Resting Electromyographic Activity of Deep Thoracic Transversospinalis Muscles Identified as Abnormal With Palpation

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Context: In the 1940s, osteopathic researchers suggested that paraspinal tissue abnormality was associated with spontaneous muscle activity, but few studies have since re-examined these reports.

Objective: To determine whether abnormal motor activity plays a role in deep paraspinal tissues that appear abnormal to palpation.

Methods: Using an observational study design, the PVG of participants with thoracic pain were palpated by two examiners for consensus on the most marked level of tissue abnormality. Dual fine-wire, intramuscular electrodes were inserted into the deep transversospinalis (multifidus, rotatores) muscles at the abnormal level and at two normal sites (above and below the abnormal level). Surface electrodes were placed over the erector spinae muscles adjacent to each intramuscular electrode site. Electromyography signals were recorded during initial prone resting, three maximal voluntary isometric contractions (MVIC), and a second prone resting. The area under the curve for a 2-second period was analyzed for each condition, and values were normalized and reported as a percentage of MVIC. Data were analyzed using a 2-factor repeated-measures analysis of variance.

Results: Twenty-five participants with mean (SD) thoracic pain of 3.3 (1.9) on a 0 to 10 visual analog pain scale completed the study protocol. There were no statistically significant differences in normalized resting activity between the three intramuscular sites ($P=0.25$) or between the three surface sites ($P=0.33$). Substantial variability in normalized resting activity at each of the three intramuscular sites was evident (mean [SD] percent of MVIC: abnormal 7.83 [8.76]; normal 9.47 [8.45], 6.65 [7.39]). No statistically significant differences existed in the intramuscular EMG values between the two resting baseline periods ($P=0.10$).

Conclusion: The lack of statistically significant differences between EMG activity at the abnormal and normal paraspinal sites suggests that factors other than muscle activity are responsible for the apparent abnormality of these tissues to palpation. Investigation of these regions for increased tissue fluid and inflammatory mediators is recommended.

J Am Osteopath Assoc. 2010;110(2):61-68

Researchers in osteopathic medicine and other manual medicine disciplines have claimed that paraspinal tissue texture irregularity is an important clinical sign of functional disturbances to the spinal joints and tissues.1,2 These functional disturbances, termed somatic dysfunction in the osteopathic literature, are identified primarily by the palpation of abnormal tissue texture, asymmetry, range-of-motion disturbance, and tenderness.2,3 Tissues in the paravertebral gutter (PVG) region—the longitudinal groove that lies between the vertebral spinous processes and the bulk of the erector spinae muscle group—are claimed to be of particular relevance to spinal assessment.1-3,6 Irregularity of segmental tissue texture in the PVG may include abnormal hardness, bogginess, or ropiness of the deep paraspinal tissues.1-3 Increased motor activity of the deep paraspinal musculature, particularly the multifidus and rotatores muscles, has been cited as a possible etiologic process for paraspinal tissue irregularity associated with somatic dysfunction.1,2,6

Although the clinical concept of somatic dysfunction has been central to osteopathic manipulative medicine for more than a century, objective evidence for the pathophysiology of the proposed dysfunction and associated tissue texture abnormality is limited. Various models for the etiologic process of somatic dysfunction have attributed tissue texture abnormalities to overactivity of segmental musculature,7-8 tissue inflammation,8,9 segmental muscle atrophy,9 or a combination of all these factors.9

As cited in the osteopathic medical literature,10-12 many researchers13-16 in the 1940s claimed evidence of increased
segmental muscle activity and segmental sympathetic motor output at spinal levels associated with palpable lesions (ie, somatic dysfunction). Denslow and Clough used needle electrodes to examine the electromyographic (EMG) activity at “lesioned” and normal segments, as identified by palpation, of the thoracic paraspinal muscles in 16 healthy participants. They reported that spontaneous electrical activity occurred at lesioned segments but was rare at normal segments. These findings were also reported in participants with postural abnormalities. A few years later, Denslow and Denslow et al reported that when graded pressure was applied to the thoracic and lumbar spine, reflex paraspinal EMG activity occurred at lower pressures in segments palpated as abnormal than in adjacent, normal segments. Although these studies are regarded as landmark research in the osteopathic medical profession, they are more than 50 years old and are, by today’s standards, poorly described with insufficient data and sometimes no statistical analysis.

