Published in final edited form as:

J Ultrasound Med. 2011 October ; 30(10): 1331–1340.

Objective Sonographic Measures for Characterizing Myofascial Trigger Points Associated With Cervical Pain

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Abstract

Objectives—The purpose of this study was to determine whether the physical properties and vascular environment of active myofascial trigger points associated with acute spontaneous cervical pain, asymptomatic latent trigger points, and palpably normal muscle differ in terms of the trigger point area, pulsatility index, and resistivity index, as measured by sonoelastography and Doppler imaging.

Methods—Sonoelastography was performed with an external 92-Hz vibration in the upper trapezius muscles in patients with acute cervical pain and at least 1 palpable trigger point ($n = 44$). The area of reduced vibration amplitude was measured as an estimate of the size of the stiff myofascial trigger points. Patients also underwent triplex Doppler imaging of the same region to analyze blood flow waveforms and calculate the pulsatility index of blood flow in vessels at or near the trigger points.

Results—On sonoelastography, active sites (spontaneously painful with palpable myofascial trigger points) had larger trigger points (mean $\pm$ SD, $0.57 \pm 0.20 \text{ cm}^2$) compared to latent sites (palpable trigger points painful on palpation; $0.36 \pm 0.16 \text{ cm}^2$) and palpably normal sites ($0.17 \pm 0.22 \text{ cm}^2$; $P < .01$). Analysis of receiver operating characteristic curves showed that area measurements could robustly distinguish between active, latent, and normal sites (areas under the curve, 0.9 for active versus latent, 0.8 for active versus normal, and 0.8 for latent versus normal, respectively). Doppler spectral waveform data showed that vessels near active sites had a significantly higher pulsatility index (median, 8.3) compared to normal sites (median, 3.0; $P < .05$).

Conclusions—The results presented in this study show that myofascial trigger points may be classified by area using sonoelastography. Furthermore, monitoring the trigger point area and pulsatility index may be useful in evaluating the natural history of myofascial pain syndrome.

Keywords

color Doppler imaging; elastography; myofascial trigger points; sonography

Myofascial pain syndrome is a substantial health problem in the United States, believed to affect about 23 million Americans.¹ Myofascial pain syndrome is a very common, complex,

yet poorly understood form of neuromuscular dysfunction consisting of motor and sensory abnormalities. It is a major progenitor of nonarticular local musculoskeletal pain and tenderness that affects every age group and is commonly recognized as “muscle knots.”² Myofascial pain syndrome is associated with numerous pain conditions, including radiculopathies, joint dysfunction, disk abnormalities, tendonitis, and many others.³ It is characterized by myofascial trigger points, which are discrete hypersensitive hard palpable nodules located within taut bands of contracted skeletal muscle. Myofascial trigger points can be found by palpation of the soft tissue performed by a trained examiner and are painful on compression. The local twitch response is an important clinical finding that confirms the presence of a trigger point. The local twitch response is a quick, localized contraction of muscle fibers produced by strumming or snapping the taut band in a direction perpendicular to the muscle fibers. Trigger points are classified clinically as active or latent.⁴ An active myofascial trigger point causes spontaneous pain and may often cause general motor dysfunction (stiffness and restricted range of motion). A latent myofascial trigger point often causes motor dysfunction without pain. Pain associated with latent myofascial trigger points requires firm palpation or a mechanical stimulus to elicit.⁵ Otherwise, latent myofascial trigger points have all of the characteristics of active myofascial trigger points, although usually to a lesser degree.^{3,6}

Although the specific pathophysiologic basis of myofascial trigger point development and symptoms is unknown, several promising lines of scientific study (ie, histologic, neurophysiologic, biochemical, sonographic, and somatosensory) have revealed objective abnormalities.^{7–13} Furthermore, our group has found that differences among active, latent, and normal sites may be evaluated objectively using diagnostic sonographic techniques, such as gray scale (2-dimensional) sonography, vibration sonoelastography, and Doppler imaging.¹²

