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Understanding the Vascular Environment of Myofascial Trigger Points using Ultrasonic Imaging and Computational Modeling

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Abstract

Myofascial pain syndrome (MPS) is a common, yet poorly understood, acute and chronic pain condition. MPS is characterized by local and referred pain associated with hyperirritable nodules known as myofascial trigger points (MTrPs) that are stiff, localized spots of exquisite tenderness in a palpable taut band of skeletal muscle. Recently, our research group has developed new ultrasound imaging methods to visualize and characterize MTrPs and their surrounding soft tissue. The goal of this paper was to quantitatively analyze Doppler velocity waveforms in blood vessels in the neighborhood of MTrPs to characterize their vascular environment. A lumped parameter compartment model was then used to understand the physiological origin of the flow velocity waveforms. 16 patients with acute neck pain were recruited for the study and the blood vessels in the upper trapezius muscle in the neighborhood of palpable MTrPs were imaged using Doppler ultrasound. Preliminary findings show that symptomatic MTrPs have significantly higher peak systolic velocities and negative diastolic velocities compared to latent MTrPs and normal muscle sites. Using compartment modeling, we show that a constricted vascular bed and an enlarged vascular volume could explain the observed flow waveforms with retrograde diastolic flow.

Keywords

Chronic pain; myofascial trigger points; Doppler ultrasonic imaging; compartment modeling

I. Introduction

Chronic pain is a significant public health problem. Myofascial pain syndrome (MPS) is a common soft-tissue pain syndrome, with prevalence varying from 21% of patients seen in a general orthopedic clinic, to as high as 85–93% of patients presenting to specialty pain management centers with chronic pain disorders [1]. In spite of its high prevalence, the

pathophysiology and pathogenesis of MPS is poorly understood. MPS is characterized by local and referred pain originating from hyperirritable nodules known as myofascial trigger points (MTrPs) that are stiff, localized spots of exquisite tenderness in a palpable taut band of skeletal muscle. Palpation of MTrPs can produce referred pain. Active, or symptomatic, MTrPs (A-MTrPs) produce spontaneous pain, are acutely tender to palpation and may be associated with stiffness and restricted range of motion, while latent MTrPs (L-MTrPs) have similar physical findings but do not produce spontaneous pain symptoms [2]. There are currently no objective criteria for the diagnosis of MTrPs or for assessing clinical outcome of treatments.

Our research group has been involved in understanding the soft tissue viscoelastic properties and blood flow environment of MTrPs based on ultrasound imaging, elastography and Doppler imaging. In preliminary studies, we have shown that MTrPs appear hypoechoic on ultrasound imaging, and are stiffer on elastography, and have unique blood flow signatures [3]. These imaging methods could lead to objective clinical outcome measures and definitive diagnostic criteria to anatomically locate MTrPs, and quantify response to treatment. In addition, these quantitative methods could elucidate the pathogenesis and pathophysiological mechanisms of MPS. The objective of this study was to further understand the vascular environment of MTrPs using a bioengineering approach that combines Doppler flow waveform analysis and computational modeling.

II. Methods and Materials

A. Participants

This study was carried out at the Rehabilitation Medicine Department of the National Institutes of Health, Clinical Research Center, Bethesda, MD, USA. Subjects with acute neck pain (< 3 months duration) were eligible and met inclusion criteria if found to have an A-MTrP in one or both upper trapezii. All subjects underwent a thorough musculoskeletal evaluation so as to rule out potential causes of their symptoms other than MTrPs. The exclusion criteria were: subjects with muscle pain due to fibromyalgia, atypical facial neuralgia and history of myopathy; neck and shoulder conditions including cervical radiculopathy, history of cervical spine or shoulder surgery and history of trigger point injections in the upper trapezius; and subjects with cancer or head, ear, eye, nose and throat infections. Based on their history and physical findings in the upper trapezius muscles, 16 subjects were recruited. Three subjects were pain free, while the rest were diagnosed with myofascial pain. Of the 13 symptomatic subjects, all but three had bilateral 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.