Few researchers have since investigated paraspinal tissue texture abnormality for empirical characteristics. In 2004, Fryer et al reported that sites abnormal to palpation in the thoracic PVG were substantially more sensitive to pressure than adjacent sites. One year later, the same researchers measured the anterior-posterior cross-sectional dimension (thickness) of the paraspinal muscle bulk directly underlying sites in the thoracic PVG using diagnostic ultrasonography. Normal and abnormal palpated regions had similar mean dimensions, suggesting that factors other than paraspinal muscle thickness were responsible for the apparently abnormal tissue texture.

Given that Denslow and Denslow et al are commonly cited as evidence of the facilitated segment concept of somatic dysfunction, it is surprising that few researchers have attempted to re-examine and expand on this body of work. There is little direct evidence to support the proposition that abnormal muscle contraction or spasm is associated with paraspinal regions identified as abnormal. A large volume of research demonstrates lumbar paraspinal motor changes associated with low back pain (increased, decreased, or delayed activity), but the relevance of these findings to the concept of somatic dysfunction and palpation is tenuous.

In the only recent study comparing the EMG activity of deep paraspinal muscle sites identified by palpation as abnormal or normal, Fryer et al found statistically significant increases in normalized EMG activity at abnormal sites under various resting and active conditions. The study, however, had a number of substantial limitations. It used a small cohort of healthy participants (N=12) who had a history of thoracic discomfort but were not symptomatic at the time of testing. The method of EMG normalization—essential for the comparison of sites within a muscle or between individuals—was calculated using the peak amplitude recorded in any activity; however, the great variation of amplitudes in individuals during different activities indicates that this method should be examined for reliability. More importantly, substantial electromagnetic noise contaminated the EMG recordings and particularly affected the resting conditions, during which activity was low. When resting conditions were excluded from the analysis, a statistically significant difference between the normal and abnormal sites still existed, but these limitations cast doubt on the veracity of the findings.

The present study re-examined the hypothesis of Fryer et al that increased paraspinal EMG activity at rest is associated with abnormal sites as determined by palpation. Sites that appeared abnormal to palpation in the PVG were chosen for the present study because practitioners in the field have claimed tissue abnormality in this region may be indicative of segmental dysfunction. In addition, increased activity of the multifidus and rotatores muscles has been proposed as the cause of this tissue abnormality, and these muscles, as well as the zygaphophysial joint, are located within the thoracic PVG region. Compared to previous studies, the current study examined a larger cohort of symptomatic participants using fine-wire intramuscular EMG procedures and used a reliable method for EMG normalization. The purpose of the present study was to determine whether abnormal motor activity plays a role in deep paraspinal tissues that appear abnormal to palpation.

**Methods**

**Participants**

Students and staff from two university campuses in Kirksville, Missouri, were recruited to participate in the present study through e-mails and posted flyers. Participants were included if they had current or frequent thoracic spinal pain during the preceding 3 weeks, were aged 18 to 50 years, and had an identifiable paraspinal region that appeared abnormal to palpation by the examiners. Participants were excluded if they had obvious spinal deformities or pathologic processes or any medical condition that precluded them from fine-wire EMG testing, such as abnormal blood pressure, current prescription anticoagulant, antiplatelet therapy, blood disorders, needle phobia, postural hypotension, skin conditions (eg, sensitivity to adhesives), or syncopal attacks. Participants were excluded if no focal tissue abnormality was detected with palpation.

All participants read an information sheet about the study and signed a consent form before participation. The study was approved by the A.T. Still University’s Kirksville College of Osteopathic Medicine Institutional Review Board.

