Stress Urinary Incontinence –
A Consequence of Failed Load Transfer Through the Pelvis?

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INTRODUCTION

The anatomical and biomechanical research in the last decade has led to a clearer understanding of how load is transferred through the low back and pelvic girdle (Hodges & Richardson 1996, 1997, Hodges 1997, 2003, Hodges et al 1999, 2001a,b,c, 2003b, Hungerford 2001, Mens et al 1999, Richardson et al 1999, Snijders et al 1993a,b Vleeming et al 1990a,b, Vleeming et al 1995, Vleeming et al 1996). From this research, an integrated model of function evolved (Lee & Vleeming 1998, 2004) and clinical extrapolations based on this model were developed (Richardson et al 1999, Hodges (Course Notes, 2004), Lee 2004, Lee & Lee 2004). Further research based on the model continued (Hungerford et al 2004, O'Sullivan et al 2002, Pool-Goodzwaard 2003, Stuge et al 2004) and it soon became evident that two conditions of failed load transfer through the pelvis, low back/pelvic girdle pain and stress urinary incontinence, had components in common. Recently, Pool-Goudzwaard (2003) conducted a multi-centered study in Holland to investigate, in part, the prevalence of low back/pelvic girdle pain and pelvic floor disorders. In this study of 66 patients, 52% reported a combination of low back and/or pelvic girdle pain and stress urinary incontinence, had components in common. Of these 52%, 82% stated that their symptoms began with either low back or pelvic girdle pain.

Our journey into considering the relationship between load transfer through the musculoskeletal components of the pelvis and the organs it contains began at the 4th World Congress on Low Back and Pelvic Girdle Pain after hearing a paper (O’Sullivan et al. 2002) which demonstrated via real-time ultrasound imaging the impact of the active straight leg raise (ASLR) (Mens et al 1999) on the position of the bladder in pelvic girdle pain patients. They noted that the bladder tended to descend during the ASLR and that this descent decreased when compression was applied to the pelvis. Our question at the time was, “How much should the bladder move when you lift your leg?” This led to a search of the literature pertaining to stress urinary incontinence and several revelations followed regarding the parallel features of both low back/pelvic girdle pain and stress urinary incontinence. We now recognize that the factors which must be optimal for effective force closure and stability of the pelvic girdle and those which must be present for optimal force closure of the urethra are the same. The intent of this paper is to briefly review the literature regarding effective force closure of the pelvic girdle, present the anatomical and neurophysiological requirements for effective force closure of the urethra and conclude with a case presentation which will clinically integrate the material. Some of the material in this paper comes from the 3rd edition of The Pelvic Girdle (Lee 2004) and is reproduced here with permission.

THE INTEGRATED MODEL OF FUNCTION

The integrated model of function has four components: form closure (structure), force closure (forces produced by myofascial action), motor control (specific timing of muscle action/inaction during loading) and emotions. The joints of the pelvic girdle are mobile (Jacob & Kissing 1995, Sturesson et al 2000) and therefore force closure of the pelvic girdle is required if loads are to be transferred optimally. According to Panjabi (1992a,b) stability (effective load transfer) is achieved when the passive, active and control systems work together. Collectively, all three systems produce approximation of the joint surfaces (Snijders & Vleeming 1993a,b); essential if stability is to be insured. The amount of approximation required is variable and difficult to quantify since it depends on an individual’s structure and the forces they need to control. The term ‘adequate’ has been used (Lee & Vleeming 1998, 2004) to describe how much approximation is necessary and reflects the non-quantitative aspect of this measure. Essentially, it means ‘not too much’ and ‘not too little’; in other words,
'just enough' to suit the existing situation. Consequently, the ability to effectively transfer load through the pelvis is dynamic and depends on:

1. optimal function of the bones, joints and ligaments (form closure – not discussed in this paper) (Vleeming et al 1990a,b).

EFFECTIVE FORCE CLOSURE AND MOTOR CONTROL - THE PELVIC GIRDLE

If the articular surfaces of the pelvis were constantly and completely compressed, mobility would not be possible. However, compression during loading is variable and therefore motion is possible (Jacob & Kissling 1995, Sturesson 2000) and stabilization required. This is achieved by increasing compression across the joint surface at the moment of loading (force closure). The amount of force closure required depends on the individual's form closure and the magnitude of the load. The anatomical structures responsible for force closure are the ligaments, muscles and fascia.

