Workplace Safety and Insurance
Appeals Tribunal

Tribunal d'appel de la sécurité professionnelle
et de l'assurance contre les accidents du travail

Limping and Back Pain

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This medical discussion paper will be useful to those seeking general information about the medical issue involved. It is intended to provide a broad and general overview of a medical topic that is frequently considered in Tribunal appeals.

Each medical discussion paper is written by a recognized expert in the field, who has been recommended by the Tribunal's medical counsellors. Each author is asked to present a balanced view of the current medical knowledge on the topic. Discussion papers are not peer reviewed. They are written to be understood by lay individuals.

Discussion papers do not necessarily represent the views of the Tribunal. A vice-chair or panel may consider and rely on the medical information provided in the discussion paper, but the Tribunal is not bound by an opinion expressed in a discussion paper in any particular case. Every Tribunal decision must be based on the facts of the particular appeal. Tribunal adjudicators recognize that it is always open to the parties to an appeal to rely on or to distinguish a medical discussion paper, and to challenge it with alternative evidence: see *Kamara v. Ontario (Workplace Safety and Insurance Appeals Tribunal)* [2009] O.J. No. 2080 (Ont Div Court).

LIMPING AND BACK PAIN

Introduction

The WSIB Tribunal is often asked to deal with appeals related to leg and knee conditions caused or aggravated by previous compensable back injuries that have progressed to the development of degenerative lumbar disc disease and mechanical low back pain. It is often claimed, for example, that a compensable back condition with acute sciatica has resulted in limping, which in turn has caused a tear of a knee meniscus. The opposite claim is also made; that a preexisting congenital or degenerative back disorder is aggravated by limping, secondary to a compensable knee injury such as a meniscal tear or post-traumatic chondromalacia of the patella. On occasion, it is claimed that a meniscal tear of one knee and a lumbar disc problem was caused by limping as a result of a compensable opposite knee condition such as a tear of one of its menisci. Bilateral plantar fasciitis has also been reported to cause aggravation of a pre-existing back, hip or knee condition secondary to limping, precipitated by foot pain. There is very little information in the medical literature about limping. Most clinicians have a limited understanding of the impact of limping on the musculo-skeletal system and in general, believe that limping causes the patient to put extra weight on the opposite normal leg, causing it and the limping individual's spine to transmit increased loads while walking. To understand the problem, some knowledge of limping as well as the pathophysiology of back pain is required.

Pathophysiology of back pain

Back pain is common. It is often multi-factorial - including mechanical, physiological and neurophysiological contributors. It is difficult, therefore, to confirm a specific anatomical diagnosis for each patient. Even if a pain generator is suspected, it is not clear how this can be reliably confirmed to be the cause of a patient's perceived pain, impairment or disability in the face of complex social, emotional and neurophysiological founders. A pain generator describes a patho-anatomic site from which the primary cause for the patient's low back pain is thought to originate and is usually considered a pathological structure as the sole cause of the patient's disability. Congenital structural conditions which may cause back pain include entities such as spondylolysis (a bony defect in the posterior portion of the lumbar vertebra), which in turn may cause spondylolisthesis (a shift of one vertebra relative to the other due to resultant instability at that particular level), spina bifida and transitional (abnormally shaped) vertebrae at the lumbo-sacral junction. Acquired causes of back pain, from a WSIB perspective, are usually related to injury of the spine involving either soft tissue elements, i.e. abdominal and lumbar musculature, spinal ligaments and discs or injuries to bone such as vertebral fractures. Other causes of back pain include infection, malignancy - either primary or metastatic, and vascular problems such as an abdominal aneurysm (Ref. WSIB Position Paper, re: Back Pain by

Dr. W. R. Harris and J. R. Fleming, March, 1997 - Revision February, 2003). These are rarely WSIB related.

Adjacent spinal vertebrae are coupled anteriorly by a soft tissue disc, posteriorly by facet joints and by interconnecting ligaments (Fig. 1). The central portion of the disc is composed of an incompressible gel, which is contained by a tough, outer fibrous wall connecting the vertebral bodies (Fig. 2). Disc herniation occurs when the outer fibrous coating, annulus fibrosis, is breached either from injury or by the normal aging degenerative wear and tear process that allows the gel-like central material to escape or herniate into the spinal canal, sometimes compressing an adjacent nerve root. This may cause sciatica.

Disc herniation is more common in younger individuals because the central disc material, the nucleus pulposus, remains in a semi-fluid gel-like state. With increasing age, the annulus fibrosis and the nucleus pulposus lose some of their elasticity and shrinkage of the disc occurs. This is seen on plain X-rays of the spine as a decrease in the height of the vertebral disc. Progressive narrowing of the intervertebral disc space with increasing age is associated with degenerative change in the posterior facet joints and causes osteophytes or bone spurs to develop along the bony margins of the adjacent vertebral bodies. With further narrowing of the disc, joint motion is reduced between vertebral segments and the joint between the vertebrae becomes stiff. From a biomechanical perspective, the normal disc is very resistant to compression. The nucleus pulposus does not alter in shape or position with compression forces and plays no active part in producing a disc prolapse. Upon compression, an adjacent vertebral body always breaks before the normal disc gives away. A combination of compression and rotation is required to produce disc herniation and sequestration.

