Table 2). Flexion and/or rotation can experimentally produce anular failure. However, segmental range of motion (ROM) during these experiments far exceeds in vivo ROM. It appears other processes must occur weakening the IVD prior to in vivo failure.

Zygapophysial joint

ZJ function is to restrict rotation and to prevent excessive flexion by restraining the forward translational component of flexion.[40] This way the joints protect the IVD from shear forces, excessive rotation, and flexion.[38] The ZJs also play a role in transmitting compressive forces over the motion segment in certain positions and may limit extension and sidebending. Bogduk[11] discussed the theoretical role of joint angle and shape during flexion and rotation. With rotation the inferior facet of the contralateral ZJ impacts its superior counterpart thus limiting rotation. A planar joint with its facets oriented parallel to the midsagittal plane will offer considerable resistance against rotation. However, it offers no resistance against the forward translational component of flexion: the inferior facets will simply slide past the superior ones. Joints oriented in the frontal plane optimally resist forward translation, but less so rotation: the facets might contact at an angle causing the inferior facets to simply glance off the superior ones. Obliquely oriented joints combine a frontal and sagittal plane orientation for resistance to both flexion and rotation. In curved joints the different portions of the joint may have different mechanical roles. The backwards facing anteromedial portions resist forward displacement; the posterior sagittally oriented two-thirds of the articular facets restrict rotation. Differences in age-related changes discussed in monograph 11.2.4. appear to support these different roles.

Excessive axial rotation can cause circumferential anular tears in vitro.[92] IVD torsion is resisted primarily by compression of the contralateral ZJ.[92] The ipsilateral joint remains unloaded with rotation.[94] Tension in the ipsilateral joint capsule may also play a role in limiting rotation; cutting this structure results in increased rotation.[95] The ZJ is the first structure to fail in forced segmental torsion at an angle of 1 to 2^0.[92] Using a mathematical model of the lumbar intervertebral joint in torsion Ueno and Liu[92] established that the ZJs transmitted 10 to 40% of the applied rotational torque. Medial facetectomy removes the anteromedial part of the joint. Surgeons use medial facetectomy to decompress the nerve root by “unroofing” the lateral recess.[96] Unilateral and bilateral medial facetectomies do not increase ROM in rotation. This underlines the different roles of the anteromedial and posterior ZJ portions: the anteromedial part which is removed in medial facetectomy does limit rotation. Total facetectomy removes the posterior as well as anteromedial ZJ portion. It is indicated sometimes for excision of an intraforaminal herniated disk or a posterior spinal tumor. Contralateral total facetectomy significantly increases rotation to the opposite side and bilateral total facetectomy significantly increases bilateral rotation.[96] Angular rotational displacement and stress in the posterior anular fibers is increased significantly in facetectomized segments as compared to intact facets.[92] Even a unilateral total facetectomy, will destabilize the segment in rotation[96], possibly predisposing it to torsional anular strain. In vivo axial torsion alone is unlikely to result in IVD injury except in cases of extreme trauma with fracture of the ZJ[97]. Degenerative changes may also compromise the segment: repeated torsional trauma could lead to thinning of the articular cartilage; a loss of 3 mm of articular cartilage permits up to 6^0 of extra rotation.[98]

ZJ asymmetry may predispose the disk to herniation. In theory a more coronally oriented facet is mechanically less suited to resist rotation. The resultant increased segmental rotation subjects the posterolateral IVD at that level to increased strain. Ultimately, the repeated strain may result in anular failure.[42,97] Haegg and Wallner[41] used computed tomography (CT) to determine facet joint angles in 17 patients with an L4-L5 protrusion and 30 patients with an L5-S1 protrusion. They compared joint angles with those in the 47 non-protruded segments. Asymmetry was present in 41 of 94 segments. They found a significant correlation between the presence of asymmetry and protrusion only at the L4-L5 level. Yet, their

conclusion was that no correlation of facet joint asymmetry with the presence of protrusions could be established due to the large error in their method of measurement. Cassidy et al[97] also used CT images to determine joint angles in 136 consecutive patients with L4-L5 or L5-S1 herniations. In 86 lateral herniations they found that a significant difference only at L5-S1: the facet on the side of herniation was on average 3^0 more coronally oriented than the contralateral facet. The authors questioned the clinical relevance of a 3^0 difference and rejected the hypothesis that ZJ orientation plays a role in lumbar disk herniation. Ahmed et al[42] more directly investigated the relation between ZJ angle and segmental rotation in vitro on 35 L2-L3 and 16 L4-L5 segments with non-degenerated disks; they found no correlation between ZJ angle and segmental axial rotation. In summary, articular tropism does not increase axial rotation to induce torsional strain sufficient for anular lesions. Ahmed et al[42] did point out that the role of ZJ asymmetry in producing coupled motions with injury potential to the disk remains undetermined.

In the absence of (iatrogenic, traumatic, or degenerative) ZJ damage, axial rotation must be coupled with other motions to cause disc injury.[42] The shape of the ZJs would suggest that flexion separates the facets to a greater degree thus permitting increased rotation before the facets make contact and rotation stops.[99] The combination of flexion and increased rotation may sufficiently strain the AF to cause trauma. Gunzburg et al[95] examined the effect of flexion on the ROM in rotation both in vitro on cadaveric lumbar spines and in vivo on volunteers who had Steinmann pins inserted into their spinous processes. They did in vitro measurements with intact and cut ZJ capsules; the in vivo measurements were in sitting and standing. Segmental and cumulative vertebral rotation decreased significantly with flexion, both in vitro and in vivo (in standing as well as sitting). Division of the ZJ capsules resulted in a significant overall increase in rotation in flexion when compared to rotation in flexion with intact capsules. Rotation was still less than in the neutral spine position. Hindle and Pearcy[98] and Pearcy[99] studied regional L1 to S1 rotation in vivo on healthy males. Hindle and Pearcy[98] compared standing axial rotation to rotation in normal and long sitting and found a significant increase in both normal and long sitting. To these postures, Pearcy[99] added rotation in 30 and 90^o forward bent standing position. Mean increase in rotation as compared to the upright position was 9^0 (38%) for the normal and 11^0 (46%) for the long sitting position; neither of the forward leaning postures resulted in a statistically different ROM in rotation.[99] We could explain the differences in the findings of Gunzburg et al[95] and the latter 2 studies by an increased tightening of posterior soft tissue structures including the ZJ capsules: beyond a certain optimum point trunk flexion restricts rather than facilitates rotation.[98] Pearcy[99] noted that a strong muscle contraction necessary to maintain the trunk in a standing forward bent position may have caused increased ZJ compression and decreased ability to rotate despite flexion. It appears that submaximal flexion does indeed increase ROM in rotation.[98] The combination of flexion and rotation could cause AF damage, especially in the seated, but possibly also in the standing forward bent position in the absence of a protective muscular contraction, such as clinically occurs with deconditioning, dyscoordination, and/or a sudden externally induced movement.

Rotation causes compressive forces in the contralateral ZJ. Gapping the ipsilateral ZJ during a forced axial rotation may be a necessary part of the mechanism of therapeutic manipulation.[100] McFadden and Taylor[100] rotated 12 L3 to S2 specimens. During manual rotation the L4 to S1 inferior recess fat pads clearly moved out of the joints through the capsular foramen. They observed gapping of the distracted side as a result of the non-thrust forces applied in 5 of 36 mobile segments. The authors concluded that axial rotation physiologically is not associated with ZJ gapping. The joints adapt to rotation by virtue of compliance of their articular cartilage and by movement of the intra- and extra-articular fat. They only observed gapping in segments with degenerative or traumatic instability. Instability may contraindicate manipulation.[100]

Medial facetectomy removes only the anteromedial part of the ZJ. This portion of the joint is important in limiting the forward translation associated with flexion.[11] The intact joint is capable of resisting forward shear forces of about 2 kN.[101] Unilateral medial facetectomy and bilateral total facetectomy significantly increased ROM into flexion underlining the importance of the joint, and especially its anteromedial portion, in limiting flexion.

The ZJ surface orientation is almost perpendicular to the transverse plane. Articular cartilage does not cover the tips of the articular processes. This is a clear indication that these joints have a primary role in transmitting shear forces rather than axial compressive loads. Adams and Hutton[90] subjected lumbar motion segments to 1,000 N compressive force in different degrees of flexion and extension. The IVD bore most of the load in neutral and 2^0 of flexion and extension. Only after prolonged 1,000 N compressive loading with loss of disk height simulating prolonged sitting or standing, did the L1 to L3 ZJs carry an average of 11 % of

the compressive load at 2^0 of extension. The L3 to S1 joints carried an average load of 19% in this position. On average, in 2^0 of extension, which corresponds to the erect standing position, the ZJs carried 16% of the axial compression load of the spine. Haher et al[102] subjected T12 to S2 cadaveric spines to compressive loads of up to 1,000 N simulating axial compression and a combination of extension and rotation (extension quadrant). The ZJs carried 20+/-3.6% of compressive forces with axial loading and 25+/-5.2% with extension-rotation. Adams and Hutton[90] found that in 2 of the segments tested the spinous processes transmitted a portion of the compressive load: these "kissing spines" carried 14 to18% of the load. Peak pressure on the articular facets increases as disk height decreases; this may predispose the ZJ to degenerative changes.[103] In segments with severe IVD degeneration and narrowing the tips of the facets transmit substantial load directly to the lamina below, or the the interarticular pars above: this may cause extra-articular impingement and limitation of trunk extension in patients with disk space narrowing.[103] In 2^0 of flexion, which corresponds to unsupported sitting, the ZJs take no part in resisting compression. The IVD, however, has the added compressive burden of resisting the tension in the posterior ligamentous structures.[90]

The role of the ZJ in resisting extension and sidebending appears minimal. Even total bilateral facetectomy does not significantly increase ROM in sidebending and extension.[96] Adams et al[104] sequentially cut the spinous processes, followed by the ZJ capsule and flaval ligament, and finally the ZJs, to determine the contribution of these structures in limiting segmental extension. The first structures limiting extension were the spinous processes and the ISL compressed between the 2 approximating spinous processes. The capsule and ligamentum flavum play a minor role in resisting extension. Only in people with wide spacing of the spinous processes did the ZJs play a role in limiting extension: load was transmitted then either between the articular facets or from the tips of the facets to the adjacent lamina or pedicle.[104] Haher et al[102] established a role for the anterior AF and ALL. With facet destruction these structures constituted an alternate path of loading during extension[102]. In vivo these ligamentous structures may protect the ZJs from excessive loading and degeneration. This establishes a connection between IVD and joint degeneration: degeneration of the (anterior) disk may cause joint degeneration and ZJ destruction will increase disk loading and accelerate degeneration of the adjacent IVD.[102]

In summary, the main ZJ function is protecting the IVD by limiting ROM in flexion and rotation. Under physiological circumstances, anular strain appears only possible during a combination of flexion and rotation. Joint destruction due to trauma, degeneration, or surgical intervention may further disrupt the protective function of the joint. The ZJ carries part of the compressive axial load through the segment during extension. It becomes more important with prolonged weight bearing and with disk space narrowing, which may lead to degenerative changes and LBP. The ZJ only has a limited role in restricting lumbar extension and sidebending.

