Intervertebral Somatic Dysfunction: A Discussion of the Manipulable Spinal Lesion

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ABSTRACT

The concept of the manipulable spinal lesion – a musculoskeletal disturbance that can be detected with manual palpation and corrected with manipulation – is examined and evidence for the theories of the dysfunction is discussed. A model for intervertebral dysfunction is presented that describes both mechanical and neurological sequelae to spinal injury, leading to a deficit in regional proprioception, changes in segmental and polysegmental muscle activity and motor control, and predisposing the segment to further strain. The evidence for the proposed model and mechanisms of manual treatment is discussed. Recommendations and directions are outlined for future research.

THE MANIPULABLE LESION

Manual therapy and manipulation of the spine is not new. There is evidence of the use of manual therapy procedures in ancient Thailand, China, Egypt and Greece, as well as its use by bonesetters from the middle ages to relatively recent times.[1] Modern manual therapy professions all have described spinal dysfunctions that are claimed to be detected with manual palpation and corrected with manipulation. These professions have stamped their own "brand names" on these allegedly "manipulable" spinal lesions. For the chiropractic profession, the manipulable spinal lesion is known as the "subluxation", "chiropractic subluxation complex" or "manipulable subluxation".[2, 3] European medical manipulators and physiotherapists have used the term "joint fixation or blockage".[4] In the field of osteopathy, it was originally called the "osteopathic lesion" or "osteopathic spinal lesion",[5] and now may be referred to as the "intervertebral lesion",[6] "intervertebral dysfunction", "segmental dysfunction" or "somatic dysfunction". [1, 6-10] Somatic dysfunction has been defined as:

> "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 [11(p. 1138)]

The term "somatic dysfunction" may be used broadly to describe a disturbance in any region of the body, whereas "segmental dysfunction" or "intervertebral dysfunction" is often used to denote a somatic dysfunction that appears to involve a single spinal level.[8] "Intervertebral dysfunction" will be used in this article because the author believes it is the most descriptive term and likely to be understood across the manual therapy disciplines.

Intervertebral dysfunction is not synonymous with spinal pain. Osteopaths have argued that intervertebral dysfunction may or may not directly cause spinal pain.[8, 9] Although acute segmental dysfunctions may create local pain, some osteopaths claim that chronic dysfunctions may be non-painful, but potentially create a region of altered mobility that can predispose to spinal strain and pain elsewhere.[8, 9] Osteopaths have also claimed that segmental dysfunction may produce distant adverse effects on segmentally related viscera by disturbing the autonomic nervous supply at that level.[7, 9, 12] Intervertebral dysfunction has been claimed to be a precursor of clinical disease and pain syndromes and so it has been argued that the treatment of intervertebral dysfunction in asymptomatic persons may prevent clinical disease.[13] It has been proposed that intervertebral dysfunction does not involve tissue pathology but is a *disturbance of function* of the related components of the musculo-skeletal system.[14] The assumption of absence of any tissue pathology is neither supported or refuted by evidence; that is, there is no current evidence that directly relates to the nature of intervertebral dysfunction. There is growing evidence of functional[15-26] and certain structural changes[27-30] associated with low back pain (LBP), but this may or may not be relevant to the clinical entity that osteopaths claim to detect with palpation.

Diagnostic indicators of intervertebral dysfunction

Authors in the field of osteopathy claim that intervertebral dysfunction can be detected by skilled manual palpation.[1, 10, 31, 32] The diagnostic indicators of segmental dysfunction are said to be segmental asymmetry of bony landmarks, range of motion abnormality (increased, decreased or a change in quality), and tissue texture changes.[1, 7] Some authors also include tenderness.[10, 31, 33, 34] The acronym "TART" is sometimes used as a memory aid for the features of tissue texture, asymmetry, range of motion abnormality and tenderness. Other manipulative professions use similar diagnostic criteria to diagnose manipulable lesions. Physiotherapists have placed emphasis on the altered quality of joint motion, or "end-feel",[35, 36] whereas the chiropractic profession have traditionally emphasised static asymmetry, but have more recently advocated motion palpation, palpation of soft tissues and tenderness.[2, 3, 37]

Reliability of detection of intervertebral dysfunction

Given that the different manual therapy professions – osteopaths, chiropractors, physiotherapists, and medical manipulators – have all independently described a manipulable spinal lesion, it seems likely that such a lesion exists. Several authors, however, have recently lamented the lack of inter-examiner reliability in attempting to detect such dysfunctions, and have raised the possibility that intervertebral dysfunction may be a figment of the collective manual therapy imagination.[38-40] There is presently an urgent need to develop objective means of examining and validating the aetiology of intervertebral dysfunction and to establish reliable methods for its clinical detection.

