Laryngeal and Global Somatosensation in Primary Muscle Tension Dysphonia

[*,†]Adrianna C. Shembel, [*]Ted Mau, [‡]Jason Zafereo, [*,†]Robert Morrison, [†]Caroline Crocker, [†]Avery Moore, and [†]Arlin Khan, *‡*Dallas, and* †*Richardson, Texas*

Summary: Introduction. Primary muscle tension dysphonia (pMTD) is a functional voice disorder that reduces communicative abilities and adversely impacts occupational productivity and quality of life. Patients with pMTD report increased vocal effort, fatigue, discomfort, and odynophonia. Although laryngeal and paralaryngeal muscle tension and hyperfunction are the most commonly proposed mechanisms underlying these symptoms, recent studies suggest pMTD may have more to do with the somatosensory system. However, relationships between voice symptoms and somatosensory mechanisms are poorly understood, creating challenges for mechanistic-based pMTD management. The first objective was to compare laryngeal, paralaryngeal, and global somatosensation between subjects with and without pMTD. The second was to determine relationships between pMTD symptoms and somatosensation.

Methods. Fifty-two (20 pMTD and 32 control) subjects underwent laryngeal sensory testing with aesthesiometers, as well as peripheral mechanosensory and dynamic temporal summation testing to paralaryngeal and limb regions. Voice symptom severities (vocal effort, fatigue, discomfort, and odynophonia) were collected on 100-mm visual analog scales before and after laryngeal sensory testing. Participants also completed the Central Sensitization Inventory.

Results. Patients with pMTD reported significantly higher laryngeal sensations ($P = 0.0072$) and voice symptom severities ($P < 0.001$) compared with the control group, and had significantly more vocal tract discomfort postlaryngeal sensory testing compared with the prelaryngeal sensory testing timepoint ($P = 0.0023$). However, there were no significant group differences in laryngeal airway protection responses suggestive of peripheral laryngeal hypersensitivities ($P = 0.444$). There were also no significant group differences on paralaryngeal or global sensitivities ($P > 0.05$), and no correlations between severity of voice symptoms and perceptual laryngeal sensations or hypersensitivities ($P > 0.05$).

Conclusion. Patients with pMTD perceive more sensitivities in the larynx and feel more sensations related to the voice (vocal effort, fatigue, discomfort, and pain). However, in general, patients with pMTD do not have abnormal peripheral laryngeal hypersensitivities, increased global somatosensation, or heightened central sensitivity. The lack of significant correlations between peripheral laryngeal hypersensitivities and voice symptom severity ratings suggests these outcome variables target distinct mechanistic constructs.

Key Words: Vocal effort—Vocal fatigue—Vocal tract discomfort—Odynophonia—Mechanosensation—Central sensitization.

INTRODUCTION

Primary muscle tension dysphonia (pMTD) is a functional voice disorder that reduces communicative abilities and adversely impacts daily living, occupational productivity, and quality of life.[1] Of the 20 million individuals in the United States with voice disorders, an estimated 2–8 million struggle with pMTD and calculations suggest pMTD causes about $2 billion in lost annual productivity costs due to absenteeism.[2] Although pMTD leads to the same emotional, social, financial, and occupational hardships as organic, structural, and neurological voice disorders, there is significantly less effort to study its underlying mechanisms or understand its pathophysiology. Patients with pMTD report increased vocal effort, vocal fatigue, vocal tract discomfort, and odynophonia, and may exhibit aberrant vocal qualities.[3–5] However, these voice symptoms are also common in many other types of voice disorders, making it challenging to accurately diagnose pMTD.[6,7]

The level of vocal dysfunction resulting from pMTD voice symptoms does not align with the relatively normal laryngeal presentations (ie, lack of overt structural or neurological laryngeal abnormalities) observed on laryngoscopy.[5,6,8] These challenges can lead to protracted periods before a formal diagnosis can be reached, and end in a diagnosis based on exclusion after other potential diagnoses have been ruled out.[8] The lack of diagnostic indicators and poor understanding of pathophysiological mechanisms underlying clinical presentations in pMTD

Accepted for publication August 2, 2024.

This work was supported by NIDCD grants R21DC019207 and R01DC021447 (PI: Adrianna Shembel). The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institute on Deafness and Other Communication Disorders or the National Institutes of Health.

From the *Department of Otolaryngology – Head and Neck Surgery, University of Texas Southwestern Medical Center, Dallas, Texas; †School of Behavioral and Brain Sciences, University of Texas at Dallas, Richardson, Texas; and the ‡Department of Physical Therapy, University of Texas Southwestern Medical Center, Dallas, Texas.

Address correspondence and reprint requests to Adrianna C. Shembel, 2001 Inwood Road, WCB3 7878, Dallas, TX 75235. E-mail: adrianna.shembel@utsouthwestern.edu

Journal of Voice, Vol xx, No xx, pp. xxx–xxx

0892-1997

© 2024 The Voice Foundation. Published by Elsevier Inc. All rights are reserved, including those for text and data mining, AI training, and similar technologies.

https://doi.org/10.1016/j.jvoice.2024.08.003

also makes the voice disorder challenging to treat with precision. A better understanding of pMTD etiology and its pathophysiological mechanisms is needed to refine diagnostic paradigms and optimize treatment for more effective management.

The most commonly proposed pathophysiological mechanisms underlying pMTD symptoms are laryngeal and paralaryngeal muscle tension and hyperfunction. However, these theoretical mechanisms have traditionally been based largely on anecdotal observations in the voice clinic and have often been defined without well-validated quantitative metrics or control subject comparisons. More recent developments in novel, well-vetted physiologic metrics to quantify tension and hyperfunction in the laryngeal and paralaryngeal muscles have challenged this theory. Across multiple studies—including investigations using shear wave elastography,[9] optical flow,[10] and motion capture[11] technologies to track extrinsic laryngeal and paralaryngeal motor patterns, vocal fold tracking[12] to quantify vocal fold adduction, and the use of various supraglottic metrics[5,13–15] to quantify supraglottic compression—patients with pMTD had similar motor patterns to vocally healthy individuals.[1] In contrast, across all studies, patients with pMTD reported significantly higher levels of vocal effort and vocal tract discomfort compared with vocally healthy subjects.[5,9–13] There were also poor correlations between these somatosensory voice symptoms and (para)laryngeal motor patterns, suggesting these constructs do not have direct influences on one another. Previous work also found that patients with pMTD who had more difficulties attending to body sensations had higher Voice Handicap Index-10 (VHI-10)[16] and Vocal Fatigue Index (VFI)[17] scores,[18] suggesting the somatosensory system may play a role in perceptions of vocal fatigue (VFI) and vocal impact (VHI-10).

In summary, results across these various studies suggest clinical presentations of pMTD may have more to do with somatosensation than aberrant motor patterns within and around the laryngeal system. However, the role of somatosensation in the laryngeal system, in general, and in pMTD, specifically, has been poorly understood and defined. In contrast, there is a well-established relationship between mechanosensory tactile stimulation and perceived sensations of physical effort, fatigue, discomfort, and pain in the limbs.[19–23] Altered sensitization in the limbs can result in muscle aches, pain, and tenderness (eg, myalgia, hyperalgesia), as well as increased sensitivity to musculoskeletal stimuli that do not normally provoke discomfort or pain (eg, allodynia).[24] Heightened global sensitization in the body is thought to be fundamental to various central sensitization disorders like fibromyalgia, chronic fatigue, and chronic pain disorders.[25–30] Chronic activation of peripheral nociceptive signaling has shown to underlie certain types of peripheral nociceptive musculoskeletal disorders like chronic tension-type headaches—originally thought to largely be due to neck muscle tension.[31–34] Although anecdotal, it is not uncommon for patients with pMTD to report tension headaches and migraines when asked about muscle tension in the head and neck.

Considering the significantly higher vocal effort, vocal fatigue, vocal tract discomfort, and odynophonia reported by patients with pMTD, especially in the absence of overt aberrant (para)laryngeal motor patterns, there is a possibility pMTD could share commonalities with these other types of central sensitization or peripheral nociceptive musculoskeletal disorders. Previous literature on pMTD has also alluded to the somatosensory system contributing to pMTD in the context of central sensitivity syndromes,[35] as part of the irritable larynx syndrome constellation,[36,37] and has proposed pMTD to be a somatoform disorder.[38] As such, the roles of pMTD clinical presentations specific to the sensory laryngeal system and global somatosensation in pMTD require elucidation.

The overarching goal of this study was to evaluate the role of localized (ie, laryngeal, paralaryngeal) and global (ie, upper and lower limb) somatosensation in pMTD. The first objective was to compare laryngeal, paralaryngeal, and limb somatosensation between individuals with and without pMTD. The second was to compare voice-specific symptom severity ratings of vocal effort, fatigue, discomfort, and odynophonia to somatosensory sensitivity testing. We hypothesized that (1) patients with pMTD would have more localized sensitivities in the laryngeal region and global hypersensitivities in the limbs than patients without pMTD, and that (2) there would be a significant relationship between voice symptoms and laryngeal, paralaryngeal, and global sensory testing.

METHODS

Participants

The study was approved by the University of Texas (UT) Southwestern Medical Center Institutional Review Board. Twenty patients diagnosed with pMTD and 32 vocally healthy controls provided written consent and were enrolled in the study at the UT Southwestern Center for Voice Care between 2023 and 2024 (Table 1 for demographic information). To be eligible for the pMTD group, participants had to have laryngeal examinations with no observable structural (eg, benign vocal fold lesions) or neurological (eg, vocal fold paralysis) laryngeal abnormalities and needed to meet three out of five of the following criteria, based on previous studies[9–11]: (1) score more than 11/40 on the VHI-10[16], (2) have a direct magnitude estimation of perceived phonatory effort (DME of at least 200,

[1] The one exception was increased severity of mediolateral supraglottic compression in the pMTD group. However, many vocally healthy individuals also had this supraglottic pattern, suggesting the presence of mediolateral compression on laryngoscopy is not a diagnostic indicator of pMTD. One reason why there was more *severe* mediolateral supraglottic compression in a subcohort of subjects with pMTD may have something to do with the role the ventricular folds play in airway protection, commonly observed in laryngeal hypersensitivity syndromes.

TABLE 1.
Subject Demographics*

Group	Gender	Age in Years (SD)	Race	Ethnicity
pMTD	43% men 57% women	42.91 (18.59)	57% Caucasian 5% Asian 19% Black or African American 19% Other/not disclosed	10% Hispanic/Latino 76% Non-Hispanic/Latino 14% Other/not disclosed
Controls	17% men 83% women	32.05 (14.08)	27% Caucasian 7% Asian 7% Black or African American 59% Other/not disclosed	10% Hispanic/Latino 27% Non-Hispanic/Latino 63% Other/not disclosed

Abbreviation: SD, standard deviation.

