The primal nature of core function: 
In rehabilitation & performance conditioning

Abstract  In this editorial, what is understood of the definition and function of the human core is discussed; presented in the context of evolution and holistic human modeling. It appears likely from this understanding of neural phenomena such as central sensitization, neural facilitation/inhibition, tonic and phasic motoneuron excitatory thresholds and viscero-somatic convergence that, very simply, for effective core function, optimal visceral function is a prerequisite and may be a commonly overlooked aspect of patient rehabilitation. Furthermore, not only is core function key for optimal expression of forces through the appendicular skeleton, but since it is the tonic motoneurons most readily inhibited by nociceptive (including viscero-somatic) phenomena, this will likely affect the tonic components of peripheral musculature directly; impairing both local motor control and performance at peripheral joints.

When viewed through the lens of human propensity to fads and trends, the focus on core function in the bodywork and movement professions over the last 20 years may rightly be seen as a transient and passing irritant to those whose focus for helping patients has been elsewhere. However, when viewed through an evolutionary lens the importance of a functional core cannot be overemphasized; as it is literally the core, the foundation from which all other function has grown.

To build a house, or indeed to rebuild a damaged house, the foundations must be laid first — and laid solidly — for a successful long-term project. Understanding how to do this, what the foundation consists of, and what processes must be put in place are all key in creating a successful outcome. The greater the desired performance of the building, and the desire for a trouble-free the process, the more solid the foundations must be.

In much the same way, creating and sustaining long-term human health, function and performance requires an analogous understanding of how our foundations are built, what they consist of, and what processes are entailed in their development and functioning.

The architectural blueprint

The core of the human body is the same as any other body with a triploblastic body cavity with hemocele (Erwin, 1997). To clarify, that is, the human core is near-identical in basic design to all the "cores" that pre-date it in the fossil record; through the primates, mammalia, reptilia, right the way back to the core of fish.

Prior to this, only three other key body plans were utilized and each, interestingly, were intimately related with the digestive process of the organism. Indeed this feature is retained in our own body plan; the core musculature developing embryologically from the very same sheet of tissue, the lateral plate mesoderm, as the smooth muscle of digestive tract. This should give us an early indication that, if structure and function are inter-related, so digestion should affect the abdominal musculature and vice versa.

To trace further back into deep time before multicellular organisms, the very first life-form, the single-celled organism only had a "core"... no flagellae, no legs, arms or head, just a core — containing organelles, which, billions of years later, would be recognized as analogues to our body organs; specialized structures that have developed as organisms have become increasingly complex and "organized". Indeed, the very first movement pattern would be one of radial contraction and relaxation; a movement pattern that is retained in the human digestive system, the transverse musculature of the body cavities, and the cardiorespiratory system.

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Of course, it was only when animals moved to land that the respiratory mechanism itself became increasingly associated with this radial dimension of movement — expansion contraction and, despite its shorter association, this is still clearly a very primitive relationship having been retained for hundreds of millions of years.

So, functionally, digestion and the core have always been intimately related and, since animals first moved to land, respiration and the core have been functionally interlocked, but what actually is the core? How is the core defined? There are varying definitions, but the core is often described as the functional unit of the transversus abdominis (TrA), pelvic floor musculature, diaphragm and multifidus (Lee, 2003; Richardson et al., 1999). Chek (1998), on the other hand, suggests if one were to pull off the upper extremities and the lower extremities, what’s left is the core”. This latter example fits best with both an evolutionary and a functional understanding of the core. In the former, the earliest fish had no discernible neck; their “head” was simply the front end of their body, while, in the latter, one of the major critiques of a focus on this “inner-unit” of the core is that it always works in conjunction with the outer unit musculature, such as obliques, paraspinals and girdle muscles, therefore Chek’s definition helps to get around this potential criticism.

The materials with which to build

The above should give a feel for the primacy of the core and some functions (digestion and respiration) that are integrated with the human core, but what is the core composed of? One answer to this is that it is composed of both musculature, and of a lot of connective tissue, which allows that musculature to work effectively in orchestrated teams of muscles, as required by the demands of the task. And what orchestrates these muscles? The nervous system.

It is an understanding of the nervous system’s role in orchestrating core function that is key in recognizing why the core exercises may be failing in their role. See Motor Learning and Viscerosomatic Reflexes below.

So why the recent focus on the core, hasn’t it always been recognized as important? In brief, the increased focus on the core began in the late 1980’s when it was not only recognized that movement emanates from the core (see The Spinal Engine (Gracovetsky 1988)), but also that when a patient suffers with back pain, this interferes first with the deep muscles of the core, such as the transversus abdominis and the lumbar multifidus; they appear to become neurologically inhibited (Richardson et al., 1999). The logical suggestion therefore would be to re-train these muscles to “switch back on”. The issue that has arisen is that, clinically, things aren’t so simple (Fig. 1).