Most of the investigational work on myofascial pain secondary to myofascial trigger points has involved human patients because no successful animal model has been found to elucidate how these nodules arise or how to treat them in a controlled setting. Furthermore, there are very few studies describing objective and clinically applicable methods for identifying and classifying myofascial trigger points. The few that do exist have attempted to externally quantify painful regions using electrodermal properties¹⁴ and superficial soft tissue stiffness measurements.¹⁵ Although both electrical resistance and tissue stiffness significantly changed at areas where myofascial trigger points existed, neither was able to distinguish between tissue properties of active and latent trigger points. Electromyography has also been used to measure end plate noise at myofascial trigger points before and after treatment with an acupuncture needle.¹⁶ Electromyography has shown that end plate noise at trigger point sites decreased as pain decreased after acupuncture treatment. However, only active trigger points had a local twitch response during needling.¹⁶ Human in vivo microdialysis studies of the upper trapezius muscle have found that active sites have a unique biochemical milieu compared to latent sites and palpably normal muscle. Active sites have a more acidic milieu and higher levels of inflammatory mediators, neuropeptides, catecholamines, and cytokines—substances known to be associated with persistent pain states, inflammation, and sensitization.^{9,10}

Other noninvasive methods are currently being explored to visualize and characterize myofascial trigger points via magnetic resonance elastography.¹⁷ Magnetic resonance elastography is attractive because magnetic resonance imaging is the diagnostic standard for musculoskeletal imaging; however, it is an expensive and less accessible method compared to other imaging modalities such as sonography. Sonography is a readily available, portable, and inexpensive imaging modality, suitable for use in a physiatrist’s office to complement physical examination, guide therapeutic interventions, and evaluate treatment outcomes.

Previously, our group showed the feasibility of sonography for visualizing trigger points and the surrounding soft tissue.¹² We were able to visualize trigger points and score them on an ordinal scale using gray scale B-mode images, color variance elastography, and Doppler waveforms of local blood flow from 9 patients. The goal of this study was to expand on these preliminary findings to study a larger set of patients and develop quantitative measures of the soft tissue environment of myofascial trigger points. We hypothesize that the soft tissue environments of active trigger points, latent trigger points, and normal muscle differ as measured by sonoelastography and Doppler imaging. We also wanted to explore the sensitivity of these measures to change after a common treatment, dry-needling therapy, to evaluate the feasibility of this method for monitoring posttreatment clinical outcomes.

Materials and Methods

Study Population and Clinical Examination

This study was conducted at the Rehabilitation Medicine Department of the National Institutes of Health Clinical Research Center. Forty-four patients with acute cervical pain met inclusion criteria, having either an active or a latent myofascial trigger point in one or both upper trapezii, and underwent a thorough musculoskeletal evaluation to rule out potential causes of their symptoms other than myofascial trigger points. Patients were classified as having active trigger points if they had pain consistently over the past 3 months or as having latent trigger points or normal muscle if they had no pain over the past 3 months. Patients with posttraumatic pain were also included in the study unless they met the exclusion criteria listed previously, which included fibromyalgia, atypical facial neuralgia, myopathy, radiculopathy, history of shoulder or spine surgery, and trigger point injections.¹² The Institutional Review Board of the National Institute of Dental and Craniofacial Research approved this study, and each participant provided informed consent to participate in the study.