Sacroiliac joint

In 1905 Goldthwait and Osgood[105] confirmed the existence of SIJ motion in both sexes: they observed separation of nails driven into the sacrum and ilium of male and female cadaveric pelves during a straight leg raise (SLR). The fact that normal SIJs are capable of movement is no longer disputed. However, opinions on the type of motion possible in the SIJ widely differ. In the section on ligamentous anatomy we defined sacral nutation as the motion in which the sacral promontory moves anteroinferiorly and the coccyx and apex of the sacrum move posterosuperiorly. With nutation the iliac crests approximate and the iliac tuberosities separate due to the multiplanar orientation of the joint. During counternutation the opposite movements occur.[19,46] If we view these motions as motions of the innominates on a fixed sacrum, nutation is the same as posterior and counternutation equals anterior innominate rotation. Pitkin and Pheasant[106] described 2 motions conceivable in the SIJ: sacral (counter)nutation around a transverse axis through the interosseus ligaments and sacral tuberosities and unpaired antagonistic movements of the innominates about a transverse axis passing through the pubic symphysis. They thought sacral rotation and sidebending unlikely due to the shape and interlocking of SIJ surfaces. The osteopathic literature reports a more complex view of SIJ motion. They distinguish between iliosacral and sacroiliac motion, depending on whether forces originate in the lower limb (iliosacral) or the spine (sacroiliac)[46]. Osteopaths describe 3 types of iliosacral motion.[55] The innominate may rotate anteriorly or posteriorly on a fixed sacrum. Inferior and superior innominate translation occurs in about 15% of the population[55]; prerequisites are more planar and parallel oriented joint surfaces. A reversal of the convex-concave relationship may allow the innominates to rotate about a vertical axis, inflare and

outflare. Nutation and counternutation of the sacrum occur around a transverse axis; individual anatomic differences result in different locations for this axis[55]. A second SI movement accompanies the gait cycle; motion occurs around 1 of 2 oblique axes. The left oblique axis is situated between the left upper pole of the sacrum and the right lower pole of the SIJ, the right oblique axis runs from the right upper to the left lower pole. Unilateral piriformis contraction may play a role in creating these axes[46]. Motion around an oblique axis is defined as rotation of the anterior sacral surface to one side with contralateral sidebending of the superior surface.[55]

Research into SIJ biomechanics has centered around 2 issues: the extent of motion and the axis or axes around which this motion occurs. Table 3 summarizes the ROM studies[15,52,106-114], table 4 reviews the results of research into the location of the axis of motion.[52,54,114,115]

Very few studies have specifically investigated non-sagittal plane sacral motion. Stevens[116] measured sacral axial rotation by comparing the position of both PSIS with that of the dorsal sacral surface during trunk sidebending in sitting (with transverse plane stabilization) and in standing (with and without frontal and sagittal plane stabilization). He tested 4 non-patient (n=18 to 48) and 1 pre- and post SIJ manual mobilization patient group (n=18). He noted no significant sacral rotation in sitting. Mean combined left and right rotation in the unstabilized standing group was $3.75+/-1.88^0$. The standing group with frontal plane stabilization rotated $5.16+/-2.23^0$, the group with sagittal plane stabilization $5.78+/-1.48^0$. The pre-treatment group rotated $1.1+/-1.52^0$ versus $5+/-1.85^0$ post-treatment. Sacral rotation consistently occured in the opposite direction of trunk sidebending. Stevens[116] hypothesized that sacral rotation only occurs if the ipsilateral ilium is fixed and the sacrum is forced to move following spine motion and coupling against this fixed ilium. There are a number of concerns with this study. Sacral axial rotation cannot occur without SIJ translation. Translation occurs only with joint surface distraction.[115] This seems contrary to the need for ilial stabilization by compression. The author also did not control for innominate anterior and posterior rotation possibly occuring with trunk sidebending[117]; subsequent anteroposterior displacement of the PSIS would affect his findings. In sitting compression through the ischial tuberosities should stabilize the innominates. Might this be why no the author found no sacral rotation? Perhaps the study did not measure sacral motion between the innominates, but rather interinnominate ROM?

Research has produced findings that may be clinically applicable. Male SIJs seem less mobile: ROM was 40% less in male versus female patients with transfers from supine to standing and 30% less with transfers from standing to prone with hip extension.[109] Patient age need not decrease mobility: ROM increased significantly with age for transfers from supine to sitting and standing to prone with hip extension.[109] Research may help interpret SIJ motion palpation findings. Transfers from supine to standing or long sitting, and standing trunk flexion cause sacral nutation[52,109,111,114], counternutation may occur with prone trunk extension.[109,111] Without providing research evidence, Vleeming et al[73] state that flattening the spine in standing also results in counternutation. Hypermobility may play a major role in SIJ dysfunction. Wilder et al[115] showed that in a normal SIJ rotation is always combined with translation. Pure rotation only occured when sufficient ligamentous laxity allowed for unlocking of joint surfaces; pure translation required significant distraction of joint surfaces (7.25 mm).[115] Jacob and Kissling[112] found sagittal plane innominate rotational hypermobility in a patient with unilateral SIJ dysfunction. Pathophysiology in unilateral and bilateral SIJ dysfunction may be different. Patients with bilateral symptoms had significantly greater ROM going from supine to standing than those with unilateral symptoms.[109] ROM was similar in symptomatic and asymptomatic joints of unilateral dysfunction patients when transfering from supine to standing or long sitting[111]; only ROM of the innominate about a longitudinal axis was significantly greater in the symptomatic SIJ when moving from standing to prone with hip extension.[109]

In summary, the existence of SIJ motion in the absence of severe pathology is undisputed. However, ROM reported greatly varies, possibly due to flawed research design. Only 1 seemingly methodologically flawed study[116] supports osteopathic views of multi-axial motion in normal subjects. Sagittal plane sacral rotation in combination with some translation appears to be the main motion allowed in a normal SIJ. The 3-dimensional orientation of joint surfaces may explain rotation and translation observed along the other 2 orthogonal axes and in the other orthogonal planes. Joint surface orientation, joint surface irregularities, and ligamentous laxity determine the type of motion allowed: pure translation or rotation are only possible when these factors allow for sufficient joint surface separation.[50,115] These factors, possibly combined with prolonged force application, may lead to disassociation of complementary ridges and depressions[113], producing the clinical dysfunction of SIJ subluxation. Osteopathic concepts may have a role in explaining

mechanics in SIJs with excessive mobility. As stated by Alderink[46] in 1991 and still holding true today: "...a unifying model of sacroiliac function has not been presented, supported, or verified...".

Sacroccocygeal joint

Sacroccocygeal movements are limited to flexion and extension.[18,19] Flexion or forward movement results from contraction of the levator ani and external anal sphincter muscles. Extension or backward movement is the result of increased IAP during defecation or parturition.[18] Sacrococcygeal extension increases the anteroposterior diameter of the pelvic outlet.[19] Maigne[18] compared sagittal plane radiographs of the coccyx in sitting and standing to determine sacrococcygeal mobility. In 47 painfree subjects the mean mobility (flexion or extension) was 9.3+/-5.7^0 (range 0 to 22^0). Thirteen coccyges had 5 to 15^0 extension, 8 had 5 to 22^0 flexion, 24 had 0 to 5^0 of mobility. Two subjects had an asymptomatic retrolisthesis of 20%. Whether the coccyx flexed or extended seemed determined by its orientation relative to the plane of the seat when sitting down.

Pubic symphysis

Opinions on the importance of the pubic symphysis differ. Kapandji[19] described the pelvis as a closed ring in which ground reaction forces balance gravitational forces. The superior pubic rami transfer part of the ground reaction forces to the pubic symphysis. Kapandji[19] hypothesized that decreased stability of the pubic symphysis may decrease SIJ stability. In contrast, Goldthwait and Osgood[105] describeded the symphysis to be of minor importance in maintaining pelvic stability and mention multiple case studies in which congenital absence of the pubic bones caused no difficulty with ambulation or pregnancy. Snijders et al[17] concurred, supporting this view with the observation that pelvic mechanical function can remain normal even after resection of the pubic bone because of osteosarcoma. They suggested that the symptoms of symphysiolysis may be related mainly to SIJ hypermobility. In their view the importance of the symphysis is limited to allowing sufficient deformation to make SIJ motion possible.[17] Vleeming et al[118] reported that symphysial separation induced in cadaveric specimens did not generate a massive loss of pelvic stability. Vleeming et al[73] discussed the coupling of symphysial to SIJ motion: nutation will cause cranial compression and caudal distraction in the pubic symphysis. They hypothesized that counternutation as a result of flattening of the lumbar lordosis unloads the symphysis supporting this with the clinical observation that this patients with a painful pubic symphysis (e.g. such as after delivery) habitually adopt this posture. Vleeming et al[118] also stated that hypermobility at the symphysis is only possible in case of SIJ hypermobility and/or profound bony deformation, the latter being uncommon. They concluded that hypermobility of the symphysis, therefore, is likely to be accompanied by SIJ hypermobility.[118]

There is also little agreement on the amount of symphyseal mobility. Kapandji[19] stated that the mobility is virtually non-existent. Greenman[55] described 2 different motions at the symphysis. He stated that prolonged one-leg standing result in a frontal plane (inferior-superior) translation. Alternating anterior-posterior rotation may occur with walking.[55] Walheim and Selvik[119] studied symphyseal mobility in a male subject age 27 and a female subject age 24. Separation and anterior translation as a result of several position changes was less than 1 mm. Sagittal plane rotation measured 2^0, transverse plane rotation 3^0. Vleeming et al[73] hypothesized a frontal plane inward rotation associated with nutation of the sacrum during transfer from supine to standing. Interestingly, Walheim and Selvik[119] in 2 of 3 tests found a 0.5^0 outward rotation. The authors determined AORs. The axis for supine SLR was anterior to the axis for standing 90^0 hip and knee flexion in both subjects.

