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The integrated continence system: A manual therapy approach to the treatment of stress urinary incontinence

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Abstract

Stress urinary incontinence (SUI) constitutes a large-scale public health concern. The integrated continence system (ICS) developed by the authors is an evidence-based model that demonstrates how urinary incontinence is maintained through the interaction of three structural systems (intrinsic urethral closure, urethral support and lumbopelvic stability) and three modifiable factors (motor control, musculoskeletal and behavioural). The purpose of the ICS is first, to demonstrate the important role that manual physiotherapists can play in the treatment of SUI and second, to guide clinical practice decisions in order to improve clinical outcomes among women with SUI.

Keywords: Stress urinary incontinence; Physiotherapy; Manual therapy; Rehabilitation

1. Introduction

Stress urinary incontinence (SUI) is "the complaint of involuntary leakage on effort or exertion, or on sneezing or coughing" (Abrams et al., 2003). SUI, the most common type of urinary incontinence, represents a large-scale public health concern, affecting 26% of women aged 30–59, peaking in the 40–49 year age group (Cardozo and Stanton, 1980). There is strong evidence for pelvic floor muscle (PFM) training as conservative treatment for SUI (Hay-Smith and Dumoulin, 2006). PFM training however, is commonly associated with improvement rather than cure and the benefits are not necessarily maintained in the long term (Bø et al., 2005). Recent evidence in the literature suggests that there is a multitude of factors, in addition to PFM deficits, that can contribute to the development of SUI. The integrated continence system (ICS) summarizes the impact that deficits in motor control, musculoskeletal and behavioural factors can have on the continence system and highlights the important role for manual physiotherapists in the treatment of SUI.

2. The ICS

The ICS (Fig. 1) illustrates how urinary continence is maintained through the interaction of three structural systems and three modifiable factors. The three structural systems comprise:

- the intrinsic urethral closure system,
- the urethral support system, and
- the lumbopelvic stability system.

These structural systems constitute the basic anatomical structure for urinary continence. They are functionally interdependent and are linked through complex neural control loops and extensive fascial

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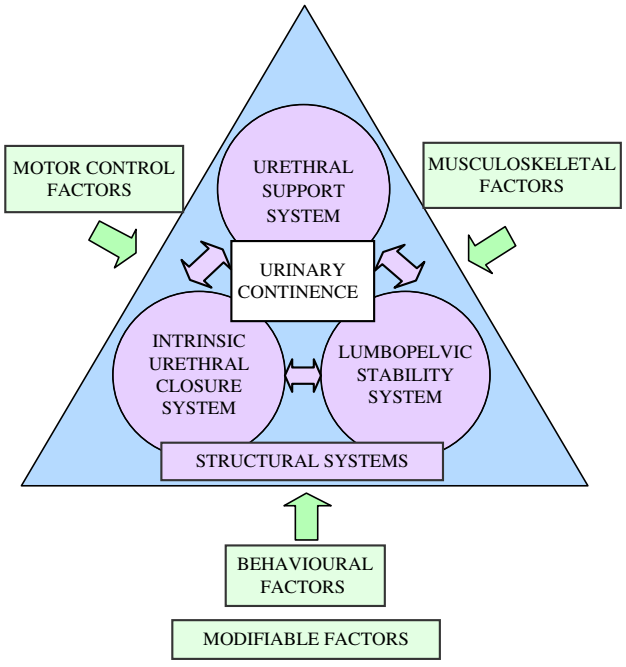

Fig. 1. The integrated continence system (Grewar and McLean, in press). Urinary continence is maintained through the interaction of three structural systems and three modifiable factors.

connections. Deficits in these systems are considered unmodifiable with physiotherapy.

The modifiable factors represent external factors that can influence the overall function of the urinary continence system. The three modifiable factors comprise:

- Motor control factors, where there is evidence to suggest that the following factors may contribute to the development of incontinence:
  - PFM dysfunction,
  - postural and movement dysfunction,
  - low back and pelvic pain, and
  - breathing disorders.
- Musculoskeletal factors, where there is evidence to suggest that the following factors may contribute to the development of incontinence:
  - decreased range of motion,
  - decreased muscle strength, and
  - decreased muscle endurance.
- Behavioural factors, where there is evidence to suggest that the following deficits may contribute to the development of incontinence:
  - chronically elevated intra-abdominal pressure,
  - physical inactivity,
  - abnormal fluid intake and voiding patterns, and
  - poor psychosocial health.

Deficits in the modifiable factors can combine to eventually strain the continence system to the point where symptoms of incontinence emerge, with or without the presence of deficits in the structural systems. Unlike deficits in the structural systems, deficits in the modifiable factors can be addressed through physiotherapy. In many cases, an individualized treatment approach aimed at addressing the key deficits in the modifiable factors may decrease the total load on the continence system to the point where the symptoms of incontinence resolve.

2.1. Structural systems

2.1.1. Intrinsic urethral closure system

The urethra is a fibromuscular tube 3–4 cm in length that extends from the bladder neck to the external urethral meatus. The urethral sphincter (Fig. 2) comprises an internal smooth muscle layer along the upper two-thirds of the urethral length and an external striated muscle layer that is circular around the upper two-thirds of the urethral length and that includes the compressor urethrae and the urethrovaginal sphincter distally (Achtari and Dwyer, 2006). The closure pressure of the urethra is further augmented by a well-developed submucosal vascular plexus (Fig. 3); a watertight mucosal coaptation occurs when the arteriovenous anastomoses fill with blood, creating a hermetic seal. These vessels also have estrogen and progesterone receptors (Achtari and Dwyer, 2006) suggesting a hormonal link.