B. Clinical Examination

Subjects underwent a physical examination by an experienced physiatrist (JPS), who determined the presence or absence of MTrPs in the upper trapezius muscle according to the standard clinical criteria defined by Travell and Simons [4]. In this study any local region of myofascial tissue in which nodules were absent to palpation was defined as “normal” or uninvolved. For our study we sought to identify 2 sites per side on each subject including a variety of A-MTrP, L-MTrP, and normal sites. Each subject was to have at least one normal site among the 4. Palpation was performed 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 L-MTrP. If less than 2 nodules were identified, palpation continued until the examiner was satisfied that only one nodule 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 at least 2 marked sites on each upper trapezius. Only the examiner knew the clinical status and classifications of the marked sites; i.e., the sonography team (SS, TG) was blinded to clinical status and identity of all sites.

C. Ultrasound Imaging

Each participant underwent US examination using a Philips iU22 clinical US system with a 12-5 MHz linear array L12-5 transducer targeted at the sites palpated during the clinical examination. Doppler ultrasound was used to visualize blood vessels and quantify the flow velocities at the sites of A-MTrPs, L-MTrPs and within normal myofascial tissue. During imaging, the sonographer applied two different levels of pressure using the ultrasound transducer, starting with minimal pressure with the transducer barely contacting the skin surface, and moderate pressure visibly compressing the muscle. The spectral Doppler velocity waveforms were analyzed to trace the peak velocity envelope throughout the cardiac cycle. The peak systolic velocity (PSV), minimum diastolic velocity (MDV), resistive index ($[PSV-MDV]/PSV$), pulsatility index ($[PSVMDV]/\text{mean velocity}$), acceleration time (AT), and time-averaged peak velocity (TAPV) were computed automatically using the standard analysis software available on the ultrasound system.

D. Statistical Analysis

Comparisons were performed between the active, latent and normal sites for each of the blood flow waveform parameters. Differences between groups were assessed using the Mann-Whitney U test, since the parameter values in our study were not normally distributed. Statistical significance was determined at the 5% level for a two-tailed test.

E. Lumped Parameter Compartment Model

To better understand the physiological significance of the observed blood flow waveforms, we developed a lumped parameter model of a vascular network with two paths. Each path consisted of a compliant volume representing the arteriolar compartment and a lumped capillary/venular outflow resistance. Lumped parameter compartment models of skeletal circulation have been previously published [5]. For our simplified model, we did not include any nonlinear variations of compliance and resistance. Figure 1 shows a schematic of the model. The volume of the compartment determines the overall resistance to flow and the rate of volume change in response to a pressure change determines the compliance. The vascular volume and outflow resistance were varied to model different scenarios. The model equations were derived based on conservation of flow, relationships between resistance and vessel diameter, and compliance and vessel volume, and definitions of vascular impedance:

$$Q_{outi}(t) = \frac{P_{in}(t) - P_{out}}{R_i(t) + R_{outi}}; Q_i(t) = \frac{dV_i(t)}{dt} = C_i \frac{dP_{in}(t)}{dt};$$

$$Q_{ini}(t) = Q_i(t) + Q_{outi}(t);$$

$$R_i(t) = \frac{128\mu L}{\pi d_i(t)^4}; C_i = \frac{\pi d_i(0)^2 L}{4P_{mean}};$$

$$V_i(0) = P_{in}(0) * C_i + V_0;$$

where P_{in} is the input arterial pressure, P_{out} is the venous pressure, P_{mean} is the mean arterial pressure, L is the length of the vessel path and V_0 is the residual volume of the compartment. For the i^{th} vessel, Q_{ini} is the inflow, Q_i is the flow through the compartment, V_i , C_i , d_i are the volume, compliance and diameter of the compartments, Q_{outi} is the outflow, and R_{outi} is the outflow resistance. All simulations were performed using the JSIM software developed at the University of Washington by the National Simulation Resource [6]. The Dopri5 method was used for numerically solving the model differential equations.