Of all the diagnostic signs of intervertebral dysfunction, palpation for tenderness appears to be the most reliable.[41-44] The reliability of palpation for static asymmetry has been reported to be poor in the examination of both the spine and pelvis.[40, 45, 46] Similarly, segmental motion testing (without pain provocation) of the spine has consistently demonstrated poor concordance.[47]

The methods that osteopaths most commonly use in clinical practice to detect intervertebral dysfunction are largely unknown because there has been no descriptive survey to establish this. It is possible that manual diagnostic techniques vary from osteopathic practitioner to practitioner, school to school, and from country to country. Most authors of modern osteopathic texts urge the practitioner not to rely on a single palpatory test, but combine multiple findings such as joint range of motion and end-feel, tissue texture, and tenderness to detect intervertebral dysfunction.[1, 7-10, 31, 33, 34] A number of studies have demonstrated that palpation using several criteria can be highly specific and sensitive for diagnosing symptomatic intervertebral joints.[35, 48, 49] Jull *et al*.[35] successfully identified symptomatic cervical zygapophysial joints, confirmed by diagnostic blocks, by using the criteria of abnormal end-feel, abnormal quality of resistance to motion, and reproduction of local or referred pain. Phillips & Twomey[49] found that manual palpation was highly sensitive and specific for detecting a symptomatic lumbar segment when they incorporated a subjective pain response from the patient.

Few studies have examined the reliability of detecting symptomatic joints without tenderness or pain provocation. Jull *et al.*[36] reported good concordance between examiners detecting symptomatic cervical joints (using passive accessory, motion palpation, and assessment of abnormal tissue stiffness associated with muscle reactivity) when no verbal report of pain was allowed. Although they presented no statistical analysis beyond percentage agreement, their results lend support to the view that, while pain provocation improves the reliability of detecting symptomatic joints (a procedure not advocated in any osteopathic text), assessment of joint motion and tissue texture are important clinical signs.

PROPOSED EXPLANATIONS FOR INTERVERTEBRAL DYSFUNCTION

There has been much speculation on the aetiology of the manipulable lesion with various authors implicating the paraspinal muscles,[8] zygapophysial joints,[50] and the intervertebral disc[51, 52]as the underlying cause of motion restriction and tissue texture change. The model that appears to be the most accepted in the field of osteopathy, and is cited in most osteopathic texts,[1, 7, 8, 10, 53] is Korr's neurological concept of the facilitated segment.

Neurological models

Osteopathic researchers J. S. Denslow and Irwin Korr conducted studies from the 1940s to the early 1960s that they claimed provided evidence of segmental spinal cord hyper-activity. They reported evidence of spontaneous electromyographic (EMG) activity and increased sympathetic output at spinal levels associated with clinically detected segmental dysfunctions.[54-59] Korr developed the "facilitated segment" concept, where he proposed that minor trauma to segmentally innervated musculature could produce a discordant barrage of afferent input from muscle spindle proprioceptors into the dorsal horn of the spinal cord and alter the firing thresholds and excitability of the interconnecting neurones. All activity passing through that segment would become exaggerated, producing increased nociception (pain), α and γ motor activity to segmental muscles, and sympathetic output. Korr's neurological model was an attempt to explain the clinical findings of segmental dysfunction: tenderness and pain due to facilitated ascending nociception, joint range of motion restriction due to the resistance of shortened and overactive muscles, and tissue texture changes due to sustained muscle contraction and sympathetic induced circulatory changes.[13]

In 1990, Van Buskirk[60] highlighted deficiencies with Korr's neurological model, in particular the proposed role of the muscle spindle. Experimental evidence, argued Van Buskirk, indicated that the muscle spindle was not capable of producing reflex muscle contraction and spindle silence was common. Van Buskirk claimed that the nociceptors (free nerve endings, pain receptors) were the only proprioceptors capable of producing reflex muscle contraction and sympathetic discharge, and had a central role in segmental dysfunction. He proposed that noxious stimuli (from viscera or somatic tissues) would produce axon reflex changes that promote inflammation at all the terminal branches of

that axon, further sensitising other nociceptors. Afferents reaching the dorsal horn would produce reflex muscle contraction and sympathetic discharge (producing visceral and immune effects). Over time the muscle would become fibrotic and if stretched or re-strained nociceptors would be activated once again.[60]

