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LEARNING OBJECTIVES

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

1. The effects of normal aging on joints and ligamentous structures of the lumbopelvic region.
2. The signs and symptoms of diseases affecting the lumbopelvic region.
3. The research base for history and examination items used in orthopaedic differential diagnosis.
4. A number of tests for orthopaedic differential diagnosis of the lumbopelvic region.
5. A biomechanical approach to orthopaedic differential diagnosis of the lumbopelvic region.
6. The research base for use of manual therapy, traction, and exercise in selected dysfunctions.
7. A biomechanical approach to treatment of dysfunctions of the lumbopelvic region.

INTRODUCTION

In this second monograph on the lumbopelvic region we will review changes as a result of normal aging, diseases affecting this region, examination, orthopaedic differential diagnosis, and treatment. Five case scenarios will illustrate the practical application of the information reviewed in both monographs.

NORMAL AGING

To distinguish the effects of aging from those resulting from pathological processes, we will review age-related changes in the joints and ligamentous structures in the lumbopelvic region.

Intervertebral disk

Aging affects all 3 components of the intervertebral disk (IVD). In the nucleus pulposus (NP), the proteoglycan (PG) synthesis rate decreases. This results in lowered PG concentrations; by age 60, PGs make up only 30% of the NP dry weight as compared to 65% in young adults.[1] The remaining PGs are also smaller. Proteoglycans contain both chondroitin- and keratan-sulphate. Chondroitin-sulphate has carboxyl- and sulphate-groups; both groups can bind water. Keratan-sulphate only has the sulphate groups. As a result, PG water binding capacity depends largely on their chondroitin-sulphate content. With aging, the keratan-sulphate concentration remains constant, but chondroitin-sulphate concentration and thus water binding capacity decreases.[1] The PGs also have a decreased tendency to bind to their core protein, hyaluronic acid, resulting in a decrease in aggregated PGs.[1,2] Cleavage of PGs with a long half-life may produce molecules without the ability to aggregate with the core protein. Older disks may change their biosynthetic activity: decreased quantity of link protein needed to connect to the core protein chain may play a role in decreased aggregation.[2] The collagen concentration in the NP increases, as does the number of bonds between the collagen and the PGs. Disk water content decreases from approximately 88% at birth to 65 to 72% at age 75. Kraemer et al[3] mentioned a decrease from 90% in the first year of life to 74% in the eighth decade. This decreased water content is the result of decreased chondroitin-sulphate concentration, decreased PG size, and increased collagen-PG interaction; the latter leaves less polar groups as binding sites for water.[1] The fibril diameter of the collagen in the NP increases; type II start to resemble type I fibers.[1] Cell viability decreases with age: 2% of cells in the newborn exhibit necrotic changes versus 50% of cells in young adults. In the elderly up to 80% of cells may show signs of necrosis.[1,4]

Collagen content of the anulus fibrosis (AF) increases, but elastic fiber content drops from 13% at age 26 to 8% at age 62. Collagen fibril diameter decreases resulting in less of a distinction between anular and nuclear fibers.[1] The number of anular lamellae decreases with age: older disks contain only 80% of the number of lamellae of younger disks. Layer irregularity increases with age: the number of incomplete layers is 10% greater in the older IVDs. A more than twofold increase in the the thickness of the remaining layers accompanies this reduction in the number of lamellae.[5] The lamellae may also be more widely separated. Anular fibers in older disks have larger interbundle spacing and a more irregular distribution of fiber bundles.[5] Successive layers may lose their opposite fiber direction.[6] Type I to II collagen ratio increases in the outer posterior AF, but decreases anteriorly.[1,7] Anular water content remains fairly constant: it decreases from about 80% at birth to 67% at age 30, and increases again to 73% at age 80.[3]

Between ages 20 to 65 the endplates become thinner.[1] The cartilaginous endplates calcify with age[4,6] and may even be replaced by bone.[6] Cell death occurs in the superficial layers and the subchondral bone gradually occludes the vascular channels.[1]

Declining nutrition affecting cell function may explain these age-related changes. Endplate calcification and a decreased number of arteries in the peripheral AF seem plausible explanations.[4] However, the biochemical changes in the disk precede endplate calcification.[1] Other possible mechanisms include accumulation of degraded matrix macromolecules and decreased water concentration interfering with diffusion, cell senescence with decreased biosynthesis in the viable cells, and accumulation of partially degraded molecules interfering with the synthesis or assembly of new molecules.[4]

The loss of disk height due to a decrease in water content may seem an attractive explanation for the loss in body height associated with aging. However, the greatest loss of water content from the NP occurs during childhood and adolescence. Dehydration is only some 6% from early adult life to old age, a period associated with the biggest height decrease.[1,8]. Disk dimensions in fact increase with age: the anteroposterior diameter increases some 10% in women and 2% in men between the second and seventh decade. Most IVDs increase about 10% in height.[1] Loss of vertebral body height is the principal reason for the overall height decrease.[8] The number of horizontal trabeculae in the vertebral body decreases, most markedly in the central area under the NP. Vertebral body microfractures increase endplate concavity.[1] The disks expand centrally by an increase in convexity of their upper and lower surface to adapt to the change in vertebral body shape. Disk dessication and narrowing is by no means universal and should invite consideration of a process other than normal aging.[1,8]

Age-related changes affect the mechanical properties of the disk. The NP may appear fibrous, but continues to exhibit hydrostatic pressure.[9] It does become less able to directly transmit weight or exert radial pressure on the AF. As a result, the AF bears more axial compression loads. Anular stiffness increases with age due to a decrease in the amount of elastic fibers and an increase in PG-collagen binding. A decreased water content may decrease the contribution of the toe region of the stress-strain curve, causing an increase in stiffness. Stress-strain curves are explained in monograph 11.2.1. In contrast, tensile strength decreases due to a decrease in total number of lamellae, an increase in the number of incomplete lamellae, more irregular fiber orientation, a decrease in fibril diameter, and a weakening of the insertion of the AF into the bone.[1,4,6] Increasing age is significantly correlated with a decrease of the energy required to cause failure.[10] Increased mechanical stresses in combination with decreased tensile properties and increased stiffness predispose the AF to failure; cracks and cavities enlarge to become clefts and overt fissures.[1] Increased stiffness in the IVD tissues is also the main cause for a reduction in the range of motion (ROM): experiments in which the posterior ligaments are removed in older spines do not greatly increase ROM.[1]

Aging also affects viscoelastic or time-dependent mechanical characteristics. Creep is the progressive deformation of a structure under a constant load well below its fracture point; it probably occurs as a result of progressive polymer distortion and fluid displacement in the disk.[11] Hysteresis describes a phenomenon whereby a distorted tissue dissipates energy; when recovering from a distortion the tissue releases less energy than was required for its initial deformation.[11] The surface area between a load-deformation curve plotted for loading and unloading represents hysteresis loss.[12] Hysteresis recovery largely depends on IVD waterbinding capacity.[11] Twomey[13] showed a trend towards increased creep in older spines under prolonged traction. Prolonged flexion loading caused greater creep and slower hysteresis recovery in older spines.[11] Decreased waterbinding capacity decreases the ability of the NP to deform rapidly in response to external force.[11] Slower NP deformation results in a relative increase in contribution of creep to total ROM. Decreased waterbinding capacity may also explain the slower hysteresis recovery.[1,11]

In summary, increased compressive loading on the AF, increased anular stiffness, and decreased tensile strength predispose the older AF to failure.[1,4,6] The increased stiffness of the IVD tissues is a major reason for decreased segmental rotation in all planes.[1] Increased creep deformation[11,13] and slower hysteresis recovery[1,11] may predipose the spine of older subjects to failure with prolonged or repeated loading more easily than those of younger subjects. Disk narrowing and dessication are not normal age-related changes.[1,8]

Zygapophyseal joint

Fetal and infant zygapophysial joints (ZJs) are oriented in the coronal plane. The sagittally oriented posterior two-thirds of the joints form during early childhood.[14] The coronal and sagittal portions serve different functions illustrated by the difference in age-related changes between the 2 components. The

changes in the anteromedial part are consistent with limiting the forward translation associated with flexion.[14] Focal changes here start in the fourth decade likely due to repeated compressive stresses and include cell hypertrophy, vertical cartilage fibrillation, and subchondral sclerosis. The changes are more pronounced in the superior than in the inferior facets.[1,14] The subchondral bone increases in thickness between age 20 to 50.[1] Thickness increases more in the anteromedial than in the posterior part of superior facet. The calcified zone of the cartilage also thickens mainly in the coronal part of the joint. The relative thinness of the subchondral bone and calcified zone in the posterior part of the joint argue against compression loading as occurs in the anteromedial part. In this sagittally oriented posterior portion of the ZJ, cartilage tends to split parallel to the cartilage-bone interface. This pattern of fibrillation suggests the presence of shearing forces. The multifidus muscle through its capsular attachment creates these posterior shearing forces between the cartilage and the underlying bone resulting in avulsions of portions of the cartilage. The damaged cartilage sometimes retains its continuity and with the capsule to which it remains attached forms a *fibrocartilaginous meniscoid inclusion*. Its origin is attested by the template-like fit into the underlying cartilage which generally shows signs of repair.[14] Taylor and Twomey[14] hypothesized that this irregular inclusion, if entrapped in the posterior aspect of the ZJ, may create a painful *acute locked back*, especially when the posterior ZJ aspect opens up with an inactive or inhibited multifidus muscle.

The cartilage thickens with aging.[1] Glycosaminoglycan (GAG) content initially increases, plateaus between the third and the fifth decade, and rises again in older age. Loss of PGs in the superficial cartilage may cause this plateau, offsetting an increase of PGs in deeper layers with age.[15] Collagen content decreases with age. Cartilage hydration increases between the second and fourth decade. Disruption in the collagen network allows for the increased hydration causing the observed thickening. However, it is also the primary cause of decreased compressive strength and cartilage destruction.[15] In areas of focal destruction and thinning fibrofatty inclusions increase in size to fill the space vacated by the cartilage.[1]

The subchondral bone thins and becomes relatively avascular during the sixth through eighth decade. The calcified zone of the cartilage becomes grossly thickened and irregular; the collagen content in the superficial layer increases. The cartilage contains fewer cells with smaller nuclei. Cartilage fibrillation is more severe in the polar than in the central ZJ regions. Older joints lose the distinction between the changes in the anteromedial and posterior portion. Osteophytes form at the attachments of the flaval ligament or capsule at the anteromedial margin of the superior articular facet. Enlarged synovial pads accompany these osteophytes forming a cushion between the inferior articular process and the osteophyte. An area of sclerotic bone develops in the lamina at the base of the superior articular process. A fat pad formed by the inferior articular joint recess separates this area of sclerotic bone from the tip of the adjacent inferior articular process. Wrap-around bumpers are extensions of the edges of the cartilage curving around the posterior aspect of the inferior articular process. They may be the result of repeated stress during rotatory movements.[1,14]

In summary, it is easier to distinguish age-related from degenerative changes in the IVD than it is in the ZJ. Loss of containment by an intact collagen network may allow for greater movement of water in and out of the cartilage during segmental movement; this may explain increased segemental mobility in axial rotation and sagittal plane posteroanterior translation in older ZJs.[1] Entrapment of a degenerative intra-articular fibrocartilaginous inclusion may play a role in acute painful locked back.[14] Structural changes may occur in ZJs of relatively young patients; these changes may cause low back pain (LBP) independent of damage to the IVD.[15]

Sacroiliac joint

At birth the auricular surfaces are sagittally oriented. This orientation changes to the adult orientation descibed in monograph 11.2.3. There are 2 types of joint surface irregularities.[16] Ridges and depressions are clearly visible, osseocartilaginous irregularities, complementary on opposing surfaces. Vleeming et al[16] found this type in all 47 sacroiliac joint (SIJ) specimens, even in the SIJ of a 12 year old boy. Joint surface texture consists of barely visible, noncomplimentary, cartilaginous irregularities. Surface texture is smoother on the sacral than the iliac side.[16] Texture differences between the opposing surfaces decrease with age with coarseness visible on both sides of the SIJ. The histological differences between the iliac and sacral sides described in monograph 11.2.3. cause earlier degeneration of the iliac cartilage[17]: early osteoarthritic changes (surface fibrillation and PG depletion) can start in the third decade. Large crevices and surface erosions may be present especially in middle-aged men. Similar changes also occur in women some 10 to

20 years later. Sacral surface changes do not occur until the fourth or fifth decade.[18] Vleeming at al[19] determined the friction coefficient of SIJ specimens; it was highest in SIJs with complementary ridges and depressions combined with coarser surface texture. The influence of texture on the friction coefficient was smaller than that of the ridges and depressions. Higher friction coefficients increase SIJ stability decreasing demands on ligamentous structures. The auricular surfaces of women have less pronounced ridges and depressions than those of men.[16] The moments on the SIJ of the superimposed body weight are larger in men than women due to a more ventral location of the center of gravity. Retaining SIJ mobility after puberty is more important in women because of child bearing.[16] The increased joint surface irregularities observed mainly in men may be the result of normal aging and adaptation to imposed mechanical stresses rather than degeneration.

Bowen and Cassidy[20] found sacral osteophytes appearing in the fourth decade. Peripheral osteophytes enlarged, especially anteriorly and superiorly, beginning to interdigitate across the joint space in the sixth and seventh decade. Some SIJs had fibrous intra-articular connections. Incidence of these changes increased in the eighth decade. An intra-articular bony ankylosis was present in 1 male specimen only. Walker[21] found incomplete chondroid ankylosis in some (but not all) SIJ specimens from the seventh through ninth decade and fibrous ankylosis in 9 of 18 SIJs. She observed no intra-articular bony ankylosis, nor chondroid or bony ankylosis of the interosseous ligaments in any specimen. The effect of these ankyloses on ROM is unclear. Studies have documented no decrease in ROM with age in patients up to age 45[22] or in normal subjects up to age 50[23]. Sturesson[24] found a significant increase in ROM with age with some transfers. Vleeming et al[25] concluded that ankylosis is not a normal result of aging; they found movement in the SIJ of 4 pelves between 73 and 83 years old.

In summary, changes in joint surface texture and irregularities are likely physiological rather than pathological.[16] Changes in joint surface orientation and increased joint surface irregularities limit multiplanar and multiaxial SIJ motion, as well as the extent of this motion. ROM decreases with age, more in men than in women, yet true ankylosis is rare.[20-25]

Sacrococcygeal and intercoccygeal joints

In younger subjects disks may be interposed between coccygeal vertebrae. In adult men these joints commonly ossify. Ossification in females generally occurs later. The sacrococcygeal disk may ossify with age.[26] No studies on incidence are mentioned. Decreased ROM may affect the length-tension relationship of the pelvic floor muscles and thus their role in lumbopelvic stability.

Pubic symphysis

In children the interpubic disk is narrow and the hyaline cartilage is quite wide. With age the disk increases in width at the expense of the cartilage.[27] The cleft in the interpubic disk is sometimes present at birth, but usually forms in the second through tenth year of life due to softening and absorption of the fibrocartilage.[26-28] Effect of these changes on symphyseal ROM have not been researched, but they would appear to favor increased ROM.

Ligamentous structures

With age, elastic fiber content of the flaval ligament decreases; collagen content increases. A shift to high molecular weight PGs favors calcium deposition leading to ossification. The resultant hypertrophy and stenosis may cause cauda equina syndrome and radiculopathy.[29] Decreased elasticity may cause inward bulging even without changes in ligament thickness.[30]

In the interspinous ligament (ISL) age-related chondrification occurs after the third decade.[29] Calcification is physiologic after age 50.[31] Yahia et al[31] showed ISL calcification in 30 to 40 year old patients with disk herniations. It is unclear whether this calcification is the cause or result of disk disease. The calcification and chondrification may diminish the ability of the thoracolumbar fascia to influence vertebral position through the ISL-supraspinous-thoracolumbar connection.[29] Fat deposition and ossification of the supraspinous ligament (SSL) occur later in life.[29]

DISEASE

Numerous diseases can potentially cause LBP. In this era of direct access to physical therapy (PT) and after minimal contact with a primary care provider due to managed care restrictions, there is an increased

need for the PT to screen patients for undiagnosed disease which may affect prognosis, intervention, and/or be a reason for referral. This review is not meant to be exhaustive, but is intended to cover some common and/or potentially serious medical problems affecting the lumbopelvic region.

Visceral disease

Visceral disease is pain originating in the internal organs; it can cause pain in a number of ways.[32,33] True visceral pain is a deep, dull, aching, and diffuse pain felt at the site of stimulation. Deep somatic pain results from irritation of the parietal peritoneum; it is sharp, intense, and often associated with reflex abdominal muscle spasm. Disorders in the viscera can cause depolarization of nociceptive fibers originating in the internal organs. Pain may be perceived as true visceral pain originating in the affected internal organ or as somatically-mediated referred pain in a more superficial tissue supplied by the same segment of the spinal cord. This is the result of convergence in the cord of multiple primary afferent (including nociceptive) neurons on a smaller number of secondary afferent neurons.[34] Visceral referred LBP is more likely to result from visceral disease in the abdomen and pelvis; intrathoracic visceral disease more commonly refers to the neck and shoulder.[32,35] Table 1 summarizes areas for pain referral from internal organs.[33,35]

Goodman and Snyder[32] noted that the prominent finding in case of LBP of visceral origin is the presence of full and painless ROM in the lumbar spine. This may not always be the case. Primary afferent neurons also diverge upon entering the cord and connect to anterior horn motor neurons and lateral horn preganglionic sympathetic neurons. Projection onto motor neurons may lower their threshold to depolarization increasing local and segmental muscle tone. Increased activity of preganglionic sympathetic neurons may cause[34]:

- Segmental hyperesthesia/hyperalgesia due to a decreased threshold in the sympathetically innervated sensory organs, such as the Pacinian corpuscles.
- Further increase in segmental muscle tone through sympathetic innervation of muscle spindles.
- Decreased segmental circulation as a result of sympathetically-mediated vasoconstriction.

Prolonged sympathetic hyperactivity will affect segmental tissue homeostasis. Therefore, prolonged lumbopelvic visceral dysfunction may present with diffuse segmental symptoms of a musculoskeletal nature extending from the low back into the lower extremities making the correct diagnosis of a primary nonmusculoskeletal problem more difficult.

