

# MUSCLE ENERGY CONCEPTS: A NEED FOR CHANGE

**Fryer G. Muscle energy concepts - a need for change. *Journal of Osteopathic Medicine*. 2000;3(2):54-59.**

**Gary Fryer**

*School of Health Science, Victoria University*

Address correspondence to Gary Fryer, School of Health Science, City Campus Victoria University, P.O. Box 14428 MCMC, Melbourne 8001, Australia.

Email: [gary.fryer@vu.edu.au](mailto:gary.fryer@vu.edu.au)

The growth in popularity of osteopathic muscle energy technique (MET) over the last two decades with osteopaths, medical practitioners, physiotherapists, chiropractors and remedial therapists is likely testament to its clinical efficacy. The techniques were originally devised and described by Fred Mitchell Snr in the 1940s and 1950s and the first technique manual published in 1979.<sup>1</sup> It is time to critically examine many of the concepts behind muscle energy as, while our understanding of biomechanics and manual medicine has increased since this time, the theory behind these techniques have remained relatively unchanged. This article aims to highlight areas of MET theory and practice that appear outdated, and offer what may be more plausible explanations and alternatives that are consistent with current knowledge.

## **EVALUATION PROCEDURES**

Fred Mitchell Snr first developed MET to treat dysfunctions of the pelvis based on his own clinical model of pelvic biomechanics, and later developed spinal techniques consistent with the Fryette model of spinal coupling. While the theories behind these

biomechanical models appeared sound at that time, more recent evidence necessitates these models be modified.

Mitchell<sup>1</sup> has described a variety of sacroiliac and pelvic somatic dysfunctions. The diagnostic criteria for these dysfunctions are a combination of static asymmetry of pelvic landmarks and forward flexion tests to determine the dysfunctional side (right or left sacroiliac joint).

### **Standing and seated forward flexion tests**

According to Mitchell<sup>1</sup>, the standing flexion test is indicative of *ilio-sacral* motion while the seated flexion test is indicative of *sacro-iliac* motion. A positive test is determined by asymmetrical excursion of one posterior superior iliac spine (PSIS) on trunk flexion, the positive side travelling further and more superior than the other side. The explanation for this behaviour is that as the sacrum nutates (flexes) with lumbar flexion, the fixed innominate is carried with it and draws the PSIS more cephalad than the uninvolved side.

A major concern behind the claimed specificity of these tests is that a multitude of factors must influence them. The forward flexion tests assume the sacrum will nutate and carry the fixed innominate with it, but one study<sup>2</sup> demonstrated the variability of sacral motion with trunk flexion. This study used trans-cutaneous implants and three-dimensional photography and revealed the sacrum was just as likely to nutate as to counternutate during flexion of the trunk. Asymmetries in lumbo-pelvic rhythm, leg length, scoliosis, hip flexion, sacroiliac joint anatomy, hamstring, piriformis and quadratus lumborum muscle length must also have a profound effect on pelvic symmetry during forward flexion.

Egan et al<sup>3</sup> found no correlation with the standing forward flexion test and static pelvic asymmetry or low back pain. While the authors' conclusion that standing flexion is not an accurate test of sacroiliac dysfunction is questionable (pelvic asymmetry and low back pain may not be indicators of sacroiliac mobility), the study does not lend any support for

the test. Furthermore, preliminary studies have not supported the inter-examiner reliability of these tests.<sup>4</sup> Yet on the basis of the forward flexion tests, a MET practitioner will decide whether to treat the right or left sacroiliac joint.

What can be reasonably said about the forward flexion tests? The proposed mechanism of the tests appears implausible (due to the variability of sacral motion in trunk flexion) and they also appear poor indicators of low back pain or static pelvic asymmetry. The tests probably indicate some functional asymmetry but do not likely differentiate whether it be articular, myofascial, or anatomical. Possibly the most significance that can be attributed to these tests is when there is an obvious difference between standing and seated. When seated, all influence of the lower extremity is removed; a positive standing but negative seated test might implicate some asymmetry in the lower extremity.

