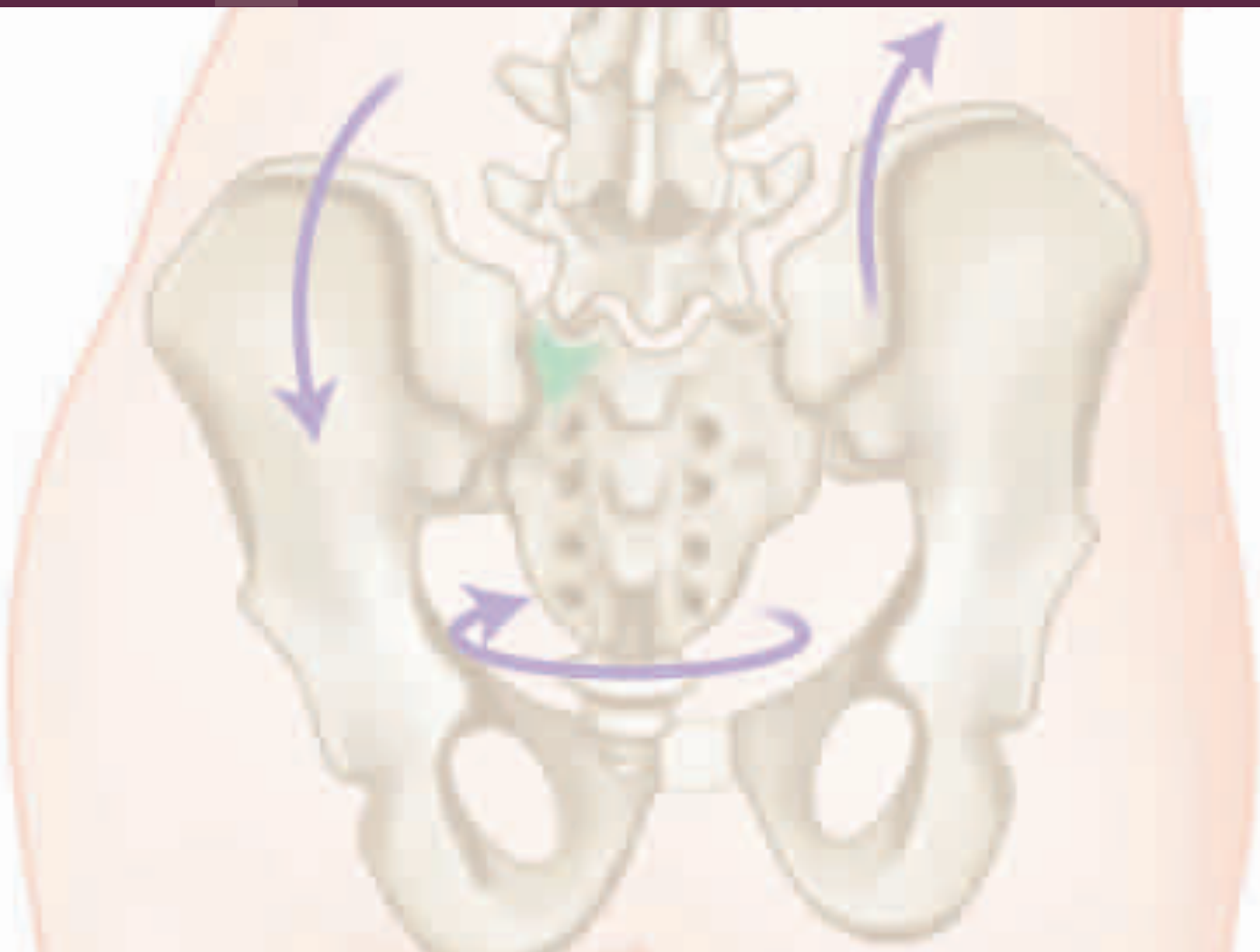
4

The Walking/Gait Cycle and Its Relationship to the Pelvis



Most of us, I would say, take walking for granted—it is something that we just do without understanding what exactly is going on … until we suffer pain somewhere in our body, and then the simple action of walking becomes very painful. What I would like to do in this chapter is examine in detail what exactly takes place when we walk (you might want to go through some of the movements yourself as they are described) and the relationship of the gait cycle to both the pelvis and the kinetic chain.

Gait Cycle

> **Definition:** A *gait cycle* is a sequence of events in walking or running, beginning when one foot contacts the ground and ending when the same foot contacts the ground again.

The gait cycle is divided into two main phases: the *stance* phase and the *swing* phase. Each cycle begins at initial contact (also known as *heel-strike*) of the leading leg in a stance phase, proceeds through a swing phase, and ends with the next contact of the ground with that same leg. The stance phase is subdivided into *heel-strike*, *mid-stance*, and *propulsion* phases.

Human gait is a very complicated, coordinated series of movements. An easier way of thinking about the gait cycle is to break it down into phases. The stance phase is the weight-bearing component of each cycle; it is initiated by heel-strike and ends with toe-off from the same foot. The swing phase is initiated with toe-off and ends with heel-strike. It has been estimated that the stance phase accounts for approximately 60% of a single gait cycle, and the swing phase for approximately 40%, as shown in Figure 4.1.

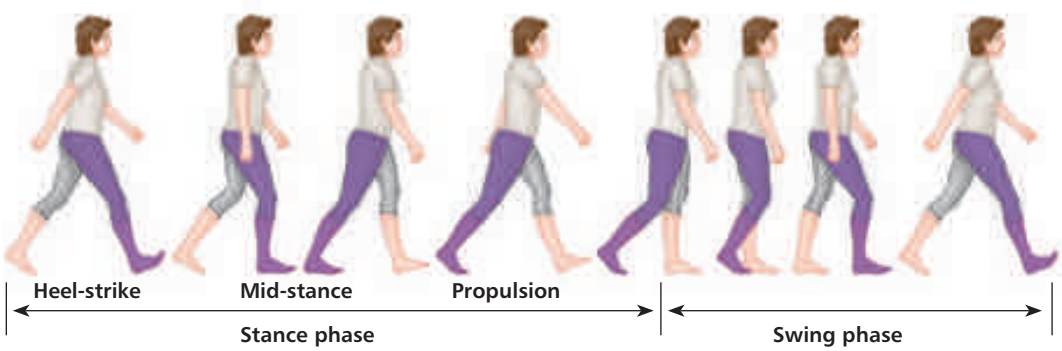


Figure 4.1. Stance and swing phases of the gait cycle.

Heel-Strike

If you think about the position of your body just before you contact the ground with your right leg during the contact phase of the stance phase, the right hip is in a position of flexion, the knee is extended, the ankle is dorsiflexed, and the foot is in a position of supination, as shown in Figure 4.2. The tibialis anterior muscle, with the help of the tibialis posterior, works to maintain the ankle/foot in a position of dorsiflexion and inversion (inversion is one part of the motion referred to as *supination*).

In normal gait, the foot strikes the ground at the beginning of the heel-strike in a supinated position of approximately 2 degrees. A normal foot will then move through 5–6 degrees of pronation at the subtalar joint (STJ) to a position of approximately 3–4 degrees of pronation, as this will allow the foot to function as a "mobile adaptor."

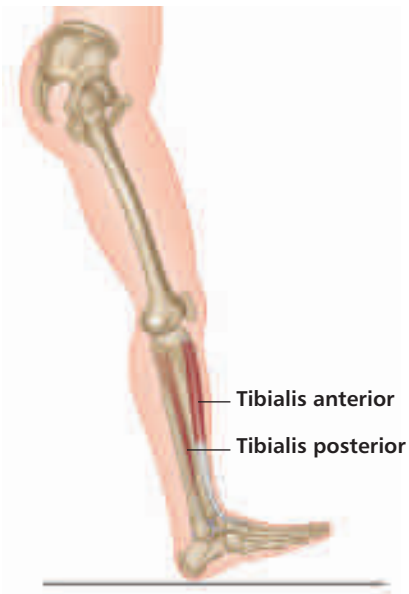


Figure 4.2. The position of the leg just before heel-strike.

A Myofascial Link

As a result of the ankle and foot being in a position of dorsiflexion and supination, the tibialis anterior (which is the main muscle responsible for this anatomical position, with an insertion on the medial cuneiform and 1st metatarsal on the foot) is now part of a link system that we will call a *myofascial sling* (see Chapter 3). This sling, starting from the initial origin of the tibialis anterior, continues as the insertion of the peroneus longus (onto the 1st metatarsal and medial cuneiform, as in the case of the tibialis anterior) to its muscular origin on the lateral side and head of the fibula. This bony landmark is also where the biceps femoris muscle inserts.

The sling now continues as the biceps femoris muscle toward its origin on the ischial tuberosity, where the muscle attaches to the tuberosity via the sacrotuberous ligament; often the biceps femoris directly attaches to this ligament rather than to the ischial tuberosity, and some authors have mentioned that potentially 30% or more of the biceps femoris attaches directly to the ILA of the sacrum. If you think back to Chapter 1, I mentioned that Vleeming et al. (1989a) found that in 50% of subjects, part of the sacrotuberous ligament was continuous with the tendon of the long head of the biceps femoris.

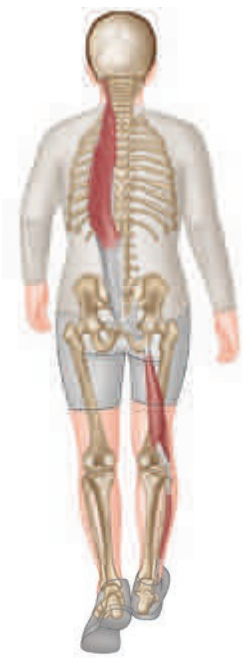


Figure 4.3. A person walking, with the posterior (deep) longitudinal sling muscles highlighted.

The sling then carries on as the sacrotuberous ligament, which attaches to the inferior aspect of the sacrum at the ILA and fascially connects to the contralateral (opposite side) multifidi and to the erector spinae, which continue to the base of the occipital bone. This myofascial sling is known as the *posterior longitudinal sling (PLS)* or the *deep longitudinal sling (DLS)*, as shown in Figure 4.3.

Even before you initiate the contact to the ground through heel-strike, dorsiflexion of the ankle (by the contraction of the tibialis anterior) initiates a coactivation of the biceps femoris and peroneus longus just prior to heel-strike. Studies have shown that the biceps femoris communicates with the peroneus longus at the fibular head, transmitting approximately 18% of the contraction force of the biceps femoris through the fascial system into the peroneus longus muscle. This co-contraction therefore serves to "wind up" the thoracolumbar fascia mechanism as a means of stabilizing the lower extremity; this results in the storage of the necessary kinetic energy that will subsequently be released during the propulsive phase of the gait cycle.

The posterior (deep) longitudinal sling as described is being fascially tensioned; the increased tension is focused on the sacrotuberous ligament via the attachment of the biceps femoris, as shown in Figure 4.4(b). This connection will assist the *force closure* mechanism process of the SIJ; in simple terms, this creates a self-locking and stable pelvis for the initiation of the weight-bearing gait cycle. You may also notice that the right ilium (see Figure 4.4(a–c)) undergoes posterior rotation during the swing phase, which will assist the force closure of the SIJ because of the increased tension in the sacrotuberous ligament.

You can also see from Figure 4.4(c), overleaf, that there is now tension developing within the right sacrotuberous ligament because of the contraction of the biceps femoris as well as the posterior rotation of the right innominate; at the same time, the left innominate is rotating anteriorly and the sacrum has rotated on the left oblique axis (L-on-L). This specific motion of the lumbopelvic hip complex occurs all at the same time as the right heel-strike.

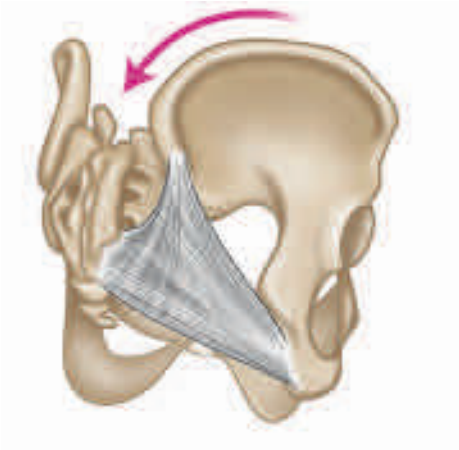


Figure 4.4. (a) Right ilium in posterior rotation—sacrotuberous ligament tensioned.

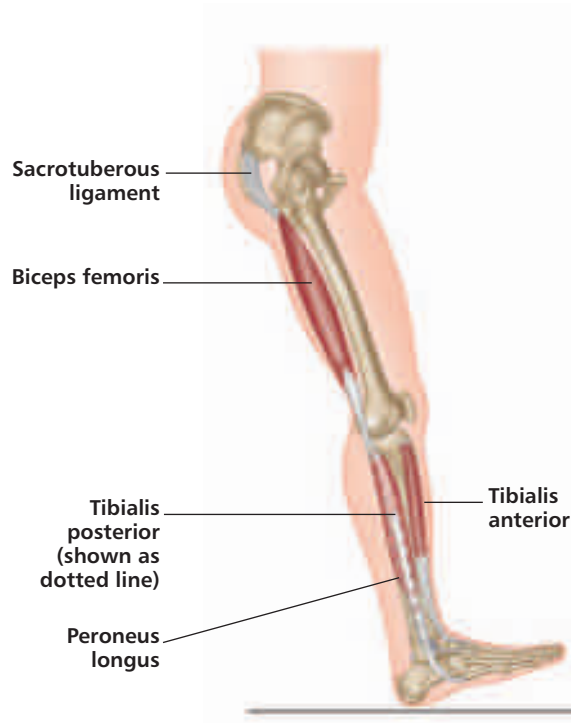


Figure 4.4. (b) Position of the leg just before heel-strike, with the biceps femoris and sacrotuberous ligament tensioned.

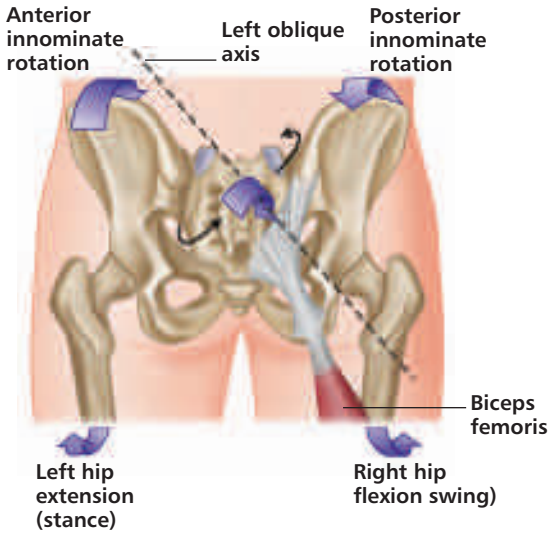


Figure 4.4. (c) Right ilium in posterior rotation—left ilium in anterior rotation and sacrum rotated on the L-on-L axis.

For the next phase, you might want to stand and slowly go through the following movements so that you can get a sense of what happens with your body in the normal walking cycle. As explained above, just before the heel-strike phase your hip will be flexed, your knee extended, and your ankle dorsiflexed with the foot supinated. The tibialis anterior and tibialis posterior maintain this position of the ankle and foot, and as you contact the ground, these two muscles are responsible for controlling the rate of pronation through the STJ by contracting eccentrically.

As your right leg moves from heel-strike to toe-off (stance phase), your body weight begins to move over your right leg, causing your pelvis to shift laterally to the right. As the movement continues toward toe-off, your right pelvic innominate bone begins to rotate anteriorly while your left innominate bone begins to rotate posteriorly.

As you proceed through the gait cycle, you now enter the mid-stance phase of gait. This is where the hamstrings should reduce their tension because of the natural anterior rotation of the pelvis and the slackening of the sacrotuberous ligament. Form closure at this point is gradually lost during the latter part of the stance phase, so that stability at this point is chiefly maintained through force closure. This is the point during the mid-stance phase where the Gmax on the right side should take the role of the continued movement of lower limb extension, as well as working in concert with the contralateral latissimus dorsi (left side). The active contraction of these two muscles increases the tension in the thoracolumbar fascia (posterior oblique sling), thus providing the necessary force closure stability to the right SIJ during the mid-stance phase of gait.

I would like to elaborate a little more on this process. Phasic contraction of the Gmax occurs in the mid-stance phase; the Gmax simultaneously contracts with the contralateral latissimus dorsi—it is this muscle that will extend the arm through what is known as *counter-rotation*, to assist in propulsion. The thoracolumbar fascia, which is a sheet of connective tissue, is located between the Gmax and the contralateral latissimus dorsi; this fascial structure is forced to increase its tension because of the contractions of the Gmax and latissimus dorsi. This increased tension will assist in stabilizing the SIJ of the stance leg through the force closure mechanism.

In Figure 4.5 you can see that just before heel-strike, the Gmax will reach maximum stretch as the latissimus dorsi is being stretched by the forward swing of the opposite arm. Heel-strike signifies a transition to the propulsive phase of gait, at which time the Gmax contraction is superimposed on that of the hamstrings.

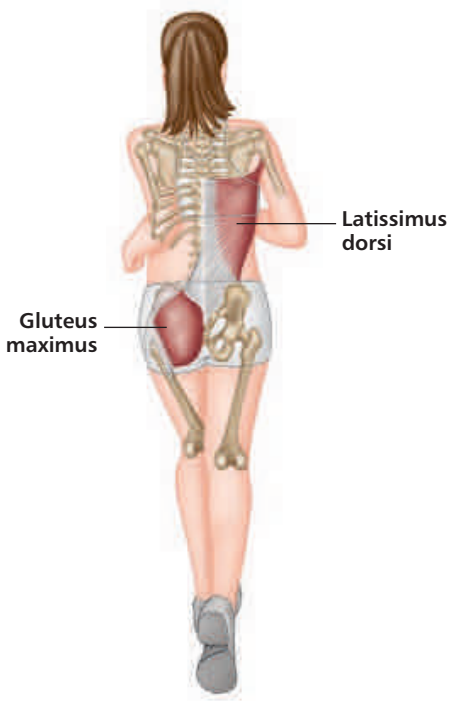


Figure 4.5. A person running, with the posterior oblique sling muscles highlighted.

As explained in the previous paragraphs, activation of the Gmax occurs in concert with contraction of the contralateral latissimus dorsi, which is now extending the arm in unison with the propelling leg. The synergistic contraction of the Gmax and the contralateral latissimus dorsi creates a state of tension within the thoracolumbar fascia, which will be released in a surge of energy that will assist the muscles of locomotion. This stored energy within the thoracolumbar fascia helps to reduce the overall energy expenditure of the gait cycle. Janda (1992, 1996) mentions that poor Gmax strength and activation is postulated to decrease the efficiency of gait. The posterior oblique sling also contains a lower component (consisting of the continuations of the Gmax), which acts to increase the tension of the iliotibial band (ITB); this helps to stabilize the knee during the stance phase of gait.

As we progress from the mid-stance phase to heel-lift and propulsion, the foot begins to re-supinate and passes through a neutral position when the propulsive phase begins; the foot continues in supination through toe-off. As a result of the foot supinating during the mid-stance propulsive period, the foot is converted from a "mobile adaptor" (which is what it is during the contact period) to a "rigid lever" as the mid-tarsal joint locks into a supinated position. With the foot functioning as a rigid lever (as a result of the locked mid-tarsal joint) during the time immediately preceding toe-off, the weight of the body is propelled more efficiently.

Pelvis and SIJ Motion

Next we will take a look at the pelvis and how it functions during the mid-stance phase of the walking cycle. As the right innominate bone starts to rotate anteriorly from an initial posteriorly rotated position, the tension of the right sacrotuberous ligament is reduced, and the sacrum will be forced to move (passively) into a right torsion on the right oblique axis (R-on-R) (recall, the motion of the pelvis and sacroiliac joint in Chapter 2). In other words, the sacrum rotates to the right and side bends to the left, because the left sacral base moves into an anterior nutation position (this is also known as *Type I spinal mechanics*, as the rotation and side bending are coupled to opposite sides—see Chapter 6); the motion is illustrated in Figure 4.6(a).

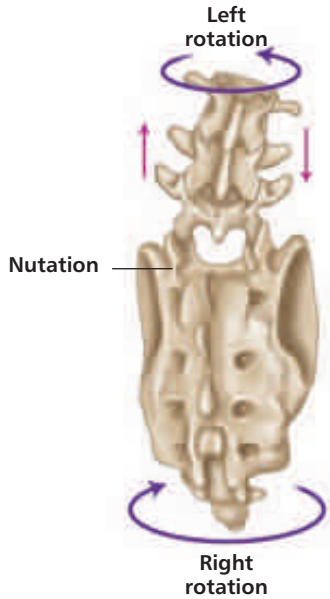


Figure 4.6. (a) Sacral rotation and lumbar counter-rotation.

We also need to mention and consider that, as the left side of the sacrum moves forward into nutation, the right side of the sacral base will move backward into counter-nutation *(R-on-R)*; this is mainly because of the slackening of the right sacrotuberous ligament and the continual anterior rotational movement of the right innominate bone during mid-stance.

Owing to the kinematics of the sacrum, the lumbar spine rotates left (opposite to the sacrum) and side bends to the right (Type I mechanics), as shown is Figure 4.6(b). The thoracic spine rotates right (same as the sacrum) and side bends to the left, and the cervical spine rotates right and side bends to the right. The cervical spine coupling is opposite to that of the other vertebrae, since its specific spinal motion is classified as *Type II spinal mechanics* (Type II means that rotation and side bending are coupled to the same side—see Chapter 6 for more details).

As the left leg moves from weight bearing to toe-off, the left innominate, the sacrum, and the lumbar and thoracic vertebrae undergo sacral torsion, rotation, and side bending in a similar manner to that described above, but with movements in the opposite directions.

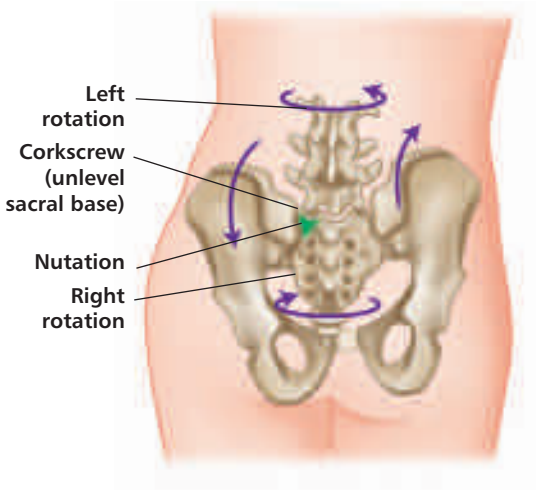


Figure 4.6. (b) Sacral rotation and lumbar counter-rotation superimposed on the pelvic girdle.

The anterior oblique also works in conjunction with the stance leg adductors, ipsilateral internal oblique, and contralateral external oblique muscles, as shown in Figure 4.7. These integrated muscle contractions help stabilize the body on top of the stance leg and assist in rotating the pelvis forward for optimum propulsion in preparation for the ensuing heel-strike.

The abdominal oblique muscles, as well as the adductor muscle group, serve to provide stability and mobility during the gait cycle.

When looking at the EMG recordings of the oblique abdominals during gait and superimposing them on the cycle of adductor activity in gait, Basmajan and De Luca (1979) found that both sets of muscles (obliques and adductors) contribute to stability at the initiation of the stance phase of the gait cycle, as well as to the rotation of the pelvis and the action of pulling the leg through during the swing phase of gait. (This was also demonstrated by Inman et al. (1981).) As the speed of walking increases to running and sprinting speeds, the activation of the anterior oblique system becomes more prominent as well as a necessity.

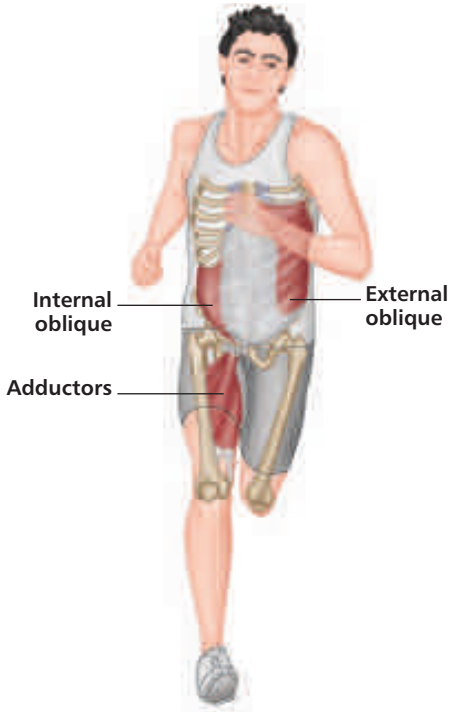


Figure 4.7. A person running, with anterior oblique sling muscles highlighted.

The swing phase of gait utilizes the lateral sling system, as we have now entered the single-leg stance position. This sling connects the Gmed and Gmin of the stance leg, and the ipsilateral (same side) adductors, with the contralateral (opposite) QL. Contraction of the left Gmed and adductors stabilizes the pelvis, and activation of the contralateral QL will assist in elevation of the pelvis; this will allow enough lift of the pelvis to permit the leg to go through the swing phase of gait. The lateral sling plays a critical role, as it assists in stabilizing the spine and hip joints in the frontal plane and is a necessary contributor to the overall stability of the pelvis and trunk.

Not only does the lateral sling system provide stability that protects the working spinal and hip joints, but it is also a necessary contributor to the overall stability of the pelvis and trunk. Should the trunk become unstable, the diminished stability will compromise one's ability to generate the forces necessary for moving the swing leg quickly, as required in many work and sports environments. Attempts to move the swing leg, or to generate force with the stance leg during gait and other functional activities, can easily disrupt the SIJs and symphysis pubis and cause kinetic dysfunction in joints throughout the entire kinetic chain (Chek 1999).

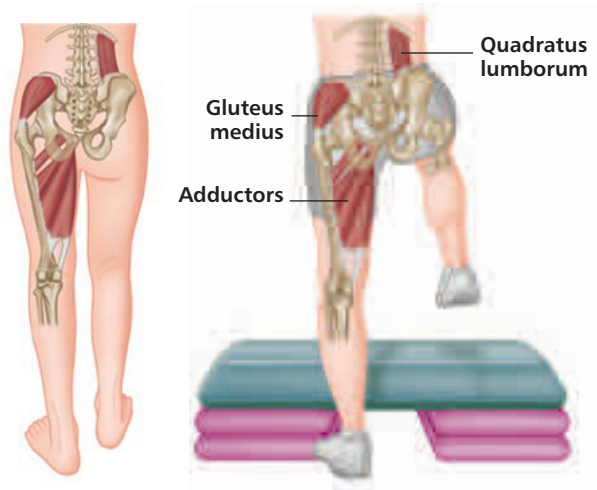


Figure 4.8. An example of the swing phase of gait, with lateral sling muscles highlighted on the single-stance leg.

Maitland (2001) mentions that proper body movement while walking is influenced by the ability of the sacrum to cope with left torsion on the left oblique axis (L-on-L) and right torsion on the right oblique axis (R-on-R). Since most walking is accomplished with the vertebral column relatively upright and vertical, for the purpose of this discussion we will assume that your spine and sacrum are in neutral while you walk.

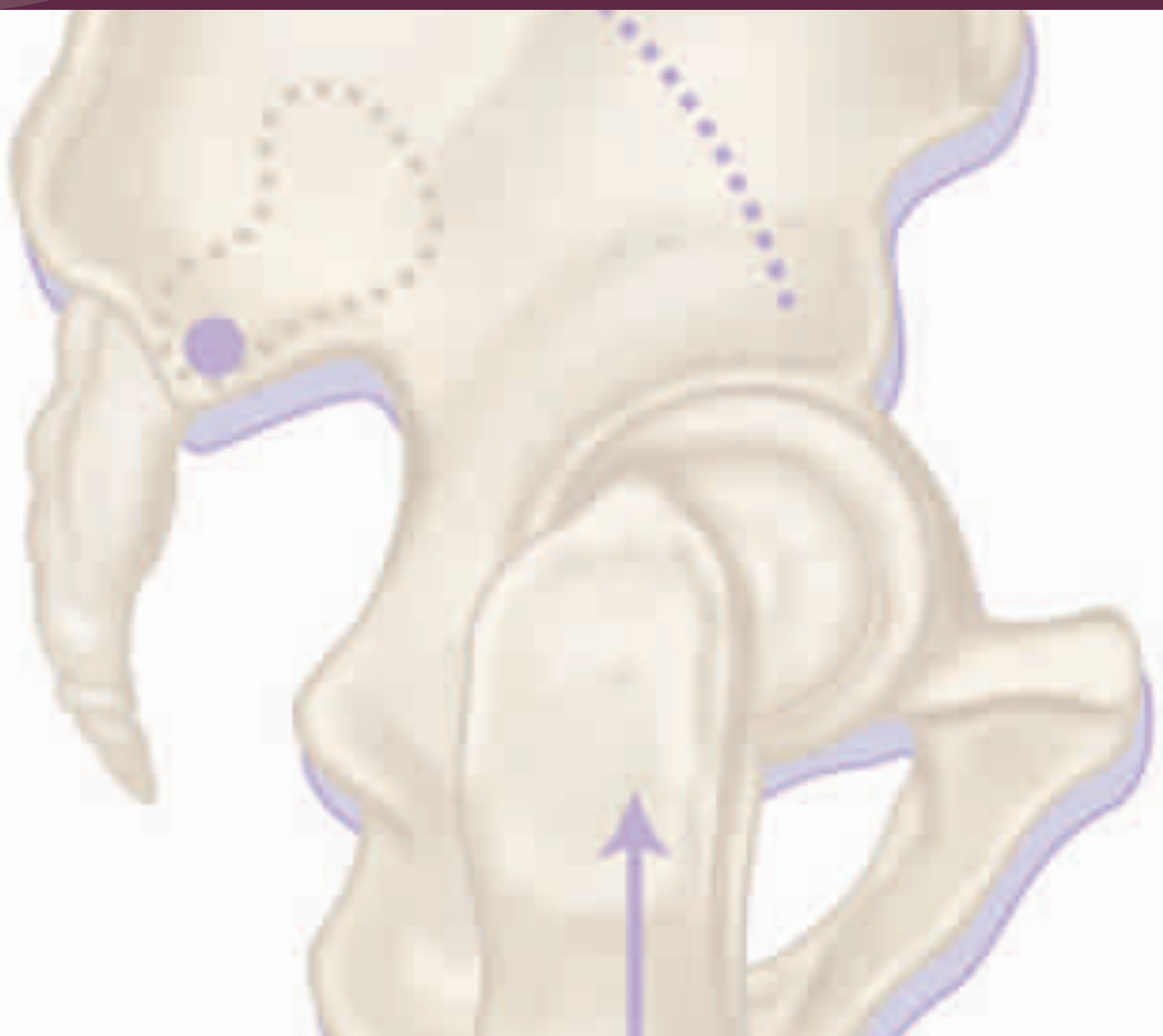
The way our axial skeletal system alternately undulates in side bending and rotation as we walk is very interesting and extremely important to our overall well-being. It is a movement that is reminiscent of the undulating action of a snake as it slithers through the grass. The big difference between a snake and a human, of course, is that our snakelike spine has ended up being given two legs on which to walk.

Summary of the Sacrum and the Gait Cycle

To summarize the gait cycle and the specific motion of the sacral spine, the sacrum is capable of left rotation on the left oblique axis (L-on-L), from which it then returns to a neutral position. From this neutral position the sacrum then rotates to the right on the right oblique axis (R-on-R) and again returns to neutral. The movement of the sacrum is *anterior* in its nature as it undergoes the earlier-described motion of nutation. The forward nutational movement during walking is anterior on one side, followed by a return to the neutral position; anterior nutation then occurs on the opposite side, before the sacrum again returns to neutral. This process is continually repeated. According to various studies, the motion of posterior nutation (counter-nutation) does not appear to extend past the neutral position during the normal walking/gait cycle.

