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CRITICAL REVIEW

The myth of core stability

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Core stability;
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Summary The principle of core stability has gained wide acceptance in training for the prevention of injury and as a treatment modality for rehabilitation of various musculoskeletal conditions in particular of the lower back. There has been surprisingly little criticism of this approach up to date. This article re-examines the original findings and the principles of core stability/spinal stabilisation approaches and how well they fare within the wider knowledge of motor control, prevention of injury and rehabilitation of neuromuscular and musculoskeletal systems following injury.

Introduction

Core stability (CS) arrived in the latter part of the 1990s. It was largely derived from studies that demonstrated a change in onset timing of the trunk muscles in back injury and chronic lower back pain (CLBP) patients (Hodges and Richardson, 1996, 1998). The research in trunk control has been an important contribution to the understanding of neuromuscular reorganisation in back pain and injury. As long as four decades ago it was shown that motor strategies change in injury and pain (Freeman et al., 1965). The CS studies confirmed that such changes take place in motor control of the trunk muscles of patients who suffer from back injury and pain.

However, these findings combined with general beliefs about the importance of abdominal muscles for a strong back, and influences from Pilates, have promoted several assumptions prevalent in CS training:

- That certain muscles are more important for stabilisation of the spine than other muscles, in particular transversus abdominis (TrA).
- That weak abdominal muscles lead to back pain
- That strengthening abdominal or trunk muscles can reduce back pain
- That there is a unique group of ''core'' muscle working independently of other trunk muscles
- That back pain can be improved by normalising the timing of core muscles
- That there is a relationship between stability and back pain

As a consequence of these assumptions, a whole industry grew out of these studies with gyms and clinics worldwide teaching the ''tummy tuck'' and trunk bracing exercise to athletes for prevention of injury and to patients as a cure

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for lower back pain (Jull and Richardson, 2000; Richardson et al., 2002). In this article some of these basic assumptions will be re-examined.

Assumptions about stability and the role of TrA and other core muscle

In essence the passive human spine is an unstable structure and therefore further stabilisation is provided by the activity of the trunk muscles. These muscles are often referred to in the CS approach as the ''core'' muscles, assuming that there is a distinct group, with an anatomical and functional characteristics specifically designed to provide for the stability. One of the muscles in this group to have received much focus is TrA. It is widely believed that this muscle is the main anterior component of trunk stabilisation. It is now accepted that many different muscles of the trunk contribute to stability and that their action may change according to varying tasks (see further discussion below).

The TrA has several functions in the upright posture. Stability is one, but this function is in synergy with every other muscle that makes up the abdominals wall and beyond (Hodges et al., 1997, 2003; Sapsford et al., 2001). It acts in controlling pressure in the abdominal cavity for vocalisation, respiration, defecation, vomiting, etc. (Misuri et al., 1997). TrA forms the posterior wall of the inguinal canal and where its valve-like function prevents the viscera from popping out through the canal (Bendavid and Howarth, 2000).

How essential is TrA for spinal stabilisation? One way to assess this is to look at situations where the muscle is damaged or put under abnormal mechanical stress. Would this predispose the individual to lower back pain?

According to Gray's Anatomy (36th edition 1980, page 555) TrA is absent or fused to the internal oblique muscle as a normal variation in some individuals. It would be interesting to see how these individuals stabilise their trunk and whether they suffer more back pain.

Pregnancy is another state that raises some important questions about the role of TrA or any abdominal muscle in spinal stabilisation. During pregnancy the abdominal wall muscles undergo dramatic elongation, associated with force losses and inability to stabilise the pelvis against resistance (Fast et al., 1990; Gilleard and Brown, 1996). Indeed, in a study of pregnant women ($n = 318$) they were shown to have lost the ability to perform sit-ups due to this extensive elongation and subsequent force losses (Fast et al., 1990). Whereas all non-pregnant women could perform a sit-up, 16.6% of pregnant women could not perform a single sit-up. However, there was no correlation between the sit-up performance and backache, i.e. *the strength of abdominal muscle was not related to backache*. Despite this, CS exercises are often prescribed as a method for retraining the abdominal muscles and ultimately as a treatment for LBP during pregnancy. There is little evidence that localised musculoskeletal mechanical issues, including spinal stability play a role in the development of LBP during pregnancy. Often cited predisposing factors are, for example, body mass index, a history of hypermobility and amenorrhea (Mogren and Pohjanen, 2005), low socioeconomic class, existence of previous LBP, posterior/fundal location of the placenta and a significant correlation between foetal weight and LBP (Orvieto et al., 1990).

Another interesting period for us, concerning the role of abdominal muscles and stabilisation is immediately after delivery. Postpartum, it would take the abdominal muscle about 4–6 weeks to reverse the length changes and for motor control to reorganise. For example, rectus abdominis takes about 4 weeks postpartum to re-shorten, and it takes about 8 weeks for pelvic stability to normalise (Gilleard and Brown, 1996). It would be expected that during this period there would be minimal spinal support/stabilisation from the slack abdominal muscles and their fasciae. Would this increase the likelihood for back pain?

In a recent study, the effects of a cognitive-behavioural approach were compared with standard physiotherapy on pelvic and lower back pain immediately after delivery (Bastiaenen et al., 2006). An interesting aspect of this research was that out 869 pregnant women suffering from back pain during pregnancy, 635 were excluded because of their spontaneous unaided recovery within a week of delivery. This spontaneous recovery was during a period, well before the abdominal muscles had time to return to their pre-pregnancy length, strength or control (Gilleard and Brown, 1996). Yet, this was a period when back pain was dramatically reduced. How can it be that back and pelvic pain is improving during a period of profound abdominal muscle inefficiency? Why does the spine not collapse? Has the relationship between abdominal muscles and spinal stability been over-emphasised?

Similarly studies on weight gain and obesity and LBP challenge the CS theory. One would expect, as in pregnancy, the distension of the abdomen to disrupt the normal mechanics and control of the trunk muscles, including TrA. According to the CS model this should result in an increased incidence of back pain among this group. Yet, epidemiological studies demonstrate that weight gain and obesity are only weakly associated with lower back pain (Leboeuf-Yde, 2000) According to the CS model we should be seeing an epidemic of back pain in over-weight individuals.