Both these models attribute the restricted range of segmental motion and palpable tissue changes principally as a result of sustained paraspinal muscle contraction. Although the concepts of "muscle spasm" and "pain-spasm-pain cycle" are popular explanations for hard and tender muscles, the evidence is lacking.[61] Lederman[62] has recently questioned the concept of "neurological muscle tone" – low level motor activity that contributes to the resting tone of a relaxed muscle – and the popular belief that this tone can be "turned up" to produce muscle spasm. "Neurological" muscle tone appears to be controversial, as some authorities in the field of EMG claim that relaxed muscles have no motor activity,[63] while other authorities (such as Guyten & Hall [64] in their "Textbook of Medical Physiology") still describe low level motor activity contributing to resting muscle tone. The assessment of very low level EMG activity in paraspinal muscles is difficult and complicated by electrical noise generated by the heart, respiratory muscles, and extraneous sources.[65]

The two main problems with the proposal of muscle contraction as a component of intervertebral dysfunction are the lack of evidence of abnormal EMG activity associated with palpatory findings and the large body of evidence that demonstrates decreased EMG activity in deep paraspinal muscles of patients with LBP.[22, 23, 66-72] Denslow and Korr's studies[54-59] are over fifty years old and were, by today's standards, poorly described with insufficient data reported and no statistical analysis.[73] Slosberg[74] suggested that the size of the needles used by Denslow were larger and more disruptive than the fine-wire electrodes used today, and thus reproduction of these studies with modern equipment would help verify their significance.

Denslow appeared to have misgivings about his original research. In 1975, thirty-four years after the publication of his original article that described increased paraspinal EMG activity,[75] Denslow stated that:

> "However, following further experience with these methods and additional studies by other researchers, it was recognised that if sufficient care was used in bolstering the subject with pillows and sandbags, the electromyographic evidence of muscle contraction in some of the abnormal areas disappeared."[75 (p155)]

Denslow conceded that, although there appeared to be muscles in the region of clinically detected osteopathic lesions that displayed increased EMG activity, not all palpable paraspinal tissue texture changes could be explained by muscle contraction. The only recent investigation of EMG activity of abnormal to palpation paraspinal muscles found no significant increased activity.[73] This pilot study should be regarded as inconclusive due to the small sample size, problems with artefact, and apparent increased activity in the only symptomatic subject. The issue of EMG activity in relaxed paraspinal muscles that appear to be abnormal to palpation requires further investigation.

Meniscoid entrapment and extrapment

Three commonly occurring meniscoid-like inclusions found within the zygapophysial joints of the spine have been well described.[76] The largest of these, the fibro-adipose meniscoid, is a fibro-fatty vascular structure up to 5mm long, and projects from the superior and inferior margins of the joint capsule. Various authors have speculated that it may be possible for the meniscus to become entrapped (swollen and inflamed from minor trauma, which may prevent the gliding of the opposing joint surfaces) or extrapped (buckled and caught on the joint margin during full flexion, which may prevent the superior joint surface from gliding downwards and backwards).[10, 34, 76]

Extrapment of meniscoid structures, Bogduk argues, is a plausible cause of the "acute locked back" that responds to manipulation. Bogduk states, however, that this theory is likely to be difficult to prove because meniscoid structures are most probably impossible to detect with diagnostic imaging.[76] Although meniscoid extrapment may explain the acute locked back that prevents the sufferer from standing upright, it does not account for the more commonly diagnosed sub-acute or chronic intervertebral dysfunction.

Zygapophysial joint sprain

Sprains of the zygapophysial joint have been postulated as a cause of spinal pain and intervertebral dysfunction.[76-78] Studies using diagnostic blocks have confirmed that the zygapophysial joint is a common source of low back pain and can produce both local spinal and referred pain, but the nature of the lesion remains elusive.[76] Bogduk reported that zygapophysial joint capsule tears, capsular avulsion, subchondral fractures, and intra-articular haemorrhages have been found in biomechanical and post-mortem studies, and these lesions may underlie zygapophysial joint pain.[76] It has been suggested that minor trauma may cause zygapophysial joint capsule sprains and produce joint effusion.[78] Sprain and effusion may account for the diagnostic signs of segmental dysfunction: pain and deep paraspinal tenderness (capsule inflammation), the restricted joint motion and end-feel (tense joint effusion), and tissue texture changes (spread of inflammation to the peri-articular muscles and tissues).