Chicken & egg

Was it pain that inhibited the musculature, or musculature not firing at the right time that resulted in pain? The likely answer was that both could occur and, indeed, one could initiate and the other could perpetuate.

Hides et al. (1996) identified that not only did the deep lumbar multifidus stop firing effectively in low back pain patients, but that even 1 year after the pain had gone, the multifidus still was not firing functionally (in a feed-forward mechanism, in anticipation of movement), unless it had been properly rehabilitated. So now, even pain wasn’t required for inhibition or inappropriate firing of the inner unit musculature. This was a case of compensation — of motor learning.

Motor learning

Motor learning is, unsurprisingly, learning of motor control which necessarily must occur primarily in the higher centers of the brain; namely the motor cortex. Although other parts of the CNS are involved and can be tuned to some degree, the cortex has the highest degree of plasticity and therefore capacity for learning. For this reason, when someone is in pain it is likely that the cortex is chiefly responsible for finding compensatory solutions. And this is part of the reason Hides found that the multifidus doesn’t start to fire again; because “learning” of multifidus activation occurs in the pre-conscious, more reflexive phase of infant development (from in utero, to 4–5 months of age), when the motor cortex is not yet able to distinguish cause &
effect mechanisms; this distinction occurs around 7–8 months of age (Wallden, 2008).

Consciousness in motor learning

In addition, motor learning is effective when the client is paying attention to the task in hand, but what happens when there is no direct focus on, for example, activating multifidus? In behavioral therapies there is a model, which suggests that dysfunctional behavior is never the goal of the individual; it is merely a circumstance they find themselves in due to a single event, or sequence of events, leading to the current status.

When a patient consults a specialist regarding back pain, a degree of dysfunction or "incompetence" would be anticipated. The patient would likely be unaware or "unconscious" that dysfunctional activation of multifidus was an aetiological feature of the back pain. Such a patient could be described unconscious incompetents. When a bodyworker or movement therapist teaches the patient how to activate the lumbar multifidi to fire appropriately - the patient becomes a conscious incompetent. If a bodyworker or movement therapist teaches the patient how to activate the lumbar multifidi to fire appropriately - the patient becomes a conscious competent. And for many patients, this is where the journey ends with that specific therapist, but it may pick up again further down the line, because this process is incomplete, as is explained below.

Milk & chilies

In the proceedings of the World Congress on Low Back & Pelvic Pain 2001, Granath et al presented findings that those women who are lactose intolerant have a higher level of post-partum pelvic pain, with a probability of 0.0107. The proposal was that this may be due to some kind of biochemical causation.

A few years prior to this, holistic fitness educator, Paul Chek, had been challenged to a chili-eating competition by his son at lunch in their local Mexican restaurant. Later that afternoon he and his son had a workout and, continuing the spirited rivalry, decided to have a prone transversus abdominis activation (see Self-Help Section) competition; who could lift the most amount of pressure (and sustain that contraction) off the biofeedback cuff, without cheating? The shocking conclusion was that neither of them could take any pressure off the cuff.

Chek's awareness of the literature on neurophysiology and viscero-somatic reflexes led him to the assumption that the inflammation in the gut caused by the excessive chilli exposure had resulted in a viscero-somatic reflex to the motor neurons feeding the abdominal wall; that his transversus abdominis was simply inhibited. The feasibility of this is discussed further below.

Anecdotally, many bodywork and movement specialists have subsequently observed this same phenomenon; when a finding in the medical history indicates visceral inflammation, there appears to consistently be a correlating inhibition of the portion of the abdominal wall that overlays that organ; compromising motor control.

Frequently, an organ that is supplied through the visceral nerves that converge on the inferior mesenteric ganglion will be seen to inhibit just the lower portion of the abdominal wall (Willard, 1997). While patients with afferent drive from the upper abdominal viscera into the middle or superior mesenteric ganglion will experience inhibition of their upper abdominal wall; palpable on the forward flexion activation test described in Hashemirad et al. (2010).

If Chek's observation were correct, we would expect to find that, clinically, any condition in which there is inflammation could inhibit the abdominal wall, so this would potentially include a raft of clinical conditions including but not limited to:

1) Chronic constipation
2) IBS
3) IBD
4) Food intolerance
5) Dysbiotic conditions
6) Dysmenorrhea
7) Cystitis... and so on

In the literature we can find some support of this notion in research conducted on bloating, and in the field of motor control, which is discussed below.