Another area that can shed light on the control and stability of the abdominal muscles is the study of abdominal muscles that have been damaged by surgery. Would such damage affect spinal stability or contribute to back pain? In breast reconstruction after mastectomy, one side of the rectus abdominis is used for reconstruction of the breast. Consequently, the patient is left with only one side of rectus abdominis and weakness of the abdominal muscles. Such alteration in trunk biomechanics would also be expected to result in profound motor control changes. Despite all these changes there seems to be no relationship to back pain or impairment to the patient's functional/movement activities, measured up to several years after the operation (Mizgala et al., 1994; Simon et al., 2004).

One area for further study would be that of subjects who have had inguinal hernia repair. In this operation the TrA is known to be affected by the surgical procedure (Berliner, 1983; Condon and Carilli, 1994). To date there is no known epidemiological study linking such surgery and back pain.

In summary we can conclude:

- That abdominal musculature can demonstrate dramatic physiological changes, such as during pregnancy, postpartum and obesity, with no detriment to spinal stability and health.
- Damage to abdominal musculature does not seem to be detrimental to spinal stability or contribute to LBP.
- No study to date has demonstrated that LBP is due to spinal instability. Despite a decade of research in this area it remains a theoretical model.

The timing issue

In one of the early studies it was demonstrated that during rapid arm/leg movement, the TrA in CLBP patients had delayed onset timing when compared with asymptomatic subjects (Hodges and Richardson, 1996, 1998). It was consequently assumed that the TrA, by means of its connection to the lumbar fascia, is dominant in controlling spinal stability (Hodges et al., 2003). Therefore any weakness or lack of control of this muscle would spell trouble for the back.

This assumption is a dramatic leap of faith. Firstly, in our body all structures are profoundly connected in many different dimensions, including anatomically and biomechanically. You need a knife to separate them from each other. It is not difficult to emphasise a connection that would fit the theory, i.e. that the TrA is the main anterior muscle that controls spinal stability. In normal human movement postural reflexes are organised well ahead in anticipation of movement or perturbation to balance. TrA is one of the many trunk muscles that takes part in this anticipatory organisation (Hodges and Richardson, 1997). Just because in healthy subjects it kicks off before all other anterior muscles (in one particular posture), does not mean it is more important in any way. It just means it is the first in a sequence of events (Cresswell et al., 1994a,b). Indeed, it has been recently suggested that earlier activity of TA may be a compensation for its long elastic anterior fasciae (Macdonald et al., 2006).

It can be equally valid to assume that a delay in onset timing in subjects with LBP may be an advantageous protection strategy for the back rather than a dysfunctional activation pattern. Furthermore, it could be that during the fast movement of the outstretched arm the subject performed a reflexive pain evasion action that involved delayed activation of TrA, an action unrelated to stabilisation (Moseley et al., 2003a,b, 2004). An analogy would be the reflex pulling of the hand from a hot surface. One could imagine that a patient with a shoulder injury would use a different arm withdrawal pattern from a normal individual. This movement pattern would be unrelated to the control of shoulder stability but would be intended to produce the least painful path of movement, even if the movement is not painful at the time. A similar phenomenon has been demonstrated in trunk control where just the perception of a threat of pain to the back resulted in altered postural strategies (Moseley and Hodges, 2006).

In the original studies of CS onset time differences between asymptomatic individuals and patients with CLBP were about 20 ms, i.e. *one fiftieth of a second difference* (Hodges and Richardson, 1996, 1998; Radebold et al., 2000). It should be noted that these were not strength but timing differences. Such timings are well beyond the patient's conscious control and the clinical capabilities of the therapist to test or alter.

Often, in CS exercise there is an emphasis on strength training for the TrA or low velocity exercise performed lying or kneeling on all fours (Richardson and Jull, 1995). It is believed that such exercise would help normalise motor control which would include timing dysfunction. This kind of training is unlikely to help reset timing differences. It is like aspiring to play the piano faster by exercising with finger weights or performing push-ups. The reason why this is ineffective is related to a contradiction which CS training creates in relation to motor learning principles (similarity/transfer principle) and training principles (specificity principle, see further discussion below). In essence these principles state that our bodies, including the neuromuscular and musculoskeletal systems will adapt specifically to particular motor events. What is learned in one particular situation may not necessarily transfer to a different physical event, i.e. if strength is required — lift weights, if speed is needed — increase the speed of movement during training and along these lines if you need to control onset timing switch your movement between synergists at a fast rate, and hope that the system will reset itself (Lederman, 2005, in press).

To overcome the timing problem the proponents of CS came up with a solution — teach everyone to continuously contract the TrA or to tense/brace the core muscle (O'Sullivan, 2000; Jull and Richardson, 2000). By continuously contracting it would overcome the need to worry about onset timing. What is proposed here is to impose an abnormal, non-functional pattern of control to overcome a functional reorganisation of the neuromuscular system to injury: a protective strategy that is as old as human evolution.

We now know that following injury, one motor strategy is to co-contract the muscles around the joint (amongst many other complex strategies, Figure 1).

This injury response has also been shown to occur in CLBP patients (Nouwen et al., 1987; Arena et al., 1991; Hubley-Kozey and Vezina, 2002a,b; Marras et al., 2005), who tend to co-contract their trunk flexors and extensors during movement (van Dieen et al., 2003a,b). This strategy is subconscious, and very complex. It requires intricate interactions between the relative timing, duration, force, muscle lengths and velocities of contraction of immediate synergists (Shirado et al., 1995a,b; Radebold et al., 2000, see Table 1). Further complexity would arise from the fact that these patterns would change on a moment-to-moment basis and with different movement/postural tasks (McGill et al., 2003; Cordo et al., 2003; Moseley et al., 2003a,b). This pattern of muscle activity observed in standing with the arm outstretched is likely to change in bending forward or twisting. Indeed, in the original studies of the onset timing of TrA delays in onset timing were observed during fast but not during slow arm movements (Hodges and Richardson, 1996). Even during a simple trunk rotation or exercise the activity in TrA is not uniform throughout the muscle (Urquhart and Hodges, 2005; Urquhart et al., 2005a).