The patients underwent a physical examination as previously described.¹² Briefly, the presence or absence of myofascial trigger points in the upper trapezius muscle was determined by the criteria of Simon et al¹⁸ according to standard clinical practice. Palpation was in the central region of the upper trapezius muscle within 6 cm of the muscle's midline (approximately midway between the cervical vertebrae and the acromion process. The examination resulted in marking of 4 sites, 2 on each side (ie, the right and left trapezius). Sites were considered normal if no palpable nodule was found. Active sites had at least 1 palpable nodule, and palpation reproduced or exacerbated the patient's spontaneous pain, thereby classifying the site as active. Latent sites had 1 or more palpable nodules; although tender to palpation, this maneuver did not reproduce or exacerbate the patient's spontaneous pain. The sites being analyzed were at least 5 cm apart from one another. Patients were asked to rate the level of pain on each side of their neck using a visual analog scale to ranging from 0 to 10 (0, least painful; 10, most painful). A pressure algometer (Pain Diagnostics and Treatment, Great Neck, NY) was used on each of the 4 sites to determine the pain pressure threshold, defined as the amount of pressure needed to produce pain. Only the examiner knew the clinical status of the patients (ie, whether they had cervical pain) and classifications of the marked sites. The sonographers were blinded to the clinical status when acquiring sonographic data. Approximately 30 minutes elapsed between the algometric procedures and sonography, and all of the test procedures were completed in less than 2 hours.

Imaging

Each participant underwent a sonographic examination using an iU22 clinical ultrasound system (Philips Healthcare, Bothell, WA) with a 12–5-MHz linear array L12-5 transducer as

previously described.¹² Briefly, the 4 marked sites were targeted to determine whether myofascial trigger points could be visualized in the upper trapezius. Typically, trigger points appear as focal hypoechoic (darker) areas with a heterogeneous echo texture.

To determine the area of these stiffer zones of muscle tissue vibration, sonoelastography was performed using an external vibration source of approximately 92 Hz. As described previously,^{12,19} a handheld vibrating massager (Mini Vibrator; North Coast Medical, Inc, Morgan Hill, CA) was applied 2 to 3 cm from each site to be imaged. The color variance mode was used to image wave propagation through the myofascial trigger points of vibrations induced by the massager (Figure 1).

The vasculature around marked sites was assessed with color Doppler imaging, and the flow velocity waveforms were measured with spectral Doppler imaging. Flow waveforms in blood vessels with pulsatile flow (arteries or enlarged arterioles, typically >1 mm in diameter) that were found within 1 to 2 cm of palpable trigger points were analyzed to calculate the resistivity index [RI; (peak systolic velocity – minimum diastolic velocity)/peak systolic velocity] and pulsatility index [PI; (peak systolic velocity – minimum diastolic velocity)/mean velocity]. In normal muscle, the RI is 1, indicating no diastolic flow. Elevated diastolic flow (RI <1) indicates decreased vascular resistance, and negative diastolic flow (RI >1) indicates increased vasculature bed resistance. Both the RI and PI are common measures used to determine the volumetric blood flow in muscle tissue²⁰ and the lower extremities²¹ on Doppler sonography.

Image Analysis

In a previous study, our group devised 2 ordinal scores to describe tissue and blood flow characteristics of the imaged sites.^{12,19} Tissue imaging scores ranged from 0 (normal, uniform echogenicity and stiffness) to 4 (abnormal structure with multiple focal hypoechoic and stiff nodules; Figure 1). The blood flow waveform score (from the Doppler flow waveform) ranged from 0 (normal arterial flow) to 2 (abnormal high-resistance flow with retrograde diastolic flow; Figure 2). In this study, more quantitative measurements were performed from the elastographic and Doppler flow waveform images using ImageJ.^{22,23}

Elastographic color variance images were imported into ImageJ, where they were scaled and cropped for the area of interest. Trigger point areas were then calculated similarly to automated cell-counting methods.²³ Briefly, the image was converted to binary; the dark areas were automatically selected; and the area was calculated on the basis of scaling (Figure 1).

Doppler flow waveform images were analyzed similarly to elastographic images. Blood flow waveforms were imported into ImageJ, scaled, and cropped for the waveform of interest. The peak systolic velocity and minimum diastolic velocity were measured. The time-averaged mean velocity was calculated by integrating the area under the velocity waveform for the entire cardiac cycle and dividing by the duration of the cardiac cycle (Figure 2).

