Review of Literature

Paraspinal Muscles and Intervertebral Dysfunction: Part Two

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Abstract

Background: One of the diagnostic characteristics of the manipulable spinal lesion—a musculoskeletal disturbance that is claimed to be detected with manual palpation and corrected with manipulation—is said to be altered segmental tissue texture. Little evidence for the nature of abnormal paraspinal tissue texture exists, but indirect evidence from experimental studies supports the plausibility of the concept of protective muscle spasm, although investigations of increased paraspinal electromyography (EMG) associated with low back pain suggests complex changes in motor control rather than simple protective reflexes.

Objectives: To review the literature for evidence that may support or refute proposed explanations for clinically observed altered paraspinal tissue texture associated with the manipulable spinal lesion. This review aims to highlight areas that require further research and make recommendations for future studies.

Data Source: MEDLINE and CINAHL databases were searched using various combinations of the keywords *paraspinal, muscle, palpation, EMG, spine, low back pain, pain, myofascial, hardness, manipulation, reliability,* and *somatic dysfunction*, along with searching the bibliographies of selected articles and textbooks.

Data Extraction: All relevant data were used.

Results: Decreased paraspinal muscle activity and strength associated with low back pain is well established, and there is evidence of changes in muscle fiber composition and localized selective multifidus atrophy. Disturbances in microcirculation have been implicated in nonparaspinal muscle pain. The effect of spinal manipulation on paraspinal EMG activity is inconclusive but promising.

Conclusion: Little direct evidence exists to support the existence or nature of paraspinal tissue texture change that is claimed to be detected with palpation. The proposal of segmental reflex paraspinal muscle contraction was not supported, at least in association with low back pain. There appears to be a complex relationship between deep paraspinal muscle inhibition during dynamic activity and nonvoluntary guarding behavior during static activity. The relationship between these findings and palpable tissue change is speculative, but increased activity, decreased activity, or both may be responsible for paraspinal tissues detected as abnormal with palpation. Recommendations are outlined for future research. (J Manipulative Physiol Ther 2004;27:348-57)

Key Indexing Terms: *Spine; Muscle; Palpation; Chiropractic; Osteopathic Medicine*

Introduction

Many authors[1-5] in the field of manual therapy have suggested that changes in the activity of deep paraspinal muscles are associated with intervertebral dysfunction (known by the various manipulative professions as the manipulable lesion, somatic dysfunction, segmental dysfunction, chiropractic subluxation, and joint fixation).[2,3,6-10] It has been proposed that these paraspinal muscles become overactive with abnormally increased and sustained contraction, interfere with normal intervertebral joint motion, and become identifiable with palpation.[11]

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Part 1 of this article reviewed the role of paraspinal muscles in intervertebral dysfunction and considered evidence for the rationale of protective muscle spasm and increased muscle activity associated with low back and muscle pain. MEDLINE and CINAHL databases were searched using various combinations of the keywords *paraspinal, muscle, palpation, EMG, spinal, low back pain, pain, myofascial, hardness, manipulation, reliability,* and *somatic dysfunction*, along with searching the bibliographies of selected articles and textbooks. Although there is little direct evidence to support the belief that sustained muscle contraction is a feature of intervertebral dysfunction, the concept of protective muscle spasm appears plausible.[12-15]

Increased paraspinal muscle activity has been observed in subjects with low back pain (LBP) during full flexion,[16-22] static postures,[18,21,23] and as a reaction to stressful imagery.[24,25] It appears that, although voluntary guarding behavior may be responsible for increased activity in some patients,[16,17] increased muscle activity is due, in part, to a nonvoluntary change in central nervous system (CNS) control, a proposed "pain mode" strategy in response to pain originating from any spinal structure.[22]

These studies support the proposal of abnormally increased paraspinal muscle activity as a consequence of spinal injury. Similar changes to those found in LBP patients may occur in the muscles adjacent to intervertebral injury and produce the commonly reported clinical findings of tenderness and altered tissue texture at a single intervertebral level. What is lacking, however, is direct evidence of increased electromyographic (EMG) activity associated with the detection of tender and abnormal to palpation paraspinal regions.