In the close-packed or self-locked (self-braced) position, the joints of the pelvic girdle are under significant compression and the ability to resist shear forces is enhanced by the tension of the passive structures and increased friction between the articular surfaces (Vleeming et al 1990b, Snijders et al 1993a,b). For the sacroiliac joints, this position is full nutation of the sacrum or posterior rotation of the innominate (Vleeming et al 1990a,b, van Wingerden et al 1993). Studies have shown (Egun et al 1978, Hungerford 2004, Lavignolle et al 1983, Sturesson et al 2000) that nutation of the sacrum occurs bilaterally whenever the lumbopelvic spine is loaded. The amount of sacral nutation varies with the magnitude of the load. Full sacral nutation (selflocking or close packing) occurs during forward and backward bending of the trunk (Sturesson et al 2000). However, function would be significantly compromised if joints could only be stable in the close-packed position. In the neutral spinal position, an osteoligamentous spine (T1 to sacrum) will buckle under approximately 20N (about 4.4 lb) of compression load (Lucas & Bresler, 1961, Panjabi 1992a,b). Consequently, stability for load transfer is required throughout the entire range of motion and this is provided by the active, or neuromyofascial, system.

In 1989, Bergmark proposed that muscles could be classified into two systems – a local and a global system (Bergmark 1989). The local system pertains to those muscles essential for segmental or intrapelvic stabilization while the global system appears to be more responsible for regional stabilization (between the thorax and pelvis or pelvis and legs) and motion (Bergmark 1989, Comerford & Mottram 2001, Richardson et al 1999). The function of the lumbopelvic local system is to stabilize the joints of the spine and pelvic girdle in preparation for (or in response to) the addition of external loads. This is achieved through several mechanisms some of which include:

- increasing the intra-abdominal pressure (Cresswell, 1993, Hodges & Gandevia 2000a,b, Hodges et al 2001a, 2003b, McGill & Norman 1987)
- increasing the tension of the thoracodorsal fascia (Cresswell 1993, Hodges 2003, Hodges et al 2003b, Vleeming et al 1995a, Willard 1997) and/or
- increasing the articular stiffness (Hodges et al 1997, Hodges 2003, Richardson et al 2002).

Research has shown (Barbic et al 2003, Bø & Stien 1994, Constantinou & Govan 1982, Hodges 1997, Hodges & Gandevia 2000a,b, Hodges 2003, Hungerford 2004, Moseley et al 2002, 2003, Sapsford et al 2001) that when the central nervous system can predict the timing of the load, the local system is anticipatory when functioning optimally. In other words, these muscles should work at low levels at all times and increase their action before any further loading or motion occurs. When the local system is functioning optimally, it provides anticipatory intersegmental stiffness of the joints of the lumbar spine (Hodges et al 2003b) and pelvis (Richardson et al 2002). This external force augments the form closure and helps to prevent excessive shearing at the time of loading. This stiffness/compression occurs prior to the onset of any movement and prepares the low back and pelvis for additional loading from the global system.
The research is still lacking which enables classification of all muscles according to this system and clinically it appears that parts of some muscles may belong to both systems. With respect to the lumbopelvic region, the following muscles fit the criteria for classification as local stabilizers - the muscles of the pelvic floor (Barbic et al 2003, Bø et al 1994, Constantinou & Govan 1982, Deindl et al 1993, Hodges 2003, Sapsford et al 2001), the transversus abdominis (Hodges & Richardson 1997a,b, Hodges 2003), the diaphragm (Hodges & Gandevia 2000a,b, Hodges 2003) and the deep fibres of multifidus (Hides et al 1994, 1996, Moseley et al 2002, 2003). As research continues, more muscles will likely be added to this list. The deep (medial) fibres of psoas (Gibbons et al 2002), the medial fibres of quadratus lumborum (Bergmark 1989, McGill 2002), the lumbar parts of the lumbar iliocostalis and longissimus (Bergmark 1989) and the posterior fibres of the internal oblique (Bergmark 1989, O'Sullivan 2000) are some likely candidates.