Statistical Analysis

Statistical analysis was done with PASW 18 software (SPSS Inc, Chicago, IL) using 1-way analysis of variance and the Tukey post hoc test for normally distributed data. Normal data are presented as mean $\pm$ SD. Non-normally distributed data were analyzed via Kruskal-Wallis 1-way analysis of variance and the Dunn test for post hoc comparison. Statistical significance was determined at $P < .05$. Receiver operating characteristic (ROC) curves were generated to measure the binary classification strength of the different objective measures in this study.

Results

The age range of the 44 patients was 22 to 57 years. A total of 169 sites were imaged via sonography, of which 84 were located medially in the upper trapezius, and 85 were located laterally. In some patients, not all of the 4 clinically identified sites could be imaged on sonography because of scheduling restrictions. Active and latent trigger points were more often located medially (37 of 49 total sites) than laterally (39 of 52 sites). Only 9 of the 68 normal sites were located medially. Approximately 83% of the active sites had a symmetric active or latent site located on the bilateral trapezius muscle.

Active and latent sites had significantly lower pain pressure thresholds than normal sites ($P < .05$) and active sites had lower pain pressure thresholds than latent sites ($P < .05$; Figure 3). Receiver operating characteristic curves were generated to evaluate the binary classification strength of pain pressure threshold scores to be able to delineate normal sites from active or latent sites and active from latent sites (Figure 4). The ROC curves determined pain pressure threshold scores to be fair classifiers of active versus normal sites (cutoff point, 4.07; area under the curve [AUC], 0.72; $P < .01$) and poor classifiers of latent versus normal sites (cutoff point, 4.07; AUC, 0.64; $P < .05$) but failed to classify active versus latent sites (cutoff point, not applicable; AUC, 0.55).

Trigger point areas assessed by vibration elastography showed trends similar to those of pain pressure threshold scores (Figure 3). Active and latent sites had significantly higher areas compared to normal sites ($P < .01$), and active sites were significantly larger than latent sites ($P < .01$). The ROC curves showed that trigger point area measurements were excellent classifiers of active versus normal sites (cutoff point, 0.35; AUC, 0.9; $P < .01$), fair classifiers of latent versus normal sites (cutoff point, 0.27; AUC, 0.79; $P < .01$), and fair classifiers of active versus latent sites (cutoff point, 0.4; AUC, 0.8; $P < .01$). The pain pressure threshold was not linearly correlated to the trigger point area, even though similar trends were observed across latent and active sites (data not shown).

On color Doppler imaging, prominent blood vessels were observed in the close vicinity of trigger points. In 27 of the 49 active sites (55%), retrograde diastolic flow was observed compared to 21 (40%) and 15 (31%) for latent and normal sites, respectively. The occurrence of retrograde flow was significantly higher in active and latent sites compared to normal tissue ($P < .01$). The PI values were significantly different in active compared to normal sites ($P < .05$; Figure 5). No differences in RI values were detected between normal (Figure 5), latent, and active sites. Using ROC analysis, it was found that the PI was not a good classifier of active, latent, or normal sites because of the large variance.

A 3-dimensional scatterplot of the visual analog scale versus the pain pressure threshold versus the area (Figure 6) showed that the visual analog scale and trigger point area separated latent and active groups well, but the pain pressure threshold did not. This finding was similar to what was shown from the ROC curves. Plotting the PI versus the visual analog scale versus the area further separated active and latent groups while also showing the increased PI variance in active trigger points.

Discussion

This study investigated quantitative methods for characterizing the soft tissue environment of active and latent myofascial trigger points compared to palpably normal muscle using sonographic techniques. We found that vibration elastography was an effective method for measuring the trigger point size and was excellent for distinguishing the site type (normal, latent, or active). Sonographic techniques can play a role in objectively identifying active and latent myofascial trigger points, developing outcome measures after therapeutic

intervention, and better describing the complex environment surrounding myofascial trigger points.