Bloating

For the abdominal wall to bloat, normal tone of the abdominal muscles must be affected. Indeed, this assertion is corroborated by Azpiroz and Malagelada (2005) who state that there are 4 factors involved in the pathophysiology of bloating: 1) subjective sensation of bloating, 2) objective abdominal distension, 3) volume of abdominal contents, 4) changes to muscular activity of the abdominal wall. (Fig. 2).

As noted, the first factor is subjective, and therefore not easily measurable; though studies conducted on IBS sufferers, where bloating is a frequent symptom, found that patients complaining of bloating (factor 1) indeed do have impaired clearance and tolerance of intestinal gas (factor 3).

Changes in abdominal girth (factor 2) is measurable – and has

Figure 2  The abdominal wall of a chronic constipation sufferer. The colon, which reflexes to the lower abdominal wall, is affecting motor drive to the lower abdominal musculature, while the upper abdominal musculature has perfect tone.
been demonstrated to be increased in symptomatic populations, but often this increase is minimal compared to controls, and symptoms are disproportionate; suggesting an element of sensitization (Tremolaterra et al., 2006). The third factor, abdominal contents, is objectively measurable and studies have confirmed increased abdominal gas in IBS: the gas surface in plain abdominal radiographs being on average 18% larger in IBS patients than in controls. However, when one considers that the normal volume of intestinal gas is about 200 mL, the extra volume in patients could elicit discomfort, but is not likely to account for objective abdominal distention. Furthermore, according to Tremolaterra, other studies failed to show any discernible increase of intestinal gas in these patients. The fourth factor — changes in muscular activity during bloating — is also measurable and has come up with the results that would be anticipated if Chek’s thesis is correct.

Compensatory muscle activation

Clinically, it would appear that when pain is present, local intrinsic musculature becomes inhibited (Richardson et al., 1999). The lumbar multifidus, for example, decreases to around 69% of its original cross-sectional area within 24 h of the onset of pain. This cannot be explained by atrophy in such a short time period; but can be explained by inhibition and therefore loss of tone and cross-sectional area as a result (Richardson et al., 1999).

The likely response of the nervous system to such inhibition is to locate compensatory structures (in the case of the lumbar multifidus it may be other more phasic “outer unit” erector spinae), to provide active support for the now unstable spinal segment(s) (Lee, 2004).

Supporting this view, Hungerford et al. (2003) found that in patients with pre-existing sacro-iliac joint pain, the feed-forward mechanism (see below) of the multifidus and lower fibers of internal oblique became inhibited and that, in compensation, the biceps femoris became facilitated, contracting first. The nervous systems of these pain patients had found a strategy to still stabilize the SIJ through the biceps femoris (an outer unit, mobilizer muscle), rather than through the normal inner unit feed-forward response, discussed above under the heading *Chicken and Egg*.

It would appear that the aberrant stimulus inhibiting the inner unit needn’t be musculoskeletal pain, but also may beafferent drive from the viscera; and especially if that afferent drive is recurrent. Bouhasira et al. (1998) investigated how irritation to the visceral afferent receptors in the rectum affected tone in the hamstring muscle. What they found was that as irritation increased in the rectum, akin to the kind of irritation that may occur in chronic constipation or irritable bowel syndrome, the EMG signal from the hamstring increased.

The relevance of this may be understood when taken in context with the Hungerford study and EMG findings from Tremolaterra et al.’s (2006) study of abdominal bloating. What Tremolaterra’s group found was that, in IBS sufferers, as gas was rectally infused into them to mimic the effects of IBS, the EMG reading from the external obliques increased. Reciprocally, the electrical activity of the transverse fibers of internal oblique decreased dramatically; it became inhibited. As pointed out by Hungerford et al. (2003), placement of the electrode in the centre of a triangle formed by anterior superior iliac spine (ASIS), the umbilicus and the inguinal ligament, means that the more horizontal fibres of IO are being targeted. These have a synergistic function with the transversus abdominis (TrA), while TrA excitability would also be registered.

Such facilitation of outer-unit musculature, such as the external oblique or the hamstring group; and, reciprocally, the inhibition of the inner unit musculature, such as the TrA or horizontal fibers of IO, suggests that aberrant stimuli, both in the form of pain signals reaching the cord, and visceral afferents reaching the cord, can be enough to “shut down” or inhibit inner unit musculature. The body’s natural compensation strategy appearing to be up-regulating neural drive to outer unit (global stabilizer) musculature.

Threshold to stimulus

If the clinical and research picture seems to be illustrating that it is the inner-unit musculature, such as the transversus abdominis or multifidus, that is inhibited by pain, what is it about these muscles that makes them prone to inhibition?