Diagnosis

Although X-rays show evidence of degenerative disc disease, (spinal arthritis), e.g. disc space narrowing and vertebral spurring, there is usually little correlation between plain X-rays of the lumbo-sacral spine and specific symptoms. A CT scan or MRI will often show a disc herniation causing sciatica but because there is no clear differentiation between disc and neural tissue, a CT scan with contrast (dye injection) may be required to clearly outline the dural sac containing the spinal cord and exiting nerve roots. An MRI is non-invasive. There is no radiation involved. Better than a CT scan, it usually provides good visualization of neural tissues. Clinical correlation, i.e. history and physical findings that correlate with radiological assessment is required in order to establish an accurate diagnosis of the possible causes of back and lower extremity pain, as abnormalities are commonly seen in CT or MRI images in people with absolutely no symptoms. Although increasingly elaborate methods of back pain assessments are being developed, i.e. CT, MRI scans etc. to identify the pain generator, no method exists to absolutely confirm that the spine abnormalities detected by radiological investigations are truly the primary source of the patient’s

back problem. The prevalence of MRI positive findings related to examinations of healthy adults indicates that a significant percentage of MRI abnormalities are present in asymptomatic subjects. The incidence of false positives tends to increase with age. For all of these reasons, it is often difficult to identify a specific pain generator of back pain. It is important, therefore, that the physical findings correlate with radiological abnormalities to be of significance.

The mechanics of limping

Minimal force in the anteriorly located abdominal or posterior erector spinal muscles (core muscles) is required to balance the spinal column. Any condition that results in major displacement of the centre of gravity of the body's mass away from the vertical axis of the spine, e.g. forward and/or lateral bending, lifting, a large protuberant abdomen and/or weak abdominal musculature, will create increased forces in the stabilizing posterior erector spinal muscles in order to balance the spine. This, in turn causes increased force transmission across the spine segments and an increase in disc pressure. Intradiscal pressures as a result of activity are the least with sleeping (lying down) and greatest with sitting, bending and lifting. Major side-to-side (lateral) displacements of the body's centre of gravity can also increase spinal load due to increased force transmission by the abdominal and paraspinal muscles (core musculature) required to stabilize spinal segments. Increased spinal motion as a result of abnormal displacements of the body's centre of gravity while walking, will also contribute to disc breakdown, particularly at the lumbo-sacral region. These are the basic biomechanical mechanisms related to limping as a cause of low back pain, i.e. the generation of increased lateral bending and rotational forces in the core musculature due to the combined repetitive side to side and vertical displacements of the body's centre of gravity and increased motion at the lumbar disc levels while walking with a limp.

Types of limping

Limp is defined as an uneven, jerky or laborious gait usually caused by pain, weakness or deformity. There are many causes of limps and it is not possible to review them all. The reader is referred to the discussion paper "Symptoms in the Opposite or Uninjured Leg" by W. R. Harris and Ian J. Harrington (August 2005). This outlines the mechanics of normal gait and how it is affected by limping. There are three basic limps; paralytic, antalgic and short leg.

Paralytic

In the paralytic type, one or more muscles are weakened by disease, e.g. poliomyelitis, or injury, e.g. to a nerve supplying a muscle. For example, if the muscles that move the leg away from the body and stabilize the pelvis (the abductors

of the hip) are weak, then, as the patient walks on the weakened or painful lower extremity, his upper (torso) body will tend to lean towards the affected leg, producing a characteristic lurch called a Trendelenburg lurch or limp. This is frequently associated with movement of the upper extremity on the limp side away from the body (Fig. 3). This motion of the upper extremity helps to shift the centre of gravity of the total body mass (c.g.) towards the affected lower extremity, thereby producing a decrease in joint force transmission at the hip, knee and ankle of the weakened, painful or shortened limb due to limited muscle activity involving these joints.

Another example of paralytic limp occurs when the muscles that lift the forefoot off the ground are weak; this causes the forefoot to drop during the swing phase of gait (a drop foot). To prevent stubbing of the toes, tripping and falling, it is necessary for the patient to lift his leg higher during its swing phase in order that his foot can clear the ground producing a characteristic gait called “steppage” or “dangle foot.” Because more time is required to get the paralyzed leg into position, its swing phase is prolonged. This, in turn, means that the stance phase of the opposite normal leg is prolonged while this limb waits for the weak leg to “catch up.” As a result, force generated by the musculature of the normal limb is transmitted over a longer period of time. In addition to these examples, hemiplegia (stroke), cerebral palsy and other neurological causes may also cause a limp due to lower extremity muscle impairment. The limp associated with amputation also fits into the paralytic type. The predominant amputee walking pattern is that of a Trendelenburg lurch on the side of the amputated limb, particularly evident with above knee amputations.