Retroperitoneal region

The kidneys, bladder, ureters, and prostate gland are located in the retroperitoneum, separated from the gastrointestinal system by the peritoneal membrane.[35] The retroperitoneal organs can refer pain to the low thoracic, lumbar, and pelvic area. Pain can also refer around the flanks into the lower abdomen, genitalia, and anterior and medial thighs.[35] Kidney pain is usually felt at the costovertebral angle just lateral to the paraspinal muscles of T12 and L1. It is a true visceral pain resulting from the acute distension of the capsule of the kidney. It is usually dull and constant in character.[33] Kidney stones can cause urethral pain. Capsular distension results in a dull flank pain; obstruction at the uretopelvic junction can cause “colicky” pain.[33] Colic is associated with spasming of a hollow internal organ.[32] Colicky pain may be felt in the testicle or vulva if the stone is lodged in ureter or bladder. The bladder can also cause a mild, diffuse LBP in patients with severe cystitis; chronic cystitis and obstruction can result in persistent LBP.[33] The prostate gland may cause a nagging lumbar or sacral pain.[35]

Pelvic organs

Nociceptive information from the female genital organs is transmitted by sympathetic nerves that travel to (T10)T11 and T12 and through parasympathetic nerves that connect with the S2 to S4 segments.[33,35] In monograph 11.2.3. we discussed the location of the preganglionic sympathetic origin segments at T10 to L2 and the symptoms thoracolumbar nociception can cause in the lumbopelvic spine and lower limbs. Pain originating in the uterus can be caused by a tumor, an abnormal uterine position, or dysmenorrhea. Fallopian tube pain often is the result of infection. Ovarian pain can occur in patients with benign ovarian neoplasms; these patients experience LBP due to torsion or compromise of blood supply to the ovary or cyst. Malignant neoplasmata can cause LBP by way of direct extension or lymphatic spread.[33] Endometriosis can cause recurrent perimenstrual sacral and lumbar pain.[35]

Gastrointestinal system

Diseases of the pancreas, duodenum, galbladder, and colon all can cause LBP.[33] Somatic pain from the pancreas is located in the midepigastric area. Referred pain is located at the L1 spinal level. Pain of the head of the pancreas is felt to the right of the spine; pain of the pancreatic body and tail are felt left to the spine. Duodenal pain is usually caused by a duodenal ulcer. A burning epigastric pain commonly starts 1 to 3 hours after eating and awakens the patient from sleep.[32,33] The pain is relieved by food and antacids.[35] Erosion of the posterior wall of the duodenum can cause back pain in a minority of patients. Galbladder pain from acute cholangitis is located in the abdomen; patients may present with local tenderness and fever. Pain can refer to the tip of the right scapula and the thoracolumbar spine.[33,35] Diseases affecting the rectum may produce colonic pain. Diverticulitis can be associated with fever and a change in bowel habits and may result in acute, persistent pain starting in the left lower abdominal quadrant and radiating to the low back.[33]

Vascular disease

Even though we could classify vascular disease as visceral, the high incidence of vascular disease and its ability to mimick musculoskeletal complaints warrant a separate discussion. The abdominal aorta bifurcates into the common iliac arteries at the level of the L4 vertebral body.[27] Vascular pain from an abdominal aortic aneurysm is due to compression of surrounding structures or extension or rupture of the aneurysm. Patients usually complain of a dull constant abdominal pain, unrelated to activity. Back pain mostly occurs together with epigastric discomfort and may radiate to hips or thighs.[33] Goodman and Snyder[32] noted that severe, tearing pain occurring with sweating and dizziness may be related to an expanding aortic aneurysm. Rupture of the aneurysm is life threatening and causes excruciating pain, circulatory shock, and an expanding abdominal mass.[33]

Atherosclerosis, the hardening of fatty substances in the arteries, is the most common cause of peripheral arterial disease.[32] Table 2 list risk factors for atherosclerosis.[25,32,36] A gradual obstruction at the level of the aortic bifurcation can produce bilateral buttock and leg pain, weakness and fatigue of the legs, atrophy of the lower extremity muscles, absent femoral pulses, and color and temperature changes in the lower extremities. Pathology of the iliac artery may cause buttock and LBP, and pain and numbness in the affected leg. Femoral artery obstruction may cause thigh and calf pain and popliteal artery obstruction may produce ankle and foot pain; both cause absent or decreased pulses below the level of obstruction.[32] The lower limb cramping, aching, numbness, tightness, or fatigue reported by patients with arterial insufficiency may mimick sciatic distribution symptoms.[36] Occlusion causes vascular claudication: symptoms are produced by walking or exercise and relieved by rest irrespective of lumbar position distinguishing this type of claudication from neurogenic claudication. Occlusion at the aorto-iliac junction is the most common site for atherosclerosis in people under age 40.[36]

Hematologic disease

Hematologic diseases capable of producing LBP are hemoglobinopathies and myelofibrosis.[33] Back pain may result from hyperplasia or replacement of bone marrow in the axial skeleton. Hemoglobinopathies are a group of diseases in which defects in the structural proteins of hemoglobin produce changes in erythrocytes. This results in obstruction of microvascular circulation. The most common hemoglobinopathies are sickle cell anemia, sickle cell hemoglobin C disease, and sickle cell beta-thallasemia. Most patients with sickle cell anemia are diagnosed in childhood as a result of a painful vaso-occlusive crisis. Sickle cell anemia is the more serious of the three hemoglobinopathies: in the musculoskeletal system it can cause bone infarction, joint effusion, hemarthroses, septic arthritis, and osteomyelitis. In adults vaso-occlusion can present as back and extremity pain which lasts 4 to 5 days. Physical findings during a vaso-occlusive crisis may include tenderness to palpation, fever, tachycardia, hypertension, tachypnea, tender and rigid abdomen, and abnormal breath sounds with signs of pleural disease. The hemoglobinopathies may also affect the SIJ.[33] Exacerbation or occurrence of LBP and limb pain and nonmusculoskeletal signs as mentioned above in a PT patient with known sickle cell anemia is cause for referral.

Trauma

Fractures may result from major trauma; stress fractures are more insidious and are often overlooked. Severe or rapidly applied forces can cause a burst fracture of the vertebral body; the vertebral body collapses and bone fragments may be forced into the spinal canal. Combined flexion and compression may cause an anterior wedge fracture; this type of fracture affects the trabeculae in the anterior portion of the

vertebral body. Extreme hyperflexion can result in an avulsion fracture of the posterior margin of the vertebral body by tension in the posterior AF.[37] Such serious lumbar fractures and fracture-dislocation through the SIJ usually also involve significant trauma and are frequently associated with other injuries.[18] Therefore, PTs are unlikely to see patients with major fractures without a proper identification and diagnosis by a physician. We are however likely to see patients with undiagnosed stress fractures. There are 2 types of stress fractures[38]: fatigue fractures and insufficiency fractures.

Fatigue fractures

Fatigue fractures occur when abnormal stress is applied to normal bone.[38] Crockett et al[38] and Weber et al[39] mentioned sacral fatigue fractures in military recruits, joggers, gymnasts, and a woman gaining 40 lbs over the course of her pregnancy. Pain as a result of sacral fatigue fractures is usually poorly localized.[39] Athletes usually present with a history of repetitive axial loading of the spine.[38] Crockett et al[38] described a 20 year old basketball player who started complaining of diffuse buttock pain after using a plate-loaded jumping machine in the off-season. Examination revealed normal strength, sensation, and range ROM in both legs with only diffuse left buttock pain on deep palpation despite bilateral fatigue fractures of the ala of the sacrum.

Another type of fatigue fracture that may go undiagnosed is spondylolysis. Spondylolysis involves fracture of the interarticular pars.[40] Long regarded as a congenital failure of fusion of 2 growth centers, it is in fact a pseudarthrosis of a childhood fracture frequently persisting into adult life.[40] It is seldom found in patients below 5 years of age, but at age 8 incidence is 4 to 5%. Incidence increases another 1 to 2% by age 18.[41] The interarticular pars may be hereditarily weakened.[40] Forces acting on the inferior articular processes cause the fracture. Forward shear, flexion, extension, and compression all exert forces on the inferior facets. In the lower lumbar spine forward shear forces during upright standing move a superior vertebra anterior and inferior in relation to the inferior vertebra. Tension in the ZJ capsules physically bends the bone of the inferior articular facets backwards about the interarticular pars. Lumbar extension and compression also bend the inferior facets backwards: hyperextension and compression may cause spondylolysis. Repetitive alternation between flexion and extension causes large stress reversals in the interarticular pars and may explain the high incidence of spondylolysis in sports that require frequent flexion and extension of the lumbar spine[37]: spondylolysis is more frequent in gymnasts, wrestlers, football linemen, and butterfly swimmers.[41,42] Spondylolysis may cause spondylolisthesis or forward slipping of a vertebra on a subjacent vertebra. The resulting spondylolisthesis is called isthmic or type II spondylolisthesis. There are 2 types of isthmic spondylolisthesis: in the lytic type there is a defect in the pars, in the healed type the pars has presumably healed and is elongated. Table 3 lists the different types of spondylolisthesis. Though often an asymptomatic radiographic finding, patients with an anterior spondylolisthetic slippage over 25% are at greater risk fo LBP.[41] Spondylolisthesis may cause bilateral neurogenic claudication or unilateral L4 or L5 distribution symptoms.[41] Spondylolisthetic LBP may originate at the IVD at the same level or the level above, the ZJs, and the segmental musculoligamentous structures.[40] Eisenstein et al[40] found a well-developed ligamentous structure bridging the spondylolytic defect in 8 patients. Sympathetic and thinly myelinated nerve fibers present in and around the ligament in 6 preparations made them hypothesize a role for this “spondylolysis” ligament in nociception.

Excessive flexion and extension in standing seem contra-indicated in an unstable listhesis: they increase segmental anterior shear forces due to the concurrent sacral nutation. Spondylolisthesis increases the stabilization demands on the IVD and polysegmental muscles, the only structures still bridging the segment. Instability may alter the response of the segment to mechanical forces. Mechanical forces should only be applied after a thorough evaluation of segmental stability.

Insufficiency fractures

Insufficiency or pathologic[43] fractures occur when normal forces are applied to abnormal or weakened bone.[38] Metabolic, infectious, neoplastic, and congenital conditions can weaken the bone predisposing it to this type of fracture. We will discuss these conditions later. At age 70 the annual incidence rate of vertebral insufficiency fractures is 20%; at age 85 the female prevalence rate for one or multiple vertebral fractures rises to 50%.[43] Resultant deformities are wedge vertebrae in which the anterior portion of the vertebral body has collapsed, a crush pattern with compressive collapse of the entire vertebral body, and a biconcavity of the body as a result of fracture of the endplates with intrusion of the IVD.[43] The acute pain associated with a compression fracture superimposed on chronic back pain may be the only symptom; a history of trauma is frequently missing. The patient may recall a “snapping” noise associated with mild pain, no pain at all, or

delayed pain; small fractures may occur in articular processes or the trabeculae supporting the vertebral endplates causing severe pain which resolves with a few days of rest[32]; larger fractures cause pain that increases with prolonged sitting, standing, and a Valsalva maneuver. This pain may resolve over 3 to 4 monts as the fractures heal, or it may persist as a result of microfractures and the biomechanical effects of the resultant deformity.[33] Percussion over the fractured vertebrae may be painful. Acute fractures may cause urinary retentention; ileus can cause a loss of bowel sounds. Because most osteoporotic fractures only involve the anterior elements, neural compression is uncommon.[33]

Sacral insufficiency fractures are not uncommon, especially in elderly women with osteopenia, and are a frequently unsuspected cause of LBP in the elderly.[38] Weber et al[39] in a prospective study of 2,366 women with an average age of 79 years old admitted for LBP found sacral insufficiency fractures in 20 patients. In their literature review Weber et al[39] found that of the 231 cases reported, 214 (93%) were female. The medical history included primary osteoporosis, a history of pelvic radiation therapy, prolonged steroid medication use, multiple myeloma, alcoholism, and/or obesity. Eleven patients reported a fall to the ground from sitting or standing. All complained of dull LBP, but only 1 reported pain radiating from the buttock to the knee. Fifteen complained of pain with direct pressure over the sacrum; the neurologic exam was negative in all patients. Plain radiography was able to detect the sacral insufficiency fracture was established in only 1 of 20 patients. Nuclear scintigraphy and CT scans were needed for the diagnosis of the other patients. Of 20 patients, 18 had longitudinal fractures lateral to the sacral foramina explaining the absence of neurologic involvement. Sixteen of 20 patients had associated fractures of pubic ramus, iliac bone, and vertebral bodies. Weber et al[39] noted that the sacral insufficiency fractures are apt to be missed by the physician.

Metabolic and endocrine disease

There are 3 metabolic or endocrine diseases with the potential affect the structural integrity of the vertebral and sacral bone: osteoporosis, osteomalacia, and Paget's disease. Diabetic radiculopathy is a differential diagnostic option for sciatic distribution pain.

Osteoporosis

Osteoporosis is a condition in which decreased density of normally mineralized bone can lead to mechanical skeletal failure resulting in fractures from minimal trauma. Osteoporosis mainly affects the more metabolically active trabecular bone explaining the seemingly disproportionate involvement of the vertebral bodies in osteoporosis. Trabecular loss is highest among the horizontal trabeculae in the vertebral bodies. These horizontal trabeculae serve as tie-beams for the vertical trabeculae and their loss decreases the ability of the bodies to resist compression. We discussed in monograph 11.2.3. how bone mineral content is directly related to compressive failure of the vertebral bodies. There are 3 types of primary osteoporosis.[43] Juvenile idiopathic osteoporosis occurs in early adolescence and mainly causes axial skeleton fragility. Type I or post-menopausal osteoporosis is 6 times more common in women, starts between age 50 and 70, and is caused by estrogen deficiency. Type II or senile osteoporosis is only twice as common in women than men, usually occurs after age 70, and affects both trabecular and cortical bone. Type II osteoporosis may be caused by decreased osteogenesis, aggravated by secondary hyperparathyroidism. Table 4 lists causes for secondary osteoporosis.[33,43] The classic presentation, however, remains that of a pale, frail, nulliparous female with a family history of osteoporosis and a personal history of early menopause. The risk of osteoporosis is further increased by a history of smoking and alcohol and caffeine intake. If previous data are available examination may reveal decreased height[43] We discussed the examination findings associated with minor fractures and acute compression fractures.

Osteomalacia

Osteomalacia is characterized by insufficient mineralization of newly formed bone matrix. The overall rate of bone formation is also decreased. The combined effect is decreased bone mass with diminished mechanical properties. Osteomalacia is most commonly caused by vitamin D deficiency. Less frequent causes include malabsorption syndromes, hepatic and renal disease, prolonged use of anti-convulsant drugs, phosphate deficiencies, and some rare genetic disorders. Examination findings may include local tenderness over the site of microfractures, enlarged costochondral junctions, muscular weakness, (hypocalcemic) tetany or muscular cramping, kyphoscoliosis, and coxa vara as part of generalized lower extremity bowing. Skeletal pain and tenderness may be generalized, but is frequently restricted to the lumbar spine.[33,43]

Paget's disease

Paget's disease is characterized by excessive activity of some osteoclastic and osteoblastic cells, bone marrow replacement by hypervascular fibrous tissue, and a resultant mosaic-like disorganization of trabecular and cortical bone. It does not seem to affect people below the age of 20, and is rare before age 40. The prevalence is equal in men and women, affecting 3 to 4% of individuals after age 40 and up to 10% by age 90. The disease affects the lumbar spine in 60% and the sacrum in 45% of patients. Central or lateral stenosis, compression fractures, and arthritic ZJ changes may cause the LBP associated with Paget's disease. This pain usually is insidious in onset, intermittent or constant, and may be related to the weather or to activity. Protrusion of the femoral head into the acetabulum, hip arthrosis, pathologic subtrochanteric fractures, and stenotic neural compression may cause leg symptoms. Nonmusculoskeletal symptoms may include increased hat size due to an enlarged skull, hearing loss, tinnitus, vertigo, headache, high-output congestive heart failure due to arteriovenous fistulae in the bone, and the neurologic sequelae of basilar invagination due to bony softening around the foramen magnum. Malignant sarcomatous degeneration of Pagetic bone occurs in approximately 1% of patients and in 10% of patients with multiple bone involvement and is usually fatal. Examination usually reveals a flat lumbar spine, a slow gait, and sometimes anterolateral bowing of the lower legs: this "saber shin" may be warm to the touch.[43]

Diabetic radiculopathy

Diabetes may also mimick musculoskeletal dysfunction. People with diabetes frequently present with a symmetrical bilateral sensorimotor polyneuropathy: hypesthesia occurs in a glove-or-stocking distribution, small muscles may be atrophied, hyporeflexia and decreased vibratory sensation are symmetrical. The polyneuropathy generally has a gradual onset and is painless. In diabetics, sudden onset unilateral sciatic distribution pain may be the result of diabetic radiculopathy. Nerve root tension signs are likely absent. Objective sensory disturbances are less pronounced in patients with diabetic versus compression radiculopathy. Naftulin et al[44] reported sphincter disturbances are rare. Asymmetrical reflex changes are due to the radiculopathy rather than to the concurrent polyneuropathy. Lumbar ROM may be unrestricted and not increase radicular pain. Important for diagnosis is the fact that some 20% of patients will have multiple episodes of diabetic radiculopathy.[44]

Infectious disease

The bony lumbopelvic area can be affected by infectious diseases. The invading organism may be viral, bacterial, fungal, or parasitic.[45]

Diskitis

Isolated diskitis (infection limited to the IVD) mainly affects pediatric patients. The cause is usually bacterial, sometimes viral. Spreading is hematogenous. The higher incidence in younger patients can be explained by the arteriolar blood supply to the disk which exists during infancy and childhood and which is slowly obliterated in the first three decades.[45] Diskitis in adults is mainly a post-operative complication of discectomy[45], but the incidence of adult diskitis has increased among intravenous drug users and immuno-suppressed patients.[46] Diskitis causes severe localized tenderness over the affected disk space with associated paraspinal muscle spasm. Pain may radiate into the abdomen, pelvis, and legs. Lumbar ROM is decreased and an SLR test may be positive. Fever is common.[32] Diskitis can progress to osteomyelitis.[45]

Vertebral osteomyelitis

Vertebral osteomyelitis is relatively rare. Roberts[47] mentioned an annual incidence of pyogenic vertebral osteomyelitis of one per 250,000 individuals, accounting for only 2 to 4% of all cases of osteomyelitis.[45] Pyogenic osteomyelitis is the result of a bacterial infection. Organisms causing an immune reponse with granuloma formation are responsible for granulomatous osteomyelitis. Parasitic osteomyelitis is rare in the developed world.[45] All infections are usually limited to the anterior and middle columns. Infection can spread into the posterior column by direct extension.[45] Roberts reported primary posterior element involvement.[47] The preference for the vertebral body is the result of hematogenous spreading through the venous plexus lining the spinal canal or the arteries supplying the vertebral bodies.[33,45]

Pyogenic disease

Staphylococcus aureus is the primary organism in 50% of patients. *Escherichia coli* is associated with infections of enteric or urogenital origin. *Pseudomonas aeruginosa* and *Candida* may be responsible in intravenous drug users and immuno-suppressed patients. Other bacteriae involved are *Staphylococcus epidermidis*, *Streptococcus*, and *Proteus*.[33,45] Pyogenic disease usually has an insidious onset. The L1 and L2 vertebral bodies are the most commonly affected in the axial skeleton.[33] Local back pain is the most common

complaint. Initially related to activity and relieved with recumbency, eventually this pain may become so severe it is no longer relieved even with complete bed rest and it is often mistaken for exaggerated illness behavior.[45] Neurologic signs are usually absent until later in the disease when collapse of a vertebra or an epidural abscess may compress neural structures. A collapsed vertebra may cause local kyphosis. The most common site for an epidural abscess is adjacent to the posterior longitudinal ligament (PLL); other epidural areas may be involved if the infection occurred as a result of posterior spinal surgery. The abscess may extend to the flank or groin.[45] A psoas abscess can cause a hip flexion contracture and a positive pain on hip extension.[33,45] Sometimes an abscess will extend to the perirectal or even popliteal area or communicate with the skin by way of a chronic, draining sinus. It may be palpated as local or remote fluctuant mass. Nonmusculoskeletal signs and symptoms may include chills, weight loss, dysuria, and photophobia. However, usually the patient is nonfebrile: Vincent and Benson[45] reported temperatures over 100 F in only 33% of patients. Table 5 summarizes risk factors for pyogenic osteomyelitis.[45,46]

Granulomatous disease

Granulomatous osteomyelitis also has an insidious onset. Massive destruction of vertebral bodies, disks, and ligaments may eventually result in collapse and angulation of the spinal column. This angulation in combination with inflammatory debris and necrotic material may compress the spinal cord and cauda equina. Paraplegia may also occur as a result of compression or inflammatory coagulation of the anterior spinal artery supplying the cord.[45] Pott's disease (tuberculosis of the spine) is the most common granulomatous infection caused by *Mycobacterium tuberculosis*. The primary visceral focus may be in the lungs, the lymphatic system, the kidneys, or another internal organs. The spread is hematogenous. In tuberculosis, 50% of the osseous involvement is found in the spine. The primary focus can be quiescent and the patient may even never have been diagnosed. Abscesses located under the anterior longitudinal ligament (ALL) and the periosteum of the vertebral body cause extension to adjacent segments and contribute to devascularization and destruction of the vertebral bodies. Other granulomatous diseases are the result of fungal infections. Infection is usually through the respiratory tract with subsequent hematogenous spread. Table 6 describes fungal and bacterial infections.[45]

Parasitic disease

Parasitic infections are rare in developed countries. Echinococcosis occurs in cattle and sheep raising areas; it is the result of infection with either *Echinococcus granulosus*, or *E. multilocularis*. Patients ingest the eggs of these parasites through water infected by cattle, sheep, or dog faeces. Embryos hatch from the eggs after ingestions, traverse the intestinal wall, and spread hematogenously. Bone is the primary focus of infection in 1 to 2%; spine (50%) and pelvis are the most common locations. Patients present with pain and deformity and are at risk for neurologic compromise due to vertebral collapse.[45]

Sacroiliac osteomyelitis

SIJ infection spreads to the joint hematogenously. Trauma, pregnancy, cutaneous infection, endocarditis, intravenous drug use, and immunosuppression may be predisposing factors.[18] Osteomyelitis usually starts in the iliac bone and extends into the joint. Patients may be febrile and appear acutely ill. They will avoid weight bearing on the affected side preferring to hold the hip in a flexed position. The distended anterior joint capsule may compress the lumbosacral plexus and cause femoral or sciatic distribution symptoms. Micro-organisms responsible include *Staphylococcus aureus*, *Streptococcus*, *Pseudomonas*, and *Cryptococcus neoformans*.[18]