5

Leg Length Discrepancy and its Relationship to the Kinetic Chain and the Pelvis



I would personally say that the majority of patients and athletes who visit my clinic in Oxford generally present with pain somewhere in their bodies. Part of my initial screening is to have the patient stand with their back to me; with them in this position, I place both my hands on top of their iliac crest to see if there is any pelvic obliquity; in other words, I look for a low or a high side, as shown in Figure 5.1. Very often I do find that there is a discrepancy in the level of the height of the iliac crest between the two sides, which could possibly indicate a leg length discrepancy (LLD), or, as it has been alternatively termed, a *short-leg syndrome* or a *long-leg syndrome*.

LLD is possibly the most significant postural asymmetry that presents itself to the physical therapist. The existence of a considerable difference between the two sides can be very detrimental to how we function on a day-to-day basis, not least during the walking/gait cycle: the discrepancy can significantly affect not just the pelvis and SIJ but also our overall posture.

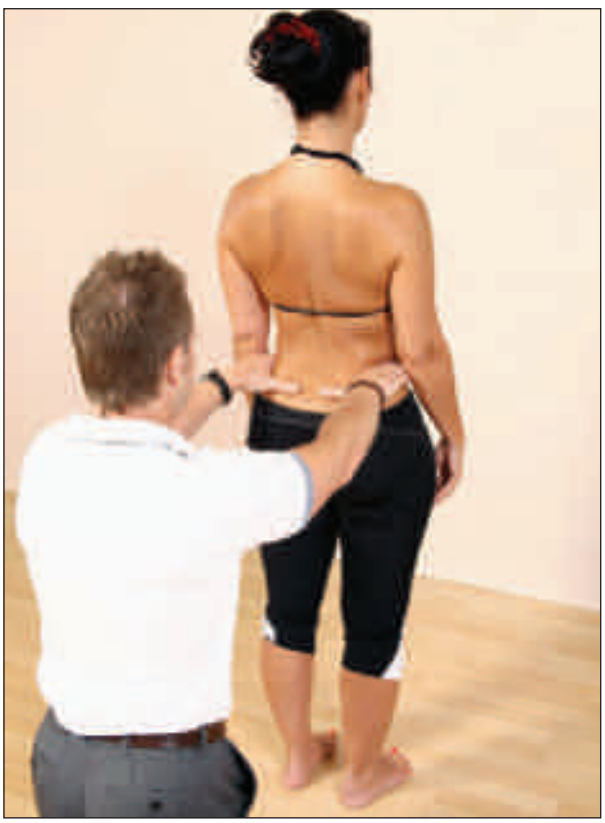


Figure 5.1. Observation of measurement of leg length by palpation of the iliac crest.

Definition: *Leg length discrepancy (LLD)* is a condition where one leg is shorter than the other.

One has to decide if there is an "actual" (or "true") anatomical LLD or an "apparent" LLD, as the condition has been implicated in all sorts of deficiencies related to our gait pattern and running mechanics. LLD has also been linked to postural dysfunctions, as well as to increased incidence of scoliosis, lower back pain, SIJ dysfunction, and osteoarthritis of the spine, hip, and knee. Even stress fractures of the hip, spine, and lower extremity have been related to changes in leg length.

Figure 5.2. True leg length measurement, taken from the ASIS to the medial malleolus.

The *actual* (true) leg length measurement is the length that is typically determined by the use of a tape measure from a point on the pelvis—the ASIS—to the medial malleolus (distal part of the tibia), as shown in Figure 5.2; the ASIS is normally used as the landmark, since it is impossible to truly palpate the femur below the iliac crest. Before taking this measurement, it is beneficial to measure the distance from the ASIS on the left and right sides to the umbilicus, as shown in Figure 5.3, to ascertain if any pelvic rotation is present. If a difference in the two measurements is found, the pelvic rotations need to be corrected before a reassessment is done (Chapter 13).

Figure 5.3. Measurement taken from the ASIS to the umbilicus.

If the measurements taken from the ASIS to the medial malleolus on both sides are the same, it can then be assumed that the lengths of the two limbs are effectively equal; on the other hand, if the measurements differ, one can assume that an actual (true) LLD is present.

The *apparent* leg length measurement is taken from the umbilicus to the medial malleolus, as shown in Figure 5.4. If the measurements taken on both sides are different, one can assume that a dysfunction exists somewhere, which will require further investigation.

Figure 5.4. Apparent leg length measurement, taken from the umbilicus to the medial malleolus.

Supine to Long Sitting

The supine to long sitting test is commonly used to ascertain the relevance of the SIJ to an apparent or true LLD. With the patient in a supine position, the therapist initially compares the relative positions of the two medial malleoli, to see if a difference exists between these two bony landmarks, as shown in Figure 5.5(a).

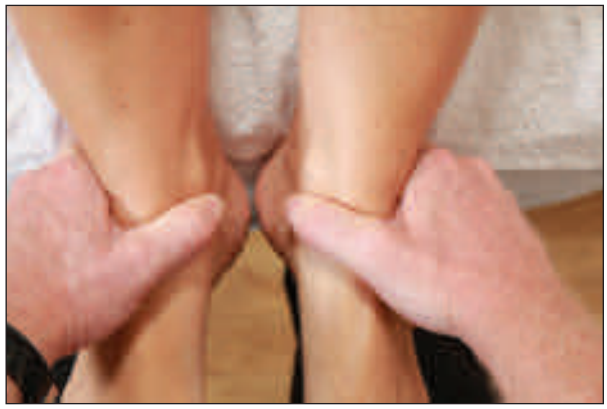


Figure 5.5. (a) Palpation for the positions of the medial malleoli (leg length) in the supine position.

Next the patient is asked to sit up, while keeping their legs extended. The positions of the two medial malleoli are compared once again, to see if there is any change, as shown in Figure 5.5(b–c).

Figure 5.5. (b) Observation of the positions of the medial malleoli (leg length) after the supine to long sitting test.

Figure 5.5. (c) Close-up view of the positions of the medial malleoli (leg length) after the supine to long sitting test. The malleoli appear to be level in this case.

If, for example, there is a *posterior* innominate present, the leg that appeared shorter in the supine position will now appear to lengthen with the sitting-up motion. If there is an *anterior* innominate present (very common on the right side), the leg that appeared longer in the supine position will now appear to shorten with the sitting-up motion, as shown in Figure 5.5(d).

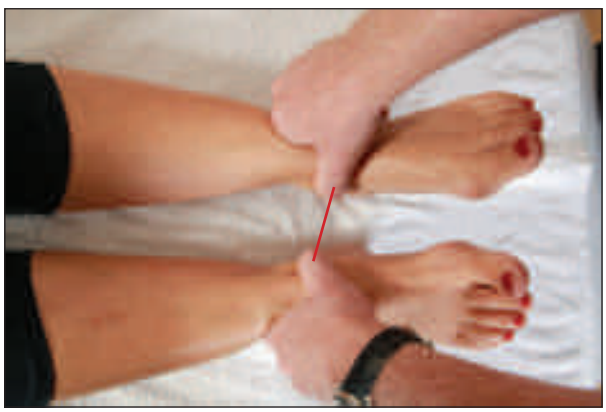


Figure 5.5 (d). Close-up view of the positions of the medial malleoli (leg length) after the supine to long sitting test. The right leg appears shorter, possibly indicating a right anterior innominate rotation.

If an actual (true) LLD is present, the true long leg will appear to be longer in the supine position as well as in the sitting position, so no obvious change will be observed (this, along with further discussion on leg length changes, will be presented in more detail in Chapter 12). For now, it is sufficient to simply note down if there are any changes in the position of the medial malleoli in the supine and long sitting positions.

Note: This test can help in differentiating between a true LLD and a sacroiliac dysfunction. Please be careful when asking your patients to perform this test: the motion of the test (sitting up) can easily exacerbate a patient's symptoms because of the forces needed to perform the action. Assistance by the therapist might sometimes be necessary.

Types of LLD

LLD can be divided into three main groups:

1. **Structural:** This is an actual (or true) shortening of the skeletal system, typically caused by one of four things:

    - Congenital defect, e.g. congenital dysplasia of the hip joint
    - Surgery, e.g. total hip replacement (THR)
    - Trauma, e.g. fractured femur or tibia
    - Disease process, e.g. tumor, osteoarthritis, Osgood–Schlatter disease, or Legg–Calvé–Perthes disease

    Fractured bones in children have been known to grow faster for many years after the healing process: this can naturally result in the limb becoming anatomically longer.

2. **Functional:** This can be a development from altered biomechanics of the lower body, such as ankle and foot over-pronation or supination, pelvic obliquity, muscle imbalances (as a result of, for instance, a weak Gmed and abdominals or tight adductors and hip flexors), hip or knee joint dysfunction, and even poor inner core stabilization, to name just a few.

3. **Idiopathic**: If there are obvious findings during the history taking and assessment process, the physical therapist may have an idea as to the cause of the patient's LLD. However, if the therapist cannot ascertain a reason for the change in the presenting leg length, the condition would be classified as *idiopathic*, which means that it arises independently, not as a result of some other condition.

Kiapour et al. (2012) estimate that a LLD of as little as 0.4" (1cm) increases the load across the SIJ fivefold.

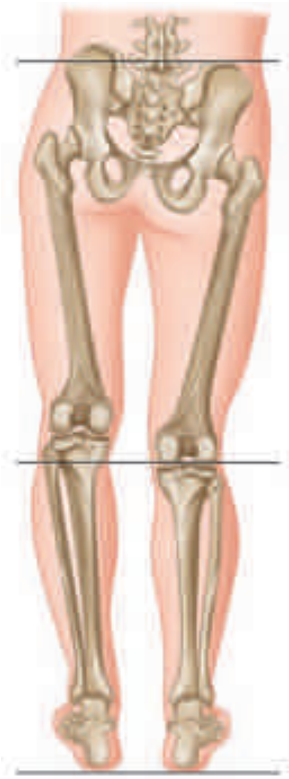


Figure 5.6. Left long-leg syndrome versus right short-leg syndrome.

Assessment

The therapist has to be very intuitive during the initial assessment. When placing their hands on top of the iliac crest of a patient in a standing position in order to ascertain pelvic obliquity, the therapist needs to be aware of a "pelvic shift" as the patient stands. Let me give you an example: if the patient has a weak Gmed muscle on the left side, the pelvis might appear to drop to the right side and deviate or laterally shift to the left; this will have the effect of the left iliac crest appearing elevated on that side (left), thus giving the natural appearance of a longer left leg.

When the patient presents to the clinic, one can assume that the pain has been present for a while; since the pain has persisted for an extended period of time we can safely say that the presenting condition is now in the chronic stage. Because of the natural overcompensatory mechanisms that occur through soft tissue chronicity, the postural muscles are probably held in a shortened and subsequently tight position; one particular lumbar spine muscle that has a natural tendency to shorten as a result of LLD is the QL. A perceived problem can arise when the patient lies on their back (supine position), so that you can observe the positions of the left and right medial malleoli when looking for any LLD. You may notice that the left medial malleolus appears nearer to the patient's head (cephalic) than the one on the right, giving the appearance of a short left leg. This apparent shortness of the leg is possibly a result of a tight left QL muscle. When the patient was standing, however, you may have convinced yourself that the patient's left leg actually looked longer!

This might seem initially confusing, but just think about it for a moment. Could it not simply mean (this is only one example because there are many potential causes of a higher iliac crest) that when the patient adopts a standing position the Gmed on the left side is weak, causing the pelvis to shift to the side of weakness, now giving the appearance of a longer left leg? Conversely, is it not possible that when the patient is in a supine position, the left QL is held in a shortened position, which is responsible for hitching up the pelvis, having the effect of pulling the leg closer to the head and thus making the left leg appear shorter?

The following phrase might help you remember this process:

"When you are standing, the weak muscles show themselves; when you are lying, the short muscles show themselves."

Foot and Ankle Position

One of the most neglected aspects of the body, especially when patients present to the clinic, is the position of the lower limb. Every single day osteopaths, chiropractors, and physical therapists see lots of patients who initially present with lower back, pelvis, and sacroiliac pain. These specialist therapists naturally spend a lot of time observing and assessing the pelvis and lumbar spine to ascertain which tissue they personally consider to be responsible for giving the person the pain. This presenting pain may, however, just be a symptom, and the cause of the pain could be somewhere else, away from the actual site of the pain.

Dr. Ida Rolf, who invented the Rolfing soft tissue technique, states: "Where you think the pain is, the problem is not." One of my popular sayings that I impress on my students (which I consider to relate to what Dr. Rolf states) is the following:

"The only person interested in the pain is your patient; you the therapist should try to find the actual cause of the pain and not simply treat where it hurts."

It is very important that when assessing your patients, you should observe the position of the lower limb and in particular the foot and ankle complex, as a faulty foot and ankle structure can profoundly affect leg length and the natural position of the pelvis. The most common asymmetrical foot position that patients present with has to be what is commonly called an *over-pronated foot* (or pes planus), as shown in Figure 5.7.

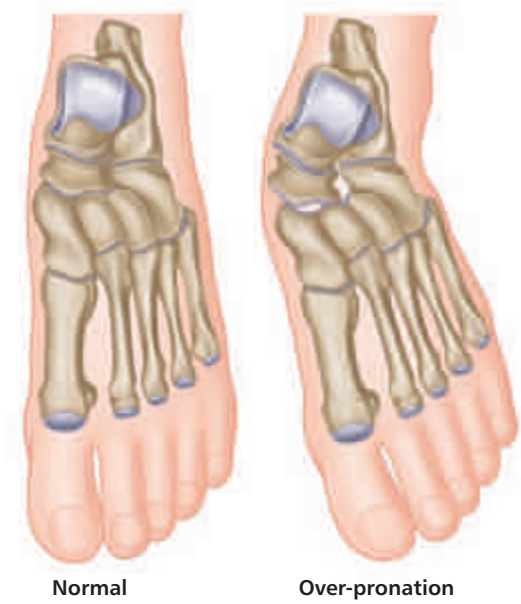


Figure 5.7. Over-pronation syndrome.

It has been widely thought that when we actually present with a true LLD, the body will try to compensate for the longer leg through lowering the medial arch of the foot by pronating at the STJ. The action of pronation is called *tri-planar motion* and consists of three movements: dorsiflexion of the ankle, eversion, and abduction of the foot complex. The position of this increased pronation is basically the body's natural compensatory mechanism to try to "shorten" the leg because it is anatomically longer.

The plantar surface of our feet has thousands of sensory receptors that are responsible for the position of the foot; the smallest shift in weight will be enough to signal the brain to induce a compensatory reaction. On the contralateral side (shorter leg), the compensatory mechanism will cause the medial arch to adopt a supinated position (tri-planar motion of plantar flexion, inversion, and adduction). The compensatory mechanism changes the position of the arch in an attempt to lengthen the apparent shorter leg. When physical therapists assess their patients they will need to check for this compensatory pattern, because, if left unchecked, excessive foot pronation caused by an anatomically longer leg (subsequent supination of the contralateral foot as compensation) will in turn cause an internal rotation of the lower extremity and an external rotation of the contralateral lower limb. This compensatory mechanism will then have the effect of altering the whole kinetic chain from the foot all the way up to the occiput.

True LLD and the Relationship to the Pelvis

Let's continue with a "thought process" just for a moment. Now consider that your patient has an actual longer leg on the left side, which you have ascertained because of the higher position of the iliac crest and the possible compensatory pronation of the STJ on the same side, as well as the leg length appearing longer

(through observation of the medial malleoli) in the supine and long sitting position. Before I continue with the discussion, just have a think to yourself about what position the innominate bone might be in if the left leg, say, is *anatomically* longer.

The innominate rotation will naturally be coupled with a LLD as a result of the compensatory mechanisms: if you look at Figure 5.8(a), you will see that the femoral head on the long-leg side forces the innominate into a superior and posteriorly rotated position. Conversely, the innominate on the low femoral head side drops down and anteriorly rotates, as shown in Figure 5.8(b). What we have now, therefore, is the left innominate being forced into a *posterior* rotation and the right innominate into an *anterior* rotation.

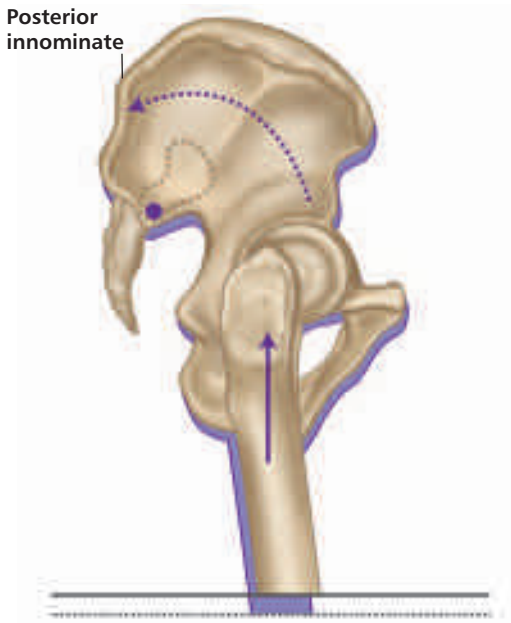


Figure 5.8. (a) Long-leg innominate compensation.

Think about what lies between the two innominate bones—yes, the sacrum. As a result of the compensatory rotation of both of the innominate bones (because of the anatomical LLD) in opposite directions to each other, a motion of the sacrum occurs—left-on-left (L-on-L) sacral torsion (Figure 5.9), which was covered in Chapter 2. Recall, a L-on-L

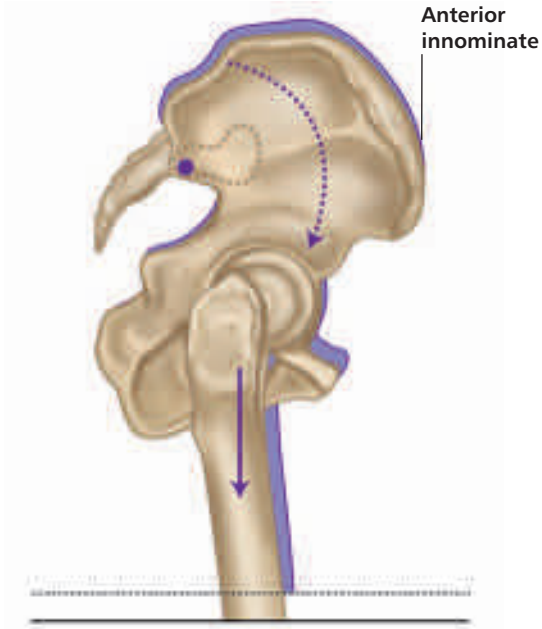


Figure 5.8. (b) Short-leg innominate compensation.

sacral torsion means that the sacrum has rotated to the left on the left oblique axis and has side bent to the right, as it is ruled by Type I spinal mechanics (rotation and side bending are coupled to opposite sides, as established by Fryette (1918) and the law of spinal mechanics, which will be discussed in Chapter 6). This complexity of the innominate rotations that are coupled with a sacral torsion is usually depicted as a *pelvic torsion*, or even a *pelvic obliquity*, and will require a good understanding before a treatment plan is introduced.

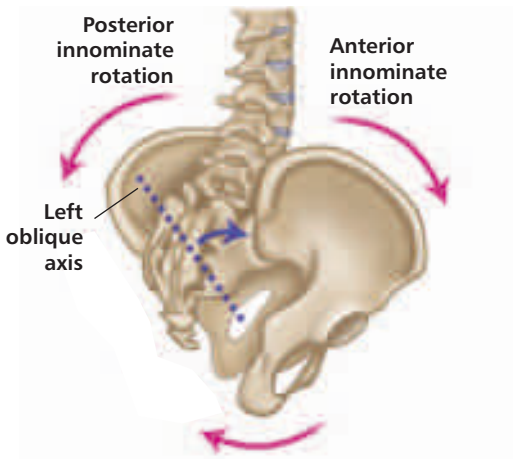


Figure 5.9. L-on-L sacral torsion.

True LLD and the Relationship Between the Trunk and the Head

You will notice in Figure 5.10(a) & (b) that on the left side there is a lower shoulder position on the high innominate side: this is a common finding in the case of a compensatory functional scoliosis. Some authors, however, have considered this to occur as a result of a "handedness pattern": for example, if you are left handed, the left shoulder might appear to be lower, and if you are right handed, the right shoulder might appear to be lower. I agree that the pattern of handedness is probably true, but only if the iliac crests are level; otherwise some form of scoliosis has to exist, especially if the iliac crest and shoulder positions are asymmetric.

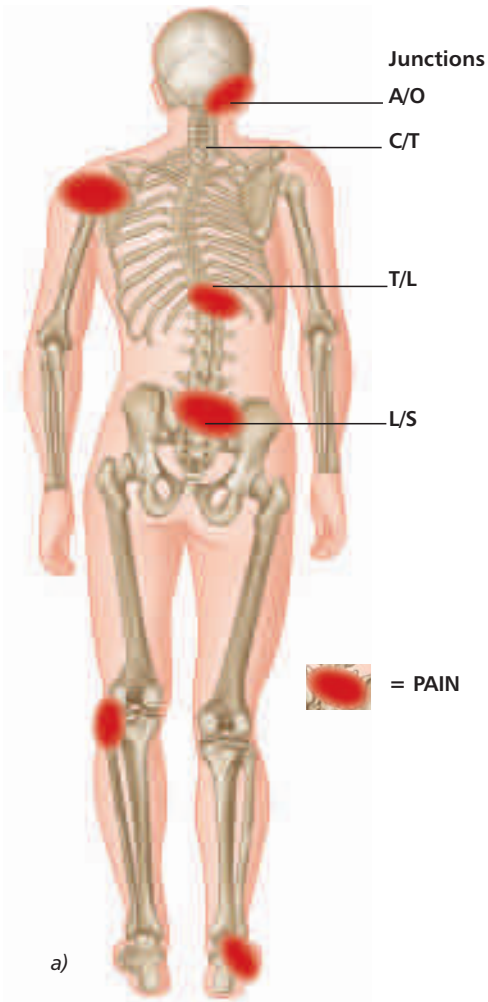


Figure 5.10. (a) Functional scoliosis compensation.

What else do you observe in Figure 5.10(b)? If you look at what is happening to the left QL, you might assume that this muscle is being held in a shortened position because of the higher left innominate. This assumption is correct, as you can also see that the lumbar spine is side bending toward the longer left leg (concavity) and rotating toward the shorter right leg (convexity).

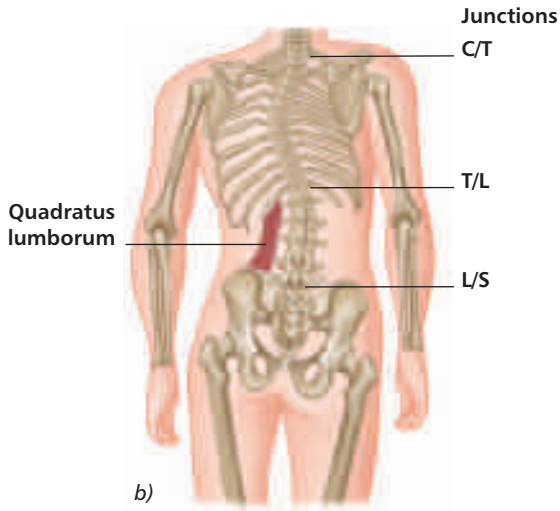


Figure 5.10. (b) Functional scoliosis compensation. Quadratus lumborum on left is short/tight.

As a result of the ascending functional scoliosis, the right shoulder is higher. You might also notice a short "C" curve in the cervical spine; this will probably cause the scalenes, sternocleidomastoid (SCM), upper trapezius, and possibly the levator scapulae muscles on the right side to adopt a shortened and subsequently tight position. This typical adaptation of muscular imbalance will help maintain an erect head position with the eyes level. The body will always want to be level no matter what and will do almost anything to accomplish this, through naturally adapting the position of the occipitoatlantal joint (OAJ), and in so doing can suffer enduring pain to maintain equilibrium. Common painful conditions that patients might present with are headaches, active trigger points, tinnitus, temporomandibular joint (TMJ) dysfunction, and even eye and facial pain.

LLD and the Gait Cycle

As we walk, if our gait cycle pattern has been altered because of an actual or an apparent LLD, the shorter leg will feel like it is stepping down, and the long leg will compensate by a sort of "vaulting" motion. It is almost like stepping into a small pothole with every step you take; imagine repeating this at least ten to fifteen thousand times a day—it will easily cause potential pain patterns of dysfunction! Common compensations are sometimes seen when patients are asked to walk: on the short-leg side, the patient might have a tendency to walk on their toes, and on the long-leg side, the patient may have a tendency to flex their knee, but this will depend on the discrepancy.

For the body to be an effective locomotor during the gait cycle, a well-aligned and symmetrical body is essential. When the positions of the innominate bones of the pelvis are altered by actual or apparent leg length discrepancies, it is easy to see how patients can present with pain, not only at the SIJ and lumbar spine but also everywhere else in their body that is going through a compensation pattern.

I mentioned earlier that there could be a weakness of the Gmed on one side of the body; this weakness could lead to a potential compensatory pattern for subsequent shortness issues with the tensor fasciae latae (TFL) and ITB on the other side. If there is weakness of the Gmed, the patient can have either a *Trendelenburg* pattern of gait or a *compensatory Trendelenburg* pattern of gait (see "Standing Balance Test," later in this chapter). Whichever way you look at this, the patient is going to have an antalgic type of gait, which simply means that they will walk with some form of limp; this compensation can only cause one thing over time and that will simply be pain.

Summary of LLD Compensations to the Sacrum

- The sacrum typically *rotates* toward the *long* leg and *side bends* toward the *short* leg.
- A posterior sacrum (counter-nutation) has been found to be associated with same-side piriformis spasm.
- An anterior sacrum (nutation) has been found to be associated with same-side Gmed spasm.

Summary of LLD Compensations to the Lumbar Spine

- The lumbar spine typically *rotates* toward the side of the low sacral base/ *short* leg and *side bends* toward the *long* leg.
- Facet pain due to compression is common on the concavity side (side-bending side) of the lumbar spine.
- The iliolumbar ligament can cause pain as it is stretched on the convex side of the lumbar spine and has been argued to refer pain to the groin, testicle, and medial thigh on the same side as the stretched ligament.

Summary of LLD Compensations to the Iliopsoas Muscle

- The iliopsoas muscle is generally considered to be tight on the side of the short leg. Note that a left iliopsoas muscle spasm can cause a pelvic side shift to the right, mimicking a shorter left leg.
- The iliopsoas muscle (iliacus in particular) can cause the innominate bone to compensate by rotating anteriorly on the side of the short leg, functionally causing a lengthening of that leg, while the innominate bone on the long side may do the opposite (posterior innominate rotation).

- Unilateral spasm (contraction) of the iliopsoas can create a lumbar concavity on the same side (because of side bending), with rotation to the opposite side, and potentially sets up a positive side shift on the side opposite to the spasm. It is important that the physical therapist always assesses for and treats iliopsoas spasm in the case of a suspected short leg or a positive pelvic lateral side shift.

- Iliopsoas spasm pain is generally worse on standing from a seated position, and less pain is perceived as the iliopsoas stretches out.

In summary, when looking at the level of the iliac crest, one has to determine if there is a LLD. If there is, one then has to ascertain if the dysfunction is a true discrepancy or a functional discrepancy, as the compensation pattern can change depending on the diagnosis. For example, if you find a true anatomical LLD, the innominate bone on the longer leg will normally try to compensate by rotating posteriorly, as seen in Figure 5.7 and explained earlier. Moreover, the femur and lower limb on the anatomically longer leg will follow the compensatory model by rotating medially, as seen in Figure 5.5, and the foot will try to pronate at the STJ, since the longer leg will attempt to *shorten* itself. At the same time, the actual shorter leg will compensate by supinating at the ankle mortise; this in effect can cause the tibia and femur to rotate externally and the innominate bone to rotate anteriorly as the leg tries to make itself appear *longer*.

Over-Pronation Syndrome

Let's look at another compensation model for a patient who appears to have a LLD. In this case it is a *functional* LLD, and the apparent shorter leg of the patient exhibits an over-pronation of the STJ, rather than the true longer leg compensating by pronating to shorten itself. As a result, the body will try to compensate by causing an internal rotation of the tibia and femur, as shown in Figure 5.11(a); this will have the effect of the innominate bone rotating anteriorly (posteriorly in a true LLD, as explained earlier), which in turn can cause an increased lumbar lordosis with subsequent lower back pain.

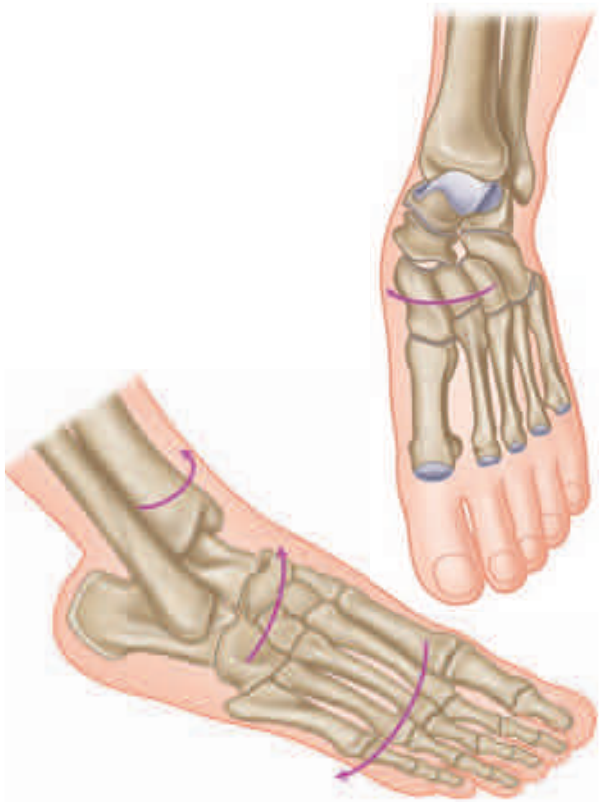


Figure 5.11. (a) A foot in over-pronation with internal tibial rotation.

According to podiatrists, over-pronation syndrome is a very common pattern found in the majority of people to some extent or another. It is best identified with the patient standing barefoot. The big clue is that one arch is lower or flatter than the other: the lower arch side is over-pronating. Sometimes, both sides may be over-pronating, but one side is generally over-pronating more than the other,

or one side might be normal and the other side lower.

It is easy to confirm the condition by simply placing one finger under the patient's arch, noting how much of your finger goes underneath, and comparing the result with the other side. Are they the same or different? If one side is noticeably lower than the other, you have found a patient with an over-pronation syndrome. Another test to confirm over-pronation is to observe the Achilles tendon from the posterior aspect: you will typically notice a bowing on the side with the lower arch.

It should be noted that over-pronation syndrome could originate not only from the foot and ankle but also from the innominate bone of that side. When the foot and ankle complex over-pronates, the innominate bone will normally rotate anteriorly. However, if we look at it the other way around, an anterior rotation of the innominate (common on the right side) can force the medial arch of the foot into an over-pronated position. This becomes a chicken and the egg situation, but that is irrelevant, as the only consideration is whatever presents itself now. In my experience, you might need to correct the innominate rotation and the over-pronation to help reduce the patient's presenting symptoms.

If you feel the pronation is attributed to the most common presentation of a right anterior innominate rotation with a compensatory left posterior innominate rotation, then you might also notice the right foot appears to be in a position of *relative* external rotation or abduction, even though the tibia is still in a position of internal rotation because of the pronation of the STJ. This is possibly caused by the anticlockwise (left) rotation of the pelvis (compared with the left foot, which appears to be in supination and relative internal rotation or adduction). As mentioned earlier, even though the foot appears to be externally rotated, the tibia is still in an overall position of internal rotation, as shown in Figure 5.11(b).