**EFFECTIVE FORCE CLOSURE AND MOTOR CONTROL - THE URETHRA**

Urinary incontinence is defined as the involuntary leakage of urine which is objectively demonstrable. Stress urinary incontinence (leakage which occurs during physical exertion) is the most common type. According to DeLancey (1994): ‘During a cough urethral closure pressure is known to rise simultaneously with abdominal pressure to keep the urethra closed in spite of great increases in intravesical pressure.’ Essentially, when one cannot effectively force close the urethra (maintain urethral closure pressure) stress urinary incontinence can result. How common is this? The prevalence of this condition varies according to age, study design and definition. Ashton-Miller et al (2001) state that 8.5% - 38% of women experience stress urinary incontinence (SUI). Nygaard et al (1994) note that this condition is not limited to women bearing children and that in a study of 144 nulliparous female athletes ages 18 to 21 years, 28% suffered from SUI. Bø & Borgen (2001) found that 41% of elite female athletes experience SUI. Fantl et al (1996) states that incontinence affects four out of ten women, about one out of ten men, and about 17% of children below the age of fifteen.

Clearly, this is a significant problem but is it a different problem than a loss of effective force closure of the musculoskeletal elements of the pelvis? It is common to hear women complain of both low back and pelvic girdle pain as well as urinary incontinence and therapists commonly note that treating one component often impacts the other. Minimal research has been done on the correlation between the two functional impairments. In reviewing the literature, it appears that Panjabi’s stability model (1992a,b) can be applied to the urethra as well as to the musculoskeletal system.

**Urinary continence**

In an excellent review article, Ashton-Miller, Howard and DeLancey (2001) clearly explain the mechanism by which continence is achieved during physical exertion. Essentially, continence relies on optimal function of two systems; the urethral support system and the sphincteric closure system.
The structures which provide support for the urethra include (Fig. 1):

- the passive system – this includes the endopelvic fascia which is anchored to a thick fascial band called the arcus tendineus fasciae which arises from the pubic bone anteriorly and inserts into the ischial spine
- the active system for this fascial hammock or sling – this includes the levator ani muscle which contains primarily type 1 fibres and exhibits constant tone.
- the control system – this includes the pudendal nerve which innervates the levator ani as well as the central control of reflex function between the detrusor muscle and the pelvic floor.

Together, the passive and active systems form a hammock of support for the urethra (Fig. 2) and the integrity/function of these tissues is essential if force closure of the urethra is to be effective. If this system gives way easily, it cannot provide a backstop against which the urethra can be compressed. A useful analogy (Ashton-Miller et al 2001) is to imagine a garden hose (urethra), with water running through it (urine), lying on a trampoline bed (the pelvic floor). Stepping on the hose will block the flow of water if the bed is very stiff and provides an equal and opposite counterforce (functional pelvic floor). If however, the bed is very flexible (i.e. loss of myofascial support), the downward pressure on the hose will cause the bed to stretch and allow the hose to indent the bed. The flow of water will continue uninterrupted.

Sphincteric Closure System

In addition, the urethra is closed by a system of both intrinsic and extrinsic muscles. The striated muscles within the wall of the urethra (intrinsic) contract prior to any pressure received by the bladder and these muscles are also comprised of type 1 fibres; well-suited to maintain constant tone.

Constantinou & Govan (1982) measured the intra-urethral and intra-bladder pressures in healthy continent women during the valsalva maneuver and coughing and found that during a cough the pressure in the urethra increased approximately 250 ms before any pressure increase was detected in the bladder. This did not occur during a valsalva (bearing down or straining). This suggests that an anticipatory reflex exists between the
pelvic floor and the urethra (the pelvic floor is active during a cough and inactive during a valsala). This anticipatory closure of the urethra was confirmed in a subsequent study by Thind et al (1991). They also noted that the urethral pressure remained elevated for a short time after the pressure normalized in the bladder.

Bo & Stien (1994) used needle EMG to measure activity in the urethral wall during a cough and valsala as well as during activation of the hip adductors, abdominals and gluteal muscles. They found that the urethral wall contracted synergistically with the pelvic floor, hip adductors and gluteals and also during a cough. They conclude that strengthening the pelvic floor will also strengthen the urethral wall – but will it restore the anticipatory reflex mechanism?

Deindl et al (1993) investigated via needle EMG the activity pattern of pubococcygeus in nulliparous, continent women during a voluntary “squeeze” and during an attempt to urinate. The voluntary “squeeze” led to a concomitant increase in activation of the targeted motor units bilaterally (this response was sustained) whereas during the attempt to urinate a marked decrease in the ongoing tonic motor unit activity occurred.

