lifts in some cases of mild anatomic LLI, plus muscular reactions to, and causes of, pelvic torsion.

The functional short leg, or unloaded leg-length alignment asymmetry

The functional short leg, or unloaded leg-length alignment asymmetry (hereafter abbreviated as LLAA) is itself a phenomenon much discussed and little understood. Essentially, when a subject lies prone or supine, unloading the pelvis, the feet are examined, most often at the welt (heel-sole interface), for the presence of a "short leg" or alignment asymmetry. Some hold the opinion that anatomic LLI can be measured in this way [1]. The examination for unloaded leg-length alignment asymmetry as a sign of "neuromuscular dysfunction" is a clinical test commonly used by chiropractors [2,3]. Given the frequent use of this test as an indicator of a functional problem, it is important to know whether the unloaded leg check test is an indicator of an anatomic short leg, or whether the test is reliable and valid as an instrument to measure functional "short leg" and whether LLAA findings are contaminated by anatomic LLI.

Anatomic LLI is caused by a natural developmental asymmetry or a variety of other factors, including fracture, disease, and complications of hip replacement surgery. Given the long-term loading, the lumbopelvic structure may be expected to adapt via Heuter Volkmanns' law [4] and soft tissue changes [4,5], establishing the compensated structural changes as "normal". This adaptive response is seen in the change of lumbosacral facet angles noted by Giles [6]. A case study followed the effect of anatomic LLI caused by hip replacement surgery on subjective symptoms, unloaded LLAA checks and pelvic unleveling, reporting that adaptive changes occurred over a period of several months [7].

Using a device to measure standing pelvic crest unleveling, Petrone et al found excellent intra and inter-examiner reliability, and validity (ICC, 0.89–0.90) relative to anatomic leg length inequality determined by x-ray measurement in asymptomatic subjects [8]. However, the correlation between the pelvic level and femoral head heights was "substantially lower" in a low back pain group. This indicates that some sort of functional pelvic tilt or torsion was present in the low back pain population that was unrelated to their anatomic LLI. While the decreased correlation between pelvic tilt and LLI in the back pain group was not examined relative to a functional short leg, the connection between back pain and the biomechanically unusual pelvic torsion stands out.

Lumbar lateral flexion was studied in a group of subjects 10 years after LLI caused by femoral fracture that occurred *after* they were skeletally mature [9]. Despite the compensatory lumbar scoliosis, these subjects had symmetrical lumbar lateral flexion, prompting the authors to comment that the "...acquired leg-length discrepancy produced little permanent structural abnormality in the lumbar spine..." [9]. Significant anatomic LLI acquired after skeletal maturity does not result in adaptive structural changes within a 10-year period.

However, another study from the same orthopedic center looked at the effects of significant (mean 3 cm) LLI acquired *prior* to skeletal maturity [10] in now mature subjects (17–38 years old, mean 28). In this group, there was considerable asymmetry of lumbar lateral flexion after placing a lift under the short leg to level the pelvis. This indicates that the body had permanently compensated to the structural changes in the spine/pelvis.

This type of permanent compensation to pre skeletal maturity LLI was also found in subjects with pelvic unleveling. Young et al [11] found that placing a lift under the foot of a subject with no pelvic unleveling resulted in greater lumbar lateral flexion towards the now high iliac crest side. In subjects with pelvic unleveling, when the lift was put under the foot on the side of the low iliac crest in order to level the crest, lateral flexion was increased towards the formerly low crest side. If the body remodels and adapts to the pelvic unleveling/torsion caused by anatomic LLI, then by putting a lift under the side of the "low" iliac crest, one is actually raising what the body has adapted to as level. In other words, the unlevel pelvis of those with anatomic LLI has been adapted to and is now "normal", and putting a lift under the low side has the same effect as putting a lift under the leg of an even pelvis (Figure 1).

These two studies [10,11] provide evidence that in pre-skeletal maturity subjects, LLI and pelvic torsion – which describe the vast majority of LLI – adaptive changes take place in the muscles, ligaments, joints and bones to compensate for the imposed asymmetry. Because these adaptive compensations to the LLI have become anatomic, they are not likely to change as the body moves from a loaded (standing) to an unloaded (supine, prone) position. The nervous system also appears to compensate as demonstrated in the study by Murrell et al [12] in which there was no loss of stability in subjects with LLI, prompting them to point to "long-term adaptation by the neuromuscular system".

The persistence of pelvic torsion in subjects with anatomic LLI is supported by Klein [13] who found that such distortion remained in both standing and sitting positions. That pelvic torsion persists with the subjects' weight off the femoral heads indicates such torsion has been incorporated into the joints as the normal position. Rhodes et al