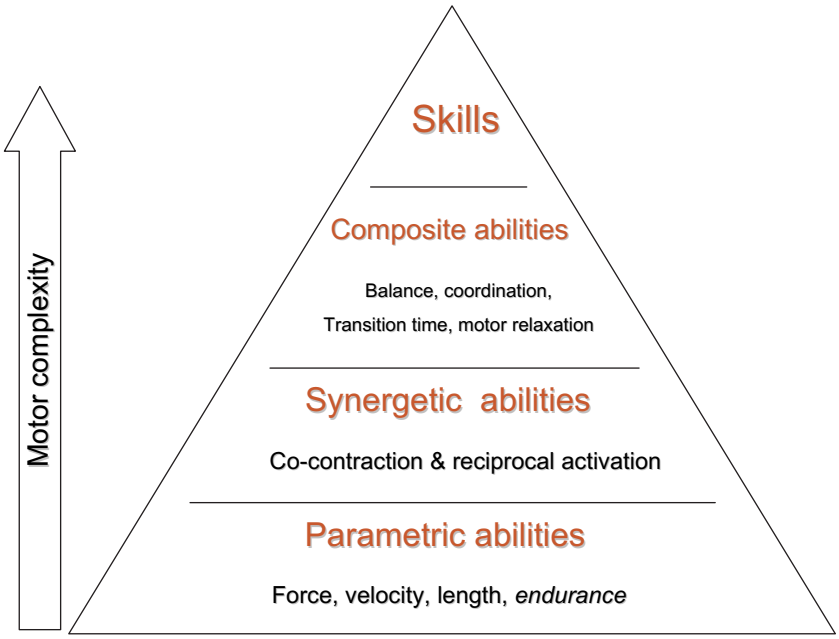

Figure 1 Motor control of movement is composed of several underlying factors which include force, velocity, range and endurance (parametric group of abilities); co-contraction and reciprocal activation which represent the synergistic level of control and the more complex composite motor abilities that include coordination, balance transition time between different activities and motor relaxation. All these motor components play a part during movement. By altering one, all the other control factors will also change. Adapted from: Lederman E, Neuromuscular rehabilitation in manual and physical therapy, to be published 2010. London, Elsevier.

These studies demonstrate the complexity that a patient re-learning trunk control may have to face. How would a person know which part of the abdomen to contract during a particular posture or movement? How would they know when to switch between synergists during movement? How would they know what is their optimal co-contraction force? If CLBP patients already use a co-contraction strategy why increase it? It is naïve to assume that by continuously contracting the TrA it will somehow override or facilitate these patterns. Furthermore no study to date has demonstrated that core stability exercise will reset onset timing in CLBP patients (Hall et al., 2007).

In summary we can conclude:

- That there is motor reorganisation of the trunk muscles in response to the experience or the fear of spinal pain
- There is no evidence that such motor reorganisation is the cause of spinal pain or recurrence of back pain
- Most prescribed CS exercise/manoeuvres are not well designed to challenge onset time of synergists and are therefore unlikely to reset the onset timing of the trunk muscles
- No study to date have as demonstrated that core stability exercise will reset onset timing in CLBP patients.

The strength issue

There is more confusion about the issue of trunk strength and its relation to back pain and injury prevention. What we do know is that trunk muscle control including force losses can be present as a consequence of back pain/injury. However, from here several assumptions are often made:

- That loss of core muscle strength could lead to back injury,
- That increasing core strength can alleviate back pain

To what force level do the trunk muscles need to co-contract in order to stabilise the spine? It seems that the answer is — not very much (Figure 2).

During standing and walking the trunk muscles are minimally activated (Andersson et al., 1996). In standing the deep spinal erectors, psoas and quadratus lumborum are virtually silent! In some subjects there is no detectable EMG activity in these muscles. During walking rectus abdominis has an average activity of 2% maximal voluntary contraction (MVC) and external oblique 5% MVC (White and McNair, 2002). During standing ''active'' stabilisation is achieved by very low levels of co-contraction of trunk flexors and extensors, estimated at less than 1% MVC rising up to 3% MVC when a 32 kg weight is added to the torso. With a back injury it is estimated to raise these values by only 2.5% MVC for the unloaded and loaded models (Cholewicki et al., 1997). During bending and lifting a weight of about 15 kg co-contraction increases by only 1.5% MVC (van Dieen et al., 2003a,b).

These low levels of activation raise the question why strength exercises are prescribed when such low levels of co-contraction forces are needed for functional movement. Such low co-contraction levels suggest the strength losses are unlikely ever to be an issue for spinal stabilisation. A person would have to lose substantial trunk muscle or force control before it will destabilise the spine.

These low levels of trunk muscle co-contraction also have important clinical implications. It means that most individuals would find it impossible to control such low

Table 1 The complexity of motor reorganisation during spinal/trunk injury and pain. All the levels of motor abilities are affected. It is an overall control reorganisation rather than failure of particular motor components. A therapeutic error is to focus on single issues such as force or co-contraction.

