Somatic Dysfunction: Updating the Concept

Special Communication

Somatic Dysfunction: updating the concept

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Introduction

Somatic dysfunction has been a concept central to osteopathic philosophy and practice for most of this century[1]. Hypothetical models like the facilitated segment have attempted to explain the clinical findings of somatic dysfunction. It is now time for a major reassessment of these concepts as current evidence forces us to question fundamental principles behind previous explanations, and search for more plausible models.

Originally called the "osteopathic lesion", synonyms of somatic dysfunction include the "osteopathic spinal lesion", "greater osteopathic lesion", vertebral articular strain, segmental somatic dysfunction and segmental dysfunction[1]. Somatic dysfunction can be used broadly to include primarily myofascial and peripheral dysfunction but this paper will focus on "segmental" somatic dysfunction, that which affects a single motion segment of the vertebral column.

> *Somatic dysfunction is impaired or altered function of related components of the somatic (body framework) system: skeletal, arthroidal, and myofascial structures, and related vascular, lymphatic, and neural elements*
>
> *The Educational Council on Osteopathic Principles (1981)*[2]

The hallmarks of diagnostic criteria for segmental somatic dysfunction are asymmetry, range of motion abnormality, and segmental tissue texture change.[3] Some authors include tenderness among these criteria.[4]

The facilitated segment

Denslow and Korr, during the 1940–60s, have provided experimental evidence to lend support to a neurological explanation for somatic dysfunction[5,6,7]

The facilitated segment is a concept that is proposed to explain the behaviour of somatic dysfunction: an injured somatic or visceral structure produces a barrage of discordant afferent impulses into the dorsal horn of the spinal cord which "sensitises" that segment. It is proposed the spinal interneuron thresholds are lowered, allowing an exaggerated response to pathways synapsing at that level: increased pain perception, sympathetic outflow, and segmentally supplied muscle tone.

An increase in gamma motor discharge to muscle spindle intrafusal fibres has been proposed to "turn up the gain" of the spindles which will produce sustained reflex muscle contraction[6] ("neurological" muscle tone). Segmental muscle contraction would produce palpable tissue texture change and restrict segmental joint motion.

The "silent gap" theory[6] had been suggested as another cause of segmental muscle contraction. Trauma or poorly controlled movements could passively shorten a muscle and momentarily produce no feedback (silence) from the spindle receptor. Higher centres, thought to need constant feedback, would react by increasing gamma motor drive to the spindle, which would dictate a shorter resting length of the muscle by sustained motor activity.

The nociceptor replaces the muscle spindle

In 1990 Van Buskirk drew attention to the shortcomings of Korr's neurological model, in particular the muscle spindle's proposed role.[8] Experimental evidence, argued Van Buskirk, indicated that the muscle spindle wasn't capable of producing reflex muscle contraction, and that spindle silence was common.

Van Buskirk argued that nociceptors (free nerve endings, pain receptors) were the only proprioceptors capable of producing reflex muscle contraction and sympathetic discharge. His model is centred on the nociceptor's role in somatic dysfunction, and proposed a cascade of events that produce it.

Noxious stimuli (from viscera or soma) produce reflex axon effects promoting inflammation at all the terminal branches of that axon, which further sensitises other nociceptors. Afferents reaching the dorsal horn produce reflex muscle contraction and sympathetic discharge (producing visceral and immune effects). Perception of pain need not be involved. Over time the muscle becomes fibrotic and if stretched or restrained activates nociceptors once again.[8]

"Neurological" muscle tone

Lederman has questioned the concept of low level sustained neurological "tone" to produce resting muscle tone.[9] He claims that scientific studies demonstrate muscles at rest display no neuromuscular activity and so "resting tone" is purely a result of biomechanical elements such as connective tissue and intramuscular fluid pressure.