This article will examine the evidence for decreased paraspinal activity and muscle atrophy associated with LBP, dysfunction in nonparaspinal muscles, the effect of manipulation on paraspinal muscle EMG activity, and the relevance of this evidence to deep paraspinal tissue changes detected with palpation. Further research is necessary to determine the nature of paraspinal tissue texture changes detected with palpation, and recommendations are made for the direction of future research.

Discussion

Evidence of Paraspinal Muscle Spasm Associated with Low Back Pain

Decreased activity of paraspinal muscles associated with LBP. There is a large body of evidence that clearly demonstrates that the lumbar paraspinal muscles of patients with LBP operate submaximally. Many studies have demonstrated reduced activity in free dynamic movements,[21,22,26,27] reduced muscle strength,[28-30] and increased muscular fatigability[27,31,32] of paraspinal muscles in LBP patients.

Sihvonen et al[21] examined 87 subjects with back pain and 25 control subjects while the subjects were performing standing flexion and extension and found that, in addition to reduced flexion-relaxation, the EMG activity of lumbar muscles in subjects with back pain during reextension was significantly lower than that of matched control subjects. Zedka et al[22] found that the lumbar paraspinal muscles operate with decreased activity during reextension as a result of experimentally induced pain. Cassisi et al[30] determined that chronic LBP patients exhibited lower peak torque and lower maximum integrated EMG activity bilaterally during isometric extension. Lee et al[29] examined 98 volunteers and found that muscle strength in the trunk (flexion, extension, and rotation) and the lower extremities (knee flexion and extension) was significantly lower in the LBP group. They argued that these results suggested the trunk weakness was not selective muscle atrophy but generalized weakness due to deconditioning or psychological and motivational factors.

Kaser et al[33] randomized 148 volunteers with chronic LBP into 3 treatment groups (active physiotherapy, muscle reconditioning on exercise equipment, and low-impact aerobics) and examined the effect of treatment on paraspinal fatigability and strength. Kaser et al[33] found that subjects in all treatment groups displayed increased isometric strength, increased activation of the erector spinae muscles during extension, and increased endurance during erector spinae fatiguing tests (Biering-Sorensen test) but no significant change in EMG-determined fatigability. They concluded that the changes in muscle activation and endurance suggested that different motor recruitment patterns were employed and that patients may have become more confident in using the lumbar muscles and did not use many "guarding" mechanisms to prevent their involvement.

The large number of studies that have demonstrated decreased paraspinal muscle activity associated with LBP support a muscle deficiency model, rather than a muscle spasm model. Although there is some evidence to suggest that decreased activity may play a role in the genesis of LBP,[26] many authors attribute these changes to deconditioning secondary to changes in activity, motivation, and pain avoidance behaviours.[29,33] This evidence does not support the common belief in manual therapies that muscles in LBP are identifiable with palpation due to their increased motor tone.

Atrophy of paraspinal muscles associated with LBP. Several studies have demonstrated paraspinal (multifidus) muscle wasting associated with LBP. Hides et al[34] used diagnostic ultrasound to study the cross-sectional area (CSA) of the lumbar multifidus muscles of 26 volunteers suffering from their first episode of unilateral acute/subacute LBP and 51 pain-free control subjects. The symptomatic spinal level was determined on the basis of reproduction of the subject's pain on direct springing in conjunction with abnormal quality or quantity of tissue resistance to segmental motion. Hides et al[34] found marked wasting of multifidus on the symptomatic side, isolated to one vertebral level. They proposed

that the wasting was not likely to be due to disuse atrophy because of the rapidity of onset and localized distribution. Interestingly, they noted that the patients had rounder muscles on the symptomatic side and suggested this may represent muscle spasm and the possibility that the spasm may have decreased the muscle's circulation, influenced its metabolism, and contributed to its atrophy.