Conditions	Parametric motor abilities				Synergistic	Composite			
	Force	Length	Velocity	Endurance	Co-contraction/ reciprocal activation	Coordination	Balance/postural stability	Transition time	Relaxation
Lower back pain	Force losses in trunk muscles in acute and CLBP patients (Airaksinen et al., 1996; Hides et al., 1994, 1996; Ng et al., 1998; Shirado et al., 1995a)	Loss of flexion relaxation in the spinal muscles during flexion in patients with CLBP.Extensors activation prevents full forward bending (Shirado et al., 1995b). Individuals with high pain-related fear had smaller excursions of the lumbar spine for reaches to all targets at 3 and 6 weeks, but not at 12 weeks following pain onset (Thomas et al., 2008). Smaller stride length (Lamoth et al., 2008)	Reduced velocity of trunk movement during induced back pain (Zedka et al., 1999)Individuals with high pain-related fear had smaller peak velocities and accelerations of the lumbar spine and hip joints, even after resolution of back pain (Thomas et al., 2008). Walking velocity significantly lower in LBP patients (Lamoth et al., 2006a,b, 2008)	Increased fatigability trunk muscles in patient with CLBP (Roy et al., 1989; Shirado et al., 1995a,b; Suter and Lindsay 2001)	Impaired postural control of the lumbar spine is associated with delayed trunk/ abdominal muscles response times in CLBP patients (Hodges and Richardson, 1999; Hodges et al., 2003a,b; Hodges and Richardson 1996, 1998; MacDonald et al., 2006; O'Sullivan et al., 1997a,b; Radebold et al., 2001; Thomas and France, 2007; Thomas et al., 2007).Increase in trunk co-contraction in CLBP patients (Cholewicki et al., 2005; van Dieen et al., 2003a)Increased co-contraction in trunk during walking and additional cognitive demands (Lamoth et al., 2008)	Lumbar spine—hip joint coordination altered in back pain subjects (Shum et al., 2005)Dis-coordination in pelvis—thorax coordination in LBP (Lamoth et al., 2006a,b)	Changes in postural control in CLBP (Leinonen et al., 2001; Popa et al., 2007)Impaired postural control of the lumbar spine associated with delayed muscle response times in CLBP patients (Radebold et al., 2001)Changes in postural control unrelated to pain in CLBP (della Volpe et al., 2006)Postspinal surgery postural control changes both in pain and pain-free subjects. However, more evident in the symptomatic subjects (Bouche et al., 2006)Hip strategy for balance control in quiet standing is affected in CLBP (Mok et al., 2004)Experimental muscle pain changes feedforward postural responses of the trunk muscles (Hodges et al., 2003)	Compared to healthy controls, persons with LBP exhibited a reduced ability to adapt trunk—pelvis coordination and spinal muscle activity to sudden changes in walking velocity (Lamoth et al., 2006a,b)Slower reaction time in LBP patients. Demonstrated recovery of reaction time with training (Luoto et al., 1996)	Not studied (but should be)

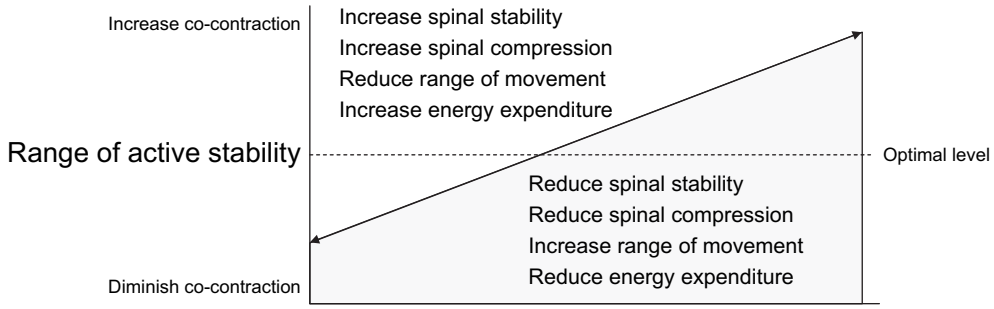

Figure 2 Co-contraction has several roles during movement such as to help stabilise the joints and refine movement. The co-contraction levels in the trunk are kept at optimal low levels — an increase in co-contraction will raise the compression force on the disc and it is more energy consuming. It also tends to rigidify the trunk which is an unsuitable control strategy where range of movement or flexibility is required.

levels of activity or even be aware of it. If they are aware of it they are probably co-contracting well above the normal levels needed for stabilisation. This would come at a cost of increasing the compression of the lumbar spine and reducing the economy of movement (see discussion below).

Is there a relationship between weak abdominals (e.g. TrA) and back pain? A common belief amongst therapists and trainers who use CS is that trunk strength will improve existing back pain. It has been shown that a muscle such as multifidus (Hides et al., 1994) can undergo atrophy in acute and CLBP (although this is still inconclusive). Furthermore, it is well established that the motor strategy changes in the recruitment of the abdominal muscles in patients with CLBP (Ng et al., 2002a,b; Moseley et al., 2003a,b), with some studies demonstrating weakness of abdominal muscles (Helewa et al., 1990, 1993; Shirado et al., 1995a,b). However, strengthening these muscles does not seem to improve the pain level or disability in CLBP patients (Mannion et al., 2001a). Improvement appeared to be mainly due to changes in neural activation of the lumbar muscles and psychological changes concerning, for example, motivation or pain tolerance (Mannion et al., 2001b). To date there are no studies that show atrophy of abdominal muscles or that strengthening the core muscles, in particular the abdominal muscle and TrA, would reduce back pain (see discussion below).

There are also examples where abdominal muscle activity is no different between asymptomatic and CLBP subjects. For instance, in studies of elite golfers, abdominal muscle activity and muscle fatigue characteristics were similar between asymptomatic and CLBP subjects after repetitive golf swings (Horton et al., 2001). Yet, this is the type of sportsperson who would often receive CS exercise advice.

Doubts have also been raised concerning the effectiveness of many of CS exercise in helping to increase the strength of core muscles. It has been shown that during CS exercise, the maximal voluntary contraction (MVC) of the ''core muscles'' is well below the level required for muscle hypertrophy and is therefore unlikely to provide strength gains (Souza et al., 2001; Vezina and Hubley-Kozey, 2000; Hubley-Kozey and Vezina, 2002a,b). Furthermore, in a study of fatigue in CLBP, four weeks of stabilisation exercise failed to show any significant improvement in muscle endurance (Sung, 2003) A recent study has demonstrated that as much as 70% MVC is needed to promote strength gains in abdominal muscle (Stevens et al., 2008). It is unlikely that during CS exercise abdominal muscle would reach this force level (Stevens et al., 2007).