### **Pelvic asymmetry and dysfunction**

The nature and degree of sacroiliac motion remains a controversial subject. Sacroiliac motions are small, complex and involve simultaneous rotation and translation with no single or simple axis.<sup>5</sup> Nutation and counternutation (flexion and extension) of the sacrum and rotation of the innominates appear the most widely accepted motions.<sup>6</sup> Bogduk<sup>7</sup> described the sacroiliac joint as having no primary motion but acted passively as a “stress relieving” joint to accommodate torsional stresses through the pelvis during ambulation.

Harrison et al<sup>5</sup> reviewed the anatomy and biomechanics of the sacroiliac joint and reported several studies that demonstrated large variations in surface configuration and orientation to the sagittal plane. Jacob & Kissling’s study<sup>2</sup> of sacroiliac mobility in healthy individuals revealed low averages for total rotation (1.7 degrees) but one subject, a former top-class track athlete who suffered from symptoms of sacroiliac hypermobility, exhibited more than 6 degrees of rotation.

Descriptions of sacroiliac dysfunction are even more controversial than sacroiliac motion and remain a clinical observation unsupported by biomechanical studies. Due to the variability of sacroiliac anatomy it may be possible that the many sacroiliac joint dysfunctions described by Mitchell all can occur, but only in individuals with susceptible sacroiliac joint anatomy. Hence one individual may be prone to inferior sacral shears while another to innominate inflares as a result of their sacroiliac joint orientation and congruence. It may also be possible that these distortions are secondary to myofascial imbalance, and treatment (such as for sacral torsions) may be primarily affecting muscles.

The chief diagnostic criteria for the dysfunctions described by Mitchell are static asymmetry of pelvic anatomical landmarks. In a recent study, Levangie<sup>8</sup> compared pelvic landmark asymmetry of 144 low back pain patients and 138 control subjects and found pelvic asymmetry was not positively associated with low back pain in any way that seemed clinically meaningful. Another study by the same author<sup>9</sup> demonstrated that four sacroiliac motion tests, including the standing and seated forward flexion tests, were not useful in identifying objectively measured innominate torsion.

Tullberg et al<sup>10</sup> showed that manipulation (high velocity and unspecified MET) appeared to improve clinical findings but did not alter the position of the sacrum in relation to the ilium as determined by roentgen stereophotogrammetric analysis. These authors were convinced that something happened when manipulating the sacroiliac joint, but it wasn't a detectable positional change between the sacrum and the ilium. Furthermore, the reliability of palpating pelvic landmarks has been questioned by the authors of several studies.<sup>11,12</sup>

Asymmetry of the pelvis is likely to be common and unrelated to biomechanical dysfunction. An asymmetrical static pelvic finding should be considered an incidental finding unless supported by positive motion, springing or pain provocation tests.

## **Clinical assessment of the sacroiliac joint**

Controversy exists concerning not just the reliability but the validity of sacroiliac joint motion and pain provocation tests. Several studies<sup>13,14</sup> have compared various manual tests to the “gold standard” sacroiliac joint anaesthetic block and have found the tests to have poor predictive value for pain arising from that joint. However, Broadhurst & Bond<sup>15</sup> in a double blind trial tested subjects who responded to three pain provocation tests with either sacroiliac blocks or control injections of saline. They found the three tests had a high predictive value for pain arising from the sacroiliac joint.

Other studies have compared clinical sacroiliac joint tests in low back pain and control populations with mixed results. Toussaint et al<sup>16</sup> found no correlation between clinically diagnosed sacroiliac dysfunction and low back pain, but Cibulka & Koldehoff<sup>17</sup> claimed a cluster of motion tests (including the standing flexion, of which three of the four tests had to be positive) had acceptable sensitivity, specificity and positive predicative value for low back pain, clinically diagnosed as “sacroiliac dysfunction”.