III. Results

Based on clinical examination, a total of 74 sites were identified either as active, latent or normal in the 16 subjects. Blood vessels with measurable flow velocities could be imaged at 56 of these sites. Of these, 20 were active, 16 were latent, and 20 were normal sites. Blood vessels were occasionally observed very close to a MTrP visualized on imaging, as shown in Figure 2(A), while in other cases they were observed in the immediate vicinity. The observed flow waveforms fell into one of four categories as shown in Figure 2(B.1–B.4). They were either high resistance flow with no diastolic flow (B.1), elevated diastolic flow (B.2), flow oscillation in early diastole (B.3) and sustained retrograde flow in diastole (B.4).

Tables 1 and 2 and Figure 3 summarize the results of blood flow waveform analysis. The peak systolic velocities at active sites were significantly higher than those at latent and normal sites, whereas the minimum diastolic velocities were significantly lower. Pulsatility indices at active sites were significantly higher than those at normal sites. No significant differences in these values were found between normal and latent sites. Other blood flow waveform measures did not show significant differences between the groups.

Figure 4 shows that the results of compartment modeling can be used to explain the blood flow waveform differences between active, latent and normal sites. Figure 4(A) shows the simulated arterial pressure waveform modeled as a superposition of sinusoids with 10 harmonics [7].

Figure 4(B) shows the flow waveform through the two vascular paths in Figure 1, where the outflow resistance and vascular volume were increased in one path. When the outflow resistance of vessel significantly exceeded that of the collateral vessel, the blood flow waveform through the constricted vessel exhibited retrograde flow in diastole, and reproduced several key features of the observed blood velocity waveform. For the simulation shown in Fig. 4, the following parameters were chosen: $P_{mean} = 100$ mmHg, $P_{out} = 10$ mmHg; $d_1(0) = 500$ μ m, $d_2(0) = 200$ μ m, $R_{out1} = 3$ dyne $\times$ s/ μ L/cm², $R_{out2} = 3 \times 10^7$ dyne $\times$ s/ μ L/cm², $L = 12.5$ mm, $V_0 = 0.1$ μ L. Figure 5 shows how the flow waveform depends upon both the initial vascular volume and the outflow resistance. A significant increase in outflow resistance for a given vascular volume decreases the flow through the path and causes some retrograde diastolic flow. However, an increase in the vascular volume increases the flow pulsatility with high systolic velocities and sustained retrograde diastolic flow. The increased volume acts as a reservoir into which the blood flows during systole, and flows out during diastole. These results suggest that a combination of obstructed vascular bed and enlarged vascular volume could lead to the observed flow waveforms with retrograde diastolic flow.

IV. Discussion

Our preliminary findings suggest that the blood flow waveforms in the vicinity of symptomatic (active) MTrPs have significantly different features compared to the waveforms in the vicinity of latent MTrPs and normal muscle sites. The flow waveforms near active sites show increased systolic velocities and flow reversal with negative diastolic velocities. Through computational modeling, we have identified two contributing factors that could lead to these observed waveforms. The first is an increase in the volume of the vascular compartment, and the second is an increase in outflow resistance. Increased outflow resistance could be either due to muscle contracture at the MTrP that compresses the capillary/venous bed, anatomical 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 using the ultrasound transducer during imaging. In our studies, we have found that active MTrPs predominantly tend to be found in the apex of the upper trapezius, and the anatomy could potentially be a contributing factor. The second contributing factor to the observed flow waveforms is an increased vascular volume. The significance of this finding in the context of pain symptoms is yet to be fully understood, but could provide some intriguing clues to elucidate the underlying pathophysiological mechanisms of MTrPs and myofascial pain syndrome. In our previous studies analyzing the biochemical milieu of MTrPs, we have observed elevated levels of pro-inflammatory mediators and other neuropeptides near A-MTrPs that are associated with vasodilatory and angiogenic effects [8]. These may play a role in the observed flow waveforms. The anatomical location of the MTrP could also play a role.