We can conclude that:

- There is no evidence that reduced trunk muscle strength or endurance will predispose the individual to LBP (Hamberg-van Reenen, 2007)
- There are inconclusive finding regarding loss of trunk muscle strength and atrophy in response to CLBP
- CS exercises do not provide an overtraining challenge that is expected to result in strength or endurance gains in these muscles.

The single/core muscle activation problem

One of the principles of CS is to teach the individuals how to isolate their TrA from the rest of the abdominal muscles or to isolate the ''core muscle'' from ''global'' muscles.

It is doubtful that there exists a ''core'' group of trunk muscles that are recruited operate independently of all other trunk muscles during daily or sport activities (McGill et al., 2003; Kavcic et al., 2004). Such classification is anatomical but has no functional meaning. The motor output and the recruitment of muscles are extensive (Hodges et al., 2000; Cholewicki et al., 2002a,b), affecting the whole body. To specifically activate the core muscles during functional movement the individual would have to override natural patterns of trunk muscle activation. This would be impractical, next to impossible and potentially dangerous; as stated by Brown et al. (2006) ''Individuals in an externally loaded state appear to select a natural muscular activation pattern appropriate to maintain spine stability sufficiently. Conscious adjustments in individual muscles around this natural level may actually decrease the stability margin of safety''.

Training focused on a single muscle is even more difficult. Muscle-by-muscle activation does not exist (Georgopoulos, 2000). If you bring your hand to your mouth the nervous system ''thinks'' hand to mouth rather than flex the biceps, then the pectorals, etc. Single muscle control is relegated in the hierarchy of motor processes to spinal motor centres — a process that would be distant from conscious control (interestingly even the motor neurons of particular muscles are intermingled rather than being distinct anatomical groups in the spinal cord) (Luscher and

Clamann, 1992). Indeed, it has been demonstrated that when tapping the tendons of rectus abdominis, external oblique and internal oblique the evoked stretch reflex responses can be observed in the muscle tapped, but also spreading extensively to muscles on the ipsilateral and contralateral sides of the abdomen (Beith and Harrison, 2004). This suggests sensory feedback and reflex control of the abdominal muscles is functionally related and would therefore be difficult to separate by conscious effort.

This simple principle in motor control poses two problems to CS training. First, it is doubtful that following injury only one group or single muscles would be affected. Indeed, the more EMG electrodes applied the more complex the picture becomes (Cholewicki et al., 2002a,b). It is well documented that other muscles are involved — multifidus (Carpenter and Nelson, 1999), psoas (Barker et al., 2004), diaphragm (Hodges et al., 1997, 2003), pelvic floor muscles (Pool-Goudzwaard et al., 2005), gluteals (Leinonen et al., 2000), etc. Basically in CLBP we see a complex and wide reorganisation of motor control in response to damage or pain.

The second problem for CS is that it would be next to impossible to contract a single muscle or specific group. Even with extensive training this would be a major problem (Beith et al., 2001). Indeed, there is no support from research that TrA can be singularly activated (Cholewicki et al., 2002a,b). The novice patient is more likely to contract wide groups of abdominal muscles (Sapsford et al., 2001; Urquhart et al., 2005a,b). So why focus on TrA or any other specific muscle or muscle group?

We can summaries that:

- The control of the trunk (and body) is whole. There is no evidence that there are core muscles that work independently from other trunk muscle during normal functional movement.
- There is no evidence that individuals can effectively learn to specifically activate one muscle group independently of all other trunk muscles.

CS and training in relation to motor learning and training issues

Further challenges for the CS model arise from motor learning and training principles.

CS training seems to clash with three important principles:

- The similarity (transfer) principle in motor learning and specificity principle in training
- Internal—external focus principles
- Economy of movement.

Similarity/specificity principles

When we train for an activity we become skilled at performing it. So if we practice playing the piano we become a good pianist, hence a *similarity principle*. We can't learn to play the piano by practicing the banjo or improve playing by lifting weight with our fingers. This adaptation to the activity is not only reserved to learning processes, it has profound physical manifestations — hence a weight trainer looks physically different to a marathon runner (*specificity principle* in training, Roels et al., 2005).

If a subject is trained to contract their TrA or any anterior abdominal muscle while lying on their back (Karst and Willett, 2004), there is no guarantee that this would transfer to control and physical adaptation during standing, running, bending, lifting, sitting, etc. Such control would have to be practiced during some of these activities (Lederman, in press, see Figure 3). Anyone who is giving CS exercise to improve sports performance should re-familiarise themselves with this basic principle.

It seems that such basic principles can escape many of the proponents of CS. This is reflected in one study which assessed the effect of training on a Swiss ball on core stability muscles and the *economy of running* (Stanton et al., 2004)! In this study it was rediscovered that practicing the banjo does not help to play the piano. The subjects got very good at using their muscles for sitting on a large inflatable rubber ball but it had no effect on their running performance. An often quoted study by Tsao and Hodges (2008) does show transfer of learning from CS training to postural activity. However, this is a low quality study, carried out on a small number of subjects ($n = 9$) without any control/sham. The transfer observed in this study is in conflict with the vast knowledge of motor control that suggests that such transfer is highly unlikely (see Schmidt and Lee, 2005 for extensive review of transfer of learning).

Trunk control will change according to the specific activity the subject is practicing. Throwing a ball would require trunk control, which is different to running. Trunk control in running will be different in climbing and so on. There is no one universal exercise for trunk control that would account for the specific needs of all activities. Is it possible to train the trunk control to specific activity? Yes, and it is simple — just train in that activity and don't worry about the trunk. The beauty of it all is that no matter what activity is carried out the trunk muscles are always specifically exercised.

Internal and external focus in training

CS has evolved over time in response to many of the model's limitations described above. Currently, the control of TrA is attempted in different standing and moving patterns (O'Sullivan, 2000). Speed of movement, balance and coordination has been introduced to the very basic early elements of CS. The new models encourage the subjects to ''think about their core'' during functional activities. One wonders if David Beckham thinks about the ''core'' before a free kick or Michael Jordan when he slam-dunks or for that matter our patient who is running after a bus, cooking or during any other daily activities. How long can they maintain that thought while multitasking in complex functional activities?