On the side that appears to be pronated with a relative external rotation or abduction of the foot (most common on the right side; the tibia is still maintained in an internally rotated position), the following musculoskeletal presentations are common:

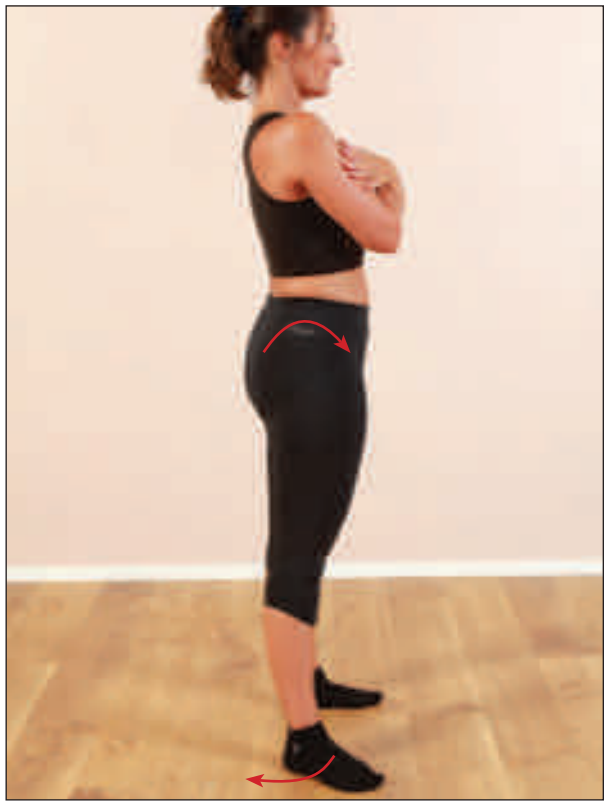


Figure 5.11. (b) External rotation/abduction of the foot and ankle with STJ pronation, commonly found with a right anterior innominate rotation.

- Medial collateral ligament and medial plica
- Groin and/or medial thigh pain
- Medial tibial stress syndrome (shin splints)
- Medial ankle ligaments sprain
- Posterior tarsal tunnel syndrome/ posterior tibial nerve
- Compression of the sural nerve at the lateral ankle
- Increased Q angle (valgus) and stress to the lateral compartment of the knee
- Lateral tracking of the patella
- Plantar fasciitis
- Sesamoiditis
- Achilles tendinopathy

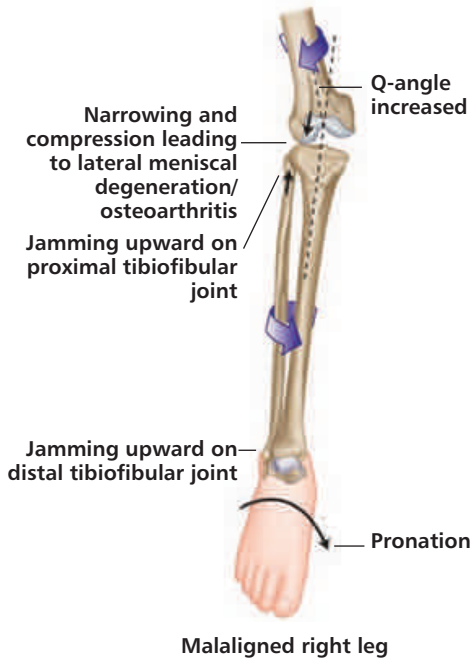


Figure 5.11. (c) Internal tibial rotation and ankle/foot with STJ pronation, commonly found with a right anterior innominate rotation.

On the side that appears to be supinated with a relative internal rotation or adduction of the foot as compared to the other side (common on the left side; the tibia is still maintained in an externally rotated position), the following musculoskeletal presentations are common:

- Strain to the hip abductors
- Greater trochanteric bursitis
- Lateral shin pain
- Lateral ankle ligaments sprain
- Nerve traction injury to the lateral femoral cutaneous and superficial peroneal nerves
- Increased pressure to the medial knee compartment due to varus position and decreased Q angle
- Traction of the lateral collateral ligament
- Tibial and metatarsal stress fractures
- Plantar fasciitis
- Morton's neuroma
- Achilles tendinopathy

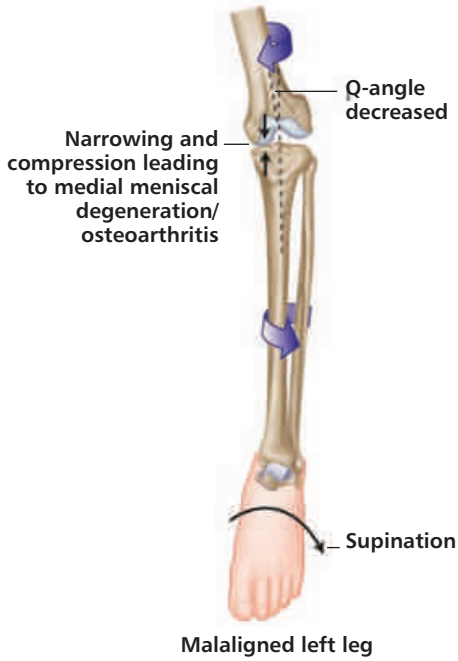


Figure 5.11. (d) External tibial rotation and ankle/foot with STJ supination, commonly found with a posterior innominate rotation.

You can see already that both compensatory mechanisms I have mentioned throughout this chapter have a pronation issue at the STJ; however, the *true* leg length compensation of the longer leg forced the innominate bone to rotate *posteriorly*, whereas the *functional* LLD of the over-pronation syndrome caused the innominate bone to rotate *anteriorly*.

You can probably gather from all the information above that there can be a lot going on at the same time throughout the kinetic chain; everything that has been mentioned can affect the *length* of the leg. It is correct to assume that this area of discussion is somewhat complex, and it might be difficult to know where to start in the assessment or even in the treatment program.

In all that has been discussed above, there lies a potential solution to the jigsaw puzzle of patients' symptoms and dysfunctions. There exists what I call a *key* to unlocking the problem; the difficulty in physical therapy, however, is finding where to start and "insert the key" (excuse the expression). I can guarantee that,

over the years, inexperienced therapists will have time and time again inserted the key into the wrong place, i.e. where the patient feels the pain and not where the problem lies. Recall the wise words of Dr. Ida Rolf!

During the practical components of my lectures to physical therapists attending my courses, I sometimes hear myself saying the following:

"Treat any dysfunctions that you find at the time during the treatment session, and the body will hopefully guide you onto the correct pathway."

After that statement I normally say this:

"If, however, after three or even five physical therapy sessions, the patient's symptoms are not reducing, then you the therapist will need to alter your thought process and reassess and potentially treat other areas of the patient's body that you initially felt might not be related to the cause of their presenting symptoms."

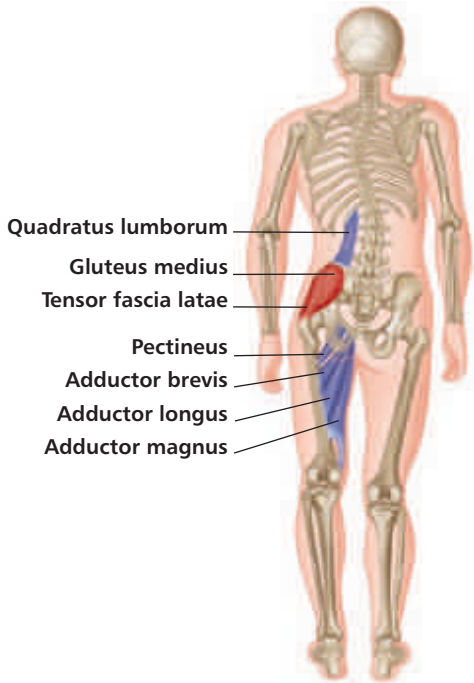


Figure 5.12. Left leg compensations—adductors and QL short and tight, with Gmed and TFL long and weak.

LLD and the Relationship to the Gluteal Muscles

So how does all this affect the glutes? When you have a compensation pattern, the femur not only rotates to compensate in the transverse plane (as explained earlier), but also experiences a compensatory mechanism of adduction and abduction in the frontal plane. Thus, in simple terms, if you have a lower limb that is held in an adducted position, then the abductor muscle group can be forced into a lengthened and subsequently weakened position, while the adductors will be held in a shortened and subsequently tight position. For a leg that is held in abduction, the situation is reversed.

If you look back at Figure 5.6 you will see that the left leg appears longer because of the higher left iliac crest, the innominate has rotated posteriorly, the femur has internally rotated, and the foot has pronated. In this compensation the left leg will be in a position of adduction (Figure 5.12), and consequently the right leg will be held in a position of abduction (Figure 5.13). This will have an effect on the musculature of the associated areas: some of these muscles will be held in a shortened position and some in a lengthened position.

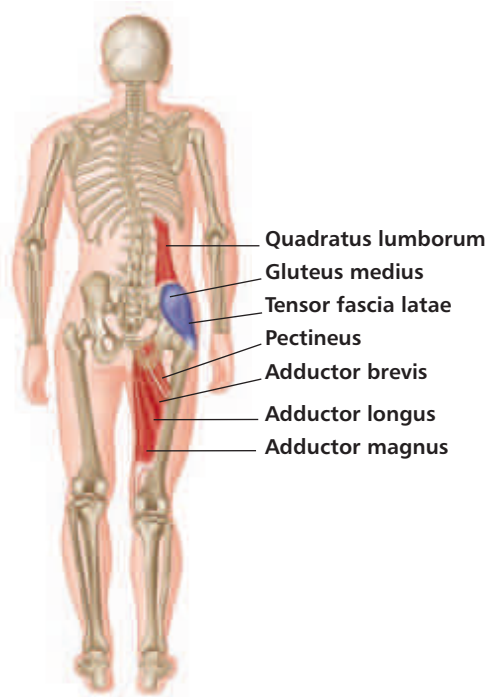


Figure 5.13. Right leg compensations—adductors and QL long and weak, with Gmed and TFL short and tight.

Standing Balance Test

When a patient is asked to stand on one leg and lift the opposite knee toward their waist, the physical therapist needs to observe the level of the PSIS as the patient transfers their weight to one leg. The patient should be able to shift their weight onto the stance leg (the right leg in Figure 5.14) with good muscular control of the Gmed of that leg. If the PSIS dips down on the leg that is being lifted (the left leg in Figure 5.15) rather than remaining level (Figure 5.14), it might be assumed that the Gmed on the opposite side (right side) is unable to control the movement; the patient might then have an altered pattern of gait when they go through the gait cycle, as shown in Figure 5.16. This altered gait pattern is called a *Trendelenburg gait* and is illustrated for a weak *left* Gmed in Figure 5.16. If this dysfunctional gait is present over a prolonged period, a compensatory Trendelenburg might develop, as shown in Figure 5.17. The reasons for this altered gait can be numerous to say the least, but one cause could be that one of the legs is held in an adducted position because of the shortening of the adductors (as mentioned above). This altered pattern will in turn result in a reciprocal inhibition (RI) to the antagonistic muscles: the abductors—in particular the Gmed—will now be held in a lengthened position that can then predispose the Gmed to becoming weak.

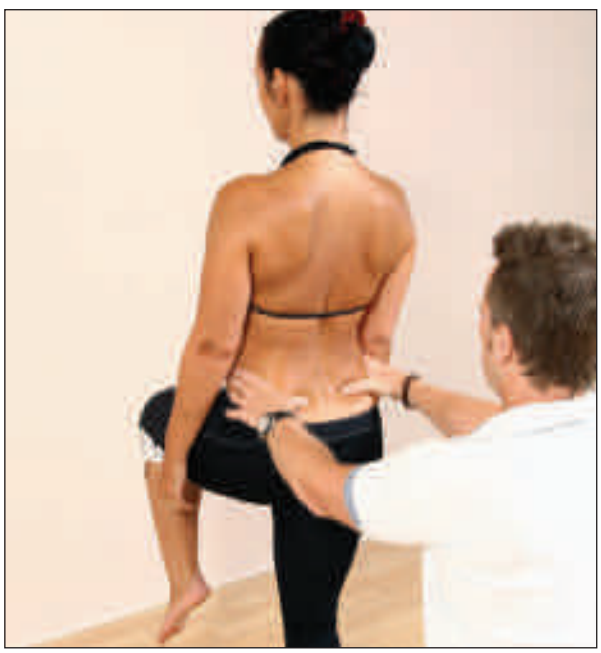


Figure 5.14. Standing balance test—normal.

Figure 5.15. Positive test for weakness of the right Gmed—the left PSIS dips down on the left.

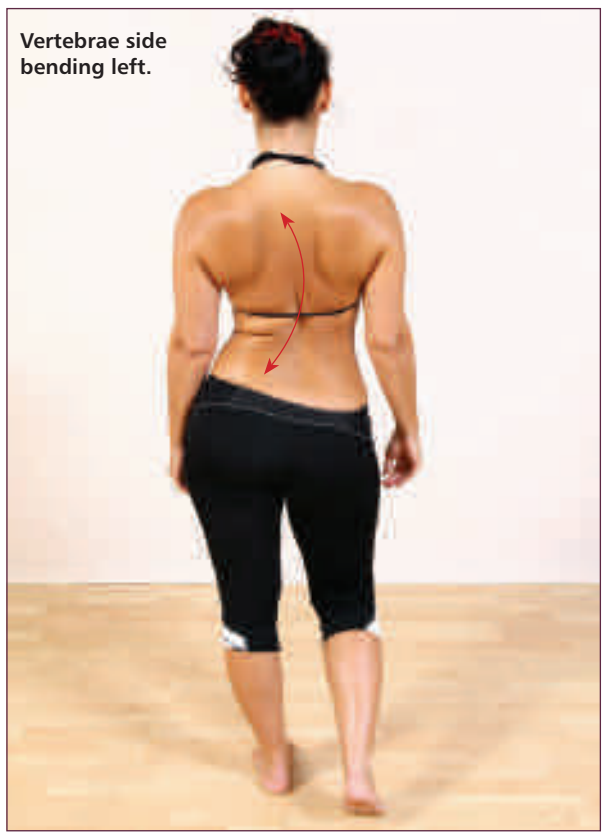


Figure 5.16. Trendelenburg gait—weak left Gmed.

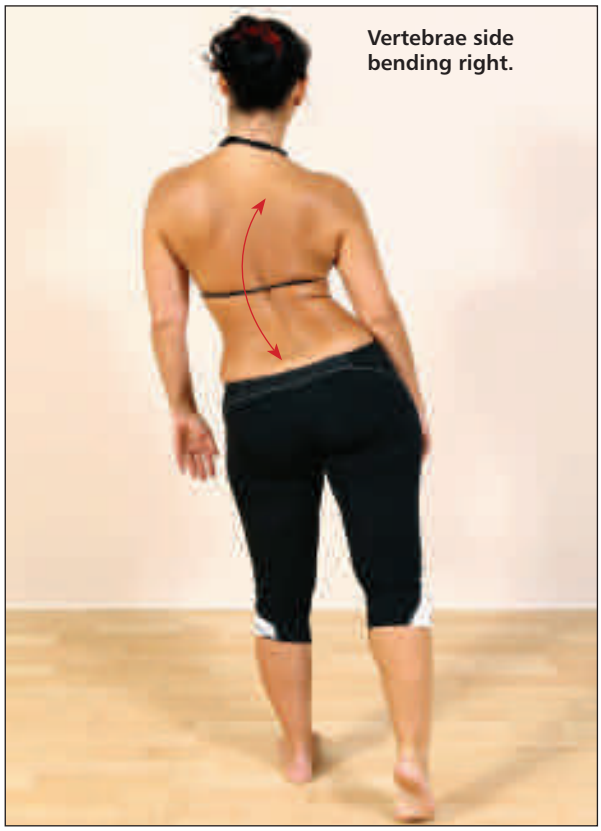


Figure 5.17. Compensatory Trendelenburg gait— weak left Gmed.

When I teach the standing balance test (Figure 5.14), I say to my students that you need to look out for three things:

1. **Position of the PSIS**

   The first, as I mentioned above, is the position of the PSIS as the patient transfers their weight from one leg to the other.

2. **Shift of the pelvis**

   The second is how much movement occurs as the patient shifts onto the weight-bearing leg; you may notice one side shifting more than the other, indicating a possible Gmed weakness.

3. **Stability on one leg**

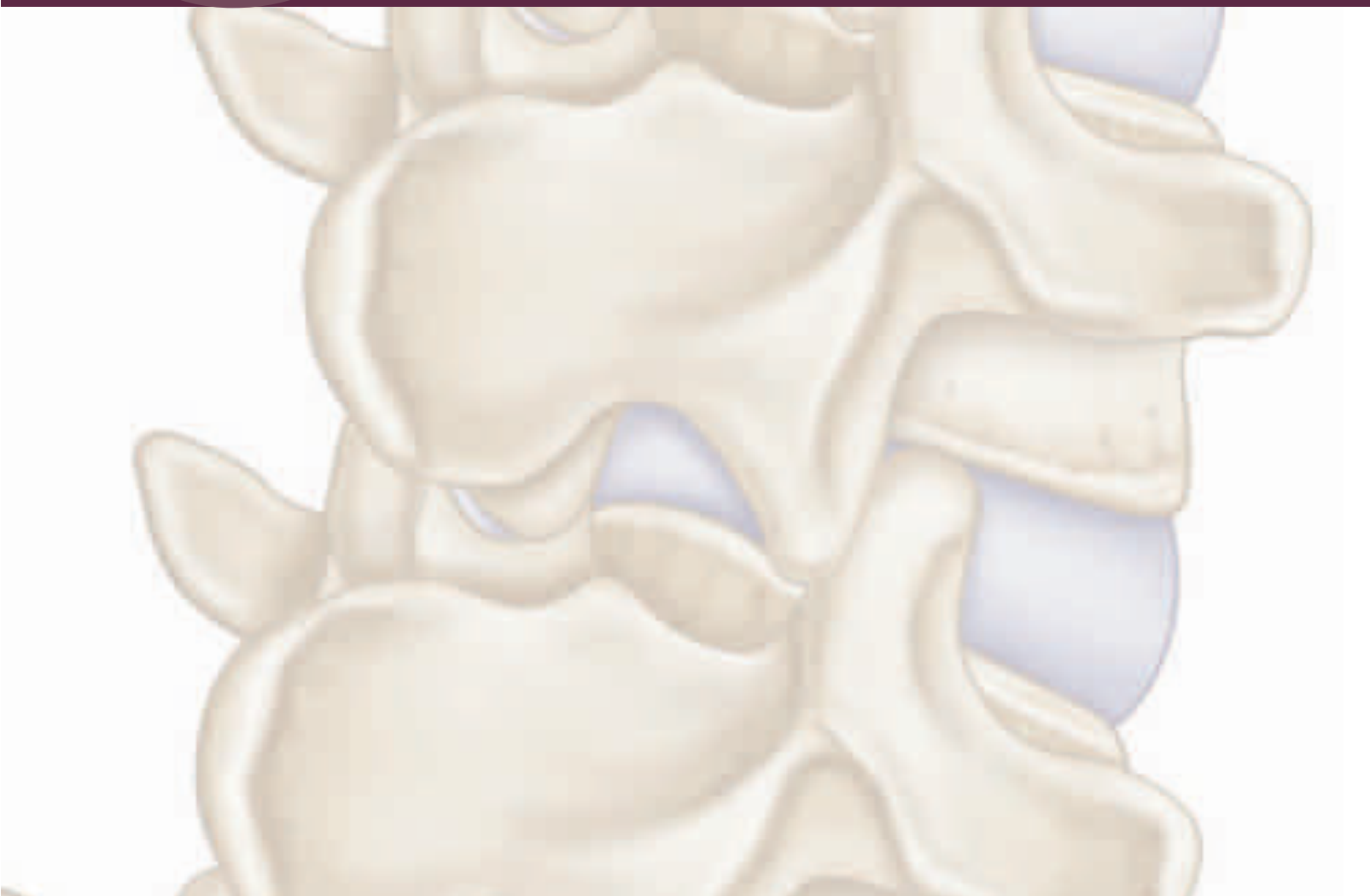
   Finally, the third is how stable the patient is when they stand on one leg compared with the other. You will be amazed how many very fit athletes struggle to stand on one leg unaided and maintain good control. This instability could be a result of a weak Gmed, but remember also that a change in a patient's ability to stabilize might be due to a previous injury/trauma to the ankle complex, thus affecting the neurological proprioceptors in controlling the specific position.

Friel et al. (2006) conducted a study about ipsilateral hip abductor weakness after an inversion ankle sprain; their results showed that hip abduction and plantar flexion were significantly weaker on the involved side. They concluded that unilateral ankle sprains led to weaker hip abduction (Gmed), and suggested exercises to strengthen the hip abductors when developing rehabilitation protocols for ankle inversion sprains.

Schmitz et al. (2002) demonstrated through an EMG study that there was an increase in Gmed activity during sudden ankle inversion motion in healthy subjects as well as in those with functionally unstable ankles.

Hopefully, after reading this chapter, you will have some understanding of what is happening when a patient presents with musculoskeletal dysfunctions, such as LLD, over-pronation syndrome, and muscle imbalances. In the next chapter I will continue with the journey on this theme by looking at the specific movement mechanics of the spine.

6 The Laws of Spinal Mechanics



Spinal Mechanics: Fact or Fiction?

A medical doctor by the name of Robert Lovett was the first person (I believe) to confirm coupled spinal motions, other than the standard primary motion of flexion and extension (Lovett 1903). He believed that side bending and rotation of the spine are parts of one compound movement and cannot be dissociated: he found that a flexible rod bent in one plane could not bend in another plane without twisting. In all his experiments Lovett proved that if the spine was in a position of lordosis, it rotated in the *opposite* direction to that of the side bending motion; moreover, if the spine was in a position of kyphosis, it rotated in the *same* direction as that of the side bending motion.

He considered that only three spinal movements were possible within the vertebral column: (1) flexion; (2) extension; and (3) side bending with rotation. He also concluded that side bending, or lateral flexion, must accompany spinal rotation (i.e. the motions are coupled).

Lovett proposed that coupled motion occurs in a second plane of motion within a joint system, and is part and parcel of the primary motion. Two or more motions are considered "coupled" when it is not possible to produce one motion without inducing the second one; spinal coupling occurs because of the morphological shape of the facet joint surfaces and the connecting ligaments and spinal curvatures.

In the early 1900s the osteopath Harrison M. Fryette contributed some pioneering work on the mechanics of spinal motion. He spent many years of his life researching this topic and eventually presented a paper in 1918 on the principles of spinal motion, which was sent to the American Osteopathic Association (Fryette 1918).

Initially, Fryette's paper was poorly received and he did not gain acceptance. Many years passed before he eventually received formal recognition for his ideas. In the late 1940s Fryette was invited to the UK by Edward Hall to do a presentation on his work. Fryette spoke about what he called "The Total Osteopathic Lesion," and he had such a profound effect on Hall that his biomechanical view of spinal motion was relabeled to "Fryette's Laws of Spinal Mechanics," which up until then had been referred to only as *principles*. (Hall wrote the first article on that subject in the 1956 yearbook of The Osteopathic Institute of Applied Technique.)

A quote from Fryette:

"No intelligent scientific spinal technic can be developed that is not based on an accurate understanding of the physiological movements of the spine."

Regarding the motion of the spine, Stoddard (1962) states the following in his manual on osteopathic technique:

"Rotation of the spine is always accompanied by some degree of lateral flexion. Likewise, lateral flexion of the spine is accompanied by some degree of vertebral rotation."

How All This Came About

Fryette utilized and established correlations with a lot of the earlier work conducted by Lovett in 1903. The research methodology consisted of cadaveric study and in vivo research via the application of gummed paper stickers. These bits of sticky paper were attached to the spinous processes of the vertebrae of a small number of individuals; the results were then obtained by observing the relative spinal motion of these gummed paper stickers.

It is hard to appreciate how much the research into spinal mechanics has progressed over the last 50–100 years, from the simple observatory techniques of Fryette employing sticky paper to the use of advanced technology such as computer simulation, computerized tomography (CT scan), magnetic resonance imaging (MRI scan), and cineradiology (viewing an organ in motion with a special movie camera). We can even detect motion of the spine and pelvis by implanting gallium balls and Steinman pins.

If we were able to jump ahead into the future by, say, 100 years, it would be very interesting to see what actually changes in terms of research and technology. It goes without saying, especially as time passes, that the more we are able to

visualize and research living spinal motion, the more complex and unpredictable the precise combination of individual spinal joint motion becomes for each particular area and segment of the vertebral column.

I have read numerous articles and many books by various authors over the last few years while researching the subject of spinal motion, before eventually deciding to write this particular book on the pelvis and SIJ. Those practitioners in question, who I consider to be experts in their field of manual therapy, all seem to have a slightly different opinion on spinal mechanics/motion, which I think is fine, as everyone is entitled to have an opinion. Some of the information that I have read, however, actually comes across as rather conflicting, because there currently does not appear to be any standardization in this field of study. Rather than having what we call definitive "laws of spinal motion" or "principles of spinal motion," there are substantial individual and regional variations, and, as yet, no true accurate model for predicting the behavior of the vertebral column as regards specific motion.

Koushik physio (2011) says in his website blog:

"The work of Fryette must be applauded for its longevity and insight, and celebrated as part of our osteopathic heritage and history, but the "laws" can no longer be viewed as such, nor do they serve as a viable explanation of physiological motion behavior. With all this uncertainty why do some of us still persist in promoting a model for physiological motion based on work conducted over 100 years ago?"

In one way I totally agree with what this author says. Yes, I am basically of the same mind that perhaps the work of Fryette might be outdated; however, another part of me totally disagrees with what is being said. Why, I hear you ask? Because I spend a lot of my time traveling the world, as well as the UK, teaching a variety of physical therapy courses; when these courses relate in particular to the motion of the spine, it amazes me that the majority of therapists who I come into contact with have not been taught anything about the specific motion of the vertebral column and the biomechanics of the pelvic girdle/SIJ during their own studies … and this truly disappoints me. This is true even in the case of qualified and experienced osteopaths, chiropractors, and physiotherapists.

These days, if I am honest, I have no idea of the current criteria for what is actually covered in terms of spinal motion in specific degree courses run in the UK. One thing I know for sure, however, is that there cannot be much time spent learning about this fascinating area during these undergraduate and even postgraduate courses, as otherwise my own physical therapy courses would not be as busy as they are.

I therefore now have a dilemma: I must at least teach a concept of spinal motion to my students, so the question is, do I teach a concept from Fryette that has been around for over 100 years and is considered by some to be outdated—even though some experts say Fryette's views should not be used any more? Or do I teach what is believed to be the most recent findings, as contradictory as they might be?

The answer is that I of course teach Fryette's Law, as my own personal approach to this is the following. I teach my students one way, one method, and as I have already said, I use the concept of Fryette's way of thinking about the way the spine moves. At least then, I honestly believe, after completing my course the students (including the more experienced therapists) will actually be able to get to grips with, and hopefully have a far better understanding of, the concept of Fryette's laws and the underpinning principles and concepts of spinal mechanics.

Ultimately I am more than happy that those therapists who have chosen to attend my courses will have a good initial grounding in applying the laws of Fryette, hopefully with specific relevance and application to their own private patients and athletes presenting with back or pelvic pain. Naturally, over time, things progress, especially in relation to current research methodology and technology. Therapists can themselves gradually evolve their techniques in practice as they become more experienced and knowledgeable, so they should be able to adapt very well to those ongoing changes.

Gracovetsky's "Spinal Engine Theory"

Serge Gracovetsky (1988) elaborated on a particular idea of spinal motion, which he discussed in his book *The Spinal Engine*. He considered the spine to be the "primary engine" in the role of locomotion and proposed that the legs were not responsible for gait, but were merely "instruments of expression" and extensions of the spinal engine. He argued that the spine was not a rigid lever during the gait cycle and that its ability to produce axial compression and torsion was a fundamental driving force during locomotion.

In his discussions Gracovetsky says that during heel-strike, kinetic energy is not displaced into the earth as in the pedestrian model, but efficiently transmitted up through the myofascial system, causing the spine to resonate in the gravitational field. He did not view the spine as a compressive loading system, whereby the intervertebral discs act as shock absorbers; he regarded the outer annulus disc fibers and their accompanying facet joints as dynamic antigravity torsional springs that store and unload tensional forces to lift and propel the body in space. He also considered that the natural process of interlocking of the facet joints and intervertebral discs transmitted virtually all of the available counter-rotational pelvic torque that is needed to aid the inner and outer core muscles for locomotion.

A quote from Gracovetsky:

"The spine is an engine driving the pelvis. Human anatomy is a consequence of function. The knee cannot be tested in isolation, as it is part of the overall function and purpose of the musculoskeletal system. The leg transfers the heel-strike energy to the spine. It is a mechanical filter. The knee is a critical part of that filter and improper energy transfer will affect spinal motion. Functional assessment of the spine ought to be part of the assessment of knee surgery."

Let's think back to the earlier concept of Lovett: it is this lumbar lateral flexion/rotation coupling that serves as the Gracovetsky spinal engine "drivetrain." For example, left lateral lumbar flexion will drive right rotation of the lumbar spine, and subsequently the SIJ and pelvis.

What I would like to do now is return to the discussion of Chapter 4 and in particular of the gait cycle, and look at this concept in a slightly different way. Some authors have considered that the biceps femoris muscle of the hamstring group, along with its connection to the posterior (deep) longitudinal sling (Figure 6.1), effectively starts the spinal engine. The biceps femoris has been likened to the *pull cord* of the spinal engine in view of its action of inducing a "force closure" mechanism in the SIJ. This closure of the SIJ will naturally lead to a subsequent transmission of force up into the osteo-articular-ligamentous tissues of the lumbosacral spine; this force will eventually continue into the muscles of the lumbar erector spinae.

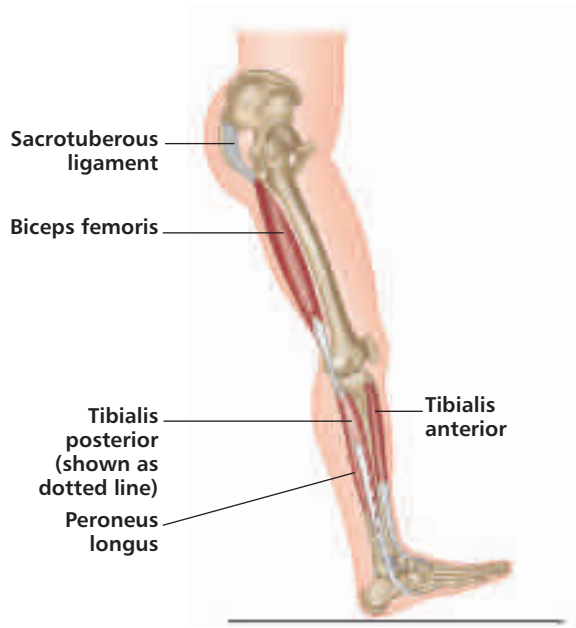


Figure 6.1. Posterior (deep) longitudinal sling.

EMG studies have demonstrated that the biceps femoris muscle is particularly active at the end of the swing phase of gait, through the early loading of the stance phase. During the transition from swing to stance, the heel contact phase of the gait cycle effectively closes the kinetic chain, and the biceps femoris can now perform its work in a manner that is commonly called a *closed kinetic chain*. Within the closed chain, the biceps femoris acts on its more proximal attachment within the chain,

namely the pelvis. The biceps femoris attaches directly to the ischial tuberosity and also to the sacrotuberous ligament, sacrum, iliac crests, and up through the multifidi and lumbar erector spinae (see figure 6.1).

At heel contact the ipsilateral (same side) hip and contralateral (opposite side) shoulder are in a position of flexion, which effectively preloads the posterior oblique sling (Figure 6.2(a)), specifically the ipsilateral Gmax and contralateral latissimus dorsi. This allows extra-spinal propulsion in a "sling-like" manner, with the superficial lamina of the thoracolumbar fascia serving as an intermediary between these kinetically linked muscles as shown by a person running by figure 6.2(b).