Above-the-knee amputees have a Trendelenburg limp where the trunk arches towards the artificial leg, which in turn activates the spinal musculature as, described above. As a result, amputees often complain of back discomfort. It is worth noting that both the Department of Veterans’ Affairs and the Workplace Safety and Insurance Board recognize this in assessing the amount of pension to be awarded. This does not occur as frequently in below-the-knee amputees who, in most cases, retain a normal gait without exaggerated displacements of the body’s centre of gravity and essentially normal energy expenditure.

Antalgic

The easiest way to picture an antalgic gait is to imagine that a stone is in your shoe or a nail is sticking through its sole. It hurts when you take weight on that foot and you lessen the discomfort by getting off it as quickly as you can. In other words, you shorten the duration of the stance phase on this side. This produces a characteristic gait with uneven strides of different duration, whereby the stance phase of the painful limb is shortened and that of the normal leg is increased. Any condition that causes pain in a lower extremity bone or soft tissue injury or referred pain such as sciatica,

can produce an antalgic gait. This type of gait abnormality is often a response to an acute short term problem. Foot and ankle injuries are a common cause of an antalgic gait.

Short Leg (leg length inequality)

In this limp, there is a dip when the short leg is in stance phase. But how short is short? A study related to the effects of limb length discrepancy on gait economy in lower extremity muscle activity in older adults (Journal of Bone and Joint Surgery, American, 83A, No. 6, p. 907-915, 2001) reported that the amount of limb length discrepancy necessary to adversely affect gait parameters in older adults was unknown and that the information provided was largely anecdotal. In this particular study, subjects were assessed while walking on a treadmill at a self-elected normal walking pace with artificial simulated limb length discrepancies of 0, 2, 3 and 4 cm. applied in a randomly selected order. Indirect calorimetry was used to measure oxygen consumption and minute ventilation. Electromyography was used to measure muscle activity of the quadriceps, plantar flexors, gluteus maximus and gluteus medius muscles of both lower extremities. Heart rate and frequency of gait compensation patterns were also measured. The results of this study demonstrated an increase in oxygen consumption and the rating of perceived exertion with a 2, 3 and 4 cm. artificial limb length discrepancy. There was a significant increase in heart rate, minute ventilation and quadriceps activity (muscle force) with a 3 and 4 cm. limb length discrepancy and an overall increase in total energy expenditure. It was concluded that both oxygen consumption and the rating of perceived exertion was increased with a limb length discrepancy and that a 3 cm or greater discrepancy was likely to induce significant quadriceps fatigue in the longer limb. As a result, it was postulated that the longer leg would be more likely to develop osteoarthritis affecting the hip, knee and ankle rather than the shorter limb, i.e. increased muscle force generates increased joint loading.

The results of studies to date, based on clinical observations, however, i.e. X-rays, clinical reviews etc. have not been conclusive. Most investigations have suggested that osteoarthritis is more prevalent in the weight bearing joints of the longer leg, whereas some have suggested that it occurs more frequently in the shorter limb. All studies, however, have concluded that leg length discrepancy increases the risk of osteoarthritis occurring in both knees.

From a purely musculo-skeletal biomechanical perspective, it is more likely that wear and tear due to increased joint bearing load would occur in the longer leg. The best way to think of this is to imagine a normal individual walking parallel to a shallow trench of variable depth, of 5 to 10 cm, with one foot in the trench while walking and the other on the adjacent level ground (simulated leg length discrepancy). With each step during the weight bearing phase of the gait cycle when one foot is in the trench, it will be necessary to flex the hip and knee and dorsiflex the foot of the opposite limb

(simulated longer leg) in order to clear the ground during the trench limb's stance phase. When the stance phase of the simulated longer limb (not in the trench) begins the knee and hip of that extremity would be flexed. Vigorous contraction of the muscles stabilizing each major joint (knee quadriceps and hip gluteus medius and maximus) of the longer limb would be required in order to elevate the centre of gravity of the body's mass so that the limb in the trench would be able to commence its swing phase of gait. This mechanism will generate significant force transmission across the hip, knee, foot and ankle in the leg not in the trench, i.e. simulated longer leg. As well, the centre of gravity of the body's mass will undergo increased vertical, lateral and rotational displacements, generating an increase in overall energy expenditure. The greater the displacement of the centre of gravity, i.e. directly related to the magnitude of the leg length discrepancy, the greater the overall force generated and transmitted by the extremity not in the trench (simulated longer leg) during that limb's stance phase. The joint motions of the lower extremity while walking are interdependent; loss of motion in one joint negatively impacts all the other joints. The inability of the knee, for example, to bear weight or bend and straighten leads to a modified gait pattern which places additional load on the other joints in the same leg, opposite leg and lower back.

