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Primary Muscle Tension Dysphonia

Melda Kunduk[1] · Daniel S. Fink[2] · Andrew J. McWhorter[2]

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Abstract
Purpose of Review Primary muscle tension dysphonia (PMTD) is a voice disorder of indeterminate cause, thought to be multifactorial. Emerging research is improving its diagnosis, but the lack of standard diagnostic testing for PMTD still creates challenges in accurate diagnosis and treatment.
Recent Findings Review of the recent literature demonstrates the scarce validity and reliability of subjective laryngeal palpation measures, which are commonly used to assess the presence of intrinsic and extrinsic laryngeal tension in PMTD. An initial study with transcranial magnetic stimulation shows promise in the differential diagnosis between adductor spasmodic dysphonia and PMTD.
Summary Current and past review of the literature continues to demonstrate the need for future investigation to determine the true pathophysiology of PMTD and its accurate differential diagnosis.

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✉ Andrew J. McWhorter
andrew.mcwhorter@ololrmc.com

Melda Kunduk
mkunduk@lsu.edu

Daniel S. Fink
daniel.fink@ololrmc.com

[1] The Department of Communication Sciences and Disorders, Louisiana State University, 68 Hatcher Hall, Field House Drive, Baton Rouge, LA 70803, USA

[2] Department of Otolaryngology-Head and Neck Surgery, Our Lady of the Lake Voice Center, Louisiana State University Health Sciences Center, New Orleans, LA 70112, USA

Keywords Primary muscle tension dysphonia · Laryngeal palpation techniques · Differential diagnosis

Introduction

Primary muscle tension dysphonia (PMTD) is a nonorganic voice disorder characterized by abnormal laryngeal posturing during phonation. As a disorder that can present with many different vocal characteristics, accurate diagnosis can be challenging and lead to misdiagnosis by a patient's primary medical care team. The potential for misdiagnosis, or failure to diagnose PMTD results in delay in delivering accurate, timely, and effective treatment approaches. The classification manual for voice disorders describes PMTD as the presence of excessive, atypical and abnormal laryngeal movements during phonation, in the absence of any obvious structural and neurological etiology [1]. PMTD differs from the diagnosis of secondary/adaptive muscle tension dysphonia (sMTD) where the excessive/abnormal laryngeal movements are considered to be due to the need for alleviating the original organic and/or neurologic causes [1]. Primary and secondary muscle tension dysphonia are common referrals to a voice center, reportedly forming 10–40 % of referrals [2, 3]. Despite the absence of organic or neurologic causes in primary muscle tension dysphonia, it can lead to the same emotional, social, financial and occupational hardships that any other organic and neurologic voice disorder can. This paper will focus on the current understanding of primary muscle tension dysphonia and a review of the current literature of the perceptual, acoustic, endoscopic, aerodynamic, and radiographic characteristics of PMTD to aid in its diagnosis and treatment.

Hyperfunctional laryngeal and extralaryngeal muscle activities are considered to be the causes of PMTD [3]. The hyperfunctional state found in PMTD is representative of a "nonadducted hyperfunction" [4], since it does not result in vocal fold tissue trauma. This type of hyperfunction differs from the "adducted hyperfunction" where the prolonged presence of heightened muscle hyperfunction together with increased impact closure during vocal fold vibration can create tissue damage (e.g. as in vocal fold edema and nodules) [4]. Both nonadducted and adducted hyperfunction eventually can result in change in voice quality, vocal fatigue and varying severity of dysphonia.

The exact causes of the excessive hyperfunctional state in the laryngeal and extralaryngeal musculature in PMTD is still unknown. However, the etiology is felt to be multifactorial [5]. The contributing factors to the increased tension in intrinsic and extrinsic laryngeal musculature include prolonged compensatory strategies begun during an acute and resolved insult [5], upper airway infection [6], and or LPR/GERD [5]. In addition, personality/psychological factors and reaction to high level stress are thought to be additives the development of PMTD [1, 7].