Active and latent trigger points have 3 clinical attributes that separate them from normal tissue and must be investigated to explain the pathogenesis and pathophysiologic mechanisms of myofascial pain syndrome. Additional attributes, which further distinguish active from latent points, may also warrant further investigation. One attribute is the change in muscle mechanical characteristics indicated by a hard palpable nodule within a taut band of contracted muscle. Another attribute is the presence of local or referred tenderness and the presence of spontaneous or induced pain. The pain can be localized to the palpable nodule, referred to a satellite location, or both.⁴ The third attribute is an increased presence of highly resistive vascular beds at or near latent and active trigger points.

As such, it would seem logical that as the trigger point area increased there would have been an associated increase in pain sensitivity (ie, a lower pain pressure threshold score). In our study, we did not find a correlation between trigger point areas and pain pressure threshold scores for either latent or active sites (Figure 3). This finding implies that although active sites have larger trigger point areas and lower pain pressure threshold scores (more tender, ie, pain elicited with less pressure) than latent sites, size and pain pressure threshold scores are not directly linked. As a result, the data suggest that other mechanisms could be contributing independently to the trigger point size and pain sensitivity. In fact, the pain pressure threshold may depend on the presence of sensitizing biochemicals such as neuropeptides and catecholamines and only secondarily on mechanical properties.¹⁰ It is believed that the stiffer hypoechoic nodules are a result of increased muscle fiber contraction and recruitment or local injury that can also cause regions of ischemia.²⁴

The pathogenesis of myofascial trigger points is not completely understood. One hypothesis is that trigger points form as a result of muscle overload. For example, patients commonly report an onset of pain associated with myofascial trigger points after acute, repetitive, prolonged, or chronic muscle overload. Piano students developed significantly decreased pressure thresholds over latent myofascial trigger points after only 20 minutes of continuous piano playing.¹⁷ Computer operators developed myofascial trigger points after as little as 30 minutes of continuous typing.²⁵ These data suggest that even low-level muscle contractions accompanying stereotypical movement patterns can contribute to the development of myofascial trigger points and have been shown to lead to muscle fiber degeneration, an increase in Ca^{2+} release, energy depletion, and the release of various cytokines, which all have been associated with the formation of trigger points.^{26–28} During low-level contractions, the intramuscular pressure increases considerably, especially near the muscle insertions, which may impair the local circulation, cause hypoxia, and eventually lead to trigger point formation.^{24,29–31} Measures that link the change in fiber structure, localized tissue stiffness, and blood flow properties to the biochemical milieu of myofascial trigger points could help elucidate the mechanobiological relationships in normal muscle and muscle affected by trigger points. Other biochemical information (eg, substances associated with muscle metabolism) is still needed to provide an adequate framework in which biochemistry is linked to the mechanical environment of the muscle. Although the cascade of changes in the muscle that lead to myofascial trigger points has not been fully mapped, the biochemical environment of the active trigger point has been found to be more acidic and to have elevated levels of inflammatory mediators, neuropeptides, and proinflammatory cytokines that are typically associated with persistent pain and tenderness (as indicated by the pain pressure threshold scores).^{9,10}

The integrated trigger point hypothesis proposes capillary constriction and increased metabolic demands as factors contributing to the development of myofascial trigger points.

Therefore, ischemia and hypoxia may result at the site of the trigger point, sensitizing peripheral and central nociceptors and causing more pain and tenderness.³¹ Increased hypoxia in muscle tissue is usually followed by increased levels of angiogenic factors and blood vessel formation, which we observed in both active and latent sites. Blood vessel formation or expansion may result from vascular endothelial growth factor, chemokines, and chemotactic agents (tumor necrosis factor α and transforming growth factor β), which can be stimulated by factors other than ischemia.^{32–35}