2.1.2. Urethral support system

The key components of the urethral support system (Fig. 4) include the endopelvic fascia, the anterior vaginal wall, the arcus tendineus fasciae pelvis and the levator ani muscles. The endopelvic fascia is a dense fibrous connective tissue meshwork that envelops and suspends the pelvic viscera in their central location over the levator plate of the pelvic floor. Inferiorly, the urethra is suspended to the pubic bone by the pubourethral ligaments. Posteriorly, the urethra is supported by the vaginal wall and is fused with it at its terminal third. Laterally, the urethra is attached to the pelvic walls by tensile structures known as the arcus tendineus fasciae pelvis that originate at the pubic bone and insert laterally into each ischial spine (DeLancey and Ashton-Miller, 2004). The pelvic floor structure (Fig. 5) is formed primarily by the levator ani muscles and the coccygeus muscles. The levator ani comprise the pubococcygeus, puborectalis and iliococcygeus muscles. The perineal branch of the pudendal nerve, arising from the sacral roots S2–S4, provides the somatic innervation to the levator ani muscles (Achtari and Dwyer, 2006). Contraction of the levator ani elevates the levator plate and moves the bladder neck anteriorly, favouring closure of the urethra (Thompson and O'Sullivan, 2003). This action forms a resistive plate onto which the urethra can be compressed and closed by the

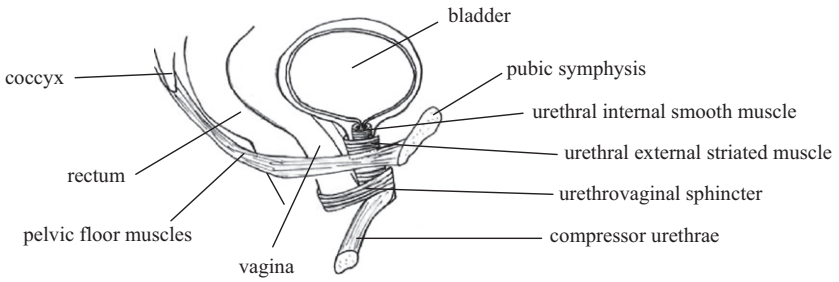

Fig. 2. The urethral sphincter is composed of an internal smooth muscle layer and an external striated muscle layer that includes the compressor urethrae and the urethrovaginal sphincter distally. (Redrawn from Newman 2003.)

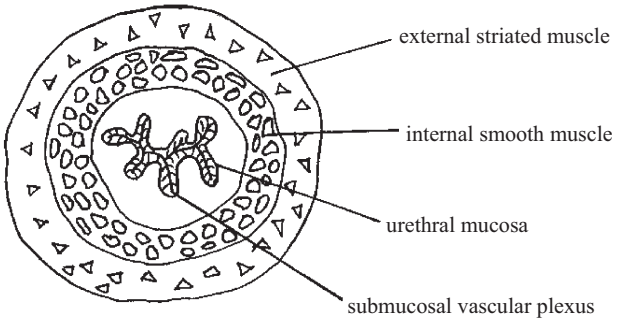

Fig. 3. The intrinsic urethral closure system The urethral striated muscles, the urethral smooth muscles and a vascular plexus within the urethral submucosa contribute to the maintenance of the urethral closure pressure greater than the bladder pressure. (Redrawn from Davila 2006.)

downward forces of intra-abdominal pressure (IAP) (Bø et al., 2001; DeLancey and Ashton-Miller, 2004). For the purposes of this paper, the levator ani will be referred to as the "PFM".

2.1.3. Lumbopelvic stability system

Optimal control of the lumbar spine and pelvis requires intact osseoligamentous factors (form closure), appropriate muscle compression forces (force closure) and control by the nervous system. The osseoligamentous structures comprise the inert structures of the lumbar spine, symphysis pubis and sacroiliac joints. The arch-like arrangement of the bones of the pelvis allows for the transfer of weight from the upper body through the lumbosacral spine, across the linea terminalis to the femoral heads (Retzky et al., 1996). Control of the lumbar spine and pelvis is dependent on the local muscle system (Fig. 6) consisting of the diaphragm (Hodges et al., 1997), the transversus abdominis (Hodges et al., 1999), the PFM (Hodges et al., 2002) and the lumbar multifidus (Bogduk et al., 1992). The coordinated activity of these muscles influences postural control by regulating IAP (Hemborg et al., 1985) and by increasing the tension in the thoracolumbar fascia (Tesh et al., 1987). For a given task, the nervous system must be able to evaluate the requirements for lumbopelvic control, determine the current status of the lumbopelvic region and develop strategies to meet those demands (Hodges, 2006).

3. Pathophysiology of SUI

The pathophysiology of SUI is complex and multifactorial. While the exact mechanisms of SUI have not yet been fully elucidated, it appears that SUI can develop as a result of one or multiple deficits within the structural systems, the modifiable factors or both. Although intact components of the continence mechanism can compensate for deficient ones, it appears that in each individual there is a threshold at which multiple factors combine to eventually strain the continence system to the point where symptoms of SUI emerge (Weber et al., 2004).