In the clinical setting, it is commonly suspected that PMTD may be a byproduct of compensatory strategies developed by the patient to address a temporary, but resolved, structural/tissue related problem reported in the history of the voice problem (e.g. history of upper respiratory infection). Despite the resolution of the original problem, the patient continues to demonstrate a hyperfunctional intrinsic and extrinsic laryngeal posture during phonation. The persisting PMTD and its voice features, may be similar or different to the original voice problem. Dysphonia due to PMTD responds to behavioral voice therapy, and in the hands of experienced clinicians, the dysphonia is quickly resolved. It is not unusual for PMTD to be completely resolved in one session with behavioral therapy techniques such as laryngeal manual therapy (LMT) [8] and manual circumlaryngeal therapy (MCT) [9]. In some cases, however, restoration of the patient's normal voice may require more than one voice therapy session. In order to set realistic goals for eliminating PMTD, restore the voice to its normal levels promptly, and prevent reoccurrence of the same voice difficulties, clinicians must be equipped with the skill set to allow them to make an accurate diagnosis of PMTD.

Accurate diagnosis of PMTD is primarily achieved by recognizing its auditory-perceptual qualities while carefully eliminating other structural or neurological pathologies with similar sounding voice features. Knowing the endoscopic, acoustic and aerodynamic features of PMTD facilitates accuracy of diagnosis [1]. However, the lack of standard tests available for the diagnosis of PMTD creates challenges in accurate diagnosis by both experienced and inexperienced clinicians. PMTD's auditory-perceptual voice qualities can mimic the voice difficulties caused by a neurologic etiology, such as adductor spasmodic dysphonia [10] or a structural etiology. The treatment approaches for PMTD and adductor spasmodic dysphonia (ADSD) are drastically different. Due to the potential misdiagnosis and mistreatment concerns, there is a significant need to improve the differentiation between the most commonly confused diagnosis of PMTD and ADSD [11]. Review of the features found to be helpful in determining the diagnosis of PMTD have been summarized in Table 1.

Recent Literature on Review Primary Muscle Tension Dysphonia

Review of the most current literature on PMTD identifies several papers on its assessment, treatment and treatment outcomes [12•, 13, 14, 15•, 16•, 17, 18••, 19–24]. Gillespie et al. [13] reported aerodynamic profiles of women with PMTD/Aphonia. Their study results demonstrated variability in the estimated subglottal pressure-flow measures in MTD/aphonia patients, with the largest percentage of patients (32 %) demonstrating a normal aerodynamic profile. Their findings emphasized the presence of varying respiratory and laryngeal function in PMTD/Aphonia patients during voice production [13]. They also quantified the anecdotal observation of abnormal breathing patterns in this patient population, namely "breath holding" where reduced phonatory airflow was observed during its presence [13]. However, the answer to why and how these patterns develop in the first place still remains speculative. The authors recommend future research to explain the variations in the aerodynamic profiles.

Laryngeal palpation to determine the presence of hyperfunction in laryngeal muscles is one of the most widely used assessment technique in the diagnosis of PMTD [25, 26]. The use of the manual laryngeal musculoskeletal tension reduction technique is extensively used as one of the most effective treatment techniques for PMTD [26, 27]. Recently, Khoddami et al. [15•] published a review on the use of laryngeal palpation methods in muscle tension dysphonia. Their review of the literature highlighted the limitations of subjective assessment methods in diagnosing laryngeal and extralaryngeal hyperfunction. The review of the protocols in assessing laryngeal tension demonstrated that the protocols vary in assessment tasks, assessed structures, and in their grading of the tension. In addition, the validity and reliability of these techniques were found to be scarce or not reported in the reviewed articles [15•].

As a method of determining extrinsic laryngeal tension in muscle tension dysphonia, several studies used surface

Table 1 Review of the features found to be helpful in determining the diagnosis of primary muscle tension dysphonia (PMTD) and its differential diagnosis from adductor spasmodic dysphonia (ADSD)