Sapsford et al. (2001) investigated the co-activation pattern of the pelvic floor and abdominals via fine wire EMG for the abdominal wall and surface EMG for the pelvic floor and found that the abdominals contract in response to a pelvic floor contraction command and that the pelvic floor contracts in both a ‘hollowing’ and ‘bracing’ abdominal command. They also found that a submaximal command of pubococcygeus elicited the greatest response in transversus abdominis. The results from this research suggest that the pelvic floor can be facilitated by co-activating the deep abdominals and visa versa. However; it is wrong to assume that all patients will be able to contract the muscles of the pelvic floor through verbal commands alone, either through the abdomen or the pelvic floor. Bump et al (1991) found that only 50% of women could actually perform a pelvic floor muscle contraction with just a verbal instruction. We have found that careful analysis is required to ensure that the reflex connection between the transversus abdominis and the pelvic floor is intact before this strategy is used.

In conclusion, the evidence suggests that the pelvic floor plays a significant role in effective force closure of the urethra and that these muscles fit the criteria for classification as local system muscles. Optimally the pelvic floor muscles function constantly at low levels of tone and increase their activation in anticipation of load. They should co-activate with transversus abdominis for lumbopelvic stabilization and play a role in increasing the intra-urethral pressure, in pre-tensing the endopelvic fascia and in facilitating a co-activation of the striated urethral sphincter muscles.

Stress urinary incontinence

Stress urinary incontinence can result when there is loss of the anatomical integrity or neurophysiological function of the pelvic floor (muscles and fascia) secondary to a single major trauma or repetitive minor trauma. Inefficient load transfer strategies through the low back and pelvis, particularly those which excessively increase the intra-abdominal pressure and result in the bladder and pelvic organs being repetitively compressed inferiorly, can lead to incontinence via repetitive microtrauma to the fascial supports or via altering optimal recruitment of the pelvic floor muscles (Sapsford 2004). Also, a history of straining during bowel movements is common in women with uterovaginal prolapses and stress urinary incontinence (Spence-Jones et al 1994). These altered motor control strategies often lead to changes in lumbopelvic posture. Nguyen et al (2000) found that women with uterovaginal prolapse had significantly less lumbar lordosis and a less vertically oriented pelvic inlet than groups without prolapase. This postural change is often seen in patients with stress urinary incontinence as well as in patients with lumbopelvic dysfunction (clinical observation). When the bladder is observed with real-time ultrasound imaging, it can be seen that these strategies cause the bladder to descend (Fig. 3).
Using real-time ultrasound imaging in a group of patients with pelvic girdle pain, O’Sullivan et al (2002) noted that the bladder descended during an active straight leg raise test. When compression was applied to the pelvic girdle, this descent was minimized. How much descent of the bladder is optimal, or normal, during functional activities? Peschers et al (2001) measured the mobility of the bladder neck via perineal ultrasound during coughing and valsala in 39 healthy, nulliparous women. They found that the bladder neck descended a variable amount (2 – 32mm) in both a cough and valsala and questioned the long held view that stress urinary incontinence was associated with urethral mobility. Like the sacroiliac joint, there appears to be a wide variation in the amount of motion possible (Buyruk et al 1995a,b, Damen et al 2001) and continence (effective force closure of the urethra) relies more on control and urethral closure rather than amplitude of motion.

Howard et al (2000) investigated the amplitude of descent of the bladder neck during a cough and valsala in three groups of women, nulliparous continent (17 subjects), primiparous continent (18 subjects) and primiparous incontinent (23 subjects). There was no statistical difference in the amount of bladder neck mobility between the groups, again suggesting that movement of the urethra is not what determines one’s continence status. When they compared the amplitude of bladder neck movement during a cough and valsala, they noted that the two continent groups exhibited less movement during a cough. Conversely, the incontinent group demonstrated no difference in the amount of movement during a cough or valsala. Clearly, something was happening during a cough in the continent women that was not happening in the incontinent group. All three groups generated the same amount of cough pressures; however, the stiffness value (pressure change divided by bladder neck mobility) was the greatest in the nulliparous continent group, next highest in the primiparous continent group and lowest in the primiparous incontinent group. They hypothesize that these differences depend on the function of the pelvic floor; assumed to be optimal in the continent women. Thind et al (1991) noted that the amplitude of the anticipatory pressure rise in the urethra was less in women with stress urinary incontinence and suggest that this is due to weakness of the pelvic floor. Indeed, Bø et al (1990) demonstrated in a randomized clinical trial that retraining the function of the pelvic floor (awareness training coupled with strength and endurance training) is effective for some women (60%) in the treatment of stress urinary incontinence.

