



## Review

## Leg length discrepancy

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**Abstract**

The role of leg length discrepancy (LLD) both as a biomechanical impediment and a predisposing factor for associated musculoskeletal disorders has been a source of controversy for some time. LLD has been implicated in affecting gait and running mechanics and economy, standing posture, postural sway, as well as increased incidence of scoliosis, low back pain, osteoarthritis of the hip and spine, aseptic loosening of hip prosthesis, and lower extremity stress fractures. Authors disagree on the extent (if any) to which LLD causes these problems, and what magnitude of LLD is necessary to generate these problems. This paper represents an overview of the classification and etiology of LLD, the controversy of several measurement and treatment protocols, and a consolidation of research addressing the role of LLD on standing posture, standing balance, gait, running, and various pathological conditions. Finally, this paper will attempt to generalize findings regarding indications of treatment for specific populations. © 2002 Elsevier Science B.V. All rights reserved.

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**1. Introduction**

Limb length discrepancy, or anisomelia, is defined as a condition in which paired limbs are noticeably unequal. When the discrepancy is in the lower extremities, it is known as leg length discrepancy (LLD). LLD is a relatively common problem found in as many as 40 [1] to 70% [2] of the population. In a retrospective study, it was found that LLD of greater than 20 mm affects at least one in every 1000 people [3]. The effects of LLD on function and the magnitude of LLD warranting treatment have been subjects of controversy for some time.

Studies have investigated the effects of LLD on low back pain (LBP) [4], osteoarthritis (OA) of the hip [5], stress fractures [6], aseptic loosening of hip prostheses [7], standing balance [8], forces transmitted through the hip [9], running economy [10] and associated running injuries [11].

LLD can be subdivided into two etiological groups: a structural LLD (SLLD) defined as those associated with a shortening of bony structures, and a functional

LLD (FLLD) defined as those that are a result of altered mechanics of the lower extremities [12]. In addition, persons with LLD can be classified into two categories, those who have had LLD since childhood, and those who developed LLD later in life. In terms of functional outcomes such as gait, persons who have developed a LLD later in life are more debilitated by LLD of the same magnitude when compared to persons who have had LLD since childhood [13].

There is disagreement regarding the role LLD plays in musculoskeletal disorders and the acceptable amount of LLD necessary to warrant treatment. Some investigators have tried to quantify a significant LLD, accepting as much as 20 [14] to 30 mm [15], while others define a significant discrepancy in terms of functional outcomes [16]. Perhaps the most controversial musculoskeletal disorder associated with LLD is LBP. Some authors have found a definite association between LLD and LBP [4,17–21] while others found none [22–25]. Regarding standing posture, several authors have found an association between LLD and scoliosis [26–28], while one study has found the association less clear [29]. Regarding gait, several authors have found that relatively small (20–30 mm), LLD created significant changes in gait such as increased ground reaction forces

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(GRF) [30,31] increased energy consumption [32], and increased lower extremity kinetic energy [33] while other authors have found that these parameters remain relatively unchanged until much larger LLD (60 mm) are realized [34,35]. Tables 1 and 2 provide an overview of the results of several studies that measured the magnitude of LLD necessary to affect subjects using both objective (Table 1) and subjective (Table 2) criteria.

This review was written to consolidate information regarding the etiology, measurement, complications and treatment of LLD. In the conclusions, an attempt is made to generalize about the amount of LLD necessary to create problems in specific populations.

Table 1  
LLD necessary to affect patients—use of objective criteria

Author	Magnitude of LLD (mm)	Problem/outcome measure
Giles (1982) <sup>a</sup>	9 mm (minimum)	Pain/arthritis changes
Giles (1981) <sup>a</sup>	9 mm (minimum)	Lumbosacral facet joint angles
Young <sup>b</sup>	15 mm (minimum)	Pelvic torsion
Cummings <sup>b</sup>	6.3 mm (minimum)	Pelvic tilt
Specht <sup>a</sup>	6 mm (minimum)	Scoliosis or altered lordosis
Papaioannou <sup>a</sup>	>22 mm	Scoliosis
Mahar <sup>b</sup>	10 mm (minimum)	Postural sway, shift of mean center of pressure
Brand <sup>b</sup>	35 mm (minimum)	Altered forces at the hip
Schuit <sup>a</sup>	10.4 mm (mean)	Altered GRF
Bhave <sup>a</sup>	49 mm (mean)	Asymmetrical stance times, GRF
Blake <sup>a</sup>	3.2 mm (minimum)	Increased rear foot eversion during midstance
Kaufman <sup>a</sup>	>20 mm	Gait asymmetry
Vink <sup>b</sup>	40 mm	Increased low back EMG activity
Delacerda <sup>a</sup>	26.7 mm (case study)	Increased kinetic energy during walking, increased oxygen consumption
Song <sup>a</sup>	5.5%	submaximal during running Increased mechanical work, greater vertical center of mass displacement with gait
Liu <sup>a</sup>	>23 mm	Gait asymmetry
Gurney <sup>b</sup>	20 mm	Increased oxygen consumption in gait
Gurney <sup>b</sup>	30 mm	Increased HR, minute ventilation, EMG activity of quad on longer leg
Gurney <sup>b</sup>	40 mm	Increased EMG activity of lower extremity, plantar flexor on shorter leg

<sup>a</sup> Actual LLD.