Participants reported their present severity of thoracic pain on a visual analog scale of 0 (no pain) to 10 (most pain experienced). They then removed clothing to expose their back (women wore disposable gowns that opened at the back) and lay prone on an osteopathic manipulative treatment table with their face at the midline face hole. Two researchers (G.F. and C.F. or B.R.) experienced in osteopathic manipulative
treatment or osteopathic manipulative therapy both palpated the deep tissues in the thoracic PVG region using deep, short gliding movements of the fingertips. A consensus was reached between both examiners on the site with the most marked tissue texture abnormality (ie, hard, boggy, or ropy deep tissues). Because sites that appear abnormal to palpation tend to be more sensitive to pressure, participants in the current study verbally indicated when a site was tender to pressure, thus reinforcing the examiners’ palpatory findings.

When an abnormal-to-palpation (AbP) site was located and agreed upon, the skin at this site was marked by impression with light pressure from the end of a plastic tube. Sites two spinal segments above and below the AbP site were palpated to ensure they were relatively normal to palpation (NP) and reported by the participants as not sensitive to pressure. If any of these sites were considered abnormal, then a different NP site was chosen instead—generally a spinal segment above or below that site. The NP sites were marked with the plastic tube in the same manner as the AbP site. These sites were used for insertion of electrodes.

**Electromyography**

Disposable paired hook-wire electrodes (44 gauge, insulated nickel alloy wire; VIASYS NeuroCare, Madison, Wisconsin) were used for intramuscular EMG data collection. Electrodes were inserted using 30 mm (27 gauge) and 50 mm (25 gauge) hypodermic needles. At the terminal ends of the wires, 2 mm of insulation were stripped, and 2 mm of one wire and 5 mm of the second wire were extended from the tip of the needle. During preliminary testing, we varied the site of needle insertion from a medial location close to the spinous process to more lateral locations using a more oblique direction of needle insertion to determine the approach that would most consistently locate the deepest muscle layer.

Location of needle placement within the muscle was visualized using diagnostic ultrasonography (iU22; Philips, Amsterdam, The Netherlands). By using a medial insertion location—approximately 2 cm lateral to the midline spinous process—and directing the needle posteriorly and slightly medially, the needle was reliably inserted in the multifidus and rotatores muscles of the deep transversospinalis muscles. These muscles cannot be distinguished by ultrasonography, making it likely that recordings include activity from both muscles.

The skin around the marked regions of each participant was swabbed with alcohol, and the surface electrode sites were abraded and swabbed. Dual fine-wire, intramuscular electrodes were inserted at the marked sites until the needle met the resistance of the lamina. The needle was then withdrawn, leaving the fine-wire electrode in situ. Spring coil connector leads were attached to the free wires, and the leads were taped to the participant’s back, keeping a loop of approximately 5 cm free to allow slack for movement. The skin was then abraded and swabbed with alcohol for attachment of the adhesive dual surface electrodes (Ag/AgCl; Noraxon, Scottsdale, Arizona), which were placed over the erector spinae bulk just laterally to each electrode (approximately 4 cm from the midline). A surface reference electrode was connected to the participant’s acromion process on the same side of the participant’s body as the other electrodes.

In total, EMG activity was collected from each patient at six separate electrode sites:

- NP site above the AbP site (NP1) via intramuscular electrode
- AbP site via intramuscular electrode
- NP site below the AbP site (NP2) via intramuscular electrode
- erector spinae adjacent to NP1 (ES1) via surface electrode
- erector spinae adjacent to NP2 (ES2) via surface electrode
- erector spinae adjacent to NP1 (ES1) via surface electrode

These sites are shown in the Figure.

Following electrode insertion, EMG activity was visually verified on the scope to ensure connectivity. Participants relaxed in the prone position for several minutes to establish resting baseline activity.

Electromyography data were collected during three periods. First, baseline data were obtained as the patient rested in the prone position (“pre-MVIC”).