Are sacroiliac blocks or detection of low back pain subjects valid indicators of the efficacy of motion and pain provocation tests? Many osteopaths<sup>18</sup> believe that hypomobile sacroiliac joints are not necessarily a source of pain but may produce compensatory strain elsewhere; hence a diagnosis of sacroiliac dysfunction need not relate directly to low back pain. Sacroiliac blocks do not anaesthetise extra-articular structures such as the posterior sacroiliac ligaments.<sup>19</sup> If dysfunction involved strain of these ligaments, the sacroiliac joint would respond to clinical testing but not to diagnostic blocks.

Much of the difficulty in studying sacroiliac dysfunction relates to its uncertain aetiology or whether it exists at all. Pelvic anatomical asymmetry appears common and unrelated to either back pain or motion testing. The sacroiliac joint has certainly been implicated in chronic low back pain but the aetiology is uncertain.<sup>7</sup> Mitchell has described functional

lesions that involve restriction of motion and malposition but motion tests appear unreliable and of questionable validity. The only study to accurately examine manipulation and sacroiliac joint position appears to refute the concept of joint malposition.<sup>10</sup> Dysfunction in the myofascial structures attaching to the pelvic girdle may play a role in clinically assessed pelvic distortion and altered motion. Sacroiliac joint capsule tears and ligament strains may be the most plausible cause of pain originating from the sacroiliac joint.

What tests might be used to determine sacroiliac dysfunction? No single test has proven reliable, but some studies<sup>15,17</sup> indicate the more tests used, the higher the likelihood of significance. If the forward flexion tests are used with caution, and various motion, springing and provocation tests are utilized, detection of sacroiliac dysfunction may be more reliable.

### **Fryette's model of spinal coupling**

MET for intervertebral somatic dysfunction has been married to the conceptual model of spinal coupled motion as first described by Harrison Fryette in 1918. Fryette<sup>20</sup> studied coupled motion in a dissected spine and designated Type I motion as sidebending with conjunct rotation to the opposite side and Type II motion as sidebending with conjunct rotation to the same side.

According to Fryette<sup>20</sup>, Type I coupling occurred in the thoracic and lumbar regions if the spine was in neutral, while Type II coupling occurred if the spine was either in a flexed or extended position and then sidebent. Type II coupling was said to always occur in the typical cervical spine regardless of position.

Evaluation of spinal segmental dysfunction, according to MET authors,<sup>1,18,21,22,23,24</sup> involves examination of the relative prominence of vertebral transverse processes (TP) when the spine is in neutral, flexion and extension. A prominent TP is thought to indicate restriction of rotation to the opposite direction; if the TP is most posterior in spinal

extension, the restriction is assumed to be extension, rotation and sidebending to the same side. The cervical spine is examined using lateral translation in both cervical flexion and extension.

When using this model, there are only three types of motion restriction combinations possible: restriction of sidebending and rotation to the opposite side (Type I), restriction of flexion, sidebending and rotation to the same side (ERS dysfunction, Type II), and restrictions of extension, sidebending and rotation to the same side (FRS dysfunction, Type II). According to authors of MET, restrictions of side bending and contralateral rotation cannot also involve restrictions of flexion or extension, and no techniques are described for such combinations.<sup>1,18,21,22,23,24</sup>

Recent studies lend support for Fryette's observations in the cervical spine but not in the lumbar spine. Coupled motion is a highly complex phenomenon that is altered by posture and involves coupled translations, flexion and extension as well as rotation and side bending<sup>25</sup>. It appears, from studies on cadavers and living subjects, that coupled motion in the lumbar spine is variable within an individual (from segment to segment) and highly variable between individuals.<sup>26</sup> There is some support for ipsilateral coupling of rotation and sidebending in lumbar flexion but not in extension.

Spinal coupling in the lumbar region appears too complex and variable for any simplistic model to hold true, so it is a particular concern when Fryette's "laws" are used as a predictive diagnostic model. On the basis of a single static finding (such as a posterior TP when the lumbar spine is in a flexed or extended position) MET authors<sup>1,18,21,22,23,24</sup> claim to know the untested motion restrictions in all three planes and perform specific techniques based on this assumed coupled motion. In the lumbar spine, at least, such assumptions are not valid.