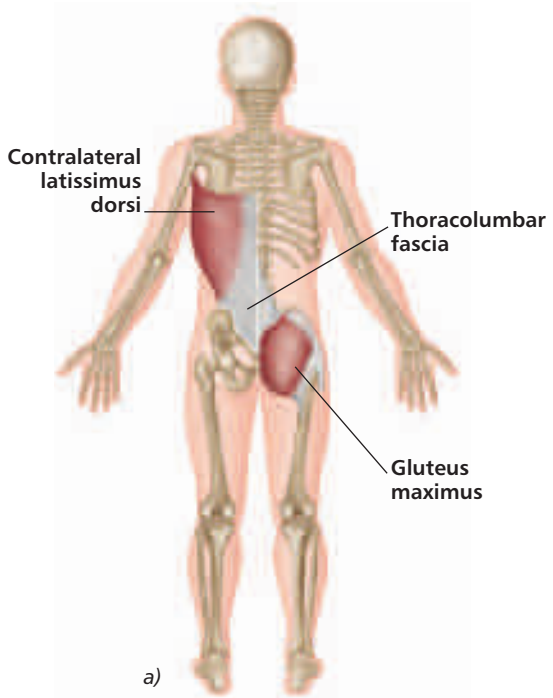


Figure 6.2. (a) Posterior oblique sling.

The force transmitted through the osteo-articular-ligamentous structures induces "form closure" of the spinal facet joints and rotation in the lumbar spine; coupled with a lateral flexion moment, the spinal engine *initiates and selects the gears* to drive the pelvis into a forward rotation. The induced lumbar rotation effectively stores elastic energy in the spinal ligaments and the annulus fibroses of the intervertebral discs, and it is this return of energy that drives gait.

In order to return the energy, the spine must be stabilized from above: this is accomplished via contralateral arm swing and trunk rotation produced by the contralateral Gmax and latissimus dorsi involvement. The coupling patterns of the spine have evolved to facilitate the return of this force. The counter-rotation is considered to be recruited directly from the spine and not from the legs.

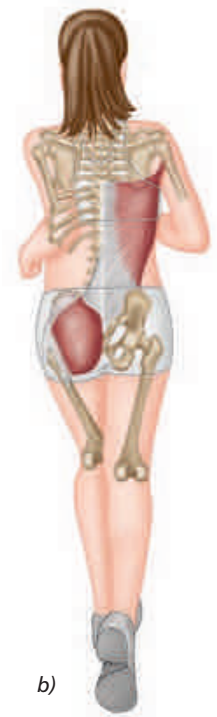


Figure 6.2. (b) Posterior oblique sling utilized whilst running.

Spinal Mechanics Explained

Each and every patient, especially from a spinal mechanics point of view, should be assessed on the basis that they are unique, especially since some people present with vertebral anomalies, which may cause abnormal physiological movement. The following information relating to spinal physiological movement is based on empirical experience rather than on scientific theory; however, it is dependable and useful in helping the budding therapist to understand what they should expect to palpate in the majority of people.

By developing good hand–eye coordination and tactile tissue tension sense, it is certainly possible to detect spinal lesions or, in modern terms, *somatic spinal dysfunctions*. These dysfunctions can be palpated as abnormal vertebral positioning; they function either statically or dynamically, while surrounding abnormal soft tissue texture is a common feature. Once the somatic spinal dysfunctions have been identified and appropriately treated, the offending vertebral segment(s) can then be re-evaluated to determine whether the treatment approach has been successful. This whole process can be demonstrated clinically, provided that the underpinning knowledge of basic spinal physiological movement has been studied and put into practice by the practitioner.

Fryette's Laws basically consist of three laws (originally known as *principles*) pertaining to spinal positioning. The first two laws were developed by Fryette in 1918, and the third law by C.R. Nelson in 1948. The laws are defined as a set of guiding principles that can be used by appropriately trained practitioners to discriminate between dysfunctions that are present within the axial skeleton.

The first two laws only relate to the thoracic and lumbar vertebrae, and the motion available is only considered to be governed by the patterns of force generated by the intervertebral discs, ligaments, and associated musculature. On the other hand, cervical spine motion, which is not classified as a motion that follows Fryette mechanics, is mainly determined by the orientation of the facet joints. We can, however, describe the cervical spine motion as Fryette-*like* mechanics because of the similarity.

Law 1: Neutral Mechanics—Type I

Neutral mechanics relates to standing or sitting in a relaxed upright position, with normal neutral spinal curves. But what is neutral? *Neutral* in the world of spinal mechanics is not defined as a single point, but rather as a range in which the weight of the trunk is borne on the vertebral bodies and intervertebral discs, and the facet joints are in an idle state.

Fryette wrote: "Neutral is defined to mean the position of any area of the spine in which the facets are idling, in the position between the beginning of flexion and the beginning of extension."

This basically means that in neutral, the facet joints are neither in a state of extension (closed) nor in a state of flexion (open)—they are simply idling or resting between these two positions.

According to Fryette, when the spine is in a *neutral* position, side bending to one side will be accompanied by horizontal rotation to the *opposite* side: this is referred to as *Type I spinal mechanics* (Figure 6.3). This first law is observed in what is known as a *Type I spinal dysfunction*, where more than one vertebra is out of alignment and cannot be returned to neutral by flexion or extension of the vertebrae. The group of vertebrae in question demonstrates a coupled relationship: when side bending forces are induced in a group of typical vertebrae to one side, the entire group will rotate to the *opposite* side, in other words obeying Type I or Law 1 spinal mechanics. This spinal motion will produce a convexity similar to a spinal curvature known as a *scoliosis*.

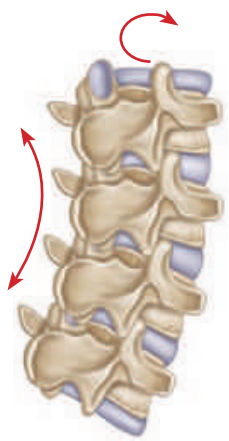


Figure 6.3. Type I spinal mechanics—side bending left, rotation right.

Type I (neutral) dysfunctions generally occur in groups of vertebrae, for example T1–7, and are typically seen in spinal conditions such as scoliosis. The vertebrae of a Type I dysfunction tend to compensate for a single Type II dysfunction, and are usually at the beginning or the end of the group dysfunction, although the dysfunction can sometimes be located at the apex of the curvature.

Another way of looking at Type I spinal mechanics is as follows. In the neutral position for the thoracic and lumbar spines, side bending will create a concavity to the same side as the side bend and a convexity to the side of the rotation (opposite side). For example, side bending to the left will create a concave curve on the left side of the body and a convex curve on the right side.

> **Note**: Neutral spinal mechanics is a naturally occuring motion of the the spine that is required to promote Gracovetsky's spinal engine, due to the side bending and rotation to the opposite side. Any spinal dysfunction that are present will reduce the overall effciency of the 'engine' (spinal).

Law 2: Non-Neutral Mechanics—Type II

Fryette states that when the spine is in a position of flexion or extension, by either standing or sitting while in a forward- or backward-bent position (also known as *non-neutral*), side bending to one side will be accompanied by rotation to the *same* side: this is referred to as *Type II mechanics* (Figure 6.4).

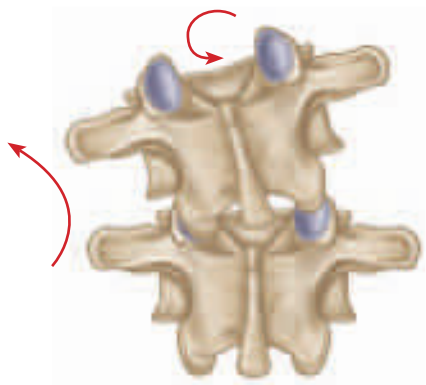


Figure 6.4. Type II spinal mechanics—side bending left, rotation left.

This second law is observed in Type II spinal dysfunctions, where only one vertebral segment is restricted in motion, and becomes much worse in a position of flexion or extension. As mentioned above, there will be a coupled spinal motion, with side bending and rotation in the *same* direction when this dysfunction is present.

Let's look at this from another angle. Put simply, if the thoracic or lumbar spine is sufficiently forward or backward bent, the coupled motions of side bending and rotation of a single vertebral unit will occur in the same direction (i.e. to the same side).

Type II (non-neutral) spinal dysfunctions are generally thought to occur in a single vertebral segment. However, two Type II dysfunctions can appear next to each other at the same time, but the occurrence of more than two is rare.

Law 3

According to Nelson (1948), when motion is introduced in one plane, the motion in the other two planes is modified (reduced). The third law basically sums up the first two laws: it simply states that dysfunction in one plane of motion will reduce the motion in all other planes. For example, if rotation is restricted, then side bending and flexion/extension will also be restricted.

Viewing Perspective

When assessing for spinal movement, the spine is usually viewed from behind. Generally speaking, a spinal dysfunction exists when we try to place the patient's spine in a neutral position, but this position of symmetry cannot to be achieved; the likely reason for this is a dysfunction of either a group of vertebrae or a single spinal segment.

It is possible to detect an abnormal ROM of the spine when patients perform flexion, extension, side bending, and rotation in the cardinal planes; one should be able to see if there is a restricted ROM.

The following sections detail the specific spinal mechanic motion for each of the areas of the vertebral column. I have already explained the term *neutral* (i.e. the facet joints are idling between flexion and extension), so this position is termed *neutral mechanics*. By contrast, the term *non-neutral mechanics* generally refers to either a spinal position of forward bending (flexion) or a spinal position of backward bending (extension).

1. Lumbar Spine

Neutral Mechanics: Type I
When side bending to one side, the vertebral bodies rotate to the *opposite* side.

Non-Neutral Mechanics: Type II
When side bending to one side, the vertebral bodies rotate to the *same* side.

Exceptions to the Rule: 5th Lumbar Vertebra (L5)
In neutral mechanics, during side bending to one side the 5th lumbar vertebra (L5) may rotate to either the opposite side or the same side (obeying non-neutral mechanics, even though the spine is in a relatively neutral position) whenever there is some asymmetry/dysfunction of the sacral base or if there are facet joint anomalies present.

2. Thoracic Spine

Neutral Mechanics—Type I
When side bending to one side, the vertebral bodies rotate to the *opposite* side.

Non-Neutral Mechanics—Type II
When side bending to one side, the vertebral bodies rotate to the *same* side.

3. Cervical Spine

As mentioned earlier, spinal motion in the cervical spine is mainly determined by the orientation of the facet joints and does not come within the realm of Fryette's laws; however, we can say these mechanics are Fryette-*like*, because the motion is similar.

Occipitoatlantal Joint
The occipitoatlantal joint (OAJ) connects the occipital bone to the atlas and always follows Type I (like) mechanics: in side bending to one side, the occiput rotates to the *opposite* side, independent of whether the occiput is in a neutral or a non-neutral position (flexion or extension).

Atlantoaxial Joint C1/C2
The atlantoaxial joint (AAJ), which lies between the atlas and the axis, is mainly considered to only rotate. There has been some discussion suggesting that, in either neutral or non-neutral mechanics, during side bending movements the atlas (C1) is capable of rotating to either side. Dysfunctions typically found at this level are thought to consist mainly of a rotational component.

The level of C2–C6 in the cervical spine is believed to only follow Type II (like) mechanics, in that side bending and the rotation are always coupled to the *same* side, regardless of whether the cervical spine is in a neutral position or a non-neutral position (flexion or extension).

C2–C6: Neutral Mechanics
When side bending to one side, the vertebral bodies rotate to the *same* side, i.e. Type II-*like* mechanics.

C2–C6: Non-Neutral Mechanics
When side bending to one side, the vertebral bodies rotate to the *same* side, i.e. Type II-*like* mechanics.

C7 has facet joints that are orientated in a similar way to the thoracic spine, so this spinal level will follow the classic law of Fryette.

Spinal Mechanics: Definitions

The specific position of a vertebra can be referred to in two different ways:

1. The position of the vertebra, relative to the vertebra below.

2. The direction of the motion restriction of the vertebra, relative to the vertebra below.

In other words, the same vertebral segment can be described from two different points of view.

For example, let's say T4 (4th vertebra of the thoracic spine) is fixed in an extended, side-bent right, and rotated-right position. This simply means that the T4 is fixed in a position of extension, side bend, and rotation, all to the right side, on top of the vertebra immediately below, i.e. T5. This is because the right T4 inferior facet is fixed in a *closed* position on the superior facet on T5. The motion restriction has to be in forward flexion as well as in left side bending and left rotation (opposite to the fixed position). This type of dysfunction obeys Type II mechanics, as already explained earlier; however, taking it a stage further, we would now classify the spinal dysfunction as a *T4 extension, rotation, side bend right*, or *T4 ERS(R)*, which will be discussed shortly.

In terms of diagnosing somatic spinal dysfunction, the positional diagnosis is determined and named according to the direction of the vertebra that has the easiest motion. Let's take a look at what I mean by that in more detail.

Spinal dysfunction is typically described as either *extended* or *flexed*, with a rotation and a side bending component to the same side, or possibly to the opposite side, as you will read later.

Before we define the terminology for spinal dysfunctions, we first need to confirm the presence of a spinal dysfunction by ascertaining the specific position and motion of the vertebra being tested. We can establish this by asking our patient to adopt three different positions of the vertebral column: neutral, extension, and flexion. The vertebral position through palpation simply becomes either symmetrical (level) or asymmetrical (not level) in these three positions, depending on the type of spinal dysfunction/facet restriction present at the time.

If there are no facet restrictions present, when you forward bend your spine, the left and right facet joints (top vertebra in relation to the bottom vertebra) will slide forward in a superior and anterior direction to open; conversely, when you backward bend your spine, the left and right facet joints will slide backward in an inferior and posterior direction to close. However, if the facet joints are for some reason restricted in either a flexion or an extension position, the restricted joint will now act as a pivot point, especially when performing the spinal motion of forward and backward bending.

To illustrate this, ask your patient to adopt a neutral position (normally a sitting position, for the thoracic spine) and lightly place your left and right thumbs on the T4/5 transverse process (TP). Lightly palpate for a few seconds and compare the left and right TPs to see if there is any asymmetry; if so, you have now identified the presence of a spinal dysfunction—in a very simple way, you have located a facet restriction. For instance, if the left thumb, while in contact with the left TP, appeared to palpate *shallow* (i.e. the TP feels closer to the surface of the skin), whereas the right thumb (on the right TP) palpated *deep* (i.e. the thumb traveled further to reach the right TP), this would be indicative of the T4 vertebra (superior) having rotated to the left side on the T5 vertebra (inferior) below, as shown in Figure 6.5(c).

What this does not tell you, however, is whether the *left* facet joint is fixed in a *closed* position or whether the *right* facet joint is fixed in an *open* position. That is why it is necessary to palpate the TPs in the position of spinal extension (backward bending) and spinal flexion (forward bending) in order to confirm or discount the presence of a fixed closed facet joint or a fixed open facet joint.

Let's try to look at this concept in a relatively simple way (I hope) by using the thoracic T4/5 as an example. As already discussed above, we should now know that when the patient is in a neutral position, the T4 vertebra has rotated to the left side, because the left TP palpated as shallow (more prominent) and the right TP palpated as deep (less prominent). From the neutral position, we now ask our patient to forward bend, while the thumbs are still in contact with the TPs; you notice the left and right TPs in the forward-bent position become more asymmetrical (the TP on one side becomes more prominent, and the TP on the other less prominent).

Imagine just for a moment that you consider the left thumb to feel more prominent (think of it as a bump) and now the right thumb feels even deeper (less prominent); this must mean that the left facet joint is fixed in a *closed* position on the *left* side, as shown in Figure 6.5(i). Why? When the patient forward bends, the right facet joint is free to glide anteriorly as normal, but the left facet joint is now fixed posteriorly; because the left facet cannot open normally during forward bending, the left facet joint that is fixed in a closed position becomes a pivot point. Because of this pivot, one could say that the right facet basically has to open even more during forward bending, but ends up rotating around the left fixed facet joint that cannot open; this is why the left and right thumbs appear even more asymmetric. In this case the left thumb has become more prominent (the bump increases because the left TP is fixed posteriorly) in the forward-bent position.

When you ask the patient to adopt a position of extension, the thumbs will now palpate level (symmetric), because the left facet joint is already fixed posteriorly in a closed position, and the right facet joint simply continues its natural motion of closing. The left and right thumb positions on the TPs therefore become symmetric (level) in extension (the bump on the left TP disappears), as shown in Figure 6.5(f).

Consider now another type of dysfunction—the case where the right facet joint is fixed in an open position. When we palpate the T4/5 TP in neutral, we will still notice the left thumb is shallow (bump) and the right thumb is deeper, indicating a left rotation, as shown in Figure 6.9(c). However, when the patient forward bends their spine, the thumbs now become symmetric (i.e. the thumbs are now level and the bump disappears), as shown in Figure 6.9(i). By contrast, when the patient backward bends their spine, you notice that the thumbs become asymmetric (i.e. symmetry is lost in extension—the bump felt by the left thumb on the TP increases), as shown in Figure 6.9(f). This time the left thumb actually appears more prominent (bump present), and the right thumb appears to travel deeper in the backward-bent position. Why? In a forward-bent position, both of the facet joints are able to open as normal, hence the thumbs becoming level in this position; however, in a backward-bent position of extension, because the *right* facet is fixed in an *open* position (even though it has rotated to the left side), the facet joint cannot close on the right side, but the fixed pivot point created by the right open facets keeps the right TP fixed anteriorly. Since the backward bending motion causes the left side to move more posteriorly, the left TP appears to move further into left rotation. The thumbs therefore appear asymmetric and now, in the backward-bent position, the left thumb on the left TP becomes more prominent (bump appears) than the right thumb.

> **Note**: The above dysfunction relates to the right facet joint fixed in an open position. This is called an FRS(L) as you will read shortly.

One way of remembering these two processes, as it will help you with your own patients, is to understand the following two rules:

Rule 1: In forward bending, if the prominent TP becomes even more prominent (bump appears), then that side is fixed closed.

Rule 2: In backward bending, if the prominent TP becomes more prominent (bump appears), then the opposite side is fixed open.

Maitland (2001) offers another explanation, which might help you better understand what I am trying to say. First, you determine rotation in a neutral position. Then, keeping your thumbs on the TPs of the rotated vertebra, forward and backward bend your client, and feel and watch what happens under your thumbs. Look for the position where the bump (the posterior or prominent vertebra TP of the rotated vertebra) disappears. Maitland says that the position where the bump disappears (or vertebral derotation appears to occur) is the position in which the facets are restricted:

"If the bump disappears in forward bending, the facets are fixed in the forward-bent position, which means the facets are fixed open (flexion fixed)."

"If the bump disappears in back bending, the facets are fixed in the back bent position, which means the facets are fixed closed (extension fixed)."

We will now look at some of the typical terminology that practitioners use in their

clinics, along with a few common examples of spinal dysfunctional patterns that patients might present with in your own clinic. Hopefully, once you have read all the information in this chapter, and practiced all the necessary spinal positions in order to confirm or discount the presence of either a fixed facet or an open facet, we can look at some form of treatment strategy, especially for the lumbar spine (discussed in Chapter 13). Specific treatment for the area of the thoracic spine, however, is not covered in this text, even though I have given some examples of the assessment process. Nevertheless, one should be able to modify the treatment techniques shown for the lumbar spine in Chapter 13 and adapt/apply them to dysfunctions in the thoracic spine.

> **Definition:** An *extension, rotation, side bending (ERS) dysfunction* involves a facet joint (inferior and superior component) which is fixed in a closed position. It is classified as a *non-neutral (Type II) spinal mechanics dysfunction*.

Extension, Rotation, Side Bending Left—ERS(L)

This is a situation in which the *left* facet joint is fixed in a *closed* position.

ERS(L) refers to the orientation of an uppermost vertebra that is fixed in an extended, side-bent, and rotated position to the left side, as shown in Figure 6.5(a).

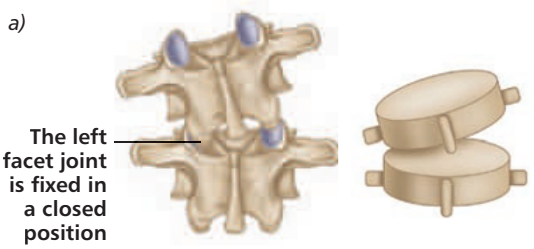
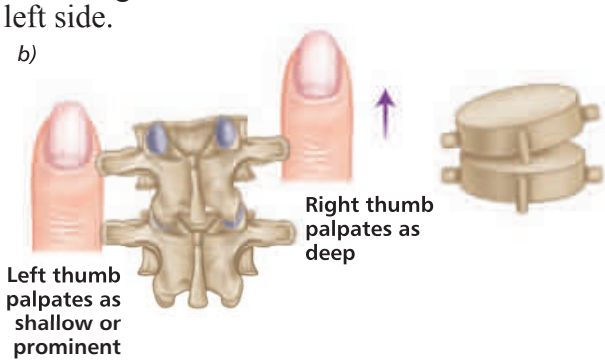


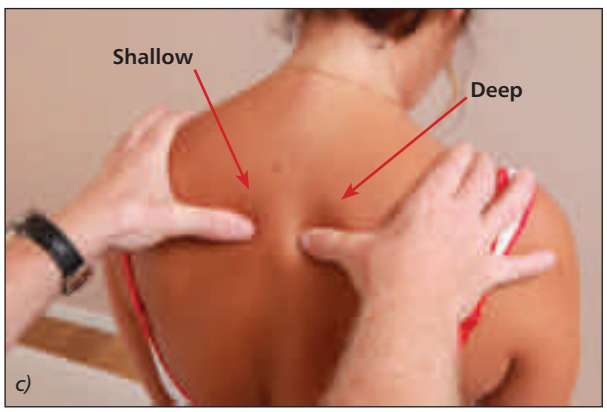
Figure 6.5. (a) Extension, rotation, side bending left—ERS(L).

Two examples will be considered: in the first, the 4th thoracic vertebra (T4) is assumed to be fixed in a closed position on the left side on the 5th thoracic vertebra (T5); in the second, the 5th lumbar vertebra (L5) is assumed to be fixed in a closed position on the left side on the 1st sacral vertebra (S1). We will now test the levels of T4/5 and L5/S1 in the three positions of neutral, flexion, and extension.

Neutral Position

With the patient in a neutral position, place your thumbs approximately 1" (2.5cm) lateral to the T4 and T5 spinous processes, so that the thumbs are in gentle contact with the left and right TPs (repeat the same process when you are ready to do the L5/S1 vertebrae). If there is an ERS(L) present, you will notice that the left thumb appears to be more prominent (shallow) and the right thumb appears to be deeper in the neutral position (see Figure 6.5(b–d)), indicating that the vertebra has rotated to the left side.





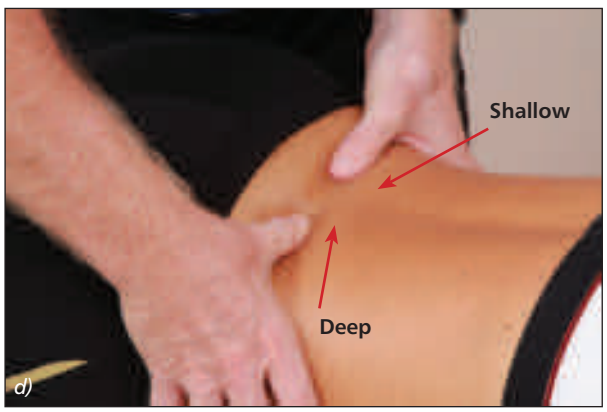
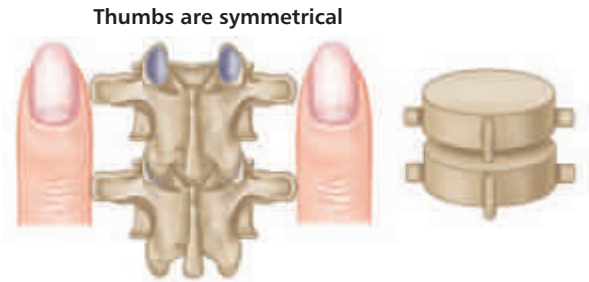
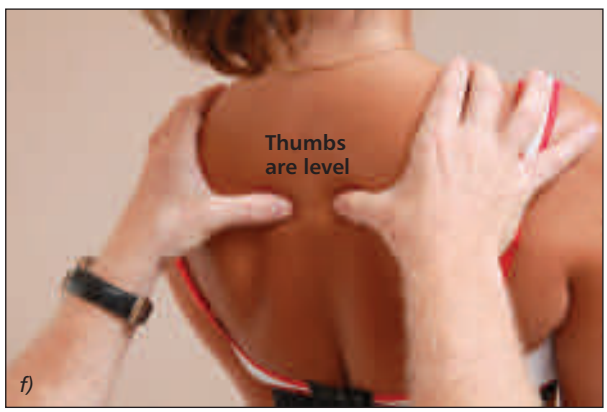


Figure 6.5. (b) Neutral position—the left thumb appears shallow and the right thumb appears deeper, indicating a left rotation of the vertebra. (c) Thoracic spine T4/5. (d) Lumbar spine L5/S1.

Extension Position

As the patient backward bends, observe the relative levels of your thumbs. You will notice that the left and right thumbs are level, as shown in Figure 6.5(e–g).





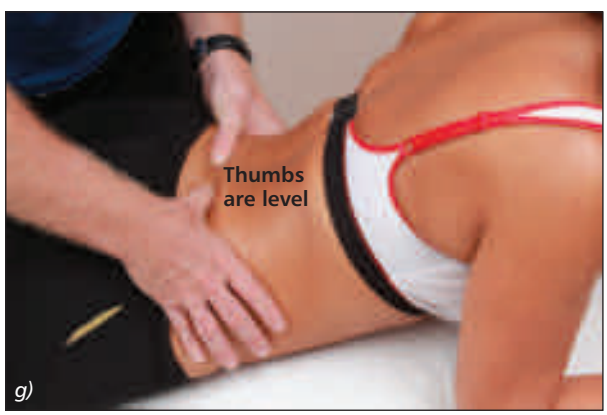
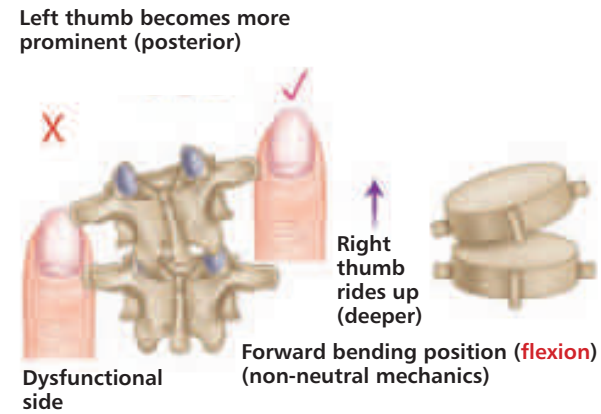


Figure 6.5. (e) Extension position—the left and right thumbs now appear to become level. (f) Thoracic spine T4/5. (g) Lumbar spine L5/S1.

Flexion Position

Next look at the relative positions of your thumbs as the patient forward bends. This time you will notice that the left thumb appears to become more prominent (bump appears) and that the right thumb appears to travel deeper, as shown in Figure 6.5(h–j). This asymmetric positioning of the thumbs indicates a fixed closed facet joint on the left side.



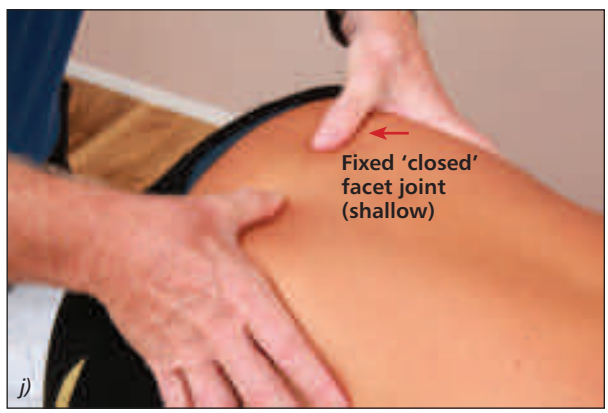


Figure 6.5. (h) Flexion position—the left thumb appears more prominent and the right thumb appears deep, indicating a fixed closed vertebra on the left side. (i) Thoracic spine T4/5. (j) Lumbar spine L5/S1.

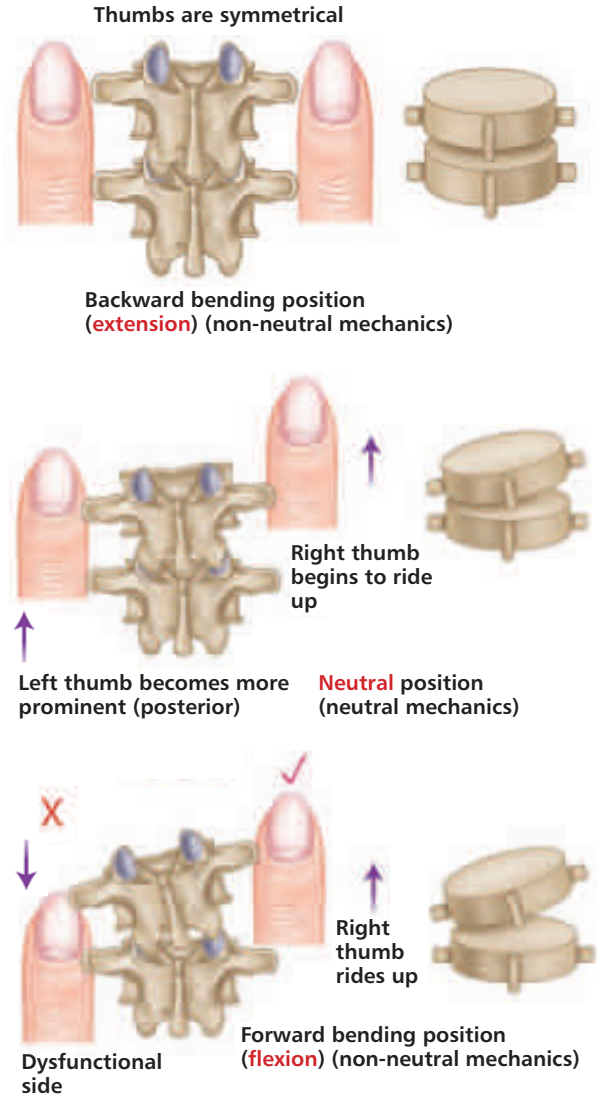


Figure 6.6. Thumb positions for ERS(L).

Extension, Rotation, Side Bending Right—ERS(R)

This is a situation in which the *right* facet joint is fixed in a *closed* position.

ERS(R) refers to the orientation of an uppermost vertebra that is fixed in an extended, side-bent, and rotated position to the right side, as shown in Figure 6.7(a).

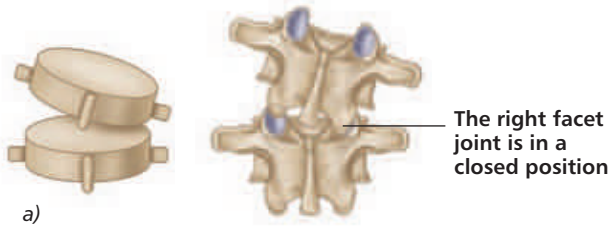
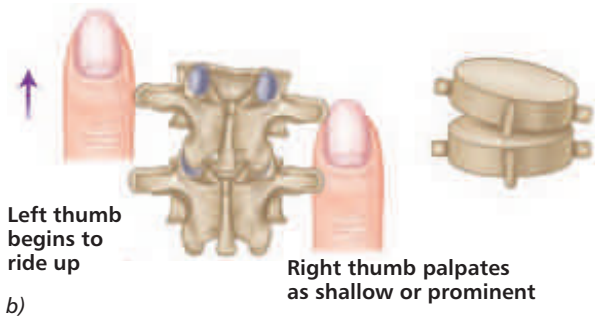


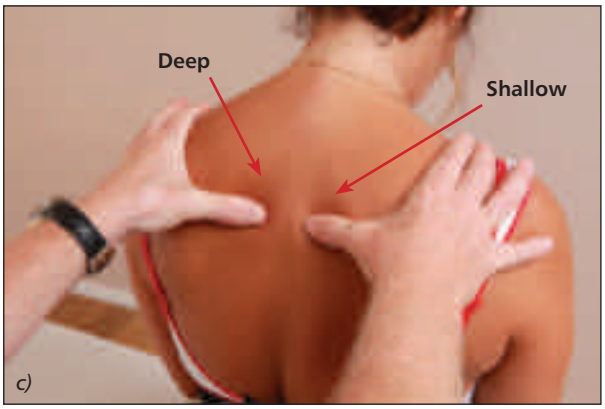
Figure 6.7. (a) Extension, rotation, side bending right—ERS(R).

