ASHA American Speech-Language-Hearing Association

JSLHR

Review Article

Integrative Review and Framework of Suggested Mechanisms in Primary Muscle Tension Dysphonia

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ARTICLE INFO

Article History:
Received October 26, 2021
Revision received December 14, 2021
Accepted January 12, 2022

Editor-in-Chief: Cara E. Stepp
Editor: Bharath Chandrasekaran

https://doi.org/10.1044/2022_JSLHR-21-00575

ABSTRACT

Purpose: Despite the high prevalence of primary muscle tension dysphonia (MTD-1), its underlying mechanisms and their interrelationships have yet to be fully identified. The objectives of this integrative review were (a) to describe and classify the suggested underlying mechanisms for MTD-1, (b) to appraise the empirical evidence supporting each of the proposed mechanisms, and (c) to summarize the information in an integrative model.
Method: PubMed, Scopus, and CINAHL were searched for all publications pertaining to muscle tension dysphonia. Papers were retained if they included theoretical or empirical data pertaining to underlying mechanisms of MTD-1. A total of 921 papers initially qualified for screening, of which 100 remained for consideration in this review. Underlying mechanisms of MTD-1 were extracted using a consensus approach.
Results: Seven broad categories of putative mechanisms involved in MTD-1 were identified: psychosocial, autonomic, sensorimotor, respiratory, postural, inflammatory, and neuromuscular. These categories were further divided into 19 subcategories detailed in the body of this review article. Based on the reviewed evidence, our proposed integrative model presents MTD-1 as an idiosyncratic motor adaptation to physiological perturbation or perceived threat. Under this model, physiologically or psychologically aversive stimuli can instigate a series of motor adaptations at multiple levels of the nervous system, ultimately disturbing muscle activation patterns and their biomechanical outcomes. Importantly, these adaptations appear to have the potential to become chronic even after threatening stimuli are withdrawn.
Conclusions: The proposed model highlights the importance of personalized rehabilitation in MTD-1 treatment. Limitations of the literature are discussed to provide guidance for future research aimed at improving our understanding of MTD-1.
Supplemental Material: https://doi.org/10.23641/asha.19586065

The Classification Manual of Voice Disorders defines primary muscle tension dysphonia (MTD-1) as a "dysphonia in the absence of current organic vocal fold pathology, without obvious psychogenic or neurologic etiology, associated with excessive, atypical or abnormal laryngeal movements during phonation" (Verdolini et al., 2014). Other common characterizations describe "excessive tension" in the intrinsic or extrinsic laryngeal muscles (Oates & Winkworth, 2008; Van Houtte et al., 2011), which is generally regarded as the proximal cause of the disorder. Patients with MTD-1 may report vocal fatigue, pain during phonation, or changes in voice quality, leading to decreased voice-related quality of life and significant interference with communicative activities (Verdolini et al., 2014). In contrast to secondary MTD, which refers to the presence of abnormal laryngeal activity in response to an organic cause (e.g., compensation for vocal fold lesions), MTD-1 is thought to occur in the absence of an obvious organic cause. An important implication of this distinction, combined with the lack of clear etiology for the disorder, is that MTD-1 is currently a diagnosis of exclusion (Kollbrunner & Seifert, 2017).

A retrospective study conducted by Van Houtte et al. (2010) showed that MTD-1—called by the corollary term *functional dysphonia* in that publication—accounted

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for approximately 30% of voice disorders in a treatment-seeking population and was the most prevalent voice condition for all adult age groups in that caseload. Despite its high prevalence, much remains to be clarified about the condition's etiology. In fact, as noted by Baker et al. (2007), the numerous terms used to refer to several conditions with similar clinical presentations suggest that the underlying causes of this class of conditions are not well understood. The most widely used umbrella term, *muscle tension dysphonia*, refers to the presumed proximal cause of the disorder and leaves room for a wide array of potential underlying distal causes (Van Houtte et al., 2011). General consensus in the literature holds that MTD-1 is a multifactorial phenomenon involving complex interactions among factors. Much remains to be discovered about how relevant factors interact and why they result in MTD-1 in certain individuals (Baker, 2008; Hillman et al., 2020; Oates & Winkworth, 2008).