Trauma, as e.g during child birth, has been shown to affect symphysial mobility. Abramson et al[558] demonstrated an increase in width of the symphysis in women mainly in the fifth through seventh month of pregnancy. The average symphysial width was 7.9 mm in primiparae, 7.7 mm in multiparae. Lindsey et al[120] reported on a 4.5 cm wide peripartum symphysial diastasis. They found no gross disruption of the SIJ. The SIJ apparently can remain uninvolved if the separation is 4 cm or less[120]. Hormonal factors during pregnancy, 7 to 10 days prior to menstruation and through the use of oral contraceptives may increase pelvic joint mobility.[14,119]

In summary, research has demonstrated multiplanar and multiaxial symphysial movement. Movements taht induce SIJ motion cause movement at the pubic symphysis as well. The axis for symphysial and therefore likely SIJ motion is force-dependent; this is evidenced by the change in position of the axis during similar weightbearing and non-weightbearing movements.[119] Hormonal and traumatic factors will influence

movement. The role of the symphysis in pelvic stability is likely minimal. Painful conditions of the joint may cause an antalgic posture and thereby indirectly affect SIJ and lumbar mechanics. Research[119] contradicts a clinical model of SIJ and symphysial motion coupling, but the number of subjects in the study is too small for strong conclusions.

Ligamentous structures

The ALL is under tension in a neutral position of the spine thus contributing to IVD pressure in neutral.[60] It resists extension and stabilizes the lumbar spine in extension positions.[59,60] The PLL also has tension in neutral: the amount of prestrain is approximately 3 times that of the ALL.[60] The PLL is not as wide, nor as thick as the ALL. It resists flexion, but due to its proximity to the center of rotation, it is less of a restraint than the flaval ligament.[59]

Like the longitudinal ligaments the flaval ligament is under tension in neutral; this pretension may prevent nipping of the anterior ZJ capsule and inward buckling of the flaval ligament during extension which would encroach upon the adjacent neural structures.[11,59]

A role in resisting flexion is very unlikely for the ISL: its fan-like oriented fibers will simply fan out more when the spine is flexed.[60] Its mainly anteroposterior orientation places it in a position to transmit tension of the TLF to the flaval ligaments preventing inward buckling of these ligaments and assisting in maintaining vertebral alignment.[59,60] The SSL also offers little resistance to flexion.[11,60] Abumi et al[96] found that division of both SSL and ISL did not affect ROM of any of the lumbar motions. Hukins et al[60] suggested its function is rather to protect the spinous processes and muscle attachments by deformation of the loose fatty tissue contained in the ligament.

Function of the iliolumbar ligaments is to restrict lumbosacral motion.[59] Willard[59] reported that section of the iliolumbar ligament increased L5-S1 rotation by 18%, extension by 20%, flexion by 23%, and sidebending by 29%. Bogduk[11] reported that the posterior band mainly restricts flexion, the anterior band sidebending, and that as a whole the ligament prevents forward sliding of L5 on S1. Leong et al[121] studied the effect of a 6 Nm load on L5 into flexion, extension, and sidebending with the iliolumbar ligament intact and after first cutting the posterior and then the anterior band of the ligament. Cutting the posterior band increased flexion significantly (61.2%), but did not affect lateral bending and extension. Cutting the anterior band also significantly increased flexion with16.3%, extension with 21.4%, and lateral bending with 105.3%. Chow et al[122] studied the role of the iliolumbar ligament in restraining rotation and found a 14.6% contribution of the iliolumbar ligament to failure torque and an 18.1% contribution to failure strain energy of L5-S1 in rotation.

The interosseus SI ligaments are the primary restraints against excessive SIJ movement.[46] They limit nutation of the sacrum.[69] Alderink[46] described the function of the posterior SI ligaments as resisting downward slipping of the sacrum between the ilia. The short posterior SI ligament also functions to limit sacral nutation. The long dorsal SI ligament, easily palpable just caudal to the PSIS, resists counternutation.[69] It is frequently painful on palpation in patients who assume an antalgic flat spine posture with resultant sacral counternutation.[73] The anatomical connection between the long posterior sacroiliac ligament and the sacrotuberous ligaments described earlier may prevent excessive slack in these 2 ligaments with an opposite function.[69] Contraction of the ipsilateral erector spinae increases tension in the ligament possibly to prevent it from becoming slack.[73]

The sacrotuberous and sacrospinous ligaments resist sacral nutation.[46,69] Vleeming et al[70] found that distally directed tension to the gluteus maximus muscle and to the long head of the biceps femoris muscle resulted in increased tension in the sacrotuberous ligament in all preparations. This occured even in preparations where the biceps femoris was fixed firmly to the ischial tuberosity due to superficial fibers passing between the ligament and the tendon in these cases.[71] Vleeming et al[123] investigated the effect of loading of the sacrotuberous ligament on the amount of nutation and counternutation of the sacrum. Minimal loads of 50 N bilateral caudolateral tension to the sacrotuberous ligament resulted in a significant decrease of nutation in 5 of 6 pelves and a significant increase in counternutation in 3 of 6 pelves. Averaged for all pelves the effect on nutation was significant; the effect on counternutation was not significant. In quadrupeds the sacrospinous ligament plays a role in movements of the tail; in humans the ligament has evolved as a support mechanism for the pelvic floor; surgeons it use as an anchor site in gynaecological surgery.[59]

Muscular structures

Placed in an upright neutral position with the sacrum fixed, a fresh cadaveric spine from sacrum to T1 that is devoid of musculature can carry a load of no more than 20 N before it buckles. Muscles are obviously necessary to stabilize and move the spine.[124] Richardson et al[72] divide the lumbar and abdominal muscles into a *global* and a *local stabilizing system* based on their main mechanical role in stabilization. Muscles of the global stabilizing system control spinal orientation and are mainly responsible for gross movements of the spine. Richardson et al[72] include in this group the thoracic parts of the longissimus thoracis and iliocostalis lumborum, the lateral fibers of the quadratus lumborum, the rectus abdominis, and both oblique abdominal muscles. Due to their origin and insertion on the lumbar vertebrae, muscles of the local stabilizing system are capable of controlling the stiffness and intersegmental relationship of the lumbar segments. The local system consists of the intertransversarii and interspinales, the multifidus, the lumbar portions of the iliocostalis and longissimus thoracis, the medial fibers of the quadratus lumborum, the transversus abdominis, and the (sometimes absent) fibers of the internal oblique muscle that attach to the TLF.[72] Many extremity muscles also play a role in movement and stability of the lumbopelvic region.

Multifidus and erector spinae muscles

There are a number of anatomical and biomechanical reasons why the deep lumbar muscles are ideally suited to a stabilizing function. Segmental innervation of the intertransversarii, interspinales, multifidi, and the lumbar portion of the iliocostalis lumborum muscles allows them to respond specifically to segmental perturbations. As noted earlier the intertransversarii and interspinales muscles are likely to have a proprioceptive rather than mechanical role. Deep segmental muscles are closer to the AOR. Segmental movement will therefore result in only small changes in muscle length allowing for shorter reaction times.[124] All bone, including that of the neural arch can be elastically deformed. Having (some) muscles closer to the AOR rather than distal on the neural arch structures eliminates the repetitive deformation of the neural arch resulting in a more energy-efficient response to perturbation. Elasticity of the neural arch will also increase the response time of the neuromuscular control system as some correction of th perturbation will be lost due to elastic deformation of the arch.[124]

Panjabi et al[124] sequentially injured a lumbar motion segment by division of SSL and ISL, and unilateral and bilateral medial facetectomy. They studied the stabilizing effect of simulated contraction of multifidi, rotatores, and interspinales muscles on the *neutral zone* and ROM in all 3 cardinal planes. Bogduk[11] defined the neutral zone as “that part of physiological intervertebral motion, measured from the neutral position, within which spinal motion is produced with minimal internal resistance”. Muscle contraction significantly increased ROM in flexion, significantly decreased ROM in extension and bilateral rotation, and non-significantly decreased ROM in sidebending. The neutral zone decreased with increasing muscle force for all injuries. In fact, 60 N of muscle force restored the neutral zone, which the most severe injury had increased by 50% in flexion, to a level less than that in the intact motion segment. Panjabi et al[124] suggested that the frequently clinically observed increased tone in the lumbar back muscles in LBP patients may represent an attempt to stabilize an unstable spine. Wilke et al[125] used L2 to S1 cadaveric spines and studied the effect of simulated multifidus, rotatores, erector spinae, and psoas major muscle forces on cardinal plane L4-L5 ROM and neutral zone. A force of 80 N on each bilateral muscle pair decreased ROM by 93% in flexion, 85% in extension, 55% in total sidebending, and 34% in total rotation. The neutral zone for flexion and extension combined decreased 83%. For right and left sidebending combined the neutral zone decreased 76%. Neutral zone effects in rotation were non-significant. In contrast to the findings by Panjabi et al[124] the authors found a 65% decrease in flexion angle using the same parameters. Intact polysegmental ligamentous structures in the multiple vertebra specimens that are absent in the isolated motion segment may play a role. The multifidus muscle was responsible for two-thirds of the increase in segmental stiffness.[125] Panjabi et al[124] suggested using the neutral zone as an indicator of segmental instability, Wilke et al[125] suggested also using ROM, especially in flexion and extension and rotation.