Next, participants were asked to complete a maximal voluntary isometric contraction (MVIC) task. To complete this...
task, participants remained in the prone position with their arms resting by their sides. They were then asked to lift their head and chest off the table as hard as possible for 3 seconds against the resistance of the examiner, whose hands were on the participant’s upper thoracic region. The resistance was not quantified, but it matched the maximal force of the subject. Participants did this task three times with a 5-second rest between contractions. This task has previously been established as having excellent repeatability (G.F., unpublished data, May 2008).

After the MVIC tasks, participants returned to a prone resting position for a second (“post-MVIC”) baseline. Participants were allowed approximately 5 to 10 seconds rest after the MVIC. Electromyography levels were monitored to ensure that this baseline was stable and at a similar level to the first baseline before recording to ensure that the participant had completely relaxed.

**Data Collection and Statistical Analysis**

Electromyography data were collected using a TeleMyo 2400G2 wireless telemetry EMG system with pre-amplified leads (Noraxon) and was processed using MyoResearch XP Master Edition software (Noraxon). Raw EMG data were pre-amplified, band-pass filtered (10-1000 Hz), rectified, and smoothed with a root mean squared (RMS) 20 millisecond window.

Two-second periods from each of the electrodes at pre-MVIC baseline, during MVIC, and at post-MVIC baseline were processed, and the area under the curve (uV) was calculated for each period. Data from each measurement of MVIC and the two baseline periods were analyzed for reliability using intraclass correlation coefficients (ICC). Baseline EMG scores were normalized to the highest MVIC value obtained from the three MVICs. Normalized baseline scores from the three intramuscular sites were analyzed for differences using a 2-factor repeated-measures analysis of variance (ANOVA) to examine the effect of site and to determine whether differences existed between the two baseline periods. Power analysis indicated that 25 participants would provide 80% power (α=0.05) to detect an anticipated medium to large effect size between abnormal and normal sites. Data were also analyzed using nonparametric Friedman tests.

**Results**

**Participants**

Twenty-seven participants were recruited for the study. Three participants were excluded—1 had no evident focal tissue change or tenderness, 1 was insufficiently symptomatic, and 1 had poor EMG recordings from several abnormal and normal sites (either no signal or contamination by artefact). The remaining 24 participants (mean age [SD], 23.8 [8.2] years; mean [SD] BMI, 24.8 [4.5]; 10 men, 14 women) were included in the study.

Participants had a current mean (SD) thoracic pain level of 3.3 (1.9) based on a 0 (no pain) to 10 (worst pain) visual analog pain scale. Two participants had no thoracic symptoms on the day of testing. Nine participants had thoracic symptoms for less than 3 months, 3 participants had symptoms for longer than 3 months but less than 1 year, and 12 participants had symptoms for longer than 1 year. All but 1 participant were right-hand dominant. The AbP site was commonly located in the mid-thoracic region (median level, 5.5; mode, 5; range, 3-9) and on the right side of the participant (18 right, 6 left). The levels were defined by the proximity to the spinal vertebra, as determined by palpation and counting of spinous processes and taking into account the obliquity of these processes (“rule of threes”).

**Normal vs Abnormal Sites**

Raw EMG values (area under the curve, uV) are presented in Table 1. Normalized baseline values for resting periods 1 and 2 are presented in Table 2. Considerable variation between individuals is evident from the large standard deviations. There were no statistically significant differences between AbP and NP sites for normalized values during either the first or second resting baseline periods for either the intramuscular sites (F(2,14)=1.53, P=.25; F(2,16)=2.84, P=.09) or the surface electrode sites (F(2,17)=1.20, P=.33; F(2,17)=0.79, P=.47). The raw EMG values of the two baseline periods were not statistically significant for either the intramuscular sites (F(1,16)=3.18, P=.10) or the surface sites (F(1,18)=1.53, P=.76). Results from the nonparametric Friedman tests were consistent with the results of the ANOVA.

**Repeatability**

For the two baseline periods for the intramuscular (intraclass correlation coefficient [ICC] range, 0.55-0.94) and surface sites (ICC range, 0.35-0.69), repeatability varied considerably. However, repeatability was excellent for the MVIC tasks for all intramuscular (ICC range, 0.96-0.99) and surface electrode sites (ICC range, 0.91-0.95) (Table 3).

