# Psoas insufficiency and its role in sacroiliac dysfunction and low back pain


**Musculoskeletal Physiotherapy Theory and Management**

Stelios Petroutsos

# Contents


**Introduction** **4**

**Anatomy-basic considerations** **5**

The ligaments……………………………………………………………………………**6**

The thoracodorsal fascia…………………………………………………………......**6**

The muscles ……………………………………………………………………………**7**

The nerves………………………………………………………………………………**8**

**Pain patterns; where pain comes from?** **9**

Lumbosacral region…………………………………………………………………..**9**

Hip joint……………………………………………………………………………….**10**

**Biomechanics/kinematics and dysfunction** **11**

**Stability of the lumbopelvic-hip region** **15**

Neutral zone, elastic zone and instability…………………………………………...**15**

Form closure. Critical issues………………………………………………………..**17**

Force closure. Critical issues………………………………………………………..**18**

**Examination of the lumbopelvic-hip region** **22**

Subjective examination……………………………………………………………...**22**

Contributing factors……………………………………………………………….....**23**

Clinical presentation of psoas insufficiency syndrome……………………………...**24**

**Physical examination-important considerations** **24**

Lumbar spine…………………………………………………………………………...**24**

Sacroiliac joints………………………………………………………………………...**25**

**Clinical diagnosis** **29**

Management…………………………………………………………………………....**29**

**Conclusions** **32**

**References** **34**

# Abbreviations

PIS: Proas Insufficiency syndrome
SIJ: Sacroiliac joint(s)
SCJ: Sacro-Coccygeal-Joint
IL: Interosseous Ligament
SSL: Sacrospinal Ligament
GM: Gluteus Maximus muscle
BF: Biceps Femoris muscle
TDF: Thoracodorsal fascia
TrA: Transversus Abdominis
ISC: Ischiococcygeus muscle
EO: External Oblique muscle(s)
RA: Rectus Abdominis
LD: Latissimus Dorsi
IVD: Intervertebral Ligament
S/E: Subjective Examination
WB/non-WB: Weight Bearing/ non- Weight Bearing
NP: Neutral Position
MVA: Motor Vehicle Accident
PPPP: Post-Partum Pelvic Pain
LBP: Low Back Pain
HL/R: Hallux Limitus/Rigitus
MRI: Magnetic Resonance Imaging
POSH test: Posterior Shear test or thigh thrust test
EZ: Elastic Zone
PS: Pubic Symphysis
ILL: Iliolumbar Ligament
STL: Sacrotuberous Ligament
LDL: Long Dorsal Ligament
PF: Piriformis muscle
ILF: Iliofemoral Ligament
TLF: Thoracolumbar Fascia
MF: Multifidous muscle
LA: Levator Ani
IO: Internal Oblique muscle(s)
ES: erector Spinae
TFL: Tensor Fascia Late
QF: Quadratus Femoris
P/E: Physical Examination
EMG: Electro-Myography(ic)
ROM: Range Of Motion
ASLR: Active Straight Leg Rising
CSA: Cross-Sectional
L/L: Left on Left axis sacral rotation
MTFJ: Metatarsophalangeal Joint
PSLR: Passive Straight Leg Rising
DLAs: Daily Living Activities

# Introduction

LBP is a costly illness that influences patients not only in a physical state but also in a broader psychosocial level. More than 60% of populations suffer from LBP in their lives (Heliovaara et al 1989). It is not surprising that many different diagnostic and therapeutic approaches exist, since pain in the lower back is such a multifactorial and complicated phenomenon. Even sophisticated imaging methods, such as CT and MRI scans, are unable in some cases to give a reasonable explanation of patient's symptoms (Ebraheim et al 1997). The complex enervation of lumbo-sacral region and the sympathetic trunk contribution makes diagnosis, even with double blocks sometimes unreliable (Willard 1997, Berrard 1997).

Depending on patient's age and lifestyle, adults commonly present a history of recurrent painful attacks, increasing progressively in frequency, duration and severity. In other cases patients are unaware of significant prior back pain episodes. Patient emotions can contribute to overall symptoms. That makes subjective examination and history taking even more complicated. Moreover physical examination routines vary among philosophies and treatment approaches.

A variety of different components, progressively built up back pain. Our clinical reasoning process helps us to identify possible sources and contributing factors of these impairments. However we are not able to detect what caused what! Psoas dysfunction is but one of the possible factors for consideration and will form the basis of this presentation.

The Psoas Insufficiency Syndrome (PIS) scenario was introduced by Bachrach in 1987 and it is well accepted as a possible source of back pain (Hammer 1992, 1995, Corn 2002). In the PIS scenario inadequacy of the lumbosacral compensatory mechanisms for contracture, shortening, or failure of adaptive lengthening of the psoas produces PIS. This increases lumbar lordosis and also the load on the facets contributing to thoracolumbar paravertebral myophascial systems shortening, the pelvis drops in anterior rotation and the abdominals are overstretched. SIJ also stiffens. There is a compensatory increase in the thoracic kyphosis, the head is forward and the cervical spine tends to flatten. Usually more often in dancers the right psoas muscle is most tight. The ipsilateral leg seems to be longer, the knee is flexed and the foot is pronated (Bachrach 1997).

The above mentioned are general observations. This essay will explain how we can structure our clinical reasoning, physical examination and treatment approach to address the impairments and symptoms that relate to this syndrome. This will be based on the work of Lee and Vleeming; the integrated model. This model analyses the lumbopelvic-hip region biomechanics as an integrated model. It has become apparent that there are common patterns of dysfunction through this region. From these observations a logical approach to examination and treatment has evolved (Lee 1999). The following chapters will summarise the most current evidence that support this fascinating approach and will correlate them to the PIS. Needless to say, this approach will be entirely bio-medical for the needs of this essay. Furthermore, due to the world limit, this essay will only refer to the Psoas Insufficiency due to muscle shortening.

## Anatomy-basic considerations

The pelvic girdle as a unit supports the abdomen and the organs of the lower pelvis and also provides a dynamic link between the spine and the lower limbs. It is a closed osteoarticular ring composed of six or seven bones including the two innominates, the sacrum, the coccyx, and the two femora as well as six joints including the two sacroiliac joints, the Pubic Symphisis (PS), the sacrococcygeal joint (SCJ), and the two hip joints (Lee, 2004). The SIJ compared with other synovial joints are unique and fascinating (Walker 1992).

It is not the purpose of this essay to focus on detailed anatomy. Thus a brief summary of the key anatomical points will follow, that will help the examiner to create a better understanding of the integrated model and the variable pain patterns that patients with psoas dysfunction may present with.

**The ligaments**

The iliolumbar ligament (ILL) is very important for the stability of the lumbosacral region in excessive spinal movements especially in females which sometimes attaches on the transverse processes of L4 as well (Doris 1997).

The ligamentous apparatus of the SIJ has a dynamic role on the stability of this amphiarthrosis (Gerlach 1992). The interosseous ligament (IL) is the main stabilizer and its thickness makes intra-articular injections quite difficult (Wingerden et al 1997).

Sacrotuberous (STL) and sacrospinal (SSL) ligaments resist sacrum nutation whereas the long dorsal ligament (LDL) resists sacrum counternutation. GlutMax, Piriformis (PF) and Biceps Femoris (BF) muscles attach also on the STL (Vleeming et al 1989) thus their activity and or dysfunction influence SIJ mobility and kinematics (Vleeming et al 1989).

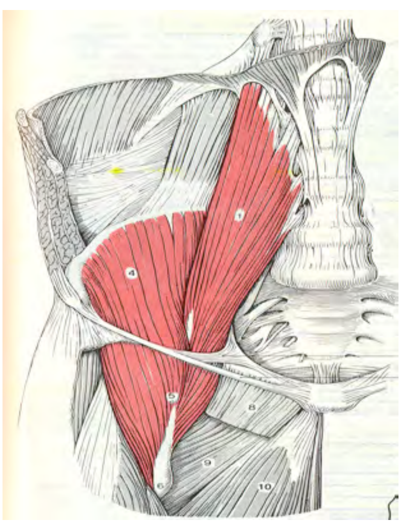
Of the hip joint ligaments, the iliofemoral (IFL) is the thickest and stronger. It mainly restricts hip extension. In the upright position the head of femur rests against IFL and its tension resists the pelvis to posteriorly rotate. Its action is reinforced by the iliopsoas muscles (IPM) (Esola et al 1996).

**The thoradorsal fascia (TDF)**

Anatomical studies have proven the extent attachments of TLF from the sacrum till the upper cervical muscles (Black et al 1996). That's why it is suggested that this fascia should be called TDF (Barker and Briggs 1999). During trunk flexion, it produces flexion momentum to the sacrum whereas neck flexion tenses the TDF even more (Sniiders et al 1993b). These observations implicate that the Slump test is not neuromeningial specific. Variations also among individuals make diagnosis harder! (Barker 1999).The posterior layer fibres cross at the L2-S2 levels, contributing to the stability of the SIJ and inferiorly they blend with the fascia of the leg assisting in the transfer of the load from the legs to the pelvis and trunk (Money et al 1997, Vleeming et al 1995,).

**The muscles**

The Psoas muscle arises from the bodies, intervertebral discs and transverse processes of the five lumbar and the twelfth thoracic vertebrae and from the membranous septae between them. It courses downward, forward and slightly laterally, crosses under the inguinal ligament in front of the hip joint and then passes posteriorly joining the iliacus to its insertion into the lesser trochanter of the femur (figures 1a, 1b). It is innervated by the L1, L2 and L3 lumbar nerves. The fascia that surrounds the PM stretches from the medial lumbocostal arch to the thigh therefore any inflammatory process in the thoracolumbar region can extend within the fascial tube to appear as wandering abscesses as far down as the thigh (Platzer 1992).



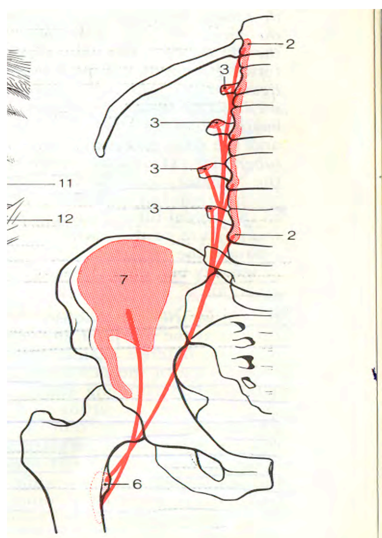


***Figure 1:** a) Iliopsoas, b: origin and insertion (modified from Platzer 1992, pp. 231)*

***a*** ***b***

There are more than 35 additional muscles attaching at the lumbopelvic-hip region. They function with the ligaments and fascia to synchronise motion and stability of the trunk and extremities (Lee 2004). The key muscles that assessment and treatment of the integrated model is relied on and their innervation are posted on table 1.

| Muscle | Innervation |
|---|---|
| Transversus Abdominis (TrA) | Anterior primary rami of T7-T12 and L1 |
| Multifudus (MF) | Lateral branch of the dorsal ramus of L1-L5 |
| Ischiococcygeus (ISC) | Ventral rami of the sacral plexus, S3 and S4 |
| Levator Ani (LA) | Anteromedial portion: pudendal nerve, while Posterolateral region: sacral plexus S3 and S4 |
| Diaphragm | Motor supply: phrenic nerves (C3-C4)<br>Sensory supply: intercostal nerves (T6-T12) |
| External Obliques (EO) | Ventral rami of T7-T12 |
| Internal Obliques (IO) | Ventral rami of T7-T12 |
| Rectus Abdominis (RA) | Ventral rami of T6 or T7-T12 |
| Erector Spinae (ES) | Lateral and intermediate branches of the segmental dorsal spinal rami |
| Latissimus Dorsi (LD) | Thoracodorsal nerve (C6-C8) |
| Iliacus | Lumbar plexus and femoral nerve (L2-L4) |
| Psoas | Lumbar plexus and femoral nerve (L1-L3) |
| Gluteus Maximus (GM) | Inferior gluteal nerve (L5-S2) |
| Tensor Fasciae Latae (TFL) | Superior gluteal nerve (L4-L5) |
| Piriformis (PF) | Ventral rami of L5 and S1 |
| Biceps Femoris (BF) | Long head: tibial nerve (L5-S1)<br>Short head: common peroneal nerve (S1-S2) |

***Table 1:** Important muscles around the lumbopelvic-hip region and their innervation*

**The nerves**

Most of the structures of the lumbosacral region receive a generous nerve supply.