Allen et al (1990) investigated 96 nulliparous women both pre and postnatally to determine if childbirth caused damage to the pelvic floor muscles and/or their nerve supply. They showed that a vaginal delivery impaired the strength of the pelvic floor and noted that recovery had not occurred at two months postpartum. They also demonstrated via needle EMG that vaginal delivery caused a partial denervation of the pelvic floor in 80% of these women. Women who had a long, active second stage of labour showed the most EMG evidence of denervation. Ashton-Miller et al (2001) feel that if the nerve to the levator ani is damaged, the denervated muscles will atrophy thus placing more stress on the passive supporting structures (endopelvic fascia) which over time will stretch and result in organ prolapse. Alternately, a paravaginal defect can occur which causes a separation in the endopelvic fascia. This effectively reduces the stiffness of the fascial layer which supports the urethra (Fig. 2) and can occur unilaterally or bilaterally. When this occurs, the pelvic floor must take over to support the organ position and provide active closure to the urethra. However, they note that ‘If the muscle is completely detached from the fascial tissues, then it may be able to contract; but that contraction may not be effective in elevating the urethra or stabilizing its position’ (Ashton-Miller et al 2001).

Deindl et al (1994) compared the activity pattern of pubococcygeus in both nulliparous, continent and parous stress urinary incontinent women. They found two differences in the incontinent group:

1. a voluntary “squeeze” of the pubococcygeus showed an endurance deficit (shorter holding times)
2. an asymmetrical and uncoordinated pattern of activation (left vs. right) commonly occurred. Sometimes the response was only unilateral.
Barbic et al (2003) also investigated the pattern and timing of muscle activation of the levator ani in both continent and incontinent women. They noted that both the left and right levator ani contracted prior to any pressure increase in the bladder and that the timing of this activation was delayed in the incontinent group. They conclude that an important aspect of a “stable bladder neck is the timely activation of the levator ani muscle. The activation, which precedes the contraction of other muscles..., might enable a pretension of the endopelvic fascia tissue, which becomes less compliant for stretching by downward forces of increased abdominal pressure.”

Therefore, continence of urine (effective force closure of the urethra) during the transference of loads through the pelvic girdle requires optimal bladder position control and this depends, in part, on the individual’s ability to effectively contract and sustain a tonic co-contraction of the local muscle system in a properly timed manner.

We believe that orthopaedic manual therapists who focus on restoring function to the local system of the low back and pelvic girdle (transversus abdominis, multifidus, diaphragm, and the pelvic floor) and therapists who specialize in pelvic floor dysfunction are treating the same condition – failed load transfer through the lumbopelvic region, manifested either through a loss of effective force closure of the joints of the low back and pelvis, or loss of effective force closure of the urethra. The research clearly supports that we are merging to a common understanding of both function and dysfunction of the whole pelvis and not just its parts. Treatment of the impaired lumbopelvic-hip region must focus on an integrated approach – one which considers the restoration of form closure, force closure and motor control of all the structures contained within the region. Ultimately, function requires stability with mobility (not rigidity) of the joints and organs, for any endeavor the individual chooses to do. The subsequent section of this paper will outline the principles for evidence-based treatment of either

1. failed load transfer through the musculoskeletal components of the pelvic girdle and/or
2. failed load transfer through the organs of the pelvic girdle.

**GENERAL PRINCIPLES - TREATMENT ACCORDING TO THE INTEGRATED MODEL OF FUNCTION**

The following material is taken from the 3rd edition of The Pelvic Girdle (Lee 2004; Ch. 10 Lee & Lee) and it’s companion DVD (Lee & Lee 2004) and is reproduced with permission (Churchill Livingstone and Lee & Lee 2004).

Treatment for the impaired lumbopelvic-hip region with or without stress urinary incontinence must be prescriptive since every individual has a unique clinical presentation. Rarely will only one dysfunction be present (one stiff joint or one poorly controlled joint); more commonly, multiple problems coexist such that the most effective treatment consists of a unique combination of techniques and exercises specific for each patient. However, there are some principles for treatment which help guide the therapist who is inexperienced in working with this model. The first step is to analyse the findings from the assessment. Does the individual appear to be:

- primarily under *too much* compression from stiff joints (form closure) or hypertonicity of the global system (force closure/motor control),
- primarily under *too little* compression due to loose joints (form closure), a poorly controlled neutral zone of motion, and/or insufficient recruitment and timing of the local system (force closure/motor control), or
- a combination of both *too much* and *too little* compression in different areas of the lumbopelvic-hip complex.