Inflammatory disease

Common spondylarthropathies include ankylosing spondylitis (AS), psoriatic arthritis, enteropathic arthritis, and Reiter's syndrome.[33,48] These diseases are characterized by peripheral joint inflammation, SIJ inflammation, a tendency towards a more diffuse spinal involvement, and, in some of these disorders, extra-articular features. Blood work usually does not show rheumatoid factor: therefore, we refer to these disorders as seronegative spondylarthropathies.[33,48] Pathologic changes occur not only in joints, but also affect entheses, the attachments of ligaments, tendons , and capsule to the bone.[48] Patients may have a genetic predisposition to these arthropathies, which is triggered by the environmental factors of trauma or infection.[33]

Ankylosing spondylitis

The prevalence reported for AS varies between 0.1 to 0.2% and 1 to 2%.[48] The classic patient is a 15 to 40 year old male presenting with a history of slowly progressive LBP and stiffness.[33] The male-to-female

ratio varies between studies from 1:1 to 4:1. As the disease is generally milder and less progressive in women, prevalence among women may be underestimated.[48] Ankylosing spondylitis (Bechterew's disease) affects the SIJs, ZJs, and costovertebral joints of the axial skeleton. The enthesopathy manifests in the spine by involvement of the insertion of the AF into the vertebral bodies: ossification of the AF produces bridging syndesmophytes, the classic "bamboo spine". Enthesopathic bony erosion and spurs at the insertion of plantar fascia and Achilles tendon into the calcaneus frequently cause heel pain. Enthesopathic involvement is common also at the ischial tuberosities, the iliac crests, epicondyles of the elbows, and shoulders. LBP and stiffness come on gradually in the second and third decade of life. Location may vary from the trochanteric and gluteal areas to the thoracic region. Radiation into the leg is common, but seldom extends below the knee. Nocturnal pain disturbs sleep, patients are stiffest and most painful in the morning, and tend to improve with exercise. Patients often adopt a flat lumbar spine posture to unload painful ZJs with a secondary forward head posture and increased thoracic kyphosis. Costovertebral involvement tends to decrease chest expansion. Over a third of patients have peripheral arthritis: frequently both hips are involved, but generally the other peripheral arthritides are asymmetric. Uveitis may cause pain and photophobia in 20 to 25% of patients. Cardiopulmonary dysfunction may also be present. Minor trauma to the rigid spine may cause fractures resulting in lumbar and thoracic pseudarthroses which may be extremely painful. Trauma or possibly infection may cause spondylodiskitis with local, use-related pain.[48] The examination may show aspecific findings of tenderness to palpation over the SIJs, a decreased lumbar lordosis, and decreased motion in all planes.[33]

Psoriatic arthritis

Psoriatic arthritis occurs in 5 to 7% of patients with psoriatic skin involvement and in 0.1% of the general population.[33] Asymptomatic oligoarthritis develops in 54% of patients with psoriatic arthritis and symmetric polyarthritis in 25%. The disease affects the spine in 20 to 21% of patients.[33,48] Patients with axial skeleton involvement tend to be men with an onset of psoriasis later in life; back pain or peripheral joint pain is frequently the initial symptom.[33] SIJ involvement in psoriatic arthritis tends to be unilateral.[48]

Enteropathic arthritis

Enteropathic arthritis refers to the association of inflammatory bowel disease with arthritis of the axial skeleton.[33,48] Patients with Crohn's disease have a higher incidence of arthritic symptoms than those with ulcerative colitis. Arthritis is more common in patients with Crohn's disease involving the colon than in those in which the disease affects the small intestine.[48] Onset is insidious with LBP and morning stiffness, but may progress to include periosteitis, bony necrosis, septic arthritis of the hip, and granulomatous inflammation of bone, synovium, and muscle. Nonmusculoskeletal signs include ulceration of the perineum, oropharynx, or rectum. Skin abnormalities of erythema nodosum and pyoderma gangrenosum (purulent abacterial skin ulcers) may be present.[33] The enteropathies may affect the ZJs and costovertebral joints[48], and often cause SIJ destruction.[18]

Reiter's syndrome

Reiter's syndrome is a reactive arthritis: it can develop soon after or during an infection elsewhere in the body.[48] The classic triad of symptoms in Reiter's syndrome is a combination of arthritis, urethritis, and ocular disease. The arthritis occurs mainly in the lumbopelvic region and the legs. Sacroiliitis may be unilateral[48] and will develop in 31 to 92% of patients.[33] Spondylitis occurs in up to 23% of patients.[33] Up to 93% of patients have genitourinary symptoms. They range from mucopurulent discharge and dysuria in men to asymptomatic vaginitis and cervicitis in women.[33] Conjunctivitis may cause redness and crusting of the eyelids; iritis will cause pain, photophobia, and scleral injection in 20% of patients. Enthesopathy may cause heel pain, Achilles tendonitis, and dactylitis (inflammation of the fingers or toes). One-third of patients have systemic signs of fever, anorexia, weight loss and fatigue. Examination findings are aspecific, but may include mucocutaneous lesions of the oropharynx, palms of the hand, soles of the feet, and nails.[33]

Other inflammatory diseases

Other rheumatic diseases which less commonly affect the lumbopelvic spine include rheumatoid arthritis, systemic lupus erythematosus, polymyalgia rheumatica, and fibromyalgia.[18,32,33] Familial Mediterranean fever is an autosomal recessive genetic disorder which may cause SIJ inflammation in children of Mediterranean ancestry following a febrile episode. Table 7 summarizes findings in inflammatory disease of the spine.

Neoplastic disease

Neoplasmata are unlikely causes of LBP.[33] Especially primary tumors are uncommon. Osteoid osteoma is a benign neoplasm. In the spine it most frequently occurs in the lumbar region. Initially intermittent and vague, the pain becomes constant with a "boring" quality. It worsens usually at night and is relieved by aspirin. Osteoid osteoma is frequently diagnosed in young adults between 20 and 30. On examination there may be a muscular hypertonicity with a resultant scoliosis: the tumor will be located in the concavity. A superficial tumor may present with swelling and redness of the overlying skin. Multiple myeloma is a malignant tumor involving plasma cells. It is the most common primary malignancy of bone in adults. Rare below the age of 40, it is found usually in patients between 50 and 70 years old. A mild intermittent LBP relieved by rest and aggravated by weight bearing is the first symptom in 35% of patients. Examination may reveal diffuse tenderness of the bone, fever, pallor, and purpura. Extradural extension of the tumor or pathologic fractures of the vertebra may cause neurologic symptoms.

Skeletal metastases are 25 times more common than primary bone tumors. Common sites for the primary tumors are prostate, lung, breasts, and kidney: autopsy studies showed that 70% of patients with primary tumors will demonstrate hematogenous spread to the thoracolumbar vertebral bodies. Patients over 50 are at greater risk for metastases. Onset of LBP is usually gradual, but of increasing intensity. Movement, coughing, and straining may increase LBP. Patients may complain of night pain.[33] A sudden exacerbation in long-standing chronic LBP in the elderly may be a signal of lumbar metastases[32]. Examination findings are very non-specific and may include pain, limited ROM, muscle spasms, and abnormal neurologic findings.[33] Sacral metastases are even harder to detect: lumbar ROM remains full and painless. Patients may only complain of sacral or coccygeal pain.[32]

Psychological influences

Perception modulates pathology, dysfunction, and nociception resulting in pain, functional limitation, and disability. Perception is influenced by pathopsychological and psychosocial factors. Pathopsychology encountered in PT practice likely fall in the categories of somatoform and *personality* disorders.[49] Table 8 describes somatoform disorders.[49] In somatoform disorders emotional factors are primary, but they are expressed as physical symptoms.[49] The incidence of personality disorders is only 1 to 2% in the general population. Despite this low incidence, the number of patients with personality disorders is high in medical or PT offices, where clinicians may tolerate dependent behavior and rarely give psychological explanations for symptoms.[49] A personality disorder is a fixed and maladaptive personal style that may result in social and/or occupational impairment. These people lack the ability to respond to social situations with a variety of styles, but rather respond in a similar fixed way irrespective of the situation. Woltersdorf[49] described personality disorders in more detail. Patients with somatoform disorders may have symptoms mimicking musculoskeletal lumbopelvic involvement. Personality disorders will not likely cause lumbopelvic-like complaints, yet recognizing their role in patient behavior may modify treatment approach and minimize clinician frustration.

Coste et al[50] studied 330 patients with local nonspecific LBP using a structured psychiatric interview based on the DSM III-classification, the manual for diagnosis of psychiatric disorders. They found a psychiatric disorder in 41.2% of these patients: 39.7% had 1 or more criteria for an anxiety disorder, 27.6% presented with depression, 33.3% had 1 or more criteria of an affective disorder, 4.8% had a generalized anxiety disorder, and 7.9% had a somatoform disorder. The nonorganic signs developed by Waddell et al[51] (Table 9) are widely used to detect inappropriate ilness behavior (IIB). Waddell et al[51] described a consistent, but small (0.18-0.29) correlation between these nonorganic signs and scores on the hysteria, hypochondriasis, and depression subscales of the Minnesota Multiphasic Personality Inventory (MMPI). Waddell et al[52] used regression analysis to determine the contributing factors to disability in 200 patients with LBP of at least 3 months' duration. They found that depression accounted for 13.4% and increased bodily awareness for 9.1% of perceived disability; psychologic testing revealed no psychiatric illness in this patient group. Waddell et al[52] suggested that the findings of positive MMPI scores are best regarded as an expression of distress rather than as evidence for pathopsychology. Polatin and Gatchell[53] note that the self-perception of disability is impacted by motivation, incentives, and reinforcement. Socioeconomic and psychosocial factors may include job dissatisfaction, involvement in litigation and secondary gain issues. Secondary gain issues may be financial in nature, but can also consist of enabling dynamics in family or other social systems to remain disabled.[53]

In summary, somatoform disorders may cause lumbopelvic region complaints. Personality disorders may affect the reponse to treatment. Patients with LBP may have psychiatric disorders.[50] These may be

concurrent, causative, or simply the result of distress caused by the LBP.[52] A careful history may identify psychosocial factors in patients with LBP.[53] Screening tools may help the PT in identifying IIB in LBP patients. Some tools may also identify the presence of a psychopathology. It is outside our scope of practice to specifically diagnose and deal with psychopathology; attempts at an appropriate referral therefore should be made. The results of screening tools may stengthen the position of the PT medicolegally and when securing an appropriate referral. Timely referral to the may curb the costs associated with the management of chronic LBP. Feuerstein and Beattie[54] offered clinically useful guidelines for referral to an appropriate mental health provider (Table 10).

Degenerative disease

We discussed how changes in the ZJ, SIJ, and ligaments were hard to clearly attribute to either aging or degeneration. The IVD is the exception: disk dessication and narrowing is not a normal age-related change.[1,8] Bogduk[1] forwarded a theory regarding degeneration specifically of the IVD and indirectly the whole lumbar motion segment. Fundamental to this theory is compressive failure of the endplate. In vitro, excessive axial compression causes endplate failure.[55-57] Ultimate endplate failure force ranges from 10,000 to 3,000 N.[1] The back muscles can exert a longitudinal force of approximately 4,000 N. Extreme exertion might cause endplate fracture in a person with vertebral bodies insufficiently conditioned to withstand compression or weakened by any of the systemic diseases affecting mechanical strength of bone. Repetitive loading decreases the forces required for endplate failure to 30 to 80% of ultimate failure strength. Endplates fail with as little as 100 repetitions.[1] Loads and repetitions of this magnitude appear feasible in many occupational situations. An endplate fracture may cause LBP; it can also go unnoticed.[1] With an endplate fracture blood from the vertebral body has access to the NP. The NP contains matrix metalloproteinases (MMPs), capable of matrix and collagen degradation.[1] Substances entering from the vertebral bodies (e.g. plasmin contained in the blood) activate these enzymes initiating degradation.[1] Proteoglycan and collagen synthesis depend on a narrow pH range[58]. An endplate fracture lowers the pH in favor of degradation.[1] Research has failed to show a proposed cellular immune response to nuclear proteins in IVD degradation. Proteolysis and deaggregation causes progressive loss of water binding capacity. The resultant deterioration of nuclear function increases the mechanical demands on the AF predisposing it to mechanical failure. This whole process is called *internal disk disruption*, a condition in which the internal surface of the disk is disrupted, but the external surface remains essentially normal.[1] Internal disk disruption may cause LBP through chemical irritation: enzymes and products of matrix and collagen degradation may depolarize nerve endings in the innervated outer one-third of the AF. An inflammation in an attempt to repair the damage may produce inflammatory mediators which may also cause chemonociception. Mechanical failure of the inner anular fibers due to NP incompetence will increase the mechanical demands on the fibers in the innervated intact outer AF resulting in depolarization of mechanonociceptors. The pain of internal disk disruption is likely constant due to its chemical component, yet aggravated by movement as a result of its mechanical component. As the external aspect of the disk remains normal, neurological symptoms are unlikely.[1]

Compressive loads acting upon a disk with an incompetent NP cause increased radial bulging of the AF and loss of disk height.[59] The loss of disk height increases compressive loads on the ZJs[60] predisposing them to degenerative changes.[1] Reactive osteophytosis may decrease the diameters of the intervertebral foramen (IVF) and central spinal canal. Loss of motion segment height decreases the pretension in the flaval ligaments. In combination with the age-related changes of hypertrophy, ossification, and decreased elastic fiber content this may cause inbulging of the flaval ligament.[29,30] Loss of motion segment height also causes ZJ telescoping and a relative descent of the superior pedicle narrowing the IVF.[61] Degeneration introduces laxity in the restraining structures of mobile lumbar segments: antero- or retrolisthesis may occur.[61] Weiler et al[62] confirmed this increased linear motion as a result of degeneration; they found a significant increase in linear translation of the L4-L5 motion segment during flexion and extension in patients with degenerative disk disease as compared to asymptomatic controls. This increased linear motion does not translate into increased sagittal plane mobility. In fact, in 412 subjects Burton et al[63] found that reduced disk height was a significantly decreased total sagittal plane ROM and decreased extension. Together all these degenerative changes can cause stenosis or narrowing in the central spinal canal or IVF and predispose the nerve roots to compression radiculopathy.

A disk weakened by internal disruption may progress to disk herniation. This may be the result of progression of the degradation process along radial fissures into the outer regions of the anulus.[1] The weakened AF is also more susceptible to mechanical failure. In vitro, only aphysiologic ranges of flexion and rotation caused anular failure[12]. Zygapohysial joint degeneration caused by disk degeneration may cause thinning of the articular cartilage allowing for excessive rotational stresses to the AF.[1,64] Gapping of the ipsilateral ZJ observed in unstable, degenerated motion segments may cause shear in the disk.[65] Degenerative spondylolisthesis may further add to shear forces in the disk. The combination of excessive mechanical forces and a weakened AF may cause mechanical failure and lead to herniation. Weber[66] subdivided disk herniations into 3 categories: protruded, extruded, and sequestered (Figure 1). He visualized a protrusion as a bulging disk with the anular wall still intact. An extrusion is a disk in which the NP has penetrated the outer anular fibers. With a sequestration, 1 or more fragments of the NP have broken free from the herniated mass and have escaped into the spinal canal. Disk herniation usually occurs in a posterolateral direction. We discussed the anatomical reasons for this predisposition in monograph 11.2.3. The absence of the PLL lateral to the midline also plays a role.[61] Posterolateral disk herniation may cause ischaemic radiculopathy, especially in a degenerated segment with already decreased IVF diameters. Posterior disk herniations may cause LBP as a result of irritation of the innervated structures. The polysegmental innervation of the dura mater may result in widespread referral. The PLL and the posterior anular fibers are other sources of nociception. A rapidly developing massive central disk herniation may compress the cauda equina. Disk herniations are frequently asymptomatic.

Disk herniations are most prevalent between the ages of 30 and 50 to 55.[61,67] It may appear that disk herniations would increase with age beyond 55 years in view of the progressive decrease in tensile strength and increase in stiffness of the AF occurring with aging.[1,4,6] This would seem to set up the disk for anular mechanical failure and subsequent herniation. However, increased collagen content and increased collagen-PG interaction makes the disk less fluid in the elderly.[1] Brinckmann[56] showed that even after anular disruption, compression would not produce a herniation of nuclear material. Herniation can only occur in case of a sufficiently fluid NP in combination with a weakened AF. Disk degradation produces both these prerequisites.

EXAMINATION

The Guide to Physical Therapist Practice[68] defined examination as the process of obtaining a history, performing a systems review, and selecting and administering tests to gather data about the patient; the initial examination is a comprehensive screening and specific testing process that leads to a diagnostic classification and/or consultation with or referral to another provider. Table 11 describes a possible format for lumbopelvic region examination.

Diagnosis within a chosen classification system occurs by way of pattern recognition. A good classification system describes clinical patterns of signs and symptoms. Historical information and physical examination tests should allow for inclusion or exclusion of a patient from a specific diagnostic category. Therefore, the findings from history items and tests need to be suficiently reliable and valid. Reliability is measured as repeatability between measurements performed by the same examiner (*intra-rater reliability*) or between measurements by different examiners (*inter-rater reliability*). Research frequently reports reliability as percent agreement. This fails to compensate for agreement on the basis of chance alone. Using different versions of the kappa statistic overcomes the problem of chance agreement.[69] Test validity is determined by how well it correctly classifies individuals with or without a particular disease.[70] A common type of validity is determined by comparing the results of a test to those of a so-called “gold standard” test; this test is accepted as being close to 100% valid.[71] Table 12 contains definitions of the 4 concepts used to describe test validity.[70] Highly sensitive tests are by definition usually positive in the presence of a disease. Clinically, a highly sensitive test is most useful when it is negative.[70] Highly specific tests are rarely positive in the absence of disease and are most helpful to the clinician when they are positive, ruling out a suspected diagnosis.[70] In this section we will review research on reliability and validity of history items and tests for the lumbopelvic region. We also describe tests somewhat unique to the examination of this region.

History

Monograph 11.2.2. reviews the general format of history taking. The section on diseases affecting the lumbopelvic region should be helpful in constructing a list of history items meant to screen for

nonmusculoskeletal causes of lumbopelvic region complaints. Research on validity of history findings in patients with LBP generally uses a classification system based on the traditional medical model. The medical model uses a structure-based classification system which assumes a direct correlation between underlying pathology and the signs and symptoms.[72] This research uses diagnostic labels of disk disease, and facet joint, SIJ, cauda equina, and stenotic syndrome.

Disk disease

Schwarzer et al[73] attempted to establish validity for the history items listed in Table 13 for diagnosis of an internal disk disruption by correlating these items to the diagnosis established by computed tomography (CT) scan. They found no significant correlation between the history findings and an established CT-diagnosis in 36 of 92 patients with chronic LBP.

Disk herniation can cause radiculopathy. Andersson and Deyo[74] described the typical presentation of a patient with diskogenic radiculopathy (Table 14). Deyo et al[67] reported a sensitivity for sciatic distribution pain in the diagnosis of a lumbar disk herniation of 95%; they calculated the likelihood of a disk herniation without sciatic pain as 0.1%. Van den Hoogen et al[75] reported a sensitivity of sciatica for the diagnosis of disk herniation of 79 to 91% and specificity of 14%. Roach et al[70] reported on the validity of history items in symptomatic subjects with radiographic evidence of rupture or displacement of disk material: sensitivity of radiating leg pain in patients with disk disease was 81%. The authors suggested using this high sensitivity to exclude the diagnosis of disk herniation in case of the absence of leg pain.[70] Van den Hoogen et al[75] reported 30 to 74% sensitivity and 18 to 58% specificity for paresthesia; specificity of sciatic pain as a result of coughing was 74%.

Zygapophyseal joint syndrome

Schwarzer et al[76] attempted to establish validity for the history items in Table 13: they found no significant correlation between any history item and a diagnosis of ZJ syndrome established by a double-block intra-articular injection. The only definitive observation was that no patient with central LBP responded to intra-articular infiltration implying that ZJs do not refer pain exclusively to the central lumbar spine. Opinions on the area of pain referral differ. Kuslich et al[77] found sharp local LBP with stimulation of the periarticular tissue; stimulation of the joint capsule caused back and rarely buttock pain, but never leg pain. Mooney and Robertson[78] infiltrated the ZJs and demonstrated not only that they can cause LBP, but also that pain from the ZJs may refer into the leg below the knee. Whether this pain was only of ZJ origin or increased due to segmental facilitation as a result of nociceptive input from a painful structure elsewhere in the segment is unclear from their study. Oesch[79] reported multisegmental pain originating from the ZJ, which never referred below the knee. Oesch[79] suggested that excessive ZJ infiltration may cause leakage with irritation of foraminal and epidural structures resulting in LBP below the knee.