Understanding the underlying mechanisms leading to a disorder is essential to develop effective therapies. While current therapy programs have been shown to improve voice quality and voice-related quality of life in some patients with MTD-1 (Eastwood et al., 2015), other patients do not benefit from mainline therapies or have poor long-term outcomes despite treatment (Van Lierde et al., 2007). This is perhaps because these approaches traditionally address the main overt manifestation of MTD-1, abnormal activation of the (para)laryngeal musculature (Van Houtte et al., 2011), but may not target the underlying mechanisms leading to chronic adverse laryngeal posturing.

Existing Models

Progress has been made in beginning to unravel the underlying mechanisms of MTD-1. Although the first proposed classifications were predominantly based on laryngoscopic findings (Koufman & Blalock, 1982, 1988, 1991; Morrison et al., 1983, 1986; Morrison & Rammage, 1993; Van Lawrence, 1987), questions have since been raised about the diagnostic and etiological significance of these profiles (Roy et al., 2007). In fact, it remains unclear whether or not these laryngoscopic phenotypes are associated with similar or different pathological mechanisms. Turning to associated symptoms, Spencer (2015) has suggested that symptomatic manifestations of MTD-1 may be amenable to positive treatment outcomes, regardless of underlying mechanisms. Although symptomatic classifications such as suggested by Spencer (2015) can help guide interventions, there is still a need to position the disorder within a broader framework to better understand the intricacies of its underlying mechanisms and move treatment research forward.

Recent frameworks aimed at describing MTD-1 have offered a deeper look at the condition. A key example is Hillman et al.'s (2020) updated framework of vocal hyperfunction, in which the proximal etiological process in MTD-1 is characterized as *nonphonotraumatic vocal hyperfunction* (NPVH; previously *nonadducted hyperfunction*; Hillman et al., 1989). In this updated framework, vocal hyperfunction is characterized as a speaker's vocal response in the context of increased vocal demand or triggering factors such as emotional stress, irritant exposure, or dehydration, potentially leading to temporary perceived vocal effort and fatigue even in vocally healthy subjects (Hillman et al., 2020; Hunter et al., 2020). Three factors are further identified as sufficient to precipitate chronic NPVH: psychosocial factors, altered biomechanics, and sensorimotor deficits (Hillman et al., 2020). Hillman et al.'s model provides a critical foundation for the study of vocal hyperfunction, and it exposes gaps in our understanding of NPVH. In fact, important questions remain regarding how the identified factors (and others) interact to sustain vocal hyperfunction in MTD-1: Whereas mechanisms perpetuating a "vicious cycle" of phonotraumatic hyperfunction are well-described, those related to NPVH remain largely undefined. As noted by the authors, "it is not known why such patients literally seem to 'get stuck' (or habituate) using these maladaptive vocal behaviors" (Hillman et al., 2020). The present integrative review complements this existing framework by providing a comprehensive overview of mechanisms proposed to be related to the development and maintenance of MTD-1 and by integrating the information with evidence from other fields of study to create a physiologically grounded model. The goal is to contribute to our understanding of the chronic nature of motor adaptations in MTD-1.

Along similar lines, the Union of European Phoniatricians Voice Committee's recently published position paper calls for an integrated framework for voice disorders more broadly (Hacki et al., 2022). The suggestion is that that dysphonia originating from temporarily disturbed psychomotor or sensorimotor systems (potentially including MTD-1) should be characterized as "malregulative dysphonia." Malregulation may purportedly arise from behavioral, sensory, or psychogenic etiologies. The current review is in agreement with this terminology framework, and it provides a comprehensive review of potential etiologies for the "dysregulated muscle activity" characteristics of MTD-1 (Roy, 2008), in addition to offering a critical appraisal of the current evidence for each suggested etiological mechanism to help identify specific areas for future research.

