Novel Applications of Ultrasound Technology to Visualize and Characterize Myofascial Trigger Points and Surrounding Soft Tissue

Siddhartha Sikdar, PhD, Jay P. Shah, MD, Tadesse Gebreab, BS, Ru-Huey Yen, BS, Elizabeth Gilliams, BS, Jerome Danoff, PT, PhD, Lynn H. Gerber, MD


Objective: To apply ultrasound (US) imaging techniques to better describe the characteristics of myofascial trigger points (MTrPs) and the immediately adjacent soft tissue.

Design: Four sites in each patient were labeled based on physical examination as active myofascial trigger points (A-MTrPs; spontaneously painful), latent myofascial trigger points (L-MTrPs; nonpainful), or normal myofascial tissue. US examination was performed on each subject by a team blinded to the physical findings. A 12–5MHz US transducer was used. Vibration sonoelastography (VSE) was performed by color Doppler variance imaging while simultaneously inducing vibrations (~92Hz) with a handheld massage vibrator. Each site was assigned a tissue imaging score as follows: 0, uniform echogenicity and stiffness; 1, focal hypoechoic region with stiff nodule; 2, multiple hypoechoic regions with stiff nodules. Blood flow in the neighborhood of MTrPs was assessed using Doppler imaging. Each site was assigned a blood flow waveform score as follows: 0, normal arterial flow in muscle; 1, elevated diastolic flow; 2, high-resistance flow waveform with retrograde diastolic flow.

Setting: Biomedical research center.

Participants: Subjects (N = 9) meeting Travell and Simons’ criteria for MTrPs in a taut band in the upper trapezius.

Interventions: Not applicable.

Main Outcome Measures: MTrPs were evaluated by (1) physical examination, (2) pressure algometry, and (3) three types of US imaging including gray-scale (2-dimensional [2D] US), VSE, and Doppler.

Results: MTrPs appeared as focal, hypoechoic regions on 2D US, indicating local changes in tissue echogenicity, and as focal regions of reduced vibration amplitude on VSE, indicating a localized, stiff nodule. MTrPs were elliptical, with a size of ~16×11cm². There were no significant differences in size between A-MTrPs and L-MTrPs. Sites containing MTrPs were more likely to have a higher tissue imaging score compared with normal myofascial tissue (P < .002). Small arteries (or enlarged arterioles) near A-MTrPs showed retrograde flow in diastole, indicating a highly resistive vascular bed. A-MTrP sites were more likely to have a higher blood flow score compared with L-MTrPs (P < .021).

Conclusions: Preliminary findings show that, under the conditions of this investigation, US imaging techniques can be used to distinguish myofascial tissue containing MTrPs from normal myofascial tissue (lacking trigger points). US enables visualization and some characterization of MTrPs and adjacent soft tissue.

Key Words: Myofascial pain syndromes; Rehabilitation; Sonoelastography; Trigger points, myofascial; Ultrasoundography.

Published by Elsevier Inc on behalf of the American Congress of Rehabilitation Medicine.

CHRONIC PAIN IS A CRITICAL public health problem.¹ A vast number of patients in specialty pain management centers and 95% of people with chronic pain disorders have MPS.² MPS is a common, nonarticular musculoskeletal disorder, characterized by MTrPs—hard, palpable, discrete, localized nodules located within taut bands of skeletal muscle that are painful on compression. MTrPs can be either active or latent.³ An A-MTrP is associated with spontaneous pain in which pain is present without palpation. This spontaneous pain can be at the site of the MTrP or remote from it. However, firm palpation of the A-MTrP increases pain locally and usually reproduces the subject’s remote pain.⁴ An L-MTrP is not associated with spontaneous pain, although pain can often be elicited in an asymptomatic subject by a mechanical stimulus.