The fascicles of the multifidus muscle run from craniomedial to caudolateral in the frontal plane. Their line of action consists of a small horizontal and a larger vertical vector. The vertical vector acts virtually at a right angle to each vertebra; its position is ideal for sagittal plane posterior rotation, but not sagittal plane posterior translation. The horizontal vector can theoretically produce transverse plane rotation. However, the multifidus muscle is inconsistently active during rotation back to neutral; it is also active in both ipsilateral, and contralateral rotation. Its main role during rotation may be to counteract the flexion component of the main trunk rotators, the oblique abdominals.[11]

Fascicles of both lumbar erector spinae muscles are oriented mainly in the sagittal plane coursing from ventrocranial to dorsocaudal. The large vertical (sagittal plane) vector of the longissimus thoracis pars lumborum runs lateral to the axis for sidebending and posterior to the axis for posterior rotation. Due to their insertion to the transverse and accessory processes close to the axis for posterior sagittal rotation, the fascicles of the longissimus muscle are less efficient at producing posterior rotation than the multifidus fascicles levering through the spinous processes. Their horizontal vector points backwards, increasingly so at the lower lumbar levels, making the longissimus muscle effective in producing posterior translation. The muscle is at a mechanical disadvantage to produce (transverse plane) axial rotation.[11] Due to its more lateral insertion to the tip of the lumbar transverse processes the lumbar iliocostalis lumborum muscle has a strong sidebending action. It is also the only intrinsic low back muscle positioned to produce rotation: it cooperates with the multifidus muscle in counteracting the flexion component of the oblique abdominal muscles.[11] The thoracic components of the longissimus and iliocostalis lumborum muscles act indirectly on the lumbar spine increasing lumbar lordosis and assisting in sidebending. The iliocostalis lumborum pars thoracis muscle can rotate the lumbar spine back to neutral from a position of contralateral sidending.[11] With trunk extension the multifidus muscle induces posterior sagittal plane rotation assisted by the lumbar erector spinae group which also produces posterior sagittal plane translation. The multifidus muscle contributes 20% to the total extensor moment at the L4 and L5 vertebral levels, the lumbar erector spinae group contributes 30%. The thoracic erector group, positioned to produce extension of the thoracic cage on the pelvis, is responsible for 50% of the total extensor moment.[72] The lumbar muscles may also contribute indirectly to the extensor moment by way of the *hydraulic amplifier mechanism*[72,74]: contraction of the erector spinae and multifidus muscles may dilate the posterior layer of the TLF and thus contribute to spinal stiffness.

Extensor moments of the lumbar multifidus and erector spinae muscles calculated based on active tension generating capacity of these muscles are insufficient to balance the loads sustained by the flexed lumbar spine. Additional mechanisms for generating the “missing” force might be intra-abdominal pressure (IAP) and tension in the TLF or posterior ligamentous sytem[1269]. Macintosh et al[126] studied the effect of flexion on the lumbar multifidus and erector spinae muscles. Central to their calculations was the premise that the total tension of a stretched muscle is equal to or greater than the maximal force exerted by this muscle at resting length: what the fascicle loses in force generating capacity with elongation, it gains by an increase in passive tension as a result of stretch of its passive elastic elements. They found relative increases in length of the muscle bellies of 15 to 59% with flexion. Moment arms decreased up to 39% with flexion, although especially among the fascicles of multifidus and iliocostalis muscles acting upon L4-L5 and L5-S1 lever arms increased by up to 30%. When compared to the upright position the sum of moments decreased by between 1 and 18%. Maximum segmental compression loads were only 2 to 5% higher with maximal contraction in flexed versus neutral position. Muscular shear forces reversed at L1 and L2 from posterior to anterior. At L3 and L4 flexion attenuated posterior shear forces. At L5 flexion reversed the forward shear forces in the upright position to substantial posteriorly directed shear forces. The anterior sagittal plane rotation of lumbar vertebrae during flexion resulted in an increased caudoventral orientation for all multifidus fascicles and a less caudodorsal orientation for all lumbar erector spinae fibers. Macintosh et al[126] stated that because of the criss-cross fascicular arrangement, changes in mechanical advantage tended to be balanced by reciprocal changes in other fibers with a result of little change in total moments and compression loads from the neutral to the fully flexed position. They hypothesized that we need not attribute the “missing” force mentioned earlier to IAP or tension in TLF or posterior ligamentous system, but to the passive elastic elements of the lumbar back muscles at full stretch.

During flexion the lumbar muscles are eccentrically active: the lumbar erector spinae muscles control anterior sagittal plane translation; multifidus and erector spinae muscles control anterior rotation.[72] During erect standing the lumbar muscles are barely electrically active. With flexion these muscles become maximally active[126], but electromyographic activity (EMG) of the lumbar erector spinae ceases at about 90% of maximal lumbar spine flexion.[72] EMG activity of the multifidus decreases muscle , but only infrequently ceases. The multifidus muscle shows continuous, tonic activity during all anti-gravity activity underlining its importance in segmental lumbar stabilization.[72] The reversal of muscular shear forces in full flexion and the increased cross-sectional area of the multifidus at L4 through S1 further support its role in especially low lumbar segmental stabilization.[72]

Transverse abdominis, internal oblique, diaphraghm, and pelvic floor muscles

By their insertion into the TLF the transversus abdominis and internal oblique muscles indirectly attach to the lumbar vertebrae making them part of the local stabilizing system.[72] The transversus abdominis is active prior to the prime movers during upper and lower limb movements when trunk stability is needed. The transversus abdominis muscle remains electrically silent when trunk stability is not at risk as with slow speed shoulder flexion or in small lever arm movements, as when only moving a wrist or thumb.[72] The transversus abdominis muscle (and multifidus muscle) contracts with a large initial phasic burst of electrical activity preceding activity of the prime mover followed by a long duration, low-level, continuous, tonic contraction. The muscle is active earlier when moving a greater mass, such as a leg rather than an arm. Transversus abdominis muscle activity is the same irrespective of the direction of movement. It contracts even when the subject does not know the direction of movement until just prior to the actual movement. It appears to contribute to an increase in non-direction-specific stiffness of the spine.[72] The diaphragm and pubococcygeus muscles display activity patterns similar to that of the transversus abdominis muscle. There are a number of possible mechanisms that allow the transversus abdominis (and internal oblique) muscle to contribute to spinal stability.[72] Cocontraction of the diaphragm, pelvic floor, and transversus abdominis muscles convert the abdomen and spine into a rigid cylinder against the relatively incompressible abdominal contents. Cocontraction of the diaphragm and pelvic floor muscles in order to contain the abdominal contents also allows the transversus abdominis muscle to increase tension in the TLF. This tension may restrict intersegmental motion and increase frontal plane spinal stiffness. Fibers in the superficial lamina of the posterior layer of the TLF pass caudomedially. This results in a small upward force vector at the midline attachment of these fibers. Fibers of the deep lamina pass craniomedially from the lateral raphe creating a small downward force vector at their midline attachment. The obliquity of this midline fiber attachment creates a horizontal and vertical component to the forces exerted due to increased tension in the TLF. The horizontal components from both sides of the TLF cancel each other out. The vertical vectors produce opposite movement approximating or at least preventing separation of the spinous processes to which they attach. The potential for lateral tension in the TLF to produce extension is only minimal. Based on a TLF fiber angle of 30^0 from horizontal and a transversus abdominis muscle cross-sectional area of 140 mm^2, lateral tension in the TLF can only contribute 2% to the maximal extensor moment. The resulting intersegmental compression may, however, increase spinal stability. The abdominal cavity may be able to act as a pressurized balloon ventral to the lumbar spine separating the diaphragm and pelvic floor thereby producing an extensor moment. Production of IAP by contraction of the oblique and rectus abdominis muscles causes a flexion moment on the spine. Counteracting this flexion moment necessitates increased activity of the lumbar muscles thus adding to spinal compression. The transversus abdominis muscle is more likely a candidate for production of the IAP: it has no flexion moment. Mathematical models show that contraction of transversely oriented fibers can cause an extensor moment, even at low levels of pressure. IAP increases with both isometric trunk flexion and extension with activity found in the transversus abdominis in both directions. These observations support the role for this muscle rather than any of the other abdominal muscle in production of IAP. Direct pressure of the abdominal contents and IAP by contraction of the transversus abdominis muscle may increase spinal stability. Finally, increased IAP may increase the stiffness of the abdominal contents thus contributing to lumbar stability.[72]

Other muscles attaching to the thoracolumbar fascia

We have described earlier the extensive attachments of muscles and ligaments to the posterior layer of the TLF. This allows a number of muscles typically classified as extremity muscles to become part of the global stabilizing system discussed earlier. As the TLF spans the lumbosacral region, increased tension in the TLF can potentially affect lumbosacral mechanics. Vleeming et al[74] studied the ability of muscles to cause displacement of and, therefore, tension in the fibers of the deep and superficial laminae of the posterior layer of the TLF. They found that traction to the cranial fascia and muscle fibers of the latissimus dorsi muscle caused limited displacement of ipsilateral fibers 2 to 4 cm away from the place where traction was applied. Traction to the caudal part of the latissimus muscle caused displacement up to the midline, 8 to 10 cm removed from the point of force application. Between L4-L5 and S1-S2 displacement even occurred contralaterally. Traction to the gluteus maximus muscle also generated displacement up to the contralateral side of the superficial lamina. The superficial lamina fibers found crossing over to the contralateral side described earlier may explain this production of contralateral movement. Traction to the gluteus medius muscle did not affect the superficial lamina; traction to the external oblique muscle had variable effects and traction to the trapezius muscle only had a small effect (up to 2 cm). Laterally directed traction to the biceps

femoris muscle conducted through the sacrotuberous ligament into the deep lamina caused displacement up to the L5-S1 level ipsilaterally and sometimes contralaterally; medially directed traction resulted in ipsilateral displacement up to the median sacral crest. Traction to the internal oblique muscles caused no displacement. Traction to the serratus posterior inferior muscle caused small displacement or rupture of TLF fibers. These in vitro findings show that contraction of muscles attached to the TLF may play a role in lumbosacral mechanics. Mooney et al[127] supported this with their in vivo study of asymptomatic individuals and patients with SIJ dysfunction confirmed by intra-articular infiltration. Elecetromyographic (EMG) data showed coordinated contraction of gluteus maximus and contralateral latissimus dorsi muscles both during walking and during isometric trunk rotation. Isometric trunk rotation yielded higher EMG activity in the ipsilateral latissimus and contralateral gluteus maximus muscles. Patients had a similar overall pattern, but had increased EMG activity in the gluteus maximus muscle on the symptomatic side. Mooney et al[127] suggested this may represent an attempt of the body to stabilize the SIJ dysfunction