Sacroiliac joint syndrome

Schwarzer et al[80] found no correlation between any history item in Table 13 and SIJ syndrome confirmed with intra-articular infiltration. They reported that groin pain was the only clinical finding distinguishing SIJ pain from pain not of SIJ origin. Fortin et al[81] performed SIJ distention arthrography on 10 asymptomatic subjects and found an area approximately 3 cm wide and 10 cm long just inferior to the PSIS that was painful in all subjects. Using patient description of pain in the aforementioned area as a diagnostic criterium for the presence of SIJ-related pain, Fortin et al[82] found interrater reliability of 96% in identifying SIJ patients. All patients thus selected had a provocation-positive SIJ infiltration. Fortin et al[82] did not report kappa values. Fortin et al[83] noted that extravasation of inflammatory mediators through a capsular recess or tear from a dysfunctional SIJ to adjacent neural structures may cause radicular symptoms.

Cauda equina syndrome

A central lumbar disk lesion may compress the spinal cord or, more commonly, the cauda equina. Kostuik et al[84] described 31 patients with cauda equina syndrome due to herniation. All patients reported urinary retention, motor weakness and decreased sensation in the legs, LBP, and bilateral or unilateral sciatic distribution pain. Saddle area hypesthesia was present in 17 patients; 8 noted sexual dysfunction (decreased sensation during intercourse, decreased penile sensation, and impotence). Kostuik et al[84] warned that central lesions, especially at L5-S1, may pose a diagnostic challenge, as they affect only the lower sacral roots and cause no motor or reflex changes in the legs. Deyo et al[67] noted a sensitivity of 90% and a specificity of 95% for urinary retention. They reported sensitivity higher than 80% for unilateral or bilateral sciatica, and sensory and motor deficits. They noted 75% sensitivity for saddle anesthesia.[67]

Stenotic syndrome

Deyo et al[67] reported 60% sensitivity for neurogenic claudication, 85% for leg pain, and 60% for neurologic abnormalities. Roach et al[70] calculated a 63% sensitivity for neurogenic claudication in patients with radiographically diagnosed spinal stenosis; sensitivity in patients with herniation and stenosis was 47%. Sensitivity of leg pain was 94% in patients with disc disease and spinal stenosis.[70]

Active range of motion tests

Schwarzer et al[73,76,80] tried to establish the validity of the physical examination items in Table 15 for the diagnosis of internal disk disruption, ZJ syndrome, and SIJ syndrome: they found no significant correlation between any of the physical examination items and the diagnosis established with the "gold standard" tests.

Neurologic examination

A neurologic examination consists of 3 parts. *Upper motor neuron screening* is indicated when cord compression is suspected. An upper lumbar central herniation may result in spinal cord compression. Pathological reflexes, hyperreflexia on deep tendon reflex (DTR) testing, clonus, and velocity-dependent hypertonicity are indicative of cord compression.[85] Magee[86] described lower extremity pathological reflexes, e.g. Babinski, Oppenheim, and Chaddock signs. A central herniation in the lower lumbar spine can cause a compression of the cauda equina. Cauda equina compression should not result in upper motor neuron signs. Kostuik et al[84] reported decreased anal sphincter tone as a neurologic sequela of cauda equina syndrome.

In monograph 11.2.3. we discussed the etiology of nerve root compression. Sufficient ischaemia may result in decreased conductive function of the nervous system; *neuroconductive testing* may reveal segmentally related paresthesia, hypesthesia, weakness, and reduced DTRs.[85] Table 16 summarizes data on the validity of neuroconductive tests for the diagnosis of disk herniation.

The nervous system adapts to trunk and limb movement by changes in tension and movement relative to the adjacent musculoskeletal structures, the mechanical interface.[87] *Neurodynamic tests* examine the movement and tensile abilities of the nervous system. Butler[87] described the technique and rationale for neurodynamic tests in the lumbopelvic region. Deyo et al[67] noted a sensitivity of 80% and a specificity of 40% for the straight leg raise (SLR) in the diagnosis of low lumbar disk herniation. Van den Hoogen et al[75] reported a sensitivity of 88 to 100% and a specificity of 11 to 44% for the SLR. Using symptom reproduction below 40^0 as a criteria for a positive SLR decreased sensitivity to 72% and increased specificity to 66%.[74] Reproduction of leg pain as the criterion for a positive SLR resulted in sensitivity of 76 to 97% and specificity of 11 to 45%; reproduction of leg or back pain increased sensitivity to 91 to 95%, but decreased specificity to 14 to 21%.[74] The crossed SLR or well leg raise reproducing pain in the affected other leg has lower sensitivity (23 to 44%), but higher specificity (86 to 100%).[67,74,75] Van den Hoogen et al[75] suggested that combining the SLR and the crossed SLR will lead to a more accurate diagnosis. The more limited the SLR, the more specific the test becomes and the greater the herniation found at surgery.[67] Meadows[85] reported on a retrospective study where 80% of patients with a positive SLR had a herniation. Only 63% had a herniation if the SLR exceeded 60^0 and only 7% of patients with a SLR $<30^0$ did not have a disk herniation.[85] The SLR test is most appropriate for testing the L5 and S1 nerve roots. Irritation of the higher lumbar roots is tested by the prone knee bend (PKB) or femoral nerve stretch test; reliability and validity of the PKB are unknown.[67]

Repeated movement tests

McKenzie[88] developed a diagnostic classification based on the assumption that sustained or repeated movements may affect nuclear position resulting in centralization or peripheralization of complaints. McKenzie[88] defined the centralization phenomenon as *the situation in which pain arising from the spine and felt laterally to the midline or distally, is reduced and transferred to a more central or near midline position when certain movements are performed*. Peripheralization is the opposite of this phenomenon.[88] As long as the anulus and the hydrostatic mechanism of the disk are intact, an off-set load on the disk in a lesion-specific direction of spinal movement may apply a reductive force on a displaced nuclear fragment, directing it towards a more central location thereby reducing symptom-generating stress on a neural or other nociceptive structure.[89] In a retrospective study of 87 patients with leg and LBP, Donelson et al[90] found that all patients with excellent outcomes of McKenzie-based treatments showed centralization during the initial evaluation. They found a significant correlation between the presence of centralization and a good or excellent outcome and, conversely, between the absence of centralization and an unsatisfactory outcome. The 4 patients who needed surgical intervention were non-centralizers; 3 actually peripheralized. Surgery

showed 3 extrusions. In a later prospective study of 63 patients with LBP, varying degrees of leg pain and altered sensation, but without neurologic deficits, Donelson et al[89] found a significant correlation between positive discograms and peripheralization or centralization; negative discograms correlated with no change on repeated testing. The incidence of a competent anulus was significantly greater in the centralizing patients with positive discograms than in their peripheralizing counterparts.[89]

Intervertebral position and motion tests

A variety of clinicians use position and motion tests to establish a diagnosis and subsequent treatment for patients with complaints related to the lumbar spine.[34,85,91-94] Lumbar spine positional palpation tests use the position of transverse and spinous processes in a neutral, flexed, and extended position to infer segmental position and possibly movement anomalies.[92] Positional changes between spinal positions do not indicate whether the underlying dysfunction is a hyper- or hypomobility.[92] Congenital or acquired anomalies of the orientation and size of the bony landmarks used obviously have the potential to make this type of tests highly invalid. Reliability and validity of positional palpation depend on the ability of practitioners to reliably palpate bony landmarks. Downey et al[95] studied the interrater reliability of palpation of the L1 to L5 spinous processes by 6 manipulative PTs on 60 patients with LBP; a kappa of 0.92 indicated almost perfect agreement. However, positional palpation involves more than just correctly identifying segmental levels. Keating et al[96] studied interrater reliability of positional palpation of 3 chiropractors evaluating 21 patients with LBP and 25 asymptomatic subjects: a kappa value of smaller or equal to 0.30 indicated poor reliability.

Motion palpation involves palpation of vertebral bony landmarks during active trunk motion.[94] Keating et al[96] reported mean kappa values between 0.00 and 0.25 indicating poor interrater reliability for motion palpation tests.

Passive intervertebral motion (PIVM) tests come in 3 categories: passive physiological intervertebral motion (PPIVM), passive accessory intervertebral motion (PAIVM), and segmental stability tests. Meadows[97] defined PPIVM as the assessment technique whereby one vertebra is moved in physiological ranges on another; PAIVM is the passive assessment of an intervertebral joint through its glides. Meadows[85] provided descriptions of these tests. Stability tests[85] attempt to examine segmental translatory mobility. Tables 17 to 19 summarize research on the reliability and validity of PPIVM and PAIVM testing. Research shows at best moderate intrarater reliability; interrater reliability is generally poor. Reliability improves when a positive response includes both perceived changes in ROM and patient report of pain rather than just decreased mobility.[98,106] Technical aspects of intervertebral motion testing affect the findings. Maher and Adams[112] found that PTs were able to reliably discrimate levels of stiffness on simulated posteroanterior (P/A) accessory motion testing in increments of 5%. However, using a thumb grip consistently resulted in higher stiffness assessments than using a pisiform grip. Visual occlusion did not decrease the ability to discriminate between stiffness on P/A, but also resulted in perception of greater stifness than with vision intact.[113] Viner and Lee[114] reported intertherapist variations in directions of force when they had PTs perform lumbar P/A tests on asymptomatic subjects; they suggested this may cause significant variations in reported pain and perceived motion. Maher et al[115] mechanically assessed P/A stiffness in prone on asymptomatic subjects; stiffness was lower on a padded than on an unpadded table. Despite low to poor reliability, several studies have shown some validity to PIVM tests. Patients with LBP are stiffer on P/A than controls.[109,111] A clinically detectable decrease in stiffness of up to 37% occurred simultaneously with a decrease in symptoms: though not necessarily a causal relationship, PAIVM tests and treatment might have a role in monitoring or influencing progress.[110] A combination of PPIVM and PAIVM tests correctly identified dysfunctional levels diagnosed with intra-articular infiltration.[104] Reliability of PIVM tests would likely benefit from standardization of procedures (e.g. surface, patient position, visual stimuli, standardized grip) and a clear definition of what constitutes a positive finding (e.g. range, endfeel, reproduction of symptoms). Uniform training may increase the ability to discriminate levels of stiffness: Latimer et al[116] showed significant reduction in the absolute error of simulated P/A examination in PT students after training with immediate quantitative feedback. These tests seem to be able to distinguish patients from controls and determine the location of dysfunction. Extrapolation of specific treatment parameters based on PIVM tests is not supported by research, yet is commonplace among manual medicine practitioners.

Cassidy and Potter[94] suggested using P/A forces in sitting to detect the excessive translation pathognomonic for segmental instability. Meadows[85] described 3 tests for detecting segemntal instability. *Anterior shear testing* (Figure 2) is performed with the patient sidelying. The PT stabilizes the superior

vertebra of the segment to be tested with both hands and applies a posterior shear force with the hips to the inferior vertebra by way of axial pressure through the femurs. The L5-S1 segment is tested at 45^0 of hip flexion; for the other segments the hips are at 80^0. The inferior vertebra is palpated with one finger for excessive motion. For *posterior shear testing* (Figure 3) the patients sits at the head of the table and puts the forearms up against the chest of the PT standing in front of the patient. The PT reaches around the patient to stabilize the inferior vertebra of the segment to be tested, while palpating the superior vertebra. Scapular protraction by the patient may result in appreciable posterior shear of the superior vertebra. For *segmental torsion testing* (Figure 4) the patient is positioned in opposite side sidelying in neutral flexion and extension. Axial rotation rotates all segments without actually locking them. Pressing the superior spinous process towards and lifting the inferior spinous process away from the bed causes pure axial rotation. Depending on the amount of pre-test rotation there should be no or minimal motion appreciable. Tilscher et al[117] tested patients with excessive mobility on impulse spring testing in prone. They found no excessive translation on radiographic examination with a sustained P/A in prone in any of the 9 patients tested. Radiographically confirmed excessive translation did occur in 11 of 22 patients with a sustained P/A in prone with hip flexion over the edge of the bed. The authors hypothesized that ZJ locking in a normal prone position makes prone P/A tests less sensitive for the diagnosis of instability. The similar test position may confer some validity to the anterior shear test described above.

Sacroiliac tests

Positive provocation test findings may include the SIJ as a source of symptoms. Potter and Rothstein[118] studied interrater reliability of SIJ tests on 17 patients with unilateral buttock pain: only the supine iliac gapping (94%) and the sidelying iliac compression test (76%) showed acceptable percentage agreement scores. Position or motion palpation tests had unacceptable reliability. Laslett and Williams[69] found substantial interrater reliability for the same gapping and compression tests, but also for the thigh thrust and Gaenslen tests. They reported the sacral thrust and the sacral cranial shear test as potentially reliable. In a systematic review of the literature on reliability of SIJ tests, Van der Wurff et al[119] as a rule found greater intra- than interrater reliability. Other authors did not support reliability for the gapping, compression, and thigh thrust test.[119] Methodological errors may have led Laslett and Williams[69] to erroneously accept the Gaenslen test as reliable.[119] In a systematic literature review on the validity of SIJ tests,Van der Wurff et al[71] reported acceptabe validity only for using the thigh thrust test for diagnosis of SIJ syndrome in pregnant women.

Positional palpation tests of the SIJ rely on palpation of asymmetric sacropelvic landmarks to determine the presence of a dysfunction. O'Haire and Gibbons[120] demonstrated poor reliability of palpation of the PSIS, sacral sulcus (SS), and inferior lateral angle (ILA) of the sacrum in prone asymptomatic subjects. Intrarater reliability for palpation of PSIS, SS, and ILA yielded mean kappa values of 0.33, 0.24, and 0.21. Kappa values for interrater reliability did not exceed 0.08. Validity of positional tests is discredited by the fact that osseous asymmetry may be perceived as proof of movement dysfunction.[120] Tullberg et al[121] compared positional tests findings to roentgen stereophotogrammetric analysis (RSA) of the spatial relationship between sacrum and innominate. Positional tests demonstrated altered SIJ relationship prior to manipulation; findings were normal after treatment. However, RSA showed an unaltered positional relationship after treatment. The authors concluded that positional tests do not provide a valid description of SIJ alignment.

Motion palpation tests attempt to provide information on the type and location of sacroiliac dysfunction. Richter and Lawall[98] reported a kappa of > 0.80 for the intrarater reliability of the standing flexion and Gillet motion palpation tests: interrater reliability was > 0.50 and > 0.65, respectively. Herzog et al[122] reported significant interrater reliability for determining the presence of decreased SIJ mobility with the Gillet test, but not for determining the side of hypomobility. Intrarater reliability was greater in examiners with less experience than in the high-experience group. Van der Wurff et al[119] reported unacceptable reliability for motion palpation tests. They questioned reliability of the Gillet test stating that statistical tests used were not appropriate.[119]

In the clinic, it is unlikely that a PT will base the entire assessment on the result of just one test. Instead, the PT will likely depend on a battery of tests to rule out or confirm a suspected diagnosis.[123] Cibulka et al[123] used a criteria of at least 3 out of 4 tests positive to determine the presence of SIJ dysfunction. They used the standing flexion, PKB, and supine-to-long-sitting test, and palpation of the PSIS in sitting. They reported a

kappa value of 0.88, establishing acceptable reliability for this battery of tests for diagnosis of SIJ dysfunction.

Passive physiological motion (PPM) tests are assessment techniques whereby a bone is moved in physiological ranges on another.[97] Van der El[34] described an *anterior innominate rotation test* in which the PT places the heel of one hand on the apex of the sacrum of the prone patient, while stabilizing the innominate closest to the PT with the other hand. The stabilizing hand also palpates for motion medial to the PSIS. A ventromedial force against the apex of the sacrum results in sacral counternutation, which equals innominate anterior rotation. In the *posterior innominate rotation test*[34] the PT reaches over the prone patient to the opposite ASIS and palpates with the other hand over the SIJ line, again at the level of the PSIS, while gently stabilizing. Pulling the innominate in a dorsal, medial, and caudal direction produces a posterior innominate rotation.

Passive accessory motion (PAM) tests refer to the passive assessment of a joint by way of its arthrokinematic glides.[97] Lee[91] described sacroiliac PAM tests. I believe these tests are theoretical constructs: the multiplanar orientation of the SIJ will not allow for this type of gliding motion to be tested, unless there is sufficient joint separation due to capsuloligamentous laxity. Therefore, we can use PAM tests to determine joint stability. A *supine thigh thrust test* can be used to asses anteroposterior SIJ translation.[92] The patient is supine with one hip flexed to 90^0 and slightly adducted. Standing on the side to be tested, the PT exerts axial pressure through the femur while palpating the posterior SIJ joint line. A *craniocaudal* and *caudocranial translation test* test for superior and inferior translation of the innominate on the sacrum in a prone patient. In the caudocranial test the PT attempts to shear the innominate upwards by pressure through the ischial tuberosity, while stabilizing the base of the sacrum with the other hand.[92] In the craniocaudal test the PT pulls down the innominate by long axis traction to the leg, while stabilizing through the apex of the sacrum. A soft endfeel[91] and the presence of more than the slightest of motion may indicate capsuloligamentous laxity.

Reliability of some provocation tests seems acceptable; acceptable validity has only been for the thigh thrust test in diagnosis of SIJ syndrome in the subpopulation of pregnant women with LBP. Provocation tests only implicate the SIJ as a source of symptoms, but give no indication for treatment. Positional palpation tests are not reliable, nor valid; therefore, motion palpation tests which depend on palpation of bony landmarks are likely to also not be reliable or valid. No data were provided on the PPM and PAM tests described.

Pubic symphysis tests

Van der El[34] described palpation of the inferior aspect of the symphysis in standing to detect asymmetry. He also described a *symphysial provocation test*, which consists of a unilateral posterior pressure on the pubic bone close to the joint in a supine patient. Lee[91] described a *superoinferior translation test*: 1 hand applies a slow and steady inferosuperior force to the inferior aspect of the superior pubic ramus, while the other hand stabilizes the superior aspect of the opposite superior ramus. Range, endfeel, and reproduction may give information regarding symphysial instability.

Sacrococcygeal and intercoccygeal tests

If sacrococcygeal dysfunction is suspected, the only physical examination test we can use to detect this, is positional palpation and PPM of the coccyx by way of a rectal exam.[34] Strong suspicion indeed of contributory or causal sacrococcygeal dysfunction would seem necessary.

Muscle function tests

Muscle length and strength tests may give us information on contributory dysfunctions in the lower extremity. It may also provide diagnostic clues: Lee[91] stated that SIJ dysfunction may cause gluteal muscle inhibition. Manual muscle tests are used in the extremities to provide us with information on the musculotendinous structures.[86] In monograph 11.2.3. we reviewed the compressive forces exerted by the lumbopelvic muscles. The already tenuous assumption that we can isolate the musculotendinous structures in the extremities by manual muscle tests in a resting position of the joint can clearly not be used in the spine. In my opinion, isometric manual muscle tests of the trunk have no value in examination of the lumbopelvic region. However, lumbopelvic muscular stability tests may give an indication of the contribution of the neuromuscular system to stability in the lumbopelvic region.

Richardson et al[124] described a test of transversus abdominis function. The sought-after muscle action is drawing in of the abdomen. After a number of practice sessions in quadruped position (Figure 5), the patient is prone with the abdomen over a pressure biofeedback unit resembling a blood pressure cuff (Figure 6). The PT inflates the pressure pad to a pressure sufficient to detect changes in position without pressing into the abdominal contents. The PT instructs the patient to draw in the abdominal wall without moving the spine or pelvis and to hold for 10 seconds while breathing normally. A successful performance of the test reduces the pressure reading on the pressure dial by 6 to 10 mm Hg. Substitution by pelvic or lumbar movement or failure to decrease the pressure is an indication of decreased motor control. The PT can test endurance by repeating the test up to 10 times. Richardson et al[124] also suggested testing the segmental lumbar multifidus in a prone position (Figure 7). First, the PT palpates the lumbar multifidi adjacent to the spinous processes feeling for a segmental loss of consistency. This may provide information on possible segmental inhibition. The patient then attempts to gently "swell out" muscles under the PT's fingers without moving the spine or pelvis and hold the contaction while breathing normally. The PT simultaneously provides proprioceptive stimulation and feels for contraction. No contraction or a rapid and superficial development of tension is unsatisfactory. The PT observes for substitutions by the thoracic erector spinae or lumbopelvic motion. A good test result is the ability to hold a tonic contraction bilaterally at each segment. Without reporting statistical data, Richardson et al[124] noted good agreement between subjects with a poor ability to decrease pressure on the clinical transversus abdominis test and a delay of contraction on EMG, and between subjects able to decrease the pressure with early activation of the transversus abdominis. They concluded that the clinical test is a good estimation for the quality of motor control of the transversus abdominis. They also reported a study on (sub)acute LBP patients in which manual identification of the inhibited multifidus corresponded with findings on real-time ultrasound imaging tests in 24 of 26 subjects.