Need for an Integrative Framework of MTD-1 and Objectives

Although the frameworks detailed in the previous section have meaningfully advanced the state of MTD-1

research beyond early laryngoscopic descriptions, the voice field still lacks a comprehensive, physiologically grounded etiological model of MTD-1. As such, MTD-1 generally remains a diagnosis of exclusion (e.g., Kollbrunner & Seifert, 2017). The objectives of this integrative review were therefore to (a) describe and classify suggested mechanisms underlying MTD-1; (b) appraise the empirical evidence that supports these proposed underlying mechanisms; and (c) integrate the information into a broader, physiologically based model of MTD-1.

Method

Search and Screening Procedures

A search strategy was used to retrieve all publications relevant to muscle tension dysphonia using the following keywords: *muscle tension dysphonia, primary muscle tension dysphonia, vocal fatigue, functional voice disorder, functional dysphonia, muscle misuse voice disorder, muscle misuse dysphonia, hyperfunctional voice disorder, hyperfunctional dysphonia,* and *ventricular dysphonia.* Keywords were combined with the Boolean operator "OR," and the initial search was conducted in three databases (PubMed, Scopus, and CINAHL) on October 9, 2019.

Current best practices dictate that integrative reviews—which are typically aimed at reviewing experimental and nonexperimental studies to synthesize concepts and theories (Whittemore & Knafl, 2005)—follow methodical review procedures to ensure a comprehensive and reliable review of the literature. After removing duplicates, the first and second authors screened 15% of the articles by title and abstract, and reached 95% agreement on their inclusion using eligibility criteria described shortly. Where ambiguities existed, the first, second, and last author discussed the papers until they reached consensus. Discrepancies were analyzed in order to specify and clarify eligibility criteria. All papers were then screened by the first author to exclude irrelevant articles, based on titles and abstracts. The remaining articles underwent full-text screening by the first author based on specific eligibility criteria, noted below. The second author performed full-text screening on 15% of the papers, selected at random, to evaluate their eligibility. The first and second authors then resolved any discrepancies between them, and the second author's comments were taken into account for the rest of the full-text screening. The first author also screened the reference lists of all included papers to retrieve any additional publications not identified through the initial search strategy. The first author conducted an updated search in the three same databases on June 7, 2021, to account for newly published MTD-1 research.

Eligibility Criteria

I. Publication

a. Type of publication: Peer-reviewed papers published in an academic journal as well as approved doctoral theses were included.
b. Language: Manuscripts published in English and in French were considered for inclusion, as first, second, and last authors are proficient in these languages.
c. Date: No limitations were applied in terms of publication year.

II. Paper Characteristics and Study Design

a. Papers had to address the topic of MTD-1 or contain information relevant to MTD-1. Note that a variety of criteria and labels for MTD-1 were accepted given the evolution of MTD-1 terminology (e.g., functional dysphonia, nonorganic dysphonia, muscle misuse dysphonia, etc.; see Table 1 for other terms accepted). We acknowledge that the lack of clear diagnostic criteria complicates the interpretation of study results; however, this limitation is inherent to the current state of the MTD-1 literature and necessary for the purposes of conducting an integrative review on the topic.
b. Any paper suggesting *an underlying mechanism for MTD-1* in adults was considered for inclusion. No exclusions were made on the basis of study design. Additionally, a subset of papers were classified as "empirical" if they provided empirical data pertaining to a proposed mechanism. For papers to be considered "empirical," participant characteristics had to conform to those listed next.

III. Participant Characteristics (for Empirical Papers Only)

a. Only studies on adult participants were included (18 years and older).

Table 1. Labels accepted for primary muscle tension dysphonia.

Label	*n*
Muscle misuse dysphonia	1
Functional dysphonia	14
(Primary) muscle tension dysphonia	18
Muscle tension voice disorder	2
Nonorganic (habitual) dysphonia	3
Primary muscle tension aphonia	1
Voice disorder in the absence of laryngeal pathology	1
Nonphonotraumatic vocal hyperfunction (NPVH)	3
Hyperfunctional dysphonia/voice disorder	4

b. At least one group of participants had to comprise solely patients with MTD-1 (not including strict diagnoses of ventricular or psychogenic dysphonia), or the study had to include analyses to compare subgroups of patients with and without MTD-1.
c. The MTD-1 group had to include at least four participants.
d. The study had to include at least one control group (either vocally healthy controls or controls with non-MTD-1 voice disorders).