The facet joint is innervated by branches from the medial divisions from the dorsal rami above and below. The L5-S1 facets are innervated by L4, L5, S1 spinal nerves. The bone and articular cartilage are innervated as well. An extensive nerve plexus is present in the anterior and posterior longitudinal ligaments. The intervertebral discs (IVD) are innervated by fibers from an elaborate plexus composed of the sinu vertebral nerve posteriorly and the somatosympathetic nerve anteriorly (Willam 1997).

SIJ receive reach innervation. The patterns are controversial but seem to be innervated anteriorly from the anterior primary rami (L4-S3) and posteriorly from

L2-S2 (Berrard 1997). Anterior SIJ ligaments are further supplied from L2-S2 (Solonen 1952). These findings challenge the sensitivity/specificity of joint blocks in SIJ. The PS is innervated from branches of the pudendal and genitofemoral nerves (Gamble et al in Lee 2004).

The hip joint receives branches from the obdurator nerve (L2-L4), the nerve to the Quadratus Femoris (QF) (L2-L4) and the superior gluteal nerve (L5, S1).

## Pain patterns; where pain comes from?

**Lumbosacral region**

The primary afferent fibers in the sinu vertebral nerve can refer pain several segments up or down in the spinal cord, as well as referring pain to the contralateral side. This is why we sometimes see symptoms shifting from the one side to the other. However, sinu vertebral nerve and somatosympathetic nerve do not innervate the muscles so when impaired can cause pain but no weakness. Pain can also be referred to the upper lumbar or lower thoracic regions due to somatosensory sympathetic nerves. This type of pain is dense, diffused and boring pain (Willard 1997). Lumbosacral dorsal rami innervate the facets as well. They can cause sharp, burning pain similar to spinal root pain and can also refer pain to areas supplied by ventral rami mimicking sciatica (Willard 1995, 1997)

In psoas dysfunction the lumbosacral hyper-lordosis can cause degenerative changes in the L5-S1 intervertebral disc (IVD) and stretch the thoracolumbar long ligament as well. Traction of the sympathetic and somatic motor fibers to the psoas may ensue (Willard in Vleeming 1997). Pain with coughing can also be provoked from SIJ dysfunction (DonTigny 1985).

Cross-sectional studies proved that SIJ is a source of pain with the most common referral to the groin area (Schwarzer et al 1995, Smidt et al 1997).

Other common patterns are pain extended 10 cm below and 3 cm lateral to the PSIS. Thigh, calf and buttock pain can also occur (Harrison et al 1997, Fortin et al 1994a, 194b). A review (Walker 1992) proposed that accessory joints occur in 8-35.8% of population. They may contribute or are responsible for SIJ dysfunction. Degeneration through age also occurs although if this is related to pain it is still unknown (Berrard 1997).

Double blocks are considered as the only reliable diagnostic tool for SIJ pain (Schwarzer et al 1995). Such studies suggest that pain in the buttock and leg which does not spread superior to L5 level has more a SIJ contribution (Harrison et al 1997, Maigne et al 1996). However, extraarticular structures can also cause pain patterns (Fortin et al 1997). Therefore, joint blocks can lead to false negatives when dealing with SIJ dysfunction (Laslett 1998). The surrounding soft tissues receive nerves that are capable to sustain a prolonged inflammation thus initiating chronic pain conditions (Willard 1997).

Common sites of Psoas dysfunction referral are the anterior thighs and central low back pain. Figure 2 shows some common pain patterns coming from SIJ dysfunction. These Symptoms may be present secondary due to psoas insufficiency!

**Hip joint**

The reach innervation of this joint (L1-S1) can cause several pain patterns. The most common areas of referral are anterior thighs and/or anterior knees, the groins and the buttocks (Roos 1997, Sims 1999a, 1999b). Psoas insufficiency can also cause pain on the anterior aspect of the hip joint. Other soft tissues around the joint can cause pain and should be differentiated through our S/E and P/E.

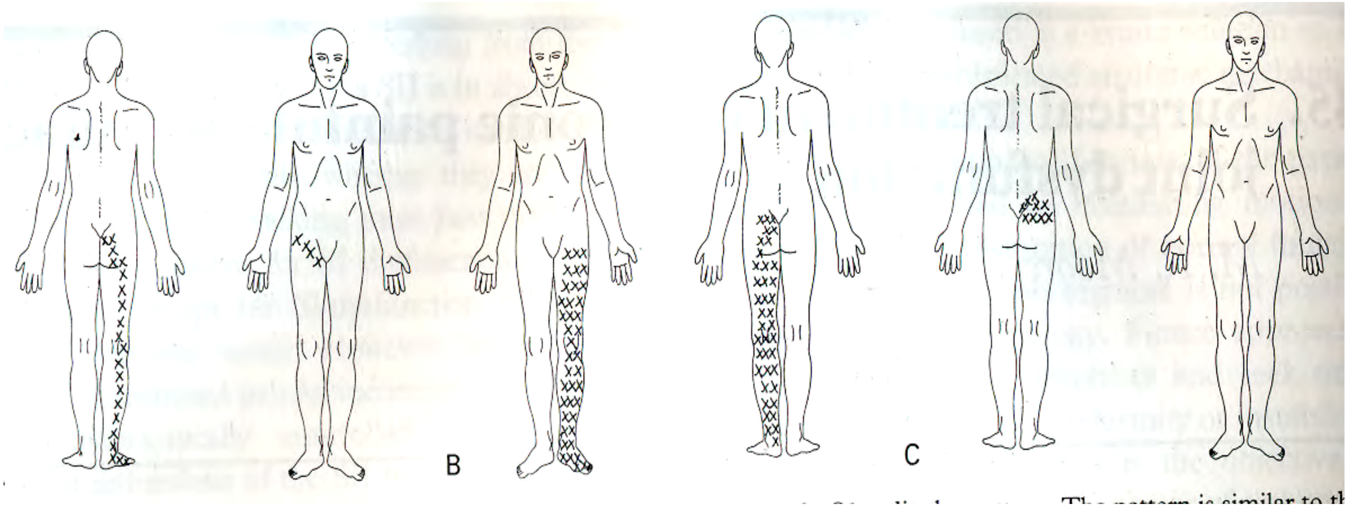



***Figure 2:** **Common pain patterns from psoas related SIJ dysfunction (modified from Moore in Vleeming 1997, pp. 564)***

## Biomechanics/kinematics and dysfunction

Huson proposed in 1997 that all changes in the kinematic position, due to one articular deficit, will have an immediate effect on kinematics of the other joints as well, proving that observation of the mechanics of the integrated model is an essential diagnostic tool (Dolan 1995). The following are general valid observations from the literature to help clinicians identify movement impairments and possible sources of symptoms during examination routine.

- *The SIJ mobility issue:* SIJ mobility is well documented in adults. Recently a stereophotogrametric analysis (RSA) study proved, in WB-tasks, that SIJ mobility remains until elderly ages. Rotation of the sacrum in younger will be more than 4 degrees! Men have 30-40% less mobility than women (Sturesson 1997). Gymnasts have slightly more range than normal adults due to the loss of form closure revealing strong force closure stability. That’s why when athletes quit gymnastics LBP problems initiate in many cases (Smidt 1997).

- Smidt in 1997 in a fresh cadaveric non-WB study noted maximum movement of the SIJ in extreme hip positions and not only in sagital but also in the transverse plain as well. He concluded that decreased hip joint mobility can influence SIJ mobility. Equally Vleeming and colleges (1992) observed on the side of decreased hip mobility after total hip reconstraction, increased SIJ mobility.

- Rotation of the L5 vertebra is limited by the articulation with the sacrum. Loss of SIJ accessories will decrease L5-S1 mobility, thus will increase stress on the L4-L5 IVD (DonTigny 1990).

- When siting on ischial tuberosities PS is stressed due to the reaction force. The contact of coccyx tends to turn sacrum into counternutation. When long dorsal ligaments (LDL) are compromised, there is no SIJ self bracing. This could be an SIJ contribution to symptoms in non-specific LBP with prolonged siting (Sniiders et al 1993). An EMG study in health subjects noted that leg crossing should be encouraged in working environments as it enables the self-bracing of the SIJ (Sniiders et al 1995). However, in the long-term this habit could reach the creep phenomena of the stabilizing tissues increasing the neutral zone and reducing instability (Vleeming 1997).

- Nutation of the sacrum stresses the PS. That's why "Osteitis Pubis" sufferers tend to evolve a counternutated position (anterior pelvis rotation) to reduce the stress. This leads to psoas tightness, unequal lumbar loading and intrapelvic instability (Vleeming 1997).

- Coupling movements occur in the lumbar spine but they are variable depending on the starting position and the individual patient (Cholewiski et al 1996). Hindle et al (1990) characteristically concluded that the word "normal" is somewhat an inappropriate term to discuss backs.

- Lumbar extension spinal narrows the spinal canal, whereas in flexion spinal the canal seems to widen (Inufusa et al 1996). Moreover, trunk flexion tends to stretch the SIJ anterior capsule thus producing pain (DonTigny 1985), whereas hyperextension corrects anterior SIJ dysfunction (DonTigny 1985). Hyperextension when psoas muscles are tight happens mostly to the L5-S1 segment, which over-extends and posteriorly displaces to compensate for restricted hip extension (Lee 2004).

- *Forward bending*: the lumbopelvic rhythm change in healthy subjects (control group) as follows. Lumbar spine flexion ratio to hip joint flexion ratio: 0-30º= 2:1, 30-60º = 1:1 and 60-90º= 1:2 (reversed). Subjects that had previous history of LBP but where asymptomatic at the time of the study noted the same range of forward flexion but impaired rhythm. More specific: 0-30º= 1:1, 30-60º= 0.72. Symptomatic subjects had 50% less range in the same movement (Esola et al 1996, Kaigle et al 1998). The lumbopelvic rhythm changes significantly in asymptomatic subjects when the spine is loaded. When carrying a box for instance it is observed that during flexion the lumbopelvic rhythm happens simultaneously whereas in extension it occurs sequentially (Nelson et al 1995).

- *Rising from forward bending*: in healthy subjects the same kinematics take place reversed: 90-60º= 1:2 (more movement happens at the hip joints), 60-30º= 1:1 and 30-0º= 2:1 (Kaigle et al 1998). Subjects with history of LBP with no symptoms at the time of the study showed altered lumbopelvic rhythm where the return to erect posture initiated from the spine. Those with symptoms returned with assistance (hands on thighs) (McClure et al 1997). All observations took place in controlled, experimental studies (level 3 of evidence).

- *Walking normal biomechanics*: since gait has been established are briefly demonstrated in figure 3. Instability of the SIJ is always reflected in the gait pattern (Lee 1997). Based on previous EMG controlled experimental studies on LBP sufferers (Bullock-Saxton et al 1993, 1994), Vleeming (1997) proposed that the posterior oblique and longitudinal slings serve to save energy during WB activities. People with back problems have insufficient posterior slings and that is why they experience difficulty with prolonged WB activities such as shopping, standing etc (Vleeming 1997).