Rhodes and Bishop,[79] in a review of the use of diagnostic ultrasound of the spine, reported a study whose author claimed to visualise inflammation of the zygapophysial capsular ligaments in a small number of low back pain patients. The quoted study has been criticised for its poorly described methods, insufficient data, lack of illustrated ultrasound images and statistical analysis.[80] Nazarian et al.[81] have published the only quality study investigating the potential of diagnostic ultrasound for detecting signs of cervical and lumbar zygapophysial joint inflammation in symptomatic patients and were unable to demonstrate abnormal echogenicity in or adjacent to these joints.

Peri-articular adhesions

Adhesions within the zygapophysial joint and thickening of the joint capsule has been suggested as a cause of restricted segmental mobility.[2, 77, 78] Although this may not account for dysfunctions of short duration or the associated tissue texture changes, adhesion formation and capsule thickening may be logical consequences of capsule sprain and explain the long-term changes to passive range and quality of joint motion following joint injury.

Intra-discal nuclear displacement

There is no question that intervertebral discs are innervated and are a common source of chronic low back pain.[76] Some authors have attributed the signs of segmental dysfunction to internal disruption of the disc and migration of the nucleus.[2, 51, 52] It is feasible that degenerative changes in the disc may affect the quality of passive motion at that segment, but it is uncertain if changes in the disc would produce segmental tissue texture change, or how manipulation might improve restricted passive motion as claimed by osteopaths. The disc is capable of producing reflex multifidus contraction,[82] and this could be responsible for palpable paraspinal tissue change, although the evidence for abnormal EMG activity associated with palpatory findings is lacking. Nonetheless, the disc has the potential to produce some of the cardinal signs of intervertebral dysfunction.

Other theories

A number of other theories have been advanced to explain intervertebral dysfunction, most with little evidence to support them. The chiropractic profession originally believed that the manipulable "subluxation" was a malposition of bones but modern chiropractic authors have largely moved away from the concept of static malposition and embraced a more complex definition with similarities to somatic dysfunction encompassing motion restriction, vascular and neurological consequences.[2, 3, 83, 84]

Axial rotation of cadaver lumbar spines has been observed to produce movement of joint fat pads (within the joint but external to the synovium) in and out of the joint capsule, presumably to keep the joint volume relatively constant.[85] It has been suggested that sprain and capsule inflammation may narrow the communicating foramen and prevent the fat pad moving in and out of the capsule, creating a change in joint pressure, resistance to motion and altered end-feel.[78] This mechanism, however, would be difficult to investigate and the proposal remains purely speculative.

A PROPRIOCEPTIVE-DEFICIT MODEL FOR INTERVERTEBRAL DYSFUNCTION

This proposed model describes concurrent patho-anatomical and functional changes that may be initiated by minor injury, and produce a deficit in local proprioception, subsequent changes in segmental and polysegmental muscle activity and motor control, and create a cycle which predisposes the segment to ongoing strain (Figure 1). The existence – let alone the nature – of intervertebral dysfunction is largely speculative and the following model, like others before it, is also speculative and in need of further corroborating evidence.

It is likely that intervertebral dysfunction is initiated by minor trauma and tissue damage as this would activate nociceptors, which appear necessary to produce the functional changes. The clinical signs produced by tissue damage would predominate in the acute stage of dysfunction. The resulting neurological disturbance would lead to impaired coordination and stability of the region, making it vulnerable to further injury and recurring acute and sub-acute episodes of pain.

Sprain of the intervertebral joint complex

As previously discussed, zygapophysial joint pain has been demonstrated to be a common source of LBP and zygapophysial joint capsule tears, capsular avulsion, subchondral fractures, and intra-articular haemorrhages have been found in biomechanical and post-mortem studies and are likely to underlie joint pain.[76] Injuries to synovial joints, such as the knee, clearly demonstrate synovial effusion and subsequent pain and limitation of motion. Zygapophysial joint capsule sprain and effusion could potentially cause typical symptoms of the acute intervertebral dysfunction: local pain and tenderness, painful limitation of motion, and altered end-feel.

Zygapophysial joint effusion has not yet been demonstrated as a cause of acute spinal pain. The only well designed study[81] that has attempted to detect signs of zygapophysial joint inflammation failed to locate evidence of inflammation in the cervical and lumbar regions. This study examined patients with neck and LBP and, as the researchers did not report the average duration of symptoms, it was likely that the subjects were suffering from sub-acute or chronic pain. It is quite possible that zygapophysial joint effusion may only be evident in the acute stage of joint injury.

Sprain of the anulus fibrosus, intervertebral ligaments, and muscles would also produce local pain and tenderness. Peripheral sensitisation of the nociceptors could enhance segmental tissue inflammation and potentially produce tissue texture change identifiable with palpation.[60] Increased tissue fluid may limit the capacity of the peri-articular soft tissues to stretch and deform with movement, and so may contribute to altered range of motion and end-feel.

