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Leg length inequality in humans: a new neurophysiological approach

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Abstract

In conditions where there is moderate or severe pain, such as in low back pain, sciatica and osteoarthritis affecting the hip, the patient often reports the sensation that the two legs are of unequal length. This sensation cannot be explained as a result of fracture or other trauma that might cause a real change in length of the legs. There is confusion as to whether this 'leg length inequality' causes the pain or vice versa. It is argued here that the sensation occurs when there is pressure on the sensory nerves, especially the dorsal spinal roots. This causes pain but, more relevantly, causes an increased discharge in muscle spindle nerves, this in turn signaling an increased length of the associated muscles. This idea is testable using percutaneous electrodes.

Keywords: Pelvic pain; Sciatica; Muscle spindle afferents; Proprioception

In most people the left and right legs are not equal in length but this does not usually create any problems unless the difference exceeds about 2 cm (reviewed in Gurney [12]). Presumably in the course of early life, adjustments are made so that at puberty a satisfactory modus operandi has been achieved. It is possible that differences of less than 2 cm may prevent top performance in occupations where a high degree of skill or endurance is demanded [3,9]. If in a mature person a leg length difference is introduced either as a result of injury or experimentally [12] then the system is thrown out of equilibrium and further adjustments have to be made.

Nevertheless, in the absence of any fracture or trauma that might cause a real change in length but in conditions where there is moderate or severe pain, such as in low back pain, sciatica and osteoarthritis affecting the hip, the patient often reports the sensation that the two legs are of unequal length. Often the patient tends to list towards the side contralateral to the injured side and there is elevation of the ipsilateral hip [15,18]. The condition goes under a variety of names: 'short leg'; 'long leg'; 'limb-length discrepancy'; 'leg length insufficiency'; 'leg length inequality'. For convenience I will use the last expression, abbreviated to 'LLI'. I exclude from consideration here conditions termed 'functional length difference' where there is no change in bone length but gross distortions of foot and pelvis occur for various reasons.

In LLI, considerable effort has been expended in the attempt to determine accurately the difference in length of the two legs. Yet there remains much uncertainty as to the primary cause of the phenomenon. Several explanations have been put forward. In some accounts low back pain, sciatica and other pain syndromes were thought to arise as a result of the distortion of the pelvis associated with LLI [2]. In other words, the LLI leads to strain and displacement at the sacro-iliac joint which in turn produces low-back pain. I believe a reverse dependency is closer to the truth, that is, the LLI is not causative for the pain but is associated with the pain. My approach stems from physiological considerations as well as from personal experience of LLI associated with sciatica. The reader is referred to some reviews on this topic for a detailed discussion [1,11,14]. In this brief article I wish to suggest a further hypothesis which does not seem to have been previously considered.

Neural basis of proprioception. How do we know where our limbs are in space? It is now accepted that the contribution made by joint receptors is important primarily during movement; in the absence of movement they make only a weak contribution except possibly at extremes of flexion or extension (cf ref. [4]). This fact is dramatically illustrated by the presence of normal proprioception in patients who have received artificial joints.

It is currently believed that the information detailing muscle length is derived from the discharge of the muscle spindle nerves, both the Ia fibres from the primary endings and the group II fibres from the secondary endings [10]. This information must be interpreted against a background of

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discharge in the gamma motor neurons, a process still imperfectly understood. Knowledge of muscle length is the neural basis of proprioception. In the rare cases where we lose our muscle spindle nerves we lose our proprioception.

A very simple but very convincing experiment is as follows. A subject stands blindfolded while physiotherapy vibrators are applied to his Achilles tendons [8]. He falls backwards and must be caught so that he does not injure himself. The physiotherapy vibrators are very effective stimulators of muscle spindle receptors, especially the primary receptors, and so set up impulses in the fibres coming from the spindles in the calf extensors of the foot. The discharge in these fibres mimics what would happen if these muscles were stretched, e.g. if the subject were pushed forward, the feet remaining on the ground. The automatic reaction – to preserve balance – is to lean backwards. I will make a leap of the imagination from here and suggest that we know the length of a limb by some kind of integration of all the information coming in along the spindle nerves.