Multifidus atrophy has also been observed in patients with chronic LBP using magnetic resonance imaging (MRI)[35] and computed tomography (CT).[36] Danneels et al[36] found the atrophy was selective for multifidus, as neither the psoas nor erector spinae muscle masses were significantly smaller compared with the matched controls. Other researchers, however, have reported no reduction of the paraspinal muscle mass in LBP patients.[37]

Atrophy of the multifidus muscle appears to occur rapidly and specifically to the side and vertebral level of pain and injury. In the lumbar spine, the process appears specific to multifidus, suggesting a mechanism other than disuse atrophy. Reflex inhibition of multifidus activity, either by a segmental reflex from injured spinal structures or a long loop-descending reflex, appears to be the most plausible explanation. Experimental evidence suggests that electrical and mechanical stimulation of deep spinal structures (such as the intervertebral disk [IVD], zygapophyseal joints, and supraspinous ligaments) produce reflex activation—not inhibition—of multifidus. Indahl et al,[38] however, demonstrated reflex inhibition of multifidus activity after injecting saline into a porcine zygapophyseal joint. They concluded that introduction of saline probably produced stretching of the joint capsule and an inhibitory reflex to the muscle. The same process may occur following joint sprain and effusion.

It is important to consider whether the evidence of multifidus wasting has any relevance to paraspinal regions that are detected as tender and abnormal to palpation. The site of specific multifidus atrophy has been reported to be located with manual palpation[39] (using motion palpation, pain reproduction, and palpation of abnormal tissue resistance) and affect one specific vertebral level. This provides support for the principle of palpating altered segmental tone, although the altered tone is usually described as being increased, despite the evidence of atrophy. It may be possible that these deep atrophied muscles feel different to palpation due to altered shape[39] or their smaller size allows the underlying bony architecture (the articular pillars and zygapophyseal joints) to be more obvious to palpation and be mistaken for "hypertonic muscle."

Paraspinal muscle fiber change. Paraspinal muscle composition of LBP subjects has been demonstrated to be different from that of pain-free control subjects. Paraspinal muscles differ from most other skeletal muscles due to their predominance of relatively large type I (slow-twitch, fatigue-resistant) fibers, which befit their function as postural muscles.[40]

Mannion et al[41] examined the lumbar paraspinal muscles of 21 volunteers with LBP and 21 matched control subjects using percutaneous needle biopsies and determined that the LBP subjects had a significantly higher proportion of type IIB (fast-twitch glycolytic) fibers than the slow-twitch type I fibers. Those subjects with LBP who experienced symptoms for the shortest time (less than 1 year) tended to have the greatest quantity of type IIC fibers, which are an intermediate type. When present in high numbers, type IIC fibers are regarded as an indication of ongoing fiber type transformation. Mannion et al[41] also reported that nonspecific pathological changes in fibers (such as moth-eaten or core-targetoid fibers) occurred more frequently in the muscles of the LBP subjects. The duration of symptoms was shown to be significantly associated with a higher proportion of type II fibers, so that the longer the duration of pain, the more glycolytic the paraspinal fiber composition. Mannion et al[41] concluded that these results support the contentious view that fiber type distribution is malleable and can change as a consequence of LBP.

Fiber type transformations in the paraspinal muscles of LBP patients probably have no bearing on the question of the nature of tissue changes detected by palpation. In both studies, Mannion et al[37,41] reported that the size of the fibers remained unchanged, and it is unlikely that a change in fiber composition would produce changes in the muscle texture that could be detected with palpation.