Lee[91] described 2 tests to diagnose insufficient force closure. In the *supine active straight leg raise test* (Figure 8) the supine patient lift 1 leg. The PT observes for compensatory motion in the trunk and reproduction of symptoms. The patient then repeats the test with the PT manually compressing the innominates or manually resisting an isometric trunk flexion with rotation towards the leg being tested. Decreased ability to raise the leg indicates poor force closure; improvement with the isometric test may indicate a good prognosis for rehabilitation through exercise.[91] In the *prone straight leg raise test* (Figure 9) the prone patient lifts the straight leg by extending at the hip. The PT can increase force closure by manual compression of the innominates or by resisting isometric extension of the opposite shoulder. A reduction of symptoms with an improvement in coordination in the isometric portion of the test again indicates rehabilitation potential with exercise.[91] Lee[91] erroneously called the manual innominate compression portion of both tests a test of form closure. However, per definition the application of a lateral force to increase SIJ stability is a form of force closure.[125] A third test is based on the findings by Vleeming et al[126] of decreased SIJ motion with the application of a SIJ-belt. If the patient can perform activities, which were painful without the stabilizing effect of a belt, with a decrease in symptoms with a belt, this screening test is positive for decreased force closure.

Vascular tests

Clinicians use vascular tests for the differential diagnosis between neurogenic and vascular claudication. Magee[86] described the *stoop test* and the *bicycle test of Van Gelderen*. A positive stoop test reproduces claudication symptoms with brisk walking within 50 m and relieves symptoms by standing or sitting trunk flexion. The bicycle test is a 2-part test. The patient cycles a stationary bike with erect trunk posture. Reproduction of symptoms is a positive first portion. The second portion is positive, if continued cycling, now in a flexed posture, results in subsiding pain and paraesthesiae. Positive tests imply the presence of neurogenic claudication, negative tests with reproduction of leg symptoms implicate vascular structures.[86] Fritz et al[127] reported a 2-stage treadmill test to differentially diagnose lower extremity degenerative joint disease and the 2 types of claudication. Patients walk on a treadmill at both a 15^0 incline and in a level position without using handrails at a comfortable speed. Rationale is that neurogenic claudication patients will benefit from the flexion posture caused by the incline, whereas other patients will not. The PT records walking time until onset of symptoms and maximum time. Patients walk for a maximum of 15 minutes. The PT records time required for symptoms to return to baseline. After a 15 minute rest patients perform the second portion of the test.

Palpation tests

In monograph 11.2.3. we discussed the monosegmental innervation of many of the paraspinal structures. By way of somatosomatic and somatosympathetic reflex circuits segmental nociceptive input may cause segmental muscle hypertonicity, segmental hyperesthesia, and segmental tissue texture changes as a result of sympathetically mediated vasoconstriction.[34] Clinicians use these changes to determine the segmental level of dysfunction.[34] Leboeuf et al[99] reported 70 to 90% interrater agreement on pain response to spinous process and interspinous palpation, and spinous percussion. Keating et al[96] reported moderate to good interrater reliability for pain response to osseous and soft tissue palpation. Palpation of paraspinal muscle tension not dependent on patient resport of pain only yielded mean kappa values between 0.07 and 0.21.[96]

DIAGNOSIS

Diagnosis is classification of patients based on some defining characteristics. Diagnosis requires a classification system. The Guide to Physical Therapist Practice[68] mentioned 3 criteria for a classification system to be useful for PT:

- The system must be consistent with boundaries placed on the profession by law or society.
- The tests necessary for confirming the diagnosis must be within the legal purview of PT.
- The label used to categorize a condition must direct the selection of interventions towards those interventions that are part of the PT scope of practice.

Classification systems are a type of clinimetric index. Clinimetric indices are rating scales and other expressions used to measure symptoms, physical signs, and other phenomena in clinical medicine. There are 3 types of clinimetric indices relevant to classification systems used for patients with LBP.[128] *Status indices* are classification systems used to define patient problems. The most commonly clinically used example of a status index is the International Classification of Diseases (ICD). The ICD is a taxonomy of diagnostic labels for the purpose of standardizing nomenclature of diagnoses for statistical and administrative reasons. PTs are all familiar with ICD-9 codes for insurance reimbursement for our services. Because the ICD manual does not describe the procedures used to apply the diagnostic labels, reliability of assigning ICD-9 codes is low.[128] The ICD-9 contains 66 codes for LBP.[128] The traditional medical, structure-based model is also a status index: it assumes a direct correlation between underlying pathology and signs and symptoms.[72] However, the medical model is unable to provide up to 85% of patients with a specific diagnosis due to the weak association between symptoms, pathological changes, and results from imaging tests.[67,75] A *prognostic index* is a classification system that allows us to to predict the patient's future status; this type of system is designed to aid the clinician in making predictions regarding the chance of a poor outcome. Waddell et al[51,52] described a prognostic index of use to PT. The third type of classification system is the *clinical guideline index*. This type of index is designed specifically to provide instructions regarding treatment. The system developed by McKenzie[88] (Table 20) and the one developed at the University of Pittsburgh[72,129,130] (Table 21) are examples of clinical guideline indices used in PT. The classification developed by the Quebec Task Force on Spinal Disorders is a *mixed index*, designed to help make clinical decisions, establish a prognosis, as well as evaluate the quality of care for patients with LBP.[128]

The goals of PT examination are to screen for undiagnosed medical disease and to establish a PT diagnosis. Physician diagnosis based on the traditional medical model is geared towards making decisision regarding surgical potential, the possibility of conservative options, and the ordering of ancillary tests.[129] Medical classification does not meet the criteria for a PT classification system: most diagnostic tests and interventions are outside the scope of PT practice. Medical diagnosis is also of little value to the therapist for selecting specific interventions.[129] To establish a diagnosis the PT needs to decide on a classification system. Prognostic indices have value for establishing relative and absolute contra-indications, but clinical guideline indices are more useful, as they allow for decisions regarding the choice of appropriate interventions.

Meadows[97] described a system of biomechanical examination and diagnosis, based on our knowledge of anatomy and pathology and the extrapolated or proven biomechanics arising from the anatomy and pathoanatomy. This type of classification system is an example of a clinical guideline index, developed based on clinical experience and judgment.[128] Central to biomechanical diagnosis is the movement dysfunction. Paris and Loubert[131] defined a movement dysfunction as *a state of altered mechanics, either an increase or a decrease from the expected normal, or the presence of an aberrant motion*. The use of tissue texture abnormality palpation, positional palpation, motion palpation, PIVM, and PPM tests may seem central to the

biomechanical examination and diagnosis. We discussed the questionable reliability and validity of these tests. However, the PT uses diagnostic clues from all aspects of the history and physical examination. Combining information from multiple tests and measures may improve reliability and validity.[123] The clinical reasoning used by the experienced clinician to arrive at a specific biomechanical diagnosis would be an interesting field of study. However, the biomechanical classification system described here remains hypothetical: speculations regarding patient presentation, pathomechanics, and treatments based on this system so far are only discussions not to be taken as definitive.[97]

Lumbar movement dysfunctions

These include diskogenic and ZJ dysfunctions, instability dysfunctions, neuromeningeal movement dysfunctions, and stenotic syndromes.

Diskogenic movement dysfunction

There are 3 different, clinically relevant diskogenic movement dysfunctions:

- A contained diskogenic dysfunction without nerve root compression.
- A contained diskogenic dysfunction with nerve root compression.
- An uncontained diskogenic dysfunction.

We discussed the progression of disk degradation from endplate failure, to internal disk disruption, to protrusion, extrusion, or even sequestration. Nuclear pressure, fundamental to normal segmental mechanical behavior, is reduced with disk degradation. Disk degradation may produce a specific movement dysfunction with a unique combination of signs and symptoms. Anatomical and mechanical features predispose the IVD to mechanical failure in a posterior or posterolateral direction.

Based on the information in this and the previous monograph we can hypothesize that a patient with a diskogenic dysfunction will report LBP and possibly leg pain. The leg pain is *somatic referred pain* due to nociceptive stimulation of the anulus, PLL, dura mater, or nerve root sleeve. Somatic referred pain is poorly delineated due to the multisegmental (and in some structures bilateral) innervation of the structures responsible for the referred pain. A posterolateral herniation may cause ischaemia of the nerve roots, dorsal root ganglion (DRG), or spinal nerve: this can cause *radicular pain*. Radicular pain is usually more delineated than somatic pain.[92] Nerve root compression seldom produces anaesthesia due to the overlap of dermatomes. A total loss of sensation invites consideration of a lesion of a peripheral nerve, multiple nerve roots, or higher centers.[85] Similarly, due to multisegmental innervation weakness is usually paresis rather than paralysis.[85] We described the validity of history items for the diagnosis of internal disk disruption and disk herniation, as well as the presentation of a typical patient with diskogenic radiculopathy. Central disk herniations may compress the cauda equina or the spinal cord. We discussed the symptoms of cauda equina syndrome and cord compression.

The typical patient with diskogenic dysfunction will complain of symptoms related to excessive imbibition, as well as excessive dehydration. Prolonged absence of compressive forces (e.g. recumbency) will allow the NP and, to a lesser extent, the AF to imbibe water. No longer restricted by an intact collagen network, excessive imbibition may cause mechanical stress on the innervated outer AF or other posterior structures making any segmental motion painful. Patients report pain and decreased mobility after getting out of bed. Prolonged weightbearing (e.g. a day of sitting or standing) will cause dehydration. This may result in nociception as the dehydrated disk allows for excessive translational segmental mobility and increases compression on normally unloaded segmental structures. Patients will report increased symptoms and likely decreased mobility as the day progresses, which can be reduced by adopting non-weightbearing positions. Maintaining any posture for prolonged periods will be painful due to decreased fluid exchange: increased intradiskal metabolite concentration may cause chemonociception. If the disk lesion protrudes onto structures in the spinal canal or IVF, any increases of intra-abdominal pressure (laughing, coughing, sneezing, straining, Valsalva maneuver) will increase nociceptive stimulation. We discussed the lack of validity of examination items in the diagnosis of internal disk disruption and the validity of neuroconductive and neurodynamic tests in the diagnosis of herniation.

Which movements a patient reports as aggravating or easing the pain depend on:

- Whether or not the disk lesion is contained.
- Whether or not the hydrostatic mechanism of the disk is intact.
- The presence or absence of an ischaemic and/or fibrotic radiculopathy or cauda compression.

Internal disk disruption and disk protrusion are examples of *contained* disk dysfunctions: the outer anular lamellae are intact and contain the NP.[85] In an extrusion or a sequestration the outer AF is ruptured: the disk dysfunction is *uncontained*. We discussed the correlation between the results of repeated movement tests and presence of contained or uncontained disk lesions.

The hydrostatic mechanism of the disk depends on 2 factors. The NP must be contained: severe endplate failure or anular incompetence will not allow the NP to convert compressive forces to tensile forces in the AF. Loss of nuclear volume as a result of a previous extrusion or sequestration, surgical herniotomy, or degradation will also render the hydrostatic mechanism less effective. Clinically, this may have the following effects. A patient with a (postero)lateral disk lesion with an intact hydrostatic mechanism will likely have increased pain with flexion, flexion with contralateral rotation or sidebending, and contralateral sideglides. Repeating these motions will increase and peripheralize the symptoms; symptoms will remain worse after these motions. Movements in the opposite directions may initially be painful depending on the position of the nuclear material, but will eventually decrease and centralize symptoms. Repeated movements in the direction causing centralization may affect the same patient differently at different times during the day due to changes in disk hydration. The effect also depends on what the patient has been doing before: walking tends to move the NP to a more central location, sitting and driving for an hour to come see the PT tends to displace the nuclear fragment to a more disadvantageous position. Placing the patient in a non-weightbearing rather than weightbearing position may be indicated in the latter case. In uncontained disk dysfunction patients with an insufficient hydrostatic mechanism, multiple directions of testing will only cause peripheralization.[89] The literature does not describe results of repeated movement testing in patients with an intact AF but a deficient hydrostatic mechanism. Per definition, patients with an intact hydrostatic mechanism, yet a ruptured outer AF do not exist.

The third factor affecting the response to movements is presence or absence of radiculopathy or cauda equina compression. An ischaemic nerve root will respond with an increase in symptoms as a result of any movement that increases compression and ischemia. These are in fact the exact movements that should reduce and centralize symptoms of the contained disk lesion causing the radicular symptoms in the first place! For example, extension and ipsilateral sidebend or sideglide will decrease the IVF diameter. Patients frequently adopt a position of non-weightbearing flexion and contralateral sidebend. This positional distraction maximally opens the IVF and allows recovery of circulation to the neural structures.[131] Flexion and contralateral sidebend motions may cause excessive tension decreasing intraneural circulation and increasing pain and paraesthesia. Fibrosis as a result of prolonged ischemia makes the neural structures more sensitive to tensile forces. Cauda equina and cord compression likely produce symptoms with movements that decrease the diameter of the spinal canal (e.g. extension, sidebending, or combinations).

Zygapophysial joint movement dysfunction

There are 4 clinically relevant ZJ movement dysfunctions:

- An extra-articular ZJ hypomobility dysfunction.
- A peri-articular ZJ hypomobility dysfunction.
- A pathomechanical ZJ hypomobility dysfunction.
- A ZJ hypermobility dysfunction.

Facet joint syndrome is a (manual) medicine term indicating pain stemming from the ZJs.[78] It is part of a structure-based classification. We discussed the different views on ZJ pain referral and the lack of valid history and examination items to identify ZJ dysfunction. Schwarzer et al[76] doubted the existence of the facet syndrome as a clinical entity. To consider the ZJ in the biomechanical classification system we need to be able to identify consistent signs and symptoms for a movement dysfunction of this joint.

The ZJs mainly limit flexion and rotation. Capsular tension limits anterior sagittal rotation in flexion; impaction of the anteromedial portions of the articular facets restricts the anterior translation component. Contralateral impaction and ipsilateral capsular tension limit rotation. Repeated joint compression may lead to inflammation. A capsular pattern restriction usually accompanies peripheral arthritis.[85] Capsulitis may progress to capsular fibrosis.[85] Excessive capsular tension can cause capsuloligamentous trauma. Subsequent muscular hypertonicity and splinting may also cause capsular restrictions. However, repetitive capsular tensile microtrauma might produce insufficient nociception to cause reflexogenic muscle splinting and may thus over time result in increased capsular length and decreased capsular mechanical stiffness.

Therefore, movement dysfunctions likely to occur in the ZJ are hypo- and hypermobility. Abnormal mobility will not be restricted to only ZJ structures. However, the assumption underlying the ZJ movement dysfunction as a diagnostic category is that the pathomechanical behavior is mainly determined by the ZJ changes. We will discuss ZJ hypermobility in the section on instability.

The signs and symptoms a patient with which a patient with a ZJ hypomobility will present are based completely on an extrapolation of (patho)anatomical and (patho)mechanical knowledge and are not supported by direct research. Hypomobile joints may cause symptoms as a result of normal stresses on abnormally shortened tissues. The primary complaint will be LBP which is likely ipsilateral due to the unilateral innervation of the joint. Somatic reffered pain may extend into the lower extremity; the pain is probably diffuse due to the multisegmental innervation of the joint. Pain below the knee invites consideration of dysfunctions in other structures in addition to ZJ hypomobility.[79] Radicular involvement does not occur in isolated ZJ hypomobility dysfunction.

Hypomobile ZJs hypothetically cause limited ROM with (possibly) end range pain. Meadows[85] suggested a subdivision into flexion and extension hypomobility. Flexion hypomobility causes restriction of flexion, contralateral sidebending, and a combination of these 2 movements, called a contralateral flexion quadrant. Paris and Loubert[131] suggested that (flexion) hypomobility will also result in an ipsilateral deviation during trunk flexion. Extension hypomobility will limit extension, ipsilateral sidebending, and the combination movement, the ipsilateral extension quadrant.[85,92] We might observe these restrictions during AROM and combined movement tests. Despite questionable reliability and validity, the biomechanical classification system uses PPIVM tests for segmental diagnosis. Unilateral ZJ hypomobility may also affect segmental rotation. Inconsistent coupling patterns make the rotational PPIVM findings aspecific to the direction (flexion or extension) of ZJ hypomobility. However, Meadows[85] suggested it may be a more sensitive clinical test for detecting the presence of segmental movement dysfunction.

After determining the presence, level, and direction of hypomobility we need to establish the cause of this dysfunction to help us determine the appropriate intervention. Meadows[85] suggested 1 extra-articular and 2 articular causes for hypomobility. Inextensible peri-articular tissues (e.g. muscle or tendon scarring, adhesions, or hypertonicity) might cause *extra-articular hypomobility*. Nociception may result in increased segmental muscle tone. Prolonged restriction of excursion may cause adaptive muscular shortening.[132] Meadows suggested that in this extra-articular hypomobility, range on PPIVM tests is decreased, but that the arthrokinematic associated glide tested with a PAIVM is normal. Meadows[85] hypothesized that due to their location muscles may restrict angular motion, but have less effect on linear motion.

Meadows[85] suggested that in an articular hypomobility both PPIVM and PAIVM tests are restricted. Meadows[85] proposed 2 types: *pericapsular* and *pathomechanical hypomobility*. The capsuloligamentous inextensibility of a pericapsular articular hypomobility produces a hard endfeel and a restricted range. A pathomechanical articular hypomobility has an abrupt, slightly springy, *jammed* endfeel. Hypotheses on the cause for pathomechanical hypomobilities include joint subluxation due to instability, mechanical locking on secondary contours in the joint, on articular surface deficiencies, or degenerative meniscoid inclusions, and muscle hypertonicity.[1,14,85]

Instability dysfunctions

There are 4 clinically relevant lumbar spine instability dysfunctions:

- A lumbar spine ligamentous instability dysfunction.
- A lumbar spine segmental instability dysfunction.
- A lumbar spine active and/or neural control subsystem deficiency.
- A lumbar spine mixed subsystem deficiency.

The neutral zone is that part of the physiological intervertebral motion, measured from the neutral position, within which spinal motion is produced with minimal internal resistance.[1] Defining instability as an increase in the range of the neutral zone is currently popular.[85] However, the neutral zone is an in vitro mechanical concept: there is no known way of measuring the neutral zone in vivo.[63] Meadows[85] proposed the more clinically useful definition of instability as *the presence of motion where no appreciable motion should exist*.

Stability of the lumbar spine depends on 3 subsystems: the passive, active, and neural control subsystems.[124,133] The passive subsystem consists of the vertebrae, disks, ZJs, and ligaments; the active

subsystem consists of the muscles and tendons acting on the lumbar spine. The neural control subsystem consists of the nerves and the central nervous system (CNS): it controls the active subsystem in its role of dynamic stabilization.[133]

Passive subsystem

We mentioned ZJ hypermobility as a possible ZJ movement dysfunction. Increased length and decreased stiffness of the ZJ and other ligamentous structures may allow for increased segmental mobility. Meadows[85] used the term *ligamentous instability* for this movement dysfunction. In the biomechanical model this type of instability is diagnosed by PPIVM test findings.[85] These tests may either demonstrate an increased ROM with a soft capsular endfeel, or a normal ROM with a muscular endfeel; the latter indicates protective muscular reactions as a result of a painful hypermobility.[85] In monograph 11.2.3. we discussed how capsuloligamentous structures are also partly responsible for limiting translatory or accessory motions. Meadows[85] stated that clinically ligamentous instability may also demonstrate excessive translatory mobility. Despite lack of reliability and validity research, the biomechanical model assumes that excessive translatory mobility may be determined by the findings on the segmental stability tests described earlier.[85]

Meadows[85] proposed a second type of instability dysfunction, *segmental instability*. This type of instability might result from disk degradation and ZJ surface degeneration.[85] Weiler et al[62] demonstrated significant increases in translatory movements in patients with disk degeneration when compared to controls. Segmental narrowing as a result of degenerative changes increases contact between the tips of the articular facets and the lamina of the vertebra below or the interarticular pars of the vertebra above.[134] Burton et al[63] found a significant correlation between reduced disk height and decreased lumbar spine sagittal plane ROM, especially in extension. In the biomechanical model, segmental instability is diagnosed by excessive mobility on segmental stability tests in combination with decreased ROM on PPIVM testing especially in extension and sidebending. The endfeel on PPIVM testing is likely harder, indicating bony impaction. In the biomechanical model, the finding of a restriction on PPIVM testing is a possible indication for joint mobilization. However, a hard endfeel on segmental extension and/or sidebending and positive stability tests in a patient diagnosed with segmental degeneration by way of imaging tests would pose a contra-indication to mobilizing techniques.