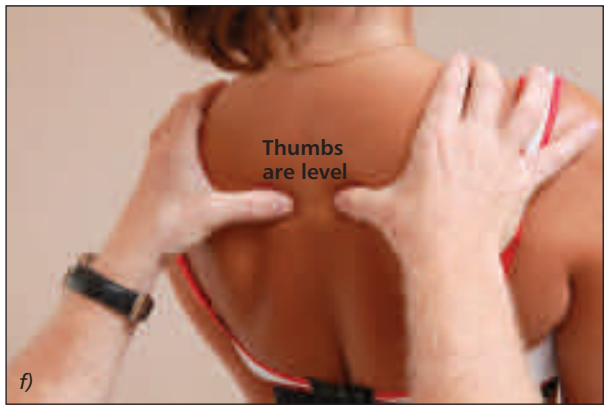
Two examples will be considered: in the first, T4 is assumed to be fixed in a closed position on the right side on T5; in the second, L5 is assumed to be fixed in a closed position on the right side on S1. We will now test the levels of T4/5 and L5/S1 in the three positions of neutral, flexion, and extension.

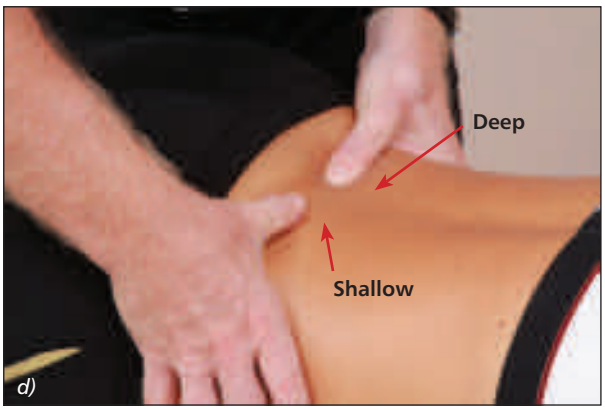
Neutral Position

With the patient in a neutral position, place your thumbs approximately 1" (2.5cm) lateral to the T4 and T5 spinous processes, so that the thumbs are in gentle contact with the left and right TPs (repeat the same process when you are ready to do the L5/S1 vertebra). If there is an ERS(R) present, you will notice that the right thumb appears to be shallow and the left thumb appears to be deeper in the neutral position (see Figure 6.7(b–d)), indicating a right rotation of the vertebra.









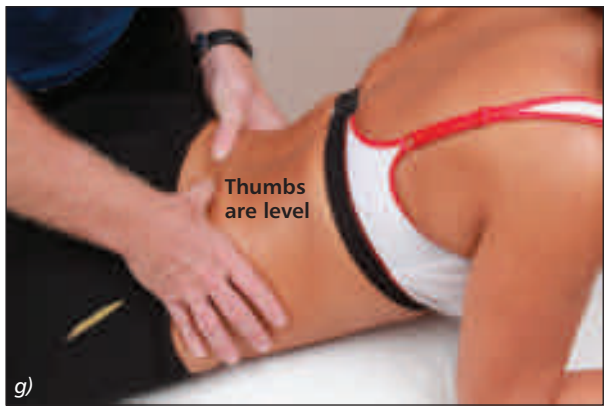
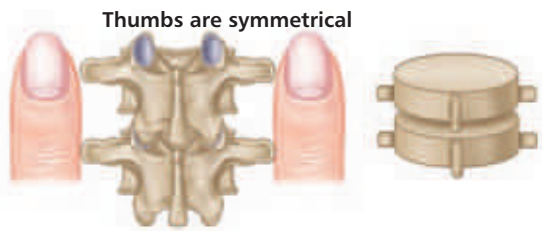


Figure 6.7. (b) Neutral position—the right thumb appears more prominent and the left thumb appears deeper, indicating a right rotation of the vertebra. (c) Thoracic spine T4/5. (d) Lumbar spine L5/S1.

Figure 6.7. (e) Extension position—the left and right thumbs now appear to become level. (f) Thoracic spine T4/5. (g) Lumbar spine L5/S1.

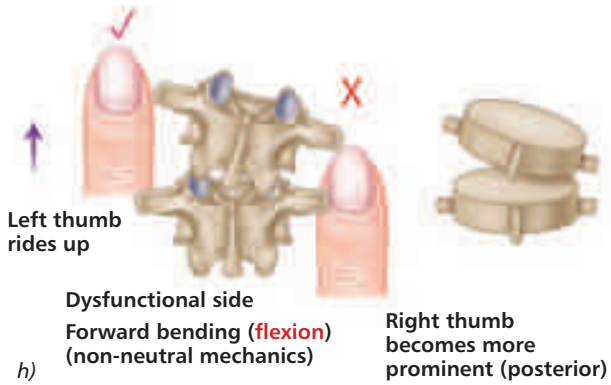
Extension Position

As the patient backward bends, observe the relative levels of your thumbs. You will notice that the left and right thumbs are level, as shown in Figure 6.7(e–g).



Flexion Position

Next look at the relative level of your thumbs as the patient forward bends. You will notice that the right thumb appears to become more prominent (bump appears) and the left thumb appears to travel deeper, as shown in Figure 6.7(h–j). This asymmetric positioning of the thumbs indicates a fixed closed facet joint on the right side.



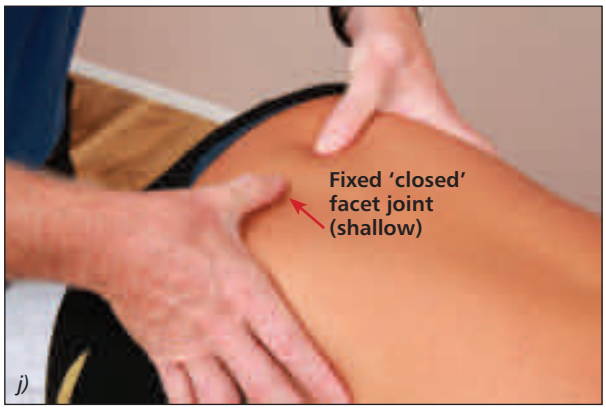
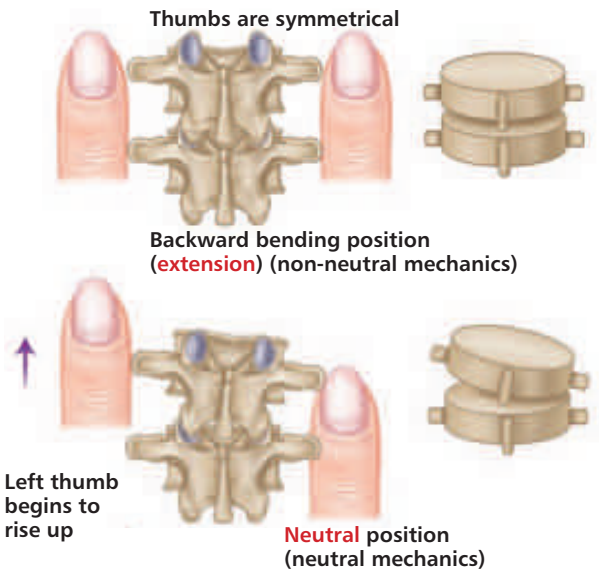


Figure 6.7. (h) Flexion position—the right thumb appears more prominent and the left thumb appears deeper, indicating a fixed closed vertebra on the right side. (i) Thoracic spine T4/5. (j) Lumbar spine L5/S1.



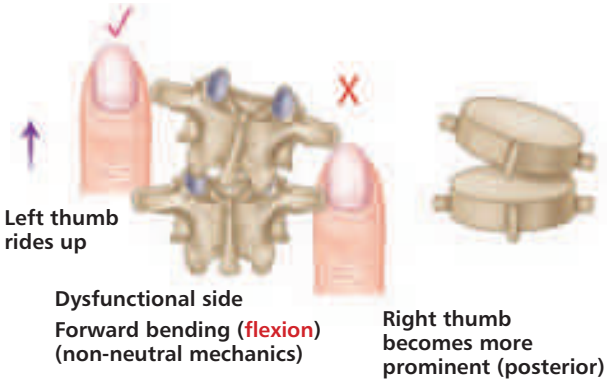


Figure 6.8. Thumb positions for ERS(R).

Definition: A *flexion, rotation, side bending (FRS) dysfunction* involves a facet joint which is fixed in an open position. It is classified as a *non-neutral (Type II) spinal mechanics dysfunction*.

Flexion, Rotation, Side Bending Left—FRS(L)

This is a situation in which the *right* facet joint is fixed in an open position.

Note that, although the dysfunction is to the *right* facet, *FRS(L)* refers to the orientation of an uppermost vertebra that is fixed in a flexed, side-bent, and rotated position to the *left* side. It is the *right* facet, however, that is dysfunctional, because it is fixed in an open position, as shown in Figure 6.9(a).

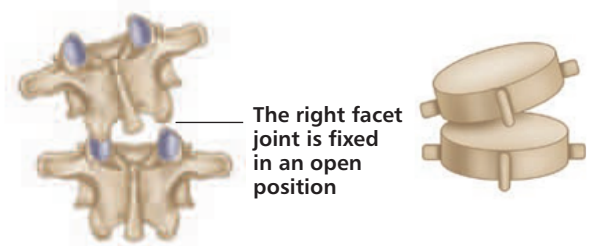


Figure 6.9. (a) Flexion, rotation, side bending left—FRS(L).

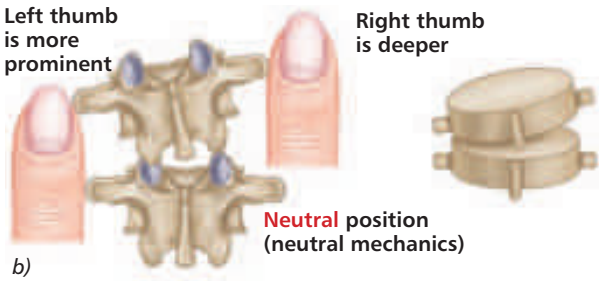
We will now test the levels of T4/5 and L5/S1 in the three positions of neutral, flexion, and extension.

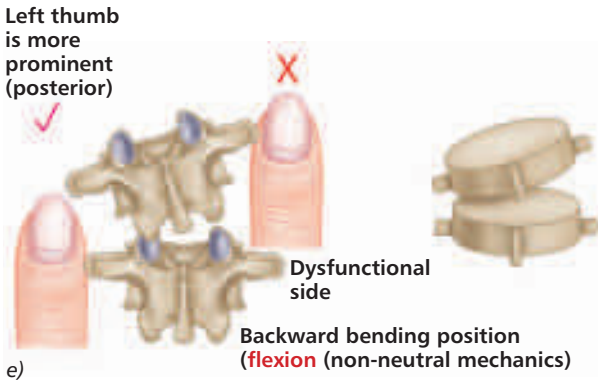
Neutral Position

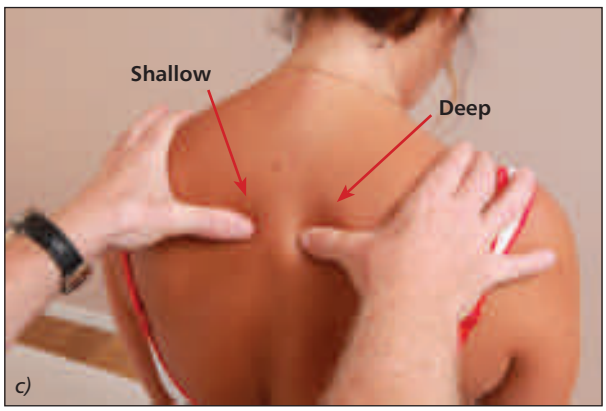
With the patient in a neutral position, place your thumbs approximately 1" (2.5cm) lateral to the T4 and T5 spinous processes, so that the thumbs are in gentle contact with the left and right TPs (repeat the same process when you are ready to do the L5/S1 vertebra). If there is an FRS(L) present, you will notice that the left thumb appears to be more prominent and the right thumb appears to be deeper in the neutral position (see Figure 6.9(b–d)), indicating that the vertebra has rotated to the left side.

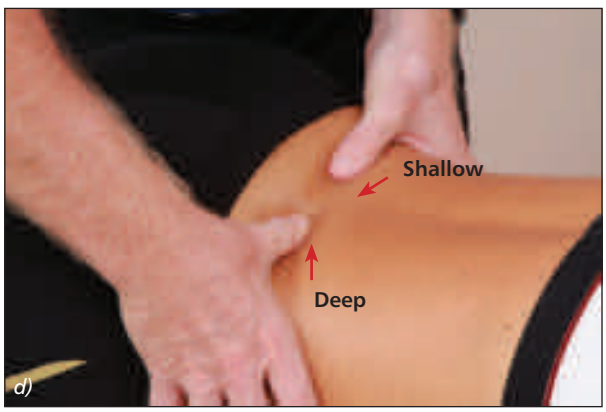
Extension Position

As the patient backward bends, observe the relative position of your thumbs. You will notice that the left thumb appears to become more prominent (bump appears) and the right thumb appears to travel deeper, as shown in Figure 6.9(e–g). This asymmetric positioning of the thumbs indicates a fixed open facet joint on the right side (side opposite to rotation).









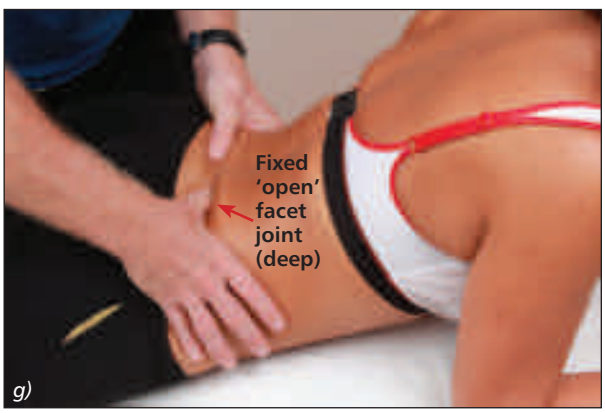
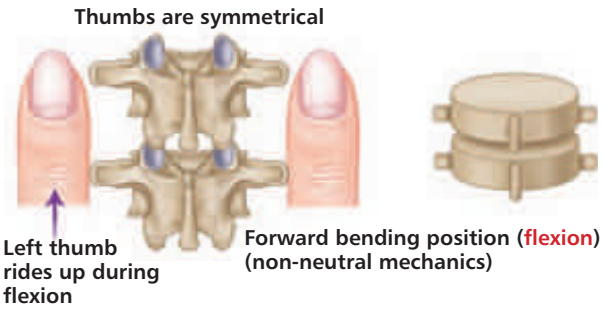


Figure 6.9. (b) Neutral position—the left thumb appears shallow and the right thumb appears deeper, indicating a left rotation of the vertebra. (c) Thoracic spine T4/5. (d) Lumbar spine L5/S1.

Figure 6.9. (e) Extension position—the left thumb appears more prominent and the right thumb appears deeper, indicating a fixed open facet joint on the right side. (f) Thoracic spine T4/5. (g) Lumbar spine L5/S1.

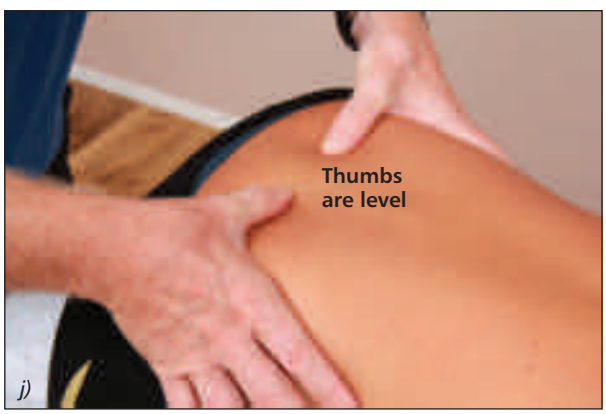
Flexion Position

Next look at the relative position of your thumbs as the patient forward bends. You will notice that the left and right thumbs are now level, as shown in Figure 6.9(h–j).



h)

i)



j)

Figure 6.9. (h) Flexion position—the left and right thumbs now appear to become level. (i) Thoracic spine T4/5. (j) Lumbar spine L5/S1.

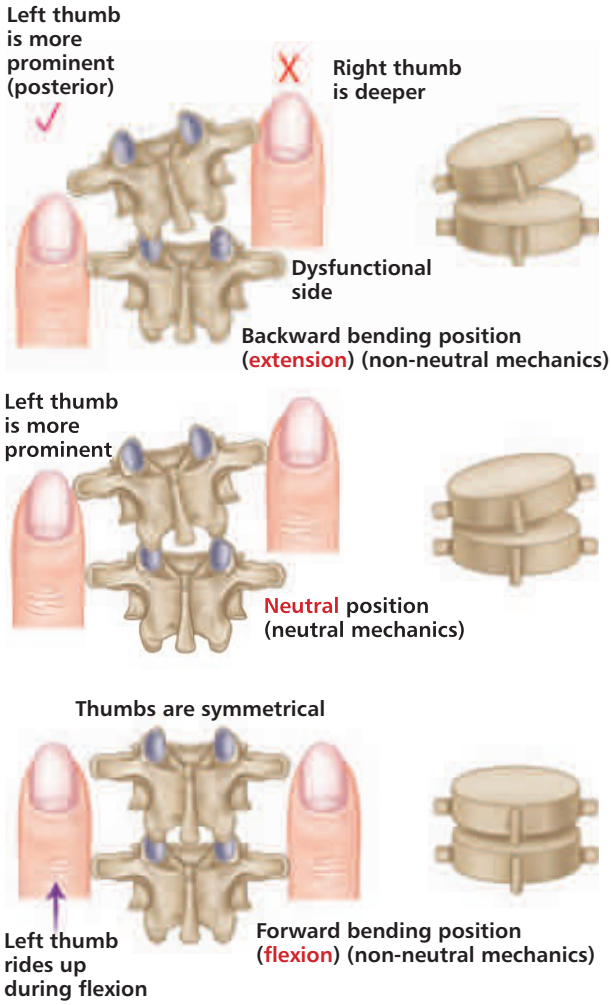


Figure 6.10. Thumb positions for FRS(L).

Flexion, Rotation, Side Bending Right—FRS(R)

This is a situation in which the *left* facet joint is fixed in an open position.

Note that, although the dysfunction is to the *left* facet, *FRS(R)* refers to the orientation of an uppermost vertebra that is fixed in a flexed, side-bent, and rotated position to the *right* side. It is the *left* facet, however, that is dysfunctional, because it is fixed in an open position, as shown in Figure 6.11(a).

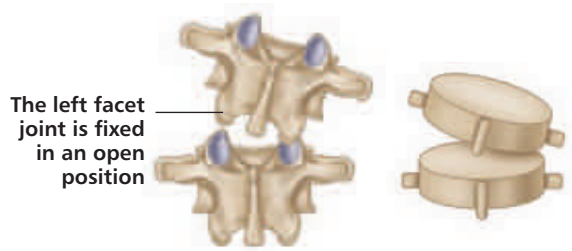
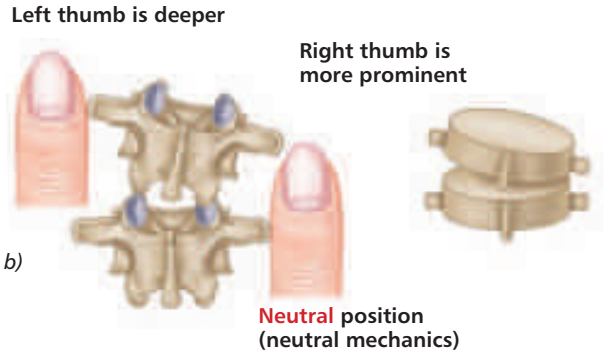


Figure 6.11. (a) Flexion, rotation, side bending right—FRS(R).

We will now test the levels of T4/5 and L5/S1 in the three positions of neutral, flexion, and extension.

Neutral Position

With the patient in a neutral position, place your thumbs approximately 1" (2.5cm) lateral to the T4 and T5 spinous processes, so that the thumbs are in gentle contact with the left and right TPs (repeat the same process when you are ready to do the L5/S1 vertebra). If there is an FRS(R) present, you will notice that the left thumb appears to be shallow and the right thumb appears to be deeper in the neutral position (see Figure 6.11(b–d)), indicating that the vertebra has rotated to the left side.



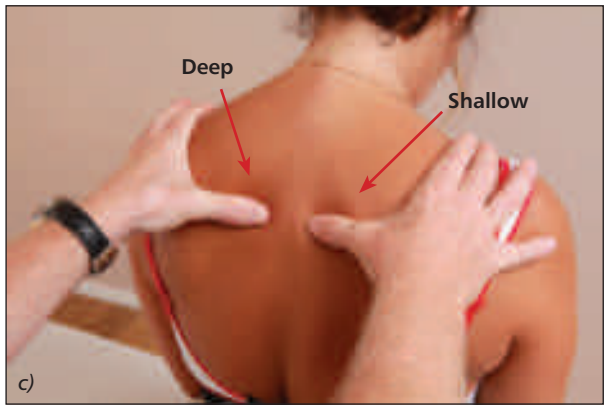
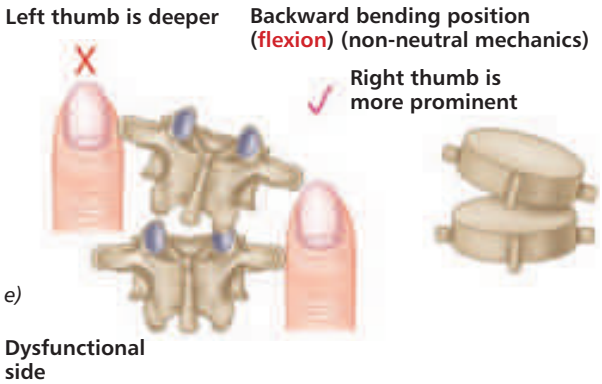


Figure 6.11. (b) Neutral position—the right thumb appears shallow and the left thumb appears deeper, indicating a right rotation of the vertebra. (c) Thoracic spine T4/5. (d) Lumbar spine L5/S1.

Extension Position

As the patient backward bends, observe the relative position of your thumbs. You will notice that the right thumb appears to become more prominent (bump appears) and the left thumb appears to travel deeper, as shown in Figure 6.11(e–g). This asymmetric positioning of the thumbs indicates a fixed open facet joint on the left side (side opposite to rotation).



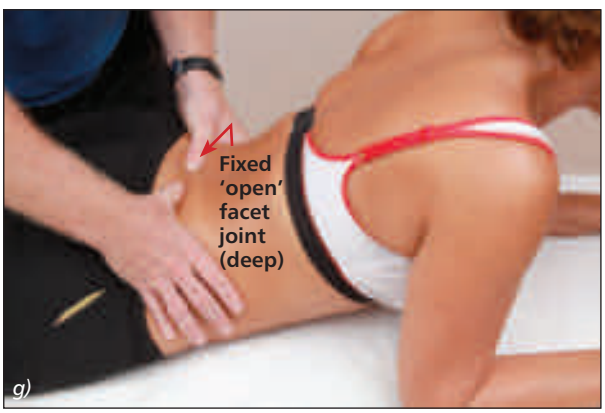
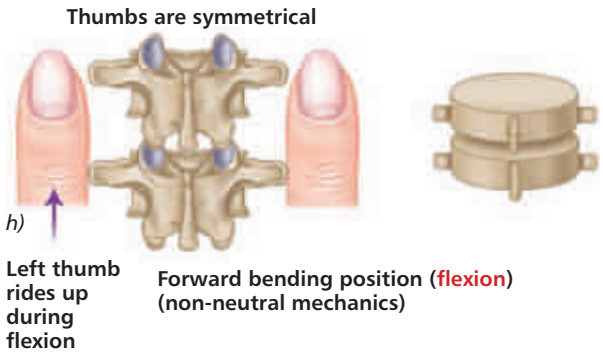


Figure 6.11. (e) Extension position—the right thumb appears more prominent and the left thumb appears deeper, indicating a fixed open facet joint on the left side. (f) Thoracic spine T4/5. (g) Lumbar spine L5/S1.

Flexion Position

Next look at the relative position of your thumbs as the patient forward bends. You will notice that the left and right thumbs are now level, as shown in Figure 6.11(h–j).



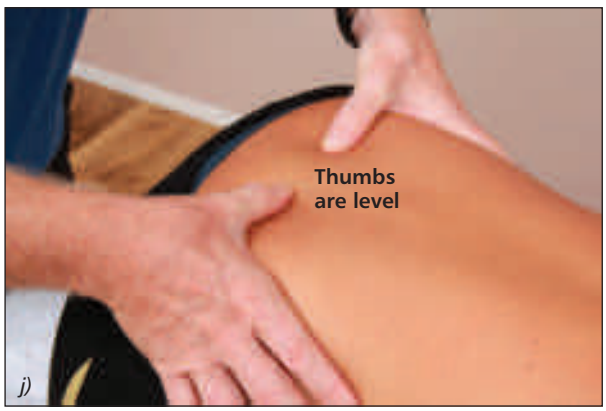


Figure 6.11. (h) Flexion position—the left and right thumbs now appear to become level. (i) Thoracic spine T4/5. (j) Lumbar spine L5/S1.

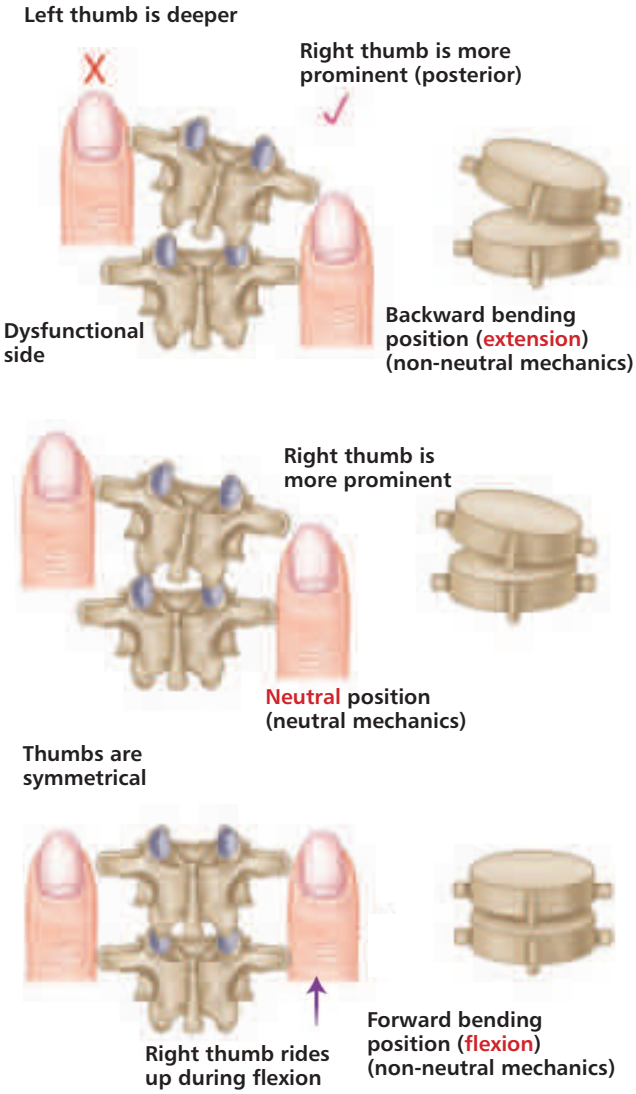


Figure 6.12. Thumb positions for FRS(R).

Definition: A *neutral mechanics (Type I) dysfunction of a group of vertebrae (NR)* typically involves a group of three or more vertebrae, and the side bending is considered to be the primary motion restriction, with a secondary rotational component. As explained earlier, this dysfunction produces a concavity on the same side as the side bending, and a convexity on the side of the rotation. This type of dysfunction is considered to be a compensatory group dysfunction as a result of a primary dysfunction; the primary dysfunctions are usually an FRS or ERS. The vertebrae in question side bend to one side and rotate to the opposite side.

Group Neutral Dysfunction, Rotation Left—NR(L)

This is a situation involving at least three vertebrae, which are side bent to the right and rotated to the left.

The rotation to the left is maintained throughout the range of backward bending, neutral, and forward bending, as shown in Figure 6.13. The amount of rotation may vary a little throughout this ROM, and will most likely be maximal in the neutral position.

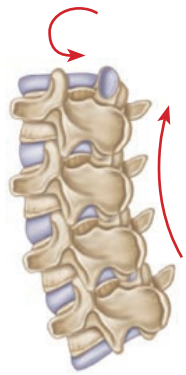


Figure 6.13. Neutral dysfunction—side bending right, rotation left—NR(L).

Group Neutral Dysfunction, Rotation Right—NR(R)

This is a situation involving at least three vertebrae, which are side bent to the left and rotated to the right.

The rotation to the right is maintained throughout the range of backward bending, neutral, and forward bending, as shown in Figure 6.14. The amount of rotation may vary a little throughout this ROM, and will most likely be maximal in the neutral position.

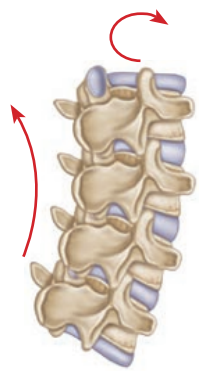
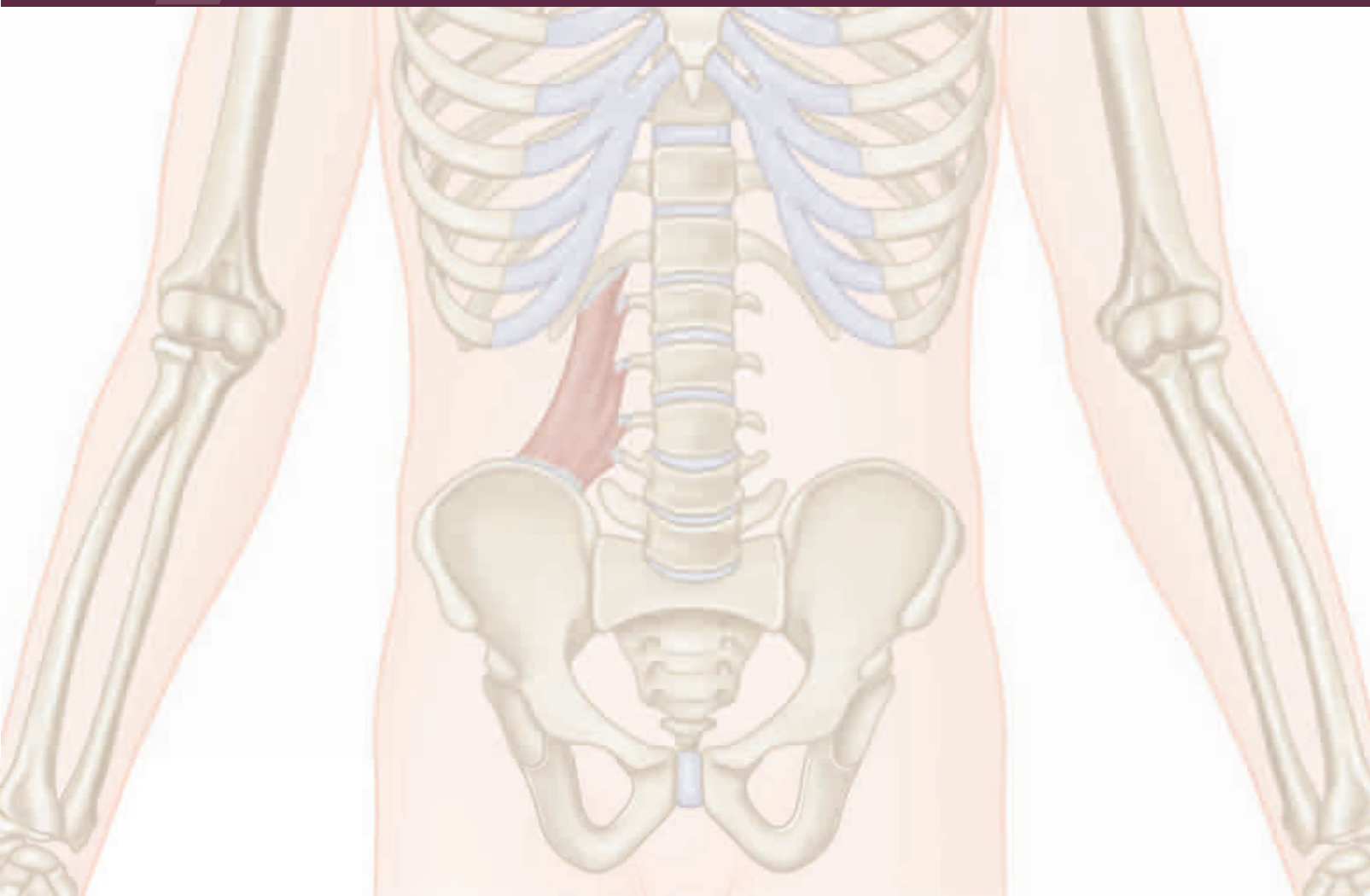


Figure 6.14. Neutral dysfunction—side bending left, rotation right—NRR

7

Muscle Energy Techniques and Their Relationship to the Pelvis



In later chapters you will read and learn about specific techniques that can be incorporated into a treatment plan to help correct pelvic and lumbar spine dysfunctions. I consider the techniques I will be demonstrating in this chapter to be some of the best soft tissue techniques that one can use to correct any soft tissue or spinal joint anomalies. You might have already guessed what these are—*muscle energy techniques (METs)*.