This view is clearly at odds with the work of Korr and Denslow. They have demonstrated spontaneous action potentials in resting paraspinal muscles where somatic dysfunctions were palpable.[5, 6,7]

Basmajian supports the notion of resting electrical silence but talks of "muscle reactivity"[10]. A muscle may be neurologically silent at rest but overeact to stimuli. It may be possible that a reactive muscle may contract when being palpated or produce "braking" when joint motion is being tested. While studies using EMG recordings to measure muscle tone in low back pain have produced conflicting results, some studies have demonstrated elevated posture-dependent EMG in such groups.[11]

To the author's knowledge Korr and Denslow's experiments have not been reproduced, and with the weight of evidence apparently supporting the contrary view, we should regard this topic as contentious and unresolved. If we accept that resting muscle tone is neurologically silent, we are forced to make substantial changes to the facilitated segment concept and its role in maintaining muscle shortening. Similarly, any notion of manual therapy altering "gamma gain" must be abandoned.

Joint strain inhibits segmental muscle activity

Jull and Richardson have investigated the stabilising role of transverse abdominus and multifidus muscles on the lumbar spine[12]. They found these muscles have an important role in stabilising and protecting the lumbar spine, and have delayed or absent contraction in people with low back pain. In such people, polysegmental muscles, such as the erector spinae, appear to substitute and increase excitability.

Jull and Richardson have demonstrated that, after lumbar injury, multifidus contraction is inhibited, followed by atrophy and degeneration. Atrophy occurs quickly; in one subject multifidus was demonstrated to decrease in size within twenty-four hours of injury. It appears joint strain produces reflex inhibition of the monoarticular "stabilising" muscles,