Scientific data obtained to date from clinical, X-ray, gait and biomechanical studies, however, has not clearly defined the magnitude of leg length discrepancy necessary to generate increased force transmission by the normal leg. From a purely biomechanical perspective, a discrepancy of less than 3 or 4 cm. would unlikely affect the back, hip or knee. Due to the complexities and uncertainties of these problems and lack of pure scientific data, each person must be looked at individually.

The biomechanical effects of limping

1) Does limping affect the low back?

It is often claimed that degenerative disc disease of the lumbo-sacral spine develops as a result of injury to an extremity that in turn results in a limp.

In the absence of a limp, standing in an erect position or level walking is unlikely to create abnormal stress levels in the coupled vertebral segments. Under these conditions, the spinal segments are primarily subjected to axial compression loading, generally of low to moderate magnitude because the centre of gravity of the body is aligned over the vertical axis of the spinal column. In this situation, direct compression of a lumbar disc is unlikely to cause disc damage. Tearing of a disc annulus is more likely to occur with a combination of compression and, in particular, rotational forces.

Musculo-skeletal disorders, however, disturb normal movements of the body segments during gait. The motion of the centre of gravity (c.g.) of the body representing the whole body system of movement is the ultimate result of both energy expenditure and motions of the body segments. The work done by muscles

to translate the centre of gravity (external work) with respect to the ground is one determinant of the energy expenditure of gait. Abnormal movements influence the motion of the centre of gravity of the body.

With limping, there is a shift of the body's centre of gravity towards the affected leg. This results in lateral bending of the trunk towards that side (Fig. 4). Depending on the magnitude of the limp, there will be an exaggerated side to side and vertical displacement of the body's centre of gravity. When weight is transferred to the good leg, the repositioning of the centre of gravity in the mid-line is in part due to the pull of the para-lumbar and abdominal musculature as well as the hip abductor muscles on the normal side. The increased muscle pull increases the force transmitted across the lumbar discs, facet joints, hip, knee and ankle due to mechanical leverage. This produces a seesaw effect where the disc centres become the centres of rotation or fulcrum for the para-lumbar muscle force, necessary to balance body weight acting through the centre of gravity through the body's mass. This is a lever system of the first class. Repetitive pull of the trunk musculature could, in time, result in increased wear and tear of the disc segments since the force transmitted across the discs by the trunk musculature would, in theory, be greater for an individual who limps than for someone with a normal gait. This, in turn, might cause or aggravate degenerative change (osteoarthritis) of the disc and facet joints. Limping as a result of muscle paralysis combined with a leg length discrepancy in the same individual, would, from a biomechanical perspective, generate the greatest lumbar disc load transmission due to major vertical and horizontal displacements of the centre of gravity of the body mass required to maintain spinal equilibrium. This type of limp would also result in the greatest energy expenditure.

Another biomechanical factor operative here is the compensatory lumbar scoliosis that occurs for those patients whose limp is due to significant leg length discrepancy. Scoliosis due to any cause accelerates the degenerative process of the spine. The above biomechanical actions are occurring in the frontal (i.e. as viewed from the front) plane. However, the same mechanism will occur in the sagittal (i.e. as viewed from the side) plane, particularly for those patients whose limp is due to limb paralysis resulting in compensatory trunk movements in both planes in order to keep the centre of gravity centered over the affected limb while walking. Extension of the spine, i.e. leaning backwards, for example, is necessary for equilibrium while walking in individuals with gluteus maximus (buttock) muscle weakness. The net result is an increase in load transmitted by the spine due to increased contraction of the core paralumbar and abdominal musculature required to stabilize the spine during repetitive side to side and forward/backward bending of the trunk and torso while walking.

Because each type of limp causes exaggerated bending and rotation of the trunk, it is probable that over time, this could accelerate normal aging change and thus cause back symptoms. The L.4 - 5 and L.5 - S.1 spinal segments of normal individuals have the greatest motion in the lumbar spine. Greater motion causes an increased

potential for lumbo-sacral disc breakdown. The incidence of herniated disc is greater at L.4 - 5 and L.5 - S.1 than at any other lumbar disc space. From a purely biomechanical perspective, increased spinal segment motion due to repetitive and exaggerated lateral bending of the spine as a result of a significant limp, would enhance the potential for disc breakdown e.g. disc herniation and degenerative change, particularly at the L.4 - 5 and L. 5 - S.1 levels.

In patients with pre-existing back discomfort, limping would probably aggravate spine symptoms in direct proportion to the magnitude of the limp. The wear rates of engineering bearings in industry are related to a number of variables, i.e. lubrication, bearing materials, friction coefficients, etc. but the two wear variables that are constant for any type of bearing is the relationship between bearing load and sliding distance relative to the individual bearing surfaces. These two factors are constant in the wear process. Overall load and sliding distance have been well established as parameters of wear in artificial joints, i.e. hip and knee replacements etc. and it is assumed that total load/sliding distance also applies to biological joints. It is known, for example, that obesity plays a significant role in accelerating osteoarthritis, as does malalignment whereby weight bearing joint stresses are increased. It has been reported that there is strong epidemiological evidence that being overweight or obese and suffering a knee injury, is associated with increased risk of developing knee osteoarthritis (Ref. Neogi, Tuhina, Zang and Yuqing. Osteoarthritis Prevention, Current Opinion in Rheumatology 23 (2); 185-191, March, 2011).