Neural structures

The biomechanics of the lumbopelvic nervous sytem can not be discussed separately from the mechanics of its mechanical interface. The mechanical interface of the neural tissues is the tissue or material adjacent to the nervous system (NS) that can move independently to the NS.[76] In the lumbopelvic region this mechanical interface consists of the vertebral canal and the IVF. Flexion of the trunk elongates the spinal canal up to 97 mm with the largest displacement occurring in the lumbar and cervical levels (28 mm) and the smallest changes in the thoracic region (3mm).[77] Extension from neutral shortens the spinal canal up to 38 mm.[77] Liyang et al[128] measured volume and sagittal plane diameter and length of the dural sac of lumbosacral cadaveric spines when moved from maximal extension to full flexion. Capacity increased 4.85+/-0.75 ml and length increased 19.40+/-6.42 mm. Anteroposterior diameter of the dural sac and spinal canal decreased significantly from flexion to extension. The cross-sectional area (CSA) of the spinal canal increases with flexion and decreases with extension.[76,77] Fritz et al[129] mentioned a reduction of the CSA of the spinal canal of a normal spine by 9% during extension. Because the AOR is further away from the posterior wall than the anterior wall of the spinal canal, during flexion the posterior wall elongates more (23 to30%) than the anterior wall (6.5 to13%).[76] Sidebending from the one endrange to the other causes a 15% unilateral spinal canal length change.The changes with rotation are unknown, but are probably smaller in the less mobile lumbar and thoracic spine than in the more mobile cervical spine. In non-degenerated cadavers flexion increases the CSA of the IVF by 30% and extension from neutral causes a 20% decrease. Ipsilateral sidebending and contralateral rotation decrease the CSA by 2 to 4% and degeneration further adds to this effect.[76]

The NS adapts to movement by gross or intraneural movement or by an increase in tension.[76] Gross movement refers to movement of the NS in relation to its mechanical interface. Intraneural movement is the movement of neural tissue in relation to its connective tissue sheaths. Trunk flexion causes a ventral displacement and lengthening of the spinal cord and cauda equina.[76,77] This elongation is partly possible because the axons are arranged in folds and spirals which straighten as the cord elongates; the posterior columns of the cord are more folded and twisted than the anterior columns as they are further away from the instantaneous axis of sagittal plane rotation than the other columns.[76] After being unfolded the spinal cord actually stretches.[77] The dura protects the cord from overstretching: dural strain with flexion is higher in areas with higher segmental mobility: strain is 15% at L1-L2 and 30% at L5-S1.[77] Extension slackens the dural sac, increases its CSA and anteroposterior diameter, and moves it posteriorly.[76,77] Transverse folds form on the posterior surface of the cord.[77] Sidebending shortens the cord and cauda on the convex side and lengthens it on the concave side.[76] Rotation may decrease cord circumference as a result of increased tension in the denticulate ligaments.[76] Traction elongates the spinal canal and all structures it contains: dural tension may explain the frequent occurrence of headaches after lumbar traction.[76] Gravity displaces the neural structures towards the down side of the spinal canal.[77] This may affect our choice for positioning with therapeutic interventions: one possible reason to choose supine over prone traction for treatment of disk herniation is that it displaces the nociceptively innervated dural sac posteriorly and away from a protrusion, whereas prone traction may cause contact between the anteriorly displaced dural sac and the posteriorly protruding disk.

During flexion the dura slides up to 3 mm caudally at the L4 spinal level. Yet, at the L5 spinal level the dural sac displaces 3 mm cranially with flexion. This seeming paradox illustrates the interaction of the

mechanical interface with the neural tissues. Shacklock et al[77] explained this paradox: high segmental mobility at L4 may allow the mechanical interface to keep up with the displacement and lengthening of the neural tissue resulting in no or low relative displacement; low segmental mobility results in increased relative displacement of the neuraxis.[77] Butler[76] called the L4 vertebral level a "tension point" and mentions a firm attachment of the dura to the PLL at this level as a possible reason for the absence of neuraxis movement at L4. The caudal neuraxis movement above L4 causes the nerve roots above L3 to become more horizontally oriented with flexion; cranial displacement below L4 results in a more vertical orientation of the lower nerve roots.[77] Flexion causes strain in the lower lumbar and sacral roots, which is greatest (16%) in the S1 nerve root.[77] With trunk flexion spinal nerve receives caudal forces due to the distal fixed attachment of the peripheral nerves. The dural sleeve of the spinal nerve gets pulled out of the IVF and acts as a plug preventing the roots from being pulled out of the foramen.[76] Fixation of the lower lumbar and sacral roots both at L4 and the IVF will further increase elongation and tension in these structures during flexion. With extension the lack of tension allows for increased movement of the neuraxis: the nerve roots droop away from the adjacent pedicles.[77] Schnebel et al[130] confirmed this experimentally: they created an experimental L4-L5 herniation compressing the L5 nerve root and found an increase in compressive forces applied to the root with flexion and a decrease with extension. Traction to the root increased the compressive forces applied to the nerve root. Sidebending slackens the spinal nerve and roots on the concave and tightens them on the convex side.[77]

Knowledge of clinical neurobiomechanics allows us to explain clinical findings. Extension decreases the CSA and anteroposterior diameter of the spinal canal, but simultaneously increases these parameters for the neuraxis. Central canal stenosis can cause compression of the spinal cord and cauda equina with extension. In contrast, flexion has an opposite effect on both spinal canal and neuraxis parameters giving it the potential to relieve symptoms in central stenosis. Flexion increases IVF-CSA and can reduce acute compression of a spinal nerve. Extension, ipsilateral sidebending, and ipsilateral rotation (and the extension quadrant as a combination of these movements) decrease IVF-CSA and can cause compression of the spinal nerve in the IVF, especially in degenerated spinal segments. However, in case of a mechanically sensitized spinal nerve, the increased tension in the lower lumbar and sacral roots due to their fixation at L4 and in their respective IVF can cause symptoms, especially if traction is applied to the spinal nerve as occurs in standing trunk flexion, slump, or SLR tests. We will discuss sensitization in the section on vascular biomechanics. Extension produces slack in the spinal nerves as they pass through the IVF. An increase in tension of neural structures results in an elongation of these structures with a simultaneous decrease in CSA. This causes higher intradural or intraneural pressure. Intraneural circulation decreases due to stretching of the extraneural nutrient vessels and closing of small vessels traversing the epineurium. This may affect nerve conduction and axonal transport.[76] If the slackening effect on the spinal nerve and its roots exceeds the effect of the decrease in foraminal CSA, trunk extension may actually improve intraneural circulation and decrease symptoms of foraminal compression. This might explain why sometimes clinically endrange McKenzie extension exercises cause peripheralization, whereas midrange extension leads to centralization of distal symptoms. Sidebending may have a similar effect because it increases contralateral IVF parameters and decreases ipsilateral dimensions. We might explain a contralateral trunk shift with unilateral radiculopathy as an effort to decrease compression in the IVF, whereas an ipsilateral shift might decrease ipsilateral nerve tension. Careful consideration of the biomechanics of neural structures and their mechanical interface can guide our diagnosis and choice of interventions.

Vascular structures

We will discuss the biomechanics of the blood vessels supplying the lumbopelvic neural structures: they may play a role in producing LBP and radiculopathy. These vessels are subject to tensile and compressive stresses. Both the distal and proximal radicular arteries, their anastomosing collateral branches, and the radicular veins are coiled.[11,79] These redundant coils ameliorate the stress resulting from intrafascicular neural movements and allow for continued vascular supply despite neural elongation.[83] Similar adaptations exist in the peripheral nerves to safeguard vascular supply.[76]

Compression of the vessels in the neural structures may occur pathologically in case of central stenosis, ZJ degeneration, and disk herniation. The arteries appear to be able to compensate and maintain function despite even severe chronic compression.[83] Slow compression first compromises the venules, then the capillaries, and finally the arterioles.[131] Radicular veins do not follow the radicular arteries: they are fewer in

number and run a separate and usually deeper course.[83] Less numerous and thin-walled, they are more sensitive to compression than the radicular arteries. Venous obstruction causes venous congestion.[83] The venous distention as a result of this congestion and possibly direct compressive damage increase microvascular permeability resulting in leakage of fluid and macromolecules from the vessels.[80] This may negatively affect nutrition of the nerve tissue by separating the axons and by altering the ionic balance within the endoneurium.[80] Increasing amounts of fluid in the endoneurial space may increase endoneurial fluid pressure interfering with conduction and axoplasmic flow.[80] Due to being enclosed in the rigid root sleeve, the spinal nerve and DRG are more likely to experience increased endoneurial fluid pressure.[80] Absence of CSF, presence of the cell bodies of primary afferent neurons, an abundant blood supply, location in the IVF inside the rigid root sleeve, a tight DRG capsule, and possibly a greater permeability of the endoneurial vessels make especially the DRG very susceptible to compressive disruption.[79-82]