* Inclusion of a demographics table, especially race and ethnicity, is a direct response to the Special Interest Group (SIG3) Voice and Upper Airway panel at the 2022 American Speech-Language and Hearing Association (ASHA) Convention on Voice as an Expression of Identity: A Cultural Construct. Collecting data on race helps understand and address health disparities and the social and economic conditions associated with them. Gathering information on ethnicity allows researchers to capture the cultural context, practices, and values that may influence health outcomes, behaviors, and access to health care. This practice will help ensure that research accurately represents the diversity of the population and is especially important for historically excluded groups whose experiences and perspectives may differ from dominant groups.

or twice as effortful compared with 100 preimpaired voices),[39] (3) have a report of muscle tension or supraglottic compression in their medical chart, (4) score more than 4 on the Vocal Tract Discomfort Scale,[40] and (5) score more than 24 on Factor 1 of the VFI.[17] All participants with pMTD took part in the study before starting voice therapy. The participants in the vocally healthy control group had to meet the following inclusion criteria: (1) no incidences of lost workdays within the last 6 months due to vocal impairments/dysfunction, (2) no laryngeal abnormalities on laryngoscopy, and (3) none of the five inclusionary characteristics defining the pMTD group. Both pMTD and control groups also had to have sufficient nasal patency and little-to-no nasal discomfort when passing a channeled flexible laryngoscope through the nasopharynx to conduct the laryngeal sensory testing.

Protocol

Laryngeal sensory testing with aesthesiometry

To quantify and compare laryngeal sensation between individuals with and without pMTD, previously validated laryngeal aesthesiometers—ie, von Frey filaments with known buckling forces directed to endolaryngeal mucosa through a channeled laryngoscope—were used to study laryngeal airway protection responses (ie, laryngeal hypersensitivities) and self-perceived laryngeal tactile stimuli severities. Previous studies have validated the use of laryngeal aesthesiometers using Nylon monofilaments of different diameters (6-0, 5-0, 4-0, and 3-0) and consistent lengths (30 mm).[41] These laryngeal aesthesiometers are more reliable than other types of laryngeal sensory testing protocols (eg, air puff through channeled laryngoscope).[42,43] Reflexive laryngeal airway protection responses (eg, laryngeal adductor reflex) to these aesthesiometers are a robust method to investigate laryngeal (hyper)sensitivities at the end organ,[44–46] while appraisal of laryngeal stimuli is a higher-order (eg, cognitive) somatosensory process.[47] Testing both laryngeal airway protection responses and appraisal of tactile severity can help differentiate the role of localized laryngeal (hyper)sensitivity at the periphery from higher-order perceptual processing of tactile stimuli.[48] Previous work using these laryngeal aesthesiometers found that positive laryngeal airway protection responses to thinner 6-0 aesthesiometer monofilaments that elicit lower force stimulation was a sign of peripheral laryngeal *hypersensitivity,* while a lack of laryngeal responses to higher 4-0 monofilament force stimulation was suggestive of peripheral laryngeal *hyposensitivity*.[41]

Before aesthesiometer testing, each participant's more patent nasal cavity was decongested and anesthetized for 5–10 minutes using cottonoid pledgets soaked in 0.05% oxymetazoline and 4% lidocaine. This approach was chosen to avoid anesthesia of the laryngopharyngeal region that can occur with standard-of-care nasal spray procedures used in the voice clinic.[49,50] After the pledget was removed, a channeled laryngoscope (Olympus ENF-VT2) was passed through the nasal cavity by one of two board-certified laryngologists on the study team who trained together on the aesthesiometer protocol to ensure consistency and reliability with the aesthesiometer protocol. Once the endoscope was in the pharynx, laryngeal aesthesiometers consisting of different monofilament diameters attached to a catheter (6-0, 5-0, 4-0, and 3-0) (Figure 1C) were passed through the working channel (Figure 1A). Perpendicular presentation of each monofilament to the aryepiglottic folds occurred until a 10%–30% bend of the monofilament of the aesthesiometer had been reached (Figure 1B). Once this brief contact was made, participants were asked to rate the perceived severity of the sensation elicited by the monofilament on a 0–10 Likert scale (0 = no sensation; 10 = maximum sensation) using the scale in Figure 2. Each monofilament diameter was presented twice. The order of the monofilaments was always presented from smallest diameter 6-0 (lowest force) to largest diameter 3-0 (highest force). This decision was made so as not to induce laryngeal airway protection responses (eg, laryngeal adductor reflexes) prematurely in the protocol, which could heighten sensation for subsequent trials.[48]

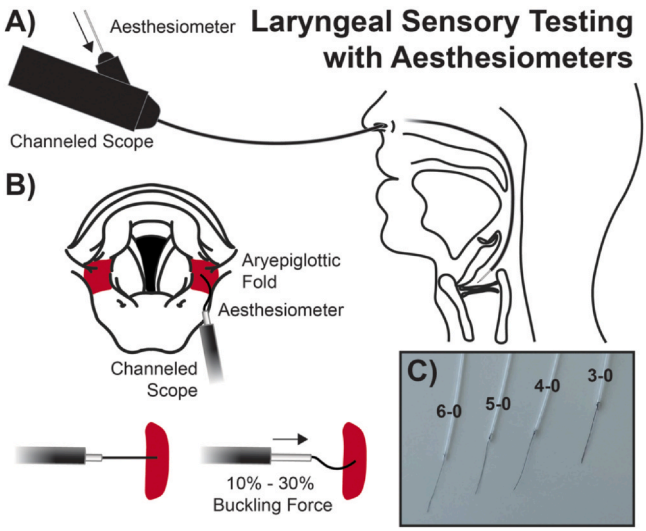

FIGURE 1. Schematic of channeled laryngoscope used for laryngeal sensory testing. **A.** Aesthesiometer presented through the channel of the laryngoscope. **B.** Aryepiglottic areas for sensory testing. **C.** Four monofilament diameters with different buckling forces. Monofilament was randomized to the left or right aryepiglottic fold.

A third negative (foil) trial was also included with the other two positive trials for each monofilament diameter. The foil trial consisted of the act of presenting the monofilament in the pharynx without touching the laryngeal tissue. Participants were asked to rate the severity of the sensation on a 0–10 scale the same way as the other two trials to test for false positives that can result from random noise in the sensory system, hypersensitivities, or to identify potential difficulties with sensory discrimination acuity.[51] Presence (yes) versus absence (no) of laryngeal adductor reflex responses and other airway protection maneuvers (coughing, swallowing, gagging, throat clearing, and guarding) in response to the monofilament presentations were determined for each monofilament across the two positive and one negative (foil) trials. Laryngeal adductor reflex responses were defined as the first visual detection of bilateral vocal fold adduction following tactile simulation. The presence of coughing was defined as rapid vocal fold adduction followed by rapid abduction with expiration. Swallowing was defined based on the presence of epiglottic occlusion followed by a whiteout period. Laryngeal priming and guarding postures in response to the monofilament were defined by brief arytenoid spasms (ie, "twitchy larynx"). The laryngeal sensory testing protocol was video-recorded via standard laryngoscopy procedures and the 0–10 severity ratings audio-recorded for offline data input and statistical analysis of self-reported perceptual symptom severity (Image Stream Medical nCare v10 at 30 fps, MPEG4 format).[44]

Paralaryngeal and global mechanosensory testing with algometry

Mechanical pressure sensitivity thresholds were determined using a pressure algometer to quantify paralaryngeal and limb sensitivities in pound force (lbf)—which measures the force applied to an area. This method targets deep somatic tissue[52,53] (eg, joints, muscles, and fascia) and preferentially activates small/pain fibers (Aδ and C nociceptive fibers) in the musculoskeletal system. Deep afferents local to the affected area are thought to be more active in individuals with peripheral sensitization (eg, nociceptive musculoskeletal pain syndromes), whereas those global to the affected area are thought to be more active in individuals with central sensitization.[54] Mechanical pressure with algometry has previously been applied to the posterior neck, shoulder, back, hand, and leg,[55–59] and validation studies found that pressure algometry yields the least variability and is the most reliable method to assess sensation in the musculoskeletal system.[53,60–62]

A pressure algometer with a 1-cm^2 probe tip (Wagner FPX, Wagner Instruments) was used for this study protocol. Paralaryngeal mechanosensory pressure regions were determined based on previously identified landmarks using Motion Capture technology,[63] as well as previous literature on paralaryngeal areas commonly targeted in circumlaryngeal massage[64,65] and manual therapies.[66–69] Mechanosensory pressures with the algometer were presented to the following regions: (1) submental region corresponding to base of tongue, (2) sternocleidomastoid muscles (bilateral), and (3) anterior tibialis of the leg (the latter used to study global sensory effects, based on previously validated methods[54]) (Figure 3A and C). To obtain quantitative metrics for group comparisons, gradual pressures were applied with the algometer to each region, per standard protocols.[55–58] Mechanosensory pressure was applied until the sensation experienced by the participant transitioned from innocuous pressure to sensation of unpleasantness (eg, discomfort, tenderness). Participants were instructed to hold up their hand with the transition to the unpleasant sensation, at which point pressures exerted via the algometer were terminated and the greatest pressure applied within each trial and to each region was documented in an Excel spreadsheet. Pressures for each region were repeated three times and trials averaged. Due to concerns

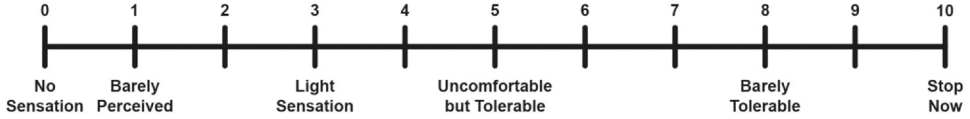

FIGURE 2. A 0–10 Likert scale used for self-perceptual measures of laryngeal sensory testing stimuli severities.

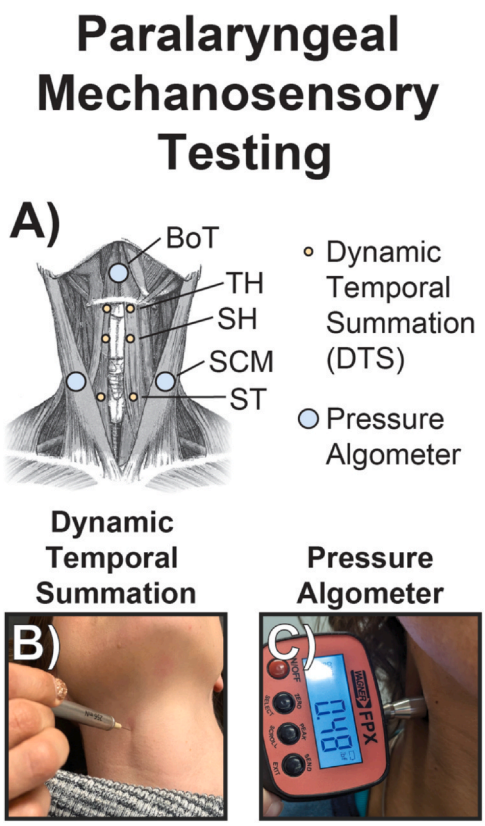

FIGURE 3. **A.** Paralaryngeal targets for mechanosensory pressure algometry and dynamic temporal summation. **B.** Example of dynamic temporal summation with weighted pin to the right thyrohyoid space. C. Example of pressure algometry to the right sternocleidomastoid muscle. BOT, submental region corresponding to base of tongue; SCM, region corresponding to sternocleidomastoid muscles; TH, thyrohyoid region corresponding to the supralaryngeal strap muscles; SH, sternohyoid region corresponding to vocal fold level; ST, sternothyroid region corresponding to infralaryngeal strap muscles.

surrounding application of blunt mechanical pressure forces to the carotid artery and laryngeal cartilage, areas closer to the larynx were only tested using dynamic temporal summation (described in detail in the following section).