Changes to motor strategy. Hodges et al[42-47] have provided evidence that the deep lumbar multifidus and transversus abdominis muscles appear to be controlled independently from the motor commands of the more superficial trunk muscles. These deep muscles are recruited prior to limb or trunk movement, are not influenced by the direction of movement, maintain contraction throughout the entire motion, and are proposed to have a major role in controlling intersegmental motion and spinal stability. Furthermore, contraction of these deep stabilizing muscles is delayed or absent in volunteers with LBP.[43,44,46,47]

There is evidence to suggest that pain–and possibly fear of pain–delays and inhibits the deep stabilizing musculature but does not affect the control of the more superficial trunk muscles.[46-48] The suppression of this aspect of motor control may, to some extent, explain the contradictory findings of researchers who have examined the EMG activity of paraspinal muscles in LBP subjects. Decreased paraspinal strength, endurance, CSA, and deep muscle atrophy may be results of the inhibition of the deep musculature, along with other factors such as deconditioning and motivational changes. Disturbance to this aspect of motor control may also provide an explanation for increased paraspinal activity observed in static postures and during motion when the muscles should be inactive, described as pain mode behavior by Zedka et al.[22] Because spinal pain appears to suppress the activation of the deep musculature, it is possible that the CNS may attempt to compensate for this deficiency of strength and stability by exaggerating the activity of the more superficial muscles. Such an attempt

to increase spinal stability without recruitment of the deeper muscles may explain the nonvoluntary guarding behavior observed in subjects with LBP.

Evidence of dysfunction in nonparaspinal muscles. Strong parallels exist between the findings of studies that have examined the effect of nonspinal muscle pain on EMG activity and those that have examined LBP and paraspinal EMG activity. Nonspinal muscle pain appears to alter muscle recruitment patterns and produce decreased activation of the involved muscles and guarding strategies that appear similar to those displayed by volunteers with LBP. There is also evidence that muscle pain is associated with decreased microcirculation within the muscle, and this may have relevance to paraspinal muscles. Additionally, there is growing evidence of localized motor endplate dysfunction that appears to produce resting activity of single muscle fibers to produce the clinical features of the myofascial trigger point (MTrP) that are claimed to occur in paraspinal muscles.

Muscle pain and EMG activity. Just as experimentally produced LBP has been demonstrated to adversely affect paraspinal muscle activation and strength,[22] several studies have confirmed that experimental muscle pain produces decreased activity in the affected muscles. Graven-Nielsen et al[49] produced experimental muscle pain in the tibialis anterior and gastrocnemius muscles and found lower maximal voluntary contraction and lower dynamic EMG activity. Muscle antagonists of the painful muscle displayed increased dynamic activity, which appeared to be a strategy to minimize use of the painful muscle. This may be similar to the altered pain mode CNS strategy that has been observed following experimental paraspinal muscle pain.[22] Similarly, other studies have demonstrated decreased activity in painful jaw muscles[50] and trapezius muscles.[51]

Increased EMG activity of muscles at rest during experimental muscle pain has been reported by some researchers,[52,53] whereas others have refuted it.[49,54,55] Stohler et al[52] found both experimental jaw muscle pain and imagined pain produced increased resting EMG activity but dismissed the clinical relevance of this finding due to the very small increases in activity and the possibility of noise from activity in adjacent facial muscles. Glaros et al[53] found patients with temporomandibular joint pain had significantly higher resting EMG activity (frontalis, temporalis, and masseter muscles), but these increases were also very small (an estimated 1% of maximal contraction).

Volunteers with neck and shoulder pain have been observed to sustain trapezius muscle activity when at rest. Vasseljen and Westgaard[56] reported increased resting EMG activity in the trapezius muscles of manual workers with neck and shoulder pain but not in office workers. Elert et al[57] reported that subjects with fibromyalgia displayed significantly higher EMG activity of trapezius and deltoid muscles than control subjects during pauses between shoulder flexion exercises. Similarly, Fredin et al[58] found volunteers with chronic neck pain and a history of whiplash had significantly higher trapezius and infraspinatus muscle EMG activity during pauses between shoulder exercises than control subjects. These findings mirror the pain mode and guarding behavior observed in LBP subjects.