Activation of nociceptive pathways

Sprain and inflammation of the zygapophysial joint capsule, the peri-articular tissues, or the anulus fibrosus of the disc would produce release of inflammatory chemicals and sensitise nociceptors (pain receptors). This peripheral sensitisation would produce local pain and tenderness. For most individuals, the pain and tenderness would progressively diminish as the injured tissues healed. Depending on the intensity of the afferent bombardment and the susceptibility of the individual, neuroplastic changes in the dorsal horn of the spinal cord may eventuate, producing chemical and physical changes in the pain processing regions of the dorsal horn and creating the phenomena known as "central sensitisation". These changes to pain processing may long outlast the original tissue injury, and result in permanent hyperalgesia (increased sensitivity to painful stimuli) and allodynia (pain evoked from normally non-painful stimuli). It is likely that most chronic pain is centrally generated, without any longer a tissue basis, despite the fact that tissues may be tender when palpated.[15, 16]

Activation of the sympathetic nervous system

Osteopaths have claimed that intervertebral dysfunction is capable of producing long-term adverse effects on segmentally innervated viscera, known as somato-visceral reflexes.[7, 9, 12, 86] Some limited experimental evidence supports the concept that noxious somatic afferents can modify visceral activity via segmental sympathetic efferent activity,[87] but this theory has been challenged.[62] Although there is no convincing evidence of segmental sympathetic hyper-activity associated with the manipulable lesion, it is likely that pain and emotional distress can certainly produce general sympathetic arousal that, in the long-term, may have detrimental effects on the patient's health.

Nociceptor activity disturbs proprioception

There is growing evidence that pain interferes with normal joint and muscle proprioception. Recent studies have demonstrated decreased proprioception in association with experimental muscle pain,[88, 89] lumbar muscle fatigue,[90] subjects with LBP,[91] lumbar spinal stenosis,[92] and chronic cervicobrachialgia.[93] Volunteers suffering with pain have demonstrated decreased awareness of direction of lumbar motion and position,[90-92] cutaneous touch perception,[88, 93] and jaw muscle spindle afferent activity.[89] Intervertebral sprains could potentially activate joint and muscle nociceptors and result in diminished proprioception from that segment.

Spinal pain and disturbed proprioception produce a change in CNS strategy and increased "guarding" activity in polysegmental paraspinal muscles

Increased lumbar paraspinal activity has been demonstrated in volunteers with LBP in full flexion (lack of flexion-relaxation),[17-23] relaxed standing,[19, 22, 24] and as a reaction to stress.[25, 26] The increased activity

is not likely due to reflex contraction, but a combination of voluntary 'guarding' behaviour[18, 21] and a complex change in motor control strategy.[23]

Zedka *et al*.[23] demonstrated that decreased flexion-relaxation occurred as a result of experimental paraspinal muscle pain alone and the increased paraspinal muscle activity was virtually unchanged even when subjects were carefully guided to perform movement at pre-experimental pain range and velocity. They concluded that pain from any spinal structure (joint, ligament or muscle) may produce a change in CNS strategy, which resulted in the muscle working in "pain mode" to protect the spine from extreme movement whenever pain was signalled.

Spinal pain and a regional deficit in proprioception may result in the inability to execute coordinated contractions of deep segmental musculature, and the "pain mode" CNS strategy appears to activate the more superficial musculature spanning several spinal segments. This attempt to compensate with increased, non-specific activity may further adversely affect the control and proprioception of that region. In the author's experience, patients often remark that their injured region "feels like it does not belong to them" and often have trouble relaxing and allowing the region to "let go", which may be a result of impaired proprioception and control.

Spinal pain produces inhibition and atrophy of deep segmental muscles

There is a large body of evidence that clearly demonstrates that lumbar paraspinal muscles of subjects with LBP operate sub-maximally. Many studies have demonstrated reduced activity in free dynamic movements,[22, 23, 66, 67] reduced muscle strength,[68-70] and increased muscular fatigability[67, 71, 72] of paraspinal muscles in volunteers with LBP. Although much of this change in muscle activity can be attributed to de-conditioning associated with modified patient activity and behaviour, some studies have reported rapid and selective atrophy of the multifidus that suggests reflex inhibition.