**Comment**

Several individuals1,2,6 have claimed that increased motor activity of the deep paraspinal musculature is present in paraspinal regions that appear abnormal to palpation, but the evidence supporting this claim rests within a body of research that lacks validation by subsequent studies. In contrast to a previous study,22 the current study found no evidence of distinguishing EMG activity in the deep thoracic paraspinal musculature at sites identified as abnormal to palpation under resting conditions compared to sites identified as normal. Given the limitations of the previous study22 and the more robust methodology of the present one, it appears likely that abnormal motor activity in the deep musculature is not a factor in the apparent abnormal tissue texture of these paraspinal regions.

The findings of the present study, which examined mul-
tifidus and rotatores activity associated with locations in the thoracic PVG identified as abnormal, do not preclude the possibility of abnormal activity in other muscles. For example, more lateral erector spinae muscles, but they did not describe their precise needle placement, location, or depth. Although we measured the activity of the erector spinae immediately lateral to the PVG sites, we used surface electrodes, which are more susceptible to crosstalk than the needle electrodes used by Denslow and colleagues.13,14 Thus, while the results of the present study do not concur with these early reports as a result of the different methodologies used, they do not invalidate the earlier studies.

Despite these differences, the present study has a number of methodologic advantages over earlier studies.13,14 We used a greater number of participants (25, as opposed to 17 or 16), all our participants had a history of thoracic symptoms with only two nonsymptomatic participants on the day of testing, and our methods were consistent for each participant. We also used modern EMG equipment with fine-wire electrodes that probably caused less tissue disruption and discomfort than the thicker concentric needle electrodes used in earlier studies.13,14 Compared with the previous study by Fryer et al,22 the present study is also more rigorous given its greater number of participants, established means of EMG normalization, and lack of electromagnetic noise in recordings during rest.25

Denslow and colleagues13,14 indicated the activity in lesioned areas varied in degree during each experiment, from occasional quiet periods to activity of single motor units, with activity fading in and out and sometimes cessation of muscle activity from changes in participant position. The EMG tracings published in these early studies showed EMG activity in the lesioned areas well above baseline levels. However, electrode placement, depth, and proximity to motor units affect the amplitude of an intramuscular EMG signal, a problem solved by EMG normalization.23,24 In the present study, rather than determining the presence of activity by visual inspection of EMG tracings, activity was calculated over a representative 2-second period, normalized against maximal activity, and analyzed with appropriate statistical methods. Results from the ANOVA used on our non-normalized data were not statistically significant, and, because comparison of non-normalized data is not considered valid, this was not reported in the results.

Normalization is more empirical than the visual interpretation of EMG tracings. Furthermore, Denslow and Hassettl4 reported no clear demarcation between a lesioned area and normal area and that if spontaneous activity was seen in a control area close to a lesioned segment, the electrode was moved to a quieter area. This methodology clearly introduced examiner bias in the selection of control sites, a flaw which did not occur in the present study.

Denslow and Hassettl4 also reported that when spontaneous activity was not present in lesioned areas, stimuli (eg, slight movement of electrodes, pin pricks or pin scratches,}