Active and neural control subsystem

Parkkola et al[135] found decreased strength, more degenerative changes, increased intramuscular fat deposits, and more more pronounced atrophy in the psoas, lumbar erector spinae, and multifidus muscles of patients with chronic LBP than in matched controls. Rantanen et al[136] reported selective fast-twitch (FT) fiber atrophy and structural abnormalities in slow-twitch (ST) muscle fibers in patients with a disk herniation. Zhao et al[137] reported significantly decreased size of ST and FT fibers in the symptomatic versus the normal side in patients with a disk herniation and a positive SLR test ($<70^0$). The ST fibers of the diseased side were significantly smaller when patients had central LBP.

Muscular changes appear to determine the outcome in surgically treated LBP. Sihvonen et al[138] reported more paraspinal atrophy in the unoperated levels in post-laminectomy patients with a poor versus those patients with a good outcome. Rantanen et al[136] compared 2 groups of patients 5 years after surgery for disk herniation: ST fiber diameter increased significantly in both groups, but only the good outcome group had significant increases in FT fiber diameter and a decrease in ST fiber abnormalities.

These changes in the active subsystem may be related to neural control subsystem failure. Sihvonen et al[138] found lesions to the dorsal ramus with corresponding paraspinal atrophy in patients with post-operative failed back syndrome. The authors pointed out that especially the medial branch of the dorsal ramus is vulnerable to traction in its course under the mammilo-accessory ligament with the lateral displacement of the lumbar muscles during surgery. Richardson et al[124] and Hides et al[139] reported decreased cross-sectional area of the lumbar multifidus at the symptomatic segment on the side of symptoms in patients with LBP. Changes occurred rapidly, in 1 subject within 24 hours after injury, making this likely the result of segmental inhibition.[124] In monograph 11.2.3. we discussed the function of the transverse abdominis in increasing non-direction specific stiffness of the lumbar spine by its activation prior to the prime movers in motions requiring trunk stability. In patients with chronic LBP the contraction of the transverse abdominis was delayed by 50 to 450 ms during arm movements; with leg movements it even followed rather than preceded contraction of the prime mover by up to several 100 ms.[124] The transverse abdominis is innervated by the T7 to L1 segments excluding monosegmental inhibition as a cause and implicating a long-loop spinal inhibitory reflex circuit or changes in motor control strategies originating in higher CNS centers.[124] Despite lacking data on

reliability and minimal support for validity, the tests for transverse abdominis and multifidus muscle function may provide the PT with information regarding active and neural control subsystem function.

The 3 subsystems interact to provide sufficient stability. Instability may occur even with an intact passive subsystem: the spinal column devoid of muscles is unable to support an axial load of even 20 N without buckling.[140] In vitro, simulated muscular forces decreased the neutral zone in intact and experimentally injured lumbar motion segments.[140,141] Paris[142] and Meadows[85] reported a number of history and physical examination findings possibly indicative of instability (Table 22). These signs and symptoms are neither sensitive, nor specific. Reliability and validity of the tests used to detect instability are poor or unknown. Passive, active, and neural control subsystem may be deficient in a number of combinations. Differential diagnosis between segmental instability and ZJ hypomobility dysfunction affects our choice of interventions: mobilization is contra-indicated in segmental instability. Because the active and neural control subsystem are the only systems we can influence with PT, treatment of all types of instability involves stabilizing exercises.

Neuromeningeal movement dysfunctions

In monograph 11.2.3. we discussed how the spinal cord needs to be able to move in relation to its meningeal covering and how the nervous system needs to be able to adapt to movement by changes in tension and movement in relation to the musculoskeletal tissues interfacing with its connective tissue sheaths. A movement dysfunction of the nervous system may occur as a result of congenital disorders, trauma, surgical complications, or degenerative changes.[87,88,143] There are 2 types of neuromeningeal movement dysfunctions: the tethered cord syndrome forms a contra-indication to PT treatment, the nerve root and dural movement dysfunctions may respond to nervous system mobilization techniques.[87]

Tethered cord syndrome

Disk prolapse or trauma may transversely stress the dura; the dura is less resistant to transverse than longitudinal stress and may tear.[87] Longitudinal traction to the dura and sudden increases in intravenous pressure may rupture the thin-walled veins of the plexus lining the spinal canal.[87] Spinal surgery may introduce blood into the spinal canal. Inflammatory byproducts of degenerative processes and myelographic contrast material may enter the spinal canal. All these processes may cause fibrous metaplasia. If bleeding and irritation occurs within the dural sac, fibrous adhesions may develop intermeningeally or even between cord and meninges.[87] The resultant *tethered cord syndrome* allows for the forces usually transmitted away from the cord through the denticulate ligaments, meninges, and nerve roots, to be transmitted directly to the conductive tissues of the cord.[87]

Tethered cord syndrome can also occur as part of a congenital malformation of the neuromeningeal structures. In *diastematomyelia*, a fibrous, cartilaginous, or bony band or spicule separates the cord into 2 hemicords each surrounded by a dural sac.[143] Diastematomyelia is frequently associated with *spina bifida*.[143] Spina bifida aperta will be obvious from birth, but spina bifida occulta can remain hidden. Diastematomyelia, but also adhesions between the spinal cord and the dura or overlying skin may cause symptoms initially, but progressive neurological deficits can also occur later in life in spina bifida occulta due to cord tethering. Symptoms may include loss of strength and sensation, pain in a dermatomal distribution, onset or increase of lower extremity spasticity, changes in bowel or bladder control, and progressive lower extremity deformities, especially in the ankles and feet.[143] Symptoms are the result of tension to the spinal cord, so treatment with interventions that may further increase tension (e.g. neural mobilizations) are contra-indicated.[87] Butler[87] mentioned a number of signs and symptoms of tethered cord syndrome related to spina bifida occulta lesions: restricted gastrocnemius, soleus, and hamstrings movement, and hair tufts and epidermal sinuses in the lumbosacral region.

Nerve root and dural movement dysfunctions

Bleeding and irritation in the spinal may also cause fibrous metaplasia between the dura mater and the epidural tissues.[87] Fibrous metaplasia or fibrosis may also affects nerve root structures. In monograph 11.2.3. we discussed how compression of the nerve root caused venous congestion and subsequent fibrosis. Appropriate (non)surgical interventions may have reduced compressive forces, but fibrosis may still be present. Nerve root fibrosis may also result from surgical procedures intended to relieve symptoms: postlaminectomy fibrosis may play a role in failed back syndrome.[87] McKenzie[88] called fibrosis affecting the nerve root *adherent nerve root dysfunction*.

Based on extrapolation of anatomical and biomechanical knowledge a patient with a dural movement dysfunction might present with pain on movements increasing dural tension (e.g.trunk flexion, slump tests,

and SLR).[87] Motor or sensory abnormalities are unlikely due to the anatomical differences between the cauda equina and nerve roots discussed in monograph 11.2.3. Pain referral may be multisegmental and bilateral based on the innervation of the dura described earlier. Butler[87] mentioned coccygodynia with dural adhesions as a result of the attachment of the filum terminale externum to the coccyx.

Again based on extrapolation, patients with nerve root movement dysfunctions will likely complain of radicular pain. Repeated tension with subsequent intraneural compression may interfere with conduction, resulting in a radicular neurologic deficit. Movements and positions that increase nerve root tension (e.g. flexion, contralateral sidebending, contralateral flexion quadrant, SLR or PKB, and slump) will increase symptoms. McKenzie[88] reported ipsilateral deviation during active trunk flexion in case of a nerve root movement dysfunction. Movements decreasing nerve root tension will decrease symptoms. Repeated movement tests may increase or decrease symptoms as a result of changes in nerve root tension during the motions, but symptoms will not lastingly change, as they would if a contained diskogenic dysfunction were responsible for the radicular symptoms. Segmental motion tests and stability tests may implicate an instability as the original cause for nerve root dysfunction.

Stenotic syndrome

Degenerative disease causes increased radial bulging, osteophytosis, and flaval inbulging. These stenotic changes may affect the spinal canal causing central stenotic syndrome. They may also affect the lateral recess or nerve root canal causing lateral stenotic syndrome.[127] Stenosis can also be congenital or traumatic.[127] For the PT, stenotic syndrome is in fact a biomechanical diagnosis of exclusion. After excluding disease and discogenic, instability, and neuromeningeal movement dysfunctions, one must consider stenotic syndrome as the etiology of neurogenic symptoms.

Based on an extrapolation of anatomical and biomechanical knowledge, patients with lateral stenotic syndrome may present with radicular pain and possibly a radicular deficit. Complaints may increase with movements and positions decreasing IVF diameter (e.g. extension, ipsilateral sidebending, and ipsilateral extension quadrant). Trunk flexion, contralateral sidebending, and contralateral flexion quadrant may decrease symptoms.

Central stenosis may result in symptoms related to cauda equina compression, aggravated by any movement that decreases the diameter of the central canal. Extension will affect truly central lesions, sidebending and extension quadrant motions may have more effect on asymmetric central lesions. Extension may reduce the cross-sectional area of the central spinal canal in degenerated spines by up to 67% versus a 9% reduction in the normal spine.[127] Postures and movements involving flexion may reduce symptoms. Weghtbearing may also produce symptoms of stenosis, non-weightbearing may relieve symptoms. Compressive loading may have an even greater effect on canal dimensions than extension.[127] Symptoms are likely intermittent, but may become constant if concomitant dysfunctions further compromise central canal and IVF diameter. Both lateral and central stenotic lesions may cause neurogenic claudication. The tests described earlier may be helpful in distinguishing neurogenic from vascular claudication.

The anatomical changes resulting from degeneration are not amenable to non-surgical interventions. In monograph 11.2.3. we discussed the kinematic effects of the lower extremity on the lumbar spine. Mobilization of hip extension, strengthening the gluteus medius, heel lifts, mobilization of adjacent segments, and lumbopelvic stabilizing exercises may all decrease the frequency of neural structure compression in stenotic lesions.

Sacroiliac dysfunctions

The only examination items for the SIJ seemingly reliable and valid are some provocation tests. Postive provocation tests implicate the SIJ as a source of complaints, but provide no further information on appropriate interventions. The biomechanical system uses the SIJ tests described earlier to diagnose SIJ dysfunctions despite their lack of reliability or validity. Diagnosis and treatment of the SIJ is based solely on extrapolation of anatomical and biomechanical knowledge. However, a uniformly accepted model for the biomechanical behavior of the SIJ does not exist. The form and force closure model of the Musculoskeletal Research Group at the Erasmus University in Rotterdam discussed in the previous monograph seems a useful theoretical construct. Adopting this model implies that all SIJ dysfunctions are viewed as a result of the failure of form and force closure.

Instability dysfunction

There are 2 clinically relevant SIJ instability dysfunctions:

- An SIJ combined subsystem deficiency.
- An SIJ active and/or neural control subsystem deficiency.

Combined subsystem deficiency

Stability of the SIJ is a function of the passive, active, and neural control subsystems. In monograph 11.2.3.we discussed aspects of form closure. The rough texture, symmetrical opposing osseochondral ridges and depressions, and undulated shape of the joint surfaces and the wedge shape of the sacrum all contribute to stability. We also discussed *self-bracing* of the SIJ: sacral nutation results in increased capsuloligamentous tension with resultant compressive forces perpendicular to the joint surfaces. This increases force closure and thus stability.

Passive subsystem deficiency can affect form closure mechanisms. Vleeming et al[126] mentioned osseochondral lesions with loosening of reciprocal ridges and depressions on the joint surfaces. Sacral and pelvic fractures may impact anatomical structure and affect form closure. Ligamentous laxity decreases the efficiency of the self-bracing mechanism. In the biomechanical system, the PAM (and possibly the PPM) tests described earlier may detect passive subsystem deficiency. Pure SIJ translation and rotation are only possible when joint surface irregularities, joint surface orientation, ligamentous laxity, and neuromuscular dysfunction allow for sufficient joint separation.[144,145]

However, passive subsystem deficiency alone need not result in symptoms. In monograph 11.2.3. we discussed how appropriate muscular contraction can increase capsuloligamentous tension and improve efficiency of the self-bracing mechanism. In the biomechanical system, the supine and prone SLR tests, in combination with the tests decribed for the transverse abdominis and multifidus muscles, can be used to diagnose active and neural control subsystem deficiency.

Active and neural control subsystem deficiency

Active and neural control subsystem deficiencies occur without passive subsytem deficiency. In monograph 11.2.3. we discussed how a flat back posture unloads a painful pubic symphysis e.g. postpartum.[125] A flat back posture may also decrease compression on a painful posterior anulus, ZJ, or neural structures. The sacral counternutation associated with a flat back posture[125] affects the ability of the SIJ to self-brace by way of nutation. Muscular contraction must play an increased role in providing sufficient capsuloligamentous tension to ensure joint stability; active and neural control subsystem deficiency may lead to instability without structural changes in the passive structures.

Again because the active and neural control subsystems are the only subsytems we can influence with PT, stabilizing exercises are part of the plan of treatment for every SIJ instability. The dysfunction responsible for the flat back posture must be addressed appropriately. Application of an SIJ-belt may decrease excessive SIJ motion in an SIJ instability dysfunction.[126]

Hypomobility dysfunction

There are 3 clinically relevant SIJ hypomobility dysfunctions:

- A pathomechanical SIJ hypomobility dysfunction.
- A peri-articular SIJ hypomobility dysfunction.
- An extra-articular SIJ hypomobility dysfunction.

Pathomechanical hypomobility

Basing our SIJ biomechanical model on the form and force closure concept means that any hypomobility has to be the result of an instability dysfunction: sufficient separation of joint surfaces is needed to allow for dissociation of complementary ridges and depressions with a subsequent pathomechanical SIJ hypomobility dysfunction.

Lee[91] reported 4 distinct positional abnormalities of the innominate on the sacrum. The innominate of the hypomobile SIJ usually rotates anteriorly possibly due to loss of self-bracing with anterior innominate rotation. Occassionally the innominate rotates posteriorly.[91] The position of the acetabulum is ventral to the SIJ: ground reaction forces tend to posteriorly rotate the innominate. However, this only seems possible if there is a greater underlying instability. Superior translation of the innominate in relation to the sacrum can also be caused by ground reaction forces. Superior translation usually occurs together with the anterior and posterior innominate rotations.[91] Superior translation of the innominate may also indicate greater instability.[91] Finally, the innominate may displace inferiorly as the result of a longitudinal traction down the leg (e.g.

falling forward with the foot caught in a ladder or stirrups).[91] In the biomechanical system, despite lack of reliability and validity positional palpation determines the type of positional abnormality.[91]

The onset is usually sudden and may be related to asymmetric lifting introducing off-center loads into the SIJs. Sudden axial compressive forces through the leg may cause superior innominate translation and/or posterior rotation.[131] Axial traction forces may produce an inferiorly translated innominate.[91] Aggravating movements depend on the particular dysfunction, but stressing the restricted endrange is painful. Patients with a superior translation of the innominate are usually severely limited in any weightbearing ADL.[92] Provocation tests may be positive, depending on stage and irritability of the dysfunction. Patients perform the supine and prone active SLR tests poorly; the tests are often painful.[91] Innominate compression as part of these tests may increase pain; the isometric contraction may improve performance with these tests.[91] Despite a lack of reliability and validity, the biomechanical system uses PPM and PAM tests for diagnosis of pathomechanical hypomobility: both show decreased ROM and a pathomechanical endfeel.[92] Palpation just inferior to the PSIS may indicate increased tension in the long dorsal SI ligament due to anterior innominate rotation.[125]

Peri-articular hypomobility

Long-term pathomechanical hypomobility may cause adaptive capsuloligamentous shortening, producing a pericapsular restriction. Capsuloligamentous restrictions may also result from the excessive tension of a subluxation, which thereafter reduces. This means positional palpation tests may be positive or negative. Positive provocation tests may stress shortened capsuloligamentous structures and thus cause pain. The biomechanical system uses motion palpation, PPM, and PAM tests in the diagnosis of a pericapsular SIJ hypomobility dysfunction: ROM is decreased with all tests, PPM and PAM tests reveal a hard capsular endfeel.[92]

Extra-articular hypomobility

Nociception originating in the SIJ may increase local muscle tone by way of somatosomatic and somatosympathetic reflex circuits.[34] Increased tone may also be an attempt to stabilize an unstable joint. The biomechanical system differentiates extra-articular from articular restrictions by a decrease in ROM on motion palpation and PPM tests, yet minimal motion with a normal endfeel on PAM tests. Positional tests may be positive. If nociception caused the increased muscle tone, provocation tests are likely painful.

The type of hypomobility determines the therapeutic intervention. Based on the assumption that hypomobility can only result from an underlying instability means we also need to diagnose and treat the causal instability dysfunction.

Pubic symphysis movement dysfunctions

There are 2 clinically relevant pubic symphysis movement dysfunctions:

- A painful symphysial instability dysfunction.
- A symphysial hypomobility dysfunction.

Symphysial instability dysfunction

In monograph 11.2.3. we discussed the limited role of the symphysis in pelvic stability.[126,146,147] Vleeming et al[126] stated that symphysial hypermobility is only possible with a hypermobile SIJ. This would suggest that finding symphysial hypermobility on examination should prompt us to examine or even a priori accept the presence of SIJ hypermobility. Conversely, Lindsey et al[148] reported that a 4 cm symphysial diastasis can occur without an effect on the SIJs. This would suggest that symphysial hypermobility is in fact a movement dysfunction occurring separately from the SIJ instability. Due to its strong, amphiarthrotic nature, symphysial hypermobility and instability implies failure of the capsuloligamentous structures and intra-articular disk. Therefore, symptomatic symphysial hypermobility automatically implies insufficiency of all 3 stability subsystems.

An unstable symphysis may be clinically less important than a painful (unstable) symphysis. The main role of the symphysis is to allow sufficient deformation to make SIJ motion possible.[147] A painful symphysis is unloaded by a flat back posture; the associated sacral counternutation disengages the SIJ self-bracing mechanism leading to SIJ instability. Therefore, the only clinically relevant symphysial instability is one that causes compensatory SIJ dysfunction. The provocation and translation tests described earlier must reproduce pain, pain during ADL and examination needs to be located in the groin area, and there must be signs and symptoms of decreased SIJ stability, before we address symphysial instability.

Symphysial hypomobility dysfunction

Symphysial instability need not alter gait and other reciprocal functions. Hypomobility will affect the ability of the innominates to rotate in an opposite direction during these activities.[93] Hypomobility will stress the peri-articular tissues during the forced deformation with reciprocal activities and can thus cause an antalgic posture affecting SIJ stability. Unilateral increases in pyramidalis muscle tone may cause a superiorly displaced pubic bone.[91] Unilateral muscular inextensibility due to adaptive shortening or increased tone may affect the symphysis with its multitude of muscular insertions. Long-standing articular restrictions may cause shortening of articular structures. The cleft in the articular disc might predispose the joint to pathomechanical hypomobility.

Sacrococcygeal dysfunctions

With its connections to the sacrotuberous and sacrospinous ligaments and the pelvic floor muscles, the position of the coccyx might influence the tension and force-length relationship in these structures. This may affect SIJ self-bracing and the coordinated contraction between the multifidus, transversus abdominis, pelvic floor, and diaphragm muscles, described in monograph 11.2.3. Thus sacrococcygeal dysfunction may affect both lumbar and SIJ stability. Conversely, SIJ dysfunction may alter tension in the myofascial structures of the pelvic floor and thus affect sacrococcygeal ROM.[149]

Maigne[150] described radiographic studies in patients with coccydynia. He considered flexion of S5-Co1 greater than 25^0, extension over 15^0, and translation over 25% of the anteroposterior diameter of the vertebra pathological. In a study of 96 patients with coccydynia he found posterior luxation in 23, anterior luxation in 3, and flexion hypermobility in 18 patients. The PPM test described earlier may be helpful in determining mobility of the coccyx. Sacrococcygeal dysfunction and coccydynia may result from child birth or other trauma. Tail bone pain may be indicative of nonorganic LBP.[52] It may be related to dural movement dysfunction.[87] In the absence of hypermobility, coccydynia may be due to a bursitis between the skin and the bony tip of the coccyx, referred pain from the SIJ, a sprain of the insertion of the sacrotuberous ligament, or psychogenic pain due to hysteria or depression.[150] Greenman[151] noted changes in urinary frequency and urgency, dysuria, dyspareunia, and rectal pruritus without clear organic reasons as symptoms of pelvic floor dysfunction. Information on PT treatment of is lacking.