In the first instance, the therapist may decide to use manual techniques and exercises which decompress the joints (increase mobility) and follow this with an exercise plan that re-establishes a more optimal stabilization strategy which emphasizes stability with mobility. In the second instance, the therapist may decide to start a program which emphasizes retraining of the local system right away (increase stability) and then add decompression techniques/exercises (increase mobility) later as necessary. The most common scenario is the third where a combination of decompression and stabilization is required. Continual assessment of form closure (mobility/stability of the joints) and force closure/motor control helps direct the therapeutic plan from treatment to treatment.

The effective management of lumbopelvic-hip pain and dysfunction requires attention to all four components – form closure, force closure, motor control and emotions. Ultimately, the goal is to teach the patient
a healthier way to live and move such that sustained compression and/or tensile forces on any one structure are avoided.

If the clinical findings suggest that decompression is necessary, the treatment principles are:
1. restore the zygapophyseal, sacroiliac and/or hip joint mobility (form closure – mobility),
2. correct the osseous alignment within and between the lumbar spine, pelvic girdle and femur,
3. restore optimal force closure and control of the neutral zone through training of the local system (force closure/motor control),
4. retrain integration of the local and global systems, including functional movements (rehearse activities of daily living, work or sport specific movement patterns - functional integration).

If the clinical findings suggest that more compression is necessary, the treatment principles are:
1. correct the osseous alignment within and between the lumbar spine, pelvic girdle and femur
2. restore optimal force closure and motor control through training of the local system (force closure/motor control),
3. provide an external support (not always necessary) to augment the training being taught (SI belt, taping),
4. restore articular mobility/stability to extrinsic joints (knee, foot, thorax) since their dysfunction may be contributing to compensatory patterns that put excessive stress on the joints of the lumbopelvic-hip region (form closure – mobility).

Restoring joint mobility

The fibrotic stiff joint

The passive structures surrounding a joint (capsule and ligaments) can stiffen following either a traumatic joint sprain or as a consequence of the joint being held in a compressed position for prolonged periods of time (weeks). Passive articular mobilization techniques are the most effective for restoring mobility to stiff joints. The technique is graded according to the irritability of the articular tissues and longstanding fibrosis requires a sustained grade 4+ passive mobilization.

The myofascially compressed joint/region

When joints are compressed due to overactivation of muscles they feel ‘stiff’ when moved passively; however their motion is not limited by passive restraints but rather the motion is resisted by myofascial forces. In this situation, there are many neuromuscular techniques which effectively decrease muscular hypertonicity and therefore restore mobility. The muscles commonly affected are those of the global system. These techniques include:
1. active mobilization or muscle energy techniques (Mitchell 2001, Schamberger 2002),
2. functional (strain, counterstrain) or craniosacral techniques,
3. trigger point techniques,
4. intramuscular stimulation (IMS - dry needling) (Gunn 1996),
6. techniques to restore optimal breathing (Chaitow et al 2002, Lee 2003, Lee 2003),
7. exercises which encourage ‘movement with awareness’ (Feldenkrais, Hanna Somatics, Pilates), finding neutral spine (Lee 2003) and the optimal lumbopelvic pyramid (postural re-education) (Lee 2001).

The fixated joint

Joints can become fixated in an abnormal position when a force exceeds the resistance ability of the passive restraints. This is not common in the spine nor the pelvic girdle, however when present, a passive articular manipulation technique (Hartman 1997, Lee 2004) is necessary to restore the joint position and mobility before motor control retraining can be prescribed.