Hides *et al*.[27] used diagnostic ultrasound to study the cross-sectional area of the lumbar multifidus muscles of patients suffering from their first episode of unilateral acute/subacute LBP and found that specific wasting of multifidus was evident at the spinal level and side determined symptomatic by clinical examination. Multifidus atrophy has also been observed in subjects with chronic LBP using MRI[28] and computer tomography.[94] Indahl *et al*.[95] demonstrated reflex inhibition of multifidus activity after injecting saline into a porcine zygapophysial joint, probably caused by reflex inhibition following a stretch of the joint capsule. This process may likely occur following zygapophysial joint sprain and effusion.

Reflex inhibition of paraspinal activity appears to target the deep paraspinal muscles, which are the muscles that contribute most to intervertebral stability.[96-98] The deep paraspinal muscles, together with the transversus abdominus muscle, are believed to be a critical factor in lumbar stability, do not automatically recover once back pain has resolved, and may leave the spine vulnerable to normally

trivial episodes of trauma.[27, 96, 97, 99, 100] These deep paraspinal muscles appear to offer most stability in the spinal mid-range "neutral zone", rather than end range of motion.[96] Lack of active control from the deep stabilising muscles, combined with impaired proprioception and non-specific guarding activity, will therefore increase the likelihood of further spinal strain, possibly in mid-range motion and in the absence of any obvious strain or trauma.

Zygapophysial joint capsule fibrosis and intervertebral disc resorption

Repeated sprain and inflammation of the joint capsule may result in proliferation of fibroblasts and shortening and thickening of the capsule. Intervertebral disc disruption and degradation may result in isolated disc resorption.[76] These long-term changes could potentially produce decreased range of motion, decreased accessory 'joint play' and altered joint 'end-feel'.

THERAPEUTIC MECHANISMS OF MANUAL TREATMENT

The mechanisms responsible for the therapeutic effect of manual techniques are poorly understood and largely speculative. The following proposed mechanisms of action are based on the author's model of intervertebral dysfunction and are consistent with available evidence. It may be possible that manual techniques produce physiological effects – such as hypoalgesia, improvement of motor recruitment, or plastic changes in connective tissue – whether or not an intervertebral dysfunction exists.

Reducing zygapophysial joint effusion and peri-articular oedema

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.[101-103] Active and passive spinal movements have been demonstrated to produce pressure fluctuations in zygapophysial joints,[104] and these fluctuations are likely to increase the rate of trans-synovial flow. If acute and sub-acute intervertebral dysfunctions involve capsule sprain, synovial effusion, and peri-articular oedema, manual techniques such as passive articulation may produce pressure fluctuations and stimulate trans-synovial flow and move the excess fluid across the membrane and out of the joint. Active techniques, such as muscle energy, may help drain fluid from the peri-articular tissues, because muscle contraction and relaxation would assist lymphatic drainage from the peri-articular lymphatics. High velocity low amplitude techniques (HVLA) or indirect techniques would probably have little effect, as a rhythmic movement would likely be needed to move fluid across a membrane and allow filling and re-filling of peri-articular lymphatics.[62]

Stretching zygapophysial joint capsule

It has been proposed that multiple sprains to a intervertebral joint may result in zygapophysial joint capsule fibrosis and shortening.[78] Repetitive, rhythmic stretching (as performed with articulation) is

likely to be the most effective means to elongate the capsule. Quick, short, ballistic stretches, such as HVLA, may be less likely to produce viscoelastic or plastic changes to the connective tissue. There is a rationale, however, for performing HVLA before other techniques, as the cavitation and subsequent gas bubble formation would increase joint volume,[105] allow greater joint surface separation, and greater capsule elongation and stretch during passive articulation.

Manipulation induced hypoalgesia

Manipulative therapy is commonly employed to relieve spinal pain. Researchers have demonstrated reductions in pain using cervical mobilisation,[106-108] cervical HVLA,[109, 110] and thoracic HVLA technique.[111] One study[110] found that HVLA was more effective than muscle energy in reducing pain, but more research is required to confirm this. It has been proposed that manipulation may produce hypoalgesia by activating descending inhibitory pathways from the dorsal periaqueductal gray area (dPAG) of the midbrain,[108, 112] release of endorphins, or inhibition of pain at the spinal cord. Christian *et al.*[113] failed to demonstrate elevated plasma levels of endorphins following manipulation, but the dPAG appears to have a role due to the observation of concurrent sympathetic nervous system activation with manipulation induced hypoalgesia.[108, 112] Experimental evidence supports the possibility of inhibition at the spinal cord according to the Gate-control theory, where mechanoreceptor afferents carried by large diameter axons inhibit nociceptor afferents at the dorsal horn.[114] Any technique that produces movement of the joint and stretch of the capsule will stimulate joint proprioceptors, and potentially may be capable of producing inhibition of pain.