	MTD	ADSD (without tremor)
Laryngeal pathophysiology		
1. Intermittent spasm in laryngeal muscles	Surface EMG studies demonstrates inconsistent/conflicting results on EMG levels [12]	Present [11, 31]
2. Reduced inhibition of laryngeal adductor response		Present [32]
Perceptual evaluation features		
1. *Voice quality*		
Strained/strangled voice quality	Present	Present [11]
Voice breaks	Present	Present [11]
Effort	Present	Present [11]
Vocal fatigue	Present	Present
2. *Task dependency* [10]	No	Yes
Sustained vowel is better than connected speech? [10]	No, voice remains the same for both tasks	Yes
Phoneme content (effects of voiced and voiceless consonants) [10]	No, voice remains almost the same for both tasks	Yes, breaks on vowels [11]
3. *Development and course of the voice symptoms*: [1, 11, 33•]		
Onset	More often "sudden" than "gradual	More often "gradual" than "sudden" [34]
Course	Voice may fluctuate and may return to normal and stay normal for a prolonged period of time [11]	Voice may fluctuate/improve but it never goes back to normal
4. *Differential performance during varying speech tasks*: [33•, 11]		
Falsetto	Affected same as in other types of speech	Less strain
Singing	Affected same as speech	Less strain to normal [11]
Whisper	May improve symptoms	Normal [11]
Shout	Affected same as speech	Normal [11]
Laugh	Affected same as speech	Normal [11]
Talking loud	Affected same as speech	May worsens symptoms
5. *Effects of speaking environment*		
Talking on the phone	Voice often remains the same	Voice often gets worse
Ordering at a drive through	Voice often remains the same	Voice often gets worse
Alcohol	Voice often remains the same	Voice improves
6. *Presence of subjective "breath hold" concept* [13]	Present	Not investigated
Fiberoptic laryngoscopic features		
1. *Glottic and supraglottic constriction patterns* [11, 35]	Sustained hyperadduction Irrespective of phoneme content May present with incomplete glottic closure, and/or limited or no vocal fold vibratory patterns	Intermittent hyperadduction/spasms, phoneme content dependent (vowels) Motion irregularity found to be significant predictor [36]
Whistling	Normal abduction and abduction	Normal abduction and abduction [11]
Symmetry	Normal	Normal [11]
Structure	Normal (may have edema)	Normal [11]
Laryngeal position and palpation measures		
1. *Radiographic measures*		
PMTD versus controls (subjects with no voice disorders) [14]		
a. Laryngeal position		
At rest	Significantly higher in PMTD than controls [14]	Normal (not investigated)
During phonation	10 mm higher than rest in PMTD but only 2 mm in controls [14]	Normal (not investigated)
b. Hyolaryngeal space		
At rest	No difference between PMTD and Controls [14]	Normal (not investigated)
During phonation	Decreased for both groups compared to rest position [14]	Normal (not investigated)
c. Hyoid position		
At rest	Significantly higher in PMTD than controls [14]	Normal (not investigated)
During phonation	Same as at rest for PMTD but lower for controls [14]	Normal (not investigated)

Table 1 continued

	MTD	ADSD (without tremor)
d. Measurements for POST-treatment of PMTD for laryngeal	position, hyolaryngeal space hyoid position	
At rest	Not investigated	
During phonation	Not investigated	
2. *Subjective common laryngeal palpation methods* [15•]		
a. Assessment tasks:		
At rest		
During speech		
Swallowing		
Singing		
b. Assessment on:		
Hyoid bone		
Thyroid cartilage		
Thyrohyoid space		
Suprahyoid muscles		
Sternocleidomastoid muscles (SCMs)		
Pharyngolaryngeal muscles		
c. Assessment criteria:	Mostly present	Mostly absent
Focal tenderness	Mostly present	Mostly absent
Laryngeal pain	Mostly reduced	WNL
Horizontal mobility	Elevated	WNL
Laryngeal elevation	Increased	WNL
Laryngeal resistance		
d. Grading Scales		
Subjective:		
Present/absent		
Ordinal scale (0–3)		
e. Validity and Reliability Measures		
Mostly not reported or low [14, 28]		
Botox injection to false vocal folds	For MTD refractory to voice therapy. Varying results with 6/7 patients needed repeated injections [16]	Considered to be a great option for a subgroup of ADSD patients. Currently no clear guidelines for patient selection criteria
Recurrent laryngeal nerve lidocaine block		
Offers little discriminatory value in the differential diagnosis of ADSD versus MTD [37]	Positive response in patient- and listener-based ratings [37]	Positive response in patient- and listener-based ratings [37]
Acoustic features		
Phonatory breaks	50 % of women demonstrated phonatory breaks during the all voiced sentence task [38]	Higher mean number of phonatory breaks in all voiced sentences [38]
	Seldom occurrence of phonatory breaks in males [38]	Longer and more frequent phonatory breaks in both man and women [38]
Aerodynamic features		
1. Airflow measures	Presence of varying airflow-pressure combinations [13]	No significant difference in mean phonatory airflow between ADSD, MTD and controls during syllable repetitions [39]
	(1) Normal estimated subglottal pressure and airflow	
	(2) High airflow-normal estimated subglottic pressure	Significantly higher amplitude-based glottal airflow parameters in women with ADSD compared to controls with no voice problems [40]
	(3) High airflow with high estimated subglottal pressure	
	(4) Normal airflow with high estimated subglottal pressure	
	(5) "Breath hold" pattern (normal established subglottal pressure with low airflow [13]	
2. Pre-post treatment	Average airflow in all voiced sentence	Improved translaryngeal airflow after Botox injections in SD patients [41]
	Significant increase in measure post treatment [17]	

