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Leg length discrepancy and osteoarthritis in the knee, hip and lumbar spine

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Osteoarthritis (OA) is an extremely common condition that creates substantial personal and health care costs. An important recognised risk factor for OA is excessive or abnormal mechanical joint loading. Leg length discrepancy (LLD) is a common condition that results in uneven and excessive loading of not only knee joints but also hip joints and lumbar motion segments. Accurate imaging methods of LLD have made it possible to study the biomechanical effects of mild LLD (LLD of 20mm or less). This review examines the accuracy of these methods compared to clinical LLD measurements. It then examines the association between LLD and OA of the joints of the lower extremity. More importantly, it addresses the largely neglected association between LLD and degeneration of lumbar motion segments and the patterns of biomechanical changes that accompany LLD. We propose that mild LLD may be an important instigator or contributor to OA of the hip and lumbar

L'arthrose est une pathologie extrêmement fréquente qui engendre des frais personnels et des coûts de soins de santé importants. Un facteur important de risque reconnu pour l'arthrose est la charge mécanique excessive ou anormale sur les articulations. L'inégalité de longueur des membres inférieurs (ILMI) est une affection fréquente qui se traduit par une charge inégale et excessive non seulement sur les articulations du genou, mais aussi sur les articulations de la hanche et les segments mobiles lombaires. Des méthodes d'imagerie précises de l'ILMI ont permis d'étudier les effets biomécaniques d'une ILMI légère (ILMI de 20 mm ou moins). Cette étude examine l'exactitude de ces méthodes par rapport aux mesures cliniques de l'ILMI. Elle se penche ensuite sur l'association entre l'ILMI et l'arthrose des articulations des membres inférieurs. Mais surtout, elle examine l'association peu étudiée entre l'ILMI et la dégénérescence de segments mobiles lombaires et les tendances des changements biomécaniques qui accompagnent l'ILMI. Nous suggérons que l'ILMI légère peut être un instigateur important ou un facteur de l'arthrose de la hanche et de la colonne lombaire, et qu'elle mérite d'être étudiée

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spine, and that it deserves to be rigorously studied in order to decrease OA's burden of disease.

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MOTS CLÉS : chiropratique, inégalité de longueur des membres inférieurs, arthrose, genou, hanche, colonne lombaire

Introduction

Musculoskeletal diseases, including osteoarthritis (OA), constitute a substantial economic burden to the community and are the most common causes of chronic pain and disability.[1] Chronic joint pain, such as degeneration of the knee, hip, and lumbar spine, affects an estimated 22% of the Australian population at any one point.[1] Similarly, the monthly prevalence of joint pain in the United States of America has been estimated at 30.7% in the general population.[2] Furthermore, musculoskeletal disease is the most common cause of chronic pain in Australia accounting for 26% of all reported chronic pain cases at any point in time.[1] Given that chronic joint pain is linked to aging, this problem is expected to become even more burdensome as the population of industrialised countries ages.[1] There is limitation of daily activities in a large proportion (43.3% – 57.9%) of people suffering from chronic joint pain.[2] In fact, low back pain, in particular, is the most common cause of long-term disability in industrialised countries.[3] In 2001 in Australia, the yearly direct and indirect costs of back pain were estimated at AUD 1.02 billion and AUD 8.15 billion respectively.[4] Similarly, in the United States in 2008, the combined total cost of all back pain cases was estimated at USD 624.8 billion.[5]

In terms of the spine, moderate to severe OA and degeneration of the intervertebral disc are commonly associated with chronic low back pain.[6,7] There is further evidence that significant disc degeneration, at least in the elderly, is associated with a twofold increase in chronic low back pain status.[6] However, the effects of OA on the spine are not confined to pain. For instance, Iguchi and co-workers, among others, have defined radiological criteria for segmental spinal instability as a result of advanced OA that would require surgical immobilization.[7]