Relation between pain and proprioception. The existence of pain leads to a large variety of responses affecting breathing, blood pressure and contraction of muscles. One of the themes suggested for LLI has been that such muscular contraction is the cause of the LLI. I wish to suggest a simpler explanation. While the pain may lead to muscular contractions and adjustment of posture it does not lead to the perception of LLI. Associated with the pain there are other features that are more relevant. In particular, in cases of prolapsed intervertebral disc, there is pressure on the dorsal root fibres. It is this pressure on the small A delta and C fibres that depolarizes these fibres and gives rise to the pain, low back pain and sciatica. But the larger the fibre the more susceptible it is to pressure – and the Ia and II fibres are among the largest fibres in the body. In such circumstances it is unavoidable that the Ia and II fibres will be discharging. Indeed, it must also follow that these nerves could be discharging in the absence of any pain, because their threshold is lower than that of the pain fibres. I am not aware of any measurements of Ia or II activity in cases of LLI but an increase in EMG on the side of the 'longer' leg has been reported [17] which is consistent with increased activity in the muscle spindle nerves. In addition to stimulation caused by pressure, if axons are also injured by the pressure they become more susceptible to all kinds of stimuli and emit ectopic discharges [16,19]. Depending on where the pressure is applied, the muscle spindle nerves will be preferentially excited from one or other myotomal group of muscles. However, it seems that for most lumbar disc ruptures it is the extensors of the lower leg, particularly gastrocnemius and soleus, that are involved. Such stimulation will give rise to the sensation that these muscles are being stretched, i.e. to the sensation of 'long leg' ipsilaterally. This is really the prime effect but the condition is often reported as 'short leg' contralaterally. It is also probable that in some cases the muscle spindle nerves from the hamstrings are excited. Because the hamstrings can act as flexors of the knee, it may be that the leg seems shorter. But it could also be argued that lengthening of any leg muscle might give the sensation of leg lengthening. There is some support for this view in the experiments of Lackner [13] who found that in most subjects vibration of either the biceps brachii or the triceps brachii caused the sensation of tilting of the body away from the side of stimulation. There is clearly a need for more experimentation in this area In other cases pressure can be exerted on nerves as a result of inflammation, swelling, compression or entrapment by muscles, neuromas or other causes. In all such cases the largest fibres have the lowest thresholds.

Treatment of LLI. The condition of LLI is commonly treated by placing a shoe lift under the shoe of the 'short leg'. Generally this appears to relieve the sensation of LLI. However, it is a moot point as to whether this is the best treatment, because it is introducing a leg-length difference where none may exist. If the preceding analysis is correct the LLI is a hallucination and the correct treatment would be to remove the cause of this hallucination. The associated pain acts as a monitor of the disability. If the pain is due to pressure on certain nerves then the correct therapy is to remove that pressure. Low back pain will often correct itself with conservative treatment [5] and the LLI will disappear. Of all the treatments short of surgery that are effective in relieving pain (except perhaps that due to spinal stenosis) probably gentle exercise has been most effective [5]. In particular, walking is recommended and I should like to comment on why this is a useful treatment. This may be because gentle exercise sets up impulses in large fibres and these have a well known suppressive effect on impulses in pain fibres. I suggest they may also have an inhibitory effect on LLI but, as far as I know, this idea has not been tested. What I can say is that, from personal experience, when I can relieve the pain of my sciatica by gentle exercise, the LLI also disappears, although not necessarily with the same time course.

The mechanism may be as follows. At the spinal level normal movement of the limbs is governed by 'reciprocal innervation'. By this is meant that when one muscle contracts the antagonistic muscle is inhibited via spinal cord circuitry. This obviously makes rhythmical movement easier. In addition there is presynaptic inhibition of the discharges in the muscle spindle nerves. This inhibition even extends to inhibition of the terminals of the muscle spindle nerves at their synapses on the neurons ascending to the cerebellum, the spino-cerebellar tracts [7]. These features were well summarized by Eccles [6]. This natural 'interference' may be enough to overcome the pathological discharges I am postulating in these muscle spindle nerves.

Immobility, on the other hand, has two opposing effects. By restricting movement it prevents surges of pressure on the nerves and allows swelling to decrease. However, immobility prevents the introduction of inhibitory effects such as those generated in movement, and therefore does

nothing to reduce the discharges in the muscle spindle nerves.

Success has been claimed for many treatments of LLI However, whatever treatment is given will appear to be successful if the underlying cause of the LLI, e.g. prolapsed intervertebral disc, is resolved, whether this is by treatment or by the passage of time.

Epilogue. Manfred Zimmermann [19] in his review of neuropathic pain mechanisms points out that there is a spread of the disease process well beyond the original site of injury. Even if the injury is peripheral, there may be spread into the central nervous system, with widespread sensitization of neurons. Injury in the pelvic region seems to follow a similar pattern, the original injury producing repercussions at several levels of organization, of which conscious proprioception is but one, albeit an important one. In all cases the aim must be to repair the initial fault, although in many cases, of neuropathic pain particularly, this may be almost impossible.

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