3.1. Structural system deficits

3.1.1. Urethral closure system deficits

Intrinsic sphincteric deficiency is caused by a reduced ability of the intrinsic urethral sphincteric mechanism to maintain mucosal coaptation either at rest or in the presence of minimal physical stress. It is commonly associated with multiple anti-incontinence procedures, radical pelvic surgery or radiation, menopause or urogenital atrophy (Vecchioli-Scaldazza and Morosetti, 2006).

3.1.2. Urethral support system deficits

Labour or vaginal delivery may cause direct damage to the PFM, such as avulsions of the inferomedial aspect of the pubococcygeus–puborectalis complex from its insertion on the arcus tendineus fascia pelvis (Dietz and Lanzarone, 2005) or damage to the nerve supply of the PFM. Stretching or tearing of the fascial supports for the urethra and the bladder can result in hypermobility of the urethra and bladder. In the case where the bladder

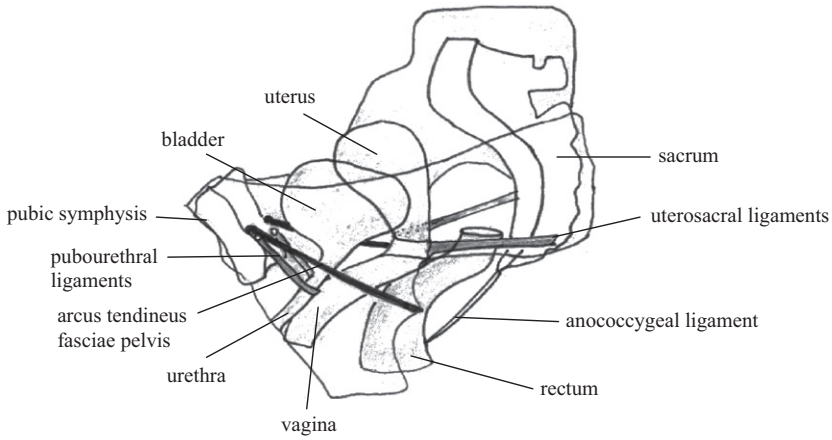

Fig. 4. The urethral support system. The primary supports to the urethra include the endopelvic fascia (not shown), the pubourethral ligaments, the vagina and the arcus tendineus fasciae pelvis. (Redrawn from Petros 2004.)

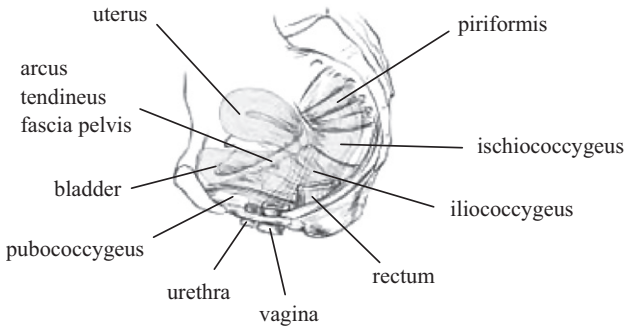

Fig. 5. The pelvic floor muscles (the pubococcygeus, the iliococcygeus and the ischiococcygeus) and their relationship with the fascia and pelvic organs. (Reproduced with permission from Diane G. Lee Physiotherapist Corp.)

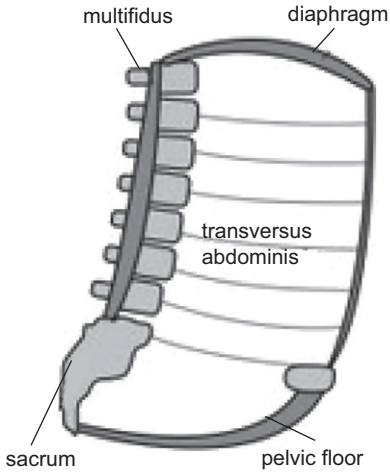

Fig. 6. The local muscles of the lumbopelvic region consist of the pelvic floor, the diaphragm, the transversus abdominis and the deep fibers of multifidus. (Reproduced with permission from Diane G. Lee Physiotherapist Corp.)

is allowed to descend as a result of a loss of support, the internal sphincter can be pulled open and the closure pressure to the urethra can be compromised (Hines and Miller, 2006). Aging, estrogen deficiency and connective tissue abnormalities may also contribute to decreased strength of the connective tissues of the PFM (Schaffer et al., 2005). If the fascia of the pelvic floor becomes overdistended, the perineum may descend over time and the PFM may lengthen and occupy a lower position in the pelvic cavity (Bø, 2004).

3.1.3. *Lumbopelvic stability system deficits*

Osseoligamentous deficits in the lumbopelvic-hip region may include joint pathology such as osteoarthritis, stenosis or joint instability. There is no evidence linking the above deficits to SUI; asymmetric sacroiliac joint laxity has however, been associated with postpartum pelvic pain (Damen et al., 2002). The association between diastasis rectus and SUI also has not been investigated; however, diastasis rectus has been linked with pubic pain and myofascial pain in the abdomen and PFM (Baker, 1998). O'Sullivan et al. (2002) demonstrated that the application of a manual compression force through the pelvis eliminated the increased descent of the bladder and PFM seen among subjects with sacroiliac joint pain ($n = 13$) during a supine active straight leg raise test. These findings provide support for the hypothesis that SUI may, in fact, be a consequence of failed load transfer through the lumbopelvic region (Lee, 2004).