A number of risk factors have been identified for OA including abnormal or excessive mechanical joint loading[8-14] as occurs with lower extremity joints or spinal discs in obesity[15,16], and with excessive occupational standing or lifting[17]. It is also widely believed that abnormal joint loading plays a major role in the development of adjacent segment degeneration following surgical fusion of a spinal motion segment.[18] Leg length discrepancy (LLD) is a very common condition that involves abnormal loading of the lower extremity and lumbar joints.[19] LLD, where one femoral head is lower than the contralateral side in the standing position, can be due to: anatomical differences in lengths of bones of the lower extremities (anatomical LLD); or functional differences in the tone of lower extremity muscles or abnormalities of joint function (functional LLD).[20] Approximately 59% of the population is affected by LLD of 5mm or more.[21] However, in 99.9% of cases LLD can be classified as mild since it falls below 20mm.[21]

Leg length discrepancy (LLD) affects up to 90% of the general population with a mean discrepancy of 5.2 mm[21,22] that involves abnormal patterns of weight bearing in the joints of the lower extremities and the spine. There is a range of studies demonstrating that LLD is associated with postural and functional changes in the lower limbs, the pelvis, and the spine. These studies document the role of LLD in: scoliosis[23]; OA of the lumbar spinal joints[10]; low back pain[20,24]; OA of the hip[8,25]; OA of the knee[2,12,13]; stress fractures in the metatarsals tibia and femur[26]; and gait disturbance[27,28]. However, the degree of LLD required to cause, or contribute to, a musculoskeletal disorder remains controversial. Some authors hold the view that LLD of less than 20 mm is clinically insignificant.[29,30] However, others suggest that LLD of less than this magnitude is of clinical significance.[13,22,31-33] It is possible that LLD can, over time, lead or contribute to the development

of OA in the lumbar spine. Understanding the contribution of abnormal joint weight bearing, as occurs in LLD, to the development of OA and disc degeneration could allow for more effective preventive strategies for OA, at least in this population of patients. This review explores the current evidence for an association between LLD, particularly mild LLD (LLD of ≤20mm), on one hand and OA of the joints of the lower extremity, the lumbar facet joints and intervertebral discs on the other. It also examines if LLD is associated with a predictable pattern of degenerative change in the joints of the lower limb and lumbar spine. Literature searches were conducted using the PubMed database as well as Scopus and Index to Chiropractic Literature. Care was taken to avoid a selection bias. However, studies that were deemed low quality, according to standard quality criteria, were excluded.

Definition of Leg Length Discrepancy (LLD)

Leg length discrepancy (LLD), also known as short leg syndrome, leg length deficiency, leg length insufficiency or anisomelia, is classified as either anatomical (also called structural) or functional.[20,34,35] Anatomical or structural LLD is caused by side-to-side differences in lower limb length, due to actual bony asymmetry existing between the level of the femur head and the calcaneus. Functional LLD is defined as LLD that is due to biomechanical abnormalities of joint function in the lower limbs.[28,36,37] For example, unilateral pronation may cause an apparent shortening of the leg. A third type is often described as an environmental LLD and is common in runners who run on a sloping or a slightly banked surface in one direction, and for long periods of time.[34] This will be considered as a type of functional LLD for the purposes of this review. Anatomical LLD can be congenital or acquired. Congenital causes include phocomelia and dysgenetic syndromes. Acquired aetiologies include: dysplasias, Ollier's disease, slipped epiphysis: poliomyelitis; neurofibromatosis; septic arthritis; osteomyelitis; fractures; pes planus; knee valgus/varus and dislocation; and also surgically induced.[38] In the case of an anatomical LLD, there may often be compensation by functional adaptation on the contralateral side, in an attempt to cause the shortening of the long leg. One example of this being pronation of the foot on the side of the long leg, which results in an anatomical LLD on one side and essentially a functional LLD on the other.[28]