In the absence of good lymphatic drainage the edematous exudate in the endoneurium of the spinal nerve and DRG is cleared very slowly thus prolonging the contact with the tissue and predisposing the nerve to reactive fibrosis.[132] Peri- and intraneural fibrosis decrease neural mobility and predisposes the nerve to further disruption due to tension and compression.[133] A normal DRG is painful with mechanical deformation.[82] Normal nerve roots are not mechanosensitive: fibrotic changes in the nerve root and DRG may explain the sciatic pain caused by pressure or stretch on an irritated nerve root.[133] Fibrotic changes in spinal nerve and DRG may be caused by biochemical irritants contained in extruded disk material. Glycoproteins, immunoglobulins, phosholipase A2, and hydrogen ions may act as irritants[134]. Other authors mention the possibility that disk material not normally exposed to the circulation may cause an autoimmune response leading to fibrosis.[132,134] Cooper et al[135] compared periradicular and epineural tissues of 11 surgically treated patients with disk herniation to those of 6 cadavers without a history of symptomatic LBP. They found a significant increase in fibrous element content and size and number of fibroblasts in the patients as compared to the cadaveric controls, but no evidence of the presence of inflammatory cells. Hoyland et al[132] examined 160 lumbar IVFs in 46 cadavers. They found 51 bulging disks, 42 posterolateral herniations, but only direct root compression in 8 foramina. Most commonly the protruding disk compressed the local venous plexus. Neural fibrosis was present in 73, DRG fibrosis in 71 specimens. The authors found a high correlation for venous dilatation in the intraforaminal plexus and the extent of fibrosis in the nerve root ($r=0.7$) and DRG ($r=0.67$). They found no inflammatory cell infiltrate in any of the fibrotic specimens. These studies lend no support to the biochemical irritant and autoimmune reaction explanation of neural fibrosis, but seem to support the venous congestion hypothesis. Venous congestion appears more important in the production of radicular compression than direct mechanical compression.

The endoneurium lacks lymphatic drainage and is therefore at risk for intraneural edema. Compression affects the endoneurial veins in the intrathecal roots of the cauda equina less than those in the spinal nerve and DRG: not only do the roots float freely in the protective cushion of the CSF, the roots need no lymphatic drainage as they are able to discharge extracellular proteins directly into the CSF.[80,83] The spinal nerve and DRG also lack the thick protective epineurial and perineurial sheath of peripheral nerves.[80] Arteriovenous anastomoses in the cauda equina are numerous and may facilitate retrograde blood flow.[83] Stenotic compression in the vertebral canal generally develops very slowly and may allow sufficient time for neuronal and vascular adaptation and compensation.[83] The acute pressure of a disk herniation may be up to 400 mm Hg[131], greatly exceeding systolic pressure and therefore totally eliminating circulation. A quickly developing herniation will not allow for neurovascular compensation. All these differences may explain why an acute compression of neural structures in the IVF may cause symptoms of nerve compression such as sensory abnormalities, paraesthesiae, motor loss, reflex abnormalities, and in case of neural fibrosis and/or DRG compression, pain.[131] In contrast, central spinal stenosis can be quite advanced, but not produce any problems.

Integrated biomechanics

Integrated cardinal plane motions

Each vertebra has 6 degrees of freedom.[84] This means it is capable of rotation and translation in each of the 3 orthogonal cardinal planes. We defined degrees of freedom as the number of independent coordinates needed to completely specify the position of an object in space.[84] Six degrees of freedom may hold true for an isolated body-disk-body specimen, but the ZJs decrease the degrees of freedom of the motion segment. A number of coordinates is no longer truly independent: in an intact non-degenerated motion segment ZJs

cause a mandatory coupling of rotation and translation. The intact lumbar spine allows for the 3 cardinal plane movements of sagittal plane flexion and extension, frontal plane sidebending, and transverse plane axial rotation.[11] All 3 movements combine rotational and translational motion. The lumbar spine also allows for the independent motions of distraction and compression covered earlier.[11] In our review of SIJ biomechanics we established that sacral nutation (posterior innominate rotation) and counternutation (anterior innominate rotation) are the main movements of a normal SIJ. We discussed hypotheses on coupling of the SIJs and pubic symphysis. We covered the biomechanics of the constituents of the motion segment. In this section we will expand on the biomechanics of the intact motion segments.

During flexion the lower lumbar motion segments rotate forward from a backward tilted to a neutral position.[11] Lumbar flexion is in effect straightening of the lordosis. Bogduk[11] and Pearcy et al[135] report that a true reversal of the lordosis takes place only at the upper lumbar levels, only occasionally including L4-L5. Macintosh et al[126] report that any degree of kyphosis is limited to the upper 2 lumbar vertebrae and then only in some people. With flexion the superior vertaebra rotates anteriorly in the sagittal plane on the inferior vertebra: this raises the inferior articular processes of the superior vertebra upwards and slightly backwards opening a small gap between the inferior and superior articular facets. Anterior sagittal plane translation then occurs as a result of gravity or muscular contraction closing this gap between the facets. Impaction of the inferior articular facets against the anteromedial portion of the superior articular facet restricts anterior sagittal plane translation.[11] Tension in the ZJ capsules limits the anterior rotation during flexion. Ultimate failure stress values for each joint capsule are up to 600 N. The tension induced in the capsules deforms the inferior articular processes bending them downwards and forwards by up to 5^0 during flexion. The resultant bending forces exerted on the interarticular pars may lead to fatigue failure.[11] With regards to the neural structures it is important to remember that flexion increases the CSA of the lumbar spinal canal and the IVF, the latter up to 30%.[76] Strain on the lower lumbar and sacral nerve roots increases with flexion due to their peripheral attachments and their anatomical connections in the spinal canal and IVF.

During flexion the sacrum nutates between the innominates; nutation provides for stability as a result of self-locking. We will discuss this in the section on form and force closure. By way of their ischial attachments the hamstring muscles control the simultaneous anterior rotation of both innominates in the hip joints around an acetabular axis.[68,73] Through its attachments to the sacrotuberous ligament the long head of the biceps femoris muscle increases ligamentous tension and therefore stability of the SIJs through force closure during trunk flexion; it also controls sacral nutation.[73] Trunk flexion increases tension transfer from biceps femoris muscle to the sacrotuberous ligament.[73] Lee[68] reported the clinically observed occurrence of counternutation of the sacrum near the end of trunk flexion. She explained this terminal counternutation by pointing to the increased tension in the biceps femoris muscle, sacrotuberous ligament, and deep lamina of the posterior layer of the TLF: these may limit sacral nutation. A continued anterior rotation of the innominates around the acetabular axis may then result in a relative sacral counternutation; this renders the SIJ vulnerable.[68] Lee[68] reported that tight hamstring muscles may cause this counternutation to occur earlier predisposing the SIJ to dysfunction with trunk bending activities. DonTigny[14] sees this hypothesized excessive anterior rotation of the innominates around an acetabular axis as an important cause for what he calls *anterior dysfunction of the SIJ*; in this dysfunction the innominate bone is locked onto the sacrum in an anteriorly rotated position. However, this clinical hypothesis seems unlikely for a number of reasons. Tightness in the hamstring muscles by way of ligamentous and osseous attachments would seem to limit both hip and SIJ movements equally. Gravity-induced forces continue to move the sacrum into nutation, perhaps even more so in the later ranges of flexion. Increased sacrotuberous tension will increase compressive forces over the SIJ; increased force closure will make any movement (nutation and counternutation alike) unlikely near the end range of trunk flexion. Finally, it appears unlikely that even if there were continued anterior rotation of the innominates around an acetabular axis, that motion in the hip joint would affect motion in the (locked) SIJ: even though the innominates are part of both joints anterior rotation of the innominates in the hip joint does not equal anterior rotation of the innominates in relation to the sacrum. Counternutation at the end of trunk flexion therefore appears unlikely. The anterior dysfunction described by DonTigny[14] would only appear possible in the case of gross failure of the SIJ force closure mechanism. Vleeming et al[73] described sacral counternutation when adopting a flat-back posture.

Extension in the lumbar motion segments consists of a posterior sagittal plane rotation combined with a small posterior sagittal plane translation.[11] We discussed the limited role of the ZJs in limiting extension. The ALL and AF restrict extension. Depending on the spacing of the spinous processes, approximation of the

spinous processes and compression of the ISL, or bony impaction of the tips of the articular processes against the lamina below or the interarticular pars above will limit extension. Extension decreases the CSA of the spinal canal and the IVF, the latter by up to 20%.[76] It decreases the tension on the lower lumbar and sacral nerve roots. During extension in standing the sacrum nutates; we discussed how the sacrum also nutates in flexion. However, passive extension in prone will produce sacral counternutation.

Impaction of the contralateral articular facets and tension in the ipsilateral ZJ capsule limit axial rotation. The joint space is very narrow. Therefore, the ROM permitted prior to impaction is very small. Compression of the articular cartilage accommodates further movement: water is squeezed out of the cartilage and later reabsorbed. Bogduk[11] calculated that every degree of segmental rotation requires 0.5 mm of cartilaginous compression. With cartilage 2 mm thick and consisting of 75% water the cartilage needs to be compressed to 62% of its resting thickness to allow for 3^0 of rotation; this involves losing 50% of its water.[11] We discussed how degenerative loss of cartilage may increase segmental rotation. In a normal non-degenerated motion segment further rotation can only occur if the upper vertebra pivots on the impacted joint. That makes this joint the new center of rotation: the vertebral body swings backwards and laterally, the ipsilateral inferior process moves backwards and medially. The shear forces thus induced cause stress on the ipsilateral ZJ capsule and the IVD.[11] McFadden and Taylor[100] found this ipsilateral ZJ gapping only in segments with traumatic or degenerative instabilty. Rotation decreases the contralateral IVF-CSA of a non-degenerated motion segment by 2 to 4%.