<sup>b</sup> Artificially induced LLD.

Table 2  
LLD necessary to affect patients—use of subjective criteria

Author	Magnitude of LLD (mm)	Problem/outcome measure
Gofton <sup>a</sup>	11 (mean)	LBP/pain levels
Helliwell <sup>a</sup>	20 (minimum)	LBP/pain levels
Rossvoll <sup>a</sup>	32 (mean)	LBP/pain levels
Tjernstrom <sup>a</sup>	60 (mean)	LBP/pain levels
Goel <sup>a</sup>	20 (case study)	Meralgia paresthetica
Friberg <sup>a</sup>	5	LBP/pain levels
Messier <sup>a</sup>	6.4	Iliotibial band syndrome, shin splints, plantar fasciitis

<sup>a</sup> Actual LLD.

## 2. Etiology of LLD

### 2.1. Etiology of structural LLD

SLLD, also known as true LLD is defined as differences in leg length resulting from inequalities in bony structure. The etiology of SLLD may be congenital or acquired. Of the congenital causes, the most common include congenital dislocation of the hip, and congenital hemiatrophy or hemihypertrophy with skeletal involvement. Acquired causes can be as a result of infections, paralysis, tumors, surgical procedures such as prosthetic hip replacement, and mechanical such as slipped capital femoral epiphysis.

### 2.2. Etiology of functional LLD

Functional, or apparent LLD is a result of muscle (tightens/weakens) or joint tightness across any joint in the lower extremity or spine. Some of the more common causes can be pronation or supination of one foot in relation to the other, hip abduction/adduction tightness/contracture, knee hyperextension due to quadriceps femoris weakness, and lumbar scoliosis.

## 3. Measurement of LLD

### 3.1. Radiography and other imaging techniques

Radiography has long been considered to be the gold standard for measuring LLD [36], although radiographic techniques vary and they are not without problems. There are three methods used that utilize radiographs to measure LLD.

The first is the 'orthoroentgenogram', which involves a single exposure of the legs including hips and ankles. It has the advantage of requiring only one exposure, but is subject to distortion by parallax error [37].

The second method is the 'scanogram' which uses three exposures, one for the hip, knee and ankle, and

negates the magnification error, but increases the time, cost, and radiation exposure of the patient.

The third method is the 'computerized digital radiograph'. This technique minimizes radiation exposure, reduces mathematical error and is accurate even in the presence of angular deformity [38]. All radiographic techniques measure from some landmark on the proximal femur/pelvis to some landmark on the ankle, and do not account for the contribution of the foot to limb length.

Computerized tomography (CT), three-dimensional (3-D) ultrasonography (US), and magnetic resonance imaging (MRI) are also used to determine leg length. CT has been shown to have sensitivity better than 1 mm and has shown good reproducibility and little exposure to radiation. Although it is considered more precise and trustworthy than using radiographs [39], especially when a flexion deformity is present in the knee [40], it is more expensive. Even though conventional US has been proven to be inferior to CT for determination of leg length, 3-D US has shown to be an accurate single step determiner of LLD without ionizing radiation [41], with a standard deviation for reproducibility of leg length measurement of 1.6 mm. MRI also has the advantage of no radiographic risks, but has been shown to have less reproducibility and accuracy when compared to CT or US [42].

### 3.2. *Clinical methods*

In general, although the above imaging techniques are considered to be the most accurate method for determining LLD, they are costly, time consuming, and, in the case of radiographs and CT, the patient is exposed to radiation. As a result, alternative clinical methods have been developed. Two methods have emerged over the years: (a) an 'indirect method' done in standing using lift blocks under the short leg and visually examining the level pelvis [2], and (b) a 'direct method' done in supine measuring the distance of fixed bony landmarks with a measuring tape. Two commonly used tape measure methods (TMM) include measuring the distance between (a) the anterior inferior iliac spine (ASIS) and the lateral malleolus [2] and (b) the ASIS and the medial malleolus [43]. There is disagreement as to the validity and reliability of these methods.

The indirect or standing method incorporates the contributions of the foot and ankle whereas the TMM does not. Woerman and Binder-MacLeod [2] compared the indirect method and both TMM against radiographs and reported that the indirect method was more accurate and precise than either of the direct methods. Of the two direct methods, they reported the ASIS to lateral malleolus measure to be superior to the ASIS to medial malleolus measure. The authors advocated the use of the indirect method, especially in cases where

FLLD may be involved. Gross et al. [44] used the indirect method with a pelvic leveling device and obtained good intratester reliability ( $ICC = 0.84$ ), fair intertester reliability ( $ICC = 0.77$ ), and validity against radiographs ranging from 0.55 to 0.76. On the other hand, Friberg et al. [45] reported that the indirect method is an inaccurate and imprecise method, with a 7.5-mm mean difference compared to radiographs, and a 1.5-mm intratester error. Clarke [36] tested the indirect method and reported that two testers were within 5 mm of the radiographic leg length in only 16 of 60 subjects. In addition, Mann et al. [46] showed poor reliability of determination of the iliac crest height, which is a requisite for the indirect method.