Maybe thinking about the core is not such a good idea for sports training. When learning movement a person can be instructed to focus on their technique (called internal focus) or on the movement goal (called external focus). When a novice learns a novel movement focusing on technique (internal focus) could help their learning (Beilock et al., 2002) For a skilled person, performance improves if training focuses on tasks outside the body (external focus) but it

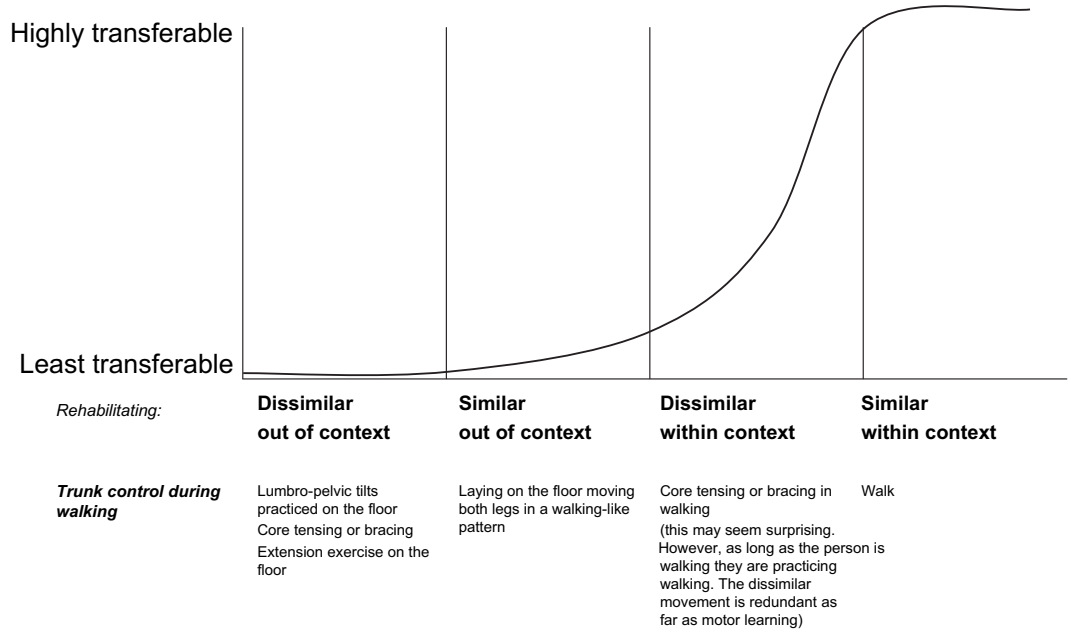

Figure 3 Similarity and context principle. Training and practice of movement can be dissimilar and out of context, similar but out of context, dissimilar within context or similar and within context. Ideal neuromuscular organisation to movement occurs when the movement is in similar patterns to the goal movement and practiced in context of the particular movement. Most CS training regimes contain movement patterns that are dissimilar and out of context to the trunk patterns used during normal activities. Adapted from Lederman E, Neuromuscular rehabilitation in manual and physical therapy, to be published 2010. London, Elsevier.

reduces when the focus is on internal processes within the body (McNevin et al., 2000, 2003). For example, there is greater accuracy in tennis serves and football shots when the subjects use external focus rather than internal-focus strategies (Wulf et al., 2002, 2003). This principle strongly suggests that internal focus on TrA or any other muscle group will reduce skilled athletic performance. Interestingly, tensing the trunk muscle has even been shown to potentially degrade postural control (Reeves et al., 2006).

What about movement rehabilitation for CLBP patients? Would internal focus on specific muscles improve functional use of trunk muscles? Let's imagine two scenarios where we are teaching a patient to lift a weight from the floor using a squat position. In the first scenario, we can give simple internal-focus advice such as bend your knees, and bring the weight close to your body, etc (van Dieen et al., 1999; Kingma et al., 2004). This type of instruction contains a mixture of external focusing (e.g. keep the object close to your body and between your knees) and internal focus about the body position during lifting. In the second scenario which is akin to CS training approach, the patient is given the following instructions: focus on co-contracting the hamstrings and the quads, gently release the gluteals, let the calf muscles elongate, while simultaneously shortening the tibialis anterior etc. Such complex internal focusing is the essence of CS training, but applied to the trunk muscles. It would be next to impossible for a person to learn simple tasks using such complicated internal-focus approach.

Economy of movement

The advice given to CS trainees is to continuously tighten their abdominal and back muscles. This could reduce the efficiency of movement during daily and sports activities. Our bodies are designed for optimal expenditure of energy during movement. It is well established that when a novice learns a new motor skill they tend to use a co-contraction strategy until they learn to refine their movement (Lay et al., 2002). Co-contraction is known to be an ''energy waster'' in initial motor learning situations. To introduce it to skilled movement will have a similar ''wasteful'' effect on the economy of movement. Minetti (2004) states: ''*to improve locomotion (and motion), mechanical work should be limited to just the indispensable type and the muscle efficiency be kept close to its maximum. Thus it is important to avoid: …. using co-contraction (or useless isometric force)*''.

Such energy wastage is likely to occur during excessive use of trunk muscles as taught in CS. In sporting activity this would have a detrimental effect on performance. Anderson (1996) in a study on the economy of running states: ''*At higher levels of competition, it is likely that 'natural selection' tends to eliminate athletes who failed to either inherit or develop characteristics which favour economy*''.

We can conclude for the evidence that:

- CS exercises are in conflict with motor learning and training principles
- CS exercises are dissimilar and out of context to normal physiological movement. This represents the most ineffective approach to learning motor skills
- The internal-focus approach on individual muscles in CS is likely to degrade motor learning as well as skilled performance
- Additional tensing of trunk muscles during daily activities or sports are likely to be more energetically taxing on the body

CS in prevention of injury and therapeutic value

Therapist and trainers have been exalting the virtues of CS as an approach for improving sports performance (Kibler et al., 2006), preventing injury and as the solution to lower back pain. No matter what the underlying cause for the complaint CS was going to save the day. However, these claims are not supported by clinical studies:

Abdominal/stability exercise as prevention of back pain

In one study, asymptomatic subjects ($n = 402$) were given back education or back education + abdominal strengthening exercise (Helewa et al., 1999). They were monitored for lower back pain for one year and the number of back pain episodes were recorded. No significant differences were found between the two groups. There was a curious aspect to this study, which is important to the strength issue in CS. This study was carried out on asymptomatic subjects who were identified as having weak abdominal muscles. Four hundred individuals with weak abdominal muscles and no back pain!