TREATMENT

The biomechanical model described earlier matches interventions and contra-indications to its different diagnostic categories (Table 23). The model matches the choice of manual techniques to the irritability of the condition; the relation between endfeel and pain determines the level of irritability (Table 24).[85] However, as with diagnosis, the interventions are selected based on an extrapolation of (patho)anatomical and (patho)biomechanical knowledge rather than on research.

Research on the efficacy of interventions in LBP using the traditional medical model for classification can only provide approximately 15% of patients with a specific diagnosis[67,72,75]; the rest is (erroneously) considered a homogenous group with possible diagnostic labels of lumbago, lumbar strain, or mechanical LBP.[72] Research considering all non-specific LBP homogenous probably does not measure the effects that we can expect from truly homogenous groups.[128] Research using a clinical guideline index as the basis for classification and diagnosis is more useful to the PT. A second avenue of useful research studies the validity of the rationales on which PTs base treatment interventions, e.g. nuclear movement as a result of repeated trunk extension. We will discuss the rationales and the research base for the use of manual therapy, traction, and exercise in the treatment of patients with lumbopelvic region neuromusculoskeletal complaints. Precise descriptions of the different techniques and a discussion of (contra)indications is outside the scope of this monograph, but is available in other sources.[34,85,88,91,92,124]

Manual therapy

There are multiple rationales for the use of manual therapy. Manual techniques may have a:

- Psychological effect as a result of patient-therapist interaction.[131]
- Mechanical effect, e.g. altering positional relationships or mobilizing joints through stretching or rupturing restrictive structures.[131,152]

- Neurophysiologic effect, e.g. activation of the gate control mechanism by stimulation of thick fiber afferents, reflexogenic decrease of muscle hypertonicity by stimulation of type III mechanoreceptors, or release of substance P and endorphines as a result of manipulative techniques.[131,152]

Altering positional relationships in the lumbar spine

Wilson and Ilfeld[153] used a regional rotatory manipulation on 13 patients with low lumbar disk herniation confirmed on a myelogram. Immediately after manipulation, a repeat myelogram showed no change in herniation in 12 patients and an increase in 1. During subsequent surgery, the authors found that the AF of this 1 patient was intact. The authors stated that it is unlikely that an extrusion can be reduced by manipulation, but, despite finding the herniation enlarged by manipulation in 1 patient, that manipulation may be beneficial in patients with disk protrusion with a still intact posterior AF.

Zhao and Feng[154] studied the effects of non-operative treatment with segmental manipulation on herniation size and location using repeated CT images in 22 patients with multi-level and 39 patients with single-level herniations. They found no changes in size, position, or volume on CT. The authors hypothesized that studies that show reduced herniation following manipulation may be flawed as a result of the natural shrinkage of the extruded tissue over time, due to different planes for pre- and post-intervention imaging, and even because forceful manipulation may progress an extrusion to a sequestration with migration of disk fragments out of the plane of the post-intervention CT image.

In a case study Zhao and Feng[155] describe the treatment of a 12 year old girl with a herniation of L5-S1 confirmed on CT and MRI. Despite treatment with rotatory manipulation of the affected motion segment and despite full functional recovery, no changes on CT scan 4 and 10.5 months after the initial onset of complaints were apparent.

Altering positional relationships in the sacroiliac joint

Cibulka et al[123] studied the effect of SIJ manipulation on the angle a line drawn through ASIS and PSIS made with the horizontal, measured with an inclinometer. They diagnosed 20 patients referred for non-specific LBP with SIJ dysfunction, based on a battery of tests described earlier. They randomly divided patients into 2 equal groups. The experimental group received manipulation, the control group received no treatment. Manipulation resulted in significant changes in innominate tilt on both sides. Innominate tilt changed in opposite directions on the manipulated and non-manipulated side. The lacking reliability and validity of positional palpation tests may invalidate the conclusions in this study.

Tullberg et al[121] used manipulation and specific muscle energy techniques to treat 10 women of 21 to 53 years old for unilateral SIJ dysfunction. Despite positional tests returning to normal after treatment, rontgenstereophotogrammetric analysis of tantalum balls inserted into the sacrum and the innominate on the symptomatic side showed no alteration in the SIJ positional relationship.

Decreasing muscle hypertonicity

Herzog et al[152] studied the EMG responses in selected trunk and proximal extremity muscles to manipulation in 10 asymptomatic subjects. Increased EMG activity occurred within 50 to 200 ms after manipulation and lasted 100 to 400 ms. EMG responses were consistent and repeatable for the level manipulated, yet extended beyond the area of application of the manipulative technique. The responses were likely non-volitional: the time of onset after manipulation was too short. Shape and duration showed a series of spatially and temporally non-synchronized motor unit action potentials indicating reflexes originating in multiple sensors (articular, skin, muscle, tendon). Herzog et al[152] also anecdotally reported a decrease in hypertonicity in symptomatic subjects immediately after the manipulation-induced EMG response. The authors stated this may support the neurophysiologic effect of manipulation decreasing muscle hypertonicity in back pain.

Clinical outcome studies

Studies using a clinical guideline index as a basis for diagnosis and involving manual therapy interventions are rare. Delitto et al[129] reported both statistically and clinically significant improved Oswestry scores in patients classified and treated appropriately based on their treatment-based system as needing SIJ mobilization and extension exercises as compared to patients with the same diagnosis treated with an inappropriate, generic flexion program. Fritz[72] reported on a study where 76 patients with acute LBP were randomized in one treatment group based on their classification and a second, generic treatment group. Treatment based on their classification resulted in improved outcomes in generic and disease-specific health status measures and return to work status at 4 weeks.

In summary, research does not support the use of rotatory manual techniques for reduction of herniations in contained or uncontained disks. Research by Nachemson[156] contradicted the rationale that negative intradiscal pressure during rotation may "suck in" a herniation[154] : in vivo, intradiscal pressure in the L3-L4 disk increased when rotation was added to trunk flexion with weights. Rotatory techniques seem contra-indicated in diskogenic dysfunctions: tensile strain to anular fibers as a result of rotation may further weaken nuclear containment.[157] Research also does not support the rational that manual therapy can affect the positional relationship in the SIJ and thus decrease complaints. Neurophysiologic mechanisms may offer a better explanation of the effects of manual therapy. Clinical outcome studies show better results when patients with non-specific LBP are classified using a clinical guideline index and treated with amnual techniques when appropriate based on this classification. Further clinical outcome studies and research on the rationale underlying manual therapy interventions is clearly needed.

Traction

We can apply traction to the lumbar spine manually or mechanically; in a supine, prone, or inverted position; in different degrees of trunk flexion or extension; with a constant or intermittent force application; and using a conventional or a split-table.[157] Table 23 reviewed the indications for traction in the biomechanical model. Rationales underlying the use of traction include:

- Decreasing compressive forces and thus circulatory compromise to neural structures.[131]
- Restoring nuclear material to a more normal position.[157]

Restoring nuclear position

A number of case studies reviewed the effects of traction on nuclear position. Matthews[158] treated 2 patients with disc protrusions confirmed on epidurography to 120 lbs of continuous lumbar traction in prone on a conventional table. A 46 year old woman with protrusions between L1 and L4 no longer had protrusions on epidurography after 38 minutes of traction; repeat epidurography showed returning defects after 14 minutes. Symptoms recurred 20 days later; the epidurography showed disk protrusions similar to the first study. A 67 year old male had an L3-L4 protrusion reduced after 4 and even further reduced after 20 minutes of traction. The protrusions returned to two-thirds of their original size 10 minutes post-traction.

Gupta and Ramarao[159] treated 14 patients with disk prolapse confirmed by epidurography with 10 to 15 days of continuous bed traction. They applied 60 to 80 lbs of tractionwas through the thighs with adhesive plaster and the foot end of the bed raised 9 to 12 inches. Patients received a 15 to 20 minute rest period from traction every 3 to 4 hours. On a repeat epidurography after 10 to 15 days of this traction treatment, 8 patients showed a return to normal on a P/A study and 11 returned to normal on a lateral study.

Onel et al[160] studied the effects of 15 minutes of traction at 45 kg in a supine position with the legs in semiflexion on 30 patients with a disk herniation confirmed on CT scan. The patient group consisted of 18 men and 12 women between ages 20 and 40. CT scans were taken before traction and after 15 minutes of continuous traction. Of the 14 patients with a median herniation, 11 showed regression of the herniation, 2 showed an increase, and 1 showed no change. Six of 9 patients with a posterolateral herniation showed a decrease, while 3 showed no change. Of 7 patients with a lateral herniation 4 decreased and 3 remained the same.

Traction may affect the position of herniated nuclear material in 2 ways. It may create a negative intradiscal pressure or even a central vacuum inside the disk, which may cause a central migration of the herniated nuclear material.[12,158,160] This hypothesis is supported by Nachemson's findings[156]: 500 N of traction in supine reduced the L3-L4 intradiscal pressure to 0. Traction may lead to tensioning of the PLL, which will then exert an anteriorly directed force on a herniation underneath this ligament.[160] Onel et al[160] stated that the moderate result of traction on reducing lateral herniations support the role of the PLL pushing back herniations: these lateral herniations are not covered by this ligament. Harrington et al[161] supported this rationale for traction; they found that traction results in an anteriorly directed force generated at the PLL at the mid-body level of L1. Although not directly applicable to the scenario of affecting a disk herniation at mid-disk level, at least this study shows the generation of an anteriorly directed force in the PLL by traction. We should remember that the PLL is wider at the level of the disk than at the level of the vertebral body; it also stands off several millimeters from the posterior surface of the body, but is intimately connected to the disk.[161] Traction may produce a greater anteriorly directed force at disk level than at mid-body level under the same traction forces.

In summary, weak evidence exists that continuous lumbar traction can temporarily influence the location of herniated nuclear material. The case studies reviewed lack control groups, study forms of traction no longer clinically used, and give no information regarding functional outcome. If mechanical compression is indeed a source of pain in patients with a disk herniation, traction could at least have a temporary effect on symptoms.

Exercise

Exercise therapy can serve numerous functions in the treatment of patients with lumbopelvic region complaints, e.g. addressing muscle strength and length deficits in the lower extremities and lumbopelvic region, teaching correct and safe lifting techniques and other ADL functions, decreasing pain, or increasing and maintaining ROM. We will review McKenzie and stabilization exercises.

McKenzie exercises

McKenzie exercises are passive and active exercises in beginning, middle, and endrange of trunk flexion, in extension, and in a combination of sidebending and rotation called sidegliding.[88] Patients perform the exercises weightbearing or non-weightbearing. Exercise prescription is based on the ability of the exercises to centralize the patient's symptoms. McKenzie[88] stated that centralization only occurs in the derangement syndrome, *the situation in which the normal resting position of the articular surfaces is disturbed as a result of the change in position of the fluid nucleus between these surfaces*. This definition equates the derangement syndrome with a diskogenic dysfunction. McKenzie's conceptual model for treatment of diskogenic dysfunctions is that an intact anular wall during spine segment movement moves the NP away from the side of compression loading, i.e. towards the convexity. Simply put, with anular fibers present to exert force on the NP, during flexion the NP will move posteriorly and during extension the NP will move anteriorly.[157] Midrange exercises may be more appropriate than endrange exercises in patients with neural compression. Table 25 reviews studies on the effect of flexion and extension on nuclear position.

All studies in Table 25 (except the study by Gill et al[163]) supported anterior NP movement with extension and/or posterior movement of the NP with flexion in normal disks. Schnebel et al[165] found posterior movement of the dye used in discography with extension in degenerated disks. Gill et al[163] found extravasation of dye into the epidural space with repeated extension. This extravasation increased with increasing disk degeneration. It is unclear whether this extravasation means that repeated extension movements can lead to further extrusion of already extruded nuclear material[163], but Schnebel et al[165] suspected that the observed discographic changes are dye movements only. The research reviewed does not allow the use of the model of anterior nuclear movement with extension to justify the McKenzie approach with degenerated or herniated disks. All in vivo studies were done in a non-weightbearing position; generalization to partial or full weightbearing McKenzie protocol exercises is not warranted. All studies reviewed sagittal plane motion; conclusions regarding frontal plane movements are not warranted.[157]

Schnebel et al[165] suggested that the clinical results of the McKenzie approach may be related to activating gate control mechanisms, neural tissue relaxation, and disk hydration. Schnebel et al[169] showed that extension decreased nerve root tension. Magnusson et al[170] supported the theory of increased disk hydration; they showed that hyperextension allowed for more hydration after sitting than did recovery in a prone position alone. The authors hypothesized that the ZJs may provide for a fulcrum in hyperextension, allowing tension to be applied to the disk, unloading the disk, and increasing imbibition. Increased disk hydration reduces posterior bulging[12,171] possibly reducing mechanical stimulation of nerve roots and other extradiscal tissues.[157]

Assuming that a disk herniation causes symptoms and assuming that mechanical stimulation of nociceptors due to changes in nuclear position is at least partly responsible for the problems reported, it appears necessary for future research to define a symptomatic versus an asymptomatic disk rather than distinguishing between degenerated and non-degenerated disks. It is clear that a degenerated disk does not show the same movement of the NP with repeated extension as does a non-degenerated disk. But how much does a disk have to degenerate to become symptomatic? A disk may be minimally degenerated, yet symptomatic, and still respond to repeated extension as would a non-degenerated disk, validating McKenzie's conceptual model.

Stabilization exercises

In monograph 11.2.3. we discussed the role of the transverse abdominis and multifidus muscles in the stabilization of the lumbopelvic region. We discussed earlier the changes in the active and neural control

subsystems in patients with acute and post-operative LBP. Richardson et al[124] described a stabilization protocol for patients with instability dysfunctions. Hides et al[139] noted decreased multifidus muscle cross-sectional area (CSA) diagnosed with ultrasound imaging ipsilateral to the location of pain in patients with first-episode LBP. The authors randomly allocated patients to a control and a treatment group. The control group received standard medical treatment: minimal bed rest and minor analgesics. The experimental group performed specific stabilization exercises aimed at the transverse abdominis and multifidus muscles. At 4 weeks there was no between-group difference in pain and disability level, and lumbar and SLR ROM measurements. However, multifidus muscle CSA recovery was significantly more rapid and more complete in the exercise group. Muscle recovery was not automatic: at 10 weeks CSA measurements were not significantly different from those made at 4 weeks, i.e. there was no spontaneous recovery of structural changes in the multifidus muscle in the control group even after remission of painful symptoms.[139]

Richardson et al[124] reported on 39 patients with first-episode unilateral LBP with ipsilateral decreased segmental multifidus muscle CSA. The study randomly allocated patients to a control or experimental group. The controls received the standard medical treatment; the experimental group performed specific stabilization exercises. The authors assessed pain, disability, ROM, and multifidus muscle CSA weekly over 4 weeks. At 4 weeks there was no between-group difference on pain and disability scores. Multifidus CSA was smaller in the controls at 4 and 10 weeks. Reassessments at 10 and 35 weeks, and at 1 year established recurrence. Recurrence over the course of a year was 80% in the control versus only 30% in the experimental group. The authors suggested that measures of pain, disability, and ROM do not correlate with recurrence rate.

O'Sullivan et al[133] studied the effect of 10 weeks of specific exercises for transverse abdominis and multifidus muscles in patients with chronic LBP and a radiologic diagnosis of spondylolysis or spondylolisthesis. The authors randomly assigned patients to a control and a specific exercise group. Control group patients carried out regular weekly general exercise routines; some were involved in supervised exercise programs and received pain-relieving PT modalities. At 10 weeks the exercise group showed a significant decrease in pain and disability levels pre- versus post-intervention and when compared to the control group. There were no significant changes in outcome measures in the control group at 10 weeks. At a 30 month follow-up the exercise group maintained a significant reduction in pain and disability; no significant changes occurred in the control group. The authors concluded that specific stabilization exercises are effective in reducing pain and disability in patients with chronically symptomatic spondylolysis and spondylolisthesis.

In summary, specific stabilization exercises appear effective in reversing structural changes in the multifidus muscles in patients with acute LBP. Specific stabilization may reduce recurrence rates after a first episode of acute LBP. In a subgroup of patients with structural abnormalities predisposing them to segmental instability, stabilization exercises reduced long-term pain and disability levels when compared to general exercise. Further research is needed before generalization of these results to other patients groups suspected of having LBP on the basis of instability dysfunctions.

CASE SCENARIOS

Case scenario #1

A 35 year old man consults with you for central LBP, left buttock pain, and left lateral leg pain of insidious onset. He complains of intermittent tingling in the lateral aspect of the left leg and decreased sensation of the medial 2 toes. Symptoms are aggravated by prolonged sitting, driving, trunk flexion, coughing, and bowel movements. The patient is an avid cyclist and notes increased symptoms when riding his bicycle. Symptoms are relieved upon recumbency and prolonged walking. The patient denies abnormalities in bowel, bladder, or sexual function. Family history and previous medical history are unremarkable. The patient scores no positives on the medical screening questionnaire.

1. After this cursory history, what is your most likely diagnosis?

a. Lumbar diskogenic dysfunction with radiculopathy.
b. Lumbar diskogenic dysfunction with cauda equina syndrome.
c. Lateral stenotic syndrome with radiculopathy.
d. ZJ hypomobility dysfunction.
e. Vacular stenosis mimicking musculoskeletal symptoms.

The most likely diagnosis is **lumbar diskogenic dysfunction with radiculopathy**. Cauda equina is unlikely due to the absence of bowel and bladder symptoms, and intact sexual function. Lateral stenotic syndrome is unlikely, because of the patient's age and becuase most aggravating activities are in flexion. This should relieve symptoms in case of stenosis: it increases IVF diameter. ZJ dysfunctions do not cause radicular symptoms. You can exclude vascular stenosis due to a decrease in symptoms with walking.

Upon observation you note no trunk deviation. Active trunk flexion and left sidebending reproduce leg and LBP. All other cardinal plane motions are painfree. Right flexion quadrant and left extension quadrant also reproduce symptoms. Repeated flexion intensifies the foot pain, repeated extension centralizes the symptoms to the central low back. Repeated sideglides have no effect. Slump test and left SLR produce leg and LBP, PKB does not. Provocation tests for the SIJ are negative. Lower lumbar muscle guarding prevents PPIVM testing. Lower extremity scan is negative. Neurologic testing reveals a weak Achilles tendon reflex, weak extensor hallucis longus (EHL), and decreased sensitivity to light touch on the dorsal aspect of the big and second toe, all on the left side. Gait reveals a slight Trendelenburg gait on the left. You diagnose the patient with a discogenic dysfunction causing left radicular pain and neurological deficit.

2. Which IVD is most likely involved?

a. L2-L3.
b. L3-L4.
c. L4-L5.
d. L5-S1.

The IVD most likelyto be involved is the **L4-L5** disk. Weakness of the gluteus medius and EHL, together with the other deficits reveal an L5 radicular lesion. A negative PKB makes higher lumbar radicular lesions (L2-L3) unlikely.[86] Discogenic lesions of the L5 root can occur centrally at L4-L5 and laterally at L5-S1. Lateral L5-S1 disc lesions should produce centralization or peripheralization of symptoms with repeated sidegliding; central L4-L5 lesions are unlikely to be affected by repeated frontal plane movements.

Based on the studies by Donelson et al[89,90] you know that this patient is a good candidate for for a McKenzie approach. You institute a program of repeated extension in standing 10 times every half hour and you educate the patient regarding avoiding prolonged or repeated flexion activities. You follow up with the patient 1 week later and the patient reports an increase in leg pain after an initial decrease in symptoms.The patient states he helped a friend move over the weekend. The patient still reports the same aggravating and easing motions and positions and denies abnormalities in bowel and bladder function, or sexual function. Re-evaluation is similar to the initial, however, repeated extension in standing now peripheralizes symptoms, and the strength of the EHL has decreased further. Repeated prone extensions centralize complaints.