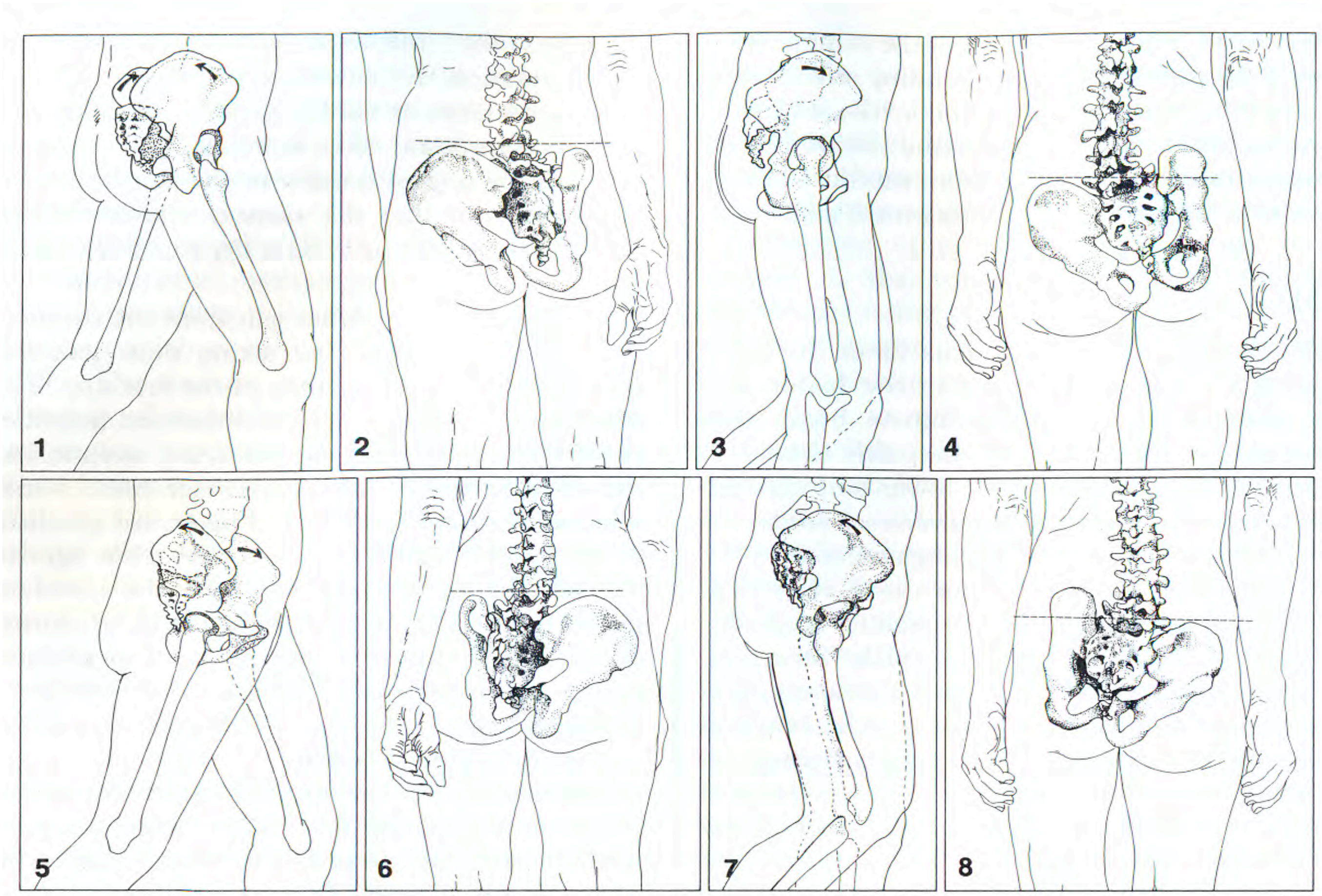



***Figure 3: Combined actions of the innominates, sacrum and spine during walking.***
***1) At right heel strike the ipsilateral inominate has posteriorly rotated while left is in anterior rotation (intrapelvic torsion),***
***2-4) during right single leg stance, ipsilateral inominate anteriorly rotates. 5) After right toe-off when left leg is on heel strike,***
***6-8) during swing the right inominate tends to rotate posteriorly. The sacrum tends always to flex, rotate and side bend away from the heel strike side (2 and 6). L5 follows the kinematics of the sacrum. The rest spine adapts to keep the erect posture.***
***(modified from Greenman in Vleeming 1997, pp. 240)***

# Stability of the lumbopelvic-hip region

## Neutral zone, elastic zone and instability

An introduction to the "neutral zone and instability hypothesis" (Panjabi 1992b) is essential to understand the principles of the integrated model. According to Panjabi (1992b pp 391):

- *Neutral Position* (NP): "is the posture of the spine in which the overall internal stresses in the spinal column and muscular effort to hold the posture is minimal".

- *Neutral Zone* (NZ): "is that part of the range of physiological intervertebral motion, measure from the NP, within which the spinal motion is produced with a minimal internal resistance". There is a high correlation between increased NZ and spinal pathology (Knutsson in Panjabi 1992b).

- *Elastic Zone* (EZ): "is that of the physiological intervertebral motion, measured from the end of the neutral zone up to the physiological limit". Within the EZ spinal motion is produced against resistance. "It is the zone of high stiffness".

- *Range Of Motion* (ROM): "is the entire range of intervertebral motion measured from the NP". It is the summation of NZ and EZ (figure 4).

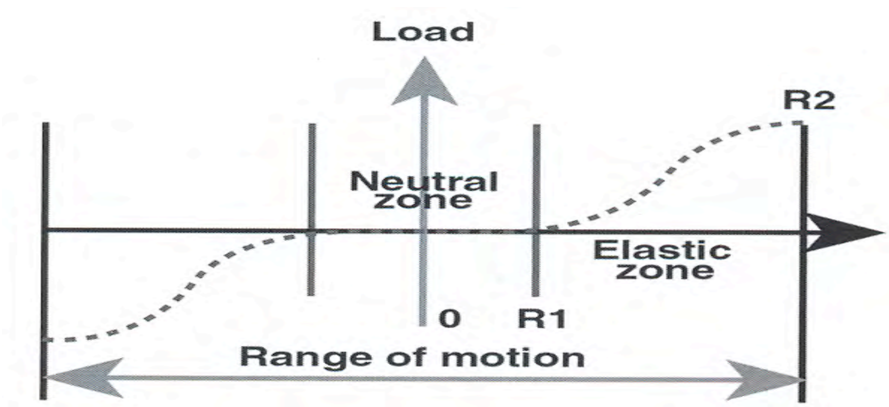



***Figure 4**: Neutral zone and elastic zone within ROM (modified from Panjabi 1992b)*

The hip joint is capable of 12 degrees of freedom of motion (figure 5). Each intervertebral segment (figure 6) and the SIJ (figure 7) have both the potential for 12 degrees of freedom of motion, but in a significant less quantity.

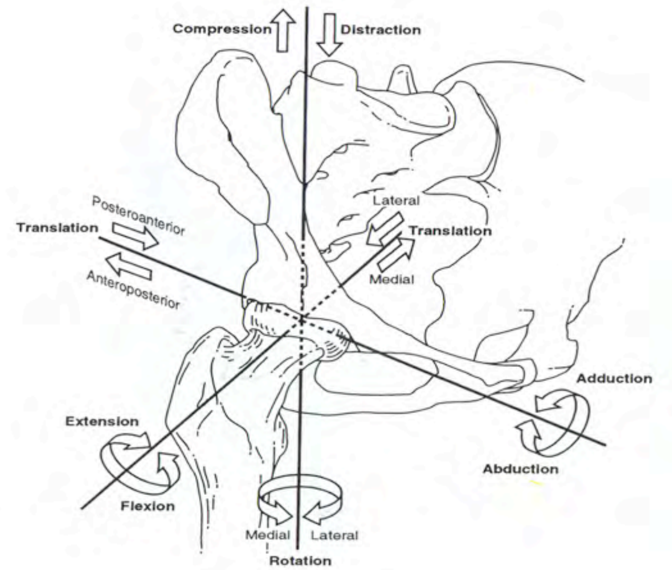



*Figure 5: Hip joint freedom of motion (modified from Lee 2004, pp. 64)*

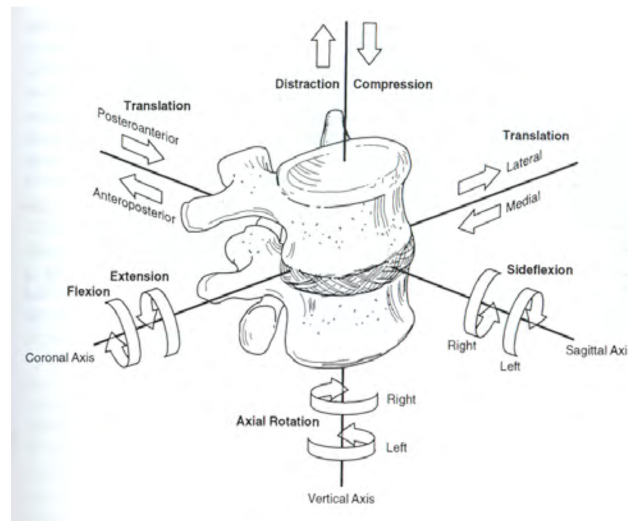



*Figure 6: Lumbar spinal segment freedom (modified from Bogduk in Lee 2004, pp. 57)*

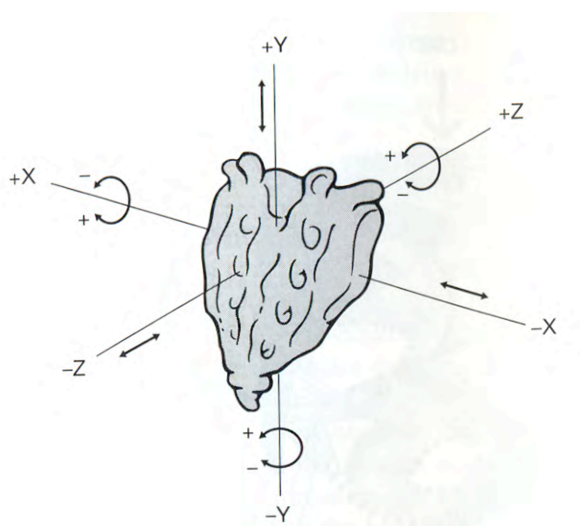



*Figure 7: Sacrum freedoms of motion (modified from Vleeming 1997, pp. 159)*

The integrated model of function has four components; three are physical and one psychological (Lee and Vleeming in Lee 2004) (figure 8). It is essential to be aware of how the passive (bones, ligaments, joints, IVD and capsules), the active components (muscles and their fascia) and the coordination provided by the neural system, co-operate in functional tasks. Needless to say that emotions and awareness have a great influence on the moving patterns of individuals which can create tissue loading. This is important to understand the vigour of assessment and treatment procedures described in this essay.

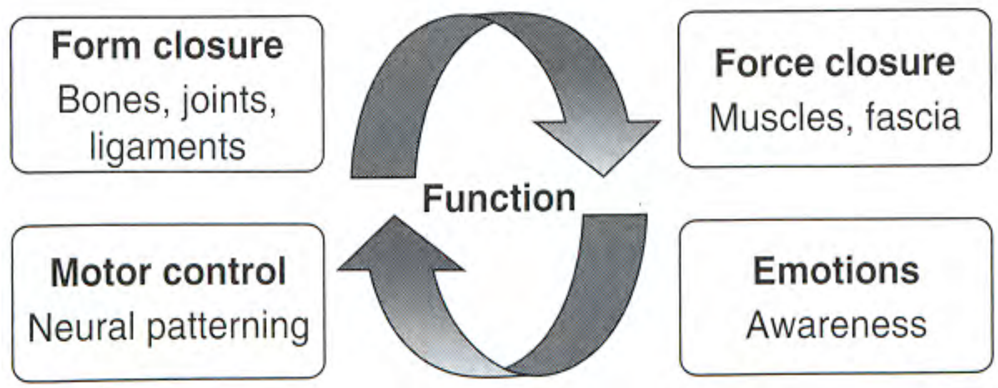



***Figure 7:  The integrated model of function (modified from Lee 2004, pp. 42)***

**<u>Form closure. Critical issues</u>**

- The SIJ receive 60% of the total body weight (Sniiders et al 1993). They are flat joints and have the ability to transfer great loads but are vulnerable to shearing subluxing forces (DonTingny 1985, Sniiders et al 1993).
- The ligamentous apparatus of the SIJ has a dynamic role in its stability (Gerlach 1992). Ridges and depressions are observed on the joint surfaces. They are considered as normal in these synovial joints since they are present even in 13 old boys. Their function is considered to increase friction therefore stability (Vleeming et al 1990). The banding mode of an arch, the increased friction and the muscles and ligaments perpendicular to the joint all form the “SIJ self bracing mechanism” (figure 9) (Sniiders et al 1993a, 1993b).