Feed-forward mechanism

The transversus abdominis became a focus of attention after Cresswell et al described its role in spinal stabilization in 1989, while its engagement in a feed-forward manner was first reported in the literature by Hodges and Richardson (1997); the latter giving us insights into why TrA and other inner unit musculature is prone to inhibition. Why do these stabilizer, local, deep, intrinsic, inner unit muscles fire ahead of their mobilizer, outer unit counterparts?

Practically, a logical answer is that feed-forward needs to occur in order to stabilize the body, before actual movement takes place. Nature, it would appear, is intelligent enough to recognize that “you can’t fire a canon from a canoe” and so in order to generate force a stable base is required, from which to generate that force. Failure to achieve stability results in shear, torsion and compression forces being generated which, may cause macrotrauma and certainly, across time, cumulative microtrauma, ultimately leading to macrotrauma (Solomonow et al., 2012).

Neurophysiologically, one of the features of these inner-unit muscles is that they have a low threshold to stimulus, which means that only a hint of a nerve impulse passing down the motor neurons is required for activation. By contrast, outer-unit muscles have a higher threshold to stimulus, meaning they require a stronger impulse to engage. However, it could be that, while there are the practical benefits from the feed-forward process (described above) in terms of providing a stable base, this may be a double-edge sword.

Arndt—Schultz Law

The Arndt—Schultz Law states “weak stimuli accelerate physiologic activity, medium stimuli inhibit physiologic activity, and strong stimuli halt physiologic activity.”
This means a small impulse should and will activate the transversus abdominis, for example, but that a really strong stimulus; perhaps a noxious stimulus such as pain or a viscero- somatic reflex, will override the lower threshold motoneurons quicker, resulting in earlier inhibition. It is therefore the inner unit muscles, with a lower threshold to stimulus (something that is functional in health), that may shut down first, when there is an excessive stimulus at the corresponding spinal segment (Arndt Schultz Law, 2013). See further discussion under Neurophysiology below.

Myophysiology

A brief review of the myology section of Gray’s Anatomy (1991 edition) reveals information that is useful to this discussion. Gray’s states:

"the deeper a muscle is located in the body the higher its number of slow twitch fibres, while the more superficial it is located, the higher its number of fast twitch fibers.”

Slow twitch fibres arise as a result of the type of motoneuron that supplies them, slow twitch fibers being fed by tonic motoneurons, and fast twitch fibers being fed by phasic motoneurons (Kuno, 1984). Further, it is known that in deeper, intrinsic muscles there is a preponderance of muscle spindles, while in superficial strap muscles the spindle cell number decreases dramatically (Williams, 1995).

These two pieces of information are logical; as there would be little sense in swamping muscles close to the axis of rotation of a joint with fast twitch, phasic fibers as they would have poor leverage on the very joint they were designed to move. On the contrary, it makes a lot of sense to have an abundance of both slow twitch (tonic) fibers – to hold tone around the joint and, with their high density of spindle cells, to provide intricate detail of joint position back into the nervous system; providing exquisite control of joint position across prolonged periods without fatigue.

A small explosion or refined harmonious activation?

Indeed, Bompa (1999) states that phasic motoneurons, which feed the fast twitch muscle fibers, will typically synapse one neuron to approximately 100 muscle fibers. In contrast, tonic motoneurons, which feed the slow twitch muscle fibers, typically synapse one neuron to approximately 5 or 6 muscle fibers. In other words, a nerve impulse traversing a phasic motoneuron will, upon reaching the muscle, be like a small explosion going off, whereas a nerve impulse arriving through the tonic motoneurons is like the honed and refined skill of a symphony conductor, orchestrating each fiber to do it’s job at the right time, in the right sequence.

In addition, while the deeper muscles are carrying out their raison d’etre to stabilize and maintain the optimal instantaneous axis of rotation of the joint they serve, they have an additional benefit. They function rather like a sesamoid bone, inasmuch as they create a fulcrum about which the outer unit musculature can gain leverage; moving the outer unit musculature further from the axis of rotation of the joint, thereby creating a greater moment arm or mechanical advantage. It would be unsurprising, therefore, that an athlete whose inner unit is inhibited, as well as having an increased the risk of injury, would lose power, speed, agility and performance in general.

Knee pain & quadriceps inhibition

In 2009, Hodges et al published a study investigating the effect of pain on the quadriceps muscle group; simulating knee pain through injecting the infrapatella fat pad with saline. He found that under normal conditions the vastus medialis obliquus (VMO), a muscle well known for being inhibited, weak or deconditioned in anterior knee pain, would fire ahead of the vastus lateralis. The VMO is considered a part of the inner-unit of the knee, which is why, like the transversus abdominis, it fires ahead of its counterparts, and is inhibited by pain.