3. What is your next step? You are going to:

a. Request a surgical consult due to progressive neurological deficit.
b. Request diagnostic imaging tests.
c. Continue to treat the patient as before.
d. Continue to treat the patient, but now using non-weightbearing repeated extension.

The correct answer is **continue to treat, but use non-weightbearing exercises**. History and examination lead you to believe your initial diagnosis is correct. Helping with the move may have caused increased displacement of nuclear material, but the patient can still be made to centralize indicating the presence of an intact anular wall; the patient remains a good candidate for non-operative management. Reducing compressive forces allows for reduction of nuclear material no longer possible in weghtbearing positions.

You continue to see the patient once a week and you are able to progress the patient to weightbearing extension. Further evaluation shows negative PPIVM and PAIVM tests in the lumbar spine, but indicates segmental atrophy of the left L4-L5 multifidus muscle and you add lumbar stabilization exercises. Free of LBP the patient decides to go for a 3 hour cycle trip, followed by some yard work. Upon lifting a heavy bag of leaves the patient feels a "snap" in his back. The next day he presents with once again increased leg and back symptoms. The patient complains of numbness of the left side of the scrotum and difficulty initiating urine flow. Re-examination shows multisegmental neurologic bilateral deficit, left more than right. No movement centralizes complaints.

4. What do you do? You will:
a. Treat the patient with prone lumbar traction;
b. Suggest bedrest and re-examination in 1 week;
c. Refer the patient to the physician due to worsening of the neurological deficit.

The correct answer is **refer to physician**. Urinary retention and saddle anaesthesia combined are a strong indication of cauda equina syndrome, as is multisegmental, bilateral neurological deficit.[67,84] Because of potentially serious complications quick surgical decompression is indicated.[84]

Case scenario #2

A 50 year old man presents with left buttock, groin, and calf pain after missing a step and landing hard on his extended left leg. He complains of tingling in the left calf and bottom of the foot which increases upon walking. Provocation tests to the SIJ reproduce moderate buttock pain. The thigh thrust test also reproduces the groin pain. Positional tests reveal a high left iliac crest, equal PSIS bilateral, equal trochanter height, and higher ASIS left. Range on PPM and stability tests is decreased with a "jammed" endfeel. Trunk extension and left extension quadrant cause calf pain and tingling. Flexion and repeated flexion cause no pain except for some discomfort in the groin. Repeated extension and sidegliding cause no peripheralization or centralization. Gait reveals a left modified Trendelenburg (ipsilateral leaning of trunk over stance leg).
1.What causes the radicular symptoms?
a. Extravasation of inflammatory products from the SIJ to adjacent neural structures.
b. A lumbar diskogenic dysfunction compressing a nerve root.
c. Compensatory lumbar hypermobility/instability due to SIJ dysfunction.
d. Piriformis hypertonicity.

The correct answer is **compensatory lumbar hypermobility**. Extravasation of inflammatory products may irritate the neural structures and produce sciatic distribution complaints, but in this case the provocation tests do not lead us to believe that a massive inflammatory response is going on in the SIJ. A disk dysfunction is unlikely due to lack of a response to repeated movements. Setting up for the thigh thrust test stretches the piriformis and should have reproduced symptoms in case of a piriformis syndrome. SIJ hypomobility puts increased loads on the lumbosacral junction: pre-existing subclinical degenerative stenotic changes may cause nerve root compression with the forced increase in mobility. Based on the eval findings above we suspect a SIJ dysfunction.
2. Which dysfunction is present?
a. Pericapsular hypomobility with left innominate rotated posteriorly.
b. Pathomechanical hypomobility with left innominate rotated posteriorly.
c. Pathomechanical hypomobility, left innominate rotated posteriorly and translated superiorly.
d. Extra-articular hypomobility, left innominate rotated posteriorly and translated superiorly.

The correct answer is **pathomechanical hypomobility with left innominate rotated posteriorly and translated superiorly**. With greater trochanters equal, the higher iliac crest left most likely means a superiorly translated left innominate. The higher ASIS and the equal PSIS in combination with the superior innominate mean that the innominate is posteriorly translated. Stability (or PAM) tests are restricted excluding an extra-articular cause for hypomobility. The endfeel implicates a pathomechanical hypomobility.

After manipulation and stabilizing exercises to address the underlying deficient form closure, sacroiliac provocation tests, positional tests, AROM tests, PPM, and PAM tests return to normal. Radicular symptoms decrease. However, groin pain on the thigh thrust test and on endrange trunk flexion remain, as does the Trendelenburg gait left. Repeated and combined movement tests of the lumbar spine are negative. Lumbosacral posterior shear is increased on stability testing.
3. What is the most likely cause for the groin pain?
a. L5 radicular deficit causing gluteus medius weakness and excessive loading of the left hip;
b. Radiculopathy upper lumbar spine referring pain to groin area.
c. SIJ dysfunction with gluteal inhibition.
d. Hip dysfunction.

The correct answer is **hip dysfunction**. An L5 radicular deficit can cause gluteus medius weakness[86], but would likely cause other leg symptoms as well, especially in response to repeated and combined trunk movements. An upper lumbar radiculopathy could cause groin pain, but not gluteal weakness and should also respond to trunk movements. SIJ dysfunction can inhibit the gluteal muscles[91], but is unlikely after appropriate intervention and with said negative battery of tests. Endrange trunk flexion also involves endrange hip flexion. A thigh thrust test resembles a scouring test with hip flexion, adduction, and axial compression through the femur.[86] Gluteus medius weakness and the resultant increased compressive (peak) forces over the hip may play a role in degeneration. Further testing reveals a capsular pattern restriction of the left hip, decreased joint play with a hard capsular endfeel, and weakness of the left gluteus medius.
4. This hip restriction may have been causal or contributory to the SIJ and lumbar dysfunctions for all reasons EXCEPT:
a. Increased compressive forces left ZJs due to excessive right rotation with gait.
b. Decreased force closure SIJ due to decreased strength left gluteus medius.
c. Increased shear forces over left SIJ due to Trendelenburg gait on the left.
d. Increased compression loading left lumbosacral disk during ambulation.

The correct answer is **decreased force closure due to gluteus medius weakness**. Vleeming et al[172] found no change in tension or displacement of the TLF with simulated contraction of the gluteus medius. The other mechanisms have been discussed in monograph 11.2.3. in the section on the biomechanics of kinetic chain influences.

Case scenario #3

A 72 year old woman is referred for evaluation and treatment for left lower extremity pain of insidious onset which increases with standing and walking. Pain is relieved by recumbency, sitting, and pool aerobics. Her walking distance has been progressively reduced as a result of paresthesiae and pain involving the calves, the bottom of both feet, and the dorsum of the left foot. The patient notes decreased endurance in both legs when walking. The patient denies bowel, bladder, and sexual dysfunction. Her previous medical and family history are positive for unspecified heart disease. She reports an L4-L5 chemonucleolysis 10 years ago. Evaluation reveals a left structurally long leg, weak right quadriceps and weak left gluteus medius; LBP on AROM extension, bilateral sidebending, and extension quadrant. Flexion and return from flexion are full range and well coordinated. Repeated trunk movements have no effect, all other trunk movements are negative.The neurological examination, SIJ provocation tests, and PAIVM tests are negative. L4-S1 extension, sidebending, and rotation are reduced with a hard capsular endfeel on PPIVM testing. Both hips are restricted into extension, the left as part of a capsular pattern, the right as a result of adaptive shortening of psoas and quadriceps.
1. What is the most likely diagnosis?
a. Diskogenic dysfunction with left radiculopathy.
b. Diskogenic dysfunction with cauda equina syndrome.
c. Central stenotic syndrome.
d. Combined lateral and central stenotic syndrome.
e. Degenerative linear instability.

The correct answer is **combined stenotic syndrome**. Diskogenic dysfunctions are unlikely at this age due to the increased crosslinking and subsequent decreased mobility of the NP. Repeated trunk movements may not have an effect, even if a disk lesion would exist, due to a deficient hydrostatic mechanism, but prolonged extension (walking) and flexion (sitting) activities might: the flexion preference of this patient further discounts a disk problem. Cauda equina syndrome is discounted due to the lack of bowel, bladder, and sexual dysfunction, even though the multisegmental distribution of pain and paraesthesiae speaks in favor of cauda compression. Instability is discounted due to obvious good coordination with return from a flexed position and negative PAIVM tests. A previous medical history of chemonucleolysis with decreased nuclear volume reduces segment height and predisposes the motion segment to degeneration with central stenosis as the endresult. Compensatory rotation due hip restrictions may have contributed. The structurally longer left leg and weak gluteus medius predisposed the left side of the lumbar spine to degenerative changes and lateral stenosis, which may explain the predominantly left leg symptoms. Quadriceps weakness,

likely a sequela from the L4-L5 herniation 10 years ago, will manifest itself during terminal knee extension, and may contribute to a functionally short right leg.
2. We set up a treatment plan. The following interventions may be helpful, EXCEPT:
a. Strengthening left gluteus medius and right quadriceps.
b. Unloaded treadmill ambulation.
c. Extension mobilization L4-S1.
d. Joint mobilization left hip.
e. Stretching right hip flexor muscles.

The correct answer is **L4-S1 extension mobilization**. The strengthening exercises may decrease the functional aspects of leg length discrepancy. Unloaded treadmill ambulation may restore local muscular endurance. Joint mobilization and stretching about the hip will increase extension (and internal rotation) of the hips reducing rotation-extension demands on the lumbar spine. Restriction of L4-S1 motion is likely the result of degenerative narrowing of the segment rather than due to pericapsular hypomobility: mobilization will not result in increased range.

To exclude a contribution of intermittent vascular claudication, the PT has the patient do the bicycle test of Van Gelderen[86] and the 2-phase treadmill test.[127] The patient scores negative, excluding vascular claudication. After a 3 week intervention period characterized by improvement in symptoms and walking distance, the patient starts complaining of orthopnea necessitating semi-recumbent sleeping, increased leg and low back symptoms during increases in intra-abdominal pressure, and she develops hypertension. Trunk motions have minimal effects on the symptoms. A repeat bicycle test produces general fatigue and shortness of breath.
3. What is the likely cause of these symptoms?
a. Vascular stenosis of the abdominal aorta or more peripheral vessels.
b. Venous hypertension with distention epidural venous plexus.
c. Increased stenotic syndrome.

The correct answer is **venous hypertension**. Vascular stenosis is unlikely to come on this rapidly after initially negative tests. General rather than lower extremity symptoms with the bicycle test indicate congestive (right) heart failure rather than vascular stenosis, as does orthopnea. Musculoskeletal causes are unlikely as trunk motions do not affect symptoms. Venous hypertension can cause engorgement of the epidural venous plexus aggravating pre-existing stenotic changes. Referral to the physician is indicated.

Case scenario #44

A 52 year old female nurse complains of left buttock and groin pain, increased with sitting, standing, trunk flexion. The patient reports difficulty with extension in the morning. She notes no easing factors. Coughing, sneezing, and straining cause LBP and after a day of standing some left buttock pain. The patient notes decreased sensation in the left popliteal fossa. An MRI showed L4 to S1 disk disease. Flexion, extension, and left extension quadrant cause left-sided LBP. AROM left rotation and sidebending are restricted as compared to motions to the right. The slump test causes LBP, the SLR is negative. PPIVM testing revealed decreased L5-S1 extension, left L5-S1 sidebending, and right L5-S1 rotation with a hard capsular endfeel. Repeated flexion increased buttock pain, repeated endrange extension both in standing, and prone cause LBP. Hip joint tests are negative, but the thigh thrust test produces mild buttock and groin pain left.
1. What is the most likely diagnosis?
a. Left lumbosacral ZJ extension hypomobility.
b. Lumbosacral diskogenic dysfunction with S1 radiculopathy left.
c. A combination of the both previous diagnoses.
d. Left SIJ hypomobility.

The correct answer is a **combination of discogenic and zygapophysial dysfunction**. Radicular symptoms implicate a process which could cause radicular compression, as do provocation with flexion, repeated flexion, pain with increased abdominal pressure, and prolonged sitting or standing. Restriction of L5-S1 on PPIVM and left unilateral LBP with repeated endrange extension both weightbearing, and non-

weightbearing implicate a ZJ dysfunction. A moderately positive thigh thrust test is the only indication of SIJ problems.

Further SIJ evaluation reveals a soft endfeel and increased range on PPM and stability tests. Positional tests are normal. The segmental multifidus test[124] shows bilateral segmental inhibition of the lumbosacral multifidi. She performs the left supine SLR test[91] with some rotatory compensation from the trunk; the test produces some groin discomfort. Performance improves with an isometric left trunk rotation and manually applied compression through both innominates.

2. What is the most likely SIJ diagnosis?
a. Active and/or neural control subsystem deficiency lumbosacral and SIJ region.
b. Pathomechanical SIJ hypomobility dysfunction without innominate positional change.
c. Inflammatory condition SIJ joint with muscular reflex-inhibition.
d. SIJ combined subsystem deficiency.

The correct answer is **sacroiliac combined subsystem deficiency**. Hypomobility is excluded by findings of increased rather than decreased range and soft rather than hard or pathomechanical endfeel on PPM and stability testing. An inflammatory condition is unlikely due to moderately positive results on only one provocation test. Active and neural control subsystem deficiency is present, but based on PPM and stability test findings, so is passive subsystem deficiency.

Clinical impression is that lumbosacral ZJ hypomobility has contributed to both SIJ instability and lumbosacral diskogenic dysfunction due to decreased fluid exchange in the disk resulting from decreased segmental motion. Treatment priority is ZJ mobilization.

3. What is the most appropriate technique?
a. Right L5-S1 rotation manipulation with impulse from cranial to protect SIJ.
b. Specific contract-relax technique (CR) to increase L5-S1 extension and left sidebending.
c. Prone posteroanterior (P/A) grade II oscillations through the L5 spinous process.
d. Prone grade IV P/A through L5 spinous process.
e.CR followed by sustained endrange stretch extension-left sidebend L5-S1, and then again CR at the new endrange.

The last answer (E) is the correct answer. Rotational techniques may increase the underlying disk lesion. Only applying CR techniques may address a concomitant muscular restriction, but fails to address the peri-articular found on evaluation. Grade II oscillations do not provide the endrange needed for mobilization.[85] P/A techniques lack segmental specificity. The CR-endrange stretch-CR technique specifically mobilizes the lumbosacral restriction, and adresses issues of possible concomitant muscular restrictions, peri-articular restrictions, and neuromuscular re-education at endrange.[85]

Specific mobilization has decreased zygapophysial restrictions on PPIVM. Repeated extensions in standing still cause LBP, but prone extensions centralize buttock and groin pain to the central low back. The patient wants to return to work.

4. What is the best treatment approach to address residual problems?
A. Provide with lumbar flexion orthosis to allow for healing of joint, disk, and nerve root.
b. Provide with lumbar region belt to aid in lumbar stability.
c. A program of specific stabilizing exercises to increase SIJ and lumbar stability.
d. Said stabilization, prone extension exercises and a SIJ-belt.

The correct answer is **provide with stabilization and extension exercises and a sacroiliac belt**. A flat lumbar spine position will only cause failed SIJ self-locking, increasing demands on the neural control and active subsystems. A lumbar belt does not address the SIJ instability. Our goal is to increase SIJ force closure by all means available (exercise, SIJ-belt), as well as to address the contained diskogenic dysfunction, now a McKenzie protocol is possible with the ZJ extension restriction resolved.

Case scenario #5

An expectant mother at the end of her second trimester seeks out your help for bilateral LBP. When asked to indicate her pain she points at an area just caudal to both PSIS. Observation reveals an antalgic flat back posture. All trunk movements are extremely painful except for seated rotation. Positional tests for the

SIJ reveal no abnormalities, except for a posterior pelvic tilt. Neurologic and adverse neural tension tests are negative. Muscular hypertonicity limits lumbar PPIVM and PAIVM tests. The supine and prone SLR test cause sacral region pain bilateral, and clicking in either SIJ; pain is relieved by manual compression, but not by isometric contractions. Palpation just inferior to the PSIS is painful bilateral.
1. What is the most likely diagnosis?
a. Lumbar ZJ dysfunction.
b. Lumbar diskogenic dysfunction.
c. SIJ hypomobility dysfunction.
d. SIJ instability dysfunction.
e. Lumbar instability dysfunction.

The correct answer is **sacroiliac instability dysfunction**. We used to assume that pregnancy caused an increase in lordosis, but Snijders et al[147] showed that the lordosis in fact decreases. This, combined with hormonal effects, causes a failed SIJ self-locking. A flat back posture causes sacral counternutation, stressing the long dorsal SI ligaments resulting in palpatory pain just below the PSIS.[125] ZJ dysfunction is unlikely due to the decrease rather than increase in lordosis and the free rotation. Seated rotation introduces only minimal off-center forces in the SIJs unlike all other trunk motions. Symptoms do not support diskogenic dysfunction. Lumbar instability may be present, and in fact the flat back posture may serve to decrease anterior shear forces at an unstable low lumbar segment, but we are unable to confirm this. Pain, clicking, and relief of pain with manual compression in the SLR tests[91] implicate an unstable SIJ.

As isometric tests during the SLR tests did not provide relief, force closure was enhanced by the use of a SIJ pregnancy belt. The symptoms decreased. The patient returns to you 2 weeks post-partum with symptoms of bilateral groin and buttock pain and constant right leg pain in the calf and dorsal thigh. Walking greatly increases symptoms. All trunk motions cause LBP, with leg pain on flexion and left flexion quadrant. A neurologic examination reveals no abnormalities. The slump test and right SLR test reproduce leg pain. Repeated trunk movements do not affect the symptoms in a lasting manner. Lumbar PPIVM tests are normal. Simple SIJ provocation tests (compression and distraction) cause sacral region pain. The patient is unable to active raise either leg from a supine position. A symphysial provocation test is positive and an inferosuperior glide reveals hypermobility.[91]
2.. What is the most likely diagnosis?
a. Lumbar diskogenic dysfunction with right radiculopathy.
b. Symphysial instability.
c. Symphysial and SIJ instability.
d. Lumbar ZJ flexion hypomobility.

The correct answer is **symphysial and sacroiliac instability**. No effect on repeated movement testing and PPIVM makes lumbar dysfunction unlikely. Natural child birth may cause symphysial dissociation and hypermobility.[148] The symphysial provocation and mobility tests confirm this dysfunction. A painful symphysis is unloaded by sacral counternutation as occurs in a flat back posture.[125] This causes failed self-locking and SIJ instability.[125] Some authors state symphysial instability can not occur without concomitant SIJ instability.[126] Repetitive microtrauma or trauma induced by child birth may cause an inflammatory SIJ condition; extravasation of inflammatory products may cause what in this case appear to be a chemically irritated neural structures with constant pain.[83]

Intervention includes appropriate anti-inflammatory medications prescribed by the patient's physician, anti-inflammatory modalities applied by the patient (cold packs) and the therapist (pulsed ultrasound), decrease of torsional stresses to pelvic region (decreased walking, trunk motions involving sidebending, pelvic belt and tight spandex pants) and gentle stabilization exercises. Symptoms of leg pain decrease over the next 4 weeks. The patient continues to complain of pain with walking, especially longer steps. A posteriorly rotated right innominate with a superior innominate is treated with manipulation, mobilization, and stretching, but the patient continues to present with the same recurrent positional fault every treatment. She now also complains of thoracolumbar pain and dysesthesia in the right buttock. AROM tests of the trunk reveal thoracolumbar pain on flexion, extension, and right rotation. Repeated motion tests are negative, as is a neurological examination of both legs. PPIVM reveals restrictions in extension and right rotation at T12-L2. Palpation of the superior cluneal nerve is painful where it passes the right iliac crest.

3. What is most likely maintaining the pelvic dysfunctions?
a. S1 radiculopathy causing gluteus maximus weakness and loss of force closure.
b. L5 radiculopathy with gluteus medius weakness causing loss of force closure.
c. Right hamstrings hypertonicity caused by lumbosacral dysfunction and segmental facilitation.
d. T12-L2 ZJ dysfunction and segmental facilitation.

The correct answer is **T12-L2 dysfunction**. No evidence of L5 or S1 involvement was found explaining gluteal or hamstrings involvement. Gluteus medius does not contribute to force closure.[172] Thoracolumbar dysfunction can cause symptoms in the buttock region by way of the superior cluneal nerves.[26] It can also cause segmental facilitation and hypertonicity of the pyramidalis muscle which may maintain a pelvic dysfunction.[91] Treatment of the symptomatic thoracolumbar segments may allow for successful intervention in the pelvic region.

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