The magnitude of force (load) transmitted by biological joints is directly related to:

- obesity
- joint deformity
- stride length
- limb length discrepancy
- walking speed
- presence of a Trendelenburg gait or any gait pattern where there are major displacements of the centre of gravity of the body’s mass.

Conclusion

In all probability, from a biomechanical perspective, limping can cause back pain and aggravate pre-existing back pain. Clinical data, however, i.e. patient studies directly related to the incidence of back pain in the general population for individuals walking with a limp, are limited and inconclusive. Therefore, each case must be considered individually and all the above factors considered.

However, in the case of an antalgic gait secondary to leg pain due to sciatica, it would probably be necessary for the limp to be severe and prolonged, meaning years, for it to have a significant impact on the initiation or aggravation of arthritis of the spine. As well, it would also be necessary for the Trendelenburg gait pattern to have been severe and present for an extended period of time, probably years, to have any permanent effect on the spine.

It is also unlikely that injuries such as a meniscal tear involving either the medial or lateral meniscus or any condition, e.g. chondromalacia of the patella etc., that caused a mild or moderate degree of limping over a relatively short period of time would have a major detrimental effect on the lumbar spine or opposite lower extremity.

Foot and ankle problems causing a temporary limp of low magnitude are also unlikely to create load transmission of significant magnitude to cause additional stress on the spine or other leg.

Evaluating appeals

In evaluating these appeals, a Panel must establish:

1. That the limp was documented.
2. The limp was caused by the compensable injury
3. If possible, the type and magnitude of the limp and leg length discrepancy, if present.
4. Did the limp pre-exist the compensable injury? If so, were there associated back symptoms?
5. The duration of the limp
6. Was there pre-existing back discomfort? If so, was it aggravated by the work related limping?

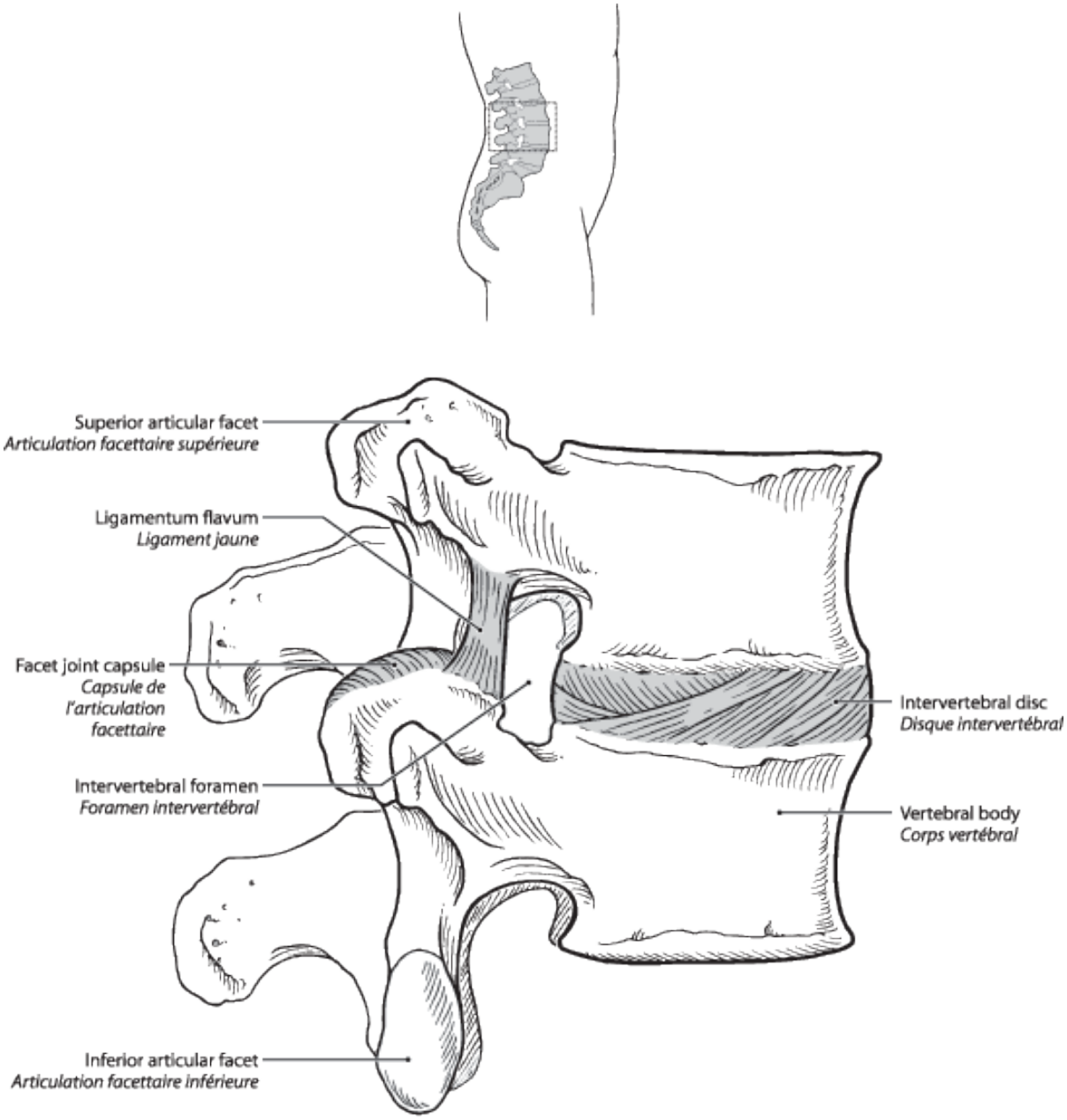
Can a back condition affect the knee?