And this is exactly what Hodges found when he simulated knee pain via the saline injection; the VMO became inhibited and fired later than the vastus lateralis. However, when he increased the saline, the vasti became inhibited. Finally, only with even further irritation through larger volumes of saline did the rectus femoris become inhibited (Hodges et al., 2009).

What this appears to illustrate is that when there is a pain stimulus, the afferent drive to the cord appears to create a central sensitization which will affect musculature sequentially depending on how deep, tonic or inner-unit it is.

It would seem that the interaction between the nervous system and the musculature functions well when not in the presence of pain, but somewhat more clumsily when pain or aberrant stimuli are present. This is neuromotor compensation, a compromise which, across time, may lead to cumulative microtrauma, decompensation and more chronic pain states.

Wheat, rice, soy & stress

Central sensitization

There is much discussion in the literature regarding the role of central sensitization in chronic pain conditions, for example, see Woolf (2011).

According to Latremoliere and Woolf (2009) central sensitization “represents an enhancement in the function of neurons and circuits in nociceptive pathways caused by increases in membrane excitability and synaptic efficacy as well as to reduced inhibition and is a manifestation of the remarkable plasticity of the somatosensory nervous system in response to activity, inflammation, and neural injury. The net effect of central sensitization is to recruit previously subthreshold synaptic inputs to nociceptive neurons, generating an increased or augmented action potential output: a state of facilitation, potentiation, augmentation, or amplification.”

The focus of this definition is facilitation of pain pathways leading to chronic pain conditions. However, what they do not comment on is that many of these nociceptive neurons (from both somatic tissues and from the viscera) synapse with motor neurons at the cord, known as convergence, and influence their behavior. It would appear that, with high or recurrent stimulation, inhibition of low threshold motoneurons is the most common outcome.
Visceral motor nerves also carry primary sensory fibers, which are divided into two systems: the A-afferent and the B-afferent systems. The A-afferent system converges with the motor neurons in the ventral horn of the spinal cord and may influence local muscle postural reflexes, as described by Bouhasira et al. (1998).

The B-afferent fibers are far more prevalent than the A-afferents and have similar characteristics to nociceptive fibers. The B-afferent system is activated by noxious stimuli, such as inflammation within an organ — irritable bowel syndrome could be such an example — and has a larger influence centrally at the cord than the A-afferent system. Neural drive, especially repetitive stimulation, through these fibers can facilitate (i.e. sensitize) spinal segments.

According to Willard (2002), the B-afferent system is key in initiating the sensitization at the cord. Cervero and Laird (1999) state that nociceptive discharges in visceral afferents can evoke profound changes in the central nervous system; repetitive noxious stimulation of the viscera evokes increases in the excitability of visceral somatic neurons in the spinal cord. What could be more repetitive than the irritation from a medical drug taken daily, a food irritant that is eaten daily, or the presence/overgrowth of a pathological organism such as Candida?

Clinically therefore, it is key to consider what may evoke an inflammatory response within the viscera. Of course, conditions such as pelvic inflammatory disease, inflammatory bowel disease, gastritis, and so on, give away the inflammatory nature of the pathology through their very name. However, there are many simple everyday activities that can cause an inflammatory response in the gut.

Foods that are consumed by people who are intolerant to them will induce an inflammatory response (Shils et al. 2005). In the West, the most common foods that cause inflammation in the digestive tract through intolerance are wheat and dairy  (Shils et al. 2005) dairy intolerance often being a sequel to wheat intolerance due to the inflammatory process obliterating lactase production. While in the East, the most common food intolerances are to rice and soya. Hence it would appear that intolerance may be the result, of both the allergenicity of the food and the level of a) refinement and b) exposure.

Adding weight to this observation, corn is particularly poorly tolerated in the US population, whereas it is better tolerated in European populations, a possible explanation being over-exposure to corn in the US through the extensive use of high-fructose corn syrup (HFCS) as a sweetener in place of cane sugar, which is more commonly used in Europe (Wallinga et al. 2009).

What Granath et al., 2001 found when they presented their paper on milk intolerance (see Milk & Chilies section above) and post-partum pelvic pain at the World Congress on Low Back Pain in 2001, may, in fact, have been not all a biochemical reaction, as they suspected, but a neurophysiological consequence of dairy intolerance.