How should we attempt to diagnose segmental dysfunction? It would be prudent not to rely on a single positional finding (as advocated by most authors of MET) but attempt to confirm such findings with motion testing and treat accordingly. While it is true that

motion palpation has not proved to be reliable,<sup>27</sup> when it is used with other criteria, such as end-feel, tissue texture, tenderness and pain provocation, several studies have demonstrated greatly increased reliability in examination of the cervical and lumbar spine.<sup>28,29,30</sup>

## **TREATMENT: HOW DOES IT WORK?**

The physiological mechanisms responsible for therapeutic effect of most manual techniques are controversial and poorly understood. At best, we may say what explanations appear likely and are supported by limited evidence and what explanations appear unlikely. Yet it is important to continually examine the theory behind our methods as changes in scientific thought can have practical implications and failure to critically reflect may result in the therapy being discredited by implausible dogma.

### **The shortened segmental muscle theory**

The explanation for the therapeutic action of MET in spinal dysfunction that has gained the widest currency is that shortened monoarticular muscles, which restrict joint motion, are stretched.<sup>18</sup> This explanation is based on Korr's<sup>31</sup> proprioceptive model of somatic dysfunction that attributes joint restriction to abnormal sustained contraction of segmental muscles. Korr proposed that disturbed afferent input into a segment of spinal cord would facilitate the spinal interneurons and produce increased motor activity of segmentally innervated muscles. The use of muscle energy is said to inhibit motor activity via the Golgi tendon organs<sup>32</sup> or the muscle spindles.<sup>18</sup>

The biggest problem with this model is the lack of evidence to support increased muscle activity associated with segmental dysfunction or spinal pain. Denslow and Korr's<sup>33</sup> research during the 1940's and 50's provided some foundation for this concept, but there has been little supporting evidence since.



The role of muscle activity in spinal pain and dysfunction is still unclear. While some studies<sup>34,35,36</sup> have recorded higher levels of lumbar paraspinal muscle EMG activity in certain postures, possibly due to muscle guarding and pain avoidance, many studies have demonstrated *decreased* dynamic activity and increased fatiguability<sup>36,37,38</sup> of these muscles in low back pain subjects. Deep segmental muscles, such as multifidus, have been shown to rapidly atrophy in the location of spinal pain,<sup>39</sup> just as suboccipital muscles have been shown to atrophy with chronic neck pain.<sup>40</sup> Richardson et al<sup>41</sup> believe that spinal pain is accompanied by inhibition and atrophy of the deep segmental “stabilizing” muscles, and overactivity of the longer superficial “global” muscles. Whether segmental muscle inhibition is a cause or effect of spinal pain is unclear, but it contradicts the MET belief that these muscles are overactive and restrict segmental motion.

Possibly the only area of research that lends support for the muscle contraction theory is that which has investigated myofascial trigger points. Spontaneous electrical activity indicating active contraction of muscle fibres has been detected at the nidus of muscle trigger points by several studies.<sup>42,43</sup> However, on the balance of current evidence, sustained contraction of segmental muscle as a common component of spinal dysfunction appears unlikely.

### **Post-isometric relaxation**

It is a common belief within schools of manual therapy that isometric contraction and relaxation of a long muscle under stretch enhances that stretch. Although some studies have offered conflicting results, several studies have demonstrated improved flexibility using isometric contraction compared to static stretch.<sup>44,45</sup> Other studies have showed MET to be effective in increasing range of motion in the cervical spine<sup>46,47</sup> and the lumbar spine.<sup>48</sup>

This “post isometric relaxation” phenomenon has been attributed to neurological inhibition as discussed above.<sup>18,32</sup> Yet many studies have demonstrated that reflex EMG

activity does not occur in a slow stretch and so cannot limit range of motion.<sup>49,50</sup> A more plausible explanation may lie with the biomechanics of connective tissue.