LLD can exist from childhood or it can develop in adult life.[20,39] In addition, functional activities of the individual play a role in determining whether LLD becomes symptomatic. Athletes may suffer complications such as patella tracking disorder or trochanteric bursitis in cases of unilateral foot pronation with a much smaller degree of LLD than non-athletes. Subotnick[28] proposed that 18 mm of LLD in a non-athlete equates to 6 mm of LLD in an athlete since during running approximately three times the body weight is transmitted through the supporting limb compared to walking. Additionally, Friberg has demonstrated using his rigorous method of measuring LLD, a positive correlation between the degree of LLD and incidence of stress fractures in 547 Finish Army conscripts involved in very strenuous training.[26] In fact 89% of these fractures occurred in those with an LLD of greater than 3mm. Of the 130 stress fractures in this study, unilateral fractures occurred in the tibia, metatarsals and femur in 73% and 16% of cases in the longer or shorter leg respectively. Bilateral fractures occurred in subjects with equal leg length or LLD not exceeding 3mm. It is important to note that stress fractures in these army conscripts with LLD occurred predominantly on the side of the longer leg. From a clinical biomechanical perspective, this is consistent with the longer leg being under increased mechanical stress during strenuous physical activities associated with military training.

Measurement/Assessment of LLD

Clinical Methods of Assessment

Valid and reliable measurement of LLD has been an area of considerable controversy. There is a range of clinical methods of measuring LLD that suffer from inaccuracy and poor inter- and intra-examiner reliability.[40-45] These include the so called 'direct' and 'indirect' methods of clinical assessment. An example of the direct method uses a tape measure to determine the distance between the anterior superior iliac spine (ASIS) and the medial malleolus with the subject lying supine. The indirect method of assessment of LLD relies on palpating the levelness of the iliac crests to determine lateral pelvic inclination and then placing boards of known thickness under the perceived shorter leg until the iliac crests are thought to be level. Several variations of the direct method of measurement have been described and include measuring from

the anterior iliac spine to the lateral or medial malleolus, from the umbilicus to the medial malleolus, and from the xiphosternum to the medial malleolus.

Direct and indirect measurement of LLD relies on the palpation of bony landmarks, which is prone to error. For example, assessment may fail to detect iliac asymmetries that may mask or accentuate LLD. Furthermore, measurement may be affected by asymmetry in the position of the umbilicus, or affected by unilateral deviations of the long axis of the limb (e.g. genu valgus). These measurements also fail to include the floor to malleolus distance and therefore entirely ignore the significant effects of foot posture in upright stance. Indirect methods may be only slightly more reliable. Clark compared radiographic evaluation to clinical assessment using iliac palpation and found two examiners agreed to within 5 mm in only 16 out of 50 subjects.[45] Fisk and Baigent compared the iliac palpation and block correction method to radiographic measurement in 107 subjects.[42] They also found that the clinical assessments of the examiners were incorrect by greater than 5mm in 29% of subjects. In a clinical situation, palpation of pelvic landmarks with block correction has the strongest support[46,47], with tape measurement methods found to have the weakest reliability[46]. Chiropractors routinely assess LLD using visual analysis with the patient prone or supine and compare medial malleolus, sole-heel interface or bottom of the shoes for relative position. The finding may be used in determining the exact biomechanical treatment given. Often a post-treatment re-check is performed to reassess LLD and if leg lengths become even then it is assumed that any pelvic or spinal imbalances have been corrected. Otherwise an anatomical LLD is suspected. A thorough literature review on the research data available on this method of assessment is available.[37] The finding of that review was that questionable methodologies and statistical analyses used in these studies meant that there was no convincing evidence on the validity of the quick visual leg length assessment.

A number of small studies have reported a high degree of inter- and intra-examiner reliability in terms of detection of the side of the shorter leg by clinical assessments of LLD using an antigravity position (prone or supine) in primarily asymptomatic volunteers[48-52], while detection of the magnitude of LLD using these methods has been found to have lower levels of inter-examiner reliability[46,48,53]. Caveats with these small studies include methodological errors that seriously compromise these findings, the use of asymptomatic participants, utilization of methods that are not commonly practised in the clinical setting[49,50,54], and the lack of comparison to a radiographic assessment to demonstrate validity. Other authors have found clinical methods of measurement to be of low reliability.[40,45] Crude clinical methods of LLD detection have complicated the effort to define the clinical significance and biomechanical effects of LLD.