The literature testing the TMM has shown similar conflicting results. Friberg et al. [45] reported that the ASIS to medial malleolus measurement is an inaccurate and imprecise method; with a mean difference in LLD measure of 8.6 mm compared to radiographs, and a 1.1 mm intratester mean error. The authors of this study used a single measurement of the ASIS to the medial malleolus. In agreement with this finding, Beattie et al. [43] found validity estimates of LLD to have an intraclass correlation coefficient (ICC) of  $r = 0.683$  when utilizing a single measure using the ASIS to medial malleolus measure. However, when the average of two measures was taken from ASIS to the medial malleolus, the validity of the TMM was good, the ICC with radiographs was  $r = 0.793$ . In addition, Gogia and Braatz [47] reported an ICC with radiographs of  $r = 0.98$  and a between- and within-tester reliability of  $r = 0.98$ . Hoyle et al. [48], noted an intertester reliability of  $r = 0.98$  and an intratester reliability ranging from  $r = 0.89$  to 0.95 for the ASIS to medial malleolus measurement.

In conclusion, radiographs or other imaging techniques should be used when accuracy is critical. There is still disagreement regarding the validity and reliability of both the indirect methods using a pelvic leveling device and TMM. The average of two measures between the ASIS and the medial malleolus appears to have acceptable validity and reliability when used as a screening tool.

## 4. Treatment

Prediction of LLD at skeletal maturity is an important prerequisite for determining the necessary treatment to equalize leg length. In order to determine this, future growth potential must be estimated. The study of growth as it pertains to LLD involves the relationship between leg length, maturity or skeletal age, and chronological age. The three relationships must be examined individually to help predict future growth and therefore, intervention [49].

Treatment of LLD ranges from shoe inserts to various surgical techniques including limb lengthening and shortening, and epiphysiodesis. There is disagreement regarding the correct treatment in regards to magnitude of LLD. Reid and Smith [15] suggest dividing LLD into three categories, mild (0–30 mm), moderate (30–60 mm), and severe (> 60 mm), where mild cases should either go untreated or treated non-surgically, moderate cases should be dealt with on a case by case basis and some should be dealt with surgically, and severe cases should be corrected surgically. Moseley suggests a similar breakdown: 0–20 mm requiring no treatment, 20–60 mm requiring a shoe lift, epiphysiodesis, or shortening, 60–200 mm requiring lengthening which may or may not be combined with other procedures, and > 200 mm prosthetic fitting [49].

#### 4.1. *Non-surgical intervention*

The most common treatment for mild LLD is the use of shoe lifts, which consists of either a shoe insert or building up the sole of the shoe on the shorter leg. In general, up to a 20 mm of correction can be made with an insert, while further corrections should be done on the sole of the shoe. Reid and Smith [15] state that full correction with shoe inserts is possible with up to a 10 mm LLD. They suggest that discrepancies between 10 and 30 mm should be partially corrected with shoe lifts, but that correction need only be made to 10 mm LLD. Moseley suggests that shoe lifts can be used with LLD up to 60 mm [49].

#### 4.2. *Surgical intervention*

In children with LLD of between 20 and 50 mm, some authors suggest epiphysiodesis. Epiphysiodesis is the surgical interruption of the epiphyseal plate on the longer leg [50]. The surgery involves the use of a curette to remove part of the growth plate resulting in a permanent premature closure, or fusion of the epiphysis.

Physeal stapling is the placement of staples across the epiphysis to temporarily arrest bone growth. Several studies have reported good results with this procedure in adolescents [51] as well as children [52] in management of knee deformities. There are several complications associated with this procedure including permanent arrest of the epiphyseal plate [53] resulting in over correction [49] as well as rebound longitudinal growth after removal of staples [54]. Because of the tendency of permanent arrest of epiphyseal plate growth, several authors have suggested that epiphyseal stapling should be considered a permanent form of growth arrest [54].

In patients with skeletal maturity who can accept the loss of stature, limb shortening by bone resection proce-

dures is sometimes performed. The maximum shortening that can take place in the femur is about 50–60 mm before serious muscle function is compromised, in the tibia, 20–30 mm [55].

Limb lengthening is generally reserved for LLD greater than 40–50 mm. This usually involves cortical osteotomy (corticotomy) followed by the extremity being fitted with an external fixation device that applies continuous longitudinal distraction across the osteotomy site. The lengthening rate is usually about 1 mm per day to optimize proper osteoblastic activity. One of the more commonly used devices is the Ilizarov system, which is a cage that surrounds the surgical site consisting of rings and small wires that apply the tensile force. Although significant gains can be made in bone length (over 150 mm), the procedure has a host of complications including fractures above and below the fixator, bending and fracture of the regenerate bone, pin-tract infections, joint stiffness, and cyst formation in the lengthened bones [56]. Other possible complications include sciatic nerve compression due to piriformis tightening [57], subsequent severe physeal growth retardation [58], and popliteal artery injury [59].