In this study, results from the Doppler waveforms of blood flow showed significantly different blood flow characteristics in active sites compared to normal tissue (Figure 5). The arteries in the neighborhood of active and latent sites had highly pulsatile blood flow with retrograde diastolic flow more often than normal tissue. Two factors could explain highly pulsatile blood flow. The first is an increase in the compliance and volume of the vascular compartment, and the second is an increase in outflow resistance. The importance of altered retrograde blood in the context of variable pain characteristics (eg, its presence or absence, intensity, location, and referral) and the tenderness associated with active versus latent myofascial trigger points is yet to be fully understood. However, the increased presence of blood vessels could provide some intriguing clues to the pathophysiologic mechanisms underlying the development of myofascial trigger points and the natural history of myofascial pain syndrome. Increased outflow resistance could be due to muscle contracture at the trigger point that compresses the capillary/venous bed, anatomic factors related to the geometry of the apex of the upper trapezius muscle that apply external compressive pressure, local vasoconstriction due to inflammation, or externally applied pressure from the ultrasound transducer during imaging.

Although a significant difference was found between active and normal sites for the PI, the RI values were very similar across all sites (Figure 5). We suspect the large variance in the PI and similar RI values between sites was due to the proximity normal sites had to latent and active trigger points and vice versa. Because all of the patients in this study had at least 1 palpable trigger point, the blood flow characteristics we observed in the upper trapezius do not represent the normal variation. The impaired muscle tissue could result in abnormal blood flow characteristics in normal sites. However, monitoring blood flow characteristics could still prove useful for monitoring areas around trigger points and how they may change over time or after treatment.

Data for a pilot study including 3 active cases and 1 latent case were gathered at different time points (Figure 7) to observe the behavior of trigger points after dry needling treatment. The visual analog scale for 2 of the 3 active cases decreased with time, whereas the latent case remained constant. The pain pressure threshold scores increased for all active cases, whereas the latent group remained constant. The trigger point area and PI decreased for all 3 active cases with time but increased slightly for the latent case. For these few cases treated with dry needling, we did observe fairly consistent changes and improvements regarding the size and blood flow of active sites in the trapezius (Figure 7). The latent site did not show any changes, but the changes in the active sites agree with other work showing decreased end plate noise at active sites.¹⁶ Although there were too few follow-up cases to draw conclusions, the data do show promise that these measures can be used to follow patient responses to treatment.

We used 3-dimensional plots to analyze data to detect interactions that would separate latent and active sites (Figure 6). The data plotted included active site data for patients with active sites (thus excluding all normal and latent site data for that patient) and only latent site data for patients not having any active sites (thus excluding normal site data). This analysis was done for 2 reasons: First, it was suspected that latent sites in patients with active sites might

not be true latent trigger points. In other words, latent sites in patients with active sites could provide confounding information because of the pain elicited by neighboring active sites. Second, visual analog scale data were reported for the left and right sides, not by site. Thus, the visual analog scale score would be attributed to the most symptomatic site (ie, active over latent or latent over normal). The 3-dimensional plots show that the visual analog scale and trigger point area separated latent from active sites most successfully, as confirmed by the ROC curves. It is not surprising that the visual analog scale score would have such a large impact on grouping data because it is one of the main scores clinicians use to screen for myofascial trigger points.

Although most trigger points are located and classified on the basis of the patient's history and a physical examination, results can vary depending on the clinician's training and experience. We sought to develop methods to locate and quantitatively assess myofascial trigger points that can aid in classifying tissue types and monitoring changes over time. The visual analog scale, pain pressure threshold scores, and palpation are the reference standards for detecting and classifying trigger points, but they are still subjective measures.³⁶ Pain pressure threshold scores are more objective and serve as fair measures for distinguishing normal sites from trigger point sites, but pain pressure threshold scores still have the perception of what each patient determines the pain threshold to be.³⁶ Blood flow measures from current symptomatic patients did not perform well as classifiers of the site type because of the large variance. The role of blood flow and the presence of enlarged blood vessels in or around trigger points has not been studied. The trigger point area proved to be a very good classifier of the site type in the upper trapezius and was also quick and simple to implement using a clinical sonographic system (Figure 4).