Other instigators of inflammatory damage to the gut wall include a raft of medical drugs (especially the NSAID’s), alcohol, dysbiotic conditions caused by poor nutrition and lifestyle choices, antibiotics, over-consumption of sugar, chlorinated water, or sympatheticonia (Bland, 2000).

Indeed, a chronic state of increased sympathetic arousal as often seen in middle-aged city dwellers, over-medicated patients, chronic pain patients and over-trained endurance athletes will often result in digestive dysfunction, including dysbiosis and inflammation. (Merck, 2013) This is due to the fact that, when in a state of sympathetic (fight/flight) dominance, peristalsis is inhibited, transit of ingested foods is slowed and conditions for dysbiotic proliferation in the gut are optimized.

All of these factors may individually or collectively create inflammation, which stimulates the B-afferent receptors returning to the cord to create sensitization at the associated segments. This sensitization, it appears, will create inhibition of low threshold motor neurons, such as those that feed the postural fibres of the transversus abdominis, multifidus and other inner unit musculature.

Important clinically, Willard (2002) also explains that, once initiated, such sensitization can survive transection of the dorsal (sensory) roots. In other words, you can completely remove the noxious input; the pain, the inflammation, the dysbiotic condition, the stress, the provocative foodstuff, and facilitation (sensitization) may remain. This is in alignment with Hides et al. (1996) findings that even 1 year after the painful afferent stimulus (low back pain) has resolved, the multifidus was still not firing normally.

Beyond the core

There is little doubt functional core musculature is key in function of both the spine and the appendicular skeleton. This is explained in greater detail in both the Self-Help piece associated with this article and in the paper by Miyake et al. (2013), "Core exercises elevate trunk stability to facilitate skilled motor behavior of the upper extremities", in this section of this edition of JBMT.

There has already been discussion of some of the peripheral mechanisms and strategies that the CNS appears to utilize when there is compromise of central stabilization mechanisms (Bouhasira et al., 1998; Hungerford et al., 2003). While it may be that the effects of pain inhibition, or viscerosomatic inhibition are most clearly observed centrally, and where these phenomena have been most deeply studied, it could be that there are similar processes occurring peripherally as well.
Cross-segmental central sensitization

In early embryological development, the spinal cord is a largely homogenous neural tube without differentiation into segments. The emerging nerve roots arise by differentiation of the neural tube into somites. In their adult arrangement, despite the segmental differentiation, there is significant overlap with adjacent spinal segments. Hence there are no intrinsic physical barriers to the spread of information across ascending or descending segments of the cord.

In research on rats, Brooks et al. (2012), found that chronic nociceptive stimulation is able to provoke extensive sensitization to as many as 6–10 segments in the superficial and deeper laminae of the cord. This rostro-caudal spread across multiple spinal segments is an important factor when considering not just the effect of aberrant stimuli on the core, but also how that may cross-over to affect muscles of the periphery. Since both somatic tissues and visceral tissues have B-afferent fibers, either of these sources can independently or together initiate facilitation (sensitization) (Willard, 2002).

Building on the understanding that it is the tonic musculature that becomes inhibited during pain and in the presence of visceral inflammation, it may be opportune to return to the discussion of tonic versus phasic motoneurons.

Kuno (1984) describes how the tonic motoneurons feed the Type I, slow twitch, muscle fibers and how the phasic motoneurons feed the Type II, fast twitch, muscle fibers. While, clinically and in the lab, the result of aberrant neural drive and central sensitization seems to be that “tonic muscles” become inhibited, Ng et al. (1998) explain that all muscles have a mixture of tonic, slow twitch, Type I fibers and phasic, fast twitch, Type II fibers. For example, the soleus, a “tonic muscle”, has around 85% slow twitch muscles (fed by tonic motoneurons) and 15% fast twitch fibers (fed by phasic motoneurons); while the gastrocnemius, a “phasic muscle”, has around 50% slow and 50% fast.

Therefore it may be technically more accurate to state that a given muscle has a “dominance” in function; as all muscles have both tonic and phasic motoneurons giving rise the fiber type and the dominant function of the muscle. In this instance, the TrA and multifidus would be seen as having a tonic dominance, while the hamstrings, gluteus maximus and latissimus dorsi would have a phasic dominance; but either can be called on to recruit its non-dominant fibers. The hamstrings can stabilize, and if something sharp or potentially injurious was moving toward the belly, the phasic fibers of the TrA will be recruited to “get out the way quickly”.

What this means from a neurological point of view is that it is not the “muscle” that is inhibited by pain, but a given proportion of its fibers. The ramifications of this functionally are uncertain, but it is likely that this means, in the case of the highly tonic muscle, such as the TrA, that it will be largely electrically silent when pain is present, whereas a highly phasic muscle, such as the external oblique will be preferentially recruited (as Tremolaterra et al. 2006 found in their IBS patients).