Correcting alignment

Loads are transferred more effectively through joints which are properly aligned such that the compression and tension forces induced are shared amongst all structures. Malalignment can create excessive stress on individual structures (tension or compression) which ultimately leads to tissue breakdown (inflammation and pain). Therefore, techniques which correct alignment and restore the path of the instantaneous centre of rotation for joint movement (PICR) (Hall & Brody 1999, Sahrmann 2001) are necessary in most treatment plans. They include:
1. active mobilization/alignment techniques (muscle energy) (Mitchell 2001, Schamberger 2002),

Restoring force closure and motor control

Recent research has increased our understanding of muscle and joint function and consequently changed the way exercises for back pain, dysfunction and stress urinary incontinence are prescribed (Barbic et al 2003, Bergmark 1989, Bullock-Saxton et al 1993, Danneels et al 2000, Hides et al 1994, 1996, Hodges 2000, Hodges 1997, 1999, 2003, Jull & Richardson 2000, Moseley et al 2002, O’Sullivan at al 1997, Richardson et al 1999). New concepts of how joints are stabilized and how load is transferred through the body (including its organs) highlight the importance of proprioception, automatic muscle activity, balance of forces, and motor control for regaining optimal movement after injury. It is clear from this body of evidence that successful rehabilitation of back pain, dysfunction and stress urinary incontinence requires exercises that differ from those used for conditioning and training the healthy, non-painful, non-injured population.

When planning rehabilitation, exercises should be prescribed as part of an integrated treatment plan (Lee & Lee 2004), not as a stand alone treatment. If exercise is prescribed without first restoring joint mobility (form closure), or correcting skeletal alignment, the patient’s pain and dysfunction often gets worse. This may lead to the conclusion that certain exercises are ‘bad’ or ‘unsuccessful’, when it may merely be a problem of inappropriately timed exercise intervention.

Similarly, the type of exercise prescribed is of utmost importance. For back and pelvic pain as well as stress urinary incontinence, the evidence supports correcting deficits in motor control rather than focusing on strength and power of individual muscles (Hodges 2000, 2003, Jull & Richardson 2000, Richardson et al 1999). Patients who go mindlessly through a routine of exercises will have limited success in retraining motor patterns and may get worse with exercise if poor patterns and control are reinforced, resulting in irritation of joint structures and symptom exacerbation. The problem may not be which exercise was prescribed, but how the exercise was performed. Three people performing a squat can do so with three different movement strategies, with three different combinations of muscle recruitment and timing. Therefore, when planning exercise intervention clinicians must remember that ‘exercise A’ does not guarantee the use of ‘muscle A.’ It is up to the
clinician to observe, assess, and decide if ‘exercise A’ is reaching the goal of training ‘muscle A’ (with appropriate recruitment, timing, endurance, etc.) for each patient. The key to correcting dysfunctional patterns of muscle activation is teaching awareness of movement; this requires mindfulness on the part of both the therapist and the patient.

The goal of restoring force closure and motor control for the lumbopelvic-hip region is to restore stabilization strategies and movement patterns such that load transfer is optimized through all joints of the kinetic chain. Optimal load transfer occurs when there is precise modulation of force, coordination, and timing in the local and global systems, ensuring control of the neutral zone for each joint (segmental control), the orientation of the spine (spinal curvatures, thorax on pelvic girdle, pelvis in relation to the lower extremity), and the control of postural equilibrium with respect to the environment (Hodges 2003). The result, and our goal for our patients, is stability with mobility, where there is stability without rigidity of posture, without episodes of collapse, and with fluidity of movement. In addition, the strategy used for stabilization should not induce excessive bladder descent (O’Sullivan et al 2002, 2003). The exercises are prescribed in the context of this goal; the focus is to balance compression and tension forces by using manual cues, imagery, and movement to address alterations in the motor control system.

Optimal coordination of the local and global systems will produce optimal stabilization strategies. These patients will have:

- the ability to find and maintain control of neutral spinal alignment both in the lumbopelvic region and in relationship to the thorax and hip.
- the ability to consciously recruit and maintain a tonic, isolated contraction of the local stabilizers of the lumbopelvis to ensure segmental control (control of the neutral zone) (Hodges 2003, 2004, Richardson et al 1999, O’Sullivan 1997) and to control the position of the bladder and effectively keep the urethra closed.
- the ability to move in and out of neutral spine (flex, extend, laterally bend, rotate) without segmental or regional collapse.
- the ability to maintain all the above in coordination with the thorax and the extremities in functional, work specific, and sport specific postures and movements.

This paper will conclude with a case presentation which demonstrates how we have integrated the related research into an ‘apparently’ effective protocol for the management of patients with both lumbopelvic pain and stress urinary incontinence. The next step is to investigate the efficacy of this integrated approach with a well designed clinical trial.

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