Imaging Methods of Assessment

Most studies, particularly in early years of LLD research, employed clinical methods of measurement. More rigorous studies have used imaging methods of measurement that enjoy higher levels of validity and reliability.[20] Four different imaging methods have been used for detection and quantification of LLD: teleroentgenography; computed tomography; slit scanography; and orthoroentgenography.[20,35] A teleroentgenogram is a single anterior – posterior exposure of the standing subject, imaging the entire lower limbs, that also includes a measuring instrument, such as a ruler. Limitations of teleroentgenography include hip and knee joint flexion contractures, and given the size of the image, magnification errors that can give a false reading.[55] Computed tomography has not been found to be any more accurate than plain radiography in detecting LLD unless the patient has hip or knee joint contractures[55], and increases radiation dose. Slit scanography, relies on a method which uses a lead diaphragm placed over the x-ray tube containing a slit, while the tube is moved along the long axis of the lower extremity during the exposure with the subject lying supine. However, as neither CT nor slit scenography are done under weight-bearing conditions, they do not amount to a postural analysis of LLD. Orthoroentgenography uses separate exposures of hips, knees and ankles in an attempt to avoid the magnification error in teleroentgenography. However, errors can still be generated due to patient movement and joint contractures. Friberg's[24,56] variation of orthoroentgenography uses a single anterior-posterior lumbo-pelvic exposure allowing for comparison of the heights of femoral heads. Friberg's method, has been found to be accurate, reproducible, and affords the advantage of lower patient radiation dose[24], and has since been widely used to study LLD. Standing antero-posterior (A-P) radiographs of the pelvis are con-

sidered an acceptably accurate and reliable method for assessing true LLD. In fact the reliability of Friberg's method has been reported to have a mean error of 0.6 mm and a range of 0 to 2.0 mm on repeated imaging.[40] Similarly, Clark and co-workers found plain radiography to be accurate, within 3 mm, for both functional and anatomical LLD.[45] Similarly, the radiographic method advocated by Giles and Taylor, involves placing the feet in line with the femoral heads in AP lumbo-pelvic X-rays which produces a mean error of only 1.12 mm.[31]

In a landmark study, Friberg and co-workers compared the inter- and intra-examiner reliability of LLD detection between radiographic methods and clinical methods using 21 subjects.[40] They demonstrated a wide variance in LLD measurements with 88% of clinical measurements being erroneous, and overestimations by as much as 20mm. In 12% of direct and 13.4% of indirect clinical measurements, the observers failed to detect the short leg even when the radiologically assessed measurement was as much as 25mm. Repeat measurements taken three months later, showed significant disagreement in 28% of cases. Similarly, Woerman and Binder-Macleod compared direct clinical methods for evaluating LLD with radiographic assessments.[46] Using a tape measure from: the ASIS to lateral malleolus had a mean error of 6.0 +/- 16mm; the umbilicus to medial malleolus had a mean error of 4.2+/-9.9mm; ASIS to medial malleolus had a mean error of 7.3+/-10.1mm; and from xiphosternum to medial malleolus had a mean error of 10.9+/-16.2 mm. Therefore, it is clear that clinical methods of measurement of LLD should be abandoned in LLD research in favour of the radiographic gold standard.

Recently, Krettek and colleagues have reported an ultrasound method for LLD measurement with error margins of less than 1mm when compared to the radiographic gold standard.[57] More recently, Rannisto and co-workers examined the accuracy of LASER-Ultrasound measurement of LLD as compared to the radiographic gold standard.[58] They reported almost perfect agreement between these methods, with interclass correlation co-efficient (ICC) for agreement of 0.97 (95% CI of 0.93-0.99). They also reported excellent levels of intra- and inter-examiner reliability for this method. These studies strongly suggest that LASER-Ultrasound may be a valid and reliable alternative to radiography in measurement of LLD, while also affording the advantage of being non-invasive. Thus, this method lends itself to a wide variety of study designs including large-scale population studies of mild LLD and its association with OA over time.

Clinically Significant LLD

The degree of LLD required to reach clinical significance has also remained controversial. However, clinical significance may be context-dependent. In the context of surgical treatment, most surgeons have advocated that LLD of less than 20 mm is clinically insignificant, as no surgery is indicated.[59] However, other authors investigating the functional effects of LLD of 3 to 20 mm have discovered clinical significance in the context of prevention of stress fractures, chondromalacia patellae, and osteoarthritis in the joints of the lower extremity. Subotnic suggests that LLD of just over 6 mm, which may be asymptomatic during walking, is sufficient to cause chronic repetitive overuse injuries such as chondromalacia patellae on the short leg side in runners.[28] LLD has been associated with many lower limb and lumbar biomechanical conditions including: foot pronation[28]; low back pain[60]; scoliosis[39] and osteoarthritis in the knee and hip joints[2,11,13,32].