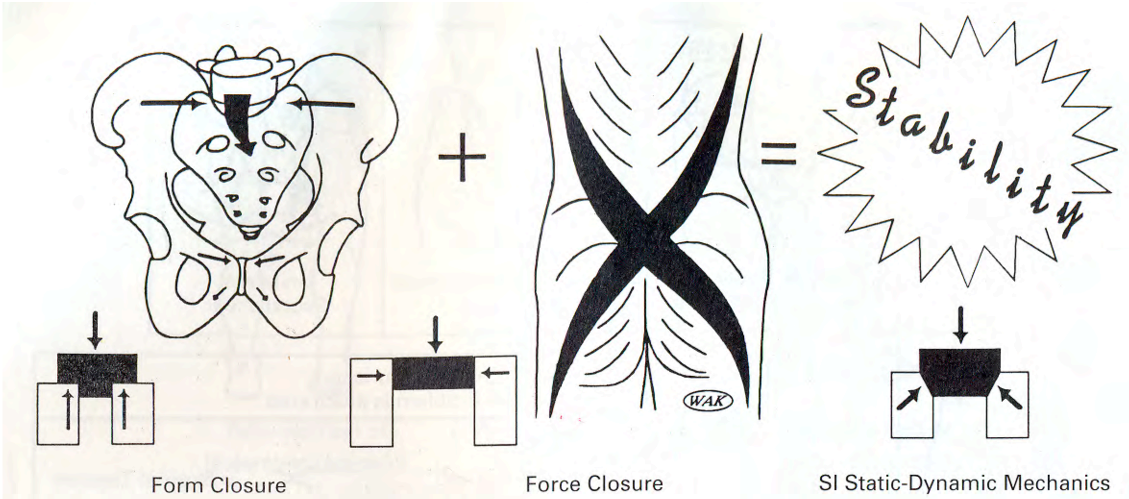



***Figure 9:*** ***The summary of the form and force closure mechanisms (modified from Snijders et al, in Vleeming ey al 1997, pp.106)***

- The iliolumbar ligament (ILL) is considered as a very important stabilizer of the lumbosacral junction. In lumbar lateral flexion it is the ipsilateral ligament and during flexion and extension the bilateral ligaments (Yamamoto et al 1990).
- It is of doubt if the pelvic ring is of any importance in loading SIJ through the limbs thus symphisiodesis is not essential for SIJ stability (Sniiders et al 1993a, 1993b).

**Force closure. Critical issues**

Joint compression before and during motion movement is essential for the stability of the lumbopelvic-hip region (Lee 2004). This is achieved by muscle action, the tensile forces applied on other passive elements such as fascias, ligaments and the ground reaction forces. There are two types of muscles attaching to the lumbopelvic-hip region. The local and global stabilizers (Lee 1998, 2004)

*Local stabilizers* are the muscles that fit the criteria for classification as local stabilizers. These muscles are the pelvic floor, the TrA, the diaphragm, and the deep fibers of MF (figure 10) (Lee 2004). Recently some others have been added to the list, like deep fibers of psoas, medial fibers of QL (McGill 2002) and the lumbar parts of the lumbar iliocostalis and longissimus and the posterior fibers of IO (O'Sullivan 2000).

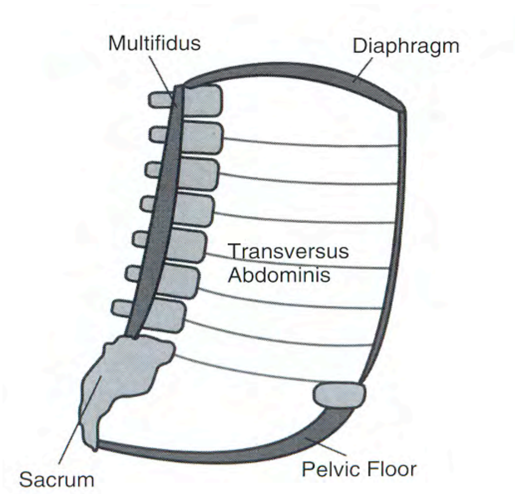



***Figure 9:*** ***Contraction of the local system increases the intra-abdominal pressure. That way contributes to trunk stabilization and intrapelvic viscera protection during functional tasks. Interestingly the valsava maneuver relaxes the pelvic floor, thus urinary bladder is stressed (modified from Lee 2004, pp. 47)***

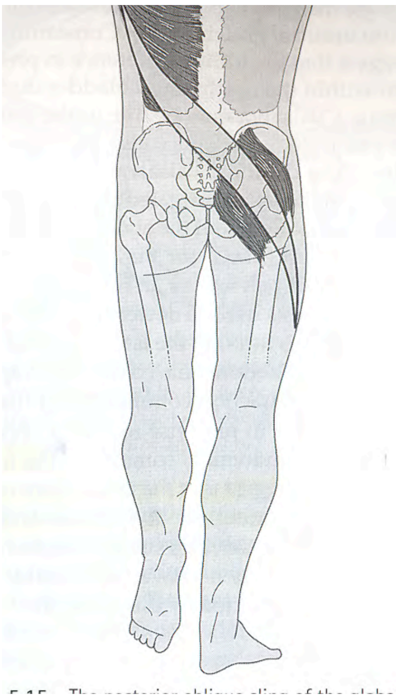
Stability is achieved through several mechanisms like:

- Increasing the intraabdominal pressure (Gardner-Morse and Stokes 1998, Richardson et al 2002)
- Increasing the tension of the TDF (O'Sallivan et al 1997, Richardson et al 2002)
- Increasing the articular stiffness (Hodges et al 2003, Wilke et al 1995)

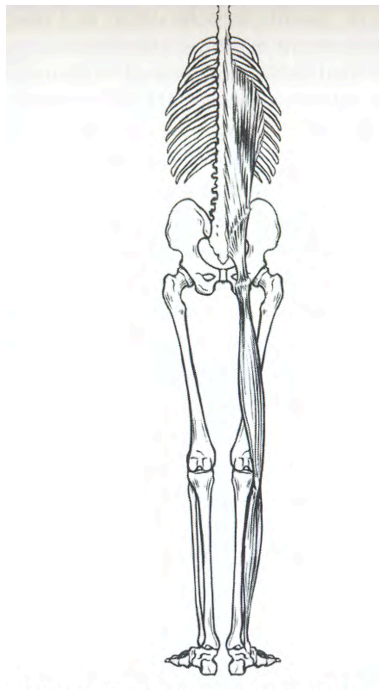
Research has shown (Hodges 2000, Hodges and Gandevia 2000, Kaigle et al 1995, Sapsford 2004) that these muscles act tonically during repetitive tasks and that they should work at low levels at all times and increase their action before any further motion or load occurs. LBP patients compared with controlled groups have a delay in deep muscles activation (Hides et al 1994, Hodges end Richardson 1996).

*Global stabilizers* have been classified into four functional slings (Vleeming et al 1995a, 1995b, Sniiders et al 1993a).

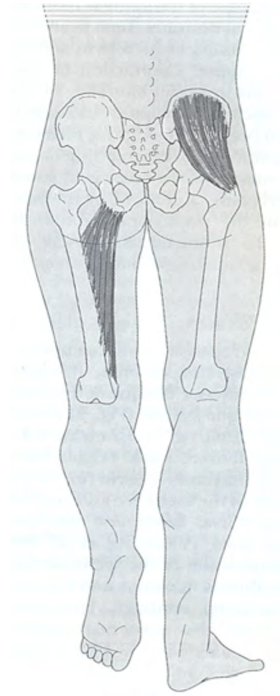
- The posterior oblique sling (figure11.1)
- The longitudinal sling (figure11.2)
- The lateral sling (figure11.3)
- The anterior oblique sling (figure11.4)



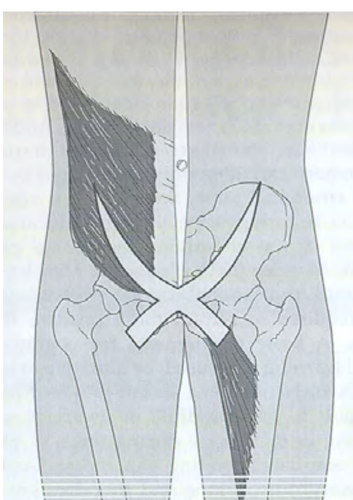
***11.1***



***11.2***



***11.3***



***11.4***

***Figure 11.1-11.4)***
***1) The posterior oblique sling is formed by the action of GlutMax, BF, the contralateral LatDorsi and the intervening TDF, 2) the longitudinal fascia connects the peroneii, the BF, the STL, the deep lamina of the TDF and the ipsilateral ES muscles, 3) The lateral sling is formed by the action of the GlutMed and the contralateral Adductors, 4) The anterior oblique sling is formed by the EO the contralateral IO and Adductors and the intervening anterior abdominal fascia (modified from Lee 2004, pp. 2-3)***

These muscles act phasic. None of these muscles cross the SIJ however they effect joint compression. Research has shown that these muscles produce forces that spread beyond their origins and insertions. Their action is transmitted to other muscles, tendons, ligaments, capsules, fasciae and bones that lie in series or parallel to the active muscle. It is also noticed that LBP sufferers compared with controls have weak key muscles and/or wrong coordination of the slings (Comerford and Mottram 2001, Hides et al 1994, Hodges and Richardson 1996, O'Sallivan et al 1997, Richardson et al 2002).

# Examination of the lumbopelvic-hip region

As manipulative physiotherapy students that we are, we all have developed advanced examination skills throughout this year. The purpose of this essay is not to repeat the knowledge gained. Moreover, the reliability, the specificity and validity of most of the examination procedures oriented on the integrated model of function are controversial throughout the literature. Therefore this chapter compromises of dot points and summaries of the most valid literature concerning examination procedures. It also includes observations of high quality studies that will help the reader to form advanced-multiple hypotheses of the contributing factors and impairments that may characterize the PIS. Taking into consideration how compound this syndrome can be, some of the discussions are not directly orientated on the dysfunctional psoas.

## Subjective examination

- Asking patients to *draw their pain patterns* is considered an accurate indicator to identify those that are not likely to have SIJ contribution to their symptoms.

- Clinically the *Fortin Finger Test* seems a sensitive predictor for SIJ dysfunction (Dreyfuss et al 1996, Fortin and Falco 1997). In this test the examiner asks the patient to point with his finger the most painful spot on their back. If the spot is exactly medial to the PSIS, the test is positive. However, little evidence supports this test.

- *History:* A traumatic like fall on the buttock or MVA should cause alert for possible SIJ dysfunction (Fortin et al 1997) especially with adult females if they state PPPP (Kristiansson 1997, Ostgaard 1997).