3.2. *Modifiable factor deficits*

3.2.1. *Motor control factors*

3.2.1.1. *PFM dysfunction*

3.2.1.1.1. Poor PFM awareness and ability to perform a correct contraction. It is well established that many women are unable to perform a correct PFM contraction. In many women, a Valsalva manoever (a straining strategy) can cause depression rather than elevation of the levator plate (Thompson and O'Sullivan, 2003). Fig. 7c illustrates the resultant caudodorsal movement

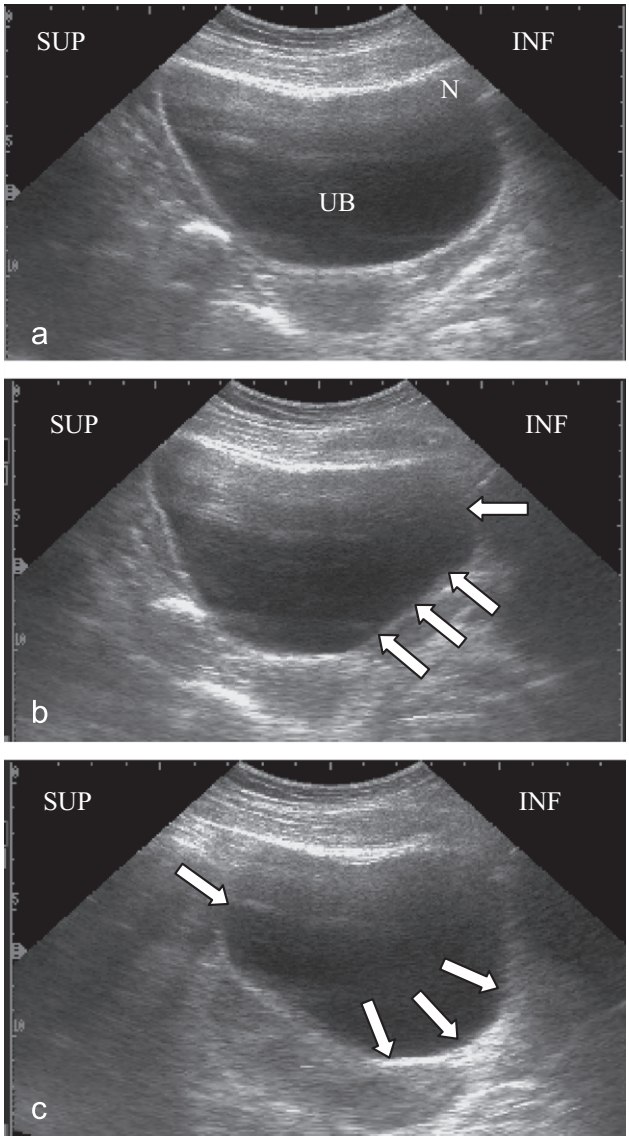

Fig. 7. Ultrasound image of the bladder (parasaggital view). (a) Bladder at rest. UB, urinary bladder; N, neck of the bladder; SUP, superior; INF, inferior. (b) Correct recruitment of the PFM results in an indentation of the posteroinferior aspect of the bladder (arrows) and a cranioventral shift (arrow). (c) A Valsalva maneuver results in a deformation of the bladder shape (arrows) and a caudodorsal shift (arrow). (Reproduced with permission from Diane G. Lee Physiotherapist Corp.)

of the bladder during a Valsalva manoever. Over time, this strategy can be detrimental; repetitive depression of the levator plate may have long-term implications for prolapse and incontinence (Thompson and O'Sullivan, 2003). A global contraction of the abdominal, gluteal and hip adductor muscles is another common pattern observed when women attempt to contract their PFM. In a study carried out by Morin et al. (2004), women with SUI ($n = 59$) were found to demonstrate a slower rate of force production ($p = 0.012$) and a lower capacity to generate repeated maximal PFM contractions within a 15 s period ($p = 0.011$), as compared to continent women ($n = 30$). These findings highlight the importance of confirming a correct PFM contraction and taking into account parameters other than strength, such as the rate of force production and the ability to perform repeated fast contractions.

3.2.1.1.2. Delayed or absent PFM automatic activity. As noted above, PFM activity has yet to be studied in a rigorous manner during normal functional activities. There is weak evidence to show that women with SUI demonstrate asymmetric or delayed PFM activation during coughing (Deindl et al., 1994; Barbič et al., 2003). Preliminary findings from more recent electromyography studies however, indicate that some women with mild symptoms of SUI actually activate their PFM to a larger extent when coughing (Goncalves et al., 2006) as compared to their continent counterparts. A randomized control trial that investigated the effects of teaching women to do a PFM contraction prior to and during a cough was found to effectively reduce urine loss by an average of 98.2% ($p = 0.009$) and 73.3% ($p = 0.003$), during medium and strong coughs, respectively, among 27 women with mild to moderate SUI (Miller et al., 1998). This finding suggests that the timing of the PFM contraction is a key factor in preventing leakage episodes.