A retrospective study of the radiographs of 106 chiropractic patients reported that more than half of those with LLD of 6 mm or more also had scoliosis or an abnormal degree of lumbar lordosis, indicating abnormal weight bearing in the joints of the lumbar spine.[61] However, this study did not compare this incidence of postural abnormalities with that of the general population, which compromises one's ability to link LLD and postural asymmetries. In addition, Giles and Taylor using 1,309 subjects with (and 50 volunteers without) chronic low back pain found that 18.3% of chronic low back pain sufferers had LLD of 10 mm or more compared to 8% of controls.[31] More importantly, a subsequent study by the same authors reported that subjects with LLD of greater than 9 mm had significantly altered lumbosacral facet joint angles compared to subjects with LLD of less than 3 mm.[10] This suggested that the joint loading abnormalities associated with LLD might affect the development of facet joints. Moreover, Cummings studied the effect of varying increases in leg length in healthy college women and noted that posterior innominate rotation occurs on the side of the lengthened limb and anterior innominate rotation occurs on the side of the shorter limb and a concomitant pelvic obliquity occurs in an almost linear relationship

from 6mm through to 22 mm.[62] Taken together these studies suggest that LLD is associated with abnormal or asymmetrical loading of lower extremity and spinal joints, which may well be significant in the context of OA.

However, there are studies suggesting that mild LLD is clinically insignificant. These include a 1975 study by Fisk and Baigent[42] that suggested that moderate LLD had little or no involvement in causation of back pain. Similarly, Hoikka radiographed 100 chronic low back pain subjects with a mean age of 40 and a mean LLD of 5 mm (+/-3 mm) and reported that while LLD correlated well with iliac crest tilt and lateral sacral tilt, its correlation with the degree and direction of scoliosis was poor.[63]

LLD and OA in the Lower Extremity and the Lumbar Spine

OA is characterised by degeneration of articular cartilage, hypertrophy of bone at the joint margins, and thickening of synovial membrane.[8] In advanced stages the surfaces of articulating bones, where cartilage has deteriorated, become significantly deformed. Osteoarthritis may be categorised as primary, often referred to as idiopathic, or secondary to other pathology.[8] Secondary OA follows a precipitating event such as fracture or dislocation, or disease such as Perthe's disease, or developmental abnormality such as scoliosis. Solomon questioned the existence of idiopathic OA after finding signs of trauma or other pathology in the hip joint in all of the 327 cases of OA of the hip that he examined.[8] On the basis of these findings, he proposed three pathogenic groups of secondary arthritis: abnormal or incongruous loads causing failure of essentially normal cartilage; cartilage breaking up under normal conditions of loading due to damage or defective cartilage, defective subchondral bone causing break-up of articular cartilage. However, the cause-effect relationships in the associations that Solomon observed remain unclear. Nevertheless, pelvic tilt or torsion resulting from LLD may place unequal stresses on the foot, ankle, knee, hip, sacro-iliac, and lumbar spinal joints in the upright posture. Tilting of the pelvis shifts the centre of gravity, resulting in compensatory muscle activity, which may increase the magnitude of internal joint load. Pelvic tilt may also reduce the contact area of articular cartilage within the joint due to a disruption of normal skeletal alignment. These two effects, of increased joint loading and reduced articulating joint surface area, may translate to increased pressure on the cartilage and the underlying bone thereby leading to the development of osteoarthritis.[64]

LLD and OA in the Knee and the Hip Joints

A large population study of 926 participants by Golightly and colleagues[12] found a positive association between LLD of greater than or equal to 20 mm and knee osteoarthritis, in an African American and Caucasian general population sample in North Carolina, USA. In adjusted models for covariates including gender, race, age, knee injury/surgery, hip pathology, BMI, and height, radiographic knee OA was 80% higher in participants with LLD. However, this study suffers from several limitations. For instance, whilst radiographic examination was used for OA of the knee, a tape measure was used to determine the presence and magnitude of LLD. In addition, the distance between the malleolus to the floor was not measured and a 20mm threshold was used for LLD. These factors may have distorted or underestimated a significant relationship between LLD and knee and hip OA.