Table 1 continued

	MTD	ADSD (without tremor)
Laryngeal muscle activity surface EMG		
Surface EMG measures of laryngeal activity [12•]	Conflicting results varied from higher EMG levels in MTD group to no group differences [12•]	Yes (see first section on the table)
1. Tasks		
At rest		
Phonation		
During spontaneous speech		
Reading		
2. Varying type of electrodes		
3. Varying electrode Positioning		
4. Control group (normal voice/nodule)		
5. Varying outcome measures		
Cortical excitability differences (transcranial magnatic stimulation (tms))		
Cortical silent period duration (CSP) [18••]	Shorter in MTD group than Control group	Significantly shorter than in MTD and control groups suggesting widespread dysfunction in $GABA_B$ mechanism
Trial speech/non speech stimubility tests (clinical)		
1. Inhalation phonation	Voice often remains the same	It is one of the management techniques but hard to use consistently
2. Cough	Normal	Normal
3. Throat clearing	Normal	Normal
4. Lib/tongue trills	Most of the time unable to do it with voice with tension visible on the lips and face	Usually hard to initiate the voice on a vowel, but it improves after the phonation starts
5. Reading voiced phoneme sentences with high pitch	Voice remains the same	Voice usually improves
6. Reading voiced phoneme sentences with loud voice	Voice remains the same	Voice usually deteriorates
7. Whisper	Articulatory patterns may improve	Normal
8. Counting from 60 to 69	Voice remains the same	Easier to
9. Counting from 80 to 89	Voice remains the same	Harder to count

EMG technique (measurements mostly from supra and infrahyoid muscles [12•]) with conflicting results in its effectives in the assessment of muscle tension [28–30]. Stepp et al. using neck surface EMG studied the alteration in vocal hyperfunction before and after injection laryngoplasty [28]. They concluded that despite the significant improvement in perceptual ratings of strain and false vocal fold compression; there were no significant changes in anterior sEMG values [28]. In a more recent study, Van Houtte et al. [12•] reported their findings in PMTD patients. The PMTD patients in the study did not demonstrate higher levels of sEMG values during rest, phonation or reading tasks compared to a normal control group [12•]. The study results did not support the use of sEMG as a diagnostic tool for differentiating patients with and without PMTD or to investigate the presence of extrinsic laryngeal tension in this population. Results of the previous and more recent articles further emphasize the need for further research in standardizing the assessment techniques in the PMTD population.

More recently, Samargia et al. investigated the cortical excitability differences between MTD, ADSD and healthy controls using transcranial magnetic stimulation (TMS) [18••]. The results of the study find significantly shorter cortical silent period (CSP) duration in ADSD than MTD and healthy controls in masseter and first dorsal interosseus (FDI) muscles. The authors interpreted these findings as a widespread dysfunction of the $GABA_B$ mechanism in ADSD and this dysfunctional $GABA_B$ mechanism as a pathophysiologic feature of ADSD. However, the authors found no significant correlation between CSP and perceptual voice ratings (CAPE -V and voice breaks analyses). They interpreted this lack of correlation as the inability of neurophysiologic measures to determine the severity of symptoms and highlighted the importance of information from other behavioral measures. The authors recommended

further research on the use of TMS in aiding differential diagnosis of MTD and ADSD. Even though TMS is considered a noninvasive method to assess cortical excitability and has been used to better understand focal dystonia, currently, it is not a technique used routinely in the clinic setting [18••].