Muscle pain and impaired blood flow. Larsson et al[59,60] have examined the intramuscular blood flow rate within the trapezius muscle using single fiber laser Doppler flowmetry (LDF). They reported that the technique accurately measured the flow of blood within the trapezius muscle and the intramuscular LDF probe caused no disturbance to microcirculation, as rhythmical vasomotion remained unobstructed. Larsson et al[60-62] examined the blood flow rates in the trapezius muscles of subjects with pain and history of soft-tissue injury of the neck,[60] chronic neck pain,[61] and with a diagnosis of chronic trapezius myalgia.[62] They found that significantly lower blood flow was evident in the muscles on the painful side relative to the nonpainful side, compared with control subjects, and especially at low contraction intensities.

The cause of the impaired intramuscular blood flow has not been established. Impaired circulation can be caused by increased intramuscular pressure, but it has been argued that relatively low pressures have been reported for the trapezius muscles at varying angles of arm elevation, and the differences were particularly evident at low contraction intensities.[60,62] Larsson et al[62] suggested that chronic neck pain likely elicits increased transmitter activity of neuropeptides in the upper cervical medulla and brain stem, and this may cause a local lack of vasodilatory neuropeptides secreted axonally. The impaired circulation may contribute to muscle pain by causing an accumulation of metabolites and producing pain and a further disturbance of the microcirculation in a vicious circle.[62] If this process occurs in association with trapezius myalgia, it is also possible that impaired microcirculation within the paraspinal muscles may occur with, and contribute to, spinal pain.

Myofascial trigger points. Travell and Simons[63] have described myofascial trigger points as hyperirritable spots occurring in palpable taut bands of skeletal muscle fibers that are tender on compression and produce characteristic referred pain and autonomic phenomena. The reliability of clinical detection of MTrPs, and therefore the prevalence, has not yet been firmly established, but they are claimed to be a common source of musculoskeletal pain. These tender and palpable bands are believed to result from minor trauma and overload. Travell and Simons[63] claimed that they involve sustained sarcomere shortening and potentially occur in any skeletal muscle, including the paraspinal muscles.

The etiology of MTrPs is still the subject of debate, but recent research implicates a disturbance of the neuromuscular motor endplate. Although some studies[55,64] have indicated there are no EMG abnormalities associated with MTrPs or tender spots, several researchers have used needle EMG to record the presence of spontaneous electrical

activity (SEA) in minute areas (the "nidus") of the MTrP within a resting muscle.[65-67]

Hong and Simons[68] argued that a special technique using high-sensitivity recordings and a very gentle insertion movement of the EMG needle is required to record SEA, as fast movement may miss the small signal. It is likely that those researchers who failed to detect SEA did not use this exacting technique, which requires the researchers to carefully probe and search for the active loci. Most studies have not been blinded; they may have been influenced by examiner bias, because the researchers may have searched for SEA with more zeal in the MTrPs sites than the control sites. Recently, Couppe et al[67] conducted a blinded and controlled study that examined MTrPs and non-MTrPs in the infraspinatus muscles of 20 subjects for spontaneous EMG activity and found that the MTrPs had higher EMG activity than the control points. Although MTrPs were more likely to elicit SEA, not all MTrPs did so, and some non-MTrPs produced spontaneous activity. The SEA appeared to be similar to endplate activity, with features of endplate noise (frequently recurring irregular low amplitude, monophasic, negative potentials, 10 to 50 μV in size and 1 to 3 milliseconds [ms]in duration) and endplate spikes (20 to 300 μV in size and 1 to 3 ms in duration).[67]

Although the evidence supporting SEA and dysfunctional endplates is growing, evidence of histopathological changes in the MTrP currently appears slight. Travell and Simons[69] cited several studies that they claimed support the existence of a "contraction knot," a fusiform swelling of muscle fiber with a region of shortened sarcomeres. Most of these studies are dated, unblinded, and not controlled. Furthermore, Roth et al[70] has reviewed muscle biopsy and muscle fiber hypercontraction and concluded that hypercontracted fibers (those having extreme shortening of the sarcomeres) are likely to be artifacts related to the muscle biopsy procedure.