In concert with this notion a recent large prospective study, by Harvey and colleagues[28] using radiographic LLD measurements in 3,026 subjects aged 50 to 79, found that LLD of 5 mm was associated with an increase in prevalent symptomatic and progressive osteoarthritis of the knee. This large cohort study involved a follow up of 30 months, and was the first prospective study to define mild LLD as a risk factor for knee osteoarthritis. Subotnick completed a survey over a six-year period of athlete patients attending his office, and found that approximately 40% of his 4,000 patients suffered from some form of LLD. He reported that in most of these cases of LLD, sufferers externally rotated the short leg, which caused excessive medial strain on the entire limb leading to: overuse knee injuries; chondromalacia patella; greater trochanteric bursitis; iliotibial band strain; flexor group shin splints or anterior tibial shin splints; medial ankle synovitis; posterior tibial tendonitis; and medial plantar fasciitis.[28] Of equal importance are reports suggesting that LLD or altered weight bearing of the hip joint[8,11,32] is a potential contributing factor to OA in the hip. For instance, a report of 100 consecutive patients immediately prior to hip arthroplasty found that in LLD, hip OA was 84% more common on the side of the longer limb.[32]

LLD and Degeneration of the Lumbar Intervertebral Disc

As an avascular structure, the intervertebral disc derives its nutrients by diffusion from the end plates.[65] Whether sustained or abnormal mechanical load interfered with this diffusion in vivo had remained unknown until recently. To answer this question, Arun and co-workers simulated the effects of upright posture on all five lumbar intervertebral discs in 8 volunteers in sitting and standing positions and employed contrast magnetic resonance imaging (MRI). The volunteers' spines were loaded in a sustained fashion with 50% of body weight in the supine position for 4.5 hours, and MRI scans were done at 1.5, 3 and 4-hour time points as well as 2 and 3.5 hours following the end of loading. The investigators found that this amount of sustained creep loading reduced the transport of small solutes into the centre of the human intervertebral disc. Furthermore, this study found that it took 3 hours for levels of diffusion of small solutes to reach pre-loading levels. These findings support the notion that sustained mechanical loading may predispose the intervertebral disc to degeneration by impairing the diffusion of nutrients entering the disc and metabolites exiting the disc. These findings also support earlier work by Buckwalter, showing that one of the main causes of disc degeneration is reduced nutrition of the disc, in particular the nucleus pulposis which becomes fibrotic and leads to a reduction in disc height and annulus fibrosis fragmentation especially posteriorly.[66] A degenerated disc displays properties of being solid, while healthy discs have more fluid properties.[67] It has also been shown by Adams and colleagues that age-related degeneration in the lumbar intervertebral discs compromises the weight-bearing capacity of the nucleus pulposis by 50%, and substantially increases the stress on the annulus fibrosis. They also found that the posterior aspect of the annulus was more affected than the anterior annulus, and that degeneration had a greater effect on intradiscal stresses than ageing.[68]

Similarly, Sato and colleagues measured intra-discal pressure in vertical and horizontal positions in 28 subjects with either ongoing lower back pain, sciatica or both, and 8 healthy controls using advanced pressure sensors placed into the L4-L5 disc.[69] They found that intra-discal pressure significantly changed in negative correlation with MRI-demonstrated disc degeneration. Additionally, Adams and Hutten examined the effect of sustained load on lumbar discs and facet joints using eighteen cadaveric lumbar spines.[70] They found that the discs took most of the compressive loads in all postures. However, after about three hours of compressive loading at a level equivalent to standing, the joints lost approximately 9% of their height, causing the apophyseal joints to bear approximately 16% of the compressive load compared to zero in the equivalent of an unsupported sitting position. Moreover, in four severely degenerated discs in this study, large proportions of the load were transferred to the apophyseal joints.