While treatment for PMTD is primarily based on laryngeal manipulation and restructuring the voice through voice therapy techniques, other interventions are sometimes utilized. Pacheco et al. recently reported their experience with treatment of refractory MTD with false vocal fold botulinum toxin injections in a retrospective study over 4 years [16]. All seven patients in the study were reported to have had at least 4–6 weeks of voice therapy prior to botulinum toxin injections and were encouraged to continue with voice therapy following the injections. The dosage of the injection (neurotoxin) varied from 30 to 33 units during the initial injections to 40–45 units for subsequent injections. The authors reported repeated injections for 5 of 7 subjects (one subject was lost at follow up) with varying voice outcomes (three subjects reported improvement from "poor" to "fair, one from "fair" to "very good", and another "poor" to good" and with one subject not improving on VRQOL scores). Six subjects received 14 injections in total and the authors reported 10 of these fourteen injections successful in reducing the voice difficulties. One patient had sustained benefit following one injection, whereas another patient received five injections. The authors also reported the gradual loss of the voice benefits, especially in patients who were noncompliant with voice therapy, as the botulinum toxin wore off [16].

Some of the recent articles on PMTD also reported on combining physical therapy to the battery of existing treatment approaches [19], compared telepractice to in-person delivery of voice therapy [21] and evaluated treatment outcomes [17, 20] in the PMTD population. Dastolfo et al. reported that following the treatment of PMTD, average airflow measures in the all-voiced sentence significantly increased but no change in airflow measurements were observed in syllable production [17]. In another treatment outcome study, Watts et al. reported the positive effects of stretch and flow exercises in secondary and PMTD patients in 6 treatment sessions [23].

Rangarathnam et al. demonstrated that PMTD could be treated using the flow phonation approach using telepractice [21]. They reported that perceptual measures were improved significantly in both in-person delivery and telepractice groups after each group of subjects received 12 treatment sessions. Acoustic and aerodynamic measures also improved, but they did not reach statistical significance. However, the authors did not report if the voices returned to normal or if any group had subjects that achieved significant voice improvement in earlier sessions. Future studies are warranted to determine if the addition of laryngeal manual therapy [8, 9] to an in-person delivery group, which cannot be delivered via telepractice, will shorten the voice recovery time in this patient population. The study's results are encouraging, and as the authors suggested, telepractice may address possible compliance issues due to the hardships sometimes experienced by the subjects from frequent travelling to voice centers. Future studies are needed to determine if telepractice can deliver further effective treatment techniques quickly and efficiently.

Conclusion

The review of the recent literature reveals new assessment and treatment concepts for PMTD. However, the concerns for the lack of standard assessment techniques and the heavy reliance on clinicians' experience and auditory-perceptual voice quality for the accurate diagnosis of PMTD still persist. In conclusion, the current and past literature continue to demonstrate the need for future studies to determine the true pathophysiology of primary muscle tension dysphonia and its accurate differential diagnosis. These studies should be geared towards developing standard testing to supplement the auditory perceptual evaluation to better distinguish PMTD from other similar sounding voice disorders. The establishment of clinically reliable, validated objective assessment tools for the presence of excessive, atypical, and abnormal laryngeal movements during phonation and their correlation with the currently available subjective assessment protocols will further the accurate diagnosis of PMTD in wider clinic settings. The establishment of a standardize assessment protocol for PMTD would minimize the dependence of accurate diagnosis on the level of clinicians' experience. In addition, further research should help to differentiate subgroups of PMTD (e.g. PMTD due to over lasting compensatory voice production (as in initiating but resolved upper respiratory infection versus due to personality/psychological factors/high stress versus malingering/secondary gain) as well as the differentiation from other etiologies such as ADSD. This knowledge will help to reduce the cost of assessment, diagnosis and treatment of PMTD for the individual and the society due to the loss of personal and professional productivity in this patient population.

Compliance with Ethical Guidelines

Conflict of Interest Dr. Melda Kunduk, Dr. Daniel S. Fink, and Dr. Andrew J. McWhorter declare that they have no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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