Data Extraction

Based on data from all included publications, the first author extracted the main proposed mechanistic categories for MTD-1 before discussing them with the second and last author until they reached consensus. Next, categories were further divided into subcategories based on specific causal hypotheses discussed in the literature. The authors then extracted the essential features (associated characteristics) of each mechanistic category as well as associated triggers identified as probable precipitating factors for the disorder based on each causal hypothesis.

For each paper providing empirical data (i.e., publications meeting inclusion Criteria IIIa, IIIb, IIIc, and IIId), the following elements were extracted and reported in Supplemental Materials S1–S6: author(s) and year of publication, label (terminology used by the authors to refer to MTD-1), criteria for MTD-1 diagnosis (specific eligibility criteria to be included in the MTD-1 group), groups of participants being compared (sample size and type of voice disorder/lack thereof), main assessment measures (main measures relevant to a specific category and used to compare groups of participants and inform on the mechanisms involved in MTD-1), and main results relevant to the suggested mechanism (brief summary of the results and associated p values when available).

Results

Overview

The literature search process initially produced 1,006 articles; 847 papers remained after duplicates were removed. An additional 74 papers were identified through other sources and through the second search on June 7, 2021, for a total of 921 papers that qualified for title and abstract screening. Of these, 341 papers qualified for full-text screening, and 241 of those were eliminated based on eligibility criteria. In total, six papers did not meet Criterion Ia, four papers did not meet Criterion Ib, 84 papers did not meet Criterion IIa, 100 papers did not meet Criterion IIb, one paper did not meet Criterion IIIa, 31 papers did not meet Criterion IIIb, three papers did not meet Criterion IIIc, eight papers did not meet Criterion IIId, and four papers were unavailable for review.

The 100 papers that remained, of which 42 were classified as "empirical," became the corpus of literature used to write the current integrative review. Seven broad categories of putative mechanisms involved in MTD-1 emerged: *psychosocial*, *autonomic*, *sensorimotor*, *respiratory*, *postural*, *inflammatory*, and *neuromuscular*. These categories were further divided into 19 subcategories, displayed in Tables 2 to 8 and discussed in further sections. These tables present a summary of the specific characteristics and triggers (when applicable) associated with each subcategory of mechanism, as well as the names of authors who have discussed and contributed to our understanding of each of those mechanisms. The observable manifestations of MTD-1, in terms of biomechanical repercussions (kinematic and laryngoscopic findings), were also summarized. Together, results from the review generated an integrative framework of MTD-1, adapted from an existing theoretical model of motor adaptation to aversive stimuli (Hodges, 2011; Hodges & Tucker, 2011).

Additionally, data extracted specifically from the 42 empirical studies are available in Supplemental Materials S1–S6, as mentioned in the Data Extraction section, for the following categories: psychosocial (Supplemental Material S1), autonomic (Supplemental Material S2), sensorimotor (Supplemental Material S3), respiratory (Supplemental Material S4), inflammatory (Supplemental Material S5), and neuromuscular (Supplemental Material S6). Note that there is no table available for the postural mechanism category because no publications in this category met the eligibility criteria to be considered as strictly "empirical."

Tables 1 and 9 show the labels and criteria used to describe and diagnose MTD-1 and the number of empirical studies in which they appeared (one study can contain more than one label or diagnostic criterion). Note that, for consistency, the term *MTD-1* will be used in the present review, unless the reported study clearly included participants with secondary MTD, which is associated with a known organic condition.