Furthermore, in a comprehensive and rigorous recent study, Rajasekaran and colleagues used contrast MRI for direct examination of the effects of dynamic and static weight bearing on diffusion of nutrients into 21 IVDs in 6 adolescent idiopathic scoliosis patients prior to surgery.[71] They also assessed cell viability in nucleus pulposis biopsy material taken from the convex and the concave regions of the disc, which corresponded to regions where the disc was stretched and compressed respectively. These findings were then correlated with histopathological and biochemical analyses. These investigators found that all discs and end plates were damaged by asymmetrical pressure, regardless of location or severity and showed affected diffusion patterns through the endplate. A subsequent study by the same group found that: end plate junction failure preceded disc herniation; the nucleus pulposis tended to migrate to the convex side of the curve; and compression as well as tension was damaging to the end plate as well the disc.[72] Taken together, these studies strongly support asymmetrical joint loading of the spinal motion segment as a mechanism for intervertebral disc (IVD) degeneration.

It is important to note that there is recent evidence suggesting that age-related degeneration, disc prolapse and OA may be inter-related but different entities. For instance, a recent study by Kanna suggests that patients with multi- or pan-lumbar degeneration are a different patient group to single-level disc prolapse and degeneration patients.[73] Disc prolapse and resultant degeneration has been shown to be accompanied by end plate avulsion and failure, particularly following injury involving combined flexion and torsion forces affecting the lumbar spine.[74] It is also becoming clear that disc degenerative disease (without herniation or prolapse) may be a result of end plate failure through sustained loading affecting diffusion of nutrients.[71,75]

LLD and OA in the Facet Joints

The function of the facet joints (also known as zygapophyseal or apophyseal joints) is to control and guide spinal movements prevent forward displacement of vertebrae and, in the lumbar region, inhibit sideways movement.[76] The human erect posture creates a lordosis in the lumbar spine that causes the lower lumbar joints to be subjected to a sheering force even in the relaxed upright posture. The sacral articular processes resist the sheering force that attempts to displace the L5 segment anteriorly.

In addition to the IVD, the facet joints of the spine can be loaded abnormally as a result of LLD. Giles and Taylor[10], showed that when the intervertebral disc degenerates, the zygapophyseal joints also frequently display degenerative changes. Biomechanical studies have shown that during combined compression and bending, zygapophyseal joints carry from 12% to 16% of the total load.[70] This load on the zygapophyseal joints is known to increase up to 70% when the intervertebral disc height is reduced.[70] At the L5-S1 level, there are also significant shear forces in correlation to the sagittal angle of L5 upon S1. Hicks and associates, in a study of OA-related chronic low back pain, reported minimal facet joint degeneration in the upper lumber region followed by a steep rise in the prevalence of facet joint OA at the lower levels with the greatest change at L5-S1.[6] They also found that facet joint degeneration typically appeared in the lumbo-sacral spine prior to the fourth decade of life and continued to increase until the age of 60+ when it became extremely prominent. Therefore, both increased or abnormal loads and ageing seem to increase the likelihood of OA in the facet joints. In this context, it is important to note that there is good evidence for a link between LLD and facet tropism at the L5/S1 level.[77]

It is also important to note that OA-related changes in zygapophysial joint hyaline articular cartilage may be different from age-related changes.[78] Unlike ageing, in OA the hyaline cartilage often develops areas of disintegration and erosion, even early in life. OA results in diffuse degradation and repair rather than general thinning as found in aging. In addition, in OA the water content of hyaline articular cartilage is normal or increased whereas in aging the water content is reduced.[78]

Patterns of skeletal asymmetry due to LLD

There is a general consensus that LLD has postural consequences. These include torsional changes in pelvic posture with posterior rotation of the ilium on the longer leg side and anterior rotation of the ilium on the short leg side, relative to the contralateral ilium, in both anatomical and artificially induced LLD.[62,79] Clearly, LLD also causes a lateral tilt of the pelvis, consequent to which a functional lumbar rotatory scoliosis can develop with the convexity usually found on the short leg side. Although one investigator described the curve as being convex toward the side of the long leg.[63] These effects are said to be common to both functional and anatomical LLD.[46] Whilst not universally accepted, some authors believe functional scoliosis may become structural over time.[24,28,77] These adaptations may be the cause of permanent spinal changes such as asymmetrical facet joint angles, disc degeneration, osteophytic spurs, facet joint OA, disc herniation, muscle imbalances and scoliosis.