List of Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>A-MTrP</td>
<td>active myofascial trigger point</td>
</tr>
<tr>
<td>L-MTrP</td>
<td>latent myofascial trigger point</td>
</tr>
<tr>
<td>MPS</td>
<td>myofascial pain syndrome</td>
</tr>
<tr>
<td>MRE</td>
<td>magnetic resonance elastography</td>
</tr>
<tr>
<td>MTrP</td>
<td>myofascial trigger point</td>
</tr>
<tr>
<td>PO₂</td>
<td>partial pressure of oxygen</td>
</tr>
<tr>
<td>PPT</td>
<td>pain pressure threshold</td>
</tr>
<tr>
<td>RI</td>
<td>resistive index</td>
</tr>
<tr>
<td>3D</td>
<td>3-dimensional</td>
</tr>
<tr>
<td>2D</td>
<td>2-dimensional</td>
</tr>
<tr>
<td>US</td>
<td>ultrasound</td>
</tr>
<tr>
<td>VSE</td>
<td>vibration sonoelastography</td>
</tr>
</tbody>
</table>

From the Department of Electrical and Computer Engineering (Sikdar) and Center for the Study of Chronic Illness and Disability (Gerber), George Mason University, Fairfax, VA; and Rehabilitation Medicine Department, National Institutes of Health, Bethesda, MD (Shah, Gebreab, Yen, Gilliams, Danoff, Gerber).

Supported in part by the Intramural Research Program, National Institutes of Health, Bethesda, MD (Shah, Gebreab, Yen, Gilliams, Danoff, Gerber).

Supported by the Intramural Research Program, National Institutes of Health, Bethesda, MD (Shah, Gebreab, Yen, Gilliams, Danoff, Gerber).

Supported by the Intramural Research Program, National Institutes of Health, Bethesda, MD (Shah, Gebreab, Yen, Gilliams, Danoff, Gerber).

Correspondence to Siddhartha Sikdar, PhD, Department of Electrical and Computer Engineering, George Mason University, 4400 University Dr, MS 1G5, Fairfax, VA 22030, e-mail: ssikdar@gmu.edu.

such as finger pressure over the L-MTrP. In someone with a spontaneous pain complaint, thorough palpation of the myofascial tissue is required to identify and differentiate an A-MTrP from an L-MTrP. Pain elicited by palpation of an L-MTrP in a symptomatic subject is qualitatively different from the subject’s pain complaint.

Despite the high prevalence of MPS, its pathophysiology is unclear. It is unknown whether the nodules are a result of ongoing anatomic and physiologic abnormalities, or whether they occur independently of other abnormalities in surrounding soft tissue. The current diagnostic standard for myofascial pain is based on palpation for the presence of trigger points in a taut band of skeletal muscle and an associated symptom cluster that includes referred pain patterns. Unfortunately, few physicians receive training in the clinical diagnosis of myofascial pain. Moreover, the physical examination has been reported to be unreliable, and until recently, there has been no clearly demonstrable underlying pathology associated with the physical findings of trigger points and taut bands.

Current approaches for pain relief include needling (with or without injection) and massage therapy. The lack of objective clinical outcome measures has been a barrier for critically evaluating the efficacy of these therapeutic methods. All of these factors have led to a lack of consensus on myofascial pain as a clinical entity and have contributed to the uncertainty about the pathogenesis and pathophysiology of trigger points.

Therefore, there is a need to develop objective, repeatable, and reliable diagnostic tests for identifying MTrPs and evaluating treatment outcome measures. Such measures can be used to properly diagnose and understand the natural history of MTrPs and to determine the underlying mechanisms relevant to the development, amplification and resolution of myofascial pain.

Diagnostic US is used extensively for noninvasive real-time imaging of muscle, tendon, fascia, blood vessels, and other soft tissues and is suitable for use in a medical office setting. US has the potential to characterize viscoelastic properties of myofascial tissue, to quantify hemodynamic changes resulting from compression of blood vessels, to provide dynamic measures of tissue performance (e.g., muscle contraction), and to demonstrate structure/function correlations. US is a low-risk method for obtaining descriptive information of tissue (e.g., presence of fat, fiber, fluid) and its mechanical properties.