Paralaryngeal and global dynamic temporal summation testing with weighted pin

The most common method to evaluate small/pain fibers and deep-tissue sensitivity (eg, determine the presence of hyperalgesia) and assess the role of central sensitization in musculoskeletal pain disorders (eg, fibromyalgia) is dynamic temporal summation with a weighted pin.[51,54] The method works via repeated, equally intense noxious stimuli presented at a consistent rate. Individuals with deep-tissue sensitivity will perceive heightened stimuli intensities. Individuals with central sensitization syndromes not only experience exponential increases of unpleasant sensations but will also report sensations that last longer after termination of the stimuli.[70–72]

The dynamic temporal summation protocol works by presenting 10 repetitive stimuli with a weighted pin (MRC PinPrick Stimulator) with an interstimulus interval of 1 second within an area 1 cm in diameter of the target region. Participants are asked to rate the level of unpleasantness experienced on a 0–10 Likert Scale immediately after the first stimuli, the 10th stimuli trial, 15 seconds after the 10th stimuli trial, and 30 seconds after the 10th stimuli trial. Local target regions in this study were the paralaryngeal regions marked DTS in Figure 3A and B (thyrohyoid [TH], sternohyoid [SH], and sternothyroid [ST]), and the dominant thenar eminence of the palm to test global sensitivities in the body. Ratings were recorded on an Excel spreadsheet. Because temporal summation has not been validated in the neck, the weighted pin that 10 subjects with and without pMTD were able to feel but still tolerate without severe pain (10 on the Likert scale) after 10 trials was used. The 256 mN pin was subsequently used across all subjects to maintain consistency across stimulations and subjects.

Central sensitization inventory

Participants completed the Central Sensitization Inventory,[73] a 25-item self-reported questionnaire that is widely used to characterize central sensitivities. Previous work has shown that this inventory can not only determine the presence of central hypersensitivities, but also correlates well with various quantitative sensory testing methods.[54] Scores ≥33.5/100 are considered significant for central sensitization (more widespread and persistent hypersensitivities, eg, pain).[54,73]

Voice-specific symptom severity ratings

Visual analog scales capture symptom severities in the moment—whereas validated voice indices, like the VHI-10, capture summative experiences like vocal impact over a longer period of time, and are thus better metrics for inclusion criteria. As such, visual analog scales were used to capture state voice symptom severities. Participants were asked to rate their perceived levels of vocal effort, vocal fatigue, vocal tract discomfort, and odynophonia by placing a tick mark on separate 100-mm visual analog scale lines for each parameter (0 = no symptoms, 100 = maximum symptoms). Each voice symptom parameter was collected twice, once before and once after laryngeal sensory testing to see if sensory testing in the larynx had an impact on perceptions of voice symptom severities in either group.

Acoustic voice sample recordings

Three trials of the sustained vowel /i/, each held for 3–5 seconds at modal pitch and conversational loudness (60–75 dB), were recorded in *Praat* (version 6.6.16) at a sampling rate of 44.1 kHz. Audio recording was based on ASHA's 2018

Instrumental Assessments Recommendations,[74] and our previous papers using the same methods.[9–12,75] Recordings were conducted twice, once before and once after laryngeal sensory testing, to determine if there were group differences and to study the effects of sensory testing on acoustic vocal quality.

Data processing

Laryngeal sensory testing perceptual ratings and laryngeal airway protection responses

Self-perceived ratings of laryngeal sensory testing stimuli severities for each monofilament on the 0–10 Likert scale were inputted into an Excel spreadsheet. The presence (yes/no) of laryngeal adductor reflex responses and other airway protection maneuvers via posthoc frame-by-frame analysis of the video recordings was also documented in an Excel spreadsheet by two independent raters and any disagreements were reconciled by a third independent rater. Laryngeal airway protection responses and self-perceptual ratings of monofilament stimuli were excluded if there was no 10%–30% monofilament buckling or the monofilament of the aesthesiometer could not be presented perpendicular to the surface of the laryngeal tissue (ie, skiving). Sensory perceptual ratings and laryngeal responses to negative (foil) trials were also documented on the spreadsheet.

Perceptual voice-specific symptom severity ratings (0–100-mm VAS)

Tick marks for each presensory and postsensory testing visual analog scale rating of vocal effort, vocal fatigue, vocal tract discomfort, and odynophonia on each respective 100-mm scale were measured with a ruler and inputted into an Excel spreadsheet.

Voice sample acoustics (cepstral peak prominence)

All segments, except for the steady-state vowel /i/, were removed from the voice sample in *Praat* so that only 10–15 seconds of vowel productions remained in each sample. Cepstral peak prominence (CPP) for each voice sample was automatically measured using a customized batch mode script in *Praat* (Phonanium CommV).

Statistical analysis

Mann-Whitney U tests were used to compare groups on severity of perceptual sensations to laryngeal tactile stimuli across monofilament diameters for both positive and foil trials (0–10 Likert Scale), and for Part A of the Central Sensitization Inventory. Fisher exact tests were used to compare groups on laryngeal adductor reflex response rates, presence of other airway protection maneuvers (eg, coughing, swallowing), and Part B of the Central Sensitization Inventory. Independent *t* tests were used to determine group differences on pressure algometry and voice symptom severity ratings of vocal effort, vocal fatigue, vocal tract discomfort, and odynophonia (100-mm VAS). Two-way analysis of variance was used to determine within- and between-group differences on dynamic temporal summation measures for paralaryngeal and hand regions across four timepoints (immediately after the first trial, immediately after the 10th trial, 15 seconds after the 10th trial, and 30 seconds after the 10th trial). All models with multiple comparisons were corrected with Bonferroni corrections to reduce type-I errors. Because there were no significant differences in laterality in the paralaryngeal regions for pressure algometry or dynamic temporal summation, right and left sides were averaged together to obtain one composite number for each paralaryngeal region for correlation analysis. Correlations between voice-specific symptom severity ratings (vocal effort, fatigue, discomfort, and odynophonia on 100-mm VAS), laryngeal sensation (aesthesiometers), paralaryngeal sensation (algometry, 10th trial dynamic temporal summation), hand (10th trial dynamic temporal summation), and leg (algometry) were determined with Pearson's correlations. Chi-square analysis was also conducted on Part A of the Central Sensitization Inventory using 33.5/100 as the cutoff score for central sensitization to determine if there was a difference in prevalence of participants in the pMTD and control groups that met criteria for a central sensitivity disorder.

RESULTS

Perceptual laryngeal sensitivities and laryngeal airway protection responses

Patients with pMTD reported significantly higher laryngeal sensations across the monofilaments compared with the vocally healthy control group ($U = 7984.0$, $P = 0.0072$; Figure 4). The largest group difference was with the thinnest 6-0 monofilament, but monofilament diameter did not have a significant effect ($P > 0.05$). The pMTD group also rated laryngeal sensations during the foil trials as significantly higher than controls ($U = 388.0$, $P = 0.0025$). However, there were no significant group differences on laryngeal adductor reflex responses (odds ratio = 0.685, $P = 0.128$) or other laryngeal airway protection maneuvers, including swallowing and laryngeal guarding (odds ratio = 1.184, $P = 0.444$), across all monofilament diameters (Table 2 for breakdown across monofilament diameters).

Paralaryngeal sensation (pressure algometry and dynamic temporal summation)

There were no significant group differences on mechanosensation with pressure algometry for the submental region corresponding to the base of tongue muscles ($t = 0.131$, $P = 0.897$), left sternocleidomastoid ($t = -0.775$, $P = 0.442$), or right sternocleidomastoid ($t = -0.229$, $P = 0.767$) muscles (Figure 5A and B). There were also no significant group differences on dynamic temporal summation stimulations for TH ($F = 1.945$, $P = 0.165$) and ST regions ($F = 2.049$, $P = 0.154$) (Figure 6). There were significant group differences in dynamic temporal summation ratings for the SH region ($F = 4.39$, $P = 0.038$; Figure 6B).

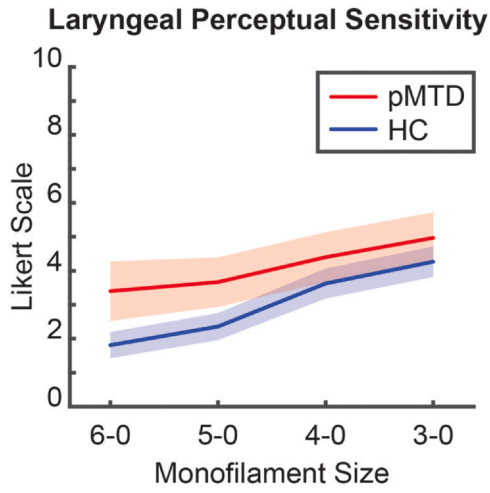

FIGURE 4. Laryngeal perceptual sensitivities on 0–10 Likert Scale across monofilaments (6-0, 5-0, 4-0, and 3-0). Shaded areas, standard error of the mean (SEM); HC, vocally healthy control subjects.

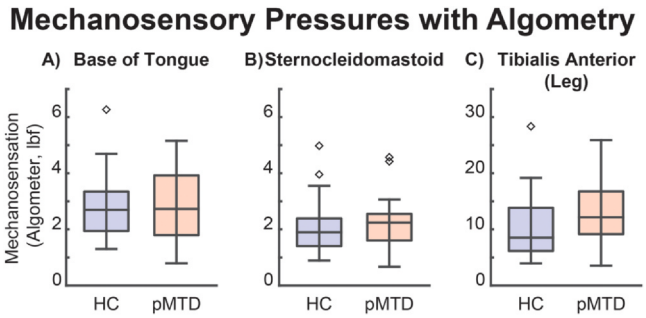

FIGURE 5. Mechanosensory testing with pressure algometry results for **A.** submental area corresponding to the base of tongue, **B.** neck area corresponding with sternocleidomastoid muscles, and **C.** tibialis anterior muscle of the leg between patients with pMTD (pMTD, red) and vocally healthy individuals (HC, blue). Error bars in standard deviation. lbf, pound force applied to an area.

Interestingly, it was the control group that had significantly higher sensations in this region across the four timepoints. As to be expected, the main effect of time was significant across the four timepoints (1st trial, 10th trial, 15 seconds post, 30 seconds post) for each paralaryngeal region ($P < 0.001$). However, there were no significant interactions between group and timepoint, suggesting that the pMTD group did not respond differently to stimulations over time compared with the control group (TH: $F = 0.237$, $P = 0.871$; SH: $F = 0.320$, $P = 0.810$; ST: $F = 2.048$, $P = 0.475$).