Since I discuss in this book how to treat specific dysfunctions associated with the SIJ, pelvis, and lumbar spine, I need to explain the role of METs, so that you have a better understanding of when and why to employ this type of soft tissue treatment. Physical therapists have what I call a toolbox of various techniques at their disposal, to help release and relax muscles, which will then assist the patient's body to promote the healing mechanisms. METs, first described by Fred Mitchell in 1948, are one such tool, which if used correctly can have a major influence on a patient's well-being. (The reader is referred to Gibbons (2011) for a fuller account of METs.)

> **Definition:** *Muscle energy techniques (METs)* are a form of osteopathic manipulative diagnosis and treatment in which the patient's muscles are actively used on request, from a precisely controlled position, in a specific direction, and against a distantly applied counterforce.

METs are unique in their application, in that the patient provides the initial effort and the practitioner just facilitates the process. The primary force comes from the contraction of the patient's soft tissues (muscles), which is then utilized to assist and correct the presenting musculoskeletal dysfunction. This treatment method is generally classified as a *direct* form of technique as opposed to *indirect*, since the use of muscular effort is from a controlled position, in a specific direction, and against a distant counterforce that is usually applied by the practitioner.

Some of the Benefits of METs

When teaching the concept of METs to my students, one of the benefits I emphasize is their use in normalizing joint range, rather than in improving flexibility. This might sound counterintuitive; what I am saying is if, for example, your patient cannot rotate their neck (cervical spine) to the right as far as they can to the left, they have a restriction of the cervical spine in right rotation. The normal rotational range of the cervical spine is 80 degrees, but let's say the patient can only rotate 70 degrees to the right. This is where METs come in. After an MET has been employed on the tight restrictive muscles, hopefully the cervical spine will then be capable of rotating to 80 degrees—the patient has made all the effort and you, the practitioner, have encouraged the cervical spine into further right rotation. You have now improved the joint range to "normal." This is not stretching in the strictest sense—even though the overall flexibility has been improved, it is only to the point of achieving what is considered to be a normal joint range.

Depending on the context and the type of MET employed, the objectives of this treatment can include:

- Restoring normal tone in hypertonic muscles
- Strengthening weak muscles
- Preparing muscles for subsequent stretching
- Increasing joint mobility

Restoring Normal Tone in Hypertonic Muscles

Through the simple process of METs, we as physical therapists try to achieve a relaxation in the hypertonic shortened muscles. If we think of a joint as being limited in its ROM, then through the initial identification of the hypertonic structures, we can employ the techniques to help achieve normality in the tissues. Certain types of massage therapy can also help us achieve this relaxation effect, and generally an MET is applied in conjunction

with massage therapy. I personally feel that massage with motion is one of the best tools a physical therapist can use.

Strengthening Weak Muscles

METs can be used in the strengthening of weak or even flaccid muscles, as the patient is asked to contract the muscles prior to the lengthening process. The therapist should be able to modify the MET by asking the patient to contract the muscle that has been classified as *weak*, against a resistance applied by the therapist (isometric contraction), the timing of which can be varied. For example, the patient can be asked to resist the movement using approximately 20–30% of their maximum capability for 5–15 seconds. They are then asked to repeat the process five to eight times, resting for 10–15 seconds between repetitions. The patient's performance can be noted and improved over time.

Preparing Muscles for Subsequent Stretching

In certain circumstances, what sport your patient participates in will be determined by what ROM they have at their joints. Everybody can improve their flexibility, and METs can be used to help achieve this goal. Remember, the focus of METs is to try to improve the normal ROM of a joint.

If you want to improve the patient's flexibility past the point of normal, a more aggressive MET approach might be necessary. This could be in the form of asking the patient to contract a bit harder than the standard 10–20% of the muscle's capability. For example, we can ask the patient to contract using, say, 40–70% of the muscle's capability. This increased contraction will help stimulate more motor units to fire, in turn causing an increased stimulation of the Golgi tendon organ (GTO). This will then have the effect of relaxing more of the muscle, allowing it to be lengthened even further. Either way, once an MET has been incorporated into the treatment plan, a flexibility program can follow.

Increasing Joint Mobility

One of my favorite sayings when I teach muscle-testing courses is:

"A stiff joint can cause a tight muscle, and a tight muscle can cause a stiff joint."

Does this not make perfect sense? When you use an MET correctly, it is one of the best ways to improve the mobility of the joint, even though you are initially relaxing the muscles. This is especially the case with the use of METs to correct any dysfunctions that you find in the pelvis, which is covered in Chapter 13. The focus of the MET is to get the patient to contract the muscles; this subsequently causes a relaxation period, allowing a greater ROM to be achieved within that specific joint.

Physiological Effects of METs

There are two main effects of METs and these are explained on the basis of two distinct physiological processes:

- Post-isometric relaxation (PIR)
- Reciprocal inhibition (RI)

When we use METs, certain neurological influences occur. Before we discuss the main process of PIR/RI, we need to consider the two types of receptor involved in the stretch reflex:

- Muscle spindles, which are sensitive to change, as well as speed of change, in length of muscle fibers
- GTOs, which detect prolonged change in tension

Stretching the muscle causes an increase in the impulses transmitted from the muscle spindle to the posterior horn cell (PHC) of the spinal cord. In turn, the anterior horn cell (AHC) transmits an increase in motor impulses to the muscle fibers, creating a protective tension to resist the stretch. However, increased tension after a few seconds is sensed within the GTOs, which transmit impulses to the PHC. These impulses have an inhibitory effect on the increased

motor stimulus at the AHC; this inhibitory effect causes a reduction in motor impulses and consequent relaxation. This implies that the prolonged stretch of the muscles will increase the stretching capability, because the protective relaxation of the GTOs overrides the protective contraction due to the muscle spindles. A fast stretch of the muscle spindles, however, will cause immediate muscle contraction, and since it is not sustained, there will be no inhibitory action (Figure 7.1). This is known as the *basic reflex arc*.

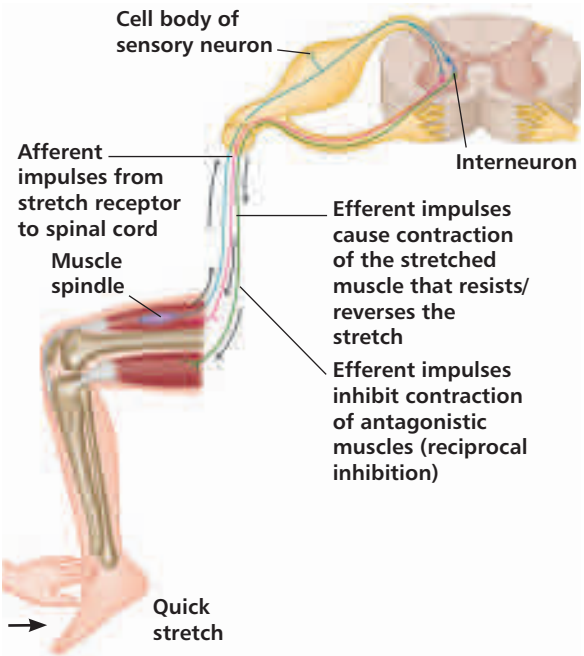


Figure 7.1. The stretch reflex arc. A quick "stretch by hand" to activate the muscle spindles.

PIR results from a neurological feedback through the spinal cord to the muscle itself when an isometric contraction is sustained, causing a reduction in tone of the muscle which has been contracted (Figure 7.2). This reduction in tone lasts for approximately 20–25 seconds, so you now have a perfect window of opportunity to improve the ROM, as during this relaxation period the tissues can be more easily moved to a new resting length.

When **RI** is employed, the reduction in tone relies on the physiological inhibiting effect of antagonists on the contraction of a muscle (Figure 7.2). When the motor neurons of the contracting agonist muscle receive excitatory impulses from the afferent pathway, the motor neurons of the opposing antagonist muscle receive inhibitory impulses at the same time, which prevent it contracting. It follows that contraction or extended stretch of the agonist muscle must elicit relaxation or inhibit the antagonist; however, a fast stretch of the agonist will facilitate a contraction of the agonist.

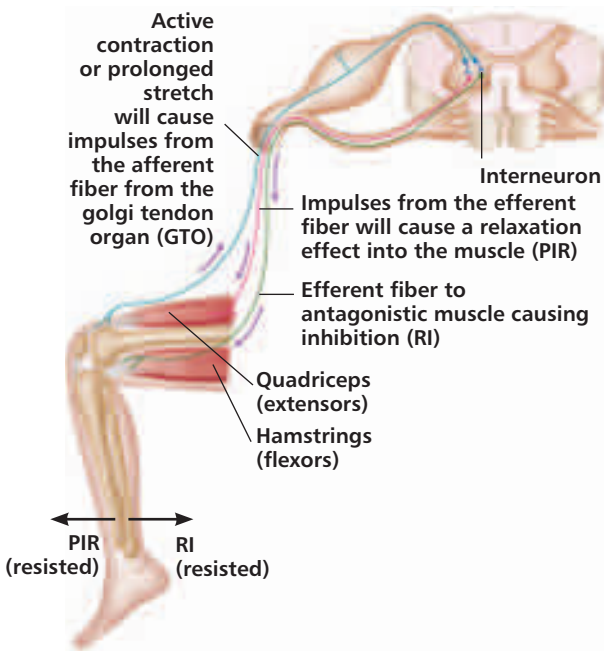


Figure 7.2. Post-isometric relaxation (PIR) and reciprocal inhibition (RI).

In most applications of METs, the point of bind, or just short of the point of bind, is the preferred position in which to perform an MET. Clearly, an MET is a fairly mild form of stretching compared with other techniques, so one can assume its use is therefore more appropriate in the rehabilitation process. It should be borne in mind that most problems with muscle shortening will occur in postural muscles. Since these muscles are composed predominantly of slow-twitch fibers, a milder form of stretching is perhaps more appropriate.

MET Procedure

- The patient's limb is taken to the point where resistance is felt, i.e. the point of bind. It can be more comfortable for the patient if you ease off to a point slightly short of the point of bind in the affected area that you are going to treat, especially if these tissues are in the chronic stage.

- The patient is asked to isometrically contract the muscle to be treated (PIR) or the antagonist (RI), using approximately 10–20% of the muscle's strength capability against a resistance that is applied by the therapist. The patient should be using the agonist if the method of approach is PIR; this will release the tight, shortened structures directly. (See the PIR example below.)

- If the RI method of MET is used, the patient is asked to contract the antagonist isometrically; this will induce a relaxation effect in the opposite muscle group (agonist), which would still be classified as the *tight* and *shortened* structures. (See the RI example below.)

- The patient is asked to slowly introduce an isometric contraction, lasting between 10 and 12 seconds, avoiding any jerking of the treated area. This contraction, as explained above, is the time necessary to load the GTOs, which allows them to become active and to influence the intrafusal fibers from the muscle spindles. This has the effect of overriding the influence from the muscle spindles, which inhibits muscle tone. The therapist then has the opportunity to take the affected area to a new position with minimal effort.

- The contraction by the patient should cause no discomfort or strain. The patient is told to relax fully by taking a deep breath in, and as they breathe out, the therapist passively takes the specific joint that lengthens the hypertonic muscle to a new position, which therefore normalizes joint range.

- After an isometric contraction, which induces a PIR, there is a relaxation period of 15–30 seconds; this period can be the perfect time to stretch the tissues to their new resting length.

- Repeat this process until no further progress is made (normally three to four times) and hold the final resting position for approximately 25–30 seconds.

- A period of 25–30 seconds is considered to be enough time for the neurological system to lock onto this new resting position.

- This type of technique is excellent for relaxing and releasing tone in tight, shortened soft tissues.

A refractory period (the brief period needed to restore the resting potential) of about 20 seconds occurs with RI; however, RI is thought to be less powerful than PIR. Therapists need to be able to use both approaches, because the use of the agonist may sometimes be inappropriate owing to pain or injury. Since the amount of force used with an MET is minimal, the risk of injury or tissue damage will be reduced.

MET Method of Application

"Point of Bind" (or "Restriction Barrier")

In this chapter the word "bind" is mentioned many times. The *point of bind*, or *restriction barrier*, occurs when resistance is first felt by the palpating hand/fingers of the therapist. Through experience and continual practice, the therapist will be able to palpate a resistance of the soft tissues as the affected area is gently taken into the position of bind. This position of bind is *not* the position of stretch—it is the position just before the point of stretch. The therapist should be able to feel the difference and not wait for the patient to say when they feel a stretch has occurred.

Acute and Chronic Conditions

The soft tissue conditions that are treated using METs are generally classified as either *acute* or *chronic*, and this tends to relate to tissues that have had some form of strain or trauma. METs can be used for both acute and chronic conditions. *Acute* involves anything that is obviously acute in terms of symptoms, pain, or spasm, as well as anything that has emerged during the previous three to four weeks. Anything older and of a less obviously acute nature is regarded as *chronic* in determining which variation of MET is suitable.

If you feel the presenting condition is relatively acute (occurring within the last three weeks), the isometric contraction can be performed at the point of bind. After the patient has contracted the muscle isometrically for the duration of 10 seconds, the therapist then takes the affected area to the new point of bind.

In chronic conditions (persisting for more than three weeks), the isometric contraction starts from a position just before the point of bind. After the patient has contracted the muscle for 10 seconds, the therapist then goes through the point of bind and encourages the specific area into the new position.

PIR versus RI

How much pain the patient is presenting with is generally the deciding factor in determining which method to initially apply. The PIR method is usually the technique of choice for muscles that are classified as *short* and *tight*, as it is these muscles that are initially contracted in the process of releasing and relaxing.

On occasion, however, a patient may experience discomfort when the agonist, i.e. the shortened structure, is contracted; in this case it would seem more appropriate to contract the opposite muscle group (antagonist), as this would reduce the patient's perception of pain, but still induce a relaxation in the painful tissues. Hence, the use of the RI method, using the antagonists, which are usually pain free, will generally be the first choice if there is increased sensitivity in the primary shortened tissues.

When the patient's initial pain has been reduced by the appropriate treatment, PIR techniques can be incorporated (as explained earlier, PIR uses an isometric contraction of the tight shortened structures, in contrast to the antagonists being used in the RI method). To some extent, the main factor in deciding the best approach is whether the sensitive tissue is in the acute stage or in the chronic stage.

After having used PIR and RI on a regular basis, I have found that the best results for lengthening the hypertonic structures are achieved with PIR (provided the patient has no pain during this technique). However, once I have performed the PIR method, if I feel more ROM is needed in the shortened tight tissue, I bring into play the antagonists using the RI method for approximately two more repetitions, as explained in the RI example below. This personal approach for my patients has had the desired effect of improving the overall ROM.

PIR Example

To illustrate the PIR method of MET treatment, we are now going to apply the procedure to the adductor pollicis muscle (*pollicis* relates to the thumb, or pollex). You might consider it more appropriate to demonstrate how METs work by means of an example related to the pelvis; however, I wanted the therapist to be

able to practice the technique on themselves first, so that they can better understand the MET concept. Once the technique has been understood and subsequently practiced using this simple example, the therapist will then be ready to tackle more complex METs with the aim of helping to restore function to the pelvis.

Place your left (or right) hand onto a blank piece of paper and, with the hand open as much as possible, draw around the fingers and the thumb (Figure 7.3).

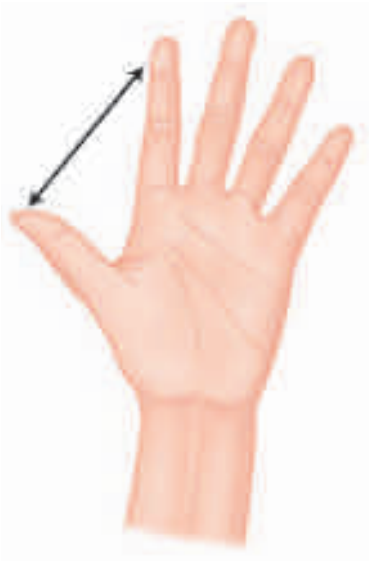


Figure 7.3. The distance between the thumb and finger is measured.

Remove the paper and actively abduct the thumb as far as you can, until a point of bind is felt. Next, place the fingers of your right hand on top of the left thumb and, using an isometric contraction, *adduct* your thumb against the downward pressure of the fingers, so that an isometric contraction is achieved (Figure 7.4). After applying this pressure for 10 seconds, breathe in, and on the exhalation passively take the thumb into further *abduction* (but do not force the thumb). Repeat this sequence two more times and on the last repetition, maintain the final resting position for at least 20–25 seconds.

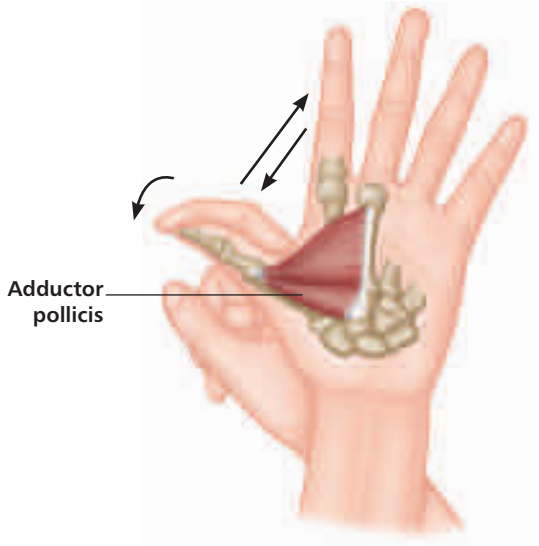


Figure 7.4. Adducting the thumb against a resistance applied by the opposite hand (PIR method).

Now place your hand back on the piece of paper and draw around it again (Figure 7.5); hopefully you will see that the thumb has abducted further than before.

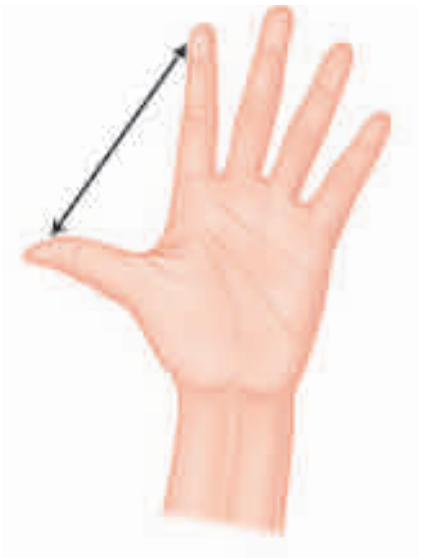


Figure 7.5. The hand redrawn after the MET treatment using PIR and RI.

RI Example

To apply the RI method, follow the same procedure as for the PIR method, i.e. go to the point of bind by still abducting the thumb. From this position of bind, instead of *adducting* the thumb (PIR) against a resistance, perform the opposite movement and *abduct* your thumb (using the abductor pollicis brevis/longus muscle) against a resistance (Figure 7.6). After applying this pressure for 10 seconds, breathe in, and on the exhalation passively take the thumb into further *abduction* (again, do not force the thumb). Repeat this sequence one or two more times and on the last repetition, maintain the final resting position for at least 20–25 seconds.

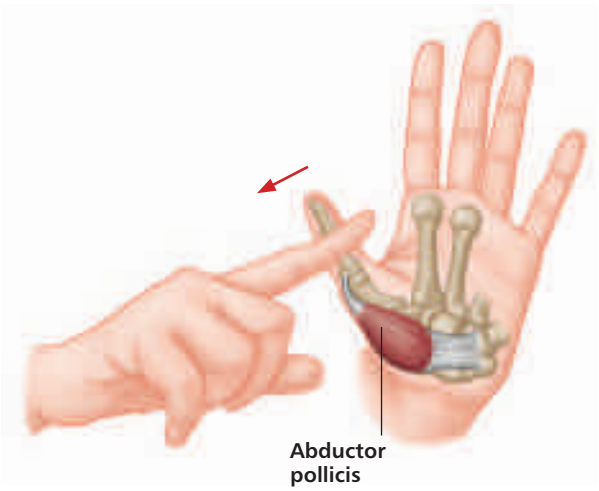


Figure 7.6. Abducting the thumb against a resistance applied by the opposite hand (RI method).

As before, place your hand back on the piece of paper and draw around it again (Figure 7.5); hopefully you will see that the thumb has abducted further than previously.

METs and the Muscles of the Pelvis

The muscles that I consider to be specifically related to the position of the pelvis and lumbar spine are:

- Iliopsoas
- Rectus femoris
- Adductors
- Hamstrings
- Tensor fasciae latae (TFL) and iliotibial band (ITB)
- Piriformis
- Quadratus lumborum (QL)

I am going to show you how to assess for each of these muscles, which have a natural tendency to shorten and subsequently become tight. After the testing procedure has been explained, I will demonstrate specific METs to encourage these short/tight muscles to lengthen, so that we can assist and hopefully normalize the dysfunctional position.

Iliopsoas

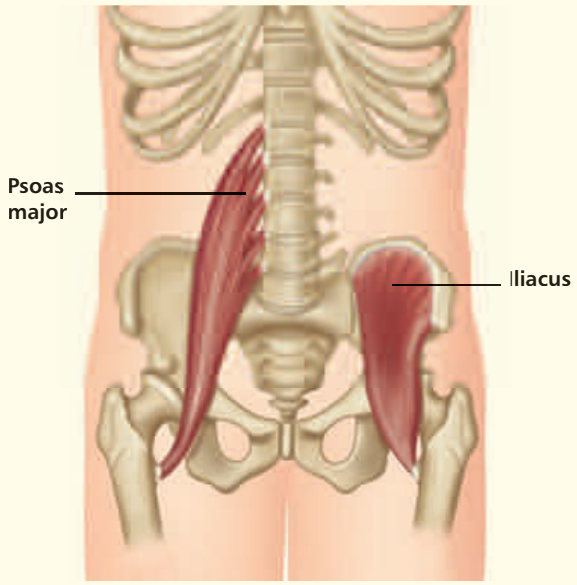


Figure 7.7. (a) Origin, insertion, action, and nerve innervation of the iliopsoas.

Origin

Psoas major: TPs of all the lumbar vertebrae (L1–L5). Bodies of the 12th thoracic and all lumbar vertebrae (T12–L5). Intervertebral discs above each lumbar vertebra.

Iliacus: Superior two-thirds of the iliac fossa. Anterior ligaments of the lumbosacral and SIJs.

Insertion

Lesser trochanter of the femur.

Action

Main flexor of the hip joint. Assists in lateral rotation of the hip. Acting from its insertion, it flexes the trunk, as in sitting up from the supine position.

Nerve

Psoas major: Ventral rami of lumbar nerves (L1–L4).

Iliacus: Femoral nerve (L2–L4).

Assessment of the Iliopsoas

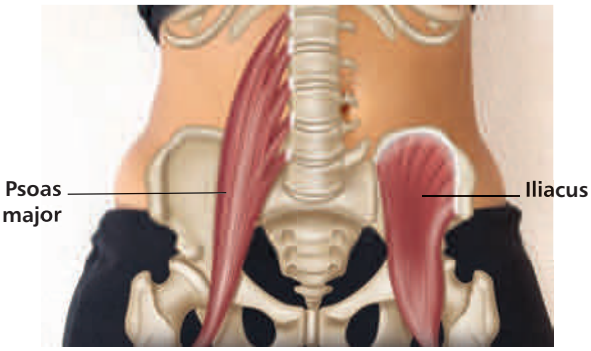


Figure 7.7. (b) The psoas major and iliacus superimposed on the abdomen.

It is important that we are able to assess for relative shortness within the hip flexor group, as Grieve (1983) says that an increased tension in the iliopsoas muscle is felt to be one of the main reasons for the recurrence of malalignment syndrome, even after the alignment of the pelvis has been corrected.

Schamberger (2013) points out that it is common to find more tension/tenderness in the left iliopsoas than in the right side. One reason for this might be that the left SIJ may actually become hypermobile as a result of the increased stress imposed if the right SIJ becomes "locked" (as a result of a right anterior innominate with a compensatory left rotated posterior innominate) or becomes hypomobile for other reasons. The left iliopsoas would contract in an attempt to stabilize the now hypermobile left SIJ.

Modified Thomas Test

To test the right hip, the patient is asked to lie back on the edge of a couch while holding onto their left knee. As they roll backward, the patient pulls their left knee as far as they can toward their chest, as shown in Figure 7.7(c). The full flexion of the hip encourages full posterior rotation of the innominate bone and helps to flatten the lordosis. From this position, the therapist looks at where the patient's right knee lies, relative to the right hip. The position of the knee should be just below the level of the hip; Figure 7.7(c) demonstrates a normal length of the right iliopsoas.

Figure 7.7. (c) The right knee is below the level of the hip, indicating a normal length of the iliopsoas.

In Figure 7.8 the therapist demonstrates with their arms the position of the right hip compared with the right knee. You can see that the hip is held in a flexed position, which confirms the tightness of the right iliopsoas in this case.

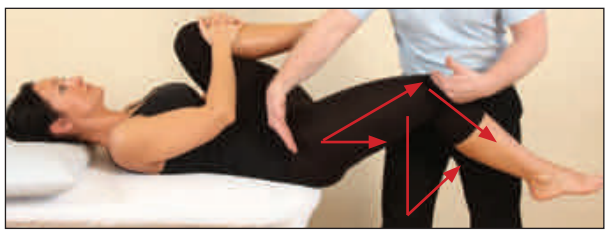


Figure 7.8. A tight right iliopsoas is confirmed. A tight rectus femoris can also be seen here.

With the patient in the modified Thomas test position, the therapist can apply an abduction of the hip (Figure 7.9), and an adduction of the hip (Figure 7.10). A ROM of 10–15 degrees for each of these is commonly accepted to be normal.

If the hip is restricted in abduction, i.e. a bind occurs at an angle of less than 10–15 degrees, the muscles of the adductor group are held in a shortened position; if the adduction movement is restricted, the TFL and the ITB are held in a shortened position.

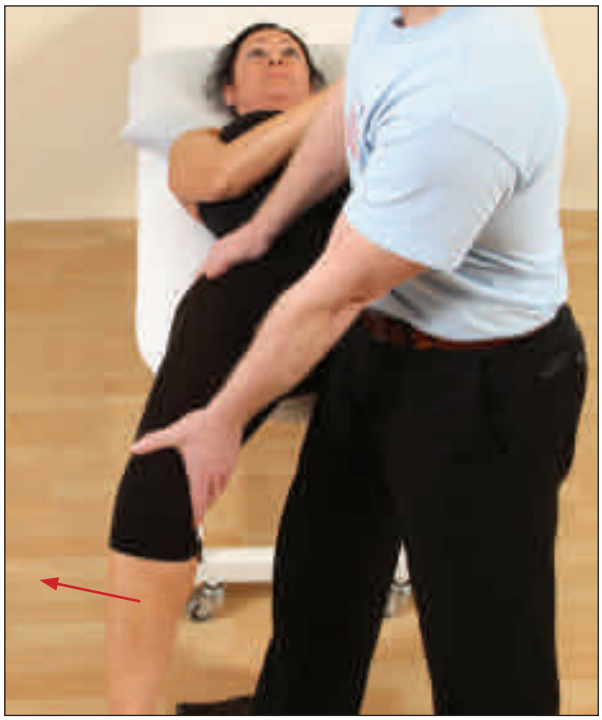


Figure 7.9. Restricted hip abduction, indicating tight adductors.

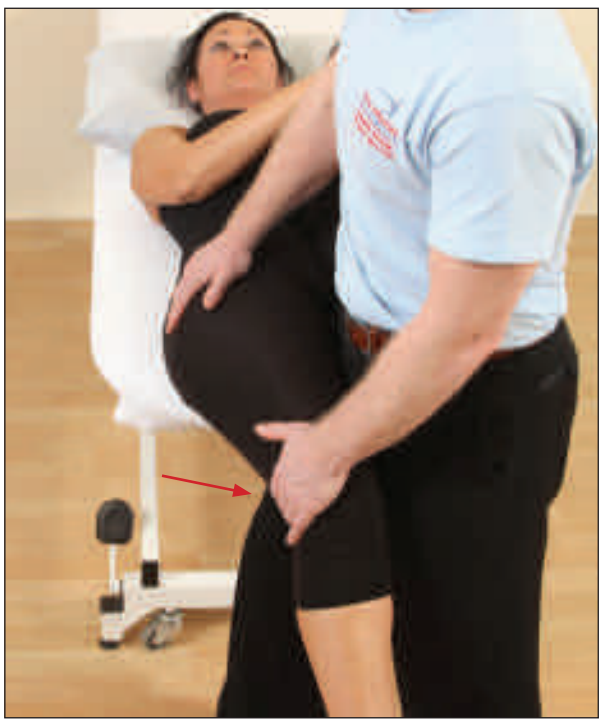


Figure 7.10. Restricted hip adduction, indicating a tight TFL/ITB.

MET Treatment of the Iliopsoas

To treat the right side, the patient adopts the same position as in the modified Thomas test above. The patient's left foot is placed into the therapist's right side and pressure is applied by the therapist to induce full flexion of the patient's left hip. Stabilizing the patient's right hip with their right hand, the therapist puts their left hand just above the patient's right knee. The patient is asked to flex their right hip against the therapist's resistance for 10 seconds, as shown in Figure 7.11; this specific contraction of the iliopsoas muscle will induce a PIR.

Figure 7.11. The patient flexes their right hip against resistance from the therapist's left hand, while the hip is being stabilized by the therapist's right hand.

Following the isometric contraction, and during the relaxation phase, the therapist slowly applies a downward pressure. This will cause the hip to passively go into extension and will induce a lengthening of the right iliopsoas, as shown in Figure 7.12. Gravity will also play a part in this technique, by assisting in the lengthening of the iliopsoas.

Figure 7.12. The therapist passively extends the hip to lengthen the iliopsoas, with the assistance of gravity.

Alternatively, it is possible to contract the iliopsoas from the flexed position, as shown in Figure 7.13. This is normally used if the original method of activating the iliopsoas

causes discomfort to the patient. Allowing the hip to be in a more flexed position will slacken the iliopsoas, which will assist in its contraction and help reduce the discomfort.