To our knowledge, Doppler US, which is routinely used clinically for vascular applications, has not been used to study MTrPs. However, VSE is a validated method to image palpably stiff nodules in tissue. When an external vibration is applied, any localized regions of stiffer tissue vibrate with lower amplitude compared with less stiff surrounding tissue. In VSE, the US color variance mode can be used to image the relative distribution of vibration amplitude in the myofascial tissue and identify any localized regions of stiffness. Several studies have demonstrated the feasibility of VSE. Transient VSE has been used to quantify the anisotropic properties of muscle and to measure the shear elastic moduli of relaxed and contracted states of both the quadriceps and biceps brachii muscles. A different imaging technique, magnetic resonance elastography, has also been used for investigating taut bands.

In this study, we investigated the feasibility of US imaging for visualizing trigger points and surrounding soft tissue. We believe that US imaging techniques will be capable of distinguishing MTrPs and adjacent structures from normal myofascial tissue and will provide objective descriptions of the underlying tissue abnormalities associated with MTrPs, which may help decipher the pathophysiology and the pathogenesis of painful MTrPs.

METHODS

Study Population

This descriptive study was carried out at the Rehabilitation Medicine Department of the National Institutes of Health, Clinical Research Center. Subjects with acute cervical pain (<3 mo) were eligible and met inclusion criteria if found to have an A-MTrP in 1 or both upper trapezius. All subjects underwent a thorough musculoskeletal evaluation to rule out potential causes of their symptoms other than MTrPs. Exclusion criteria included muscle pain due to fibromyalgia and atypical facial neuralgia, a history of myopathy, neck and shoulder conditions including cervical radiculopathy and lumbar-sacral myopathy, a history of cervical spine or shoulder surgery, and a history of trigger point injections in the upper trapezius. In addition, subjects with cancer or with infections of the head, eyes, ears, nose, or throat were excluded from the study. Based on their history and physical findings in the upper trapezius muscles, 9 subjects with a diagnosis of myofascial pain were recruited. All but 2 had unilateral pain symptoms. 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.

Clinical Examination

Subjects underwent a physical examination by an experienced physiatrist (J.P.S.), who determined the presence or absence of MTrPs in the upper trapezius muscle according to the standard clinical criteria defined by Travell and Simons. In this study, any local region of myofascial tissue in which nodules were absent to palpation was defined as normal or uninvolved.

In our study design, we sought to identify 2 sites per side on each subject including a variety of A-MTrPs, L-MTrPs, and normal sites. Each subject was to have at least 1 normal site among the 4. 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). Up to 2 nodules were found by the examiner in each upper trapezius and identified as either an A-MTrP or an L-MTrP. If fewer than 2 nodules were identified, palpation continued until the examiner was satisfied that only 1 node or none was present in the muscle. The examiner then marked “dummy” sites in the same general vicinity as the nodules and recorded them as “normal.” This process resulted in 2 marked sites on each upper trapezius (4 total per subject). Pressure algometry was performed on each of the 4 sites to determine the PPT (ie, the amount of pressure that produces the sensation of pain). Only the examiner knew the clinical status and classifications of the marked sites (ie, the sonography team [S.S., T.G.] was blinded to the clinical status, algometry results, and identity of all sites). Approximately 30 minutes elapsed between PPT procedures and sonography.

Gray-Scale Imaging

Each participant underwent a US examination using a Philips iU22® clinical US system with a 12–5 MHz linear array L12-5 transducer targeted at the 4 sites palpated during the clinical examination. An experienced sonographer (with >18y of experience) performed all US examinations. The upper trapezius was visualized in the longitudinal and transverse views with the subject sitting upright in a comfortable position. On 2D gray-scale imaging, MTrPs in the upper trapezius appeared as focal hypoechoic (darker) areas with heteroge-
neous echotexture. Because a single 2D image did not fully capture the 3D area of the trigger point and surrounding soft tissue, 3D imaging was also performed by manually sweeping the transducer over the upper trapezius along the transverse and longitudinal planes. A mechanically scanned 9/10 3MHz 3D transducer (3D9-3v) was used for real-time 3D imaging of trigger points in the upper trapezius of the subjects.