### **Connective tissue changes**

Connective tissues display mechanical properties relating to both their fluid (viscous) and elastic components. “Creep” is the temporary elongation of connective tissue during stretch as a result of its viscoelastic properties. Permanent “plastic” changes occur as a result of micro-tearing and remodelling of connective tissue fibres. Bandy et al<sup>51</sup> identified 30 seconds as the optimal duration for an effective stretch; MET, which can maintain muscle elongation for this duration, may produce increased muscle length by a combination of creep and plastic change in the connective tissues.

Taylor et al<sup>52</sup> examined the effects of repeated contractions versus passive stretch on rabbit tibialis anterior muscle. This study found that isometric contraction resulted in decreased passive tension in the muscle at neutral length, a finding normally associated with passive stretching. Passively stretching a muscle principally stretches the connective tissue elements arranged parallel with the muscle fibres. It has been proposed<sup>53</sup> that when the stretched muscle isometrically contracts, the contracting filaments place tension and stretch on the tougher “in series” connective tissue elements, which are not normally tensioned by passive stretch.

Thus post-isometric relaxation may principally be a biomechanical event: a combination of viscoelastic creep and plastic change in the parallel and series connective tissue elements of the muscle, above and beyond that obtained by passive stretch.

### **Venous and lymph drainage**

Muscle contraction and relaxation is a major mechanism of assisting movement of venous and lymphatic fluid.<sup>54</sup> Many muscle energy authors<sup>1,18,23,24</sup> have suggested that MET may help lymphatic and venous drainage. It has been suggested that injury may

produce paraspinal muscle tissue damage and cause inflammation and congestion. This in turn could produce altered segmental tissue texture changes and tenderness.<sup>55</sup> It is plausible that MET, using repetitive light muscle contractions, may increase venous and lymphatic drainage and relieve paraspinal fluid congestion.

### **Trans-synovial flow**

Minor trauma may produce zygapophysial joint capsule tears<sup>7</sup> and result in synovial effusion. Passive joint motion and rhythmic muscle contractions can produce zygapophysial joint intra-synovial pressure fluctuations<sup>56</sup> that may increase trans-synovial flow out of the joint to relieve the effusion. Such a change has been suggested to result in less pain and increased segmental range of movement,<sup>55</sup> but this has not been formally investigated.

### **Inhibition of pain**

Joint movement and isometric muscle contraction will stimulate joint and muscle proprioceptors. This may produce pain relief according to the Gate-control theory<sup>57</sup> where mechanoreceptor afferents carried by large diameter axons inhibit nociceptor afferents at the dorsal horn of the spinal cord. Several studies have demonstrated mobilization and manipulation to have analgesic effects.<sup>47,58</sup> One of these studies<sup>47</sup> compared manipulation to MET and suggested that while they both produced increases in range of motion, manipulation may be more effective for pain relief. However, this is an area that requires further investigation.

### **Motor control and muscle recruitment**

Stimulation of proprioceptors by muscle contraction may also influence motor control. Deep segmental muscles are inhibited and atrophy in people with low back pain.<sup>39,41</sup> It has been suggested that gentle, precisely controlled spinal muscle contraction as used in MET may increase the recruitment of such muscles and help the central nervous system

improve coordination of that region.<sup>55</sup> This theory holds promise in light of current beliefs concerning spinal muscle inhibition,<sup>41</sup> but remains to be formally investigated.

## CONCLUSION

It is time to critically examine many of the concepts, theories and practices that underlie muscle energy technique. It has been argued that many of the diagnostic tests used with this approach are not supported by a sound rationale.

Static pelvic asymmetry is likely to be common and unrelated to biomechanical dysfunction. Sacroiliac dysfunction should only be considered with the presence of positive motion, springing or pain provocation tests; the forward flexion tests should be used with caution as they are not likely to be good indicators of sacroiliac dysfunction. Similarly, static findings in the lumbar region should be corroborated with motion testing, altered end-feel, segmental tissue texture change and possibly pain provocation, as the Fryette predictive model does not appear to be valid in the lumbar spine.