Limb sensation (pressure algometry and dynamic temporal summation)

There were no significant group differences on mechanosensation with pressure algometry for the anterior tibialis of the leg ($t = -1.31$, $P = 0.200$; Figure 5C). There were also no group differences on dynamic temporal summation of the hand ($F = 0.946$, $P = 0.332$; Figure 6D). Although there was a significant effect of time on dynamic temporal summation measures of the hand ($F = 56.871$, $P < 0.0001$), there were no significant interactions between group and time ($F = 0.348$, $P = 0.790$).

Voice symptom severity (100-mm VAS)

Vocal effort severity ratings were significantly higher in the pMTD group compared with the control group ($P < 0.0001$). However, there were no significant differences between prelaryngeal and postlaryngeal sensory testing timepoints in either group ($P = 0.790$). Although there were trends toward significance, there were no statistically significant interactions between group and timepoint for vocal effort ($P = 0.063$) (Figure 7A). *Vocal fatigue* severity ratings were significantly higher in the pMTD group compared with the control group ($P < 0.0001$). However, there were no significant differences between prelaryngeal and postlaryngeal sensory testing timepoints in either group ($P = 0.164$), and no significant interactions between group and timepoint ($P = 0.130$) (Figure 7B). *Vocal tract discomfort* severity ratings were significantly higher in the pMTD group compared with the control group ($P < 0.0001$). Patients with pMTD also reported significantly higher vocal tract discomfort after laryngeal sensory testing compared with prelaryngeal sensory testing ($P = 0.0023$). However, there were no significant interactions between group and timepoint ($P = 0.170$) (Figure 7C). *Odynophonia* ratings were significantly higher in the pMTD group compared with the control group ($P < 0.0001$). However, there were no significant differences between prelaryngeal and postlaryngeal sensory testing timepoints between groups ($P = 0.743$), and no group-by-timepoint interactions on the odynophonia parameter ($P = 0.887$) (Figure 7D).

Central sensitization inventory

There were no statistically significant group differences on Part A ($P = 0.111$) or Part B ($P = 0.089$) of the Central Sensitization Inventory. Thirty percent of participants in the control group met criteria for a central sensitization

TABLE 2.
Prevalence of Airway Protection Responses to Laryngeal Sensory Testing

Monofilament Size	Odds Ratio	*P* Value	Present in pMTD Group	Present in Control Group
6-0	1.730	0.418	26%	16%
5-0	1.250	0.656	33%	28%
4-0	0.842	0.826	45%	50%
3-0	0.647	0.390	55%	65%

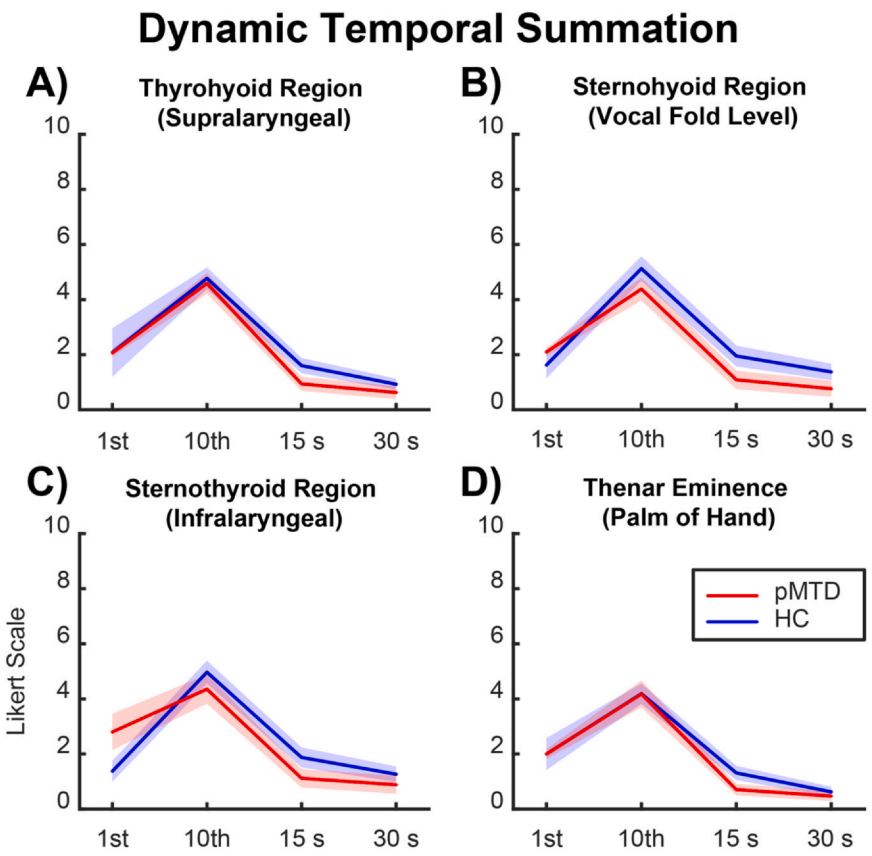

FIGURE 6. Dynamic temporal summation results for areas corresponding to **A.** thyrohyoid space, **B.** sternohyoid muscle, **C.** sternothyroid muscle, and **D.** thenar eminence of the hand. pMTD, primary muscle tension dysphonia (red); HC, vocally healthy control subjects (blue); shaded areas, SEM; 1st, first trial, 10th, tenth trial; 15 s, 15 seconds after 10th trial; 30 s, 30 seconds after 10th trial.

disorder, while 44% of participants in the pMTD group met criteria for central sensitization. There were no significant differences in prevalence of central sensitization between the two groups, using 33.5 as the cutoff score ($X^2 = 0.113$, $P = 0.737$).

Acoustic vocal quality (CPP)

There were no significant group differences on CPP vocal acoustics ($P = 0.900$) recorded before sensory testing and timepoint (pre-postsensory testing) did not influence CPP measures ($P = 0.889$). Interestingly, a subset of subjects (n = 10 controls, n = 9 pMTD) were asked to rate *their* self-perceived severity of vocal quality before and after laryngeal sensory testing. Patients with pMTD reported significantly worse vocal quality than the control group ($P = 0.013$).

Correlations across laryngeal, paralaryngeal, and global sensations and voice-specific symptom severity ratings

There were largely no significant correlations between severity of voice-specific symptoms (vocal effort, fatigue, discomfort, and odynophonia) and laryngeal, paralaryngeal, and global sensations for either pMTD (Table 3) or control (Table 4) groups. The three exceptions, all in the pMTD group, were significant positive correlations between (1) severity of vocal tract discomfort and SH region (vocal fold level) sensitivity, (2) severity of vocal tract discomfort and hand sensitivity, and (3) odynophonia and leg mechanosensation. The pMTD group also had significant positive correlations between vocal fatigue and vocal effort, as well as odynophonia and vocal tract discomfort. The control group had significant positive correlations between vocal fatigue and vocal tract discomfort, odynophonia and vocal tract discomfort, and odynophonia and vocal fatigue.

DISCUSSION

The relationships between voice symptoms and laryngeal, paralaryngeal, and global somatosensation in patients with pMTD have not been elucidated, creating challenges for mechanistic-based diagnostics and management of pMTD. The need to understand the role of the somatosensory system in pMTD is especially important considering the vital role this sensory system plays in vocal productions[76–78] and the recent studies that have suggested the somatosensory system may have more to do with pMTD than laryngeal motor tension or hyperfunction patterns.[9,10,18] To address this knowledge gap and provide a better understanding of the underlying mechanisms to

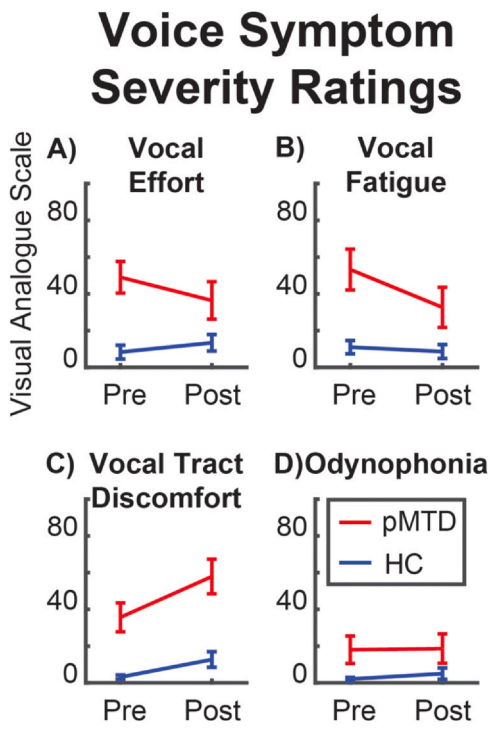

FIGURE 7. Voice-specific symptom severity ratings for **A.** vocal effort, **B.** vocal fatigue, **C.** vocal tract discomfort, and **D.** odynophonia based on 100-mm visual analog scales. pMTD, primary muscle tension dysphonia (red); HC, vocally healthy control subjects (blue). Error bars: SEM.

facilitate optimization of voice assessment and treatment approaches for patients with pMTD, laryngeal, paralaryngeal, and global somatosensation were first compared between individuals with and without pMTD. Relationships between common pMTD voice symptoms and laryngeal, paralaryngeal, and global somatosensory severities were also compared. Patients with pMTD (1) had significantly heightened perceptions of laryngeal tactile stimuli, (2) reported significantly more severe voice-specific symptoms presensory and postsensory testing, and (3) had significantly higher ratings of vocal tract discomfort postsensory testing, compared with presensory testing. However, there were no significant (1) group differences on prevalence of laryngeal motor patterns of hypersensitivity patterns (eg, laryngeal adductor reflex, laryngeal guarding), (2) group differences on paralaryngeal or global mechanosensation, (3) group differences on central sensitivity measures, or (4) correlations between perceived laryngeal sensation and self-reported voice-specific symptoms.

Higher perceptions of laryngeal sensations to tactile stimulation in patients with pMTD

Patients with pMTD reported significantly higher sensations to laryngeal tactile stimuli than control participants. Considering the laryngeal somatosensory system is involved in voice production,[41,76–78] it is not surprising that this system would play a role in a voice disorder that occurs with vocal productions. Interestingly, although there were significant group differences in the *perception* of laryngeal sensitivities, there were no significant differences in the *prevalence* of laryngeal motor response patterns indicative of hypersensitivities (eg, laryngeal adductor reflexes, laryngeal guarding). These findings suggest the subjective appraisal of laryngeal sensation—a higher-order process and perceptual construct—may better define pMTD than lower-order peripheral sensitivities at the end organ as reflected by brainstem-mediated sensitivity responses.