Elastography Imaging
Ultrasound VSE uses an external vibration source less than 1000Hz in conjunction with Doppler techniques to identify localized regions of increased tissue stiffness. This methodology has been described in detail elsewhere.22 In this study, vibrations were induced in the upper trapezius muscle using an external handheld vibrating massager (model NC70209) modified with a flat attachment head (fig 1). This vibration source was placed approximately 2 to 3cm from each of the 4 sites to be imaged. Color variance mode was then used to image the site while the massager induced vibrations of approximately 92Hz in the muscle.

Doppler Imaging
We investigated the vasculature and circulation in and around the marked sites using Doppler US. The peak systolic velocity and the minimum diastolic velocity blood flow velocities were measured in the ascending branch of the transverse cervical artery and any other arteries or enlarged arterioles that were found in the neighborhood of palpable trigger points. The RI is commonly used for vascular diagnosis and is defined as the ratio of the difference in peak systolic and minimum diastolic velocities to the peak systolic velocity. The RI is an indication of the resistance of the end-organ vascular bed. In muscle, normally RI equals 1, indicating no diastolic flow. Elevated diastolic flow (RI<1) indicates decreased vascular bed resistance, while negative diastolic flow (RI>1) indicates increased vascular bed resistance.

Ordinal Imaging Score
Two ordinal scores were devised to describe tissue and blood flow characteristics of the imaged sites. The tissue imaging score, based on gray-scale and elastography imaging, was given a range from 0 (normal, uniform echogenicity and stiffness) to 2 (abnormal structure with multiple focal hypoechoic and stiff nodules). The blood flow waveform score, based on the Doppler flow waveform, was given a range from 0 (normal arterial flow) to 2 (abnormal high-resistance flow with retrograde diastolic flow). Table 1 lists the scoring criteria.

Two investigators (T.G., S.S.) independently quantified the size of the MTrPs from the acquired US images. An ellipse was manually fitted to the most prominent MTrP, and the area was computed using the measurement tool available on the US scanner.

Table 1: Ordinal Imaging Scores

<table>
<thead>
<tr>
<th>Tissue Imaging Score</th>
<th>Blood Flow Waveform Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Score</td>
<td>Criterion</td>
</tr>
<tr>
<td>0</td>
<td>No focal lesion on echo or stiffness image (includes heterogeneity)</td>
</tr>
<tr>
<td>1</td>
<td>Evidence of focal lesion on both echo and stiffness image</td>
</tr>
<tr>
<td>2</td>
<td>Multiple focal lesions or marked heterogeneity on both echo and stiffness image</td>
</tr>
</tbody>
</table>

*RI, the ratio of the difference in peak systolic and minimum diastolic velocities to the peak systolic velocity.
Statistical Analysis

PPTs were compared among sites containing A-MTrPs, L-MTrPs, and normal myofascial tissue using t tests subject to Bonferroni correction. The nonparametric Kruskal-Wallis test was used to compare the tissue imaging score and the blood flow score among sites containing A-MTrPs, L-MTrPs, and normal myofascial tissue. Pearson’s correlation coefficient was used to assess the interrater reliability of the MTrP size measurement. For all tests, a P value less than .05 was considered significant.

RESULTS

The age range of the 9 subjects (2 men, 7 women) was 23 to 55 years. US data were acquired from 33 sites in the upper trapezius of the 9 subjects (2 sites in 1 subject and 1 site in another subject could not be imaged because of scheduling restrictions). Based on the physical examination, 13 sites were classified as A-MTrPs, 6 sites were classified as L-MTrPs, and 14 sites were classified as normal. Eleven of the 13 A-MTrPs, 5 of the 6 L-MTrPs, and 1 of the 14 normal sites were found at a more medial location on the upper trapezius (relative to the midline of the body), while the rest were found at a more lateral location. Pressure algometry was performed at 30 of these sites (algometry was not performed in 1 patient).