### 5. **Complications of LLD**

There are a plethora of studies identifying complications associated with LLD, but there is little agreement regarding how much LLD is necessary to cause them. One source suggests that a LLD of over 20 mm is unacceptable to the patient and can lead to gait anomalies and spinal deformities [3], while another source stated that many LLDs up to 30 mm can go untreated [15], while still another author stating that, with LLD up to 50 mm, disability is negligible [60]. Gross [14] found that only 50% of patients with a LLD of between 20 and 30 mm felt they were unbalanced, and only 45% of patients were willing to wear a lift with LLD up to 30 mm.

It appears that the age of onset may be one determining factor. Children can compensate for LLD more readily than adults who have experienced a sudden onset of LLD [61]. Another factor might be the functional activities of the individual. Athletes may have symptoms with significantly smaller LLD than non-athletes [62]. While Siffert [63] reported that a LLD of 10–25 mm is rarely symptomatic in the general population, Friberg [6] found that Finnish Army conscripts with as little as 10 mm LLD involved in extensive training have a greater incidence of stress fractures than controls. Subotnick [1] proposed that a 1/4 in. LLD in the athlete is as pathologically important as a 3/4 in. LLD in the non-athlete.

Complications associated with LLD can be divided into two categories: (a) functional limitations such as

gait and balance problems, and (b) associated musculoskeletal disorders such as LBP or stress fractures.

### 5.1. Functional limitations associated with LLD

LLD has been shown to affect several functional activities such as standing posture, standing balance, walking, and running. However, the conflicting information in the literature, combined with the differences in the methodology between studies, make it difficult to make many generalizations.

#### 5.1.1. The role of LLD on standing posture/balance

Compensation for LLD in standing can occur in many different ways. The longer leg is often compensated by pronation of the foot on the longer leg [64]. In addition, supination and/or plantar flexion of the foot of the shorter leg [65] can be used as a compensatory mechanism. The knee and hip can also compensate by extension of the shorter limb and/or flexion of the longer limb [66]. If the leg is left uncompensated, the anterior and posterior iliac spines are lower on the side of the short leg [67], which, in turn, may result in a sacral base unleveling [68] and/or scoliosis [26]. In electromyographic studies, it has been shown that relatively small leg length discrepancies of 10–20 mm can lead to a large increase in muscle activity of several muscle groups, making it impossible to maintain a complete resting position while standing [27]. In addition, a greater amount of pressure is transmitted through the hip of the longer leg due to both a decrease in the area of contact of the femoral head on the acetabulum, as well as an increase in tone of the hip abductors secondary to an increased distance between origin and insertion [28]. To compound this decrease in contact area, there may be an increase in axial loading through the long leg. Mahar et al. [69] reported that a 10 mm lift on one leg resulted in a significant shift in the mediolateral position of the center of pressure towards the longer leg.

The effect of LLD on standing balance has been studied. It appears that there is a difference between artificially induced LLD and true LLD. In one study, the authors measured postural sway changes in standing associated with artificially induced LLD. They found there was a statistically significant increase in postural sway with each increase in induced LLD. The authors concluded that minor LLD might be biomechanically significant [69].

On the other hand, another study [8] found conflicting findings using persons with true LLD. They found no statistical difference in postural sway between subjects with LLD and controls. The authors concluded that the long-term adaptation by the neuromuscular system found in their subjects (age range, 20–32 years old) accounted for the difference between their findings

and those of Mahar et al. [69]. In a rebuttal paper, Kirby et al. [70] stated that the differences in the findings between their study and Murrel's might be due to the small amount of average LLD used by Murrel, as well as differences in foot position during stance.

#### 5.1.2. The role of LLD on walking

Studies on the effects of LLD on walking have found gait asymmetries manifest throughout the kinetic chain [9,30,71]. In general, the individual with a LLD must step down onto the short limb and vault over the long limb resulting in an increase in vertical displacement of the center of mass, and hence an increase in energy consumption. Gait characteristics with LLD include decreased stance time and step length on the shorter leg, decreased walking velocity, and increased walking cadence [31,72]. Various compensatory mechanisms can occur to lengthen the shorter limb including increasing downward pelvic obliquity, increasing knee extension in midstance, vaulting, toe walking, or any combination of these [30]. In addition, an individual may shorten the longer leg by increasing pelvic obliquity, circumduction, increasing hip and/or knee flexion (steppage gait), increasing ankle dorsiflexion, or any combination of these [30]. In addition, Blake and Ferguson [73] found that there was a significant difference in calcaneal position of the short and long leg during midstance of gait, with the longer side being more everted by 3°. They concluded that eversion may be a compensatory shortening mechanism, as it has been postulated that eversion of the calcaneus can result in functional shortening of the leg. The patient adopts these compensatory mechanisms to minimize the displacement of the body center of mass during gait, thereby reducing energy expenditure [30].