The commonly cited explanation for the therapeutic action of MET appears unlikely, and alternative explanations have been offered. It is likely that biomechanical changes in the connective tissues, rather than neurological mechanisms, are primarily responsible for post-isometric relaxation. Lymphatic drainage, trans-synovial flow, inhibition of pain and changes in motor control and muscle recruitment may all have a role in its therapeutic action.

Muscle energy technique is seen as an effective, non-traumatic therapeutic approach by practitioners of many disciplines and is a credit to the osteopaths who pioneered its development. The challenge ahead for the osteopathic profession is to revise the way MET is practiced and taught, and to validate by research the theoretical basis and clinical efficacy of this popular technique to ensure its reputation and credibility for the future.

## References

1. Mitchell FL Jr, Moran PS, Pruzzo NA. *An Evaluation and Treatment Manual of Osteopathic Muscle Energy Procedures*. 1979, Institute for Continuing Education in Osteopathic Principles, Missouri
2. Jacob HAC, Kissling RO. The mobility of the sacroiliac joints in healthy volunteers between 20 and 50 years of age. *Clinical Biomechanics*. 1995;10:352-361
3. Egan D, Cole J, Twomey L. The standing forward flexion test: an inaccurate determinant of sacroiliac joint dysfunction. *Physiotherapy*. 1996;82(4):236-242
4. Vincent-Smith B, Gibbons P. Inter-examiner and intra-examiner reliability of the standing flexion test. *Manual Therapy*. 1999;4(2):87-93
5. Harrison DE, Harrison DD, Troyanovich SJ. The Sacroiliac Joint: a Review of Anatomy and Biomechanics with Clinical Implications. *Journal of Manipulative and Physiological Therapeutics*. 1997;20(9):607-617
6. Norkin CC, Levangie PK, *Joint Structure and Function: a comprehensive analysis*, 2<sup>nd</sup> Ed. F.A. Davis Company, Philadelphia, 1992
7. Bogduk N. *Clinical Anatomy of the Lumbar Spine and Sacrum*, 3rd Ed. Churchill Livingstone; 1997
8. Levangie PK. The association between static pelvic asymmetry and low back pain. *Spine*. 1999;24(12):1234-42
9. Levangie PK. Four clinical tests of sacroiliac joint dysfunction: the association of test results with innominate torsion among patients with and without low back pain. *Physical Therapy*. 1999;79(11):1043-1057
10. Tullberg T, Blomberg S, Branth B, Johnsson R. Manipulation does not alter the position of the sacroiliac joint. *Spine*. 1998;23(10):1124-8
11. O'Haire C, Gibbons P. Inter-examiner and intra-examiner agreement for assessing sacroiliac anatomy using palpation and observation: pilot study. *Manual Therapy*. 2000;5(1):13-20
12. Freburger JK, Riddle DL. Measurement of sacroiliac dysfunction: a multicenter intertester reliability study. *Physical Therapy*. 1999;79(12):1134-41
13. Maigne JY, Aivaliklis A, Pfefer F. Results of sacroiliac joint double block and value of sacroiliac pain provocation tests in 54 patients with low back pain. *Spine*. 1996;21(16):1889-92
14. Slipman CW, Sterenfild EB, Chou LH, Herzog R, Vresiliovic E. The predictive value of provocation sacroiliac joint stress maneuvers in the diagnosis of sacroiliac joint syndrome. *Arch Phys Med Rehab*. 1998;79:288-292
15. Broadhurst NA, Bond MJ. Pain provocation tests for the assessment of sacroiliac joint dysfunction. *Journal of Spinal Disorders*. 1998;11(4):341-5
16. Toussaint R, Gawlik CS, Rehder U, Ruther W. Sacroiliac dysfunction in construction workers. *Journal of Manipulative and Physiological Therapeutics*. 1999;22(3):134-8