PPTs determined using pressure algometry showed that sites with A-MTrPs and L-MTrPs had a significantly lower threshold compared with palpably normal sites (P<.01) (fig 2). The PPTs were not significantly different between A-MTrPs and L-MTrPs.

On US imaging, MTrPs in the upper trapezius muscle appeared as elliptically shaped focal areas of hypoechoicinity that corresponded with the location of the palpable nodule in all the subjects examined (fig 3A). In our images, trigger points often appear to coexist with multiple nodules in close proximity (fig 3B). The size of MTrPs was .16±.11cm² (mean ± SD for both examiners and for all subjects). No significant size difference was found between A-MTrPs (.16±.11cm²) and L-MTrPs (.15±.13cm²). When quantifying size, there was good agreement between the examiners (Pearson’s r=.78, P<.001, excluding 1 outlying case where the first examiner identified a single large MTrP, while the second examiner identified 2 smaller MTrPs).

In 3D imaging, the discrete trigger point was clearly visible in the longitudinal, transverse, and coronal images (fig 4). The echotexture in the nodule was heterogeneous and markedly different from a control area in the upper trapezius muscle found to be palpably normal on physical examination. The palpably normal tissue appeared isoechoic with homogeneous echotexture (compare fig 5A and 5C).

On color variance imaging, MTrPs appeared as focal areas of reduced vibration amplitude. Figure 5D shows the color variance image of the upper trapezius muscle with a palpable MTrP indicated by an arrow. The area of reduced vibration amplitude corresponds with the hypoechoic regions observed on 2D gray-scale imaging for all subjects. Figure 5B shows the color variance image of the upper trapezius muscle that was normal on physical examination. The entire region of the muscle appears to vibrate with approximately uniform amplitude, as indicated by the uniform color. In some regions, single, well-defined nodules were apparent, while in others multiple focal nodules could be observed (fig 5E, 5F).

Color Doppler and duplex Doppler examination of the upper trapezius in our subject sample revealed differences between A-MTrPs and L-MTrPs. Prominent blood vessels were found at 27 of the 33 sites. In some cases, the blood vessel passed directly through a trigger point (figs 6 and 7). In 9 (69%) of 13 sites with A-MTrPs, we observed unique Doppler flow waveforms (fig 8) with retrograde diastolic flow. Intriguingly, retrograde diastolic flow was observed in only 1 (16.7%) of 6 sites with an L-MTrP. Only 1 palpably normal site of 14 (7%) showed sustained retrograde diastolic flow. This site was adjacent to a site with an A-MTrP.
To quantify these findings, we used a composite ordinal scoring system using the tissue imaging score and blood flow score as variables (see Table 1). The number of sites and their scores based on US imaging for active, latent, and normal sites in the upper trapezius muscle are shown in Figure 9. Palpable trigger points identified on physical examination had a significantly higher tissue imaging score compared with normal myofascial tissue \((P < .002\) using the nonparametric Kruskal-Wallis test), indicating that the imaging findings correlated with the physical findings. Tissue imaging scores were not significantly different between A-MTrPs and L-MTrPs. However, A-MTrPs had a significantly higher blood flow waveform score compared with L-MTrPs in the upper trapezius \((P < .021\), Kruskal-Wallis), indicating that A-MTrPs are associated with blood flow abnormalities. Blood flow score was not significantly different between L-MTrPs and normal sites.