D'Amico et al. [72] analyzed the gait of 17 patients with an electrodynamic analyzer both before and after correction with a heel lift. They found that prior to correction, the patient's longer limb demonstrated an average cadence of 48.2 steps/min (1.25 s/step average) and the patient's shorter limb 52.3 steps/min (1.15 s/step average). After correction, the patient's longer limb demonstrated a cadence of 44.0 steps/min (1.36 s/step average) and the patient's shorter limb 45.0 steps/min (1.33 s/step average). In addition, they found that the shorter limb underwent compensatory supination and the longer limb compensatory pronation. Correction resulted in a reduction in both the pronatory forces and the supinatory forces during gait.

Bandy and Sinning [74] examined range of motion (ROM), duration, and angular velocities in the hip, knee and ankle during gait in four males with LLD ranging from 4.8 to 9.5 mm with and without corrective heel lifts. They found that correction did not significantly affect any of the above parameters, but noted that the heel lift did cause more symmetrical movement

for the maximum angle of hip extension and ROM of the swing plantar flexion phase on the ankle.

Vertical GRF during locomotion have been examined by several authors. While a normal gait has been shown to have equal GRF for both limbs [75], several authors have found that LLD creates an asymmetry in GRF, with a larger GRF consistently found on the longer leg [30,31]. Since a relationship between GRF and economy during locomotion has been established [76], GRF values might also lend some insight into energy expenditure during gait.

Bhave et al. [31] used a three parameter GRF analysis using a force plate system as their outcome measure. They found significant differences in GRF between subjects with true LLD and controls, as well as a significant difference in GRF between the longer and shorter leg in patients with LLD, with the longer leg having the greater GRF. Their study included a post-test of the same patients after corrective lengthening surgery. They reported that after the leg lengths were corrected to within 10 mm, there is no significant difference in GRF between legs. In addition, correction of LLD equalized the stance times between legs from a 12% difference before surgery to a 2.4% difference after.

Kaufman et al. [30] used five GRF parameters combined into an asymmetry index as their outcome measure, and reported that 45–70% of the variation in gait asymmetry was explained by the variance in limb length. They found that a LLD of  $> 20$  mm resulted in a gait asymmetry significantly greater than that observed in the normal population.

Liu et al. [77] performed a similar analysis to Kaufman et al. but used GRF in conjunction with several positional parameters to determine what they called a symmetry index (SI). In contrast to Kaufman et al., their results suggested that GRF alone and LLD did not correlate well (they did not report the correlation coefficient), and that differences in hip flexion at initial contact and knee minimal flexion during midstance mainly determined the alterations of subjects SI influenced by LLD. They concluded that acceptable gait symmetries were evident in individuals with mean values of LLD up to 23.3 mm.

Brand and Yack [9] examined the relationship between LLD and forces at the hip joint during walking. Unlike the previous GRF studies, these authors artificially induced LLD of 23, 35, and 65 mm in normal individuals. They found that the 35 and 65 mm lifts increased mean peak intersegmental resultant hip forces of the short leg by 2 and 12%, respectively, and decreased the forces of the long leg by 6 and 12%, respectively. The 23 mm lift produced no measurable changes. In agreement with these findings, Goel et al. [78] artificially induced LLD of 12.5 mm and noted no significant differences in joint moments between short

and long limbs. The authors suggested that the body is able to compensate for minor LLD of up to 20 mm.

Mechanical work and walking economy have been found to be correlated [79]. Song et al. [71] measured mechanical work of children with true LLD during gait. They found that the children demonstrated several compensations including toe walking, vaulting, circumduction, and increased flexion of the longer limb. In addition, they noted the long limb performed more mechanical work than the short limb. The average LLD for patients who had no observable compensatory strategy was 16.4 mm.

In a single case study, Delacerda and Wikoff [33] measured the lower extremity kinetic energy of a woman with a true LLD of 28.7 mm during gait, both with and without a corrective lift. They found that the addition of the lift decreased the overall kinetic energy expended by both legs during gait. Gurney et al. [32] used an artificial LLD (aLLD) on healthy older persons (ages 55–86) during gait and found that subjects had a significantly greater energy consumption using  $VO_2$  at 20, 30 and 40 mm aLLD compared to no LLD, significantly greater HR at 30 and 40 mm aLLD, significantly greater minute ventilation at 30 and 40 mm aLLD, and reported a higher rate of perceived exertion at 20, 30 and 40 mm aLLD compared to no aLLD. Based on the findings in studies using GRF, forces at the hip, mechanical work, and kinetic energy, and oxygen consumption, it would appear that a LLD as small as 20 mm can consistently affect the function of gait.

In apparent conflict with the above findings, however, Phelps et al. [34] reported that young adults who had as much as 60 mm of true LLD did not have a greater oxygen consumption or oxygen cost than normal adult controls during walking. Richter [35] found that only persons with true LLD of  $\geq 40$  mm had significant differences in heart rate. He also performed a repeated measures study and found that subjects ambulating with as much as a 60 mm aLLD did not have significantly higher heart rates than with no leg length difference.