Travell et al[69] have described MTrPs occurring in both superficial and deep paraspinal muscles, and these dysfunctions could explain the deep, altered tissue texture and tenderness commonly observed by osteopaths when using palpation in the paraspinal region. The concept of deep paraspinal muscle fiber overactivity and contracture is at odds with the observations of many researchers, who have reported decreased paraspinal EMG activity associated with LBP. It may be possible, however, that a muscle can be inhibited by a local segmental reflex or from general disuse, yet a few of its fibers remain taut, contracted, and unusually palpable. No study has attempted to detect paraspinal SEA using the method described by Hong and Simons,[68] so MTrPs remain a possible, but untested, cause of paraspinal tissue change and tenderness.

The effect of manipulation on paraspinal muscle EMG activity. High-velocity low-amplitude (HVLA) spinal manipulation is commonly thought to produce relaxation of paraspinal tissues adjacent to the manipulable lesion. If HVLA manipulation of clinically detected intervertebral dysfunctions can be demonstrated to produce decreased resting EMG activity, this may provide indirect evidence that the paraspinal muscles were abnormally active.

Several studies appear to provide evidence that HVLA manipulation produces a decrease in resting paraspinal EMG activity. Many of these studies share similar shortcomings: lack of controls and blinding; poorly described methods, results, and EMG data; and some unsupported conclusions. Thabe[71] claimed that continuous spontaneous EMG activity of segmental, cervical paraspinal muscles associated with cervical articular dysfunction and the S1 portion of multifidus associated with sacroiliac dysfunction were extinguished following HVLA manipulation. Unfortunately, Thabe[71] only offered a few EMG tracings to support this claim.

Shambaugh[72] examined the effect of a single HVLA manipulation on surface EMG (sEMG) activity of the trapezius, upper thoracic, and lumbar paraspinal muscles. Shambaugh[72] examined an experimental (HVLA) group of 20 subjects, of which half suffered from some form of musculoskeletal pain, and 14 control subjects. They reported a significant reduction in sEMG activity at all locations within the manipulated group but no reduction in the control group. The results were reported in terms of percentage decrease only, but the authors stated that mean lumbar paraspinal EMG recordings were 25 μV pre-HVLA and dropped to 9 μV post-HVLA manipulation.

Kelly and Boone[73] reported a significant decline in sEMG activity of the paraspinal muscles of 30 chiropractic patients over the course of a 4-week treatment regimen using HVLA manipulation. The EMG recordings were obtained from segments the full length of the spine while the patients were seated, which would make some postural activity more likely. As the study used no control group, it is uncertain whether a treatment effect was demonstrated or simply the patients on their first assessment were more anxious and tense, elevating the baseline EMG recordings.

Hayek et al[74] performed a pilot study to examine the effect of HVLA on the deep paraspinal muscles (rotatores) of the upper thoracic region. Recording of needle EMG (nEMG) activity was performed on 3 subjects using intramuscular needle electrodes inserted adjacent to the vertebrae that were determined as fixated using motion palpation (from T1 to T4) and a control electrode inserted 4 segments lower. Recordings were performed with the patients sitting while looking forward and then rotating their head fully. EMG tracings appeared to support the conclusion of Hayek et al[74] that paraspinal activity was decreased following HVLA, both with the patients looking forward and rotating, but no statistical analysis was offered.

Ellestad et al[75] investigated the effect of osteopathic treatment (using a combined approach of HVLA, muscle energy technique, and stretching) to absolute EMG activity levels of the lumbar paraspinal muscles during a sequence

of resting prone, prone with lumbar extension, standing and bending forward and backwards, and returning to lying prone. The authors reported a significant decrease in peak EMG activity between the treated and untreated LBP group, as well as between the treated and untreated control subjects. Unfortunately, the authors reported group total peak activity and did not report if any positions (ie, during flexion-relaxation) resulted in a more significant reduction of EMG activity than other positions.