No extensive biomechanical analysis exists for lumbar spine sidebending.[11] Meadows[136] described lumbar segmental sidebending as the ipsilateral superior facet sliding down on the subjacent inferior facet. Simultaneously, the contralateral superior facet slides upwards in relation to the contralateral inferior facet. Meadows[136] likened the ipsilateral ZJ motion to a unilateral extension. The contralateral motion he described as a unilateral flexion. He used these unilateral flexion and extension motions in combination with segmental flexion and extension to more thoroughly evaluate segmental mobility and to deliver more specific mobilizing forces. Similar to rotation, sidebending decreases the IVF diameter by 2 to 4%. Sidebending slackens the ipsilateral and tenses the contralateral neural structures. Describing sidebending as a uniplanar motion is a simplification. However, this simplification allows us to construct a model for evaluation and treatment. In vivo sidebending in the lumbar spine occurs coupled to rotation.

In the section on isolated SIJ biomechanics we found it is unlikely for a normal SIJ to rotate or sidebend. However, lumbar sidebending results in gravity-induced vertical forces on the superior aspect of the sacrum lateral to the mid-sagittal plane. The SIJs need to absorb these forces. The multiplanar SIJ orientation may allow dissipation of these off-center forces by the non-sagittal components of innominate rotation. Meadows[117] notes that lumbar sidebending may result in ipsilateral anterior and contralateral posterior innominate rotation. Due to coupling to sidebending, lumbar rotation may induce similar SIJ motion. The direction of this lumbar coupling should determine the SIJ motion with rotation.

Coupled motions

Fryette's first law[137] states that in lumbar spinal neutral, sidebending induces a contralateral rotation. His second law states that in full flexion or extension, rotation and sidebending occur to the same side. In his laws, Fryette discussed his findings on coupled motions. Coupled motion is the phenomenon of consistent association of one motion (translation or rotation) about one axis with another motion about a second axis; the one motion can not be produced without the other occurring.[138] Coupled motion is the motion that occurs in directions other than the direction of the load applied.[139,140] Coupled motions have implications for treatment, especially manual therapy.

The spinal column generally is symmetrical about the sagittal plane.[84] Therefore, we can expect no or only minimal coupled motion with flexion and extension. Cholewicki et al[139] noted no coupled motion when applying flexion and extension moments to cadaveric lumbosacral spines. Hindle et al[141] found no coupled motion in vivo with flexion and extension in subjects without a history of LBP. Schuit and Rheault[142] reported only very small coupled motions in asymptomatic adults. Oxland et al[140] found coupled motions of axial rotation and sidebending of less than 0.5^0 in both flexion and extension at L5-S1 in cadaveric spines. In contrast, Pearcy et al[143] did find coupled motion in asymptomatic individuals; they suggest that we consider coupled segmental rotation and sidebending greater than 4^0 in flexion and greater than 3^0 in extension abnormal.

Fryette's laws discuss motion coupling during non-sagittal plane movements. Research has studied the coupled motions occuring during axial rotation and sidebending.[139-145] Table 5 reviews the coupled mtions found with axial rotation as the primary motion, table 6 summarizes the coupling found during sidebending.

Coupled motions occur in cadaveric spines devoid of muscles: the role of muscles in producing coupling seems minor[138]. White and Panjabi[84] hypothesized a role for suboptimal muscle control to explain coupled motions during flexion and extension. Pearcy et al[146] mentioned a possible role for uni- or bilateral muscle contraction determining direction and ROM of coupling. Oxland et al[140] distinguish between *postural* and *structural coupling*. Postural coupling refers to the influence of the lumbar lordosis and occurs as a result of differences in vertebral orientation throughout the spine. Structural coupling is the result of the physical characteristics of the joints such as articular tropism[42] and IVD degeneration. Panjabi et al[145] studied the effect of 5 sagittal plane postures on motion coupling in cadaveric lumbosacral spines. The different postures did not affect the direction of coupling between sidebending and rotation. In 4 of 5 postures the associated sagittal plane motion was flexion both during sidebending and rotation. Only in a maximal flexion was extension the coupled motion. Vicenzino and Twomey[138] studied the effects of submaximal flexion and extension combined with left or right sidebending on coupled rotation in 4 cadaveric spines. Sidebending resulted in ipsilateral rotation in all 4 quadrant positions at L1-L2 and L3-L4 except for flexion-left sidebending: here L1-L2 rotated contralaterally. Rotation at L2-L3 and L4-L5 was contralateral. Coupled rotation at L5-S1 was ipsilateral independent of spine position.[138] This affects our inferences from findings on quadrant test positions. Cholewicki et al[139] found that intrinsic mechanical properties of the spine and degree of lordotic posture each determined 50% of the magnitude of coupling in their mathematical model. Oxland et al[140] found that the IVD played a major role in limiting sidebending coupled to L5-S1 axial rotation; the ZJs were mainly responsible for the flexion coupled to axial rotation and the axial rotation coupled to sidebending at L5-S1.[140] Vicenzino and Twomey[138] found no association between facet tropism and coupling despite a mean facet tropism of 10^0 in their specimens. They also reported no influence of ZJ resection or degree of IVD degeneration in their small sample, but they did note that compressive preload might play a role in producing coupled rotation in the presence of facet tropism.

No consensus exists on the direction and ROM of coupled motions associated with all 3 cardinal plane motions in the lumbosacral spine. The effects of physical characteristics of the joints involved are unknown. Regional coupling need not be the same as coupling in the individual segments. Vicenzino and Twomey[138] suggested that, in the absence of a clear consensus, the PT should use clinical assessment findings, especially those of the passive physiological intervertebral motion tests as the basis for treatment selection. Unfortunately, reliability of these tests is poor. We will discuss this in monograph 11.2.4. Blindly following a set of guidelines such as Fryette's laws to determine direction of manual techniques is not a wise clinical decision.

Form and force closure

The joint surfaces of the SIJ are relatively flat. This makes them well suited to transfer great forces, but at the same time makes them vulnerable to transverse forces near the joint.[147] Joint surface orientation of the SIJs is almost parallel to the long axis of the spine. This makes them vulnerable to the shear forces induced by lumbosacral loads. Asymmetric loading as in lifting with trunk rotation increases these SIJ shear forces unilaterally because the resultant lumbosacral force acts lateral to the midsagittal plane.[17] The Musculoskeletal Research Group at the Erasmus University in Rotterdam has developed a biomechanical model attempting to explain SI movement and stability incorporating the complex musculo-fascial-ligamentous connections of the lumbopelvic region described earlier.[17]

SIJ stability is provided by a combination of *form* and *force closure*. Form closure refers to a stable situation with closely fitting joint surfaces; no extra forces are needed to maintain the state of the system given the current load situation.[73] Aspects of form closure in the SIJs are[17,147]:

- The increased friction coefficient as a result of roughening of the articular surfaces.
- The symmetrical osseocartilaginous ridges and grooves on iliac and sacral auricular surfaces.
- The undulated shape of the joint surfaces resembling a propeller.
- The wedge shape of the sacrum between the innominate bones.

However, perfect form closure is not conducive to movement. It would not allow the SIJs to move and thus dissipate the caudally and cranially directed forces converging in this region. The combination of form

and force closure does allow for the necessary movement. Force closure becomes essential when form closure is absent or insufficient.[17] With force closure lateromedially directed, compressive forces are needed to increase intra-articular friction and thus resist the shear forces resulting from vertical lumbosacral loading.[17,73] In the SIJ the ligaments primarily provide these forces. In addition to ligamentous tension, forces in muscles and fascial structures crossing the SIJ surfaces contribute to force closure.[17]

SIJ *self-locking*[73] or *self-bracing*[147] is essential for force closure. Sacral nutation is crucial in this self-locking: it increases tension in the interosseus, short posterior SI, sacrotuberous, and sacrospinous ligaments. These ligaments pull the posterior portions of the innominate bones closer together increasing compressive forces over the SIJ surfaces thereby contributing to force closure.[73] Muscles can contribute in a number of ways to increased SIJ compression. The piriformis, gluteus maximus, iliococcygeus, and ischiococcygeus muscles connect sacrum and innominate bones[10,68]: contraction will directly approximate these bones and thus compress the SIJs. Both the lumbar multifidus and the lumbar erector spinae muscles have iliac attachments.[11] Contraction will pull the innominate bones together posteriorly.[73] Sacral attachments of the lumbar multifidus muscle will contribute to self-locking as a result of their nutation action on the sacrum.[73] The oblique abdominal and the transversus abdominis muscles attach to the anterior aspect of the innominate bones crossing the SIJs anteriorly.[10] Their orientation causes a sizeable frontal plane horizontal component to the forces they produce. Their large mechanical advantage because of their distance to the SIJs and horizontal vector results in a "nutcracker-like" effect on these joints: abdominal muscle contraction tenses the ligaments dorsal to the joint and produces compressive forces.[17] A number of muscles have their origin or insertion on the SI ligaments; contraction will increase tension in these ligaments and thus indirectly increase compressive forces over the SIJ. The gluteus maximus, the long head of the biceps femoris, the deepest laminae of the multifidus, and the piriformis muscle all have a direct anatomical connection with the sacrotuberous ligament.[59,70,71] The entire levator ani complex has an anatomical connection to the sacrotuberous ligament by way of the obturator fascia.[10,68] The iliococcygeus and ischiococcygeus muscles insert into the sacrospinous ligament[68] and therefore also into the sacrotuberous ligament to which it is intimately connected.[9] Ventrally the iliolumbar ligament attaches into the ventral SI ligament.[9] This may allow the quadratus lumborum muscle a role in SIJ as well as lumbosacral mechanics.[68] The L5 longissimus fascicle also inserts into these ventral SI ligaments.[11]

The posterior layer of the TLF plays an important role in force closure. In the lower lumbar and sacral region its superficial lamina has contralateral connections with the sacrum, PSIS, and iliac crest; its deep lamina inserts into the contralateral PSIS, iliac crest, long posterior SI and sacrotuberous ligaments, and the fascia of the lumbar erector spinae group.[74] Both laminae are strongly interconnected at the L4-L5 and sacral levels.[73] The oblique orientation, contralateral insertions, and interconnections with SIJ ligaments allow for an important role for tension in the TLF in force closure. The erector spinae muscles can directly increase tension in the posterior layer of the TLF by way of its insertion into the deep lamina and indirectly through the hydraulic amplifier mechanism discussed earlier.[73] All muscles inserting into the sacrotuberous ligament have the potential to indirectly increase TLF tension as a result of the attachment of the deep lamina to this ligament. This may explain the effect of tension to the long head of the biceps femoris muscle on sacral nutation.[70,123] We discussed the evidence for coupling of the gluteus maximus and the contralateral latissimus dorsi muscles which creates a force perpendicular to the SIJs thus adding to force closure.[73,74,127] The internal oblique and transversus abdominis muscles may have a role in general tension increase in the TLF in addition to their role in the local stabilizing system of the lumbar spine.[72] Anterior to the SIJs the deep and superficial fibers of the anterior abdominal fascia cross the midline and blend with fibers from the opposite side similar to the fibers in the posterior layer of the TLF.[68] The aponeuroses of the transversus abdominis, internal oblique, rectus abdominis, pyramidalis, adductor longus, and gracilis muscles reinforce the pubic symphysis and form connections with their contralateral oblique counterparts.[19,57] Increased tension in the abdominal fascia and prepubic aponeurosis may play a role in force closure.