Several limitations of this preliminary study should be acknowledged. We did not include measures of reproducibility of the flow waveforms. The amount of external pressure applied by the transducer was qualitatively assessed, and more quantitative measurement is needed to standardize the measurements. In our study, a single physiatrist and sonographer performed all examinations, and inter- and intra-observer variability were not assessed. We did not analyze systemic changes in the blood flow supply to the upper trapezius muscle through the transverse cervical artery. These limitations will be addressed in future studies.

In this preliminary study, we have shown that the blood flow waveforms show differences between active, latent and normal sites in symptomatic and asymptomatic subjects. We have also demonstrated that simple lumped parameter models can be used to explain the observed flow waveforms. In future studies, we will extend and apply the computational modeling approach described in this paper to investigate underlying mechanisms linking imaging observations with clinical symptoms and physical findings.

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Figure 1. Schematic of a lumped parameter compliant vessel model with two vessels simulating blood flow through muscle with a MTrP.

Figure 2.

(A). Color Doppler image showing a blood vessel very close to a palpable MTrP, which appears as a hypoechoic nodule on ultrasound images (arrow). (B) Four types blood flow waveforms that are representative of the findings in our study.

Blood Flow Waveform Measures

■ Peak Systolic Velocity ■ Minimum Diastolic Velocity ■ Pulsatility Index

Figure 3.

Difference in blood flow waveform parameters between active, latent and normal sites. The bars correspond to median values, and the error bars represent the interquartile range.

Figure 4.

Simulated blood flow waveforms reproduces several features of the observed blood velocity waveforms, such as the flow oscillation at the dicrotic notch and retrograde flow in diastole. (A) The simulated arterial pressure waveform with 10 harmonic components. (B) Inflow waveforms through the two vascular pathways in Fig. 1(B). (C) Doppler velocity spectrum near L-MTrP. (D) Doppler velocity spectrum near A-MTrP.

Figure 5. Dependence of flow waveform on the initial vascular volume and outflow resistance. As the outflow resistance increases, the inflow decreases. However for a larger initial vascular volume, the pulsatility of the flow waveform increases with larger retrograde diastolic flow.

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Table 1

Blood flow waveform measurements in the neighborhood of active and latent MTrPs and normal sites in the upper trapezius muscle. Values are median $\pm$ half the interquartile range.

	PSV (cm/s)	MDV (cm/s)	PI	RI	AT (ms)	TAPV (cm/s)
Normal	9.75 $\pm$ 7.19	0 $\pm$ 2.36	3.02 $\pm$ 6.36	0.97 $\pm$ 0.02	52 $\pm$ 12.87	1.89 $\pm$ 0.78
Latent	15.75 $\pm$ 3.97	-1.61 $\pm$ 1.99	6.25 $\pm$ 8.18	0.97 $\pm$ 0.02	52 $\pm$ 11.13	1.87 $\pm$ 0.99
Active	25.1 $\pm$ 10.3	-5.27 $\pm$ 3.35	11.78 $\pm$ 9.67	0.98 $\pm$ 0.02	59 $\pm$ 8	1.83 $\pm$ 1.17

PSV: peak systolic velocity; MDV: minimum diastolic velocity; PI: pulsatility index = (PSV-MDV)/mean velocity; RI: resistive index = (PSV-MDV)/PSV; AT: acceleration time; TAPV: time averaged peak velocity.

Table 2

Significant differences between blood flow measures at different sites.

	Peak Systolic Velocity	Minimum Diastolic Velocity	Pulsatility Index
Normal vs. Latent	$p=0.18$	$p=0.667$	$p=0.147$
Normal vs. Active	$p=0.006^*$	$p=0.04^*$	$p=0.03^*$
Latent vs. Active	$p=0.04^*$	$p=0.043^*$	$p=0.32$

*
 $p<0.05$ for a two-sided Mann-Whitney U-test