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Electromyographic Activity of Deep Thoracic Transversospinalis Muscles: Raw Electromyography Values* for Baseline and MVIC, mean (SD)</th>
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<tbody>
<tr>
<td>Site</td>
<td>Resting Baseline, Pre-MVIC</td>
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<tr>
<td>---------</td>
<td>-----------------------------</td>
</tr>
<tr>
<td>□ NP1</td>
<td>31.28 (54.07)</td>
</tr>
<tr>
<td>□ AbP</td>
<td>23.88 (21.39)</td>
</tr>
<tr>
<td>□ NP2</td>
<td>27.71 (25.58)</td>
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<tr>
<th>Table 2</th>
<th>Electromyographic Activity of Deep Thoracic Transversospinalis Muscles: Normalized Electromyography Values (Percent of MVIC) for Resting Baseline, mean (SD)</th>
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<tbody>
<tr>
<td>Site</td>
<td>Pre-MVIC</td>
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<tr>
<td>---------</td>
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</tr>
<tr>
<td>□ NP1</td>
<td>11.28 (9.02)</td>
</tr>
<tr>
<td>□ AbP</td>
<td>7.30 (8.00)</td>
</tr>
<tr>
<td>□ NP2</td>
<td>6.54 (6.71)</td>
</tr>
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Abbreviations: AbP, abnormal-to-palpation; ES1, erector spinae muscles adjacent to NP1; ES2, erector spinae muscles adjacent to NP2; EVA, erector spinae muscles adjacent to AbP; MVC, mean voluntary isometric contractions; NP1, normal to palpation, above AbP site; NP2, normal to palpation, below AbP site.

* Electromyography values were calculated as the area under the curve.
cubes of ice) were applied to the skin near electrodes in the lesioned and normal areas. There were no details of how often these stimuli were applied. The present study recorded activity over a much shorter time and may have missed transient episodes of spontaneous activity, but if motor activity has a role in the alteration of tissue texture, then more consistent activity in relatively high normalized resting activity values. These results may involve crosstalk from other muscle groups or reflect a lack of complete relaxation in participants with thoracic pain. Regardless of the cause, regions that appeared abnormal to palpation did not have a greater amount of rest activity.

Caution is required before concluding that abnormal EMG activity is not present in abnormal-to-palpation sites. The regions in the present study required deep palpation, and despite every attempt to direct the needle to the exact region that was palpated, we cannot be certain that the EMG recordings came from the tissues identified with deep palpation. Studies from the 1990s and later have reported spontaneous electrical activity in the loci of myofascial trigger points (palpable bands or nodules in skeletal muscle that refer pain on manual compression), using a special recording and needle insertion technique. Perhaps the palpable abnormalities in the PVG represent trigger points in the transversospinalis musculature. No study has investigated paraspinal regions using the method described by Hong and Simons, so myofascial trigger points remain a possible cause of paraspinal tissue irregularity and tenderness.

Inflammation and increased tissue fluid in deep paraspinal tissues may have a role in producing tenderness and abnormal texture to palpation, and future studies in this area may yield rewarding results. Investigation of tissue fluid and inflammation in these deep locations would be challenging, but modern imaging techniques, such as magnetic resonance imaging, may aid these types of investigations. Exploring these regions using measurement of intramuscular pressure with specialized probes may also be feasible. Shah et al. developed a technique for measuring the local biochemical milieu of skeletal muscle to determine the presence of inflammatory mediators in trapezius muscle trigger points, and this sensitivity to motion artifact than the concentric needle electrodes used by Denslow and colleagues, but despite the improved sensitivity, it does not seem likely that the earlier experiments can be reproduced using modern electrodes.

In the present study, substantial variability in the resting EMG values was seen, as evidenced by large standard deviations in the EMG data and variable reliability of the baseline measures. Individual variability of multifidus activation has been noted in the few studies that have examined the thoracic musculature and may be characteristic of deep muscles that function as stabilizers rather than prime movers. Variable baseline activity at many tested sites resulted in relatively high normalized resting activity values. These results may involve crosstalk from other muscle groups or reflect a lack of complete relaxation in participants with thoracic pain. Regardless of the cause, regions that appeared abnormal to palpation did not have a greater amount of rest activity.

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technique may also be appropriate for investigation of the paraspinous muscles.

Conclusion
No differences in resting EMG activity were found in the deep paraspinous muscles underlying sites in the thoracic PVG that were identified with palpation as either normal or abnormal. The results of this study do not support previous EMG investigations reported in the osteopathic medical literature, but earlier studies used different methodologies and examined different paraspinous muscles. Based on the current results, factors other than muscle activity may be responsible for the apparent abnormality of these deep tissues. Investigation of these regions for increased tissue fluid and inflammatory mediators is recommended.

References

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