**DISCUSSION**

This study was undertaken to determine whether a noninvasive, easily accessible and relatively inexpensive technology could be used to characterize MTrPs. Our preliminary findings indicate that palpable nodules (identified as either A-MTrPs or L-MTrPs) appear as 1 or more focal nodules on gray-scale and color variance US imaging and are absent in palpably normal myofascial tissue. Some hypoechoic regions were observed even when nodules were not palpable. We did not investigate the ultrasonic characteristics of taut bands in this study. A-MTrPs and L-MTrPs demonstrated different and distinct blood flow waveform patterns. Significantly more A-MTrPs than L-MTrPs were associated with retrograde flow in diastole, which would be indicative of a highly resistive vascular bed.

A-MTrPs have 2 clinical attributes that must be investigated in order to elucidate the pathogenesis and pathophysiology of myofascial pain. One attribute is a motor dysfunction of the myofascial tissue as indicated by the finding of a constant, discrete hardness, usually palpable as a nodule in a taut band within the belly of the muscle. The other attribute is a sensory abnormality characterized primarily by tenderness and spontaneous pain. The pain can be localized to the immediate area of the nodularity/taut band or be referred to a distant part of the body, or both.

Several promising lines of scientific study (ie, histologic, neurophysiologic, biochemical, and somatosensory) have attempted to explain the pathophysiologic basis of the motor and sensory abnormalities associated with MTrPs. Findings from these studies support Simons’ Integrated Hypothesis, which postulates that an “energy crisis” perpetuates an initial sustained sarcomere contracture, which leads to increased local metabolic demands in the presence of compromised capillary circulation. This leads to local hypoxia, tissue damage, or both. Shah et al found and confirmed the presence of elevated levels of inflammatory mediators, catecholamines, neuropeptides, and proinflammatory cytokines in the vicinity of A-MTrPs. These biochemicals are known to be associated with persistent pain states, myofascial tenderness, intercellular signaling, and inflammation. These attributes may help explain the sensory abnormalities associated with A-MTrPs.

Results of 2D US imaging confirm that significant tissue abnormalities and morphological changes are associated with MTrPs. Differences in echogenicity and stiffness of the MTrP compared with the surrounding tissue suggest a disruption of normal muscle fiber structure and a change in local tissue characteristics. The hypoechoic and stiffer nodules may be indicative of contraction knots resulting from increased muscle fiber contraction and recruitment, local injury, and/or localized regions of ischemia.
Fig 5. Simultaneous 2D gray-scale and color variance imaging. (A and B) Normal upper trapezius muscle. The normal muscle appears isoechoic and has uniform color variance (TIS=0). (C and D) Muscle with a palpable MTrP. A hypoechoic region and a well-defined focal decrease of color variance indicating a localized stiffer region is visible (TIS=1). (E and F) Muscle with a palpable MTrP. Multiple hypoechoic regions and multiple focal nodules are visible (TIS=2). Abbreviation: TIS, tissue imaging score.
To date, we know of only one other study that has attempted to objectively characterize the physical features of MTrPs. In this study by Chen et al., MRE was used to measure the viscoelastic properties of the upper trapezius muscles. MRE uses a modified gradient echo pulse sequence to image the propagation of induced vibration shear waves. Although they were able to demonstrate that the shear wave propagation pattern in a taut band differs from that in palpably normal myofascial tissue, the MTrP itself was not identified within the taut band. US imaging may be a better method to localize the MTrP while simultaneously investigating the neighboring tissue structure and vasculature. 2D US combined with VSE provides both visual imaging and objective measurements of the physical characteristics and mechanical properties of the MTrP. We believe that this novel application of imaging US will provide significant methodological improvement over MRE, enabling cost-effective imaging and longitudinal monitoring of MTrPs in an office-based setting. Using a transient vibration source, the velocity of the propagating shear wave could be measured using US, which would allow further quantification of the tissue stiffness. US provides new and exciting possibilities for identifying physical characteristics of MTrPs on human subjects in vivo, noninvasively and at low cost.