3.2.1.1.3. Altered PFM and abdominal muscle co-activation. The PFM have been shown to co-activate with the abdominal muscles during PFM contractions and during different abdominal maneuver across several studies in small samples of healthy continent volunteers (Sapsford and Hodges, 2001; Sapsford et al., 2001; Neumann and Gill, 2002; Madill and McLean, 2004). A number of authors report coordination problems between the PFM and the lower abdominal muscles during coughing among incontinent women (Devreese et al., 2004; Sapsford, 2004); however, to date, the normal coordination pattern is not fully understood.

3.2.1.2. Postural and movement dysfunction. There is evidence that the alignment of the lumbar spine and pelvis may affect PFM and trunk muscle co-activation. Sapsford et al. (2001) found that a voluntary PFM contraction in lumbar extension produced the greatest increase in transversus abdominis activity relative to the activity recorded in the rectus abdominus and oblique abdominal muscles ($n = 7$). Further validation is required to confirm how spinal alignment affects muscle activation patterns. A neutral spinal posture (cervical lordosis, thoracic kyphosis, lumbar lordosis and neutral pelvis) (Fig. 8a) is encouraged clinically as it has been found to improve the recruitment of the deep trunk stabilizing muscles (Lee and Lee, 2004a). In theory, a neutral spinal posture also allows some of the downward force of the abdominal contents to be absorbed at the pubis, helping to unload the PFM and the

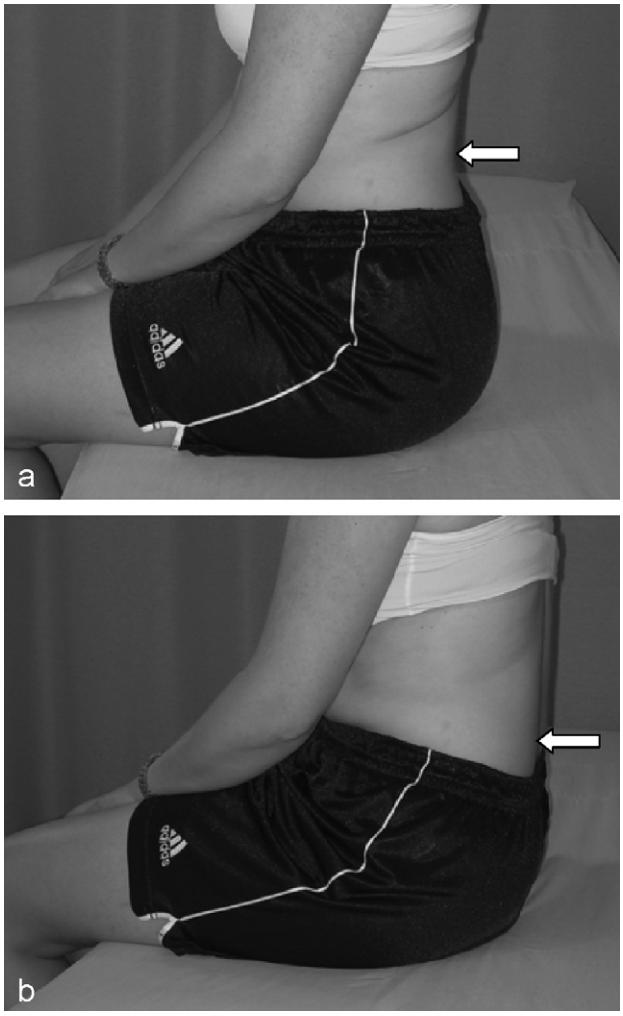

Fig. 8. (a) A neutral spinal posture in the lumbopelvic region produces a lumbar lordosis (arrow). (b) A posteriorly tilted pelvis produces a flexed lumbar spine (arrow).

endopelvic fascia. A habitual posteriorly tilted pelvis (Fig. 8b) is thought to cause increased vertical loading on the PFM and increased risk for stretch weakness from repetitive activities such as running or aerobics (Spitznagle, 2006). Inadequate force closure in the lumbopelvic region during an active straight leg raise may result in altered motor responses such as PFM descent and/or splinting of the diaphram (O'Sullivan et al., 2002). Real-time ultrasound imaging allows the visualization of the bladder during loading. Ideally, the local muscle system should co-contract and the bladder should remain relatively stationary (Whittaker, 2004).

3.2.1.3. Low back and pelvic pain. A recent large cross-sectional study indicated that disorders of continence and breathing are strongly associated with frequent back pain (Smith et al., 2006). The findings provide preliminary support for the hypothesis that reduced function of the diaphragm, abdominal muscles and PFM among individuals with incontinence and respiratory disease may compromise postural control and may contribute to the development of back pain. Many of the musculoskeletal structures in the back and lower extremities share their segmental innervation (S2–S4) with the urogenital structures, and therefore, some PFM dysfunctions may be related to denervation injuries. It is thought that increased activity of the superficial trunk muscles seen in patients with back pain may restrict the expansion of the rib cage and abdomen (Hodges, 2006), increase the IAP and potentially contribute to stretch weakness of the PFM. The ability to retrain the PFM may also be compromised in the presence of low back or pelvic pain due to the fact that the PFM may be compensating for a dysfunction in other components of the muscle system (Hodges, 2006).