**Contributing factors**

- A clinical prospective cohort study showed significant decrease in CSA of Psoas in the side of disk herniation in subjects with sciatica (Dangaria and Naesh 1998). Past upper lumbar pathology or L5-S1 disk pathology via sacral autonomic plexus through the white rami communicates can stimulate L1-L3 motor fibres which can cause chronic psoas contraction and tightness (Cibulka et al 1986).
- Previous articular pillar stress fracture in young dancers (Cibulka et al 1986).
- Exercise emphasizing hip flexion like cycling, running up hills (Hammer 1992)
- Chronic pain leads to decreased motor control and proprioception (Kaigle et al 1998, Nelson et al 1995, Taimela et al 1999,). Tight Iliopsoas produces reciprocal inhibition of GM compromising more the intra-pelvic instability (Lee 2004).
- It is clinically observed that athletes with strained hamstrings always show an anterior pelvic tilt which may put more stress on the hamstring muscle contributing to psoas insufficiency (Bachrach 1997) (expert's opinion).
- Repetitive ankle sprains (i.e. footballers) are a serious contributing factor to LBP and SIJ dysfunction. Bullock-Saxton and colleges (1993, 1994) measured significant increased reaction time of the GM of the sprained limb in healthy subjects compared with the unaffected limb. LBP subjects compared with healthy uninjured subjects had decreased firing of GM on both limbs. This influences the force closure of the SIJ via the posterior oblique sling and longitudinal slings (Vleeming 1997).
- Hip Extensors hypertonia/tightness (piriformis). The limb is externally rotated. Right piriformis insufficiency leads to SIJ dysfunction; usually a L/L axis rotation. SIJ dysfunction contributes to more asymmetrical stress on the lumbar IVD and especially the L4 and L5 (expert's opinion), (Bachrach 1997, Lee 2004).
- Tight hamstrings (due to prolonged elongation) can impair more the SIJ via STL attachment
- HL/R impedes dorsi flexion of the first MTFJ. During gait, complete hip extension is not possible leading to PIS (expert's opinion), (Dananberg 1997)

**Clinical presentation of PIS (level 6 of evidence; expert's opinion)**

- Gradual or sudden increase on set of symptoms.
- Usually observed in people with sedentary or very active life style like gymnastics athletes, trainers, aerobic instructors etc.
- Thoracolumbar, lower lumbar, anterior hip, buttock pain or a combination of symptoms may be present. They can be bilateral or unilateral.
- If symptoms exist midline then disk pathology should also be considered (Donelson et al 1997). Symptoms should not spread more distal than the knee. However if increased tension and trigger points on STL, SSL and piriformis exist, pain may refer to the calf via satellite trigger points.
- May also have low back pain on associated side and contralateral upper back and shoulder via posterior oblique sling active TrPs (Baldry 2005, Vleeming 1997).
- Usually asymmetrical psoas tightness exists, often on the right. In this case we will also observe a "psoatic lumbar rotoscholiosis" with the convex away from the side of dysfunction (Bachrach 1997). Based on the anatomy of psoas this observation makes sense. Therefore, IVDs are loaded asymmetrically.

## Physical examination-Important considerations

**Lumbar spine**

- *Spinal Mobility* assessment unless restriction is present in more than 3 planes can not predict outcome for LBP sufferers (Thomas et al 1998).
- *Instability* might co-exist with PIS. O'Sullivan (2000, 2006) established a high reliable system to identify lumbar instabilities.
- The "*lateral lumbar shift*" and "*lateral side bend*" tests for the lumbar spine are objective tools to diagnose lumbar dysfunction and guide treatment (systematic review), (Tenhula et al 1990)

- The McKenzie centralisation/peripheralisation examination method to differentiate discogenic from non-discogenic pain is reliable and accurate to MRI (prospective experimental study), (Donelson 1997). A very recent concurrent criterion-related validity study design concluded high reliability for both the McKenzie method and specific SIJ provocation tests. Specificity, sensitivity and positive likelihood ratio (95% CI) was 91%, 83% and 6.97 respectively. The diagnostic accuracy of the clinical examination and clinical reasoning process was superior to the SIJ provocation tests alone (Laslett et al 2003).
- Patient's *respiration patterns* and functional abilities should be assessed and addressed when dealing with posture defects (Hodges and Gandevia 2000)

**Sacroiliac joints**

- ***Palpation of bony landmarks****.* There is controversy among the literature concerning the reliability of pelvis bony landmarks and prediction of pelvis dysfunction. Old studies with blinded examiners support the reliability of bony palpation (Gajdosik et al 1985, Smith et al 1988, Subotnic 1976, Walker et al 1987). More recent studies dissentient these results (Cummings and Crowell 1988, Dreyfuss et al 1996, Freburger and Riddle 1999, Kelly 1995, Smith et al 1988). However two systematic reviews (Mann et al 1984, Harrison et al 1997) concluded high agreement between experienced physios. Moreover, a very recent experimental study (Haire and Gibborns 2000) concluded high intra-examiner but low inter-examiner reliability. Therefore palpation is considered an important tool in manual therapy, especially for experienced clinicians. Further studies are required to determine why agreement on both static and motion palpatory findings remain poor.

- Howeve*r* **SIJ** mobility is not a reliable predictor of SIJ pathology, **asymmetry** is (Buyrak et al 1997, Sturesson 1997). This implicates that during the Stork test, SIJ joint play testing etc, we should assess asymmetry and not mobility! Other studies suggest as well that mobility of the SIJ is very small to be detected via clinical tests (Sturesson 2000). A prospective cohort study measured the diagnostic value of SIJ asymmetry detection (sensitivity 65%, specificity 83%, and positive predictive value 77%). (Damen et al 2002).

- ***Pain provocation tests*** are source of controversy between examiners (Dreyfuss et al 1994, Potter and Rothstein 1985, Strender et al 1997). Cross sectional blinded studies proved that:
    - distraction test
    - compression test
    - POSH test (or thigh thrust test) and
    - sacral shear test,

  are reliable tools to detect SIJ pathology (Laslett 1997, Laslett and Williams 1994). Two tests must at least be positive to diagnose SIJ pathology (Laslett et al, article in press). The Gaenslen's and POSH tests show the highest inter-intra examiner reliability (Wurff et al 2000a, 2000b) (systematic reviews). A more recent cross sectional study concluded that the POSH test is the most sensitive and the distraction test is most specific to detect SIJ pathology (Laslett et al, article in press); however, by taking into consideration what happens to the PS, via the pelvic ring during the distraction test, I would argue that such a test is 100% specific to SIJ only.

- ***Thomas test:*** *V*ery tight psoas/hip flexors complex (20-30 degrees above transverse plane). Tenderness with palpation over psoas may revel in this position. This test introduces an extreme intrapelvic movement which may provoke SIJ symptoms. We should also consider lumbar disk anterior pathology (L2, L3, L4 and less common L5) (Vleeming 1997).

- ***Trigger points*** (primary and secondary) on QL, IO, Piriformis, TFL, Psoas, Iliacus, adductors, gluteal and peroneal muscles are often present and its management with dry needling is essential in symptom release. Satellite TrP down the leg, often present in non specific LBP, mimic sciatica symptoms. This must be kept always in mind when assessing LBP patients (Baldry 2005). Moreover, active TrP in the hams belies can give positive PSLR test (Simons and Travell 1984).

- ***Neuro-dynamic testing****.* Fahrni (1966), DonTigny (1985) and Bohannon et al (1985) observed that during **PSLR** innominate, posterior rotation initiates when the limb is approximately 1-1.5 cm lifted from the bed. The contralateral innominate tends to anteriorly rotate relative to the ipsilateral where during PSLR an intrapelvic torsion is produced. The sacrum follows the movement (see gait biomechanics) and a torsional momentum is produced to the L5-S1 and L4-L5 segments. Therefore, when interpreting pain responses not only should we consider neural structures but also contralateral SIJ or disk pathology. The same researchers measured that until:
    - 30-40º of PSLR the slack of neural tissue is taken up
    - *60-70*º there is no other neural tissue slide
    - *80-90*º other soft tissues are tightened like TDF etc

  Experts also suggest that a deference of only 10° between R1 and R2 during the PSLR is most suggestive of SIJ pathology. The **crossed PSLR test** is more specific for medial disk prolapse, but less sensitive than the ipsilateral PSLR (Urban 1981). Consequently tight hamstrings (common in PIS) could confuse the results via its attachment with STL and sacrum (DonTigny 1985, 1997).

- ***Active forward bending*:** Experimental EMG studies have proved that forward flexion and intersegmental mobility is significantly decreased in LBP sufferers thus they are objective tools to measure impairment in LBP (Kaigle et al 1998, Waddell et al 1992). However during trunk flexion, tight hamstrings via the longitudinal sling tend to unlock the SIJ. That may lead to instability and pain (Wingerden et al 1997). This is why we should be careful in interpreting results from WB active tests (Sniiders 1993a, 1993b). DonTigny (1985) demonstrated that pain coming from SIJ during the forward flexion test is very common. This is because sacrum widens anteriorly and shortens posteriorly and therefore tends to spread the innominates, increases ligamentous tension and produces pain.

- The ***ASLR test*** has proved by far the most reliable tool to detect dysfunction at the lumbopelvic-hip region (Mens et al 1997, 2002). A cross sectional study suggested a six point scale to be graded by the patient, (from 0-5) for each leg, grading his ability to lift the leg (how heavy it felt etc). The summary score of both legs (excellent = 10) reflects how the patient effectively transfers load from the legs to the pelvis. This is why it is an objective tool to measure disability and improvement (Mens et al 2001). Lee (1997, 2004) suggests we should apply pressure to the pelvis in various directions during this test, mimicking the local and global stabilizers action. This will help to determine which of the stabilizing systems is compromised and to also choose the most appropriate treatment.

- **Muscle tests**: depended on the overall pathology various muscle can be found weak. Two muscles that is likely to be found weak in any LBP patient from caused by PIS, related with SIJ dysfunction are:
    - the ipsilateral GM (to the side of Psoas insufficiency)
    - adduction. In a cross-sectional analysis study (Mens et al 2002) it was suggested that adductors weakness, in intra-pelvic dysfunction, appears to be caused by the inability to use the hip muscles rather than true muscle weakness; and it is a reliable tool to measure severity of dysfunction.

- ***Motor control***: Intersegmental motion, trunk biomechanics and lumbopelvic rhythm alter significantly in LBP sufferers (Hungerford et al 2003, McClure 1997, Nelson et al 1995, Taimla et al 1999). Altered activity of local and global stabilizers has also been demonstrated in LBP (Hides et al 1994, Hodges and Gandavera 2000, Hodges and Richardson 1996, Hodges et al 2003, O'Sallivan 1997, Richardson et al 2003). This suggests that motor control should be assessed and addressed respectively to optimize the lumbopelvic-hip region function in PIS.

- ***Imaging****:* The most useful plain radiographic view of the SIJ is 30 degrees cephalad tilt view (Berrard 1997). However imaging studies can not determine wether SIJ is causing pain or not. X-Rays are superior to evaluate diverticula of the SIJ capsule whereas posterioarthrography-CT is superior to evaluate anterior capsule pathology (Fortin et al 1997). Pelvic girdle real-time ultrasonography is widely accepted as a means of evaluation and treatment of the lumbopelvic-hip region dysfunction (Hides et al 1995, Whittaker 2004).

## Clinical diagnosis

Clinical diagnosis should never rely on one test alone (Rebecca 1995). A thorough examination is needed to make an accurate clinical diagnosis. In one prospective, blinded study by a manipulative physiotherapist of 30 years experience showed within manipulative clinical tests for lumbopelvic-hip dysfunction in low back patients, high agreement with available reference standards (Laslett et al 2005). Another prospective study highlighted the importance of history taking when dealing with SIJ dysfunction syndromes (Dreyfuss et al 1996).

# Management

The above mentioned principles are essential in the assessment, diagnosis and management of psoas insufficiency. The selection of treatment techniques in such a multidimensional syndrome depends on the individual needs of patients. Table 2 represents a brief guideline with the basic principles that should characterize the treatment approach.

***Surgery:*** Surveys suggest that immediate lumbar surgery is only required for midline disk rupture which causes dysfunction of the bowel and bladder (Weber 1983).

SIJ arthrodesis is a safe way of treatment (Lippit 1997), however it should be used when SIJ has accurately been diagnosed as the only symptomatic stracture and all other conservative management has failed (Moore 1997, Keating et al 1997).