In an uncontrolled preliminary study, Lehman et al[76] found that painful lumbar segments in patients displayed an exaggerated paraspinal EMG response to mechanical pressure compared with nontender segments, which significantly decreased following HVLA manipulation. The authors suggested that this response may have resulted from a segmental reflex and that the manipulation may have attenuated the reflex. Alternatively, the EMG response in the painful segments may have been due to voluntary[16,17] or nonvoluntary[22] guarding or a psychophysiological response to the pain,[24,25] and manipulation may have diminished the reactive guarding by modulation of the pain[77-79] or because the subjects may have been less fearful when the procedure was applied for the second time. Regardless of the mechanism, this is an area that deserves further investigation.

Other studies have demonstrated short-lived, reflex EMG responses from paraspinal muscles following spinal HVLA manipulation. Herzog et al[80,81] examined the effect of cervical, thoracic, lumbar, and sacroiliac HVLA manipulation on the resting sEMG responses of various trunk and limb muscles. HVLA produced very short-lived (100 to 400 ms) EMG responses in target-specific areas. Dishman and Bulbulian[82] and Dishman et al[83] examined the effect of lumbosacral HVLA manipulation on the tibial nerve H-reflex response in asymptomatic subjects, which provides a neurophysiological index of α-motorneuron pool excitability. They found HVLA (but not mobilization) produced a suppression of the H-reflex, indicating an inhibition of the α-motorneuron pool, that returned to baseline within 30 seconds. In another study, Dishman et al[84] used transcranial magnetic stimulation, reputedly a more accurate indicator of α-motorneuron pool excitability than the H-reflex, and found that lumbar manipulation produced a transient facilitation of the motorneuron pool. The authors suggested that decreases in H-reflex amplitude following HVLA were a result of presynaptic inhibition of peripheral sensory fibers, rather than an attenuation of motorneuron activity.

Both Herzog et al[80] and Dishman and Bulbulian[82] speculated that their transient findings may be related to reflex inhibition and relaxation of paraspinal muscles following manipulation, but these studies with asymptomatic subjects only indicate very short-lived responses and the clinical relevance seems tenuous. The evidence supporting reduced paraspinal EMG activity following manipulative treatment appears promising, but more studies with adequate subject numbers, blinding, and controls are needed before any conclusions can be made.

Directions for Research

Clinical detection of paraspinal regions that are tender and abnormal to palpation. Detection of paraspinal regions that are tender and abnormal to palpation (TAbP) are claimed to be an important diagnostic indicator of segmental dysfunction.[1-5] Although there is some support for reliability of the palpation of spinal tenderness,[85-88] it should be established whether an examiner can detect TAbP paraspinal regions that can be verified independently by a more objective means. Pressure algometry has been demonstrated to be a reliable instrument for the determination of pressure pain thresholds (PPT).[89-93] Pressure algometry could be used to verify that TAbP regions are more pain sensitive than control regions. Although this will not prove the validity of the palpation of tissue dysfunction, it will at least independently demonstrate that clinically detected TAbP paraspinal regions are pain sensitive and therefore different from control sites. This would enable researchers to examine these regions for other objectively measured distinguishing characteristics.

Electromyographic examination of TAbP paraspinal regions. Observations by Denslow et al[94-97] that ''lesioned'' segments displayed paraspinal muscle SEA at rest provided support for the belief that sustained muscle contraction was associated with the manipulable lesion. As previously discussed, these studies are dated and inadequate and have not been verified by any study since. The examination of TAbP and control paraspinal regions with nEMG in subjects who are resting in the prone position will determine if abnormal activity exists in these muscles. Paraspinal muscles could also be screened for MTrPs and examined for SEA using the methods described by Hong and Simons.[68]

Johansson and Sojka[98] proposed that muscle pain might increase the sensitivity of the γ-muscle spindle system, which would produce no abnormal EMG activity at rest but increased reflex muscle activity and stiffness if the muscle were stretched. TAbP paraspinal regions could be examined with nEMG while the muscle is being palpated and while motion palpation of the adjacent segment is being performed. Palpation and motion palpation are procedures commonly used by osteopaths. Abnormal EMG activity during these procedures may explain the abnormal tissue texture and resistance to passive motion that is often claimed to occur at a dysfunctional segment.