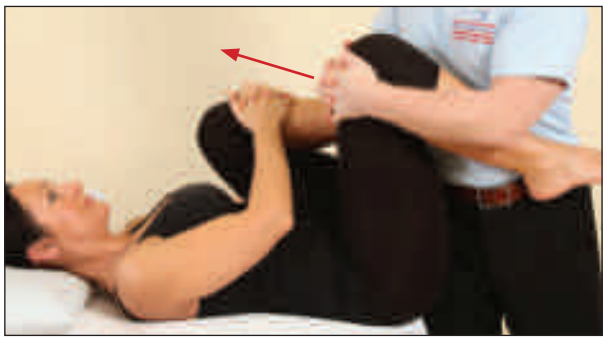


Figure 7.13. The patient resists hip flexion from a flexed position.

The patient is asked to flex their right hip against a resistance applied by the therapist's left hand (Figure 7.13). After a 10-second contraction, and during the relaxation phase, the therapist lengthens the iliopsoas by taking the hip into an extended position, as demonstrated in Figure 7.14(a).

Figure 7.14. (a) Lengthening of the right iliopsoas.

Tip: The psoas major is also known as filet mignon, which is a piece of beef taken from the tenderloin. A bilateral shortness of the psoas major can cause the pelvis to anteriorly tilt, in turn causing the lumbar spine to adopt a position of hyperlordosis and drawing the sacrum into a position of increased nutation. This can result in compression of the facet joints, leading to lower back pain.

Note: If full sit-ups are performed on a regular basis (not recommended), the iliopsoas is predominantly the muscle being used. Repeated sit-ups will make the iliopsoas stronger and tighter, and result in weakness of the abdominals; this can maintain a patient's lower back pain.

To prove the involvement of the iliopsoas (during the sit-up motion), have your patient lie on their back with their knees bent. Hold the patient's ankles and ask them to dorsiflex their ankles while you resist the movement. This will stimulate the anterior chain musculature, including the iliopsoas, which is part of this chain. The patient then performs the full sit-up movement as shown in Figure 7.14(b) (most fit individuals will be able to do many sit-ups).

Figure 7.14. (b) Dorsiflexion assists the activation for the contraction of the iliopsoas (switch on).

To deactivate or switch off the iliopsoas, the patient is asked to plantar flex their ankles (instead of dorsiflexing them), or to squeeze their glutes. Either of these actions stimulates the posterior chain musculature, causing the iliopsoas to switch off, as activation of the gluteal muscles results in a relaxation of the iliopsoas through RI. When the patient is now asked to perform the sit-up, the movement will prove to be impossible, confirming that the iliopsoas is generally the prime mover in a full sit-up, as shown in Figure 7.14(c).

Figure 7.14. (c) Plantar flexion or activating the glutes assists the deactivation for the contraction of the iliopsoas (switch off).

Rectus Femoris

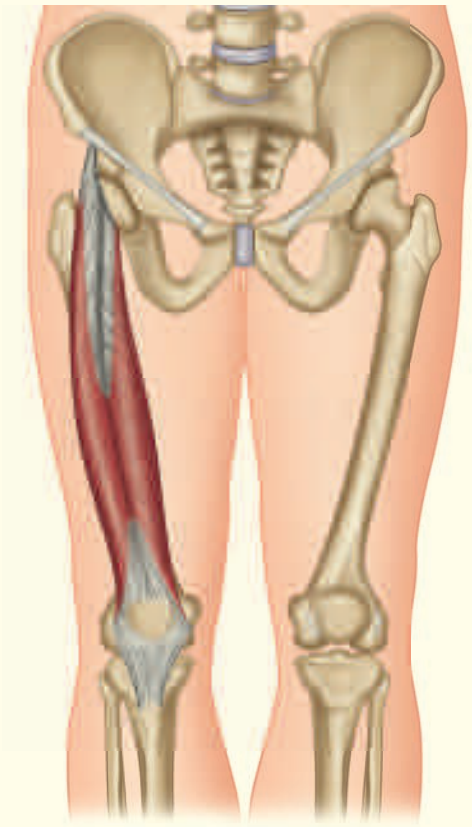


Figure 7.15. Origin, insertion, action, and nerve innervation of the rectus femoris.

Origin

Straight head (anterior head): AIIS.

Reflected head (posterior head): Groove above the acetabulum (on the ilium).

Insertion

Patella, then via the patellar ligament to the tuberosity of the tibia.

Action

Extends the knee joint and flexes the hip joint (particularly in combination movements, such as in kicking a ball). Assists iliopsoas in flexing the trunk on the thigh. Prevents flexion at the knee joint as the heel strikes the ground during walking.

Nerve

Femoral nerve (L2–L4).

Assessment of the Rectus Femoris

Modified Thomas Test

This test is an excellent way of identifying shortness not only in the rectus femoris but also in the iliopsoas as explained earlier. To test the right rectus femoris, the patient adopts the position in Figure 7.16, in which they are holding onto their left leg. The patient is asked to pull their left knee toward their chest, as this will posteriorly rotate the innominate bone on that side; this will be the test position. From this position, the therapist looks at the position of the patient's right knee and right ankle. The angular position of the knee to the ankle should be about 90 degrees; a normal length of the right rectus femoris is shown below.

Figure 7.16. To test the right rectus femoris, the patient lies on the couch and holds onto their left leg. A normal length of the rectus femoris is shown.

In Figure 7.17 the therapist demonstrates the position of the right knee compared with the right ankle. Here, the lower leg is seen to be held in a position of extension, which confirms the tightness of the right rectus femoris. You will also notice the position of the hip—it is held in a flexed position. This indicates a tightness of the iliopsoas and was discussed earlier.

Figure 7.17. The knee is held in extension, indicating a tight rectus femoris.

MET Treatment of the Rectus Femoris

The patient is asked to adopt a prone position, and the therapist passively flexes the patient's right knee until a bind is felt. At the same time, the therapist stabilizes the sacrum with their right hand, which will prevent the pelvis from rotating anteriorly and stressing the lower lumbar spine facet joints.

> **Note:** If you consider the patient to have an increased lumbar lordosis, a pillow can be placed under their stomach. This will help flatten the lordosis and can reduce any potential discomfort.

From the position of bind, the patient is asked to extend their knee against a resistance applied by the therapist (Figure 7.18), as this contraction will induce a PIR in the rectus femoris muscle.

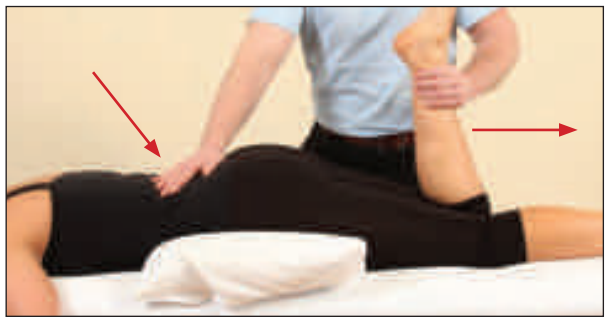


Figure 7.18. The patient extends their knee while the therapist applies resistance.

After a 10-second contraction, and during the relaxation phase, the therapist encourages the knee into further flexion, which will lengthen the rectus femoris, as shown in Figure 7.19.

Figure 7.19. The therapist passively flexes the patient's knee to lengthen the rectus femoris while stabilizing the lumbar spine.

Alternative MET Treatment of the Rectus Femoris

Some patients may find that the previous MET for the rectus femoris puts a strain on their lower back. An alternative and possibly a more effective MET for the rectus femoris is based on the modified Thomas test position.

The patient adopts the position of the modified Thomas test as described earlier. The therapist controls the position of the patient's right thigh, and slowly and passively flexes the patient's right knee toward their bottom. A bind will be reached very quickly in this position, so take extra care when performing this technique for the first time.

From the position of bind, the patient is asked to extend their knee against a resistance applied by the therapist (Figure 7.20). After the 10-second contraction, and during the relaxation phase, the therapist passively takes the knee into further flexion (Figure 7.21). This is a very effective way to lengthen a tight rectus femoris.

> **Tip:** Bilateral hypertonicity of the rectus femoris will cause the pelvis to adopt an anterior tilt, resulting in lower back pain due to the 5th lumbar vertebra facet joints being forced into a lordotic (extended) position.
>
> If one side (typically the right side) of the rectus femoris muscle is held in a shortened position, this will have the effect of pulling the innominate bone into an anteriorly rotated position, with relative counter-nutation of the sacrum to the same side.

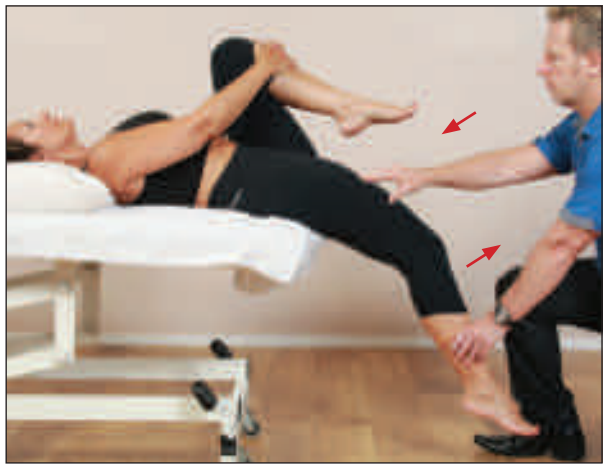


Figure 7.20. The therapist palpates the rectus femoris, and the patient extends their knee against a resistance.

Figure 7.21. The therapist passively flexes the patient's knee to lengthen the rectus femoris.

Adductors

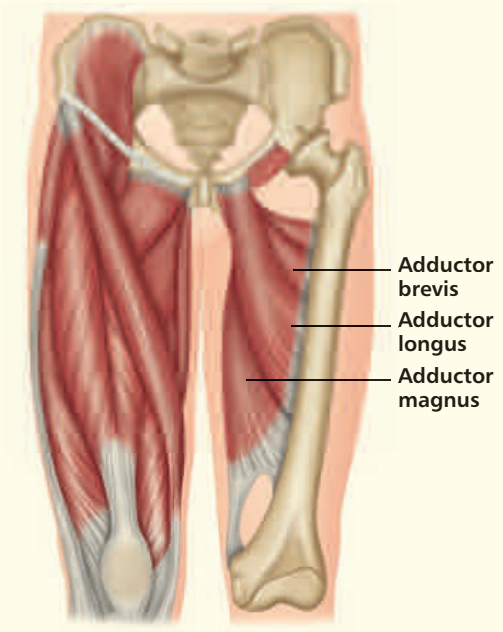


Figure 7.22. Origin, insertion, action, and nerve innervation of the adductors.

Origin

Anterior part of the pubic bone (ramus). Adductor magnus also has its origin on the ischial tuberosity.

Insertion

Entire length of the medial side of the femur, from the hip to the knee.

Action

Adduct, flex, and medially rotate the hip joint.

Nerve

Magnus: Obturator nerve (L2–L4). Sciatic nerve (L4, L5, S1).

Brevis: Obturator nerve (L2–L4).

Longus: Obturator nerve (L2–L4).

Assessment of the Adductors

Hip Abduction Test

To test the left side, the patient adopts a supine position on the couch. The therapist takes hold of the patient's left leg and passively abducts the hip while palpating the adductors with their right hand (Figure 7.23). When they feel a bind, the position is noted. The normal ROM for passive abduction is 45 degrees; if the range is less than this, a tight left adductor group is indicated.

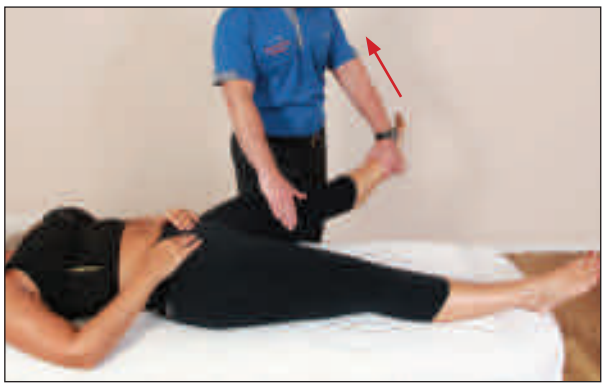


Figure 7.23. The therapist abducts and palpates the adductors for bind.

However, there is an exception to the rule. If the ROM is less than 45 degrees, it could be that the medial hamstrings are restricting the movement of passive abduction. To differentiate between the short adductors and the medial hamstrings, the knee is flexed to 90 degrees (Figure 7.24); if the range now increases, this indicates shortness in the medial hamstrings.

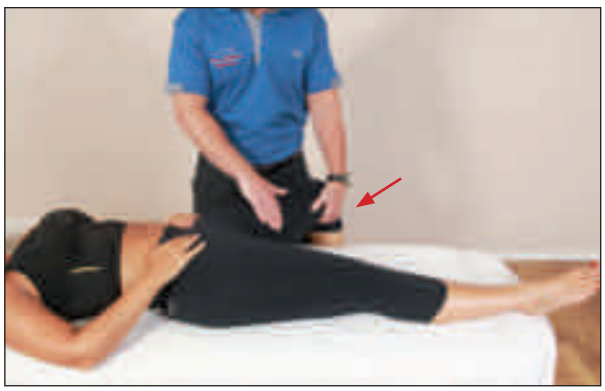


Figure 7.24. The knee is bent to isolate the short adductors.

In summary, to identify if the hamstrings are the restrictive factor, the therapist passively flexes the knee and then continues with the passive abduction. If the ROM improves, the hamstrings are the restrictive tissues and not the short adductors.

> **Note:** The term short adductor refers to all of the adductor muscles that attach to the femur, the exception being the gracilis. This particular muscle attaches to a point below the knee, on the pes anserinus (goose foot) area of the medial knee, and acts on the knee as well as on the hip.

MET Treatment of the Adductors

One of the most effective ways of lengthening the adductors (short) is to utilize an MET from the position, shown in Figure 7.25(a–b). The patient adopts a supine position with their knees bent and heels together; the hips are slowly and passively taken into abduction by the therapist until a bind is felt in the adductors.

From the position of bind, the patient is asked to adduct their hips against a resistance applied by the therapist, to contract the short adductors.

Figure 7.25. (a) The patient adducts their legs against resistance from the therapist.

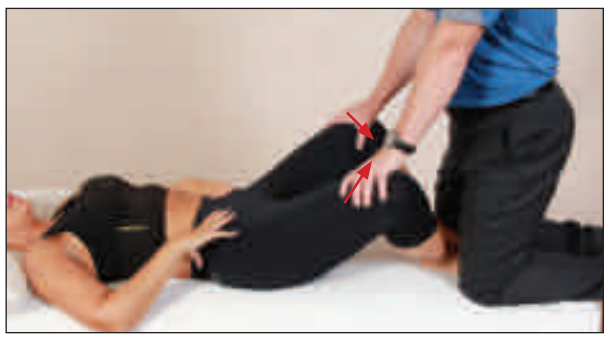


Figure 7.25. (b) Alternative position—the therapist kneels on the couch as the patient adducts their legs against resistance from the therapist.

After a 10-second contraction, and during the relaxation phase, the hips are passively taken into further abduction by the therapist, as shown in Figure 7.26(a–b).

Figure 7.26. (a) The therapist lengthens the adductors.

Figure 7.26. (b) Alternative position—the therapist lengthens the adductors.

Tip: Overactivity of the adductors will potentially result in a weakness inhibition of the abductor muscles, in particular the Gmed. This can result in a Trendelenburg pattern of gait, as explained in Chapter 5.

Hamstrings

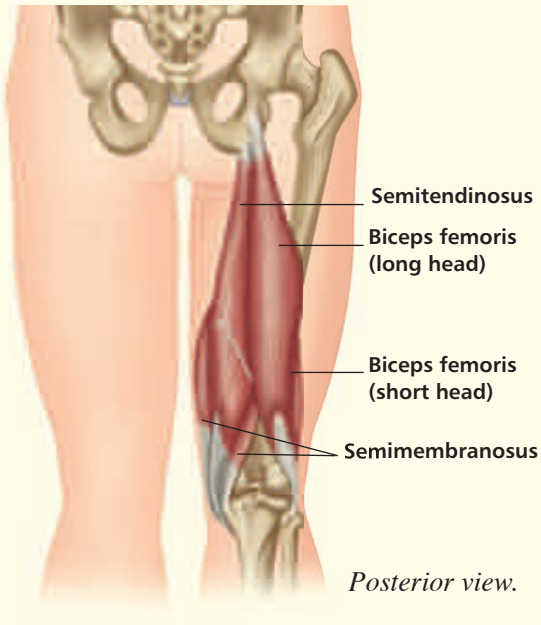


Figure 7.27. Origin, insertion, action, and nerve innervation of the hamstrings.

Origin

Ischial tuberosity (sitting bone). Biceps femoris also originates from the back of the femur.

Insertion

Semimembranosus: Back of the medial condyle of the tibia (upper inside part of tibia).

Semitendinosus: Upper medial surface of the shaft of the tibia.

Biceps femoris: Head (top) of the fibula. Lateral condyle of the tibia (upper outside part of tibia).

Action

Flex the knee joint. Extend the hip joint. Semimembranosus and semitendinosus also medially rotate (turn in) the lower leg when the knee is flexed. Biceps femoris laterally rotates (turns out) the lower leg when the knee is flexed.

Nerve

Branches of the sciatic nerve (L4, L5, S1, S2, S3).

General Assessment of the Hamstrings

Hip Flexion Test

This test helps to provide the practitioner with an overall impression of the general length of the hamstring muscles. The patient lies in a supine position with both legs extended. The therapist passively guides the patient's left hip into flexion until a point of bind is felt. The normal ROM is anywhere between 80 and 90 degrees; less than 80 degrees indicates that the hamstrings are held in a shortened position. However, "neural tension" of the sciatic nerve and a specific hamstring injury can also restrict the ROM (flexion) of the hip joint.

As you can see in Figure 7.28(a), the patient has a normal ROM in their hamstrings. Anything less than 80–90 degrees would be classified as *short*, as shown in Figure 7.28(b).

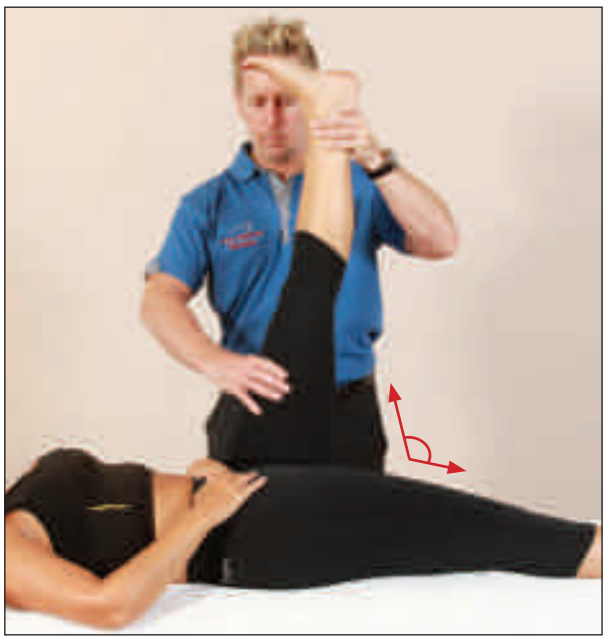


Figure 7.28. (a) Hip flexion test. A ROM of 80–90 degrees is normal.

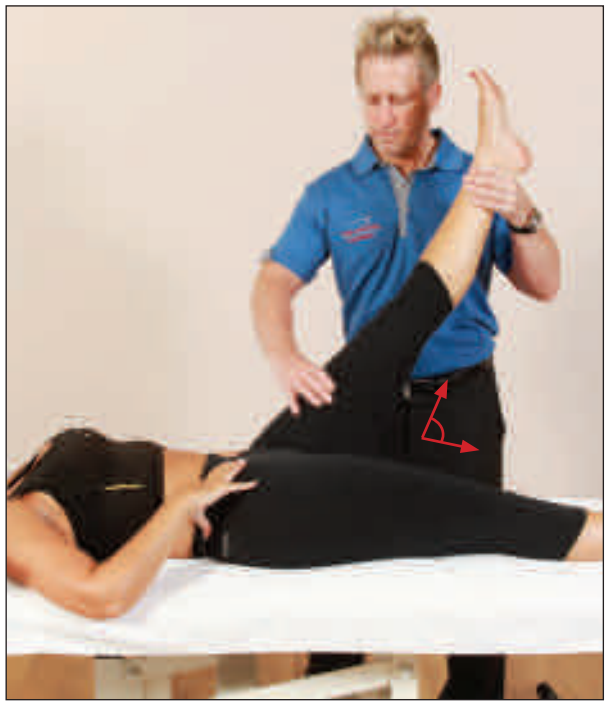


Figure 7.28. (b) Hip flexion test. A ROM of 45 degrees is demonstrated here, indicating short hamstrings.

MET Treatment of the Hamstrings (Non-Specific)

The following technique is very good for lengthening the hamstrings as a group; later in this chapter we will see how to specifically target the medial and lateral hamstrings.

The therapist adopts a standing posture and passively controls the patient's right leg into hip flexion until a bind is felt in the hamstrings. From this position, the patient's lower leg is placed on the therapist's right shoulder, as shown in Figure 7.29.

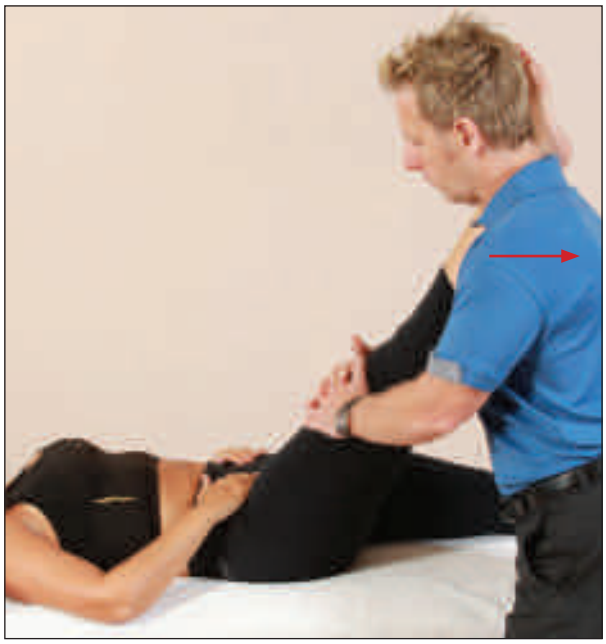


Figure 7.29. The patient pushes their right leg down against the therapist's shoulder.

The patient is asked to push down against the therapist's shoulder for 10 seconds. After the contraction of the hamstrings, and during the relaxation phase, the therapist passively takes the right leg into further flexion, as seen in Figure 7.30.

Figure 7.30. The therapist passively takes the hip into further flexion.

Alternative MET for the Insertion of the Hamstrings

This technique is very good for lengthening the insertion component of the hamstrings. The patient's hip is now flexed to 90 degrees and the lower leg is placed over the shoulder of the therapist, as shown in Figure 7.31.

Figure 7.31. With the hip flexed to 90 degrees, the patient places their lower leg over the therapist's shoulder.

From this position, the patient is asked to pull their heel toward their gluteal muscles, as this will activate the contraction of the hamstrings. After a 10-second contraction, and during the relaxation phase, the therapist passively encourages knee extension until a new point of bind is felt, as shown in Figure 7.32.

Figure 7.32. The therapist passively encourages knee extension to lengthen the hamstrings.

RI Method

The patient is asked to contact the hamstrings as described above; however, after the 10-second contraction, and during the relaxation phase, the patient is asked to slowly straighten their knee (which was flexed to start with) as the therapist passively takes the knee into further extension. The patient will be contracting their quadriceps as they straighten the knee actively; this will induce an RI effect in the hamstrings, allowing a more effective and safe lengthening to occur.

Assessment of the Medial Hamstrings (Semitendinosus and Semimembranosus)

After conducting a general assessment of the hamstrings, if the ROM is less than 80 degrees, we can conclude that there is a soft tissue restriction present within the hamstring muscle group. However, the assessment does not tell us which aspect of the hamstrings is the tighter structure.

With specific testing it is possible to identify the individual components of the hamstring muscles that are responsible. The following tests can be incorporated into the assessment to help differentiate muscle length anomalies in the medial hamstrings from those in the lateral hamstrings.

In order to investigate whether the semitendinosus/semimembranosus is the restrictive tissue, the medial hamstrings are isolated as follows. The patient's leg is controlled by the therapist, who applies an external rotation and abduction, while at the same time the hip is passively flexed (Figure 7.33(a–b)). The point of bind is noted; if the ROM is less than that in the original test, the medial hamstrings can be assumed to be the shortened muscles.

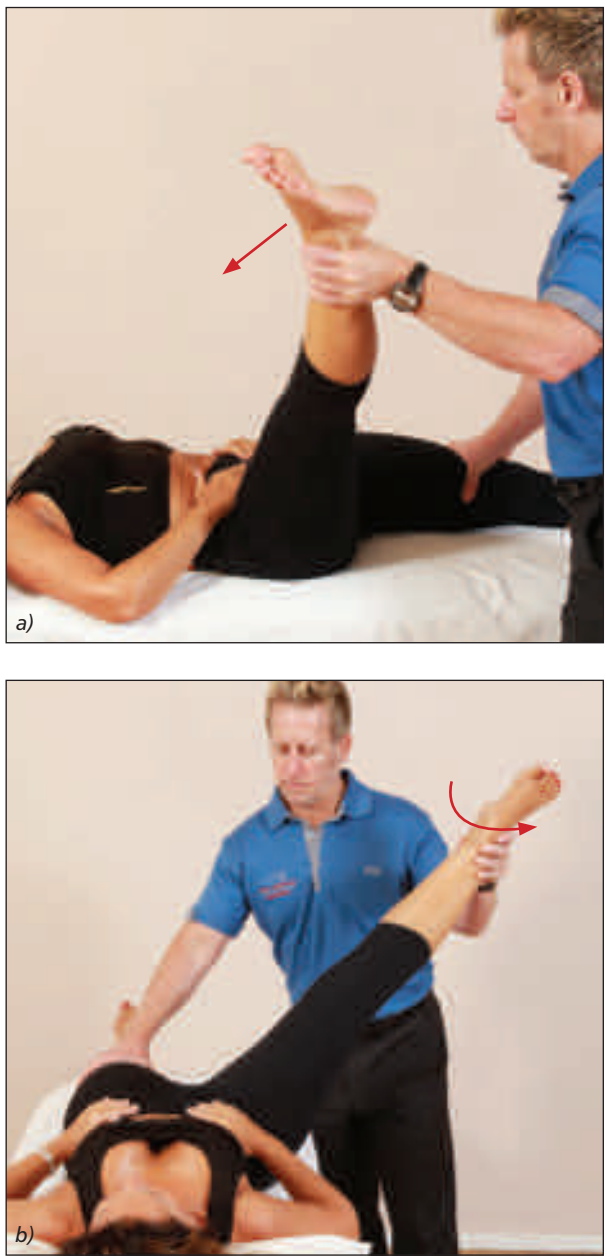


Figure 7.33. (a) To specifically identify the medial hamstrings as the restrictive tissue, the patient's leg is externally rotated and abducted while the hip is passively flexed. (b) Alternative view of the test position.

Assessment of the Lateral Hamstrings (Biceps Femoris)

This specific test will isolate the biceps femoris. The therapist applies an internal rotation and adduction, while the patient's leg is taken into passive flexion (Figure 7.34(a–b)). If the motion feels restrictive, the therapist needs to determine whether the ROM is less than that in the original hip flexion test; if it is, the lateral hamstring of the biceps femoris can be identified as the short tissue.

Tip: Remember, the medial and lateral hamstrings might need treating individually rather than together.

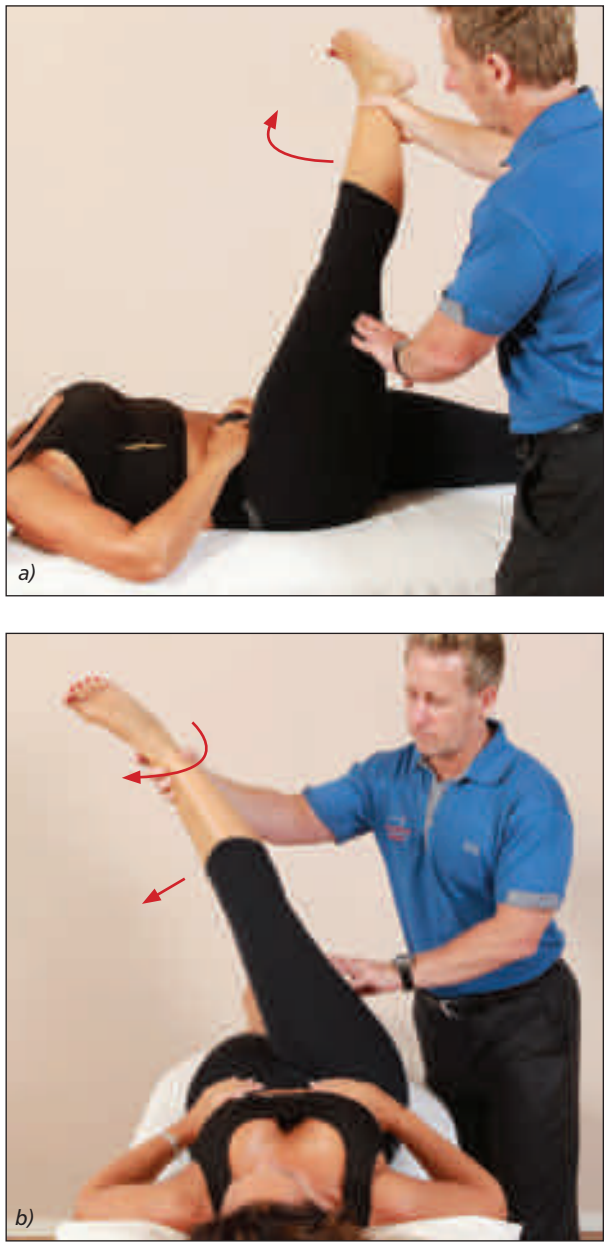


Figure 7.34. (a) To test the biceps femoris, the therapist applies an internal rotation and adduction while the leg is taken into passive flexion. (b) Alternative view of the test position.

Tensor Fasciae Latae (TFL) and Iliotibial Band (ITB)

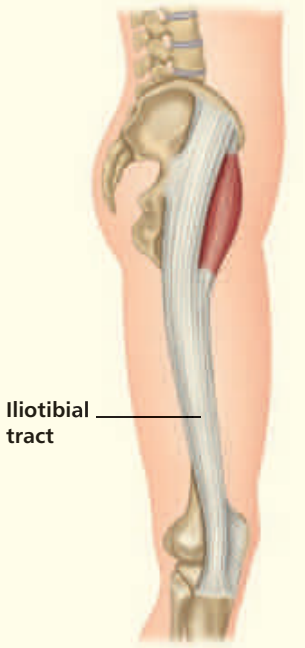


Figure 7.35: Origin, insertion, action, and nerve innervation of the TFL/ITB.

Origin

Outer edge of the iliac crest, toward the front.

Insertion

Joins the iliotibial tract (long fascia lata tendon) just below the hip, which runs to the upper lateral side of the tibia (Gerdy's tubercle).

Action

Flexes, abducts, and medially rotates the hip joint. Tenses the fascia lata, thus stabilizing the knee.

Nerve

Superior gluteal nerve (L4, L5, S1).