It also means that pain or noxious visceral afferent drive will affect not just a specific muscle, but the tonic motoneuron pool associated with the segment it returns to and, to a lesser degree, segments several levels above and below.

In this way, something as apparently innocuous to athletic performance as a food intolerance, could have a profound effect through inhibition, not just of the TrA (served through nerves of the thoracolumbar outflow), but also of the tonic motoneurons serving any musculature at or around that same spinal level, such as gluteus minimus/medius, vastus medialis obliquus, or tibialis posterior. The more tonic fibers the muscle has, the more its overall function will be affected (Fig. 3).

Respiration, bloating, emotion

Moving back to the concept that it is the musculature involved in expansion-contraction that is of primary relevance to this discussion, respiration is yet to be reviewed.

Breathing pattern disorder (BPD) is an extremely common clinical finding. Clinically, it is often identified secondary to a primary pain condition, such as low back pain or TMJ dysfunction. However, as Chaitow notes (2004), breathing pattern disorders account for 11% of all internal medicine hospital admissions in the US. Chaitow (2004) also notes that many of the symptoms of a breathing pattern disorder may mimic or, indeed, physically cause other conditions for which patients seek help.

The respiratory rate is controlled by the autonomic nervous system, and hence is in lock-step with each individual’s perceived level of stress. That stress could be running up a hill, sitting at an examination desk, standing on a stage, or being bent over double with back pain. The respiratory system will respond in the same way; by increasing its rapidity.

In addition to the rate, one of the ways the respiratory system adapts to stress is to switch strategy from a deep abdominal breathing pattern, where excursion of the diaphragm and abdominal wall accounts for around two thirds of the respiratory cycle (the remaining third being movement of the thorax). However, in people with an increased stress level, with pain, with a breathing pattern disorder, or with poor posture (upper-crossed syndrome) the likelihood is that they will have what’s termed an inverted breathing pattern — where two-thirds (or commonly all) of the movement of respiration is happening at the chest. What are the ramifications of this from a core function point of view?

Firstly, this inverted breathing pattern tends to compound postural faults by using accessory muscles of respiration, such as pectoralis minor, sternocleidomastoid, levator scapulae to become overactive and facilitated.

The law of facilitation states: when an impulse has passed through a certain set of neurons to the exclusion of others, it will tend to take the same course on a future occasion and each time it traverses this path the resistance in the path will be smaller (Onofrio, 2013). In other words, the more you use a muscle, to breathe, to hold you posture, the more facilitated it will become and the easier it will be to recruit it.

The inverse of this is also true; that the less a given set of neurons receive a nerve impulse the greater the resistance that will build. This might be termed de-facilitation;
more of a deconditioning of neural drive, than a blockage or inhibition of neural drive.

In functional breathing, there would therefore be strong facilitation of the primary muscles of respiration, the diaphragm and the transversus abdominis, which work in a push–pull relationship. As the diaphragm contracts, the transversus relaxes and controls abdominal excursion (eccentrically), then as the diaphragm relaxes, the transversus abdominis engages concentrically to drive exhalation. Ishida and Watanabe (2013) confirm this in their paper assessing activation of the abdominal wall in respiration, found in this edition of JBMT. They state: after maximum expiration, the thickness of the TrA muscle significantly increased, and there was no significant change in the thickness of the IO and EO muscles. Hence the TrA, as evidenced by ultrasound imaging, is engaged in the exhalation portion of the respiratory cycle, as described.

In patients with BPD it is very common to see no movement at the abdominal wall during respiration; all visible respiratory movement is occurring at the chest and above. This means that, as far as respiration is concerned, the transversus abdominis is now completely inactive. This may seem of little consequence, until it is considered that the average number of respiratory cycles per day is around the 20,000-level. If the pattern is dysfunctional, very quickly, the transversus abdominis could become deconditioned and the neural pathways become de-facilitated.

When considering core function, and activation of the transversus abdominis in particular, the readiness with which the nervous system can locate a muscle, how facilitated the neural pathways are to it, is key in daily function.

It is interesting to note that one of the symptoms commonly described by patients with breathing pattern disorder is abdominal bloating (Chaitow, 2004). The exact cause of this, just like breathing pattern disorder itself, is likely to be a summative effect of multiple etiological drivers. For example, aerophagia (the swallowing of air), which increases with breathing rate is a major contributor to bloating in BPD. However, as in the studies of Tremolaterra et al. (2006) on irritable bowel patients, inhibitory effects on the inner-unit musculature (TrA and transverse fibers of IO) are likely to also contribute to bloating in BPD.