17. Cibulka MT, Koldehoff R. Clinical usefulness of a cluster of sacroiliac joint tests in patients with and without low back pain. *Journal of Orthopaedic & Sports Physical Therapy*. 1999;29(2):83-92
18. Mitchell FL Jr, *The muscle energy manual, Vol 1*. 1995, MET Press
19. Tanner J. Letter to the editor. *Spine*. 1997;22(14):1673
20. Fryette HH. *Principles of Osteopathic Technic*. 1954, American Academy of Osteopathy
21. Bourdillon JF, Day EA, Bookhout MR. *Spinal Manipulation*, 5th Ed. Butterworth -Heinemann; 1992
22. DiGiovanna EL, Schiowitz S. *An Osteopathic Approach to Diagnosis & Treatment*, 2<sup>nd</sup> Ed. 1997, Lippincott
23. Goodridge JP, Kuchera WA, *Muscle Energy Treatment Techniques for Specific Areas*. In: Ward RC (ed). *Foundations for Osteopathic Medicine*. Baltimore: Williams & Wilkins; 1997:697-761
24. Greenman PE. *Principles of Manual Medicine*, 2nd Edition. 1997, William & Wilkins
25. Harrison DE, Harrison DD, Troyanovich SJ. Three-dimensional spinal coupling mechanics: Part I. A review of the literature. *Journal of Manipulative and Physiological Therapeutics*. 1998;21(2):101-13.
26. Gibbons P, Tehan P. Muscle energy concepts and coupled motion of the spine. *Manual Therapy*. 1998;3(2):95-101
27. Troyanovich S, Harrison DD, Harrison DE. Motion palpation: its time to accept the evidence (Commentary). *Journal of Manipulative and Physiological Therapeutics*. 1998;21;8:568-571
28. Jull G, Bogduk N, Marsland A. The accuracy of manual diagnosis for cervical zygapophysial joint pain syndromes. *Medical Journal of Australia*. 1988;148:233-236
29. Jull G, Zito G, Trott P, Potter H, Shirley D, Richardson C. Inter-examiner reliability to detect painful upper cervical joint dysfunction. *Australian Journal of Physiotherapy*. 1997; 43;2:125-9
30. Phillips DR, Twomey LT. A comparison of manual diagnosis with a diagnosis established by a uni-level lumbar spinal block procedure. *Manual Therapy*. 1996;2;82-87
31. Korr IM. The neural basis of the osteopathic lesion JAOA 1947:191-198. In: *The Collected Papers of Irvin Korr*. 1979, Indiana: American Academy of Osteopathy
32. Kuchera WA, Kuchera ML. *Osteopathic Principles in Practice*, 2nd Ed. 1991; Missouri: KCOM Press
33. Denslow JS, Korr IM, Krems AD. Quantitative studies of chronic facilitation in human motorneuron pools. *American Journal of Physiology*. 1947. In: *The Collected Works of JS Denslow*, 1993 Year Book, Indiana: American Academy of Osteopathy
34. Arena JG, Sherman RA, Bruno GM, Young TR. Electromyographic recordings of 5 types of low back pain subjects and non-pain controls in different positions. *Pain*. 1989;37:57-65
35. Arendt-Nielsen L, Graven-Nielsen T, Sværre H, Svensson P. The influence of low back pain on muscle activity and coordination during gait: a clinical and experimental study. *Pain*. 1995;64:231-240
36. Sihvonen T, Partanen J, Hanninen O, Soimakallio S. Electric behaviour of low back muscles during lumbar pelvic rhythm in low back pain patients and healthy controls. *Arch Phys Med Rehab*. 1991;72:1080-1087