It is often claimed that a previous compensable back injury with degenerative disc disease and resulting mechanical low back pain can cause or aggravate osteoarthritis of the knees due to increased knee joint stress precipitated by the back problem. The exact cause of knee osteoarthritis is uncertain but it is known that factors such as excessive body weight, injury or any activity that creates excessive force transmission across the bearing surface of the hip, knee and ankle joints can initiate or aggravate an existing arthritic condition due to the wear and tear phenomena.

Lower extremity joint force tends to increase with walking speed so that overall force transmission is greater for young, fit individuals than for those who are elderly and disabled and have a slow shuffling gait. It is commonly believed that injury to one leg can precipitate symptoms in the opposite uninjured limb insert (see discussion paper “Symptoms in the Opposite or Uninjured leg” by W. R. Harris and I. J. Harrington). From a biomechanical perspective, however, it is unlikely that an injury to one leg can cause major problems with the opposite uninjured leg except for certain specific conditions, i.e. a major leg length discrepancy where the injured leg becomes significantly shorter than the normal leg, there is a significant limb deformity, or when a severe Trendelenburg lurch develops as a result of injury or paralysis of a lower extremity. It follows then, that a back problem is unlikely to cause increased force transmission at either hip, knee, foot or ankle unless the spinal injury is of such severity that it results in lower extremity muscle paralysis due to spinal cord (myelopathy) or nerve root damage (radiculopathy) that in turn causes a pronounced limp or significant lower extremity functional abnormality; the opposite normal leg (hip, knee and ankle) could then be subjected to increased load (force transmission) - (see discussion paper “Symptoms in the Opposite or Uninjured leg” by W. R. Harris and I. J. Harrington and Ref. Harrington IJ, Harris WE: Can “Favouring” One Leg Damage the Other? Editorial - Journal of Bone and Joint Surgery, Vol. 76-B, No.4, Page 519. July, 1994).