In LBP subjects, paraspinal EMG activity has been demonstrated to be abnormally increased in several static postures, particularly standing.[18,21,23] This has been explained as an altered CNS strategy to ''guard'' the painful region.[22] The nEMG activity of TAbP sites can be examined during quiet postural activity (such as standing or sitting) and during active motion (such as neck or trunk rotation) to determine if these regions also exhibit guarding behavior.

This may also confirm the findings of Hayek et al[74] that activity adjacent to dysfunctional segments increases during active rotation.

Muscle size and composition of TAbP paraspinal regions. Deep unisegmental multifidus muscle atrophy has been identified at the same level as clinically detected symptomatic lumbar segments.[39] Altered segmental tissue texture that is claimed to be associated with intervertebral dysfunction may be due to deep paraspinal muscle atrophy and exposure of the underlying zygapophyseal joint and bony architecture. Investigation of TAbP paraspinal regions with diagnostic ultrasound could determine if there are significant differences between the CSA of the muscles in TAbP and control sites.

Evaluation of the paraspinal muscle fiber composition can be performed using percutaneous muscle biopsy. Muscle biopsies at TAbP and control sites should determine if there is a change in the fiber composition or nonspecific pathological changes, as seen in the back muscles of LBP patients,[40,41] or if there is evidence of the MTrP "contraction knot," as claimed by Travell and Simons.[69]

Diagnostic imaging of deep paraspinal structures. Zygapophyseal joint sprain and effusion has been proposed as a cause of symptomatic intervertebral dysfunction.[99-101] Diagnostic ultrasound, MRI, and CT scanning have all been used to determine paraspinal muscle CSA and examine the spine for pathology.[34-36,102] Diagnostic imaging could be used to determine what bony structures underlie the medial paraspinal groove commonly palpated for tenderness and abnormal texture.[2] Furthermore, imaging may be able to detect signs of periarticular inflammation or zygapophyseal joint effusion in patients with symptomatic intervertebral dysfunctions, which would support joint sprain and effusion as an etiology for the manipulable lesion.

Conclusion

Paraspinal tissues that are tender and feel abnormal with palpation are claimed to be a clinical indicator of intervertebral dysfunction, yet the nature and existence of either intervertebral dysfunction or paraspinal tissue change have not been established. Various authors in the field of manual therapy have proposed these tissue changes represent abnormally active musculature, but the evidence for this is lacking.

Experimental research supports the possibility of deep paraspinal muscle contraction as a protective reflex in response to injured joints and ligaments or increased reflex-mediated stiffness as a consequence of muscle inflammation. Increased activity of the paraspinal muscles in volunteers with LBP has been observed under certain conditions, and it appears to be a result of voluntary guarding and nonvoluntary changes in CNS motor control, modified by psychophysiological responses to perceived stress. Many studies have also demonstrated decreased activity, strength, endurance, and atrophy of paraspinal muscles in LBP subjects. There appears to be a complex relationship between deep muscle inhibition during dynamic phases of activity (in the agonist phase, when muscles should be strongly active) and increased pain mode guarding behavior during static postures (when muscles should be relaxed). It is not possible to directly infer that these same changes occur in the paraspinal muscles associated with intervertebral dysfunction, but it is possible these changes may occur at a specific vertebral level following intervertebral sprain. If so, such changes might be detected with palpation.

The challenge ahead for researchers is to establish the reliability of palpation of paraspinal regions reported as tender and detected as abnormal, examine the nature of these regions for reliable changes in their character, and investigate if any of these changes can be influenced by manipulative therapy. This may support the validity of manual diagnosis and treatment. It will also enable clinicians to be more confident concerning the meaning of their physical examination findings and help them formulate appropriate and effective treatment, concerns which are at the heart of the manual therapies.

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