Improvement of proprioception and motor control

Changes in proprioception and motor control may underlie both short and long term benefits for patients receiving manual therapy. Researchers have demonstrated that LBP patients exhibit impaired proprioception[90-92] and altered muscle recruitment,[17-26] and it is hypothesised that this impairment may occur regionally as a result of intervertebral sprain. Manual techniques often involve applying gentle stretch and pressure to muscles, and specific mobilisation of spinal articulations, which will stimulate joint and muscle proprioceptors. It has been proposed that careful, specific, and purposeful joint movement in a relaxed patient will stimulate joint proprioceptors, highlight a different pattern of afferent activity in the proprioceptive-impaired region, and allow the CNS to normalise the proprioceptive and motor coordination from that segment.[78, 115] Similarly, indirect techniques, such as functional and counterstrain, may highlight a different pattern of afferent feedback to alter and improve regional proprioception and motor control.

In the author's experience, it is not unusual for patients to volunteer feelings of being more "balanced", with the painful region "back in place" and "part of the body again", following treatment. Such comments could be attributed to a placebo response, but also suggest an improvement in proprioception and motor control. Although active exercise is considered more effective than passive treatment for

motor learning,[62] minor specific regions of impaired proprioception may respond to specific passive stimulation, and subsequent normal patient activity may be sufficient for improved proprioceptive integration and motor learning.

Few studies have attempted to investigate the effect of manipulation on proprioception and motor control. Cervical HVLA has been reported to improve head re-positioning after active displacement in patients with chronic neck pain,[116] and patients with vertigo and dizziness.[117] Cervical mobilisation has been demonstrated to improve body sway in patients with whiplash trauma,[118] and decrease superficial neck muscle recruitment during staged cervical flexion.[108] HVLA has been claimed to diminish quadriceps muscle inhibition,[119] and mechanical spinal manipulation (activator) has been claimed to increase the mean voluntary contraction of the paraspinal muscles.[120] These studies support the proposition that manual therapy can influence proprioception and motor control, but more research is needed.

DIRECTIONS FOR RESEARCH

Reliable detection of intervertebral dysfunction

The reliability of the clinical detection of intervertebral dysfunction has not been clearly established. Recent studies have failed to show acceptable inter-examiner agreement for detection of static asymmetry of bony landmarks,[40, 45, 46] motion palpation,[47] or which segment to manipulate.[38, 39] The detection of symptomatic segments using combined criteria (including pain provocation) has been found to be sensitive and reliable,[35, 48, 49] but these studies may simply be confirming the reliability of pain provocation rather than the TART criteria. Researchers need to re-examine the diagnostic criteria of the manipulable lesion and find ways to make the detection of dysfunction more reliable. Only after acceptable reliability has been established can the validity of the diagnosis be tested.

Diagnostic imaging of zygapophysial joint effusion

Zygapophysial joint sprain and effusion has been proposed as a potential component in intervertebral dysfunction.[76-78] Diagnostic imaging, such as diagnostic ultrasound, MRI and CT, may be capable of detecting signs of peri-articular inflammation or zygapophysial joint effusion in patients with symptomatic intervertebral dysfunctions, which would support joint sprain and effusion as having a role in spinal pain and the manipulable lesion. One study found no sign of inflammation in the cervical and lumbar regions of patients with neck and LBP,[81] but it could be possible that effusion may be visualised only in acute episodes of sprain.

Determine the nature of altered tissue texture associated with the manipulable lesion

Denslow and Korr's[54-57] observations that "lesioned" segments displayed spontaneous paraspinal muscle activity at rest provided the foundation of much osteopathic theory. As previously discussed, these studies are dated and inadequate and have not been verified by any study since. The nature of relaxed paraspinal muscles Detected as tender and abnormal with palpation needs re-investigation with EMG. Such regions could be examined at rest, during quiet sitting and standing, and during dynamic movements to determine if these muscles are abnormally overactive or under-active relative to normal regions. Diagnostic imaging could be employed to determine if the size or shape of these muscles was different to the surrounding musculature.

Investigate the effect of spinal pain on proprioception and the efficacy of osteopathic treatment to improve it

Further evidence is needed to confirm that patients with spinal pain or clinically detected intervertebral dysfunction have a deficit in proprioception and motor control. Head re-positioning, awareness of body position and motion, and postural balance and sway can be used to confirm proprioceptive deficit of patients and as outcome measures to determine the efficacy of osteopathic treatment.