There is little research addressing the effects of LLD on muscle activity during gait using electromyography (EMG) as a measure. Vink and Huson [80] artificially induced LLD of 10, 20, 30 and 40 mm and examined back extensor muscle activity with surface EMG (sEMG). They found a significant increase in sEMG activity of the back extensor muscles during heel strike of the raised limb when LLD was 30 mm or greater. The authors suggested that the increase in sEMG was due to an exaggerated trunk flexion induced by an increased deceleration of the pelvis during heel strike of the raised limb. Gurney et al. [32] found that there was a significant overall increase in muscle recruitment (the sum of quadriceps femoris, gastrocnemius, gluteus maximus and gluteus medius bilaterally) with a 40 mm

aLLD compared to no LLD, but not with a 20 or 30 mm aLLD. In addition, they found that the quadriceps on the longer leg showed significant increases in activity only with a 40 mm aLLD, and the plantar flexor on the shorter leg showed a significant increase in activity with both a 30 and 40 mm aLLD.

### 5.1.3. *The role of LLD on running*

The mechanics of running differs considerably from those of walking, and the effect that LLD has on running reflects those differences. The vertical oscillation is greater in running compared to walking, and there is no double support in running, so the weight is not shared between the two limbs. In addition, the stance phase of walking is about 60% of the total gait cycle as compared to 30% with running [81]. These differences lead to stresses on the lower extremity that are three times that of walking [12]. It has been suggested that biomechanical abnormalities due to LLD are three times more significant in running compared to walking [1].

The findings of the literature vary regarding the effects of LLD on running dynamics. Studies have addressed parameters including rear foot position and energy consumption. Blake and Ferguson [73] examined rear foot position of persons with true LLD during running. They found that there are significant differences between the calcaneus-to-vertical angle of the short and long legs in both early midstance and midstance, with the longer leg exhibiting greater calcaneal eversion. Overall, the difference in calcaneal position was greater in running when compared to walking. Unfortunately, the authors did not report the magnitude of the LLD in their subjects except to say they were over 1/8 in. (3.2 mm). In contrast to these findings, Bloedel and Hauger [82] reported no significant difference in the amount of maximum calcaneal inversion or eversion between the long and short limbs. The range of true LLD in their subjects was from 12.7 to 19.0 mm. However, the authors did not control for shoe type, which has been shown to influence the rear foot during initial contact to midstance [83].

Delacerda and McCrory [10] found that a corrective lift resulted in a reduction of oxygen consumption in a 30-year-old runner with a 28.6 mm true LLD when running at a sub-maximal rate compared to running without a lift. In a similar study, Kern (1995, unpublished data) examined oxygen consumption in runners with true LLD of 3–11 mm. He found no differences in oxygen consumption of subjects with and without a corrective shoe lift. In fact, he noted that the running economy actually worsened in some subjects with the use of the orthotic correction. In conclusion, based on studies that reported LLD magnitudes, it appears that the LLD needs to be in excess of 19 mm before running parameters are affected.

In apparent conflict with this generalization, however, one study performed by Reid, Smith and Raso (1982, unpublished data) found no statistically significant difference with regard to the amount of oxygen required to run, even with a 30 mm aLLD.

## 5.2. *Associated musculoskeletal disorders*

LLD has been implicated in a variety of disorders including LBP, scoliosis, pelvic and sacral malalignments, arthritis of the spine, hip pain and OA, lower extremity stress fractures, aseptic loosening of the prosthetic hip, trochanteric bursitis, myofascial pain syndrome (MPS) of the peroneus longus, patellar apicitis, and meralgia paresthetica.

### 5.2.1. *Low back pain*

Perhaps, the most equivocal pathological condition associated with LLD is LBP. LBP is a general term that includes pain in the lumbar spine, the lumbosacral junction, and the sacroiliac joint (SI). LLD appears to affect the lumbar spine, at least in part, by creating a lumbar scoliosis. It has been shown that LLD leads to pelvic obliquity on the frontal plane and a scoliosis that has been described as compensatory, non-structural, and non-progressive [26]. Friberg [17] described the scoliosis as a lumbar convexity toward the short leg side with a concomitant vertebral axial rotation. He goes on to say that the curve is produced solely by the intervertebral joints, and the subsequent wedging on the intervertebral discs in combination with the axial rotation are predisposing factors to LBP. Young et al. [84] examined the immediate effects of a simulated LLD of a magnitude necessary to create a lateral tilt of the pelvis of 1.2° and above on pelvic torsion and trunk flexion. They found the innominate contralateral to the lift rotated anteriorly compared to the ipsilateral side and that lateral flexion of the trunk increased toward the side of the lift. Giles and Taylor [85] found that patients with LLD demonstrated abnormal radiologic findings compared to controls including wedging of the fifth lumbar vertebra, concavities of the vertebral endplates in the lumbar spine, and traction spurs and osteophytes of the vertebral bodies. Morscher [28] reported that the asymmetrical loading forces acting on the spine secondary to scoliosis caused by LLD result in early degeneration affecting both the intervertebral disc spaces in the form of osteophyte formation as well as arthrosis of the facet joints on the concavity of the scoliosis. Hoikka et al. [29] found that the relationship between LLD and scoliosis was not as clear. They reported that although LLD correlated well ( $r = 0.843$ ) with pelvic tilt (on the frontal plane), it only correlated moderately with sacral tilt ( $r = 0.639$ ), and poorly with lumbar scoliosis ( $r = 0.338$ ). They proposed that the body compensates for asymmetry associated with LLD

progressively up the kinetic chain. They concluded that although there may be an association between LLD and LBP, LLD might not be the cause of LBP.