3.2.1.4. Breathing disorders. As noted above, the links between respiratory disorders, back pain and incontinence suggest that coordinated activity of the muscles that surround the abdominal cavity is required for optimal function of breathing, postural control and continence. Respiratory dysfunctions are commonly seen in patients with low back pain, pelvic floor dysfunction and poor posture (Carrière, 2006). Disrupted function of either the diaphragm or the PFM may alter the normal mechanisms for regulating IAP (Carrière, 2006). The increased frequency of coughing associated with certain respiratory conditions may increase the demands on the PFM.

3.2.2. Musculoskeletal factors

3.2.2.1. Decreased range of motion. Articular, muscle or fascial restrictions in the lumbopelvic-hip region or thorax may contribute to the development of pain, compensatory movement patterns and maladaptive postures. Soft tissue scarring in the perineal area and abdominal cavity resulting from tearing after prolonged labor, episiotomies, the use of forceps, caesarian sections and surgical procedures in the abdominal cavity may contribute to pain and limited mobility of the affected structures (Carrière, 2006). Scar tissue or adhesions following conditions such as endometriosis may affect the function, position and motility of the abdominal or pelvic viscera. In cases of dysfunction, mobilization of the pelvic viscera, specifically the bladder, uterus and rectum, by a health professional trained in visceral mobilization may improve the function of the pelvic floor (Vleminckx, 2006).

3.2.2.2. Decreased muscle strength. There are controversies in the literature about whether PFM strength differs between continent and incontinent women. Many discrepancies may be related to differences in the instruments used, the techniques employed or the

definition of continence status. To date, there is no strong evidence indicating that women with SUI demonstrate clinically significant lower mean maximal PFM strength values. Efficacy studies do however, provide some support for PFM training in the treatment of women with urinary incontinence. Statistical heterogeneity with respect to variations in incontinence type, training and outcome measures make the results difficult to interpret (Hay-Smith and Dumoulin, 2006). Intensive PFM strength training is believed to cause hypertrophy and increase the stiffness of the PFM and connective tissues, to improve recruitment efficiency, to elevate the levator plate position to a higher position inside the pelvis and ultimately, to facilitate an automatic PFM contraction during rises in IAP (Bø, 2004).

3.2.2.3. Decreased muscle endurance. Devreese et al. (2004) and Morin et al. (2004) found significantly decreased PFM endurance in incontinent women as compared to continent women ($p = 0.006$ and 0.001, respectively). Neither of the studies however, reflect the tonic activity of the PFM during functional activities since endurance during repeated maximal PFM contractions was assessed rather than a more functional sustained or repeated submaximal contraction.

3.2.3. Behavioural factors

3.2.3.1. Chronically elevated intra-abdominal pressure. Chronically elevated IAP may place excessive loads on the PFM. Transient high IAP is associated with activities such as coughing, lifting, opera singing, aerobic exercise and high-impact activities (Hines and Miller, 2006). Chronically elevated IAP is found in women who are overweight (Hines and Miller, 2006). Interestingly, Subak et al. (2005) showed that a weight reduction program consisting of exercise, decreased caloric intake and education is an effective treatment for stress, urge, and mixed urinary incontinence in overweight and obese women. In this case however, it is not possible to determine the separate effects of weight loss, exercise and behavioural modification on the results. Cigarette smoking alone has been found to increase the risk of genuine SUI in women by 2.5-fold, independent of other risk factors (Bump and McClish, 1994). Possible reasons for this may include the more frequent coughing among smokers, the anti-estrogenic effects associated with smoking (Baron et al., 1990) or the interference with collagen synthesis (Last et al., 1990).

3.2.3.2. Physical inactivity. According to the results of two large cross-sectional population-based studies, women with more frequent leakage episodes are more likely to report low levels of physical activity (Brown and Miller, 2001) and women who participate in low-intensity activities for 1 hour or more per week report less incontinence than their sedentary counterparts (Hannestad et al., 2003). It is unclear from the findings whether low-intensity activity produces a protective effect on incontinence or whether incontinent women tend to stop exercising because of leakage symptoms. Depression, chronic low back or pelvic pain and respiratory disorders may also constitute barriers to regular exercise.

3.2.3.3. Abnormal fluid intake and voiding patterns. Women who drink excessive amounts of fluids may experience urinary leakage as a direct result of placing an unnecessary overload on the bladder. In contrast, women who intentionally limit their fluid intake can develop bladder irritation and constipation (Hines and Miller, 2006). Caffeine, carbonated beverages, alcohol, decaffeinated coffee or tea, and artificial sweeteners may contribute to symptoms of urinary incontinence (Hines and Miller, 2006). Repetitive straining at defecation associated with chronic constipation has been linked to damage of the innervation of the puborectalis muscle (Snooks et al., 1985). The accumulation of stool in the rectum as a result of constipation can alter the position of the pelvic organs and press on the bladder, potentially reducing its capacity to hold urine (Hines and Miller, 2006).

3.2.3.4. Poor psychosocial health. The experience of incontinence may be associated with feelings of anxiety and social withdrawal (Hines and Miller, 2006). These and other emotions may influence posture and motor control (Umphred, 2006) via the limbic system. There is also undoubtedly a link between social withdrawal, fitness, nutrition, smoking status and incontinence, whereby it is difficult to assess the causal factors relative to the effects.