Proposed Mechanisms and Their Associated Evidence

Psychosocial Mechanisms

Conflict over speaking out and Emotion-Processing Deficits Model. Conflict Over Speaking Out (COSO; House & Andrews, 1988) is triggered by stressful experiences and has two requirements: (a) the individual's strong commitment in the scenario and (b) conflict requiring the individual to react, where responding will exacerbate the conflict (e.g., spousal disagreement). In support of their theory, the authors claimed that people with functional

dysphonia experienced a greater number of COSO events than vocally healthy controls in the year prior to or at dysphonia onset (House & Andrews, 1988). Baker (2008) and Baker and colleagues (2013, 2014) adopted COSO as one trigger for MTD-1 in their Emotion-Processing Deficits Model, which posits that stressful life events can trigger negative emotions that are experienced implicitly, viscerally, and in an undifferentiated manner. As a result, simultaneous autonomic and voluntary nervous arousal may lead to strong vocal fold adduction along with concurrent abduction, the latter to facilitate the body's respiratory and metabolic needs during fight or flight reactions (Aronson, 1990; Baker, 2008). Baker et al. (2014) presented mixed evidence for the Emotion-Processing Deficits Model while studying people with MTD-1 or secondary MTD (MTD-2) and corresponding healthy control groups. Results indicated that people with MTD-1 scored lower on the use of both adaptive and maladaptive coping skills when compared to people with MTD-2 and healthy control groups. However, in stronger support of the model, patients with MTD-1 scored higher in alexithymia (trouble with identifying and expressing emotions) than people with MTD-2 and healthy controls (Baker et al., 2014). A note is that the sample size of this study was limited—10 patients with MTD-1 were included—and additional evidence is warranted.

Medically unexplained symptoms, cognitive behavioral therapy model, and perceived control. Several authors proposed that MTD-1 be considered under the rubric of Medically Unexplained Symptoms (MUS)—a group of disorders without obvious etiology including fibromyalgia, chronic fatigue syndrome, and irritable bowel syndrome (Daniilidou et al., 2007; Deary & Miller, 2011; Deary et al., 2018; Kollbrunner & Seifert, 2017; Miller et al., 2014; Misono et al., 2020; O'Hara et al., 2011). Deary et al., who incorporated MUS into their novel Cognitive Behavioral Therapy Model of Functional Dysphonia (CBT Model), pointed to predisposing, precipitating, and perpetuating factors in MTD-1. Proposed predisposing factors (i.e., causing increased susceptibility) included genetics, frequent voice use, neuroticism, introversion, anxious coping style, childhood trauma, and unhealthy perfectionism (Deary & Miller, 2011; O'Hara et al., 2011). Precipitating factors (i.e., triggers) included life events—especially those involving COSO (House & Andrews, 1988)—coping style, frequent voice use, and having repeated respiratory tract infections (Deary & Miller, 2011). Perpetuating factors (i.e., maintaining factors) included general fatigue, anxiety, depression, avoidance of symptoms, and other cognitive, physiological, behavioral, and social factors (Deary & Miller, 2011; O'Hara et al., 2011). Clinically, authors of the CBT Model suggest that speech-language pathologists (SLPs) should identify a patient's specific predisposing, precipitating, and perpetuating factors, and then address them using voice therapy incorporating CBT exercises. Misono et al. (2019) discussed perceived control as a possible perpetuating factor, and reported that greater voice-specific perceived control was adaptive, potentially leading to lessening of MTD-1 symptoms. Interestingly, Haselden et al. (2009) found a greater internal locus of control in patients with MTD-1 when compared to those with laryngeal and nonlaryngeal dystonia but no difference compared to normative data. The authors noted that these results could have been influenced by the proactive nature of their sample—voice therapy–seeking patients with MTD-1—and the young age of participants.

Evidence surrounding MUS/CBT is mixed and largely pertains to higher or lower levels of depression or anxiety—both considered to be predisposing and perpetuating factors (Deary & Miller, 2011)—in people with MTD-1 versus various comparison groups (people with other voice disorders or healthy controls). Higher levels of depression or anxiety in people with MTD-1 were found in works by Andrea et al. (2017), Buck et al. (2007), Dietrich et al. (2008), Mirza et al. (2003), Roy, McGrory, et al. (1997), and Roy et al. (2000a, 2000b). Additionally, Piersiala et al. (2021) reported greater odds of MTD-1 in voice patients with depression when compared to those without depression. However, no difference in scores or even lower anxiety/depression were found in people with MTD-1 versus people with vocal fold nodules, according to reports by Falanga et al. (2020) and Montgomery et al. (2016). Siupsinskiene et al.'s (2011) evidence was mixed; these authors reported that people with MTD-1 had similar rates of depression but higher amounts of anxiety, in comparison with vocally healthy controls.