Although we believe that measuring the trigger point area and PI at symptomatic sites can aid in distinguishing between active, latent, and normal sites, there were some limitations to this study. First, our study did not include a control group of pain-free patients. Instead, pain-free sites were marked at least 5 cm apart from latent and active sites. Site locations were standardized as mentioned in "Materials and Methods" in an attempt to measure the symmetry of site locations. Second, no biochemical analysis was conducted to link biochemical markers to differences in trigger point sizes and blood flow properties. Therefore, universal generalization to myofascial pain syndrome and myofascial trigger points is premature, as is development of a definitive model to explain the etiology of myofascial trigger points. Last, the vibrating massager used in this study was an off-the-shelf handheld device with only a single vibrating frequency. Acquiring trigger point area measurements at different frequencies would confirm that area measurements are frequency independent and furthermore would provide data necessary to measure shear elasticity.

Future work needs to focus on linking the physical properties measured here and the biochemical changes in the muscle tissue. More data on the mechanical properties of trigger points need to be obtained. The heterogeneity of the fiber structure and the compressive or shear moduli of these areas may provide important clues to the pathogenesis or pathophysiologic mechanisms of myofascial trigger points. Last, tracking the changes in mechanical properties and the muscle fiber structure along with the biochemical milieu in trigger points after treatment may provide a better understanding of the physiologic environment in the muscle tissue.

The approach presented here provides a quick and effective method for quantitatively classifying myofascial trigger points in the upper trapezius. Measuring the trigger point area could serve as an acceptable outcome measure for assessing different treatments.

Acknowledgments

This research was supported in part by the Intramural Research Program, National Institutes of Health (NIH), the Clinical Center and Office of the Director, NIH, and grant 1R01-AR057348 from the National Institutes of Arthritis and Musculoskeletal and Skin Diseases, NIH.

Abbreviations

AUC	area under the curve
PI	pulsatility index
RI	resistivity index
ROC	receiver operating characteristic

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Figure 1.

Color Doppler images (top row) of the vibrating trapezius muscle and binary images (bottom row) for trigger point area calculation of stiffer tissue zones. Muscle tissue was scored as follows: 0, uniform echogenicity and stiffness; 1, noisy image, no clear nodule; 2, taut band; 3, focal stiff nodule; and 4, multiple nodules. Arrows denote focal trigger points, and red outlined areas denote measured trigger point areas.

Figure 2.

Color Doppler waveform images (top row) of blood flow through the trapezius muscle and binary images (bottom row) for pulsatility and resistivity index calculations. Waveforms were scored as follows: 1, high-resistance flow with no diastolic flow; 2, elevated diastolic flow; and 3, sustained retrograde flow in diastole.

Figure 3. Plots of pain pressure threshold (PPT) scores and trigger point areas. *Significant difference from normal sites; +significant difference from latent sites ($P < .05$).

Figure 4.

Receiver operating characteristic curves for pain pressure threshold (PPT) scores (top row) and trigger point areas (bottom row) comparing the classification strength between latent and normal, active and normal, and active and latent sites. Red lines indicate receiver operating characteristic curves; black diagonals, random classifiers; and circles, cutoff points for a successful test.

Figure 5. Plots of the pulsatility and resistivity indices for normal, latent, and active sites. *Significant difference ($P < .05$).

Figure 6.

Three-dimensional plots of the visual analog scale (VAS) versus the pain pressure threshold (PPT) versus the trigger point area (left) and the pulsatility index versus the visual analog scale versus the area (right) for patients with latent and active sites. The visual analog scale and trigger point area measures separate latent and active data the most, whereas the pulsatility index shows the largest amount of variance in patients with active sites.

Figure 7.

Follow-up data plots of the visual analog scale (VAS), pain pressure threshold (PPT), trigger point area, and pulsatility index versus time. Data are from 3 patients with active sites and 1 patient with latent sites that underwent dry needling after the initial visit and sonographic examination.