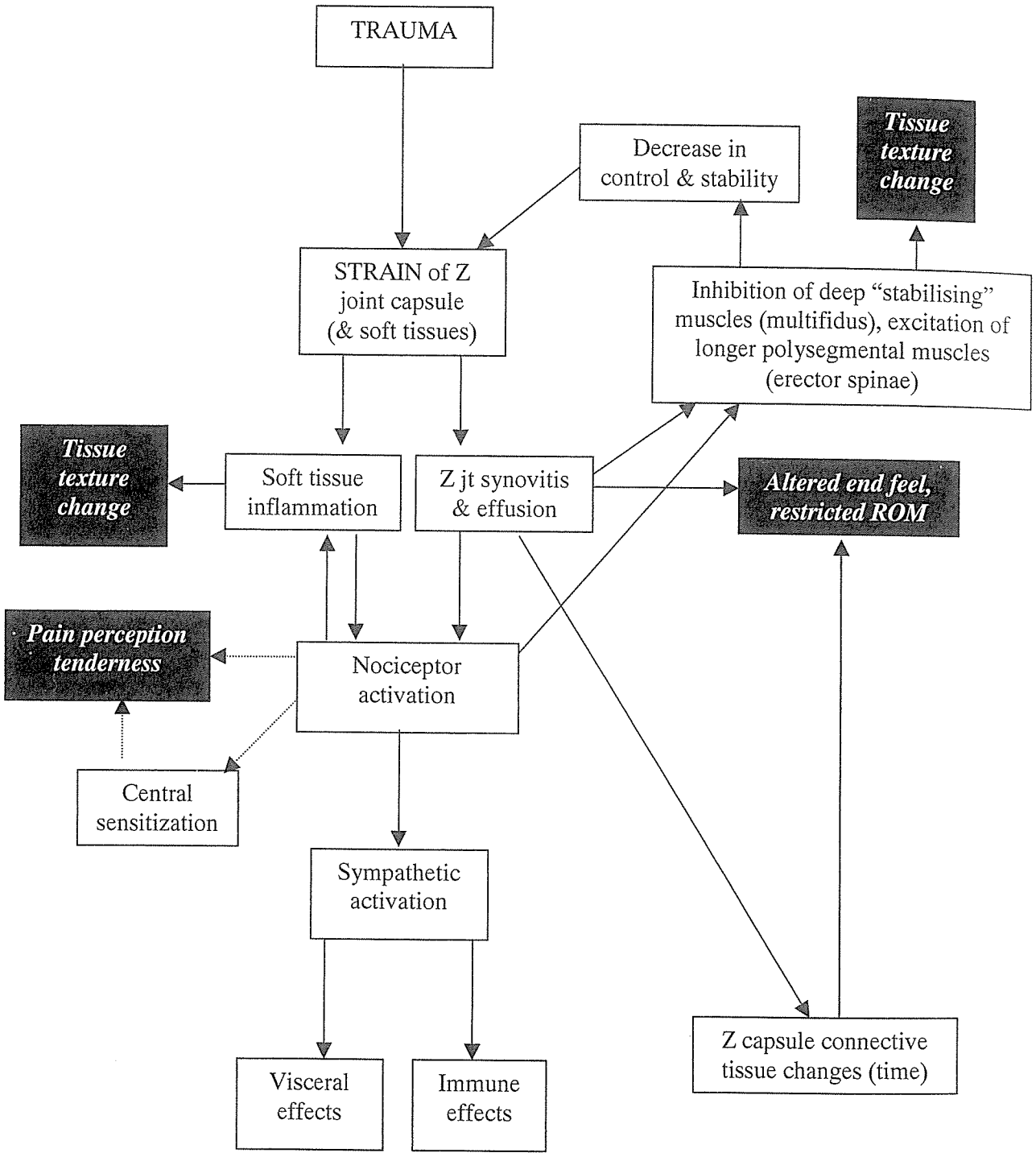

Figure 1: Somatic dysfunction – a model

Trauma produces strain to zygapophysial joint capsule and adjacent soft tissues. Synovial effusion produces changes in segmental range of movement and end feel, and activates nociceptors which produce sympathetic stimulation, inhibition of segmental muscles and possibly pain perception.

producing further joint instability and overwork of the substituting polysegmental muscles[12].

How do we reconcile these findings with the traditional osteopathic belief that, following joint strain, monoarticular muscles contract and restrict joint movement? When we palpate deeply in the paraspinal gutter we often detect "altered", often tender, tissue texture changes. What is it that we are palpating?

Altered segmental motion: the muscle or the joint?

Osteopathic wisdom has usually cited segmental muscle shortness as the cause of restricted joint mobility.[13] This concept must be reviewed if we accept that resting muscle is neurologically silent and deep segmental muscles are actually inhibited by joint dysfunction.

Lewit has questioned the hypertonic muscle theory as he has perceived no change in restricted spinal segmental motion in anaesthetised patients with myorelaxants where protective muscle contraction should be absent.[14] Hartman recalled unpublished studies where experimenters perceived improvement in segmental motion after cavitation in cadaver spines.[15]

What muscles could be capable of restricting vertebral motion at one level? The rotatores are unisegmental, but although they are present in the thoracic spine they are absent or insignificant in lumbar and cervical regions. In the lumbar spine, the only unisegmental muscles are the interspinales and intertransversarii muscles, but these are weak and operate at considerable mechanical disadvantage. It is suggested their main role is proprioceptive as they contain a high density of muscle spindles[16]. Multifidus is the largest and most medial of the lumbar paraspinal muscles but its shortest fibres transverse two vertebral levels. Longer polysegmental muscles such as the erector spinae group could not restrict motion at one segment alone.

If muscle is not a likely candidate, what articular possibilities are there? Bogduk concedes that intra-articular meniscus entrapment is a possibility, at least in acute low back pain.[16] Connective tissue shortening and adhesions have been suggested[17] but these could not account for more acute episodes of somatic dysfunction. Discogenic causes, via reflex muscle contraction, is unlikely as the spinal segments without intervertebral discs (occipitoatlantal and atlantoaxial joints) manifest the same palpable dysfunctions.[18]

The zygapophysial joints can definitely be a source of back pain.[16] Experiments using diagnostic blocks have confirmed that zygapophysial joint pain is common and can occur independently of discogenic or sacroiliac pain. Zygapophysial capsular tears, capsular avulsion, subchondral fractures and intra-articular hemorrhage have been found in both biomechanical and post-mortem studies. Bogduk speculates that these lesions may underlie zygapophysial joint pain, and cannot be detected by radiography, MRI or CT scans.[16]

A hypothetical model

In order to adapt the concept of segmental somatic dysfunction to previously discussed issues, the following hypothesis is proposed (Figure 1).

Strain to zygapophysial joint capsule and ligaments creates inflammation, synovitis, synovial effusion and activates nociceptors. Axon reflexes produce vasodilation and inflammation at the terminal ends of all the axon branches, producing segmental tissue texture change and tenderness (possibly even segmental muscle inflammation and engorgement). Range of movement and end feel is altered due to tissue engorgement and joint effusion.