In addition to greater perceptions of laryngeal tactile stimuli, patients with pMTD also had significantly higher false-positive responses to foil trials (ie, when no tactile stimulation was presented to the larynx). There could be several reasons for these observations. Higher somatosensory appraisal might involve greater engagement of top-down cognitive processes, such as anticipation and expectation. These cognitive processes can modulate sensory processing, making individuals more responsive to stimuli that they are primed to detect, even if those stimuli are part of a foil trial. A second reason could have to do with psychological factors. Patients with higher somatosensory appraisal might also have heightened vigilance related to sensory experiences. This increased psychological arousal can enhance their perceptual responses to tactile stimuli, including those in foil trials. Previous work has eluded to relationships between stress responses and pMTD clinical presentations. However, the majority of these studies included only vocally healthy cohorts, were retrospective, or were case studies with small sample sizes.[79–82] Additional investigations into the relationships between somatosensation, higher-order sensory processing, and psychological factors (eg, anxiety, stress) are needed to determine the exact mechanisms underlying these clinical presentations.

A third reason could have to do with how sensory information is processed, such as differences in neural pathways or neurotransmitter levels related to sensory processing. A fourth reason could have to do with reduced somatosensory discrimination acuity in this cohort. Patients with pMTD in this study often required additional coaching to help them discriminate between sensations created by the laryngoscope in the nasal cavity from sensations in the larynx with monofilament stimulation. The inability to distinguish sensations between the nose and larynx (or other areas of the vocal tract, as suggested by the significantly higher vocal tract discomfort ratings in the pMTD group postsensory testing) could have confounded perceptions of tactile stimuli targets. The need for coaching to improve somatosensory acuity could be one reason why voice therapy techniques provided by a licensed clinician (ie, the coach) can be successful. Incorporation of voice therapies that require the clinician to coach patients to feel resonance in the nasal and oral cavities instead of the throat (ie, somatosensory discrimination acuity) or increase airflow through the vocal tract (eg, stretch-and-flow phonation) are some of the best empirically supported therapeutic approaches for patients with hyperfunctional voice disorders.[83–85]

TABLE 3.
pMTD Group Pearson's Correlations

	Effort VAS	Fatigue VAS	Disc. VAS	Odyn. VAS	TE DTS	TH DTS	SH DTS	ST DTS	BOT Algo.	SCM Algo.	TA Algo.	6-0 Mono.	5-0 Mono.	4-0 Mono.	3-0 Mono.	6-0 Foil	5-0 Foil	4-0 Foil	3-0 Foil	CSI-A
Effort VAS	1.00	0.86*	0.25	0.23	0.00	−0.06	−0.11	0.01	−0.04	0.03	−0.27	0.10	0.08	0.16	0.24	−0.14	0.57	0.15	−0.04	0.19
Fatigue VAS	0.86*	1.00	0.23	0.18	−0.05	−0.06	−0.19	−0.04	0.02	0.09	−0.26	−0.17	−0.41	−0.07	−0.13	−0.47	0.10	−0.12	−0.40	0.38
Disc. VAS	0.25	0.23	1.00	0.83*	0.75†	0.66	0.72†	0.51	−0.15	0.49	0.63	−0.10	−0.18	0.15	0.06	−0.37	−0.15	0.22	−0.05	−0.19
Odyn. VAS	0.23	0.18	0.83*	1.00	0.64	0.60	0.61	0.31	0.23	0.51	0.79†	0.18	−0.10	0.31	0.40	−0.33	0.17	0.09	−0.06	−0.05
TE DTS	0.00	−0.05	0.75†	0.64	1.00	0.92‡	0.57†	0.37	−0.47	−0.20	0.14	0.31	0.23	0.19	−0.18	0.22	−0.06	−0.13	−0.41	−0.64
TH DTS	−0.06	−0.06	0.66	0.60	0.92‡	1.00	0.74§	0.60†	−0.38	−0.22	0.04	0.40	0.35	0.20	−0.07	0.32	−0.09	0.02	−0.29	−0.48
SH DTS	−0.11	−0.19	0.72†	0.61	0.57	0.74*	1.00	0.86‡	−0.19	−0.16	−0.07	0.26	0.35	−0.01	0.11	0.31	−0.02	0.25	0.06	−0.43
ST DTS	0.01	−0.04	0.51	0.31	0.37	0.60†	0.86‡	1.00	−0.23	−0.20	−0.24	0.01	0.21	−0.14	0.19	−0.04	−0.17	0.66	0.57	0.24
BOT Algo.	−0.04	0.02	−0.15	0.23	−0.47	−0.38	−0.19	−0.23	1.00	0.74*	0.48	0.13	0.15	0.29	0.55	−0.14	0.29	−0.36	0.02	0.48
SCM Algo.	0.03	0.09	0.49	0.51	−0.20	−0.22	−0.16	−0.20	0.74§	1.00	0.71*	−0.12	0.03	0.35	0.32	−0.48	−0.10	−0.57	−0.20	0.05
TA Algo.	−0.27	−0.26	0.63	0.79†	0.14	0.04	−0.07	−0.24	0.48	0.71*	1.00	0.00	−0.08	0.18	0.23	−0.21	−0.09	−0.29	−0.14	−0.19
6-0 Mono.	0.10	−0.17	−0.10	0.18	0.31	0.40	0.26	0.01	0.13	−0.12	0.00	1.00	0.80§	0.69*	0.28	0.83†	0.57	0.16	−0.05	−0.42
5-0 Mono.	0.08	−0.41	−0.18	−0.10	0.23	0.35	0.35	0.21	0.15	0.03	−0.08	0.80§	1.00	0.73*	0.51	0.96§	0.71	0.36	0.29	−0.58
4-0 Mono.	0.16	−0.07	0.15	0.31	0.19	0.20	−0.01	−0.14	0.29	0.35	0.18	0.69*	0.73*	1.00	0.51	0.43	0.30	−0.03	−0.03	−0.13
3-0 Mono.	0.24	−0.13	0.06	0.40	−0.18	−0.07	0.11	0.19	0.55	0.32	0.23	0.28	0.51	0.51	1.00	−0.07	0.61	0.65	0.70	0.48
6-0 Foil	−0.14	−0.47	−0.37	−0.33	0.22	0.32	0.31	−0.04	−0.14	−0.48	−0.21	0.83†	0.96§	0.43	−0.07	1.00	0.51	0.14	0.03	−0.75
5-0 Foil	0.57	0.10	−0.15	0.17	−0.06	−0.09	−0.02	−0.17	0.29	−0.10	−0.09	0.57	0.71	0.30	0.61	0.51	1.00	0.53	0.26	−0.27
4-0 Foil	0.15	−0.12	0.22	0.09	−0.13	0.02	0.25	0.66	−0.36	−0.57	−0.29	0.16	0.36	−0.03	0.65	0.14	0.53	1.00	0.89†	0.46
3-0 Foil	−0.04	−0.40	−0.05	−0.06	−0.41	−0.29	0.06	0.57	0.02	−0.20	−0.14	−0.05	0.29	−0.03	0.70	0.03	0.26	0.89†	1.00	0.42
CSI-A	0.19	0.38	−0.19	−0.05	−0.64	−0.48	−0.43	0.24	0.48	0.05	−0.19	−0.42	−0.58	−0.13	0.48	−0.75	−0.27	0.46	0.42	1.00

VAS, 100-mm visual analog scale; Disc., discomfort; Odyn., odynophonia; DTS, dynamic temporal summation; Algo., algometry; mono., monofilament (aesthesiometer); foil, negative foil aesthesiometer trials; TE, thenar eminence (hand); TH, thyrohyoid; SH, sternohyoid; ST, sternothyroid; BOT, base of tongue; SCM, sternocleidomastoid; TA, tibialis anterior (leg); CSI-A, Central Sensitization Inventory-Part A.

* Correlation is significant at the 0.01 level (2-tailed).

† Correlation is significant at the 0.05 level (2-tailed).

‡ Correlation is significant at 0.0001 level (2-tailed).

§ Correlation is significant at the 0.001 level (2-tailed).

TABLE 4.
Control Group Pearson's Correlations.

	Effort VAS	Fatigue VAS	Disc. VAS	Odyn. VAS	TE DTS	TH DTS	SH DTS	ST DTS	BOT Algo.	SCM Algo.	TA Algo.	6-0 Mono.	5-0 Mono.	4-0 Mono.	3-0 Mono.	6-0 Foil	5-0 Foil	4-0 Foil	3-0 Foil	CSI-A
Effort VAS	1.00	0.26	0.20	0.16	−0.15	−0.12	−0.13	−0.12	0.02	0.08	0.01	0.28	0.40	0.29	0.38	0.08	−0.05	0.06	0.14	−0.027
Fatigue VAS	0.26	1.00	0.53*	0.62*	−0.10	−0.05	0.01	0.00	−0.03	−0.09	−0.25	0.08	−0.05	0.19	0.31	−0.14	0.10	0.03	0.08	0.256
Disc. VAS	0.21	0.53*	1.00	0.84†	−0.01	0.06	0.10	0.15	−0.24	−0.20	−0.11	−0.26	−0.31	−0.11	0.08	−0.30	−0.22	−0.27	−0.13	0.207
Odyn. VAS	0.16	0.63*	0.84†	1.00	−0.03	0.05	0.01	0.09	−0.25	−0.08	−0.10	−0.21	−0.27	−0.06	0.18	−0.23	−0.11	−0.08	0.04	0.233
TE DTS	−0.15	−0.10	−0.01	−0.03	1.00	0.90†	0.78†	0.76†	−0.17	−0.18	0.11	0.37	0.51‡	0.36	0.24	0.14	−0.19	−0.33	−0.59	0.231
TH DTS	−0.12	−0.05	0.06	0.05	0.90†	1.00	0.93†	0.91†	−0.33	−0.35‡	−0.07	0.34	0.51	0.40	0.28	0.34	0.10	−0.42	−0.49	0.329
SH DTS	−0.13	0.01	0.10	0.01	0.78†	0.93†	1.00	0.94†	−0.28	−0.32	−0.07	0.27	0.44	0.28	0.22	0.30	0.21	−0.37	−0.39	0.333
ST DTS	−0.12	0.00	0.15	0.09	0.76†	0.91†	0.94†	1.00	−0.31	−0.39‡	−0.08	0.23	0.35	0.26	0.27	0.29	0.16	−0.33	−0.34	0.347
BOT Algo.	0.02	−0.03	−0.24	−0.25	−0.17	−0.33	−0.28	−0.31	1.00	0.84†	0.81†	−0.37	−0.32	−0.36	−0.38	−0.36	−0.32	0.14	0.00	−0.157
SCM Algo.	0.08	−0.09	−0.20	−0.08	−0.18	−0.35‡	−0.32	−0.39‡	0.84†	1.00	0.80†	−0.34	−0.41	−0.32	−0.30	−0.43	−0.41	0.16	−0.07	−0.226
TA Algo.	0.01	−0.25	−0.11	−0.10	0.11	−0.07	−0.07	−0.08	0.81†	0.80†	1.00	−0.21	−0.25	−0.21	−0.20	−0.33	−0.41	0.24	0.02	−0.144
6-0 Mono.	0.28	0.08	−0.26	−0.21	0.37	0.34	0.27	0.23	−0.37	−0.34	−0.21	1.00	0.67*	0.54‡	0.34	0.55	0.20	−0.13	0.10	0.420
5-0 Mono.	0.40	−0.05	−0.31	−0.27	0.51‡	0.51‡	0.44	0.35	−0.32	−0.41	−0.25	0.67	1.00	0.47	0.20	0.72*	0.38	−0.44	−0.09	0.485
4-0 Mono.	0.29	0.19	−0.11	−0.06	0.38	0.40	0.28	0.26	−0.36	−0.32	−0.21	0.54‡	0.47	1.00	0.88†	−0.14	−0.11	0.36	0.27	−0.048
3-0 Mono.	0.38	0.31	0.08	0.18	0.24	0.28	0.22	0.27	−0.38	−0.30	−0.20	0.34	0.20	0.88†	1.00	0.08	0.10	0.68‡	0.64‡	0.046
6-0 Foil	0.08	−0.18	−0.29	−0.23	0.14	0.34	0.30	0.29	−0.36	−0.43	−0.33	0.55	0.72*	−0.14	0.08	1.00	0.77*	−0.05	0.57	0.543
5-0 Foil	−0.05	0.10	−0.22	−0.11	−0.19	0.10	0.21	0.16	−0.32	−0.41	−0.41	0.20	0.38	−0.11	0.10	0.77*	1.00	−0.11	0.57	0.428
4-0 Foil	0.06	0.03	−0.27	−0.08	−0.33	−0.42	−0.37	−0.33	0.14	0.16	0.24	−0.13	−0.44	0.36	0.67‡	−0.05	−0.11	1.00	1.00†	−0.320
3-0 Foil	0.14	0.08	−0.13	0.04	−0.59	−0.49	−0.39	−0.34	0.00	−0.07	0.02	0.10	−0.09	0.27	0.64‡	0.57	0.57	1.00†	1.00	0.092
CSI-A	−0.03	0.26	0.21	0.23	0.23	0.33	0.33	0.35	−0.16	−0.23	−0.14	0.42	0.49	−0.05	0.05	0.54	0.43	−0.32	0.09	1.00