Giles [86] examined patients with LLD radiographically and found that the lumbosacral facet joint angles on the short side were smaller with the horizontal compared to the controls. He postulated that the asymmetry of joint angles could predispose patients to osteoarthritic changes at the lumbosacral joints.

It has been demonstrated that LLD is associated with sacroiliac malalignment. Greenman [68] stated that LLD can result in sacral tilt, and that patients with an unlevel sacral base were twice as likely to complain of low back syndromes as patients without an unlevel sacral base. He concluded that a sacral base unleveling of greater than 4 mm as determined by X-ray should be considered clinically significant. Schuit et al. [87] found that nine of the 14 subjects with LLD had a SI malalignment of some kind. Pitkin and Pheasant [88] described an innominate bone rotation of between 3 and 19° in patients with LLD. Cummings et al. [89] reported the same relationship and described it as a posterior innominate bone rotation on the side of the lengthened limb and an anterior rotation on the side of the shorter limb. The authors also suggested that innominate rotation could limit movement strategies or patterns during activities that involve movement of the pelvis, that may result in asymmetrical loading of the SI ligaments, predisposing them to chronic strain. In addition, they proposed that a constant asymmetrical loading on the SI joint surfaces might lead to degenerative changes of the joint.

There have been many studies showing a positive relationship between LLD and LBP. In the introduction of an article by Giles and Taylor [4], the authors combined five studies with a total population of 1806 subjects. They chose to look only at studies that used radiographic measurements of LLD. They reported that about 7% of the subjects with no history of LBP have a LLD of 10 mm or more, while four of the five studies showed 13–22% of subjects who sought medical treatment for their LBP have LLD. In their own study, Giles and Taylor tested 217 persons. Of these, 8% who had no complaints of LBP had a LLD of 10 mm or more, while 18.3% of those with LBP had a LLD of 10 mm or more. Friberg [17] compared 653 patients with LBP with 359 controls with radiography and found the relative prevalence of leg length inequality of > 5mm was 1.7 times greater in the LBP group compared to the control group. The prevalence grew to 5.3 times greater with 15 mm LLD. In a population of 132 persons with LLD of 10 mm or greater who were experiencing LBP and radiating leg pain, 104 had pain radiating into the shorter leg ( $P = 0.02$ ) [18].

If LLD does lead to LBP, then treatments aimed at equalizing the LLD should help with the symptoms.

Giles and Taylor [4] treated 50 LBP patients with LLD with shoe lifts. After a 4-month follow-up, their results showed less working days lost to LBP, a marked decrease in subject-reported symptoms and numbers of LBP episodes, and an increase in overall ROM. In a clinical study by Gofton, ten patients with LLD and LBP who were treated with a shoe lift on the short side experienced major or complete pain relief that lasted upon follow-up ranging from 3 to 11 years [19]. In a similar clinical study by Helliwell [20], 18 patients with LBP and a LLD of 20 mm or more were treated with corrective shoe lifts. Upon follow-ups of at least 3 months, 44% experienced complete pain relief, and 45% had substantial or moderate pain relief. Similarly, Friberg [17] found that, of the 211 persons with LBP treated with shoe lifts, 157 were symptom-free after a mean follow-up of 18 months. In a study by Rossvoll et al. [21], 22 LBP patients with an average of 32 mm LLD were surgically managed with shortening osteotomy. After surgery, their average LLD was 4.3 mm, and their LBP was significantly reduced. Finally, Tjernstrom and Rehnberg [90] performed 100 leg-lengthening procedures. Before lengthening, 18 of these patients experienced LBP, whereas, after an average 6-year post-surgical follow-up, six patients experienced LBP.

On the other hand, several investigators have found no relationship between LBP and LLD. Hellsing [22] examined over 600 military trainees over a period of 4 years and found no correlation between LLD and back pain or pain provoking tests. Similarly, Nadler et al. [23] showed in a prospective study on 257 college athletes that LLD was not associated with future LBP treatments. Soukka et al. [24] found no association between mild LLD measured radiographically and LBP in 247 men and women. Yrjonen et al. [25] found LLD and lumbar scoliosis correlated poorly with LBP in 96 patients with Perthes' disease.

### 5.2.2. Hip pain

The literature appears less equivocal regarding hip symptomology. Brunet et al. [11] surveyed 1493 male and female runners and found that hip pain is over twice as common a complaint in persons with a self-reported LLD compared with persons with no LLD. Friberg [17] reported that of 254 patients with LLD complaining of chronic hip pain, 226 have pain on the longer extremity. Twenty-seven of these subjects had severe idiopathic hip arthrosis, with the arthrosis occurring on the longer leg in 24 of the 27 subjects. Friberg treated 79 of these hip patients with shoe lifts, 56 of which became symptom-free after treatment. Gofton and Trueman [5] had similar results. They examined 67 patients with hip OA, of which 62 were idiopathic. Of these, 36 patients demonstrated significant LLD. All 36 patients had superolateral type OA, and 29 of the 36 patients had the arthritic findings in the longer leg.