Nociceptor activation sends action potentials to the dorsal horn and stimulates sympathetic activation, possibly producing visceral and immune sequelae.[8] Segmental "stabilising" muscles, like multifidus, are reflexly inhibited; excitability of polysegmental muscles like the erector spinae increase, making the joint less stable and vulnerable to further strain.
Over time connective tissue changes in the strained capsule occur, producing long term joint range of movement asymmetry. The multifidus atrophies, functional stability and control are impaired and the joint undergoes continuing strain. Nociceptor activation produces further segmental tissue inflammation and sympathetic stimulation and the cycle becomes self-sustaining.

Pain perception need not be involved. However, nociceptive processing in the dorsal horn may become disturbed, producing what has been described as "central sensitisation"[11], leading to hyperalgesia and chronic pain.

Manual techniques: possible therapeutic action

The mode of action of manual techniques in segmental somatic dysfunction is even more speculative. In the acute segmental spinal joint dysfunction manual techniques may act principally on fluid mechanics and motor control.

Active, passive and accessory movements have all been demonstrated to produce pressure fluctuations in zygapophysial joints[19]. Movement of synovial joints has been shown to promote "trans-synovial flow", moving fluids in and out of the joint through the synovial membrane, as well as stimulating lymphatic and blood flow around the joint[9]. Techniques such as passive articulation, muscle energy technique and possibly high velocity

technique may alter the pressure within the inflamed zygapophysial joint, improving motion and end feel.

Active techniques, such as muscle energy, may play a role in improving motor control.[9] Gentle, precise contraction of segmental muscles with slow, precise and painless joint movement may increase recruitment of inhibited multifidus and stimulate joint and muscle proprioceptors to help reprogram the motor control of that segment. Hence, following treatment, passive movements may not provoke polysegmental muscle "braking" and active movements may be performed in a more coordinated, painless and confident fashion.

It is hypothesised that chronic segmental somatic dysfunction involves fibrosis and thickening of the zygapophysial joint capsule in the region of strain and this will produce a range of motion restriction. Articulation, muscle energy and high velocity techniques may all act to stretch the capsule and capsular ligaments to improve movement. Improvement of motor control and fluid drainage may also play a role. These changes should decrease nociceptor activity[8] which in turn may decrease local inflammation and abnormal sympathetic discharge.

High velocity technique may act to free entrapped menisci in the acute "locked" low back[16]. It has been noted that axial rotation in cadaver lumbar spines produced movement of fat pads (intracapsular, extrasynovial) in and out of the joint capsule, presumably to keep the joint volume relatively constant[20]. Might this mechanism be disturbed when capsule inflammation occurs and, if so, might high velocity technique facilitate its movement?

It is possible that any of the above techniques will inhibit incoming messages of pain according to the "gate control" theory of Melzac and Wall[21]. Action potentials from joint mechanoreceptors are conducted by fast large diameter axons which reach the dorsal horn of the spinal cord before the nociceptor potentials, and can "close the gate" on the incoming pain messages. Whether manual techniques produce a significant analgesic effect that lasts longer than the manual event is yet to be determined.

Spinal manipulative therapy has been proposed to reduce motor neuron excitability and produce reflex muscle relaxation. Results of studies testing this proposal have been conflicting[11]. A recent study has demonstrated reflex electromyographic responses in spinal and limb muscles follow high velocity technique but the responses were short lived (100 – 400msec) and no study has examined the effects in symptomatic patients.[22]

Conclusion

The widely held osteopathic view that sustained segmental muscle contraction is responsible for the clinical findings of segmental somatic dysfunction seems untenable. Although not resolved, it appears resting paraspinal muscle has no motor activity and monoarticular spinal muscles are inhibited and atrophy with low back pain.

Discussion of concepts and mechanisms of manual therapy often produce more questions than answers. Are the palpable tissue changes in the paravertebral gutter electrically active? If so, what muscles are they and can they be responsible for segmental joint motion changes? Zygapophysial joint capsule strains and tears appear to be common; can capsule strain produce synovial effusion and disturb joint motion and end feel? If so, can manual therapy influence healing and decrease effusion?

Time and research may answer these questions and endorse or dispose of this model. There is a pressing need to critically examine and research our hypothetical concepts, to abandon those which are obsolete, and to search for more plausible explanations for the therapeutic action of osteopathic practice.

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Assessing competence in the understanding and use of Muscle Energy Techniques (continued from page 10)

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