Commentary

The Ileoceleal Valve Point and Muscle Testing: A Possible Mechanism of Action

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INTRODUCTION

The muscle test procedure is a common test used by manual therapists. It is primarily used for the determination of muscle strength in orthopaedic testing, but its application is not limited to such use. The muscle test procedure has acquired other uses as a part of diagnostic systems that include largely multi-step diagnostic protocols of techniques such as Neuro-Emotional Technique (NET) and Applied Kinesiology (AK), amongst other techniques.

These procedures are both loved and loathed, depending on the end of the chiropractic spectrum with which one identifies (vitalistic or mechanistic). Fundamental to the vitalistic diagnostic protocols is the application of the muscle test before and after stimulation of points on the body said to represent various anatomical sites and functions.¹,² One such point is the ileocecal valve point. This point is said to represent the function of the ileocecal valve and is associated with various syndromes, including low back pain.¹,²

The ileocecal valve point is located at McBurney’s Point,² which is found 25mm from the anterior superior iliac spine on a 45⁰ line towards the umbilicus. If a muscle that tests strong weakens after stimulation of the ileocecal valve point (ICVP), it is said to indicate dysfunction of the valve. The consequences of a malfunctioning ICV can mean much more than low back pain according to Walker¹ and Walther,² who note the effects of dysfunction as bowel changes, low back pain, headaches, shoulder pain, nausea, neck stiffness, sinusitis, dizziness, fatigue, pallor, dark circles under the eyes, to name a few examples.

In this paper, we will attempt to synthesise new research that provides evidence for the existence of a mechanism to explain this process. Discussion of this literature is important in light of an explanation of a mechanism that has hitherto not been explained adequately in terms of published peer-reviewed literature.

BACKGROUND

The ICV is located at the junction of the ileum and the caecum. NET and AK doctrine suggest that the skin above the ICVP, when stimulated, would make a previously strong-testing muscle weak if there is an underlying imbalance in the functioning of the valve.¹,² This test forms part of a multiple diagnostic protocol in both NET and AK. It is important to note that this discussion will focus only on 1) the potential for the ICV to change skin function, 2) that stimulation of the skin by touch could cause muscle weakness, and 3) stimulation of the skin (at the ICVP) might cause a previously strong muscle to test weak after the stimulation of the skin at the ICVP.

CONTROVERSY

Research associated with technique groups lags behind claims of efficacy often made by some practitioners within the groups. What research has been attempted has sometimes attracted criticism, however some of this criticism appears to be based on fundamental, unproven concepts associated with the techniques rather than on the content of the studies.³ We wonder why such positions are taken against reasonable attempts to begin a body of literature in an area where one does not already exist.

It is frequently said of emerging techniques such as NET and AK that they must investigate each step of their multi-step evaluation and treatment protocols.⁴ Research has begun to investigate some of this work with genuine attempts to establish a body of peer-reviewed research publications to support some of the fundamental procedures. For example, we have examined the test-retest inter-examiner reliability of muscle testing.⁴ Following this work we attempted to investigate a diagnostic use of muscle testing in another study.⁵ We examined whether the ICV procedure was reliably associated with what the technique groups purported to do. That is, could the ICVP be reliably associated with the presence of low back pain.¹,² This seems to be a reasonable proposition to start an investigation of these complicated techniques. In other publications, background conceptual papers have been published,⁶⁻¹¹ as have case history papers documenting objective outcomes associated with non-musculoskeletal condition management,¹²⁻¹⁴ however no implication for successful management of any condition was implied in the recent ICVP study.¹⁵

It is apparent to the authors that some criticism has been appropriately levelled at groups that have made claims that
were not supported by quality scientific data, however other criticism appears to stem from the belief that the techniques are “unscientific,” and therefore all they do is unscientific. Such criticism must be relevant to the evidence presented; otherwise it clearly represents as much bias as that frequently referred to by the detractors of the technique.

The key issue relevant to this discussion is the possibility (or not) of the muscle test being able to measure function of a visceral anatomical structure: the ileocecal valve. Vitalists propose various connections, while mechanists suggest that no evidence exists to support any association between the ileocecal valve, the ileocecal valve point and the outcome of the muscle test.

THE ILEOCECAL VALVE

The basic question to ask is, Is there a plausible mechanism that the ICV structure can affect the strength of a muscle?

Muscle strength can be modulated by local, peripheral and central mechanisms. These mechanisms include local muscle factors such as dysfunction of muscle chemistry (or fatigue-based processes) and disorders of neuromuscular functioning; peripheral factors including hormone effects; and other spinal and supraspinal mediated effects. It is also known that there is diurnal variation to muscle strength.