There are reports of a strong association between asymmetrical disc degeneration and degenerative lumbar scoliosis.[80,81] However, in children with LLD-related functional scoliosis, a small LLD is often asymptomatic and hence may be ignored by clinicians.[23] Nevertheless, LLD may cause an increase of mechanical load on the foot on the side of the longer leg by up to 6% of body weight.[23] Importantly, using an internal shoe lift, these investigators reported that the functional scoliosis could be eliminated.[23] Even though Hoikka and colleagues have reported a poor correlation between scoliosis and mild LLD[63], many other authors have reported a significant correlation between the two[24,36,39,60,63,82].

Many investigators have examined the effects of artificial (experimentally-induced) LLD on pelvic torsion and scoliosis. Young and colleagues artificially increased the leg length on one side in 29 healthy young adults and reported that this produced a contralateral innominate rotation anteriorly and a posterior rotation on the ipsilateral innominate.[83] Also lateral flexion increased towards the side of the lift. This assessment was based on an abrupt induction of LLD that does not allow for postural compensations over time. Similarly, Betsch and colleagues assessed the effects of simulated LLD on spinal posture and pelvic position using dynamic rastersterographic analysis in 115 volunteers and found a significant correlation with pelvic tilt, torsion and scoliosis.[84] Similarly, Timgren and colleagues found that 87% of 150 consecutive neurologic patients presenting to a physiatrist had LLD with asym-

metry of the pelvis and spine.[85] They described two types of scoliosis in these patients; a) an S-shaped scoliosis associated with an elevation of the iliac crest and the ipsilateral scapula, and b) a c-shaped scoliosis associated with an elevation of the iliac crest and the contralateral scapula. These shapes represented approximately equal proportions of patients. The patients with the c-shaped scoliosis exhibited apparent leg lengthening on the side of the elevated crest, and the s-shaped patients showed a shortening of the leg on the side of the elevated crest. Taken as a whole, these studies demonstrate that the postural changes induced by LLD may be complex and may depend on the magnitude of LLD and the compensatory mechanisms operative in particular individuals. These compensatory mechanisms may include: asymmetrical foot pronation/supination, genu valgus, knee degeneration, alterations to spinal kinematics and gait disturbances.

Conclusion

Much of the LLD literature is compromised by the use of invalid and unreliable clinical methods of LLD quantification. This has largely been the cause of the controversy that still exists about the clinical significance of mild LLD. However, the literature that is based on the gold standard of radiographic assessment has allowed elucidation of the subtle postural effects of mild LLD and their consequences in terms of excessive and abnormal loading of lower extremity and at least lumbar spinal joints. Given that excessive weight bearing is a known predisposing factor in OA, this may well have implications in the development of OA in these joints. Clinically, standing radiographic assessment would be an indispensable tool in accurately assessing LLD, if it is clinically suspected. The introduction of accurate ultrasound methods of LLD detection also has promising clinical applications particularly in children and adolescents, in terms of early LLD management and OA prevention. In addition, there is a significant body of literature linking LLD and knee OA, and to a lesser extent hip OA. However, there is little research attention that has been paid to date to the relationship between mild LLD and OA of the lumbar facet joints or lumbar disc degeneration. This relationship needs to be more thoroughly investigated. This effort will ideally involve long-term population studies to properly establish and quantify the impact of mild LLD on OA in terms of a cause-effect relationship. It will also need to be rigorously studied in subpopulations such as athletes in an effort to improve sports performance and prevent injuries. Furthermore, this link needs to be interrogated in adolescent populations to possibly prevent the development of OA later in life. If this cause-effect relationship is established by further research, interventional studies using heel-lifts, shoe lifts or other orthoses could be carried out to assess the value of this relatively simple and inexpensive measure in reducing OA's burden of disease. This is a promising area of clinical research that may well have important public health implications.

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