Vleeming et al[73] named a number of reasons for failed SIJ self-locking. Ligamentous laxity can result from repetitive microtrauma as occurs in high-level athletics and gymnastics or it can be related to the relaxin-induced decrease in mechanical characteristics of ligaments during and after pregnancy[73] and around the menstrual period.[14] Decreased muscle performance can occur in deconditioning or as a result of detraining in high-level athletes with traumatically induced ligamentous laxity.[73] Counternutation disengages the self-locking mechanism of the pelvis. This counternutation may be an antalgic position. Vleeming et al[73] mentioned a painful symphysis postpartum as a possible cause. Flattening the spine counternutates the

sacrum and mechanically unloads the symphysis.[73] A flat-back posture with associated sacral counternutation and unlocking of the pelvis may also be the result of an attempt to unload a painful ZJ, posterior IVD, lumbar nerve root, or pinched ISL. Adaptive shortening of the hamstrings muscles may play a role.

The biomechanical model of form and force closure has a number of diagnostic and therapeutic implications. The body can only compensate for failed self-locking by increased tension in those muscles that are connected to the sacroiliac ligaments and the TLF. Increased piriformis muscle tone may alter sacroiliac biomechanics[46] and cause sciatic distribution pain as a result of sciatic nerve compression. Contraction of the lumbar multifidus and erector spinae muscles alters lumbar, lumbosacral, and SIJ biomechanics and increases compressive forces in the lumbosacral spine. Increased pelvic floor muscle tone may cause seemingly non-musculoskeletal complaints. Greenman[55] names changes in urinary urgency and frequency, dysuria, dyspareunia, and rectal pruritus without clear organic reason as indicators for dysfunction of the pelvic diaphragm. Shortening of the hamstring muscles frequently observed in patients with LBP may be an attempt of the body to increase tension in the sacrotuberous ligament and TLF to increase force closure. Hamstring muscle contraction also posteriorly rotates the innominates with resultant sacral nutation and increased force closure.[73] Indiscriminately including hamstring muscle stretches in the rehabilitation program for all LBP patients, therefore, needs to be questioned. Prolonged increases in muscle tone will affect local muscular homeostasis resulting possibly in nociceptive stimulation originating in these muscles. The increased muscle tone will also affect the homeostasis in the structures it compresses or restricts in motion. The effect on avascular structures such as the joint surfaces of the lumbopelvic joints and the inner lumbar IVDs that depend on motion for adequate nutrition seems obvious. Restoring the self-locking mechanism is of paramount importance. This means that our examination needs to review all possible causes for this possibly antalgic posture, so we can direct our intervention at the cause rather than the symptoms resulting from compensatory muscular overuse. Vleeming et al[118] studied the effect of a pelvic belt applied just above the greater trochanters on total nutation-counternutation of the sacrum in 6 cadavers. They hypothesized that the belt would decrease sacral motion by increasing compressive forces and thus force closure. Tightening the belt with a force of 50 N caused a significant decrease in sacral movement of 18.8%; 100 N decreased motion significantly with 18.5%. The authors suggested applying a pelvic belt with a force of 50 N in patients with pelvic instability. Snijders et al[17] mentioned using tight-fitting elastic or spandex pants if the belt is not tolerated and reported positive effects in patients with peripartum pelvic pain. They also noted that other solutions need to be considered if there is no relief of symptoms within 15 minutes of applying the pelvic belt. Symphysiodesis is a surgical treatment in case of postpartum or posttraumatic symphysiolysis: a bone graft is inserted under strain in the pubic symphysis.[118] Vleeming et al[118] advised against this procedure: it will increase tension in the thin ventral, but slacken the posterior SI ligaments. This surgically induced posterior ligamentous laxity will likely further compromise the pelvic self-locking mechanism.

How can we integrate the model of form and force closure with the model of a local and global stabilizing system as proposed by Richardson et al?[72] Vleeming et al[73] suggest strengthening of the gluteus maximus, latissimus dorsi, and erector spinae muscles to increase force closure by increased tension generation in the posterior layer of the TLF. Mooney et al[127] report good results of dynamic trunk rotation strengthening targeting these muscles in treating patients with SIJ dysfunction. Sufficient strength, endurance, and coordination of these muscles are undoubtedly needed for ADL function. However, concentrating on just these muscles will not restore lumbar segmental stability and independent motion: they would force the spine to function as a rigid structure.[72] We need to address deficits in the muscles of the local stabilizing system. Careful examination to determine the existing deficits and appropriate interventions integrating training of both the global system active in force closure and gross spinal orientation and the local system needed for segmental control appear indicated.

Kinetic chain influences

Structural and functional changes in the lower extremities will alter direction and magnitude of forces directed through the lumbopelvic region. Friberg[148] used a radiographic method to evaluate leg length discrepancy (LLD) in 359 asymptomatic subjects, 653 chronic LBP patients, with and without sciatica, and 254 patients with chronic unilateral hip symptoms, with or without arthrosis.

He found a discrepancy of greater than or equal to 5 mm in 75.4% of patients versus 43.5% of controls. The incidence of a LLD of 15 mm or more in patients was 5.32 times that of the symptom-free control group. Sciatica referred into the longer limb in 179 (78.5%) of 228 patients, hip pain was located on the side of the

longer limb in 226 (88.9%) of 254 patients, and he found degenerative changes in the hip joint were on the long limb side in 24 of 27 patients with unilateral hip arthrosis. All arthrotic changes were located superolaterally. Friberg[148] explained the hip symptoms by the varus position of the long limb hip; this subjects the load-bearing surface of this hip to greater stresses than that of the opposite, short limb side. An LLD causes a functional scoliosis convex to the side of the shorter limb. This scoliosis causes a sidebending of the lumbar motion segment to the long limb side compressing the concave side of the disk and increasing the posterolateral disk bulge towards the spinal nerve roots.[148] Friberg[148] also assumed that a contralateral rotation takes place in the motion segment further adding to the stress on the IVD; he implied that the increased loads on the disk and the increased chance of a compressive nerve root lesion are responsible for the greater incidence of LBP and sciatica found. Friberg[148] reported positive results when using a shoe or heel lift for patients with a functional scoliosis due to LLD.

We can hypothesize a number of additional ways in which LLD affects lumbopelvic function. The hip on the long limb side is in a varus or relatively adducted position. This may cause stretch weakness of the hip abductor muscles resulting in a Trendelenburg or modified Trendelenburg type gait. Either gait type increases lumbar sidebending to the long limb side intermittently reducing the already decreased diameter of the IVF and compressing the IVD. As Friberg[148] noted increased load-bearing stresses predispose the hip of the long limb to degenerative changes. The capsular pattern of the hip is initial restriction of medial rotation, followed by extension, abduction, and finally lateral rotation.[149] From midstance to preswing the stance leg needs to medially rotate and extend. Capsuloligamentous and muscular restrictions to extension and medial rotation will force the lumbar spine to increasingly rotate contralaterally to compensate for hip restrictions and maintain normal stride length. Contralateral lumbar rotation will cause increased compressive forces over the ipsilateral impacted ZJ potentially predisposing this joint to premature degenerative changes. The reactive osteophytosis may further compromise IVF diameter. Joint line orientation of the SIJ on the side of the short limb becomes more horizontal increasing the compressive forces over the joint and thus increasing its stability. The more vertical orientation of the SIJ on the long leg side increases the vertical shear forces over this joint. To increase force closure the muscle tone of the lumbar erector and multifidus, pelvic floor, and hamstring muscles may increase. Increased lumbar muscle tone results in increased compressive stresses in the lumbar spine affecting local homeostasis, possibly contributing short-term to decreased nerve root perfusion and long-term to degenerative changes in disks and ZJs and to nerve root fibrosis. It also further compromises IVF diameter by increased disk bulging. Increased hamstring muscle tone may compromise the sciatic nerve by direct compression or by decreasing neural mobility. Limitation of hip ROM with trunk flexion further increases lumbar spine stresses.

Equalizing leg length by way of an orthotic may be a wise therapeutic choice especially when the LLD has occurred only recently as with e.g. lower extremity fracture. Orthotics seem contraindicated in case of a structural, fixed scoliosis.[148] Sometimes using an orthotic may unload contralateral structures temporarily even if no LLD is present. A functional LLD may result from dysfunctions in the kinetic chain including a posteriorly rotated innominate on the short leg side, an anteriorly rotated innominate on the long leg side, a knee flexion contracture on the short leg side, or a decreased dorsiflexion of the talus on the long leg side.[136] Of course, the dysfunctions of increased muscle tone, decreased muscle strength and length, and joint instability and restriction can occur separate from LLD. Evaluation and treatment needs to include all dysfunctions in the lower limbs that can affect lumbopelvic mechanics. Sometimes treatment of lower limb dysfunctions contributing to the symptoms of a structural problem such as lumbar central stenotic syndrome may be the only therapeutic intervention available to the PT.

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