VAS, 100-mm visual analog scale; Disc., discomfort; Odyn., odynophonia; DTS, dynamic temporal summation; Algo., algometry; mono., monofilament (aesthesiometer); foil, negative foil aesthesiometer trials; TE, thenar eminence (hand); TH, thyrohyoid; SH, sternohyoid; ST, sternothyroid; BOT, base of tongue; SCM, sternocleidomastoid; TA, tibialis anterior (leg); CSI-A, Central Sensitization Inventory-Part A.

§Correlation is significant at the 0.001 level (2-tailed).

* Correlation is significant at the 0.01 level (2-tailed).

† Correlation is significant at 0.0001 level (2-tailed).

‡ Correlation is significant at the 0.05 level (2-tailed).

Higher voice symptom ratings in patients with pMTD

Patients with pMTD rated all of their voice-specific symptoms (effort, fatigue, discomfort, and odynophonia) as significantly higher than vocally healthy controls. These findings parallel previous studies that found significantly higher ratings of vocal effort and vocal tract discomfort in patients with pMTD, compared with vocally healthy cohorts, even when laryngeal and paralaryngeal muscle tension and hyperfunctional motor patterns were similar between groups.[86–88] There were also significantly higher ratings of vocal tract discomfort at the postlaryngeal sensory testing timepoint, compared with the pretesting timepoint, that occurred in the pMTD group but not the control group in the present study. These findings may explain, at least in part, why patients with pMTD experience symptoms for longer and why symptoms may persist after extinguishment of the original stimulus or instigating event (eg, upper respiratory infections, increased vocal demands).

No significant group differences in paralaryngeal mechanosensation, limb mechanosensation, or central sensitization

Patients with pMTD did not perceive more mechanosensation than the control group or exhibit significant group differences on central sensitization measures. These findings suggest differences in sensory experiences may be specific to the larynx and may not be present externally, or the result of central sensitization. The results of this study contrast with previous retrospective studies that found a higher prevalence of central sensitization disorders, including fibromyalgia, chronic pain, and irritable bowel disorders in patients with pMTD.[38,89] The implications of this study are that although some patients with functional voice disorders *can* have comorbid central sensitization disorders, the presence of this comorbidity is not a central feature in patients with pMTD.

No correlations between perceived laryngeal stimuli severities and voice-specific symptoms

There were no significant relationships between levels of perceived laryngeal stimuli severities with aesthesiometer testing and severity ratings of vocal effort, fatigue, discomfort, and odynophonia on 100-mm VAS. There are several potential reasons for these findings. The first is that these outcomes may be targeting different underlying physiological or perceptual constructs. Another reason may have to do with the timing of when sensations and symptoms are experienced and reported. For example, while laryngeal sensations during sensory testing might be triggered by specific events, vocal symptoms like vocal fatigue and effort may build over time or during specific uses of the voice, leading to nonaligned reporting and poor correlations.

The possibility of a central sensitization or hypersensitivity phenotype in patients with pMTD

It is important to note that although there were no significant correlations between laryngeal stimuli severities and voice-specific symptoms, patients with pMTD did have significant correlations between vocal tract discomfort and global sensitivity ratings. These data may suggest a central sensitivity phenotype in some individuals with pMTD where more sensory input to the body globally correlates with greater vocal tract discomfort. Furthermore, although not significant, there was a slightly higher prevalence of certain laryngeal airway protection responses (eg, swallowing, laryngeal guarding) in the pMTD group (odds ratio = 1.184). These airway protection maneuvers could either be a *volitional behavioral* response (eg, swallowing in an attempt to alleviate perceived sensations in the larynx) or specific to a *subcohort* of patients with pMTD. These clinical presentations in some individuals with pMTD could, in part, explain some of the high variability and heterogeneity of clinical presentations common in the pMTD population. However, because there were no differences between groups, these voice-specific clinical presentations and their relationships to central sensitivities are not central to the pMTD diagnosis.

Limitations and future directions

Several limitations of this study are worth mentioning. The first is that not everyone had the nasal anatomy to easily pass a channeled laryngoscope with minimal discomfort. The decision to be conservative with inclusion criteria for passing the laryngoscope was based on potential confounding issues with conflicting somatosensory feedback between the laryngoscope in the nose and sensory testing in the larynx. This phenomenon, known as *conditioned pain modulation*, involves a process where a pain stimulus can diminish the perception of another sensory stimulus.[90] In other words, too much pain in the nose could influence somatosensory perceptions to tactile stimulations in the larynx. This phenomenon could potentially skew results, especially if these anatomical difficulties occurred more often in one group than the other. However, all subjects included in the laryngeal sensory testing data analysis cohort experienced low levels of nasal discomfort (as per inclusion criteria). Participants were instructed to report any pain experienced when passing the laryngoscope and the protocol was terminated if participants reported pain or if the laryngoscope could not be easily passed through the nasopharynx (as per exclusion criteria). Because the prevalence of subjects who could not tolerate the laryngoscope was similar between groups (36% control vs 25% pMTD), it is highly unlikely that this factor confounded study findings.

The second limitation is that measurements of laryngeal sensitivities are limited by the accuracy of aesthesiometer testing and ability to provide reliable tactile stimuli to specific laryngeal sites via the aesthesiometers. Aesthesiometers were initially presented to the following laryngeal regions:

arytenoids, epiglottis, ventricular folds, pyriform sinuses, and aryepiglottic folds. The arytenoids, epiglottis, and ventricular folds were too sensitive for laryngeal sensory testing in vocally healthy controls during initial protocol optimization. These laryngeal regions were subsequently excluded from the laryngeal sensory testing protocol, especially in the context of potential hypersensitivities in the pMTD cohort. It was also challenging to consistently apply aesthesiometer buckling forces without skivvying the monofilaments to the lateral pyriform sinuses. As such, monofilament forces were only applied randomly to either the right or left aryepiglottic fold for optimal consistency and precision. Further investigations utilizing sensory testing methods with greater precision are needed.

Different laryngeal regions are innervated by different branches of the superior laryngeal nerve (superior, middle, and inferior internal branches).[91] It is therefore possible that because laryngeal testing did not cover all three branches of the internal superior laryngeal nerve, the omission of sensory testing across these different laryngeal sites could have resulted in a missed opportunity for a more comprehensive investigation of the laryngeal somatosensory system. However, because the methods applied were consistent between groups, omission of laryngeal sites should not impact group comparison findings. Considering these limitations, future work should focus on validation of alternative approaches with greater sensitivity and precision for more comprehensive laryngeal sensory testing.

The third limitation is the lack of concrete anchoring with laryngeal sensation ratings on the 0–10 Likert scale. Participants were initially presented with a 3-0 monofilament to the midline lower lip until 10%–30% buckling of the monofilament had been reached, which served as an anchor of 10 on the Likert scale, per previous methods.[92] However, subjects were often confused by these anchors because sensations on the lip substantially differ from sensations in the larynx. As such, we ended up basing the 0–10 scale on relative sensations (eg, 0 = no sensation; 3 = light sensation; 5 = uncomfortable but tolerable; 8 = barely tolerable; Figure 2). Although this method alleviated some confusion, patients with pMTD continued to have more difficulties ascribing numbers representing laryngeal sensory testing intensity levels on the 0–10 scale compared with the controls. It is possible that the reason for this could have to do with differences in somatosensory acuity in patients with pMTD, which could be assessed using two-point discrimination testing at laryngeal structures. Future investigations on the role of somatosensory acuity in pMTD symptomology are needed.

A fourth limitation is related to the procedure for quantitative sensory testing. Our protocol only focused on static and dynamic testing of Aδ and C nociceptive fibers, both locally and globally. A more comprehensive protocol could have included static testing of Aβ fibers (eg, vibration) to inform potential deficits in large-fiber somatosensory discrimination acuity, as well as a dynamic measure of conditioned pain modulation (eg, cold pressor test) to provide insight into the role of top-down cognitive processing in somatosensory appraisal. Finally, the protocol for our dynamic measure of temporal summation was condensed to one trial for each site due to the number of sites tested. Future studies may condense the protocol to only include those sites showing potential for between-group differences or correlation to voice severity (eg, SH and hand), which would then allow for up to four additional trials to be added at each site, since windup is a frequency-dependent increase in excitability that reaches a plateau after about five stimuli.[93]

CONCLUSION

Patients with pMTD perceive laryngeal sensations as more intense than vocally healthy controls and have significantly higher levels of vocal effort, vocal fatigue, vocal tract discomfort, and odynophonia. The lack of group differences on prevalence of laryngeal motor airway protection responses suggestive of hypersensitivities, peripheral mechanosensory, and central sensitivity measures in the perilarynx and limbs suggests these mechanisms may not play a central role in pMTD pathophysiology, and that heightened sensory perceptions or appraisals may be specific to the larynx. Finally, the lack of significant correlations between perceived somatosensory laryngeal stimuli and voice symptom severity ratings suggests these parameters target distinct underlying mechanistic constructs, and as such, their relationships require further investigation.

Declaration of Competing Interest

The authors have no conflicts of interest to disclose.