Margulies end colleges (1997) characteristically state: *"Although spinal arthrodesis is widely practiced, it should be appreciated that fusion is a crude solution to the malfunction of sophisticated anatomic mechanisms that occur in nature. Artificial disks and ligaments may become the state of the art in future lumbosacral surgeries".*

| Treatment approach | Technique application |
|---|---|
| **Restore form closure (mobility/allignment)**<br>• **Lumbar spine/thoracic spine and ribs**<br>• **SIJ**<br>• **Hip joint** | • **Passive mobilization**<br>• **Mobilization with movement**<br>• **Manipulation** |
| **Use sacroiliac belts or taping to ensure stability** | • **The ASLR is used to determine exactly where and how much compression is needed** |

| | |
|---|---|
| **Prolotherapy** | • **When there is loss of form closure and the local system can not apply sufficient compression to stabilize the pelvis joints under load** |
| **Restore breathing patterns** | • **Oscillate diaphragm**<br>• **Correct asymmetries**<br>❖ **Inhibit overactive/tight muscles (usually RA, IO, EO, ES)**<br>  ➢ **Myofascial release**<br>  ➢ **Re-educate with verbal and manual cues**<br>  ➢ **Dry needling (EO, IO, ES, SM, GM, QL, IC)**<br>❖ **Stretch tight muscles (EO, IO, ES, LatDorsi, RA)** |
| **Restore force closure (motor control)** | • **Re-educate neutral position for all joints**<br>• **Oscillate local stabilizers (MF, TrA, Pelvic floor)**<br>❖ **Educate**<br>❖ **Use real-time ultrasound**<br>• **Coordinate global stabilizers (proprioception exercises)**<br>❖ **Start from supine with ASLR**<br>❖ **Progress to WB activities**<br>❖ **Inhibit overactive/tight muscles**<br>  ➢ **Myofascial release**<br>  ➢ **Correct "butt grippers"**<br>  ➢ **Massage**<br>  ➢ **Dry needling (PF, OI, LD, Gluteals, TFL)**<br>❖ **Stretch tight muscles (psoas, iliacus, Gluteals, Hamstrings, Adductors, RF, TFL,-ITB, PF** |
| **Endurance training for the local system** | • **10 reps of 10 sec duration each – integrate with breathing**<br>• **Progress to integrate with upper/lower limb exercises**<br>• **More progress to DLAs**<br>• **Advanced progress to sport specific activities (use Swiss balls, thera-bands, pulley machines)** |
| **Address other contributing factors** | • **Ankle joint problems**<br>• **Knee joint complex**<br>• **Shoulder problems**<br>• **Sporting equipment**<br>• **Technique**<br>• **Other health issues (urinary incontinence in female athletes etc)**<br>• **Yellow flags (psychosocial factors)** |

***Table 2: Treatment guideline for the management of Psoas insufficiency and SIJ dysfunction***

# Conclusions

❖ Psoas insufficiency syndrome is a multifactorial pathology which perpetuates through chronic shortening, often asymmetric, of the psoas major muscle. It is commonly associated with weakness of this muscle, the abdominals and the gluteals, and hamstring tightness.

- ❖ Anterolateral disc pathology at L1, L2 and/or L3 can be result of chronic psoas shortening, or reversed ways can be the cause of psoas shortening due to direct pressure on the muscle belly or through reflex excitation of motor nerves.

- ❖ Anterolateral pathology of the L5-S1 IVD can also excite the sympathetic fibers of the sacral autonomic plexus, which, through the white rami communicantes may stimulate L1, L2 and L3 motor nerves; thus result in chronic asymmetric psoas contraction.

- ❖ Low back pain may initiate the PIS due to inhibition-imbalances of muscles and movement patterns. From the other hand PIS may initiate low back pain.

- ❖ SIJ dysfunction may contribute to LBP following chronic PIS or reversed ways and so on.

- ❖ Multiple previous lower limb injuries or repetitive microtraumas in athletic populations such as ballet dancers and footballers can be significant contributors to PIS, LBP and/or SIJ dysfunction.

- ❖ Unfortunately when someone is seeking our help he is usually already in pain. Because all the above happen progressively until the initiation of symptoms, we usually have to deal with all these pathological entities. A very detailed history combined with a thorough examination, based on the integrated model of function, is the key to rehabilitation and successful return to sport.

- ❖ Thus, we should always investigate for pelvic dysfunction in low back pain patients. High quality literature supports that there are reliable diagnostic tools to detect abnormalities and accelerate recovery.

# References


1. Adams MA, Dolan P (1995): Recent advances in lumbar spinal mechanics and their clinical significance. *Clinical Biomechanics* 10(1): 3-19

2. Bachrach RM (1997): Psoas dysfunction/insufficiency, sacroiliac joint dysfunction and low back pain. In Leeming A, Mooney V, Dorman T, Snider C, Stocker R*: Movement, Stability and Low Back Pain*. Churchill Livingstone, pp. 309-318

3. Barker PJ, Briggs CA (1999): Attachments of the Posterior Layer of Lumbar Fascia. *Spine* 24(17): 1757-1764

4. Baldry PE (2005): Chapter 17: Low Back Pain in: Acupuncture, Trigger Points and Musculoskeletal Pain. Third Edition, Elsevier Churchill Livingstone, pp. 275-314

5. Baldry PE (2005): Chapter Abdominal and Pelvic Pain in: Acupuncture, Trigger Points and Musculoskeletal Pain. Third Edition, Elsevier Churchill Livingstone, pp. 343-358

6. Bernard TN (1997): The role of sacroiliac joints in low back pain: basic aspects of pathophysiology and management. In Leeming A, Mooney V, Dorman T, Snider C, Stocker R*: Movement, Stability and Low Back Pain*. Churchill Livingstone, pp. 73-88

7. Black KM, McClure P, Polansky M (1996): The Influence of Different Sitting Positions on Cervical and Lumbar Posture. *Spine* 21(1): 65-70

8. Bohannon R, Gajdosik R, LeVeau BF (1985): Contribution of Pelvic and Lower Limb Motion to Increases in the Angle of Passive Straight Leg Raising. *Physical Therapy* 65(4): 474-477

9. Bohannon R, Gajdosik R, LeVeau BF (1985): Contribution of Pelvic and Lower Limb Motion to Increases in the Angle of Passive Straight Leg Raising. *Physical Therapy* 65(4): 474-477

10. Bullock-Saxton JE, Janda V, Bullock MI (1993): Reflex Activation of Gluteal Muscles in Walking. An approach to restoration of muscle functions for patients with low-back pain. *Spine* 18(6): 704-708

11. Bullock-Saxton JE, Janda V, Bullock MI (1994): The Influence of ankle Sprain Injury on Muscle Activation during Hip Extension. *International Journal of Sports Medicine* 15:330-334

12. Buyruk HM, Stem HF, Snider CF, Leeming A, Lameris FS, Holland WPF (1997): Measurement of sacroiliac joint stiffness with color Doppler imaging and the importance or asymmetric stiffness in sacroiliac pathology. In Leeming A, Mooney V, Dorman T, Snider C, Stocker R*: Movement, Stability and Low Back Pain*. Churchill Livingstone, pp. 297-307

13. Cibulka MT (1992): The Treatment of the Sacroiliac Joint Component to Low Back Pain: A Case Report. *Spine* 72(12): 917-922

14. Cholewicki J, Crisco JJ, Oxland R, Yamamoto I, Panjabi MM (1996): Effects of Posture and Structural 3 Diamensional Coupled Rotations of the Lumbar Spine. A biomechanical analysis. *Spine* (21): 2421-2428

**15.** Comerford MJ, Mottram SL (2001): Movement and stability dysfunction – contemporary developments. *Manual Therapy* 6(1): 15-26

**16.** Corn R (2002): Putting the maximus back into your gluteus. Common causes of disruption in neuromuscular efficiency. C:\Documents and Settings\Stelios\Desktop\ROM-DEEP STABILIZERS OF THE TRUNK\Putting the MAXIMUS Back in your GLUTEUS Pt_ 2.htm. *Copyright National Academy of Sports Medicine 2002.* Accessed on 16/08/2005

**17.** Cottingham JT, Porges SW, Richmond K (1988): Sifts in Pelvic Inclination Angle and Parasympathetic Tone Produced by Roling Soft Tissue Manipulation. *Physical Therapy* 1364-1370

**18.** Cresswell AG, Thortsensson A (1994): Changes in intra-abdominal pressure, trunk muscle activation and force during isokinatic lifting and lowering. *European Journal of Applied Physiology* 68: 315-321

**19.** Cummings GS, Crowell RD (1988): Source of Error in Clinical Assessment of Inominate Rotation. A special communication. *Physical Therapy* 68(1): 77-79

**20.** Damen L, Buyrak HM, Gyler-Uysal F, Lotgering FK, Snider CJ, Stem HJ (2002): The Prognostic Value of Asymmetric Laxity of the Sacroiliac Joints in Pregnancy-Related Pelvic Pain. *Spine* 27(24): 2820-2824

**21.** Damen L, Spoor CW, Snider CJ, Stem HJ (2002): Does a pelvic belt influence sacroiliac joint laxity? *Clinical Biomechanics* 7: 495-498

**22.** Dananberg HF (1997): Lower back pain as a gait-related repetitive motion injury. In Leeming A, Mooney V, Dorman T, Snider C, Stocker R*: Movement, Stability and Low Back Pain*. Churchill Livingstone, pp. 253-267

**23.** Dangaria TR, Naesh O (1998): Changes in Cross-Sectional Area of Psoas Major Muscle in Unilateral Sciatica Caused by Disc Herniation. Spine 23(8): 928-931

**24.** Donelson R, Aprill C, Medcalf R, Grant W (1997): A Prospective Study of Centralization of the Lumbar and Referred Pain. A predictor of symptomatic discs and annular competence. *Spine* 22(10): 1115-1122

**25.** DonTigny R (1990): Anterior Dysfunction of the Sacroiliac Joint as a Major Factor in the Etiology of Idiopathic Low Back Pain Syndrome. *Physical Therapy* 70: 250-265

**26.** DonTigny R (1985): Function and Pathomechanics of the Sacroiliac Joint. A Review. *Physical Therapy* 65(1): 35-43

**27.** DonTigny RL (1997): Mechanics and treatment of the sacroiliac joint. In Leeming A, Mooney V, Dorman T, Snider C, Stocker R*: Movement, Stability and Low Back Pain*. Churchill Livingstone, pp. 461-476

**28.** Dreyfuss P, Michaelsen M, Pauza K, McLarty J, Bogduk N (1996): The Value of Medical History and Physical and Physical Examination in Diagnosing Sacroiliac Joint Pain. *Spine* 21(22): 2594-260

**29.** Dreyfuss P, Dreyer S, Griffin J, Hoffman J, Walsh N (1994): Positive Sacroiliac Screening Tests in Asymptomatic Adults. *Spine* 19(10): 1138-1143

**30.** Ebraheim NA, Mekhail AO, Wiley WF, Jackson WT, Yeasting RA (1997): Radiology of the Sacroiliac Joint. *Spine* 22(8): 869-876

**31.** Esola M, McClure PW, Fitzgerald GK, Siegler S (1996): Analysis of Lumbar Spine and Hip Motion During Forward Bending in Subjects with and without a History of Low Back Pain*. Spine* 21(1): 71-78

**32.** Fahrni WH (1966): Observations on straight leg-raising with special reference to nerve root adhesions. *Canadian Journal of Surgery* 9: 44-48

**33.** Fortin JD, Aprill CN, Ponthieux B, Pier J (1994): Sacroiliac Joint: Pain Referral Maps Upon Applying a New Injection/Arthrography Technique. Part II: clinical evaluation. *Spine* 19(13): 1483-1489

**34.** Fortin JD, Dwyer AP, West S, Pier J (1994): Sacroiliac Joint: Pain Referral Maps Upon Applying a New Injection/Arthrography Technique. Part I: Asymptomatic volunteers. *Spine* 19(13): 1475-1482

**35.** Fortin JD, Falco FJE (1997): The Fortin Finger Test: an Indicator of Sacroiliac Pain. *The American Journal of Orthopedics* pp. 477-480

