an anatomic LLI and structurally compensatory pelvic torsion moves from a loaded (standing) to an unloaded (prone/supine) position, the torsion of the pelvis remains intact and the leg length at the feet/shoes would appear "even" on a visual check. The pelvis – joints, ligaments and muscles – have adapted to the anatomic LLI, making any torsion structural. It is this putative biomechanical adaptation that makes unloaded leg-length alignment asymmetry tests – the functional "short leg" tests – unreliable as a measure of anatomic LLI [14].

Unloaded LLAA is suspected to result from hypertonicity of suprapelvic muscles [15-17]. In a study of subjects with and without supine LLAA, Knutson & Owens found those with LLAA had significantly decreased endurance times for the erector (Biering-Sorensen test) and quadratus lumborum muscles [18]. Further, the side of LLAA significantly correlated with the side of the QL muscle quickest to fatigue. One of the causes of increased susceptibility of muscles to fatigue is hypertonicity. These results stand in contrast to Mincer et al [19] who suspected altered muscle fatigue profiles with anatomic leg-length inequality, but did not find such, providing further evidence that LLAA is a pathological process distinct from LLI.

When standing, the actions of the QL depend on whether the spine or the pelvis is stabilized. If the pelvis is stabilized, QL contraction laterally flexes and extends the spine [1,20,21]. With the spine stable, QL contraction pulls cephalically through its attachment to the posterior aspect of the hemipelvis [1,21]. This load on the posterior aspect of the iliac crest could act to rotate the ipsilateral anterior hemipelvis lower – an AS ilium – causing the pelvis to torque and having the opposite effect on the contralateral hemipelvis – a PI ilium. The degree of torsion (if any) would be dependent on the tension in the QL and the freedom of movement of the pelvis, and any pre-existing pelvic torsion due to anatomic LLI. However, if the subject now adopts an unloaded posture – supine or prone – QL hypertonicity is freed from the load of the body and able to lift the ipsilateral hemipelvis, hip and leg in the cephalic direction, producing leg-length alignment asymmetry at the feet. This model is in agreement with Travell and Simons who write, "In recumbancy, active TrPs [trigger points] shorten the [quadratus lumborum] muscle and can thus distort pelvic alignment, elevating the pelvis on the side of the tense muscle" [1].

Clinical considerations

Now we can return to the dilemma of how lifts may have a positive effect on back pain and muscle activity given that most anatomic LLI is not clinically significant. Torsion of the pelvis as an adaptive structural compensation in anatomic LLI has been shown to be limited. If a person has pelvic torsion due to anatomic LLI near the limits of the body's ability to adapt, and QL hypertonicity with its ability to cause pelvic torsion is superimposed, muscular bracing reactions and pain could be the result. Indahl et al [22] found that stimulation of the sacroiliac joint capsule (in pigs) caused reflexive muscular responses, depending on what area of the joint (dorsal/ventral) was stimulated. They note that, "Irritation of low threshold nerve endings in the sacroiliac joint tissue may trigger a reflex activation of the gluteal and paraspinal muscles that become painful over time". Interestingly, stimulation of the ventral area of the SI joint produced reflexive contraction of the quadratus lumborum. It may be that a positive feedback loop could be established where QL hypertonicity leads to lumbar curvature and pelvic torsion which stimulates the SI joint leading to more QL hypertonicity, more lumbar curvature and pelvic torsion. It will be interesting to see if a similar muscular reflex to SI stimulation is found in humans.

Based on their research, Allum et al [23] proposed that rotation of the trunk excites joint receptors in the lumbar spine triggering muscular contractions – paraspinal muscles – for balance correction. While these receptors likely have adapted to any pelvic/lumbar rotation caused by anatomic LLI, further pelvic torsion caused by QL hypertonicity may stimulate the balance receptors causing reflexive muscular contraction. A lift would reduce the pelvic torsion and lower the proprioceptive balance triggers below threshold, eliminating chronic, painful muscular contraction.

In a case of additive effects of anatomic LLI and QL/suprapelvic hypertonicity on pelvic torsion, a lift used to level the pelvis would take the strain off the sacroiliac and associated joints and ligaments and decrease potentially painful muscular bracing. Thus, lifts can work to decrease back pain in people with what seem to be clinically insignificant amounts of anatomic leg-length inequality. Of course, it would be important for the clinician to explore reasons for any quadratus lumborum and other suprapelvic muscle hypertonicity and eliminate them to provide a complete correction. On the other hand, pure anatomic LLI in the range of and above 20 mm – the upward limit for adaptive compensation – may stimulate sacroiliac and/or lumbar proprioceptors causing reflexive and ultimately painful muscular contractions that will only be relieved by a lift to level the pelvis.

Reliable detection of LLI and LLAA is difficult, but not impossible. Research has shown the examination procedures for putative LLAA both prone [24] and supine [25] to have intra- and inter-examiner reliability. In a controlled setting, Cooperstein et al investigated the accuracy of a compressive prone leg check in subjects with proscribed amounts of artificial LLI [26]. They found the procedure