Acknowledgments

Thanks to Harsha Haridas, Maxwell Ugochukwu, Gauri Surendran, Asma Nasir, and Kayla Coreno for constructing the laryngeal aesthesiometers, Lesley Childs and Laura Toles for help with recruitment, Shumon Dhar for conducting the laryngeal sensory testing with the aesthesiometers, and Youri Maryn at Phonanium CommV for providing the customized *Praat* script for acoustic analysis.

References

1. Roy N, Merrill RM, Gray SD, et al. Voice disorders in the general population: prevalence, risk factors, and occupational impact. *Laryngoscope.* 2005;115:1988–1995. https://doi.org/10.1097/01.mlg.0000179174.32345.41.PMID: 16319611.
2. Cohen SM, Kim J, Roy N, et al. Factors influencing the health care expenditures of patients with laryngeal disorders. *Otolaryngol Head Neck Surg.* 2012;147:1099–1107. https://doi.org/10.1177/0194599812454838.
3. Bonetti A, Bonetti L, Čipčić O. Self-assessment of vocal fatigue in muscle tension dysphonia and vocal nodules: a preliminary analysis of the discriminatory potential of the Croatian Adaptation of the Vocal Fatigue Index (VFI-C). *J Voice.* 2019;35:325.e1–325.e15. https://doi.org/10.1016/j.jvoice.2019.08.028.

4. Van Houtte E, Van Lierde K, Claeys S. Pathophysiology and treatment of muscle tension dysphonia: a review of the current knowledge. *J Voice.* 2011;25:202–207.
5. McDowell S, Morrison R, Mau T, et al. Clinical characteristics and effects of vocal demands in occupational voice users with and without primary muscle tension dysphonia. *J Voice.* 2022. https://doi.org/10.1016/j.jvoice.2022.10.005.
6. Shembel AC, Lee J, Sacher JR, et al. Characterization of primary muscle tension dysphonia using acoustic and aerodynamic voice metrics. *J Voice.* 2021;37:897–906. https://doi.org/10.1016/j.jvoice.2021.05.019.
7. Morrison MD, Rammage LA. Muscle misuse voice disorders: description and classification. *Acta Otolaryngol.* 1993;113:428–434. https://doi.org/10.3109/00016489309135839.
8. Spencer ML. Muscle tension dysphonia: a rationale for symptomatic subtypes, expedited treatment, and increased therapy compliance. *Perspect Voice Voice Dis.* 2015;25:5–15. https://doi.org/10.1044/vvd25.1.5.
9. Shembel AC, Morrison RA, Fetzer DT, et al. Extrinsic laryngeal muscle tension in primary muscle tension dysphonia with shear wave elastography. *Laryngoscope.* 2023;133:3482–3491. https://doi.org/10.1002/lary.30830.
10. Morrison RA, Fetzer DT, Patterson-Lachowicz A, et al. Optical flow analysis of paralaryngeal muscle movement. *Laryngoscope.* 2024;134:1792–1801. https://doi.org/10.1002/lary.31063.
11. Hogue S, Guo X, Morrison RA, et al. Use of motion capture technology to study extrinsic laryngeal muscle tension and hyperfunction. *Laryngoscope.* 2023;133:3472–3481. https://doi.org/10.1002/lary.30829.
12. Crocker C, Toles LE, Morrison RA, et al. Relationships between vocal fold adduction patterns, vocal acoustic quality, and vocal effort in individuals with and without hyperfunctional voice disorders. *J Voice.* 2024.
13. Shembel AC, Morrison RA, McDowell S, et al. Relationships between laryngoscopic analysis metrics of supraglottic compression and vocal effort in primary muscle tension dysphonia. *J Voice.* 2023.
14. Sama A, Carding PN, Price S, et al. The clinical features of functional dysphonia. *Laryngoscope.* 2001;111:458–463. https://doi.org/10.1097/00005537-200103000-00015.
15. Behrman A, Dahl LD, Abramson AL, et al. Anterior-posterior and medial compression of the supraglottis: signs of nonorganic dysphoniaor normal postures? *J Voice.* 2003;17:403–410.
16. Jacobson BH, Johnson A, Grywalski C, et al. The voice handicap index (VHI): development and validation. *Am J Speech Lang Pathol.* 1997;6:66.
17. Nanjundeswaran C, Jacobson BH, Gartner-Schmidt J, et al. Vocal fatigue index (VFI): development and validation. *J Voice.* 2015;29:433–440. https://doi.org/10.1016/j.jvoice.2014.09.012.
18. Smeltzer JC, Chiou SH, Shembel AC. Interoception, voice symptom reporting, and voice disorders. *J Voice.* 2023. https://doi.org/10.1016/j.jvoice.2023.03.002.
19. Brooks J, Allen TJ, Proske U. The senses of force and heaviness at the human elbow joint. *Exp Brain Res.* 2013;226:617–629.
20. Lafargue G, Paillard J, Lamarre Y, et al. Production and perception of grip force without proprioception: is there a sense of effort in deafferented subjects? *Eur J Neurosci.* 2003;17:2741–2749.
21. Luu BL, Day BL, Cole JD, et al. The fusimotor and reafferent origin of the sense of force and weight. *J Physiol.* 2011;589:3135–3147.
22. Sanes JN, Shadmehr R. Sense of muscular effort and somesthetic afferent information in humans. *Can J Physiol Pharmacol.* 1995;73:223–233.
23. Slobounov S, Hallett M, Newell KM. Perceived effort in force production as reflected in motor-related cortical potentials. *Clin Neurophysiol.* 2004;115:2391–2402.
24. Kuppuswamy A. The neurobiology of pathological fatigue: new models, new questions. *Neuroscientist.* 2022;28:238–253. https://doi.org/10.1177/1073858420985447.
25. Arendt-Nielsen L, Morlion B, Perrot S, et al. Assessment and manifestation of central sensitisation across different chronic pain conditions. *Eur J Pain.* 2018;22:216–241.
26. Gur A, Oktayoglu P. Central nervous system abnormalities in fibromyalgia and chronic fatigue syndrome: new concepts in treatment. *Curr Pharm Des.* 2008;14:1274–1294.
27. Kindler LL, Bennett RM, Jones KD. Central sensitivity syndromes: mounting pathophysiologic evidence to link fibromyalgia with other common chronic pain disorders. *Pain Manag Nurs.* 2011;12:15–24.
28. Meeus M, Nijs J. Central sensitization: a biopsychosocial explanation for chronic widespread pain in patients with fibromyalgia and chronic fatigue syndrome. *Clin Rheumatol.* 2007;26:465–473.
29. Nijs J, Meeus M, Van Oosterwijck J, et al. In the mind or in the brain? Scientific evidence for central sensitisation in chronic fatigue syndrome. *Eur J Clin Invest.* 2012;42:203–212.
30. Phillips K, Clauw DJ. Central pain mechanisms in chronic pain states–maybe it is all in their head. *Best Pract Res Clin Rheumatol.* 2011;25:141–154.
31. Bendtsen L. Central and peripheral sensitization in tension-type headache. *Curr Sci Inc.* 2003;7:460–465. https://doi.org/10.1007/s11916-003-0062-9.
32. Edelmayer RM, Le LN, Yan J, et al. Activation of TRPA1 on dural afferents: a potential mechanism of headache pain. *PAIN®.* 2012;153:1949–1958.
33. Yan J, Edelmayer RM, Wei X, et al. Dural afferents express acid-sensing ion channels: a role for decreased meningeal pH in migraine headache. *PAIN®.* 2011;152:106–113.
34. Yan J, Melemedjian OK, Price TJ, et al. Sensitization of dural afferents underlies migraine-related behavior following meningeal application of interleukin-6 (IL-6). *Molecular Pain.* 2012;8:1744–8069.
35. Morrison M, Rammage L. The irritable larynx syndrome as a central sensitivity syndrome. *Can J Speech-Language Pathol Audiol.* 2010;34.
36. Vertigan AE, Bone SL, Gibson PG. Laryngeal sensory dysfunction in laryngeal hypersensitivity syndrome. *Respirology.* 2013;18:948–956. https://doi.org/10.1111/resp.12103.
37. Morrison M, Rammage L, Emami AJ. The irritable larynx syndrome. *J Voice.* 1999;13:447–455.
38. Misono S, Dietrich M, Piccirillo JF. The puzzle of medically unexplained symptoms—a holistic view of the patient with laryngeal symptoms. *JAMA Otolaryngol Head Neck Surg.* 2020;146:550–551. https://doi.org/10.1001/jamaoto.2020.0559.
39. Verdolini K, Titze IR, Fennell A. Dependence of phonatory effort on hydration level. *J Speech Hear Res.* 1994;37:1001–1007. https://doi.org/10.1044/jshr.3705.1001.
40. Lopes LW, de Oliveira Florencio V, Silva POC, et al. Vocal tract discomfort scale (VTDS) and voice symptom scale (VoiSS) in the evaluation of patients with voice disorders. *J Voice.* 2019;33:381.e23–381.e32. https://doi.org/10.1016/j.jvoice.2017.11.018.
41. Strohl MP, Young VN, Dwyer CD, et al. Novel adaptation of a validated tactile aesthesiometer to evaluate laryngopharyngeal sensation. *Laryngoscope.* 2021;131:1324–1331. https://doi.org/10.1002/lary.28947.
42. Kaneoka A, Pisegna JM, Krisciunas GP, et al. Variability of the pressure measurements exerted by the tip of laryngoscope during laryngeal sensory testing: a clinical demonstration. *Am J Speech-Lang Pathol.* 2017;26:729–736. https://doi.org/10.1044/2017_AJSLP-16-0006.
43. Kaneoka A, Krisciunas GP, Walsh K, et al. A comparison of 2 methods of endoscopic laryngeal sensory testing: a preliminary study. *Ann Otol Rhinol Laryngol.* 2015;124:187–193.
44. Strohl MP, Chang JL, Dwyer CD, et al. Laryngeal adductor reflex movement latency following tactile stimulation. *Otolaryngol Head Neck Surg.* 2022;166:720–726. https://doi.org/10.1177/01945998211025517.
45. Domer AS, Kuhn MA, Belafsky PC. Neurophysiology and clinical implications of the laryngeal adductor reflex. *Curr Otorhinolaryngol Rep.* 2013;1:178–182. https://doi.org/10.1007/s40136-013-0018-5.
46. Sinclair CF, Téllez MJ, Ulkatan S. Human laryngeal sensory receptor mapping illuminates the mechanisms of laryngeal adductor reflex control. *Laryngoscope.* 2018;128:E365–E370. https://doi.org/10.1002/lary.27248.
47. Price DD. Central neural mechanisms that interrelate sensory and affective dimensions of pain. *Mol Interv.* 2002;2:392. https://doi.org/10.1124/mi.2.6.392.