**36.** Fortin FD, Pier F, Falco F (1997): Sacroiliac joint injection: pain referral maping and arthrographic findings. In Leeming A, Mooney V, Dorman T, Snider C, Stocker R*: Movement, Stability and Low Back Pain*. Churchill Livingstone, pp. 271-285

**37.** Freburger JK, Riddle DL (1999): Measurement of Sacroiliac Joint Dysfunction: A Multicenter Intertester Reliability Study. Research Report. *Physical Therapy* 79(12): 1134-1141

**38.** Gajdosik R, Simpson R, Smith R, DonTigny RL (1985): Pelvic Tilt. Intertester Reliability of Measuring the Standing Position and Range of Motion. *Physical Therapy* 65(2): 169-17

**39.** Garner-Morse MG, Stokes IAF (1998): The Effects of Abdominal Muscle Coactivation on Lumbar Spine Instability. *Spine* 23(1): 86-92

**40.** Garrison DE, Harrison DD, Troyanovich SJ (1997): The Sacroiliac Joint: a Review of Anatomy and Biomechanics with Clinical Implications. *Journal of Manipulative and Physiological Therapeutics* 20(9): 607-617

**41.** Gerlach UJ, Lierse W (1992): Functional Construction of the Sacroiliac Ligamentous Apparatus. *Acta Anatomica* 144: 97-102

**42.** Greenman PE (1997): Clinical aspects of the sacroiliac joint in walking. In Leeming A, Mooney V, Dorman T, Snider C, Stocker R*: Movement, Stability and Low Back Pain*. Churchill Livingstone, pp. 235-242

**43.** Hammer W (1995): Iliopsoas. *Dynamic Chiropractic* 13(24). C:\Documents and Settings\Stelios\Desktop\ROM-DEEP STABILIZERS OF THE TRUNK\Soft Tissue.htm. Accessed on 16/08/2005

**44.** Heliovaara M, Sievers K, Impivaara O, Maatela J, Knekt P, Makela M, Aromaa A (1989): *Annals of Medicine* 21: 327-337

**45.** Hesch F (1997): Evaluation and treatment of the most common patterns of sacroiliac joint dysfunction. In Leeming A, Mooney V, Dorman T, Snider C, Stocker R*: Movement, Stability and Low Back Pain*. Churchill Livingstone, pp. 535-545

**46.** Hides JA, Richardson CA, Jull GA (1996): Multifidus Muscle Recovery is not automatic after Resolution of Acute, First-Episode Low Back Pain. *Spine* 21(23): 2763-2769

**47.** Hides JA, Stokes MJ, Saide M, Jull GA, Cooper DH (1994): Evidence of Lumbar Multifidus Muscle Wasing Ipsilateral to Symptoms in Patents with Acute/Subacute Low Back Pain*. Spine* 19(2): 165-172

**48.** Hindlle RJ, Pearchy MJ, Cross AT, Miller DHT (1990): Three-dimentional Kinematics of the human back. *Clinical Biomechanics* 5: 218-228

**49.** Hodges PW (2000): The Role of Motor System in Spinal Pain: Implication for Rehabilitation of the Athlete Following Lower Back Pain. *Journal of Science and Medicine in Sport* 3(3): 243-253

**50.** Hodges PF, Gandevia SC (2000): Changes in intra-abdominal pressure during postural and respiratory activation of the human diaphragm. *Journal of Applied Physiology* 89: 967-976

**51.** Hodges PW, Gandevia SC (2000): Activation of the human diaphragm during a repetitive postural task. *The Journal of Physiology* 522(1): 165-175

**52.** Hodges P, Holm AK, Holm S, Ekstrom L, Cresswell A, Hansson T, Thortsensson A (2003): Intervertebral Stiffness of the Spine is Increased by Evoked Contraction of Transversous Abdominis and the Diaphragm: In Vivo Porcine Studies*. Spine* 28(23): 2594-2601

**53.** Hodges PW, Moseley GL (2003): Pain and motor control of the lumbopelvic region: effect and possible mechanisms. Journal *of Electromyography and Kinesiology* 13: 361–370

**54.** Hodges PW, Richardson CA (1996): Inefficient Muscular Stabilization of the Lumbar Spine Associated with Low Back Pain. *Spine* 21(22): 2640-2650

**55.** Hungerford B, Gilleard W, Hodges P (2003): Evidence of Altered Lumbopelvic Muscle Recruitment in the Presence of Sacroiliac Joint Pain. *Spine* 28(14): 1593-1600

**56.** Huson A (1997): Kinematic models and the human pelvis. In Leeming A, Mooney V, Dorman T, Snider C, Stocker R*: Movement, Stability and Low Back Pain*. Churchill Livingstone, pp. 123-131

**57.** Inufusa A, An HS, Lim TH, Hasegawa T, Haughton VM, Nowicki B (1996): Anatomical Changes of the Spinal Canal and Intervertebral Foramen, Associated with Spinal Flexion-Extension Movement. *Spine* 21(21): 2412-2420

**58.** Irvin RE (1997): Suboptimal posture: the origin of the majority of idiopathic pain of the musculoskeletal system. In Leeming A, Mooney V, Dorman T, Snider C, Stocker R*: Movement, Stability and Low Back Pain*. Churchill Livingstone, pp. 133-155

**59.** Kaigle AM, Holm SH, Hansson TH (1995): Experimental Instability in the Lumbar Spine. *Spine* 20(4): 421-430

**60.** Kaigle AM, Wessberg P, Hansson TH (1998): Muscular and Kinematic Behaviour of the Lumbar Spine During Flexion-Extension. *Journal of Spinal Disorders* 11(2): 163-174

**61.** Keating FG, Avillar MD, Price M (1997): Sacroiliac joint arthrodesis in selected patients with low back pain. In Leeming A, Mooney V, Dorman T, Snider C, Stocker R*: Movement, Stability and Low Back Pain*. Churchill Livingstone, pp. 573-586

**62.** Kelly KD (1995): The Relationship of Pelvic Girdle Asymmetries and Low Back Pain. *Hons Thesis University of North Carolina*, Chapel Hill

**63.** Kissling RO, Facob HAC (1997): The mobility of sacroiliac joints in healthy subjects. In Leeming A, Mooney V, Dorman T, Snider C, Stocker R*: Movement, Stability and Low Back Pain*. Churchill Livingstone, pp. 177-185

**64.** Kristiansson P (1997): S-Relaxin and pelvic pain in pregnant women. In Leeming A, Mooney V, Dorman T, Snider C, Stocker R*: Movement, Stability and Low Back Pain*. Churchill Livingstone, pp. 421-424

**65.** Kuchera ML (1997): Treatment of gravitational strain pathology. In Leeming A, Mooney V, Dorman T, Snider C, Stocker R*: Movement, Stability and Low Back Pain*. Churchill Livingstone, pp. 477-499

**66.** Laslett M (1997): Pain provocation sacroiliac joint tests: reliability and prevalence. In Leeming A, Mooney V, Dorman T, Snider C, Stocker R*: Movement, Stability and Low Back Pain*. Churchill Livingstone, pp. 287-295

**67.** Laslett M (1998): Mail to the editor. *Spine* 23(8): 962-963

**68.** Laslett M, April CN, McDonald B, Young SB (article in press): Diagnosis of Sacroiliac Joint Pain: Validity of individual provocation tests and composites of tests. *Manual Therapy.*

**69.** Laslett M, McDonald B, Tropp H, Aprill CN, Oberg B (2005): Agreement between diagnoses reached by clinical examination and available reference standards: a prospective study of 216 patients with lumbopelvic pain. *BMC Musculoskeletal Disorders* 6:28. http://www.biomedcentral.com/1471-2474/6/28. Accessed on 13/08/2005

**70.** Laslett M, Williams M (1994): The Reliability of Selected Pain Provocation Tests for Sacroiliac Joint Pathology. *Spine* 19(11): 1243-1249

**71.** Laslett M, Young SB, Aprill CN, McDonald B (2003): Diagnosing painful sacroiliac joints: A validity study of a McKenzie evaluation and sacroiliac provocation tests. *Australian Journal of Physiotherapy* 49: 89-97

**72.** Lee D (1997): Instability of the sacroiliac joints and the consequences for gait. In Leeming A, Mooney V, Dorman T, Snider C, Stocker R*: Movement, Stability and Low Back Pain*. Churchill Livingstone, pp. 231-233

**73.** Lee D (1997): Treatment of pelvic instability. In Leeming A, Mooney V, Dorman T, Snider C, Stocker R*: Movement, Stability and Low Back Pain*. Churchill Livingstone, pp. 445-459

**74.** Lee D (1999): *The Pelvic Girdle. An approach to the examination and treatment of the thoracolumbar-hip region*. Second Edition, Edinburgh, Churchill Livingstone

**75.** Lee D (2004): *The Pelvic Girdle. An approach to the examination and treatment of the thoracolumbar-hip region*. Third Edition, Edinburgh, Churchill Livingstone

**76.** Levin SM (1997): A different approach of the mechanics of the human pelvis: tensegrity. In Leeming A, Mooney V, Dorman T, Snider C, Stocker R*: Movement, Stability and Low Back Pain*. Churchill Livingstone, pp. 157-167

**77.** Lippit AB (1997): Percutaneous fixation of the sacroiliac join. In Leeming A, Mooney V, Dorman T, Snider C, Stocker R*: Movement, Stability and Low Back Pain*. Churchill Livingstone, pp. 587-594

**78.** Maigne JY, Aivaliklis A, Pfefer F (1996): Results of Sacroiliac Joint Double Block and Value of Sacroiliac Pain Provocation Tests in 54 Patients with Low Back Pain. *Spine* 21(16): 1889-1892

**79.** Mann M, Glasheen-Wray M, Nyberg R (1984): Therapist Agreement for Palpation and Observation of Iliac Crest Heights. *Physical Therapy* 64(3): 334-338

**80.** McClure PW, Esola M, Schreier R, Siegler S (1997): Kinematic Analysis of Lumbar and Hip Motion While Rising From a Forward, Flexed Position in Patients With and Without a History of Low Back Pain. *Spine* 22(5): 552-558

**81.** McGill SM, Grenier S, Kavcic N, Cholewicki J (2003): Coordination of muscle activity to assure stability of the lumbar spine. *Journal of Electromyography and Kinesiology*. 13: 353–359. www.elsevier.com/locate/jelekin. Accessed on 16/08/2005

**82.** Means JMA, Leeming A, Snider CF, Stem HF (1997): Active straight leg raising test: a clinical approach to the load transfer function of the pelvic girdle. In Leeming A, Mooney V, Dorman T, Snider C, Stocker R*: Movement, Stability and Low Back Pain*. Churchill Livingstone, pp. 425-431

**83.** Means JMA, Leeming A, Snider CJ, Koes BW, Stem HJ (2001): Reliability and Validity of the Active Straight Leg Raising Test in Posterior Pelvic Pain Since Pregnancy. *Spine* 26(10): 1167-1171

**84.** Means JMA, Leeming A, Snider CJ, Koes BW, Stem HJ (2002): Validity of the Active Straight Leg Raise Tast for Measuring Disease Severity inpatients with Posterior Pelvic after Pregnancy. *Spine* 27(2): 196-200

**85.** Means JMA, Leeming A, Snider, Ronchetti I, Stem HJ (2002): Reliability and Validity of Hip Adduction Strength to Measure Disease Severity in Posterior Pelvic Pain Since Pregnancy. Spine 27(15): 1674-1679

**86.** Michaud TC (1997): *Foot Orthoses and other forms of conservative foot care*: Chapter three: Abnormal motion during the gait cycle. Newton, Massachusetts, pp. 57-180

**87.** Mooney V, Pozos R, Leeming A, Gulick F, Swenski D (1997): Coupled motion in contralateral latisimus dorsi and gluteus maximus: its role in sacroiliac stabilization. In Leeming A, Mooney V, Dorman T, Snider C, Stocker R*: Movement, Stability and Low Back Pain*. Churchill Livingstone, pp. 115-122