CONCLUSION

Concepts, theories and supporting evidence for the manipulable spinal lesion have been reviewed. A model for intervertebral dysfunction has been presented that details mechanical and functional changes following minor trauma and it is hoped this will stimulate discussion and research into the nature of intervertebral dysfunction and the mechanisms of manual therapy. Future research should examine methods for reliable detection of intervertebral dysfunction, signs of joint effusion, changes in associated paraspinal muscle size and activity, and the effect of spinal pain on proprioception. Once objective signs of dysfunction are established, researchers can accurately investigate the efficacy of manual therapy.

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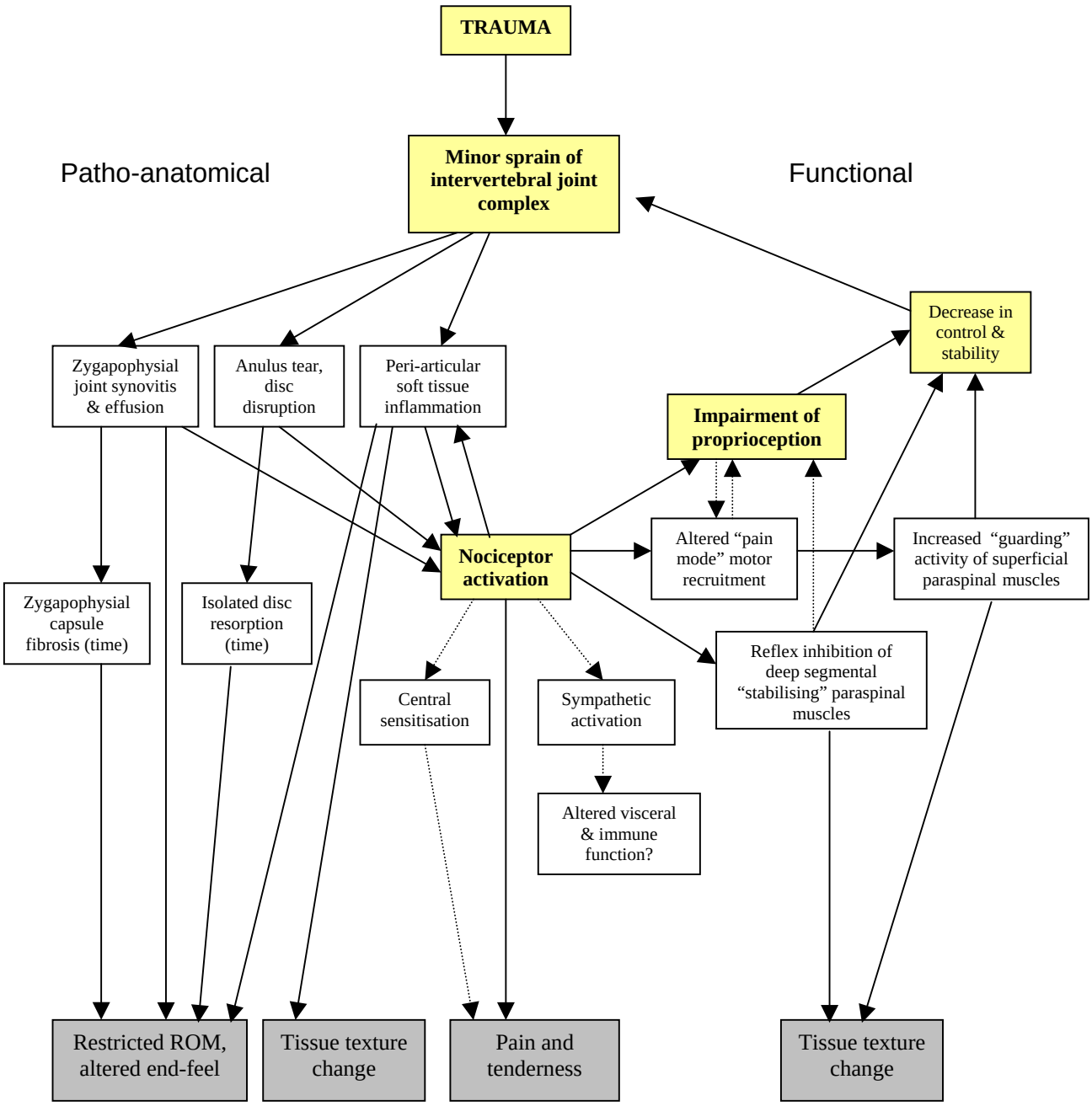

Figure 1: A Proprioceptive-Deficit Model of Intervertebral Dysfunction

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