Regarding general fatigue and connections between MTD-1 and other MUS, O'Hara et al. (2011) reported greater fatigue, along with greater perfectionism, in people with MTD-1 in comparison with healthy controls. Furthermore, Piersiala et al. (2020) documented that, among voice patients, those with MUS (chronic pain syndromes) were more likely to present with MTD-1 than those without chronic pain syndromes. This effect was especially marked for patients with fibromyalgia, who were at least twice as likely to have MTD-1 than controls.

The theory of the dispositional bases of functional dysphonia and vocal nodules. Also known as The Trait Theory of Voice Disorders or Trait Theory (Roy & Bless, 2000; Roy et al., 2000a, 2000b), this theory was the most frequently addressed psychosocial theory pertinent to MTD-1 in the current review (Dietrich & Abbott, 2012; Dietrich et al., 2020; Roy, 2011; Roy et al., 2019; van Mersbergen et al., 2008). The Trait Theory was developed based on a fusion of Eysenck and Gray theories of personality (Roy & Bless, 2000). Eysenck's model of personality includes three continuous dimensions: extraversion–introversion, neuroticism–stability, and psychoticism–control

(Eysenck & Eysenck, 1994). Gray's model consists of three systems, the Behavioral Activation System (BAS; approach behavior), Behavioral Inhibition System (BIS; freeze or flee), and the Nonspecific Arousal System (NAS; heightens BAS/BIS responses; Gray, 1970; McNaughton & Corr, 2004; McNaughton & Gray, 2000). The Trait Theory suggests that people with MTD-1 tend to have high trait introversion and neuroticism, thereby leading them to interpret environmental stimuli negatively. This interpretation triggers the BIS, magnified by the NAS, and passive avoidance of stimuli is facilitated by essentially "withholding" effective vocal communication (Roy & Bless, 2000). In fact, recent brain imaging data showed diminished cortical input for voicing in individuals who are less extroverted and more stress-reactive, potentially requiring greater muscular effort to compensate (Dietrich et al., 2020). Within the Trait Theory, Extraversion corresponds with BAS, introversion is congruous with BIS, and neuroticism/NAS heightens an individual's extravert/introvert signal sensitivities (Roy & Bless, 2000). Largely supporting this theory, Roy et al. found that people with MTD-1 (or "functional dysphonia") had lower extraversion/positive emotionality than people with vocal fold nodules (2000a, 2000b) and vocally healthy controls (Roy et al., 2000b). In addition, Roy et al. (2000a) reported higher neuroticism/negative emotionality in people with MTD-1 when compared to patients with nonfunctional voice disorders and vocally healthy controls.

Additional empirical evidence supports the role of high anxiety (implied in high neuroticism/NAS) in MTD-1, as previously noted. Mirza et al. (2003) specifically stated that some individuals with MTD-1 demonstrated high interpersonal sensitivity and distrusted other people, which is reminiscent of Gray's BIS per interpretation of environmental stimuli as a threat.

Data points unaligned with the Trait Theory are findings that people with MTD-1 had less anxiety than people with vocal fold nodules (Falanga et al., 2020; Montgomery et al., 2016). Adding to the complexity of the matter, van Mersbergen et al.'s (2008) data indicated that people with MTD-1 may have higher anxiety than healthy controls yet lower anxiety than vocally healthy people who have a diagnosis of social anxiety. These findings suggest that anxiety may contribute to MTD-1 but that other factors are involved.