In 2003, Hodges assessed the function of the TrA to understand better its role as both a stabilizer of the back and its role as a respiratory muscle. In synopsis, Hodges found that when under stress of perturbation (i.e. the structures of the lumbar spine were potentially in jeopardy) the nervous system would utilize the transversus abdominis to stabilize preferentially until such time as respiratory requirements predominated (the subject began to move into oxygen debt); when the respiratory role of the transversus prevailed and its stabilizer role was sacrificed Hodges, 2003. This suggests a logical hierarchy of physiological function; that when respiratory homeostasis is compromised, the body’s priority is to restore that homeostasis, over and above to stabilize the low back. Hence people suffering with breathing pattern disorders may be more prone to low back pain than the general population (described in more detail by Chaitow, 2004).

**Figure 3** Image from Gray’s anatomy demonstrating the motoneuron pools overlapping for associated muscles associated with different musculature of the lower limbs; the proximity, integration and polysegmental nature of innervation suggests that inhibitory influences on the tonic motor neurons could have far reaching effects impacting on musculature deep into the periphery.
Emotion

On screening the diaphragm for trigger points and tender points, these can often readily be found (Simons et al. 1999). Those bodyworkers who specialize in working with somato-emotional release commonly describe the diaphragm as the seat of emotion; where the body stores trapped emotion.

Offering these observations some face value, physiologically this can make some logical sense when it is understood that the normal physiological response to emotion in human infants is to take a sharp inhalation of air (diaphragmatic contraction), often followed by what can seem like an inordinately long pause, and finally concluded with a real belly-driven cry of emotion. Indeed, this full reaction is like an inordinately long pause, and finally concluded with a real belly-driven cry of emotion. Indeed, this full reaction is seemingly suppressed of the latter phase of this process of emotional release is socialized out of acceptable adult behavior, usually at some point during the teenage years.

Physiologically, the way a Westernized adult may typically respond to an emotional stressor is to, a) take the same sharp inhalation of air on exposure to the emotional stressor (diaphragmatic contraction, transversus abdominis inhibition), and then to b) hold that contractile tension in the diaphragm, until such time as it is c) cried out in private, d) talked out (usually in private) or e) manually released in a one-to-one consultation with a trained practitioner. For many adults, none of these three options are realized and tension remains held.

Interestingly, Simons et al. (1999) state that a finding characteristic of diaphragmatic trigger points is that there is pain on the exhalation phase of respiration, as this is when the diaphragm becomes domed upward and stretched. Of course, this would be the same time that the transversus abdominis is/should be contracting. Therefore, if there is pain at height of the expiratory phase of respiration when, as Ishida and Watanabe (2013) have shown, the transversus abdominis should be most active, what will be the likely response of the nervous system? The likely solution is to inhibit the transversus abdominis—especially in its inner-range contraction.

Summary

In conclusion, it appears there are many factors that can influence core function and, fundamentally, they involve any factors associated with the primal dimension of space (radial contraction); namely eating, breathing and exercise of the deep core musculature.

Since 90% of low back pain is defined as non-specific low back pain (Krismer and Tulder, 2007) which, in turn, is defined as pain not attributed to a recognizable pathology, such as infection, tumor, osteoporosis, rheumatoid arthritis, fracture, or inflammation, (Heijden, 1991) it is entirely feasible that, with a more comprehensive evaluation of visceral function, the pain may be attributable to sensitization; either through pain sensitization directly, or through cumulative micro-trauma as a result of inhibition to the inner unit musculature. To have optimal core function it would appear that the prerequisites are a sound diet, low in foods or habits that may contribute to inflammation, resulting in repetitive firing of the B-afferent system; ample management of stress summation and emotional expression to allow for functional breathing patterns; and activation of the muscles of stabilization through the use of directly targeted (isolated) and indirectly targeted (integrated) exercise modalities. Examples of these are discussed in the associated practical section.

Application of this advice should mean that the model of consciousness described earlier in this piece can be carried through from unconscious incompetent (not knowing what’s wrong), to conscious incompetent (knowing what but not how), to conscious competent (knowing what and how), and finally to unconscious competence. This unconscious competence; the level of competence where the client is operating functionally without having to engage the conscious mind, is the point bodyworkers and movement therapists should strive to reach with their client; but which is so commonly fallen short of. Hopefully, this way of looking at core function, and indeed function of the tonic motor neurons as a whole, may prove useful in creating more unconsciously competent people in the world.

Simply put, without getting the most primal nature of human function correct; breathing and eating, attempts to optimize core function may be limited, or even futile.

References


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