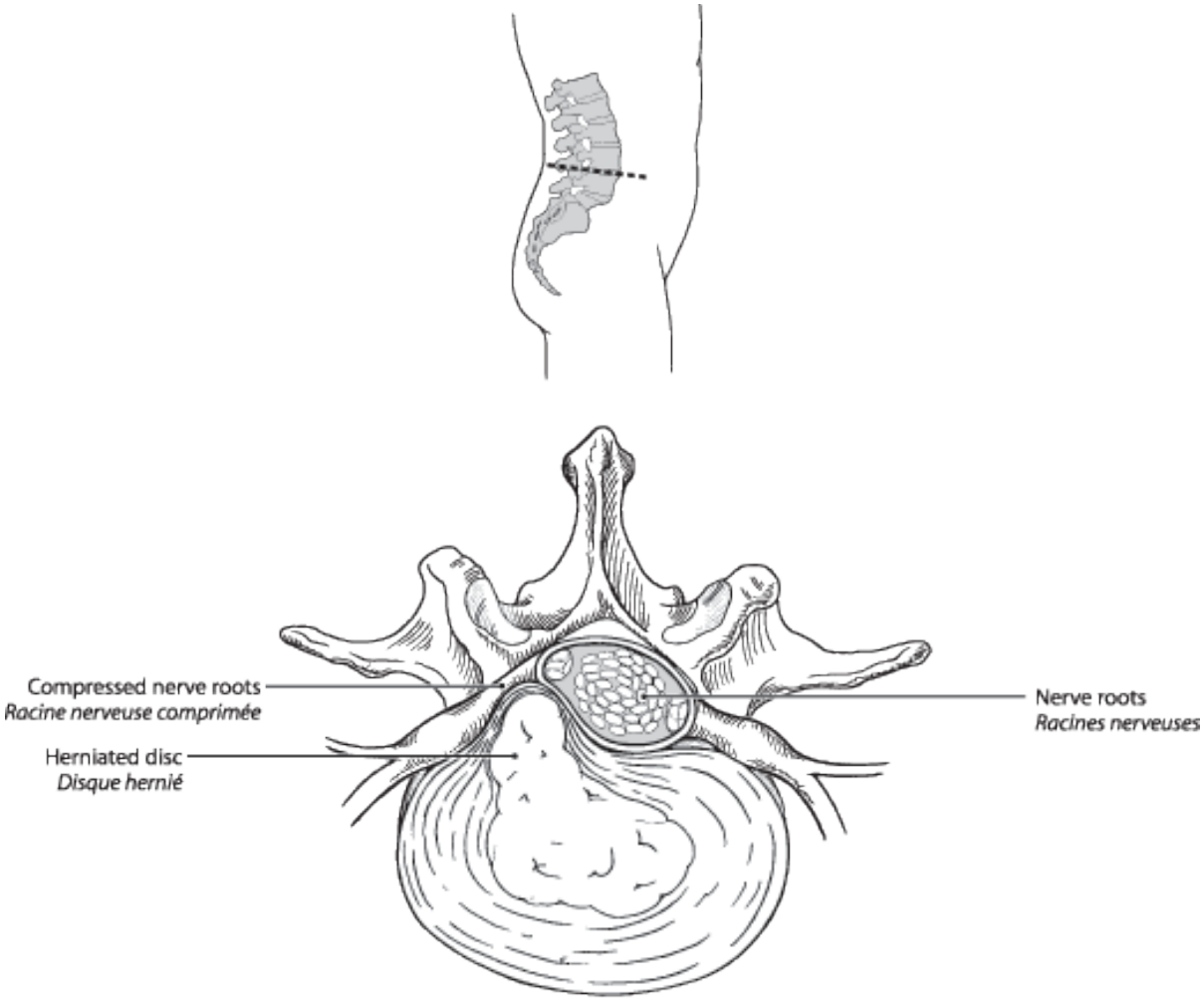
Since the activity level of patients with chronic low back pain is usually limited, i.e. decreased stride length and walking speed, it is unlikely that either lower extremity would be subjected to greater than normal force since the magnitude of joint force transmission for both lower extremities is related to walking speed and the generation of acceleration forces by the limb segments while walking.

Back problems of lesser magnitude causing mechanical low back pain without a significant alteration in gait pattern, i.e. major limp with Trendelenburg lurch, are unlikely to cause increased force transmission in the weight bearing joints of the lower extremities. These types of back conditions are not likely to generate the magnitude of force necessary to cause a meniscal tear, aggravate pre-existing osteoarthritis or cause osteoarthritis of the hip, knee or ankle. Meniscal tears are usually caused by a major combined compression and twisting force (torque) which would not occur from a minor alteration in gait pattern. Age related degenerative change with tearing of the meniscus, however, are common.



Side view of normal vertebrae showing ligaments and intervertebral disc
Vue latérale d'une vertèbre normale montrant les ligaments et le disque intervertébral

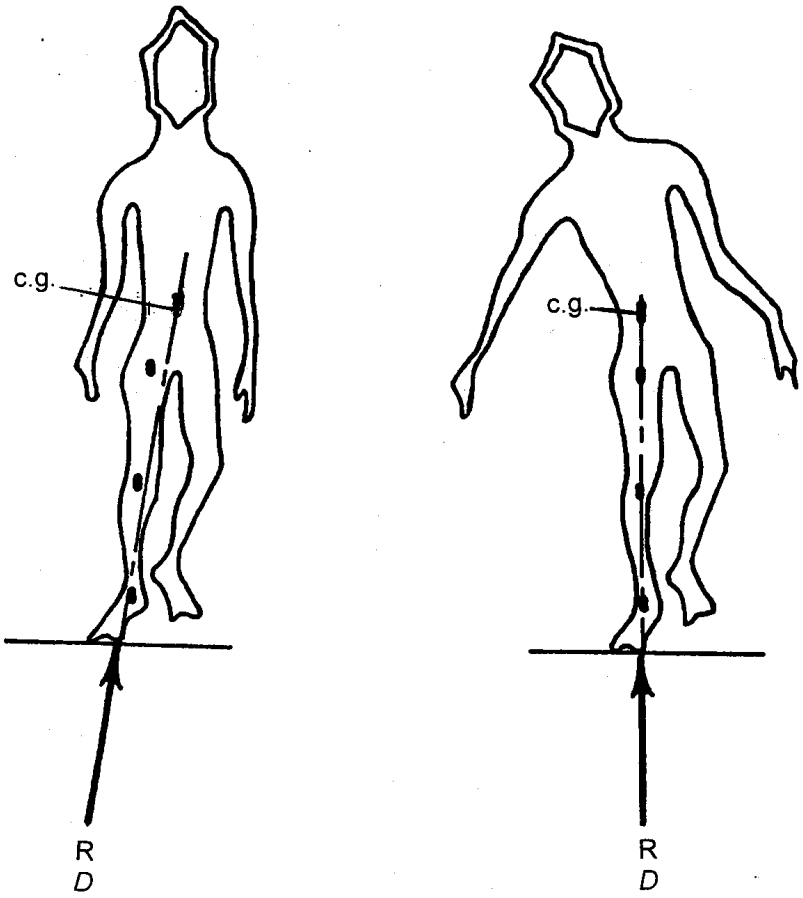
Figure 1 - Side view of normal vertebrae showing ligaments and intervertebral disc



A ruptured (herniated) nucleus pulposus compressing the nerve root, as seen on cross-section.