The role of stress, life events, and trauma. Stress is implicated as a trigger for MTD-1 in multiple theories: Emotion Processing Deficits, COSO, MUS, and Perceived Control. However, Dietrich et al. (2008) reported no difference in stress between people with MTD-1 and those with other voice disorders including vocal fold nodules, paradoxical vocal fold motion disorder, and glottal insufficiency. Moreover, Falanga et al. (2020) found greater levels of perceived stress in people with vocal fold nodules compared with MTD-1. Nonetheless, in both Dietrich and Falanga's studies, test scores of people with MTD-1 were indicative of greater perceived stress when compared with healthy controls or normative data. Therefore, as with anxiety, stress may contribute to MTD-1 as part of a group of risk factors. The heterogeneity of factors associated with dysphonia onset in patients with MTD-1 was in fact highlighted by Kridgen et al. (2021), who found that anxiety/stress, upper respiratory infection (URI), and increased voice use were reported with similar frequencies by patients with MTD-1 during case history, which was not the case in patients with phonotraumatic vocal hyperfunction who reported "increased voice use" more frequently than any other factor.

Life events and trauma are posited as triggers in the Emotion-Processing Deficits Model, the MUS/CBT Model, and Perceived Control Model. However, the only study included in this review that provided empirical data regarding life events and trauma (House & Andrews, 1988) found no difference between people with MTD-1 and healthy controls. More empirical data are needed to clarify the role of life events and trauma in MTD-1 onset.

Rammage et al.'s six theories. Rammage et al. (1987) authored the chronologically earliest article in this section on psychosocial factors in MTD-1. In this strictly theoretical review article, the authors described six theories, all of which bear resemblance to those later discussed in the papers noted above. Specifically, parallels are seen between what the authors describe as (a) tensional symptoms (a.k.a. functional dysphonia, vocal hyperfunction, or muscular tension dysphonia) and Trait Theory; (b) symbolic symptoms (a.k.a. conversion disorder or hysterical aphonia) and the Emotion-Processing Deficits Model; (c) hypochondriacal symptoms and MUS; (d) depressive-type symptoms and COSO; and finally, (e) combined organic and psychogenic processes and the novel CBT Model of functional dysphonia.

Summary. Overall, empirical evidence supporting the psychosocial theories described in this section is mixed. Additionally, much of the evidence presented in this section is cross-sectional and is therefore limited in its ability to inform on causal relationships. However, a group of factors emerges that may contribute to MTD-1: low social potency, depression, stress and anxiety, the presence of other *MUS*, and poor coping skills. Table 2 summarizes the different psychosocial mechanisms presented in this section, along with their main associated characteristics and triggers that are thought to participate in the development and maintenance of MTD-1.

Autonomic System Reaction (Fight or Flight Mechanism)

Demmink-Geertman and Dejonckere (2002) suggested that external and internal "threats," such as life situations, emotions, thoughts, and so forth, are stressors

Table 2. Psychosocial mechanisms: subcategories, characteristics, and triggers.

Author(s), year	Subcategories	Associated characteristics	Potential triggers
Baker (2008); Baker et al. (2013, 2014)	Emotion Processing Deficits	Implicit, general emotional arousal (undifferentiated emotions) experienced viscerally (somatization) Fight or flight state	Post–upper respiratory illness or surgery, organic voice disorder, phonotrauma, increased vocal load, life events, stressful situations, COSO
House & Andrews (1988)	COSO	Two requirements: Strong commitment within the communicative scenario; individual required to respond, but they do not to avoid exacerbating the conflict Likely no conscious awareness of the conflict	Stressful experiences
Dietrich et al. (2019); Dietrich & Verdolini Abbott (2012); Roy (2011); Roy & Bless (2000); Roy et al. (2019, 2000a, 2000b); Van Mersbergen et al. (2008)	The Dispositional Bases of Functional Dysphonia and Vocal Nodules, also known as The Trait Theory of Voice Disorders or Trait Theory	Low trait extraversion (introversion) with high trait neuroticism Behavioral inhibition system intensified by nonspecific arousal system (tendency not to perform a behavior is increased; Gray, 1970) Laryngeal muscle activity heightened or disorganized; passive avoidance of stimuli	Environmental signals/cues interpreted as punishment, frustrative nonreward (lack of expected reward in a scenario leads to frustration), novelty, or threat
Daniilidou et al. (2007); Deary & Miller (2011); Deary et al. (2018); Kollbrunner & Seifert (2017); Miller et al. (2014); Misono et al. (2020); O'Hara et al. (2011); Piersiala et al. (2020)	Medically Unexplained Symptoms (MUS) Novel expanded Cognitive Behavioral Therapy model of functional dysphonia (CBT model; Deary et al.)	Predisposing, precipitating, and perpetuating factors have a feedforward/feedback relationship with physical symptoms Presence of other MUS. CBT Model: Predisposing factors: Family history of dysphonia, anxiety, depression, coping style, frequency of vocal use, unhealthy/general perfectionism, high neuroticism, emotional inhibition, responsibility for others, trauma Perpetuating factors: general fatigue, anxiety, depression, avoidance of symptoms	MUS: Stress, loneliness, COSO, chronic somatic concerns CBT Model: Precipitating factors: frequency of vocal use, anxious coping style, viruses/ respiratory tract infections, sense of powerlessness, life events