Another advantage of US imaging is the ability to investigate the vasculature and blood flow characteristics in and around MTrPs. Results of our blood flow investigation associate blood flow disturbances with the pathophysiology of A-MTrPs. Doppler flow waveforms of arteries in the neighborhood of A-MTrPs showed high-resistance blood flow with retrograde diastolic flow, which differed from the blood flow around L-MTrPs and normal, uninvolved myofascial tissue. An increase in vascular resistance in A-MTrPs is consistent with blood vessel compression caused by sustained contracture at a trigger point or constriction caused by oxidative stress or hypoxia. The blood vessel compression may be sufficient to cause the local hypoperfusion, hypoxia, and other pathophysiologic developments leading to the pain, tenderness, and nodularity of an A-MTrP.

Jarvholm et al. found that high intramuscular pressure in the supraspinatus muscle significantly impeded local muscle blood flow, which would lead to hypoperfusion and local ischemia. Measurements of the Po2 in myogeloses (which presumably are accumulations of several MTrPs) showed that the Po2 is extremely low (close to 0) at the center of an MTrP area. The low Po2 is likely to be associated with a lack of adenosine triphosphate, a condition that may contribute to the local hypercontractions (contractures) found in this area. Adenosine triphosphate is needed to break the bonds between the myofilaments and to end the muscle contraction. The available data on myofascial pain suggest that, in fact, pain associated with A-MTrPs may be caused indirectly by decreased blood flow to the MTrP. The hypoperfusion may lead to the observed low Po2 and the contractures. A low Po2 is known to be a powerful factor for the release of bradykinin, a sensitizing agent for muscle nociceptors.

The US techniques presented in this article can be used to identify anatomic and physiologic abnormalities associated with MTrPs. These findings will help to establish objective diagnostic criteria for the identification and classification of MTrPs, which would be more reliable, sensitive, and specific than physical examination alone. Changes in these objective measures in response to treatment, such as alterations in the blood flow or changes in the size, appearance, or stiffness of trigger points, or both, could then be used as treatment outcome measures. Further studies are needed to confirm these findings and their clinical usefulness.
Study Limitations

We believe that our observations of echogenicity, stiffness, and blood flow may enable differentiating A-MTrPs from L-MTrPs through US visualization. However, this study is exploratory and descriptive, and the findings are from a small number of subjects. Therefore, universal generalization to MPS and MTrPs is premature, as is development of a definitive model to explain etiology of MTrPs.

In addition, there are some technical difficulties, which we are currently addressing. For example, the operator technique (amount of pressure and the angle at which the US transducer head is held to scan the tissue) is difficult to control. We are attempting to standardize the scan technique for future investigations. The vibrator used in our study for qualitative sonoelastography imaging was a modified off-the-shelf handheld device; for more quantitative studies improved designs are
possible. While our methodology was designed to keep the sonography team blinded to the clinical findings, unintentional bias cannot be ruled out because the nodules were readily visualized on imaging. Our study did not include a control group of pain-free subjects. All but 2 subjects had unilateral symptoms, and the contralateral side in the same subject was used as the control. However, some bias resulting from the anatomic location of the MTrPs cannot be ruled out. Finally, interobserver and intraobserver variabilities were not assessed in this feasibility study. Nonetheless, we believe that the reported findings expand the understanding of MTrPs and bring us closer towards a model of the pathophysiology of the MTrPs and their surrounding milieu.

CONCLUSIONS

Under the conditions of this investigation, gray-scale echogenicity and color variance imaging based on relative stiffness were used to differentiate palpable nodules in soft tissue (either active or latent) from normal myofascial tissue, and blood flow waveform characteristics were used to differentiate A-MTrPs and L-MTrPs. Our preliminary findings show that US, a convenient, accessible, and low-risk technique, can be a useful method for differentiating MTrPs from surrounding tissue.

References


Suppliers
b. Philips Healthcare USA, 22100 Bothell Everett Hwy, Bothell, WA 98021-8431.
c. North Coast Medical, Inc, 18305 Sutter Blvd, Morgan Hill, CA 95037-2845.