37. Cassisi JE, Robinson ME, O'Conner P, MacMillan M. Trunk strength and lumbar paraspinal muscle activity during isometric exercise in chronic low back pain patients and controls. *Spine*. 1993;18(2):245-251
38. Sihvonen T, Huttunen M, Makkonen M, Airaksinen O. Functional changes in back muscle activity correlate with pain intensity and prediction of low back pain during pregnancy. *Arch Phys Med Rehab*. 1998;79:1210-1212
39. Hides JA, Stokes MJ, Saide M, Jull GA, Cooper DH. Evidence of lumbar multifidus muscle wasting ipsilateral to symptoms in patients with acute/subacute low back pain. *Spine*. 1994;19:165-172
40. Hallgren RC, Greenman PE, Rechten JJ. Atrophy of suboccipital muscles in patients with chronic pain: A pilot study. *Journal of the American Osteopathic Association*. 1994;94(12):1032-38
41. Richardson C, Jull G, Hodges P, Hides J. *Therapeutic Exercise for Spinal Segmental Stabilization in Low Back Pain*. London: Churchill Livingstone, 1999
42. Hubbard DR, Berkoff GM. Myofascial trigger points show spontaneous needle EMG activity. *Spine*. 1993;18:1803-1807
43. Chen JT, Chen SM, Kuan TS, Chung KC, Hong CZ. Phentolamine effect on the spontaneous electrical activity of active loci in a myofascial trigger spot of rabbit skeletal muscle. *Arch Phys Med Rehab*. 1998;79:790-794
44. Wallin D, Ekblom B, Grahn R, Nordenborg T. Improvement in muscle flexibility. A comparison between two techniques. *American Journal of Sports Medicine*. 1985;13(4):263-8
45. Handel M, Horstmann T, Dickhuth HH, Gulch RW. Effects of contract-relax stretching training on muscle performance in athletes. *Eur J App Physiol Occup Physiol*. 1997;76(5):400-8
46. Shlenk R, Adelman K, Rousselle J. The effects of muscle energy technique on cervical range of motion. *Journal of Manual & Manipulative Therapy*. 1994;2(4):149-55
47. Cassidy JD, Lopes AA, Yong-Hing K. The immediate effect of manipulation versus mobilization on pain and range of motion in the cervical spine: A randomized controlled trial. *Journal of Manipulative and Physiological Therapeutics*. 1992;15:570-5
48. Shlenk RJ, MacDiarmid A, Rousselle J. The effects of muscle energy technique on lumbar range of motion. *Journal of Manual & Manipulative Therapy*. 1997;5(4):179-83
49. Condon SM, Hutton RS. Soleus muscle electromyographic activity and ankle dorsiflexion range of movement during four stretching procedures. *Physical Therapy*. 1987;67(1):24-30
50. Magnusson SP, Simonsen EB, Aagaard P, Sorensen H, Kjaer M. A mechanism for altered flexibility in human skeletal muscle. *J Physiol (Lond)*. 1996;497(Pt 1):291-8
51. Bandy WD, Irion JM, Briggler M. The effect of time and frequency of static stretching on flexibility of the hamstring muscles. *Physical Therapy*. 1997;77(10):1090-6
52. Taylor DC, Brooks DE, Ryan JB. Viscoelastic characteristics of muscle: passive stretching versus muscular contractions. *Medicine & Science in Sport & Exercise*. 1997;29(12):1619-24
53. Lederman E. *Fundamentals of Manual Therapy*. London: Churchill Livingstone; 1997

54. Guyton AC. *Textbook of Medical Physiology*, Eighth ed. W.B. Saunders Company;1991:182-3
55. Fryer G. Somatic Dysfunction: updating the concept. *Australian Journal of Osteopathy*. 1999;10;2:14-19
56. Giovanelli B, Thompson E, Elvey R. Measurement of Variations in Lumbar Zygapophyseal Joint Intracapsular Pressure: A Pilot Study. *The Australian Journal of Physiotherapy*. 1985; Vol 31;3:115-121
57. Melzack R, Wall PD. Pain mechanisms: a new theory. *Science*. 1965; 150:971-979
58. Vincenzo B, Collins D, Wright A. The initial effects of a cervical spine manipulative physiotherapy treatment on the pain and dysfunction of lateral epicondylalgia. *Pain*. 1996;68:69-74