Coupe transversale montrant un nucleus pulposus hernié comprimant la racine nerveuse.

Figure 2 - A ruptured (herniated) nucleus pulposus compressing the nerve root, as seen on cross-section



TRENDELENBURG LURCH DUE TO
LIMPING CENTRE OF GRAVITY SHIFT
Boiterie de Trendelenburg entraînée par
un déplacement du centre de gravité

Figure 3 - Trendelenburg lurch due to limping centre of gravity shift

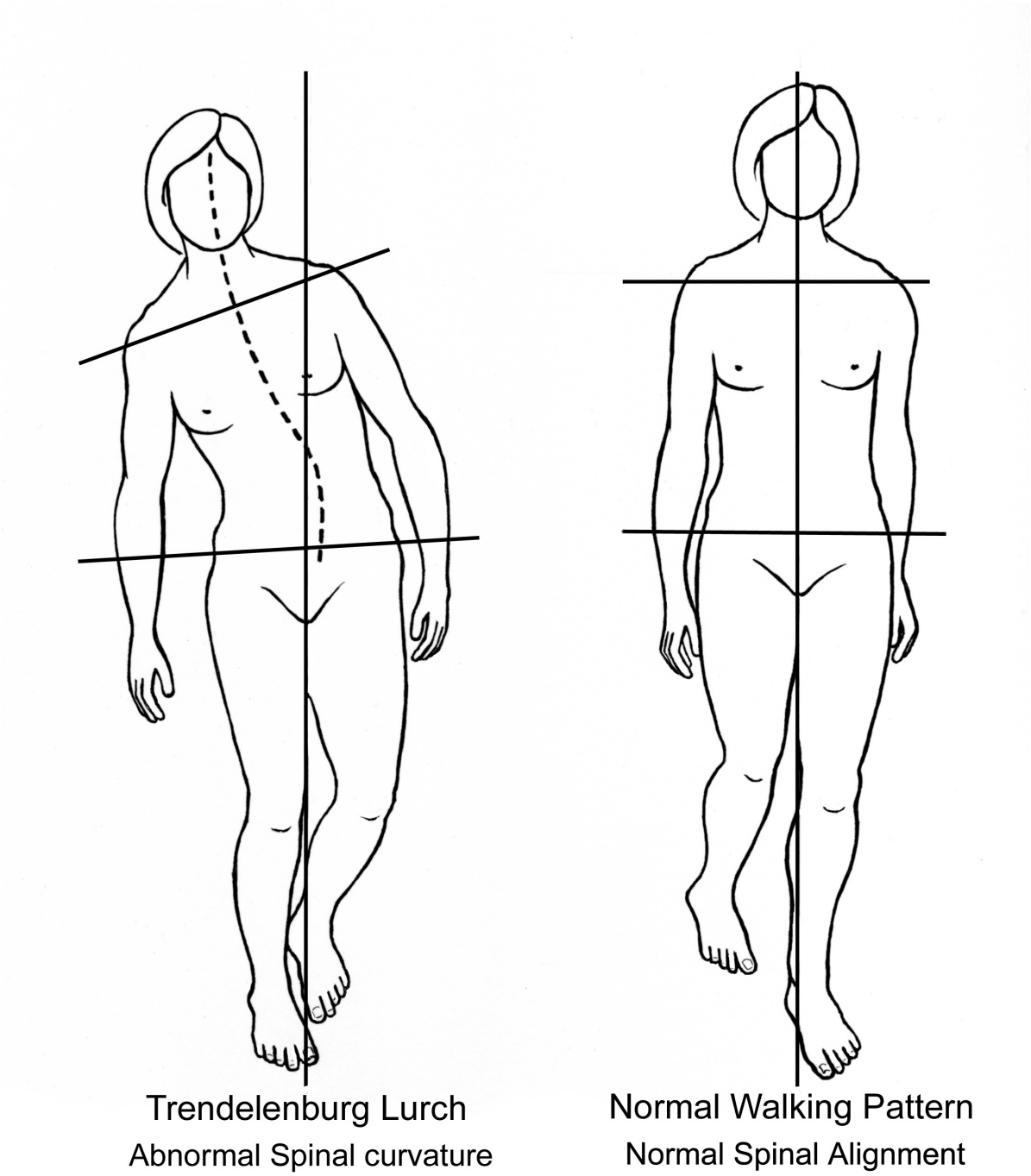


Figure 4 - Trendelenburg Lurch (abnormal spinal curvature) and normal walking pattern (normal spinal alignment)

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