4. Implications for clinical practice

The purpose of the ICS is (i) to assist physiotherapists to identify and prioritize the key deficits in the modifiable factors, (ii) to develop treatment strategies that effectively target the key deficits in the modifiable factors and, (iii) to guide clinical practice decisions in order to improve clinical outcomes among women with SUI. Tables 1, 2 and 3 outline the key modifiable factor deficits among women with SUI: deficits in motor control factors, deficits in musculoskeletal factors and deficits in behavioural factors, respectively. Each table includes the key subjective and objective assessment findings for each deficit and highlights clinical recommendations to guide clinical practice decisions. Reference can be made to the sources cited for further reading.

It is important to note that the clinical recommendations do not constitute evidence-based intervention approaches since they simply reflect, for the most part, the results of case-control studies. Each individual presenting with SUI will present with a different cluster of factors that is contributing to their symptoms;

Table 1
Deficits in motor control factors

Key subjective findings	Key objective findings	Clinical recommendations
PFM dysfunction		
Poor PFM awareness and ability to perform a correct contraction		
Absent sensation of a voluntary PFM contraction	Absent palpable contraction of the PFM	Electrical stimulation, biofeedback and/or cones are recommended for women who cannot voluntarily contract the PFM (Laycock et al., 2001) Manual techniques can be used to facilitate a PFM contraction (Brown, 2006)
Poor understanding of how to perform a correct PFM contraction	Depression of the levator plate or a global contraction of the hip abductor adductor and/or gluteal muscles	Digital palpation of the PFM is the recommended technique for physiotherapists to teach and give feedback to clients about the correctness of the PFM contraction (Bø and Sherburn, 2005) Real-time ultrasound is increasingly being used clinically as an assessment and biofeedback tool (Bø and Sherburn, 2005; Whittaker, 2004) Teach clients to monitor the PFM contraction by (i) palpating the upward movement of the perineum using external hand contact (Sapsford, 2004), (ii) palpating the co-contraction of the transversus abdominis muscle (Lee and Lee, 2004a, Chapter 8) and/or (iii) palpating the PFM digitally Teach strategies to encourage functional integration of the PFM (Gödl-Purrer, 2006, Lee and Lee, 2004b, Chapter 10)
Poor ability to contract the PFM quickly and/or perform repeated fast maximal PFM contractions	Decreased rate of PFM force production and/or decreased contractile force with repeated fast maximal PFM contractions	Train fast maximum PFM contractions followed by complete relaxation with repetitions to the point of fatigue
Delayed or absent PFM automatic activity		
Urinary leakage during coughing	Palpable descent of the perineum during coughing (Sapsford, 2004)	Train a voluntary PFM contraction immediately prior to and during coughing (Miller et al., 1998), nose blowing, sneezing, laughing (Sapsford, 2004) and other activities where leakage is anticipated
Altered PFM and abdominal co-activation		
Descent of the perineum and bulging of the abdominal wall during coughing	Palpable descent of the perineum and bulging of the abdominal wall during coughing (Sapsford, 2004)	Train a coordinated PFM and abdominal co-contraction during coughing and other functional expiratory patterns (Sapsford, 2004)
Postural and movement dysfunction		
Poor postural awareness	Loss of the neutral spinal posture during functional postures and movements	Retrain a neutral spinal posture during functional postures and movements (Hides et al., 2004, Chapter 14; Lee and Lee, 2004b, Chapter 10)
Muscle weakness in the lumbopelvic-hip region	Active straight leg raise with loss of lumbopelvic stability (Lee and Lee, 2004a, Chapter 8) One-leg standing with failed load transfer through the pelvis or hip (Lee and Lee, 2004a, Chapter 8)	Restore adequate form closure, force closure and motor control in the lumbopelvic-hip region (Lee and Lee, 2004b, Chapter 10). Train the PFM as part of a progressive lumbopelvic stabilization program (Hodges, 2003; Hides et al, 2004, Chapter 14; Lee and Lee, 2004b, Chapter 10; Richardson and Hides, 2004a, b, Chapters 15 and 16; Richardson et al., 2004, Chapter 13; Sapsford, 2004)
Low back and pelvic pain		
Low back or pelvic pain	Dysfunction of form closure, force closure, and/or motor control in the lumbopelvic-hip region (Lee and Lee, 2004a, Chapters 8 and 9)	Restore adequate form closure, force closure and motor control in the lumbopelvic-hip region (Hodges, 2003; Hides et al., 2004, Chapter 14; Lee and Lee, 2004b, Chapter 10; Richardson and Hides, 2004a, b, Chapters 15 and 16; Richardson et al., 2004, Chapter 13; Sapsford, 2004)

Table 1 (*continued*)

Key subjective findings	Key objective findings	Clinical recommendations
Breathing disorders		
Shallow breathing or poor awareness of breathing pattern	Apical breathing or breath holding	Train a correct diaphragmatic breathing pattern (Hides et al., 2004, Chapter 14; Lee and Lee, 2004b, Chapter 10; Sapsford, 2004)
	Signs of excessive global muscle activity (Hides et al., 2004, Chapter 14; Lee and Lee, 2004b, Chapter 10)	Teach strategies to reduce excessive global muscle activity (Hides et al., 2004, Chapter 14; Lee and Lee, 2004b, Chapter 10)
	Poor coordination of breathing with local muscle co-contraction	Train tonic co-contraction of the local muscle system while maintaining normal breathing (Hides et al., 2004, Chapter 14; Lee and Lee, 2004b, Chapter 10)