Since OA is generally considered to cause shortening of the involved leg, the authors dismissed the possibility that the OA is the cause of the LLD noted in these patients. They suggested that a longer leg might be a predisposing factor to hip OA.

This relationship between hip OA and the long leg in LLD is consistent with the calculations of Krakovits [91] who mathematically modeled a relationship between LLD and reduction of weight-bearing area of the femoral head. Based on his formula, a 10 mm increase in leg length would result in a reduction of weight-bearing area of 5%, and an increase in leg length of 50 mm would cause a reduction of contact area of 25.1%.

The work of Morscher [28] also supports these findings. He found a greater amount of pressure transmitted through the hip of the longer leg due to the decrease in area of contact of the femoral head on the acetabulum, as well as an increase in tone of the hip abductors secondary to increase distance between origin and insertion. These findings coupled with the finding of several authors that larger GRF are found on the longer leg [30,31] suggest the longer leg to be at risk. Gurney et al. [32] found greater EMG activity on the quadriceps of the longer leg with an aLLD, which could be due to the greater GRF that leg would have to overcome. Finally, the findings of Visuri et al. [7] seem to support the greater force transmission through the longer leg. They found that overlengthening of the prosthetic leg is the most important single variable predisposing patients to aseptic loosening of the prosthesis after total hip arthroplasty.

The issue of greater forces through the hip of the longer leg is still in question, however. Brand and Yack [9] found that when subjects were given aLLD of 35 and 65 mm, they demonstrated decrease intersegmental resultant forces through the hip on the longer leg of 6 and 12%, respectively. In addition, Schuit et al. [92] found that patients with LLD ranging from 4.8 to 22.2 mm demonstrated a maximum vertical force that was significantly greater in the short leg before correction with a lift compared to after correction.

### 5.2.3. Stress fractures

Several studies have shown a higher incidence of stress fractures in the lower extremities in people with LLD. Bennel et al. [93] reported that LLD is a risk factor for stress fractures in female athletes, in that a significantly greater number of women in the stress fracture group (70%) displayed a LLD compared to the non-stress fracture group (36%). Brunet et al. [11] surveyed 1493 male and female runners and noted that over twice as many runners with a self-reported LLD develop stress fractures when compared to runners who reported no LLD. Friberg [6] noted an association between the quantity of LLD and the incidence of stress fractures. While 15.4% of Finnish conscripts with

no LLD experienced stress fractures, 46.2% of those with a 10–14 mm LLD had stress fractures, and 66.7% of those with LLD of 15–20 mm had stress fractures. He also reported that stress fractures occurring in the tibia, metatarsals, and femur were found in the long leg 73% of the time. The increased incidence of stress fractures on the longer limb seen by Friberg appears consistent with the greater forces emitted through the longer leg described under hip pain.

### 5.2.4. Other pathology

In addition to the above problems, there appears to be an association between LLD and several other lower extremity problems. Swezey [94] found a relationship between LLD and trochanteric bursitis. He found a LLD of 25.4 mm or greater in 13 of 20 patients diagnosed with trochanteric bursitis. Kujala et al. [95] found significantly more patellar apicitis ( $P < 0.001$ ) and patellofemoral joint incongruencies [96] in patients with LLD than in controls. Saggini et al. [97] found that LLD was related to MPS of the peroneus longus. When they treated MPS patients with shoe lifts, they reported a moderate, significant reduction in pain reports. Finally, in a single case report, Goel [98] found that when he treated a patient with long standing meralgia paresthetica with a shoe lift in his shorter leg, the pain was resolved.

## 6. Conclusion

There is still controversy regarding the magnitude of LLD necessary to cause musculoskeletal problems. The articles that find associations between LLD and pathological conditions must be interpreted with caution, as association does not prove a cause effect relationship, and may just represent a coincident finding.

It is clear that studies using persons with long standing true LLD are able to cope with larger LLD than those who are subjected to artificial or induced LLD. This is sensible since given enough time most individuals would be able to reduce the energy and mechanical costs of LLD. It also appears that younger persons on the whole are able to adapt to larger LLD than older persons. Again, this is sensible since it has been shown that gait patterns differ considerably between old and young individuals [99,100], and that older persons have greater difficulty in mastering novel motor tasks [101,102]. The level of activity of the person also seems to play a role. Individuals who are on their feet most of the day [17] or who are involved in sports [1] seems to be more sensitive to LLD than those who are less active. Finally, studies that use patient pain levels or satisfaction as an outcome measure seem to differ from those that use more objective outcome measures (Tables 1 and 2).

The question of whether to treat individuals with LLD should be taken on a case by case basis, although the breakpoint of 20 mm is often used, this number could be considerably higher for younger persons who are inactive and have had LLD their entire life and considerably lower for older persons who are active and have acquired LLD later in life.

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