Another large-scale study examined the influence of a core-strengthening programme on low back pain (LBP) in collegiate athletes ($n = 257$). In this study too, there were no significant advantage of core strengthening in reducing LBP occurrence (Nadler et al., 2002).

CS a treatment for recurrent LBP and CLBP

At first glance, studies of CS exercise for the treatment of recurrent LBP look promising — significant improvements can be demonstrated when compared to other forms of therapy (O'Sullivan et al., 1997a,b; Hides et al., 2001; Moseley, 2002; Rasmussen-Barr et al., 2003; Niemisto et al., 2003; Stuge et al., 2004; Goldby et al., 2006). Indeed, systematic reviews found stabilisation exercise to be better than general practitioner care, but not from any other form of physical therapy (Rackwitz et al., 2006; Ferreira et al., 2006; Macedo et al., 2009).

However is could be argued that none of these studies actually showed a relationship between improvement in LBP and spinal stabilisation or core control. In all the studies there was no attempt to effectively identify patients who had timing or other control issues or had underlying instability. There was no attempt to evaluate how well the subjects learned CS manoeuvres and whether they were able to maintain that learning throughout the duration of the studies. Furthermore, there was no attempt to evaluate if there is a correlation between improvement of the condition and the recovery of stabilisation. It should also be noted that many of these studies did not have a control group. This means that although CS training may be better when compared to another form of therapy, we still don't know if it is any better than a placebo/sham treatment.

An interesting trend emerges when CS exercise are compared to general exercise (Table 2). *Both exercise approaches are demonstrated to be equally effective* (Ariyoshi et al., 1999; van der Velde and Mierau, 2000; Franke et al., 2000; Reeves, 2006; Nilsson-Wikmar et al., 2005; Koumantakis et al., 2005; Cairns et al., 2006). Systematic reviews repeat this message (van Tulder et al., 2000; Abenhaim et al., 2000; Hurwitz et al., 2005).

These studies strongly suggest that improvements are due to the positive effects that physical exercise may have on the patient rather than on improvements in spinal stability (it is known that general exercise can also improve CLBP) (Ariyoshi et al., 1999; van der Velde and Mierau, 2000).

So why give the patient complex exercise regimes that will both be expensive and difficult to maintain? Perhaps our patients should be encouraged to maintain their own preferred exercise regime or provide them with exercises that they are more likely to enjoy. This of course could include CS exercise. But the patient should be informed that it is only as effective as any other exercise.

We can thus conclude:

- That CS exercise may better than general medical care (which is not difficult to achieve)
- CS exercise is no better than other forms of manual or physical therapy or general exercise
- Find out what exercise the patient enjoys and add it to the management plan.

CS in relation to aetiology of back pain

Why has CS not performed better than any other exercise? In part, due to all the issues that have been discussed above. More importantly, in the last decade our understanding of the aetiology of back pain has dramatically changed. Psychological and psychosocial factors have become important risk and prognostic factors for recurrent back pain and the transition of acute to chronic pain states (Hasenbring et al., 2001). Genetic factors (MacGregor et al., 2004) and behavioural/''use of body'' are also known to be contributing factors. Localised, structural factors such as trunk/spinal asymmetries, have been reduced in their importance as contributing factors to back pain (Dieck, 1985; Nadler, 1998; Franklin and Conner-Kerr, 1998; Levangie, 1999; Fann, 2002; Norton, 2004; Poussa, 2005; Reeves, 2006; Mitchell et al., 2008). This shift in understanding LBP would include stability issues which are an extension of a biomechanical model.

It is difficult to imagine how improving biomechanical factors such as spinal stabilisation can play a role in reducing back pain when there are such evident biopsychosocial factors associated with LBP conditions. Even in the behavioural/biomechanical spheres of spinal pain it is difficult to imagine how CS can act as prevention or cure. This can be clarified by grouping potential causes for back injury into two broad categories:

- Behavioural group: individuals who use their back in ways that exert excessive loads on their spine, such as bending to lift (Gallagher et al., 2005) or repetitive sports activities (Fairclough et al., 1986; Renström, 1996; Reid and McNair, 2000).

Table 2 CS studies, description of study, CS compared to other therapeutic modalities and outcome.

	Description of condition	CS compared to	Result	Notes
O'Sullivan et al., 1997a,b	CLBP (spondylolysis/ spondylolisthesis)	General exercise consisted of swimming, walking and gym work + pain relief including heat application, massage and ultrasound	CS better	General exercises were not of the same duration as CS exercise.The pain relief methods chosen are known to have little effect on back pain
Hides et al., 2001	First episode LBP	General practitioner care + medication	CS better	
Moseley, 2002	CLBP	CS + MT compared to medical management	CS/MT better than medical care	We still don't know if CS is better because it was combined with MT
Rasmussen-Barr et al., 2003	CLBP	Manual therapy (muscle stretching, segmental traction, soft tissue and facet mobilisation	CS better in the short term but not long-term	The duration of MT was shorter than the CS exercise
Stuge et al., 2004	LBP in pregnancy	Physical therapy	CS better	
Niemisto et al., 2003	LBP	CS + MT + physician care compared to: physician care	CS/MT better	We still don't know if CS is better because it was combined with MT
Goldby et al., 2006	CLBP	Back education and MT	CS > MT > education	Generally considered to be poor quality study
Bastiaenen et al., 2006	LBP postpartum	Cognitive-behavioural therapy (CBT)	CBT better	
Nilsson-Wikmar et al., 2005	LBP in pregnancy	General exercise	**Same**	
Franke et al., 2000	CLBP	General exercise	**Same**	
Koumantakis et al., 2005	Sub-acute or CLBP	General exercise	**General exercise slightly better**	
Cairns et al., 2006	Recurrent LBP	Exercise + MT	**Same**	
Ferreira et al., 2007	CLBP	CS + CBT compared to: 1. General exercise + CBT + stretching and strengthening all main muscles groups in body, + cardiovascular exercise 2. Spinal manipulation (SM)	**SM and CS same outcome but slightly better than general exercise in the short but not long term**	Other studiers suggest that CS is better than MT..
Critchley et al., 2007		1. MT 2. Pain management + CBT 3. General exercise	**No difference between the groups**	

- Bad luck group: individuals who had suffered a back injury from sudden unexpected events, such as falls or sporting injuries (Fairclough et al., 1986).