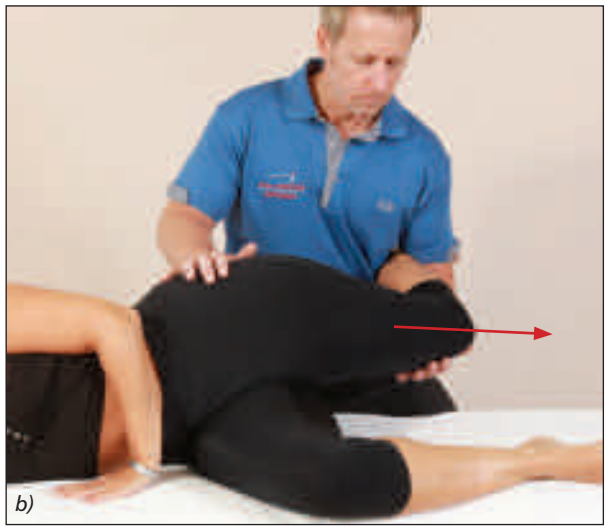
Assessment of the TFL and ITB

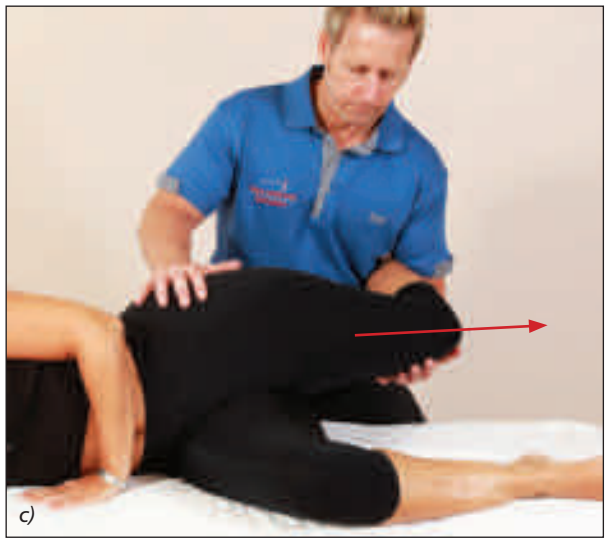
Ober's Test

An orthopedic surgeon by the name of Frank Ober wrote an article called "Back strain and sciatica" (Ober 1935a) and first described this test in 1937. He discussed the relationship of a contracted TFL muscle and the ITB to lower back pain and sciatica.

The patient is asked to adopt a side-lying position, and the therapist (with the assistance of the patient) places the patient's shoulder, hip, and knee in alignment as shown in Figure 7.36(a).

When the therapist feels that the patient has sufficiently relaxed, they slowly bend their own knees (as in a half squat) while maintaining control of the patient's left knee as it is lowered to the couch. If the knee is seen to drop below the level of parallel, the TFL/ITB is classified as *normal*, as shown in Figure 7.36(b); if the thigh remains (or drops only slightly below) parallel, the TFL/ITB is classified as *short*, as shown in Figure 7.36(c).





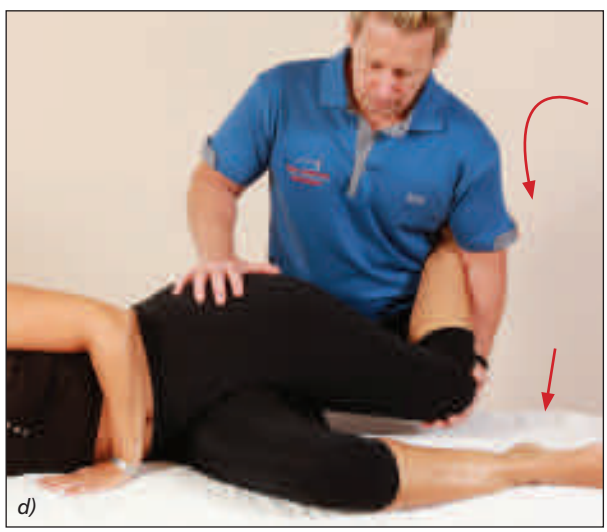


Figure 7.36. Ober's test: (a) The therapist controls the patient's left knee and asks the patient to relax fully before the knee is lowered toward the couch; (b) a normal length of the TFL/ITB, indicated by the knee dropping down; (c) a tight TFL/ITB, indicated by the knee remaining where it is; (d) allowing the hip to "fall" into hip flexion and internal rotation can lead to the incorrect conclusion of a normal ITB/TFL.

Note: If there is perceived shortness within the TFL and ITB, the leg will remain relatively abducted, but the hip will naturally want to "fall" into hip flexion with internal rotation as the therapist lowers the leg (see Figure 7.36(d)). If this is allowed to happen as the leg approximates the couch, one could mistakenly assume the length of the TFL and ITB to be normal, but a tight TFL/ITB will take the hip into this dysfunctional position. It is therefore important to be very diligent when controlling the patient's leg during the test and not allow the hip to flex and internally rotate.

Please bear in mind the following: when Ober's test is positive, we naturally assume that the TFL and ITB are short and subsequently tight, and that the treatment protocol is to "stretch" and/or perform manual therapy techniques to encourage lengthening of the ITB. However, research has now shown that altering the length of the ITB through manual therapy is next to impossible: according to Chaudhry et al. (2008), a change in length of the ITB by only 1% will require approximately 1 ton (925kg) of pressure. Hence, there is little chance of actually causing any significant deformation to the ITB and making it less tight through manual therapy and stretching techniques.

A study by Tenney et al. (2013), however, demonstrated that the activation of the abdominals and hamstring muscles in subjects who experience lumbopelvic pain (with a positive Ober's test) led to an improvement in the position of the pelvis, and subsequently to an overall improvement in the results of Ober's test.

MET Treatment of the TFL and ITB

I consider the following MET an appropriate procedure to be included within a treatment plan, as I feel that it will assist in some way to alter the "tone" of the TFL muscle, rather than altering the length of the connective tissue component, i.e. the ITB. It makes a lot more sense to me to use the PIR effect of METs on the TFL muscle, as I personally believe that this technique helps relax the TFL and hopefully will induce some reduction in tone in the ITB. This is my preferred way of treating these structures, rather than spending a great deal of time performing deep massage techniques (generally called *stripping* the ITB), or advocating the use of a foam roller, to help lengthen the ITB. Techniques of this type (foam roller in particular) may not actually be doing what they have supposedly been designed for in the past, because the soft tissue structure of the ITB (as already discussed) has been shown through research not to change in length by even 1% using almost one ton of pressure.

Let me give you an example to illustrate the above. When the weather is particularly warm at my clinic in Oxford, and especially when I am lecturing, I tend to look out of the window and across the running track to a small area of grass. Almost every day (only when the weather is good), I see a young man spending anywhere between two and three hours rolling his legs using a foam roller. He has been given the nickname "Roller Dave" for obvious reasons, and I have even questioned him on why he does this to his legs. He simply says to me that his therapist recommends doing that every day to *release* his ITBs … I will leave that thought process with you! I am sure some of the students who have attended my courses in Oxford will be smiling when they read about Roller Dave, as they too have undoubtedly seen him rolling his legs many times!

Anyway, back to the treatment protocol. The patient adopts a supine position, and the therapist crosses the patient's flexed left leg over the right leg. The therapist controls the patient's left knee with their right hand and holds onto the patient's right ankle with their left hand. The patient's right leg is then placed into an adducted position until a bind is felt. From the position of bind, the patient is asked to abduct their right leg against a resistance applied by the therapist, as shown in Figure 7.37.

Figure 7.37. The patient abducts their right leg against a resistance.

After a 10-second contraction, and during the relaxation phase, the therapist passively takes the patient's right leg into further adduction (Figure 7.38(a)). This will encourage a lengthening of the right TFL and may possibly have an effect on the ITB as well (albeit small).

Figure 7.38. (a) The patient's left knee is stabilized, while the therapist adducts the right leg, lengthening the TFL muscle.

If you look at Figure 7.38(b), the patient has adopted a side-bent position to the left; this will encourage a lengthening of the right QL muscle as well as a lengthening of the TFL and ITB.

Figure 7.38. (b) The patient side bends to the left, while the therapist adducts the right leg, lengthening the QL and TFL muscles.

Piriformis

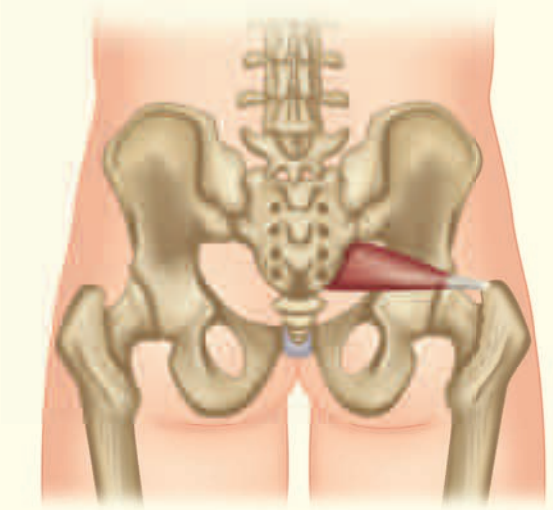


Figure 7.39. Origin, insertion, action, and nerve innervation of the piriformis.

Origin

Internal (front) surface of the sacrum S2–S4.

Insertion

Greater trochanter (top) of the femur.

Action

Laterally rotates and extends the hip joint. Abducts the thigh when the hip is flexed. Helps hold the head of the femur in its socket.

Nerve

Ventral rami of the lumbar nerve (L5) and the sacral nerves (S1, S2).

Observation Assessment of the Position of the Hip

The initial assessment for the relative length of the piriformis muscle is by observation. The patient is asked to adopt the supine position, and the patient's lower limbs are observed from the cephalic end of the couch. The focus of attention will be on the relative position of the foot.

As you can see in Figure 7.40, the patient's left foot appears to be further away from the midline than the right foot. The actual movement has come from the hip, which is in a position of external rotation. This possibly relates to a shortened piriformis on the left side.

The piriformis is a very important muscle regarding sacral torsions, because of its attachment onto the anterior surface of the sacrum. Since its pull is in a diagonal direction, the muscle can rotate the sacral base posteriorly and downward relative to the innominate bone. This motion can then cause a wedging against the innominate, resulting in a loss of mobility of the SIJ, and consequently hypomobility of the joint.

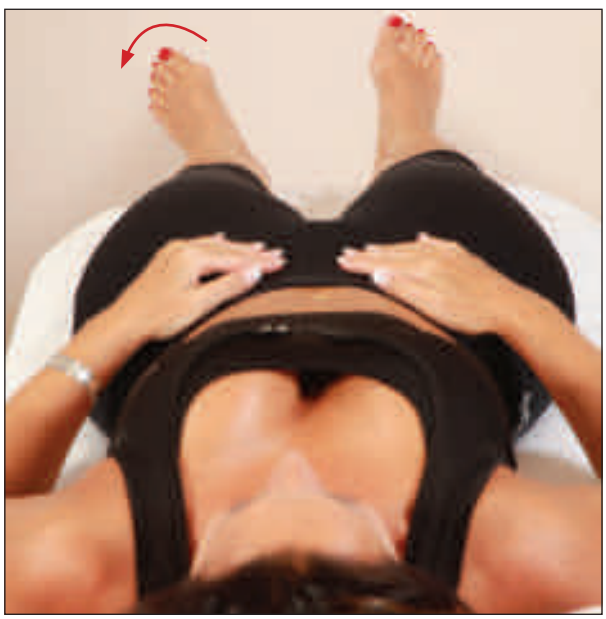


Figure 7.40. The left leg is held in an externally rotated position.

Passive Assessment of the Piriformis

In order to look at the position of the hip to help us decide whether the piriformis is held in a shortened position, we ask the patient to adopt a prone position. One of the patient's knees is flexed to 90 degrees, and the hip is passively controlled by the therapist and allowed to internally rotate. This is repeated with the other knee flexed to 90 degrees. The side that has the least ROM possibly indicates relative shortness of the corresponding piriformis (Figure 7.41).

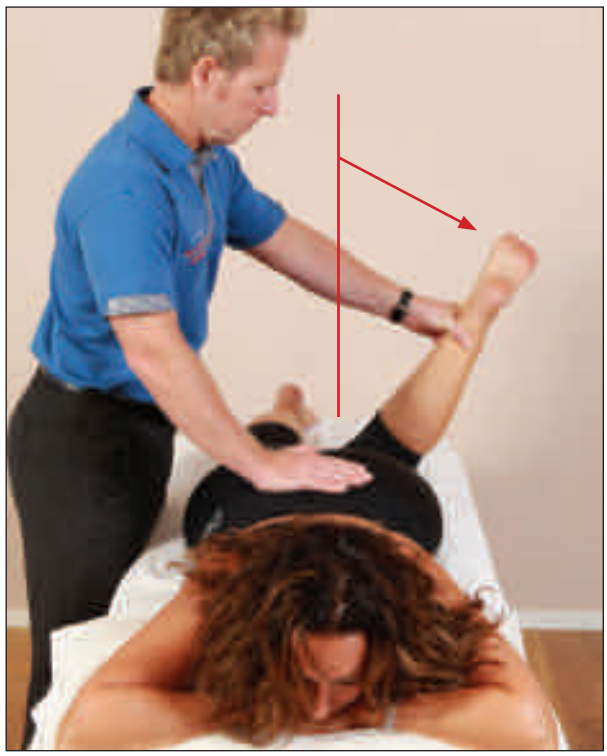


Figure 7.41. The left hip is passively taken into internal rotation to assess for shortness of the piriformis.

Another way of assessing the relative length of the piriformis is as follows. The patient is asked to adopt a prone position with both of their knees bent, and then let their legs "flop out"; this will induce internal rotation of the hip joints.

From the cephalic position of the patient, the therapist observes the position of the lower limb. As you can see in Figure 7.42, the lower limb appears to be asymmetric on one side. One can assume that the patient's left side is the dysfunctional side, as the hip is in a position of external rotation. In this case, internal rotation of the hip is restricted, which means that the piriformis on that side is held in a shortened position.

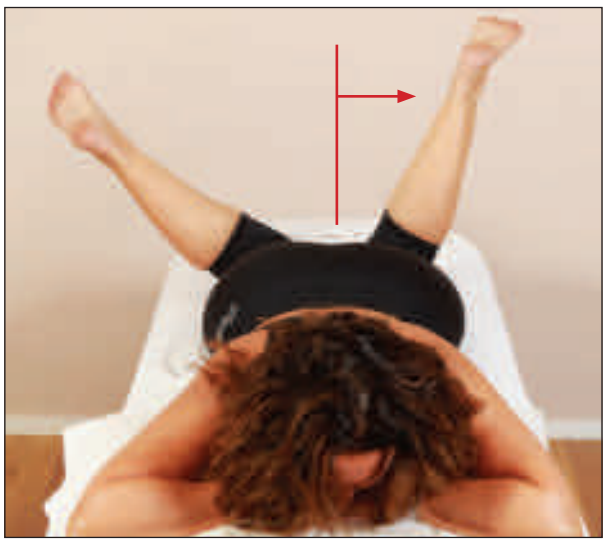


Figure 7.42. A decreased ROM of the left hip, indicating a short left piriformis.

MET Treatment of the Piriformis

The patient adopts the position of the test as described above, but with the right leg straight and the left knee bent. The therapist makes sure that the pelvis/sacrum is stabilized with their right hand, while controlling the patient's left leg with their left hand. The patient's left leg is passively taken into internal rotation until the position of bind is felt, and the patient is asked to contract the piriformis by pulling their leg against resistance applied by the therapist's left hand. This will induce an external rotation of the hip joint (Figure 7.43).

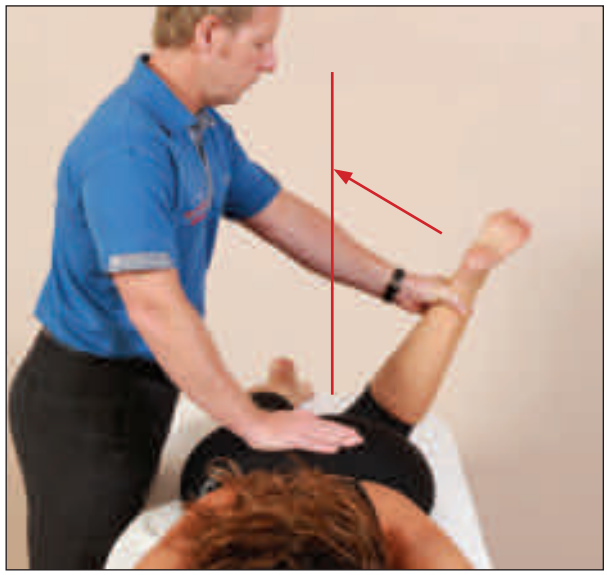


Figure 7.43. The patient is asked to pull their left leg across their body against a resistance. The therapist stabilizes the lumbar spine with the right hand.

After a 10-second contraction of the piriformis, and during the relaxation phase, the therapist takes the patient's left hip into further internal rotation. This will lengthen the piriformis, as shown in Figure 7.44.

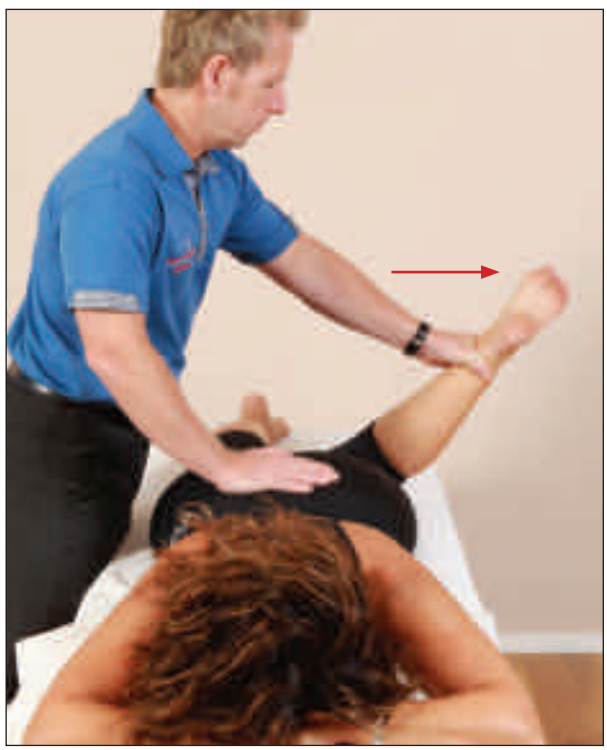


Figure 7.44. The therapist lengthens the piriformis while stabilizing the lumbar spine.

Alternative MET Technique for the Piriformis

Technique 1

This time the patient is asked to adopt a supine position, and the therapist passively takes the patient's left leg and crosses it over the right leg. Controlling the movement of the patient's left innominate with their right hand, the therapist applies pressure to the patient's left knee, passively inducing adduction of the hip to the point of bind.

The patient is asked to abduct their left leg (the piriformis is an abductor), while the therapist resists the movement, as shown in Figure 7.45.

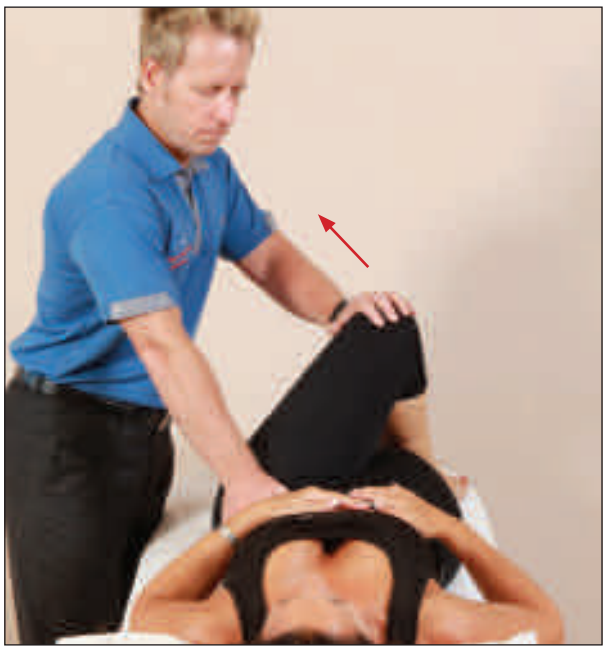


Figure 7.45. Technique 1: from the point of bind, the patient abducts against the pressure applied by the therapist in the direction of the arrow.

After a 10-second contraction, and during the relaxation phase, the therapist passively takes the patient's left leg into further adduction, as shown in Figure 7.46.

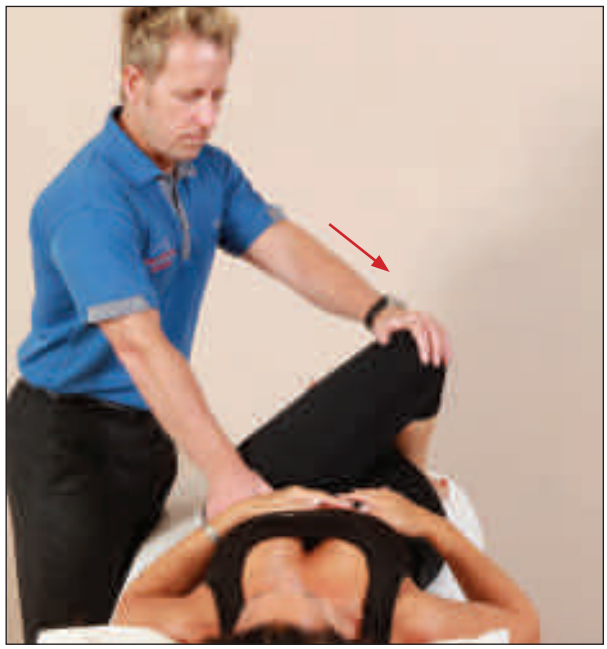


Figure 7.46. Technique 1: the therapist takes the patient's left leg into further adduction and stabilizes the innominate/lumbar spine with the right hand.

Technique 2
This is my preferred way of lengthening the piriformis muscle.

Controlling the patient's left leg, the therapist tries to encourage flexion of the hip, while at the same time externally rotating the hip with some adduction. This technique will place the piriformis into a position of relative bind, but will need fine-tuning by the therapist and feedback from the patient to finally achieve the optimum position. From this finely tuned position of bind, the patient is asked to push their knee away, into the abdomen of the therapist. This will induce a contraction of the piriformis, as shown in Figure 7.47.

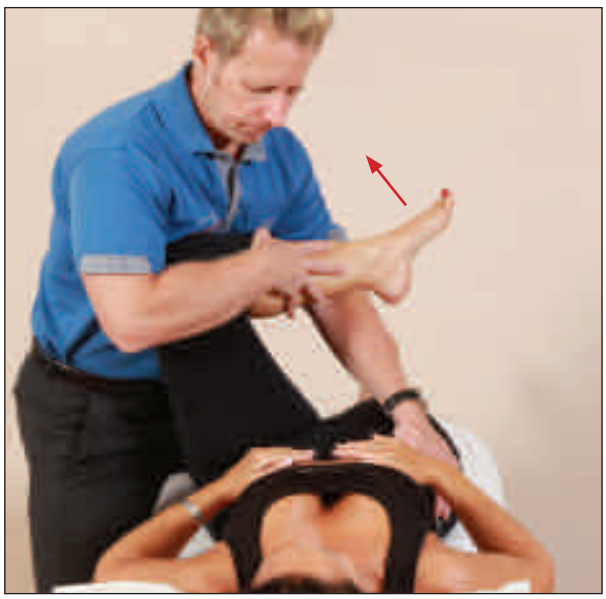


Figure 7.47. Technique 2: this technique will need fine-tuning to get to the optimum position. From the position of bind the patient is asked to push their knee away.

After a 10-second contraction, and during the relaxation phase, the therapist passively encourages the hip into further external rotation while applying some hip flexion/adduction as shown in Figure 7.48.

Figure 7.48. Technique 2: using their chest and hand, the therapist encourages further external rotation and adduction of the patient's left hip.

Tip: The sciatic nerve of one in five of the population (20%) passes through the piriformis muscle. This can result in buttock and leg pain, but generally no back pain is present, so make sure you eliminate disc/lumbar spine pathology from your hypothesis.

Note: It is considered that after 60 degrees of hip flexion, the piriformis changes from an external rotator to an internal rotator—this is because of its anatomical attachments. If you look closely at Figure 7.48, this is the reason why the patient's left hip is placed in an externally rotated position. This will now lengthen the piriformis, as the left hip has exceeded 60 degrees of flexion.

Quadratus Lumborum (QL)

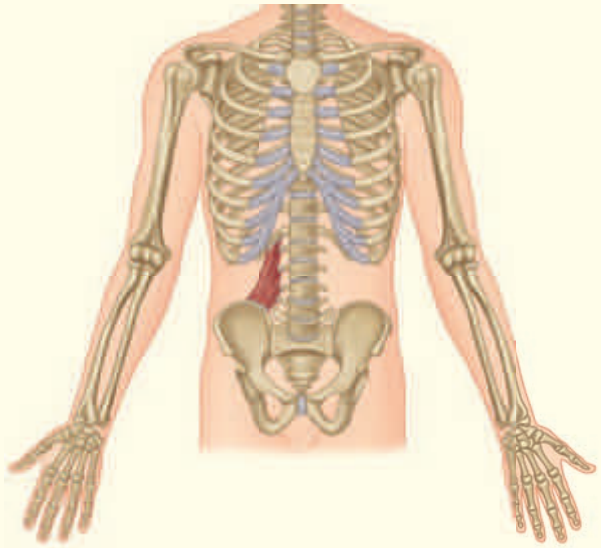


Figure 7.49. Origin, insertion, action, and nerve innervation of the QL.

Origin

Iliac crest. Iliolumbar ligament (the ligament from the 4th and 5th lumbar vertebrae to the ilium).

Insertion

12th rib. TPs of the upper four lumbar vertebrae (L1–L4).

Action

Laterally flexes vertebral column. Fixes the 12th rib during deep respiration (e.g. helps stabilize the diaphragm of singers exercising voice control). Helps extend the lumbar spine component of the vertebral column and gives it lateral stability.

Nerve

Ventral rami of the subcostal nerve and upper three or four lumbar nerves (T12, L1, L2, L3).

Assessment of the QL

In my experience I find that the standing side flexion test is relatively good for indicating tightness of the QL.

The patient stands upright and maintains a neutral position of the lumbar spine. From the standing position, the patient is asked to side bend to the left, and at the same time slide their left hand down the outside of their left leg, as shown in Figure 7.50. When the position of bind is reached (this is felt by the therapist palpating the right side of the QL as the patient side bends to the left), the patient's left middle finger should be in contact with the head of the fibula on the left side.

If the middle finger is close or touches the fibula head on the left side, the QL on the right (contralateral) side is classified as *normal*; if there is a restriction, the QL on the right is classified as *tight*.

> **Note:** This test is not conclusive for determining shortness of the QL, as many other lumbar spine factors will affect the overall result. For example, any intervertebral lumbar disc pathology or facet joint restriction/pain will be affected during this test and will give the therapist a false-positive result.

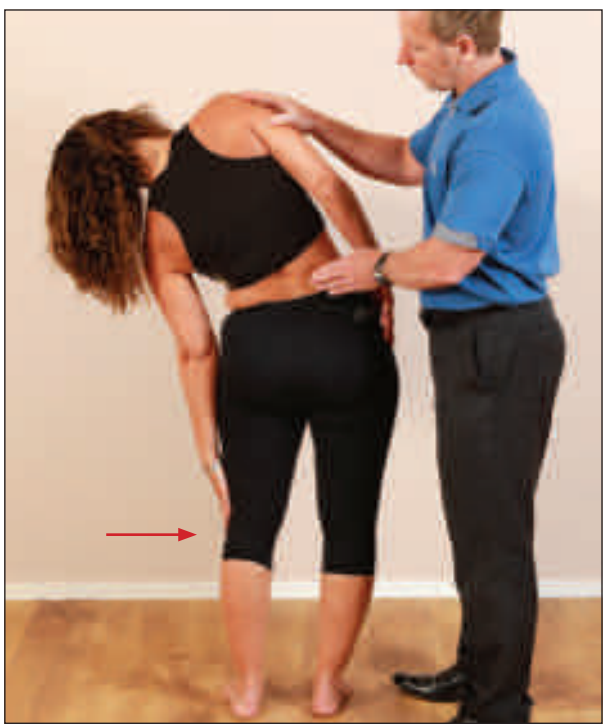


Figure 7.50. The left hand approximates the head of the fibula if the right QL is of normal length.

MET Treatment of the QL

PIR Method

The patient is asked to adopt the shape of a banana on the couch: this is achieved by the patient assuming a side-lying supine position, with their right hand placed underneath their head, and their right leg over their left leg. The left leg overlies the edge of the couch, as shown in Figure 7.51.

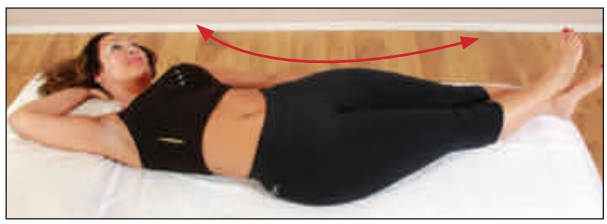


Figure 7.51. The patient's right QL is taken to the point of bind by assuming the "banana" position.

Once the patient has adopted this position, the therapist places their right hand under the head of the patient and cradles the right axilla. The left hand of the therapist stabilizes the patient's left pelvis.

From this position, the patient is asked to side bend to the right against the resistance applied to their axilla by the therapist's right hand (Figure 7.52). This will induce a PIR method of contraction in their right QL.

Figure 7.52. The patient side bends to the right while the therapist's left hand stabilizes the patient's left pelvis.

After a 10-second contraction, and during the relaxation phase, the therapist induces further side bending to the left, which will lengthen the QL on the right side.

Reciprocal Inhibition (RI) Method

The position of the patient and the procedure are similar to those explained for the PIR method, the only difference being that when the therapist encourages the new position of bind, the patient is asked to reach their left hand toward their left leg (Figure 7.53). This will induce a contraction of the left QL and cause the right QL to relax through RI, allowing a lengthening to occur.

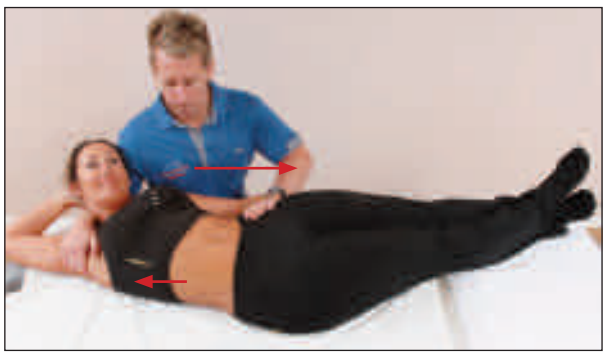


Figure 7.53. The patient is asked to slowly side bend to the left as the therapist guides the motion. This induces an RI effect in the right QL.

Alternative MET for the QL

For an alternative MET for the QL, the patient is placed in a side-lying position, with their left leg off the side of the couch, as shown in Figure 7.54. The therapist stabilizes the lower ribs (attachment of the QL) with their right hand and controls the patient's left leg with their left hand. The patient is asked to abduct their left leg against the resistance applied by the therapist's left hand; this will induce a PIR contraction of the left QL muscle.

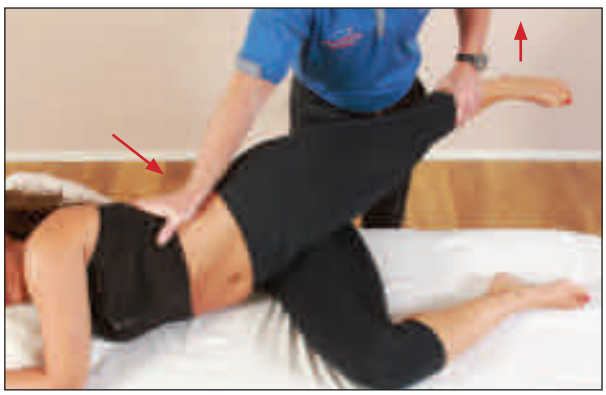


Figure 7.54. The patient abducts their left leg, while the therapist's right hand stabilizes the lower ribs.

After a 10-second contraction, and during the relaxation phase, the therapist slowly and passively takes the patient's left leg into further adduction while stabilizing the patient's lower back (Figure 7.55). This will lengthen the QL on the left side.

Figure 7.55. The therapist stabilizes the lower back and gently applies cephalic pressure to the 12th rib with their right hand while encouraging adduction of the left leg.

Tip: The QL can become overactive and subsequently shorten if the contralateral (opposite) Gmed is weak. It can also be strained by overreaching to one side, for example to the right; in this case, the strain sustained to the left side will result in a protective spasm of the left QL. If the left QL becomes shortened, it will appear as if an iliosacral upslip (see Chapter 12) has occurred on the left side of the innominate bone.