Table 2
Deficits in musculoskeletal factors

Key subjective findings	Key objective findings	Clinical recommendations
Decreased range of motion		
Pain and/or stiffness in the lumbopelvic-hip region or perineum	Articular, muscle or fascial restrictions in the lumbopelvic-hip region (Lee, 2004, Chapter 9)	Restore the normal articular, muscle and fascial mobility in the lumbopelvic-hip region (Lee and Lee, 2004b, Chapter 10)
	Articular or soft tissue restrictions within the pelvic floor (Brown, 2006)	Restore the mobility of the sacrococcygeal joint and connective tissue structures in the pelvic floor (Brown, 2006)
		Refer to a therapist who performs visceral mobilization if visceral involvement is suspected (Vleminckx, 2006)
Decreased muscle strength		
Weak PFM contraction and/or reduced ability to generate repeated sustained maximal PFM contractions	Grades 0, 1 or 2 on the MOS[a]	Electrical stimulation, biofeedback, and/or cones are recommended for grades 0–2 on the MOS (Laycock et al., 2001)
	Grade 3 or greater on the MOS	PFM strength training is recommended for grades 3 or greater on the MOS: 8–12 maximum repetitions (Kraemer et al., 2002) sustained to the point of fatigue (Laycock et al., 2001) 2–4 times per week (Kraemer et al., 2002; McArdle et al., 2007) for 15–20 weeks (Laycock et al., 2001)
	Contractile force decreases with repeated sustained maximal PFM contractions	Continue the PFM exercises as part of maintenance program (Laycock et al., 2001)
Decreased muscle endurance		
Poor PFM endurance	Reduced ability to sustain a tonic PFM contraction and/or perform repeated contractions	Train repeated sustained tonic holds of the PFM to the point of fatigue (Laycock et al., 2001; Lee and Lee, 2004b, Chapter 10; Sapsford, 2004)

[a]Modified Oxford Scale (Laycock, 1994). (See Appendix A.)

physiotherapists therefore, need to tailor the exercise program and goal setting to each individual. With respect to PFM training, there is no standard exercise prescription that would provide optimal strength gains across different individuals. Instead, physiotherapists need to apply to each individual the principles of motor learning (Gödl-Purrer, 2006; Umphred, 2006) and strength training including exercise of sufficient intensity, repetitions, frequency and duration to bring about a training change (Kraemer et al., 2002; McArdle et al., 2007). In cases where there are severe structural deficits, the effectiveness of the ICS may be limited and surgical intervention may be indicated.

5. Conclusions

Manual physiotherapists have the knowledge base and skills required to assess the multitude of factors that can contribute to SUI and to develop and implement effective treatment strategies. The ICS represents a unique integrated treatment approach that takes into account the key motor control, musculoskeletal and

Table 3

Deficits in behavioural factors

Key subjective findings	Key objective findings	Clinical recommendations
Chronically elevated intra-abdominal pressure		
Overweight or obese	BMI $\geqslant 25\,kg/m^2$ or $\geqslant 30\,kg/m^2$, respectively (Lau et al., 2007)	Provide the client with information regarding a structured weight management program (Lau et al., 2007)
Cigarette smoking	Self-report increased frequency of coughing	Provide the client with information regarding a smoking cessation program
Physical inactivity		
Reduced regular physical activity	Exercise diary indicates reduced regular physical activity	Identify barriers to physical activity (Brown and Miller, 2001) and encourage a regular structured physical activity program (Haskell et al., 2007)
Abnormal fluid intake and voiding patterns		
Excessive or reduced fluid intake, habitual preventative emptying of the bladder, regular consumption of caffeinated products, and/or constipation with repetitive straining during defecation	Bladder diary indicates excessive or reduced fluid intake, increased frequency of voiding, regular consumption of caffeinated products, and/or regular straining during defecation	Encourage an approximate fluid intake of 1.5 Litres per day (Ospelt, 2006) with voiding approximately every 3–4 hours during the day (Hines and Miller, 2006) Provide education regarding the reduction or elimination of caffeinated products and other potential bladder irritants (Hines and Miller, 2006) Provide education regarding sources of dietary fiber, regular fluid intake and effective defecation strategies (Ospelt, 2006)
Poor psychosocial health		
Feelings of anxiety or social withdrawal	Feelings of anxiety or social withdrawal identified on self-report outcome measures	Provide the treatment in a supportive and motivating environment Set attainable goals that minimize lifestyle changes, provide education and positive feedback at every visit (Laycock et al., 2001) and encourage the client to participate in group classes (Gödl-Purrer, 2006)

behavioural factors that can influence the continence system. At this point, the ICS constitutes a preliminary framework—confirmatory research is still lacking within many subgroups of the model and the ICS needs to be scientifically evaluated before it can be accepted as a valid conservative treatment approach for women with SUI. That said, the model reflects the current state of knowledge and is a logical approach to the treatment of SUI. Its application to other presentations such as urge and mixed incontinence, prolapse and incontinence in males needs to be determined. The effectiveness of prevention, which to date has been sparsely documented, constitutes another important area for future research.

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Appendix A. Modified Oxford Scale

0	No contraction
1	Flicker
2	Weak
3	Moderate
4	Good (with lift)
5	Strong

Laycock (1994).

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