In the behavioural group, bending and lifting is associated with a low level increase in abdominal muscle activity, which contributes to further spinal compression (de Looze et al., 1999). In patients with CLBP lifting is associated with higher levels of trunk co-contraction and spinal loading (Marras et al., 2005). Any further tensing of the abdominal muscles may lead to additional spinal compression. Since the spinal compression in lifting

approaches the margins of safety of the spine, these seemingly small differences are not irrelevant (Biggemann et al., 1988). It is therefore difficult to imagine how CS can offer any additional protection to the lumbar spine during these activities.

Often in CS advice is given to patients to brace their core muscle while sitting to reduce or prevent back pain. Although sitting is not regarded as a predisposing factor for LBP (Hartvigsen et al., 2002), some patients with existing back pain find that standing relieves the back pain of sitting. This phenomenon has been shown in CLBP patients who during sitting exhibit marked anterior loss of disc space in flexion or segmental instability (Maigne et al., 2003). Sitting, however, is associated with increased activity of abdominal muscles (when compared to standing) (Snijders et al., 1995). Increasing the co-contraction activity of the anterior and back muscles is unlikely to offer any further protection for patients with disc narrowing/pathology. Conversely, it may result in greater spinal compression. It is unknown whether core tensing can impede the movement of the unstable segments during sitting. This seems unlikely because even in healthy individuals creep deformation of spinal structures will eventually take place during sitting (Hedman and Fernie, 1997). The creep response is likely to be increased by further co-contraction of trunk muscles.

In the bad luck group, CS will have very little influence on the outcome of sudden unexpected trauma. Most injuries occur within a fraction of a second, before the nervous system manages to organise itself to protect the back. Often injuries are associated with factors such as fatigue (Gabbett, 2004) and overtraining (Smith, 2004). These factors when combined with sudden, unexpected high velocity movement are often the cause of injury (Fairclough et al., 1986). It is difficult to see the benefit of strong TrA, abdominals or maintaining a constant contraction in these muscles in injury prevention.

Potential damage with CS?

Continuous and abnormal patterns of use of the trunk muscles could also be a source of potential damage for spinal or pelvic pain conditions. It is known that when trunk muscles contract they exert a compressive force on the lumbar spine (van Dieen et al., 2003a,b) and that CLBP patients tend to increase their co-contraction force during movement (Cholewicki et al., 1997). This results in further increases of spinal compression (Marras et al., 2005; Brown et al., 2006). Another recent study examined the effects of abdominal stabilisation manoeuvres on the control of spine motion and stability against sudden trunk perturbations (Vera-Garcia et al., 2007). The abdominal stabilisation manoeuvres were — abdominal hollowing, abdominal bracing and a ''natural'' strategy. Abdominal hollowing was the most ineffective and did not increase stability. Abdominal bracing did improve stability but came at a cost of increasing spinal compression. The natural strategy group seems to employ the best strategy — ideal stability without excessive spinal compression.

An increase in intra-abdominal pressure could be a further complication of tensing the trunk muscle (Cresswell et al., 1994a,b). It has been estimated that in patients with pelvic girdle pain, increased intra-abdominal pressure could exert potentially damaging forces on various pelvic ligaments (Mens et al., 2006).

Maybe our patients should be encouraged to relax their trunk muscle rather than hold them rigid? In a study of the effects of psychological stress during lifting it was found that mental processing/stress had a large impact on the spine. It resulted in an increase in spinal compression associated with increases in trunk muscle co-contraction and less controlled movements (Davis et al., 2002).

Psychological factors such as catastrophising and somatisation are often observed in patients suffering from CLBP. One wonders if CS training colludes with these factors, encouraging excessive focusing on back pain and re-enforcing the patient's notion that there is something seriously wrong with their back. Perhaps we should be shifting the patient's focus away from their back. (I often stop patients doing specific back exercise).

Furthermore, CS training may shift the therapeutic focus away from the real issues that maintain the patient in their chronic state. It offers a simplistic solution to a condition that may involve complex biopsychosocial factors. The issues that underline the patient's condition may be neglected, with the patient remaining uninformed about the real causes of their condition. Under such circumstance CS training may promote chronicity.

Conclusion

- Weak trunk muscles, weak abdominals and imbalances between trunk muscles groups are not a pathology just a normal variation.
- The division of the trunk into core and global muscle system is a reductionist fantasy, which serves only to promote CS.
- Weak or dysfunctional abdominal muscles will not lead to back pain.
- Tensing the trunk muscles is unlikely to provide any protection against back pain or reduce the recurrence of back pain.
- Core stability exercises are no more effective than, and will not prevent injury more than, any other forms of exercise or physical therapy.
- Core stability exercises are no better than other forms of exercise in reducing chronic lower back pain. Any therapeutic influence is related to the exercise effects rather than stability issues.
- There may be potential danger of damaging the spine with continuous tensing of the trunk muscles during daily and sports activities.
- Patients who have been trained to use complex abdominal hollowing and bracing manoeuvres should be discouraged from using them.

Epilogue

Many of the issues raised in this article were known well before the emergence of CS training. It is surprising that the researchers and proponents of this method ignored such important issues. Despite a decade of extensive research in

this area, it is difficult to see what contribution CS had to the understanding and care of patients suffering from back pain.

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