**88.** Moore MR (1997): Surgical treatment of chronic painful sacroiliac joint dysfunction. In Leeming A, Mooney V, Dorman T, Snider C, Stocker R*: Movement, Stability and Low Back Pain*. Churchill Livingstone, pp. 563-572

**89.** Moseley L (2002): Combined Physiotherapy and education is efficacious for chronic low back pain. *Australian Journal of Physiotherapy* 48: 297-302

**90.** Nelson JM, Walmsley RP, Stevenson JM (1995): Relative Lumbar and Pelvic Motion During Loaded Spinal Flexion/Extension. *Spine* 20(2): 199-204

**91.** O'Haire C, Gibbons P (2000): Inter-examiner agreement for assessing sacroiliac anatomical landmarks using palpation and observation: pilot study. *Manual Therapy* 5(1): 13-20

**92.** O'Sullivan PB (2000): Lumbar segmental "instability": clinical presentation and specific stabilizing exercise management. Manual Therapy 5(1): 2-12

**93.** O'Sullivan PB, Twomey L, Allison GT (1997): Evaluation of Specific Stabilizing Exercise in the Treatment of Chronic Low Back Pain with Radiologic Diagnosis of Spondylolysis or Spondylolisthesis. *Spine* 22(24): 2959-2967

**94.** Ostagaard HC (1997): Lumbar, back and posterior pelvic pain in pregnancy. In Leeming A, Mooney V, Dorman T, Snider C, Stocker R*: Movement, Stability and Low Back Pain*. Churchill Livingstone, pp. 511-420

**95.** Panjabi MM (1990): The Stabilizing System of the Spine. Part I. Function, Dysfunction and Enhancement. *Journal of Spinal Disorders* 5(4): 383-389

**96.** Panjabi MM (1992): The Stabilizing System of the Spine. Part II. Neutral Zone and Instability Hypothesis. *Journal of Spinal Disorders* 5(4): 390-397

**97.** Paris SV (1997): Differential diagnosis of lumbar and pelvic pain. In Leeming A, Mooney V, Dorman T, Snider C, Stocker R*: Movement, Stability and Low Back Pain*. Churchill Livingstone, pp. 319-330

**98.** Platzer W, Leonardt H, KAhle W (1992): Color Atlas/Text of Human Anatomy, Vol.1. Locomotor System. 4$^{th}$ edition, Thieme Medical Publishers, New York

**99.** Petersen, Laslett M, Thorsen H, Manniche C, Ekdahl, Jacobsen S (2003): Diagnostic classification of non-specific low back pain. A new system integrating patho0anatomic and clinical categories. *Physiotherapy Theory and Practice* 19: 213-237

**100.** Potter N, Rothstein JM (1985): Intertester Reliability for Selected Clinical Tests of the Sacroiliac Joint. *Physical Therapy* 65(11): 1671-1675

**101.** Richardson CA, Snider CJ, Hides JA, Damen L, Pas MS, Storm J (2002): The Relation between the Transversus Abdominis Muscles, Sacroiliac Joint Mechanics, and Low Back Pain*. Spine* 27(4): 399-405

**102.** Roos HP (1997): Hip Pain in Sport. *Sports Medicine and Arthroscopy Review* 5: 292-300

**103.** Ruta DA, Garratt AM, Wardlaw D (1994): Developing a Valid and Reliable Measure of Health Outcome for Patients with Low Back Pain. *Spine* 19(17): 1887-1896

**104.** Saal JA, Firtch W, Saal JS, Herzog RJ (1992): The Value of Somatosensory Evoked Potential Testing for Upper Lumbar Radiculopathy. A correlation of Electrophysiological and Anatomic data. *Spine* 17(6) supplement: S133-S137

**105.** Sapsford R (2004): Rehabilitation of pelvic floor muscles utilizing trunk stabilization. *Manual Therapy* 9:3-12

**106.** Schultz RWM (1995): Malidation of the Shear Test for the Lumbar Spine. A pilot study. *Ola Grimsby Institute, San Diego*, California

**107.** Schwarzer AC, Aprill CN, Bogdub N (1995): The Sacroiliac Joint in Chronic Low Back Pain. *Spine* 20(1): 31-37

**108.** Sims K (1999): Assessment and Treatment of Hip Osteoporosis. *Manual Therapy* 4(3): 136-144

**109.** Sims K (1999): The development of hip osteoarthritis: implications for conservative treatment. *Manual Therapy* 4(3): 127-135

**110.** Smidt GL (1997): Interinnominate range of motion. In Leeming A, Mooney V,Dorman T, Snider C, Stocker R*: Movement, Stability and Low Back Pain*. Churchill Livingstone, pp. 187-191

**111.** Smith RL, Sebastian BA, Gajdosik LG (1988): Effect of Sacroiliac Joint Mobilization on the Standing position of the Pelvis in Healthy Men. *The Journal of Orthopaedic and Sports Physical Therapy* 10(3): 77-84

**112.** Smidt GL, Wei SH, McQuade K, Barakatt E, Sun T, Stanford W (1997): Sacroiliac Motion for Extreme Hip Positions. Spine 22(18): 2073-2082

**113.** Snider CJ, Slater AHE, van Strike R, Leeming A, Stocker R, Stem HJ (1995): Why Leg Crossing? The influence of common postures on abdominal muscle activity. *Spine* 20(18): 1989-1993

**114.** Snider CJ, Leeming A, Stocker R (1993): Transfer of lumbosacral load to iliac bones and legs. Part 1: Biomechanics of self-bracing of the sacroiliac joints and its significance for treatment and exercise. *Clinical Biomechanics* 8: 285-294

**115.** Snider CJ, Leeming A, Stocker R (1993): Transfer of lumbosacral load to iliac bones and legs. Part 2: Loading of the sacroiliac joints when lifting in a stooped posture. *Clinical Biomechanics* 8: 295-301

**116.** Snider CF, Leeming A, Stocker R, Means FMA, GF Kleinrensink (1997): Biomechanics of the interface between spine and pelvis in different postures. In

Leeming A, Mooney V, Dorman T, Snider C, Stocker R*: Movement, Stability and Low Back Pain*. Churchill Livingstone, pp. 103-113

**117.** Strender LE, Sjoblom A, Sundell K, Ludwig, Taube A (1997): Inter-examiner Reliability in Physical Examination of Patients with Low Back Pain. *Spine* 22(7): 814-820

**118.** Sturesson B (1997): Movement of the sacroiliac joint: a fresh look. In Leeming A, Mooney V, Dorman T, Snider C, Stocker R*: Movement, Stability and Low Back Pain*. Churchill Livingstone, pp. 171-176

**119.** Sturesson B, Uden A, Leeming A (2000): A Radiostereometric Analysis of Movements of the Sacroiliac Joints During the Standing Hip Flexion Test. *Spine* 25(3): 364-368

**120.** Subotnick SI (1976): The Short Leg Syndrome. *Podiatric Sports Medicine* 66(9): 720-723

**121.** Tenhula JA, Rose SJ, Delitto A (1990): Association between Direction of Lateral Lumbar Shift, Movement Tests and Side of Symptoms in Patients with Low Back Pain Syndrome. Research Report. *Physical Therapy* 70(8): 480-486

**122.** Thomas E, Silman AJ, Papageorgiou AC, Macfarlane GJ, Croft PR (1998): Association between Measures of Spinal Mobility and Low Back Pain. An analysis of new attendrers in primary care*. Spine* 23(3): 343-347

**123.** Simons DG and Travell JG (1984): Myophascial pain syndromes. In Wall PD, Melzack R (1984): *Textbook of Pain*. Churchill Livingstone, Edinburgh, p. 271

**124.** Urban LM (1981): The Straight-Leg-Raising Test: A Review. *The Journal of Orthopaedic and Sports Physical Therapy* 2(3): 117-133

**125.** Van Diee¨n JH ,Selen LPJ, Cholewicki J (2003): Trunk muscle activation in low-back pain patients, an analysis of the literature. Journal of Electromyography and Kinesiology 13: 333–351

**126.** Van Wngerden FP, Leeming A, Kleinrensink GF, Stocker R (1997): The role of hamstrings in pelvic and spinal function. In Leeming A, Mooney V, Dorman T, Snider C, Stocker R*: Movement, Stability and Low Back Pain*. Churchill Livingstone, pp. 207-210

**127.** Vleeming A, Pool-Goudzwaard AL, Stocker R, Van Wingerden JP, Snider CJ (1995): The Posterior Layer of the Thoracolumbar Fascia. Spine 20(7): 753-758

**128.** Vleeming A, Stocker R, Snider CJ (1989): The sacrotuberous ligament: a conceptual approach to its dynamic role in stabilizing the sacroiliac joint. *Clinical Biomechanics* 4(4): 201-203

**129.** Vleeming A, Stocker R, Volkers ACW, Snider CJ (1993): Relation Between Form and Function in the Sacroiliac Joint. Part I: Clinical Anatomical Aspects. *Spine* 15(2): 130-136

**130.** Vleeming A, Snider CF, Stocker R, Means FMA (1997): The role of the Sacroiliac joints in coupling between spine, pelvis, legs and arms. In Vleeming A, Mooney V, Dorman T, Snider C, Stocker R*: Movement, Stability and Low Back Pain.* Churchill Livingstone, pp. 53-71

**131.** Vleeming A, Van Wingerden JP, Dijkstra PF, Stocker R, Snider CJ, Stinjnen T (1992): Mobility of the sacroiliac joints in the elderly: a Kinematic and radiological study. *Clinical Biomechanics* 7: 170-175

**132.** Vleeming A, Van Wingerden JP, Snider CJ, Stocker R, Stijnen T (1989): Load application to the sacrotuberous ligament; influences on sacroiliac joint mechanics. *Clinical Biomechanics* 4(4): 204-209

**133.** Vogt L, Pfeifer K, Bazner W (2002): Comparison of angular lumbar spine and pelvis kinematics during treadmill and overground locomotion. *Clinical Biomechanics* 17: 162-165

**134.** Waddell G, Somerville D, Henderson I, Newton M (1992): Objective Clinical Evaluation of Physical Impairment in Chronic Low Back Pain. *Spine* 17(6): 617-628

**135.** Walker JM (1992): The Sacroiliac Joint: A Critical Review. *Physical Therapy* 72: 903-916

**136.** Walker ML, Rothstein JM, Finucane SD, Lamb RL (1987): Relationships between Lumbar Lordosis, Pelvic Tilt, and Abdominal Muscle Performance. *Physical therapy* 67(4): 512-515

**137.** Weber B (1983): Lumbar disk herniation: a controlled prospective study with ten years observation. *Spine* 8: 131-140

**138.** Wilke HJ, Wolf S, Claes LE, Arand M, Wiesend A (1995): Stability Increase of the Lumbar Spine With Different Muscle Groups. A biomechanical In Vitro Study. *Spine* 20(2): 192-198

**139.** Willard FH (1997): The muscular, ligamentous and neural structure of the low back ant its relation to back pain. In Leeming A, Mooney V, Dorman T, Snider C, Stocker R: *Movement, Stability and Low Back Pain*. Churchill Livingstone, pp. 3-35

**140.** Whittaker J (2004): Abdominal Ultrasound Imaging of Pelvic Floor Muscle Function in Individuals with Low Back Pain. *The Journal of Manual & Manipulative Physiotherapy* 12(1): 44-49

**141.** Wurff P, Meyne W, Hagmeijer RHM (2000): Clinical tests for the sacroiliac joint. A systematic methodological review. Part 1: Reliability. *Manual Therapy* 5(1): 30-36

**142.** Wurff P, Meyne W, Hagmeijer RHM (2000): Clinical tests for the sacroiliac joint. A systematic methodological review. Part 2: Validity. Manual Therapy 5(2): 89-96

**143.** Yamamoto I, Panjabi MM, Oxland R, Crisco JJ (1990): The Role of Iliolumbar Ligament in the Lumbosacral Junction. *Spine* 15(11): 1139-1141