48. Young VN, Kidane J, Gochman GE, et al. Abnormal laryngopharyngeal sensation in adductor laryngeal dystonia compared to healthy controls. *Laryngoscope.* 2022;133:2271–2278. https://doi.org/10.1002/lary.30462.
49. Fife TA, Butler SG, Langmore SE, et al. Use of topical nasal anesthesia during flexible endoscopic evaluation of swallowing in dysphagic patients. *Ann Otol Rhinol Laryngol.* 2015;124:206–211. https://doi.org/10.1177/0003489414550153.
50. Johnson PE, Belafsky PC, Postma GN. Topical nasal anesthesia and laryngopharyngeal sensory testing: a prospective, double-blind crossover study. *Ann Otol Rhinol Laryngol.* 2003;112:14–16. https://doi.org/10.1177/000348940311200104.
51. Kostek M, Polaski A, Kolber B, et al. A protocol of manual tests to measure sensation and pain in humans. *J Vis Exp.* 2016;19:e54130. https://doi.org/10.3791/54130.
52. Treede RD, Rolke R, Andrews K, et al. Pain elicited by blunt pressure: neurobiological basis and clinical relevance. *Pain.* 2002;98:235–240.
53. Mutlu EK, Ozdincler AR. Reliability and responsiveness of algometry for measuring pressure pain threshold in patients with knee osteoarthritis. *J Phys Ther Sci.* 2015;27:1961–1965.
54. Zafereo J, Wang-Price S, Kandil E. Quantitative sensory testing discriminates central sensitization inventory scores in participants with chronic musculoskeletal pain: an exploratory study. *Pain Pract.* 2021;21:547–556. https://doi.org/10.1111/papr.12990.
55. de Oliveira AK, Dibai-Filho AV, Soleira G, et al. Reliability of pressure pain threshold on myofascial trigger points in the trapezius muscle of women with chronic neck pain. *Rev Assoc Méd Bras.* 2021;67:708–712.
56. Grimby-Ekman A, Ahlstrand C, Gerdle B, et al. Pain intensity and pressure pain thresholds after a light dynamic physical load in patients with chronic neck-shoulder pain. *BMC Musculoskelet Disord.* 2020;21:1–10.
57. Wang-Price S, Zafereo J, Brizzolara K, et al. Psychometric properties of pressure pain thresholds measured in 2 positions for adults with and without neck-shoulder pain and tenderness. *J Manip Physiol Ther.* 2019;42:416–424.
58. Heredia-Rizo AM, Petersen KK, Arendt-Nielsen L, et al. Eccentric training changes the pressure pain and stiffness maps of the upper trapezius in females with chronic neck-shoulder pain: a preliminary study. *Pain Med.* 2020;21:1936–1946. https://doi.org/10.1093/pm/pnz360.
59. Rolke R, Baron R, Maier C, et al. Quantitative sensory testing in the German Research Network on Neuropathic Pain (DFNS): standardized protocol and reference values. *Pain.* 2006;123:231–243. https://doi.org/10.1016/j.pain.2006.01.041.
60. Arant KR, Katz JN, Neogi T. Quantitative sensory testing: identifying pain characteristics in patients with osteoarthritis. *Osteoarthr Cartil.* 2021;30:17–31.
61. Park G, Kim CW, Park SB, et al. Reliability and usefulness of the pressure pain threshold measurement in patients with myofascial pain. *Ann Rehabil Med.* 2011;35:412–417.
62. Coronado RA, Simon CB, Valencia C, et al. Experimental pain responses support peripheral and central sensitization in patients with unilateral shoulder pain. *Clin J Pain.* 2014;30:143–151.
63. Hogue S, Guo X, Morrison RA, McDowell S, Shembel AC. Use of motion capture technology to study extrinsic laryngeal muscle tension and hyperfunction. *Laryngoscope.* 2023;133:3472–3481.
64. Aghadoost S, Jalaie S, Khatoonabadi AR, et al. A study of vocal facilitating techniques compared to manual circumlaryngeal therapy in teachers with muscle tension dysphonia. *J Voice.* 2019;34:963.e11–963.e21. https://doi.org/10.1016/j.jvoice.2019.06.002.
65. Roy N, Bless DM, Heisey D, et al. Manual circumlaryngeal therapy for functionaldysphonia: an evaluation of short- and long-term treatment outcomes. *J Voice.* 1997;11:321–331. https://doi.org/10.1016/S0892-1997(97)80011-2.
66. Ahmadi N, Abbott KV, Rajati F, et al. Effects of laryngeal manual therapy on primary muscle tension dysphonia (MTD-1): implications for MTD-1 type. *J Voice.* 2022. https://doi.org/10.1016/j.jvoice.2022.04.002.
67. Chatterjee I, Das L, Majumdar A, et al. Determining the outcome of laryngeal manual therapy in female subjects with muscle tension dysphonia: an experimental study. *Int J Phonosurg Laryngol.* 2021;11:10–15. https://doi.org/10.5005/jp-journals-10023-1194.
68. Mathieson L, Hirani SP, Epstein R, et al. Laryngeal manual therapy: a preliminary study to examine its treatment effects in the management of muscle tension dysphonia. *J Voice.* 2009;23:353–366. https://doi.org/10.1016/j.jvoice.2007.10.002.
69. Woźnicka E, Niebudek-Bogusz E, Morawska J, Wiktorowicz J, Śliwińska-Kowalska M. Laryngeal manual therapy palpatory evaluation scale: a preliminary study to examine its usefulness in diagnosis of occupational dysphonia. *Medycyna Pracy.* 2017;68. https://doi.org/10.13075/mp.5893.00463.
70. Middlebrook N, Heneghan NR, Evans DW, et al. Reliability of temporal summation, thermal and pressure pain thresholds in a healthy cohort and musculoskeletal trauma population. *PLoS One.* 2020;15:e0233521. https://doi.org/10.1371/journal.pone.0233521.
71. Nie H, Arendt-Nielsen L, Andersen H, et al. Temporal summation of pain evoked by mechanical stimulation in deep and superficial tissue. *J Pain.* 2005;6:348–355. https://doi.org/10.1016/j.jpain.2005.01.352.
72. Graven-Nielsen T, Arendt-Nielsen L. Assessment of mechanisms in localized and widespread musculoskeletal pain. *Nat Rev Rheumatol.* 2010;6:599–606. https://doi.org/10.1038/nrrheum.2010.107.
73. Neblett R, Cohen H, Choi Y, et al. The Central Sensitization Inventory (CSI): establishing clinically significant values for identifying central sensitivity syndromes in an outpatient chronic pain sample. *J Pain.* 2013;14:438–445.
74. Patel RR, Awan SN, Barkmeier-Kraemer J, et al. Recommended protocols for instrumental assessment of voice: American Speech-Language-Hearing Association expert panel to develop a protocol for instrumental assessment of vocal function. *Am J Speech Lang Pathol.* 2018;27:887–905. https://doi.org/10.1044/2018_AJSLP-17-0009.
75. Toles LE, Shembel AC. Acoustic and physiologic correlates of vocal effort in individuals with and without primary muscle tension dysphonia. *Am J Speech Lang Pathol.* 2024;33:237–247.
76. Wyke BD. Laryngeal myotatic reflexes and phonation. *Folia Phoniatr Logop.* 1974;26:249–264. https://doi.org/10.1159/000263784.
77. Bradley RM. Sensory receptors of the larynx. *Am J Med.* 2000;108(4, suppl 1):47–50. https://doi.org/10.1016/S0002-9343(99)00339-3.
78. Gould WJ, Tanabe M. The effect of anesthesia of the internal branch of the superior laryngeal nerve upon phonation: an aerodynamic study. *FPL.* 1975;27:337–349. https://doi.org/10.1159/000264005.
79. Dietrich M, Verdolini Abbott K, Gartner-Schmidt J, et al. The frequency of perceived stress, anxiety, and depression in patients with common pathologies affecting voice. *J Voice.* 2008;22:472–488.
80. Dietrich M, Andreatta RD, Jiang Y, et al. Preliminary findings on the relation between the personality trait of stress reaction and the central neural control of human vocalization. *Int J Speech Lang Pathol.* 2012;14:377–389. https://doi.org/10.3109/17549507.2012.688865.
81. Dietrich M, Katherine VA. Psychobiological stress reactivity and personality in persons with high and low stressor-induced extralaryngeal reactivity. *J Speech Lang Hear Res.* 2014;57:2076–2089. https://doi.org/10.1044/2014_JSLHR-S-12-0386.
82. Roy N, Dietrich M, Blomgren M, et al. Exploring the neural bases of primary muscle tension dysphonia: a case study using functional magnetic resonance imaging. *J Voice.* 2019;33:183–194. https://doi.org/10.1016/j.jvoice.2017.11.009.
83. Gillespie AI, Gartner-Schmidt J. Immediate effect of stimulability assessment on acoustic, aerodynamic, and patient-perceptual measures of voice. *J Voice.* 2016;30:507.e9–507.e14. https://doi.org/10.1016/j.jvoice.2015.06.004.
84. Vahid M, Mansuri B, Farzadi F, et al. Immediate effects of combining kinesio tape with voice therapy in patients with muscle tension dysphonia. *J Voice.* 2022. https://doi.org/10.1016/j.jvoice.2022.09.006.
85. Watts CR, Hamilton A, Toles L, et al. A randomized controlled trial of stretch-and-flow voice therapy for muscle tension dysphonia. *Laryngoscope.* 2015;125:1420–1425. https://doi.org/10.1002/lary.25155.
86. Shembel AC, Morrison RA, Fetzer DT, et al. Extrinsic laryngeal muscle tension in primary muscle tension dysphonia with shear wave

elastography. *Laryngoscope.* 2023;133:3482–3491. https://doi.org/10.1002/lary.30830.

87. Morrison RA, Fetzer DT, Patterson-Lachowicz A, et al. Optical Flow Analysis of Paralaryngeal Muscle Movement. *The Laryngoscope.* 2024;134(4):1792–1801. https://doi.org/10.1002/lary.31063.
88. Hogue S, Guo X, Morrison RA, et al. Use of motion capture technology to study extrinsic laryngeal muscle tension and hyperfunction. *Laryngoscope.* 2023;133:3472–3481. https://doi.org/10.1002/lary.30829. lary.30829.
89. Piersiala K, Akst LM, Hillel AT, et al. Chronic pain syndromes and their laryngeal manifestations. *JAMA Otolaryngol Head Neck Surg.* 2020;146:543–549. https://doi.org/10.1001/jamaoto.2020.0530.
90. Ramaswamy S, Wodehouse T. Conditioned pain modulation-a comprehensive review. *Neurophysiol Clin.* 2021;51:197–208. https://doi.org/10.1016/j.neucli.2020.11.002.
91. Foote A, Thibeault S. Sensory innervation of the larynx and the search for mucosal mechanoreceptors. *J Speech Lang Hear Res.* 2021;64:371–391. https://doi.org/10.1044/2020_JSLHR-20-00350.
92. Bearelly S, Cheung SW. Sensory topography of oral structures. *JAMA Otolaryngol Head Neck Surg.* 2017;143:73–80. https://doi.org/10.1001/jamaoto.2016.2772.
93. Herrero JF, Laird JMA, Lopez-Garcia JA. Wind-up of spinal cord neurones and pain sensation: much ado about something? *Prog Neurobiol.* 2000;61:169–203. https://doi.org/10.1016/S0301-0082(99)00051-9.