We propose a mechanism that distension of the ileum could provide the stimulus by which a central action on muscles could take place. These central effects may include motivational factors; descending inhibitory effects of the higher centres that include the limbic, cortical and hypothalamic systems. Important in these mechanisms is the knowledge that the higher centres output universally and in particular to the intermediolateral centres of the cord to influence body-wide muscular functioning as well as visceral function (and not just to the site of origin). So it is possible amongst these, or through a hitherto unknown mechanism, that muscle strength may be reduced globally in the presence of appropriate stimuli.

It has been stated that there is no known anatomical neurologic, hormonal or other physiologic relationship between a muscle test in a limb and the ileocecal valve. We suggest, based on the work outlined in this commentary, that there is evidence for such a relationship, albeit indirect.

Much recent research has appeared in an area of the literature referred to as the brain-gut axis. This model is based upon evidence that may provide explanation of some of the effects noted with muscle testing after stimulating the ICVP (and possibly other points also).

TOUCH

Touch is an active process, according to Fanselow and Connors, but, they ask, How does the body’s somatic sensors influence its movement? They suggest that positive feedback is triggered by a sensorimotor loop. Such a loop has been demonstrated in the cat for the first time only recently.

Stimulation of the Skin

Nicholas has shown that scratching the skin can alter the maximum muscle contraction possible in an adjacent muscle. In addition, there is evidence that stimulation of tactile afferents of the foot can provide short-term latency of foot motoneurone pools, including low-threshold cutaneous mechanoreceptors. In addition, Nakajima et al. have demonstrated that such innervation occurs in a highly topographical manner. Collectively, this research has demonstrated that stimulating the skin can cause changes in muscle function. Further research should replicate these methodologies on trunk muscles utilising similar stimulation protocols to that used by the NET and AK groups.

Another study has taken a different approach. Electrical impedance of specific dermatomes has been measured reflecting the role of internal organ pathologies on function. The authors concluded that electrical dermal-visceral impedance measurement has the potential to serve as a screening tool for inner organ pathologies.

Changes in organ function are not limited to muscle changes. Another study by Uchida et al. in an ovarian model demonstrated altered blood flow following a noxious cutaneous stimulation. They confirmed that local and supraspinal pathways mediated the efferent arc of the reflex action. Their work implied that the more lateral the stimulus location (to the midline), the more likely it would follow a supraspinal path. Most significant, Kurosawa and co-workers demonstrated changes in hepatic blood flow following mechanical stimulation of the skin with noxious stimuli. Whilst this does not support the concept of light touch creating this change, it does provide a reflex pathway for skin afferents affecting visceral function.

Recent work on the brain-gut axis may help to elucidate a common pathway for the assessment and treatment of functional visceral and related disorders. Studies such as these are required in human models with similar stimulation protocols to provide direct evidence of relevance these of effects and mechanisms in any proposed model to explain the ICVP reflex.

It is known that referred pain from visceral structures can affect the skin, and the mechanism may also be relevant to the ICVP. In a recently published paper, stimulation of the ileum reportedly caused pain and mechanical hyperalgesia in the skin of the abdominal area. Further support for this concept results from the knowledge that when an indelible dye is added to an inflamed uterus in a rat, the dye is expressed into the skin over the abdomen. Together these studies provide evidence that trophic changes are associated with areas of visceral referred pain. Moreover, opinion exists that these changes are whole-gut in nature and stimulated by many different specific sites.

Taken together, these studies establish a potential explanation for how the ICVP may change in abdominal skin and how stimulation of that skin may cause reflex changes in many systems that could result in a change in muscle strength.

ICV AND LOW BACK PAIN

The ICVP muscle test is said to be important in the diagnosis of low back pain. Because of this, it is important to explain the relationship between the anatomical structure that is the ICVP and the potential for pain arising from the
musculoskeletal structures of the back, or at least perceived to be from that location.

Researchers have demonstrated that stimulating the region of the skin above the ICVP may activate a stretch reflex that changes the tonicity of the abdominal muscles (rectus abdominis and obliques). Further research has demonstrated that these muscles may activate or be associated with the function of the transversus abdominis muscles that in turn may affect function of the back. In addition, dysfunction of lumbar muscles in timing of the recruitment of muscle contraction has been implicated in the generation of lower back and sacroiliac pain. Indahl et al. have suggested that the sympathetic and parasympathetic pathways may regulate function by controlling the intricate neuromuscular balance in lumbar motion segments. Thus, lumbar musculature could be less efficient in stabilising the lumbar neuromuscular feedback system after changes in abdominal function, changes that could result from dysfunction of the abdominal viscera.

The exact nature of the neurophysiological control associated with the abdominal muscles is attracting much attention, but further work is required to fully elucidate their role in the mechanism proposed here, particularly how they are stimulated by visceral structures.

Houghton and Whorwell discuss symptoms associated with functional gastrointestinal disorders. In their paper, they discuss the role of pathophysiology including psychological factors, intestinal gas accumulation, fluid retention, food intolerance and malabsorption of sugars, weakness of abdominal musculature, and altered sensorimotor function. Thus, the role of these bowel signs and symptoms in the generation of low back pain may not be so tenuous after all. To illustrate, cases of abdominal pathology presenting as low back pain have been recorded. Despite this, gastrointestinal dysfunction as a cause of low back pain needs further investigation before any definitive statement can be made about its clinical utility.

A Biomechanical Explanation

The evaluation of the muscle test is often only considered from a mechanistic viewpoint. This occurs in parallel with the ongoing anecdotal claims of efficacy from field practitioners who continue using the ICVP in a non-mechanistic, vitalistic manner. Another potential explanation for the role of the ICVP in low back pain sufferers is that the ICVP may be the result of psoas muscle dysfunction. The psoas muscle has been suggested as a major contributor to many cases of low back pain. Perhaps it would be more palatable to think of the ICVP point in such anatomical terms. It appears to us that such an explanation may be as reasonable as the one offered by the technique founders, but one that is more “orthopaedic in nature.” Despite this, both are untested hypotheses in terms of aetiology, and neither has been tested in any study to date.

The ICV Is a Sphincter

An interesting side note to this discussion is that researchers now refer to the ICV as a sphincter rather than as a valve, and they report the function governed by inhibitory and excitatory neurogenic influences. Important in any discussion of the neurogenic influences is the connection of the viscera to higher centres, as this connection is the link for the possible connection with muscle strength testing. Alterations in afferent signals to the limbic and paralimbic areas of the brain have been demonstrated in irritable bowel syndrome patients. Changes have been shown to occur in the anterior insula and dorsal anterior cingulate cortex, and the amygdala, rostroventral ACC, and dorsomedial frontal cortical regions. These changes are known to affect the emotional, autonomic and descending modulatory responses to pain and are associated with visceral intolerance.

Visceral intolerance to distension can be gut-wide in patients with multiple symptoms, or specific in those exhibiting a specific site of symptoms. Furthermore, noxious stimuli applied at a site can interact with others to diminish the sensation elicited from another site.

Intolerance can grow secondary to distension and result in the gut being damaged from the build-up of noxious chemicals from chyme. The damage takes the form of alterations in gut mucosa and blood flow, and these changes influence feedback to the spinal cord and affective pain centres in the brain producing changes in the output of higher centres known to affect general muscle tone. Shafik et al. suggested that a balance between chyme delivery and gastric filling affected gastric emptying and ultimately the degree of distension of the ileus. This was termed a cephalic excitatory reflex and could be the catalyst for the for the initiation of gastrointestinal disorders secondary to neurogenic and myogenic influences.

It is noteworthy that chronic constipation and bowel distension are common in low back sufferers, even if secondary to analgesic use.

In fact, several pathways have been elucidated regarding the ileosphincteric reflex. These include excitatory muscarinic and inhibitory nitrogentic and beta-adrenergic pathways, inhibitory ileosphincteric reflex mediated by nitrogentic and beta-adrenergic postganglionic neural pathways, in the pig at least. So perhaps there is a possibility of such reflexes affecting variables not yet confirmed in the human.

It appears that there is substantial indirect evidence to support a testable hypothesis that bowel dysfunction can alter muscle function, probably through the action of higher brain centres. It is important to note that Monti et al. have demonstrated that false (incongruent) statements could change muscle strength while testing with a muscle test. Thus, it is possible that the changes in output from the higher centres can affect muscles globally. This mechanism may explain the claim of many kinesiologists that any muscle may be used in a diagnostic muscle test. Despite this, some muscles appear to be more reliable for diagnostic purposes.

FURTHER RESEARCH

True science has observation as its cornerstone. Such observation often begins in the clinic and then progresses to the laboratory. The fact that the exact mechanism is currently unknown does not preclude the possibility that the effects of the mechanisms can be observed, hence the objective documentation of case outcomes. Such research, however, should be followed by higher level research in the form of randomised controlled trials before any firm claims of efficacy can be made for the techniques.
As mentioned in our paper, several groups suggest that the ICVP is representative of the anatomical structure that is the ICV. At present there is no direct evidence for such a statement, however absence of evidence is not evidence of absence. As presented in this commentary, there is interesting new evidence to support the possibility—evidence that may not have been viewed by many, as it does not fall within the usual readings of the average chiropractor.

**CONCLUSION**

This paper has provided evidence for the existence of a mechanism to explain how the ileocecal valve could cause muscle weakness when in lesion and how stimulation of the skin could affect the function of muscles. These papers bring evidence to the debate that may help uncover the mechanisms underlying some of the diagnostic protocols used by NET, AK and other related practitioners.

Closing one’s mind to advances in science because of a competing paradigm limits progression of science. Our collective goal should be to evaluate recent and exciting developments in science and look for new methods to test our clinical observations. Only through such investigation will the untested areas of chiropractic truly advance into the realms of respectability.

**REFERENCES**

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