(*table continues*)

Table 2. *(Continued).*

Author(s), year	Subcategories	Associated characteristics	Potential triggers
Misono et al. (2019)	Perceived Control	Maladaptive emotional and behavioral reactions due to low present perceived control over voice difficulties	Life events and trauma, heavy vocal demand, stressors, sensations of the voice problem, environmental irritants, reflux
Rammage et al. (1987)	Tensional symptoms (aka functional dysphonia, vocal hyperfunction, or muscular tension dysphonia) Symbolic symptoms Hypochondriacal symptoms Depressive-type symptoms Symbolic, tensional, and hypochondriacal symptoms Combined organic and psychogenic processes	Hyperactive nervous system leads to muscle hypertonicity Laryngeal muscular involvement unconsciously substituted for psychological conflict Anticipation of voice problems Suppression of the urge to cry or verbally display anger Combination of symbolic, tensional, and hypochondriacal symptoms Organic, psychological, and social factors predispose, precipitate, or perpetuate laryngeal symptoms	Disproportionate arousal and anxiety; personality Psychological conflict Physical sensations; personality traits (obsessive-compulsion, dependency, hypochondria). Urge to cry or express anger Symbolic, tensional, and hypochondriacal symptoms Edema, infection, polypoidal change, neoplasia, reflux esophagitis, acid laryngitis, health or voice-related anxiety

Note. MUS = medically unexplained symptom; COSO = conflict over speaking out; CBT = cognitive behavioral therapy.

capable of triggering overactivity of the autonomic nervous system (fight or flight response). In this model, the abnormal laryngeal activation observed in MTD-1 is one of many manifestations of the fight or flight reaction, which is likely to occur in individuals with an overly sensitive autonomic nervous system. Data from two studies were consistent with this hypothesis, although the authors cautiously did not characterize noted relationships as "causal." Specifically, the studies showed a higher prevalence of neurovegetative complaints in patients with "nonorganic habitual dysphonia" when compared to healthy controls, particularly in females (Demmink-Geertman & Dejonckere, 2002, 2008). Examples included cold hands and feet, excessive perspiration, tinnitus, lack of energy, allergies, sore throat, hyperventilation, and so forth (Demmink-Geertman & Dejonckere, 2002). The second study revealed a significant reduction in neurovegetative symptoms (voice and non–voice-related) following voice therapy, which the authors attributed to better regulation of the autonomic nervous system due to improved coping abilities (Demmink-Geertman & Dejonckere, 2008). Also relevant to the fight or flight mechanism, Helou et al. (2013, 2018) found an increase in laryngeal muscle activation (adductors and abductors) concurrent with autonomic nervous system activation in healthy speakers in both a cold pressor task and a public speech preparation task—providing oblique support regarding the viability of the autonomic system reaction hypothesis of MTD-1. A summary of the characteristics and triggers associated with the autonomic nervous system mechanism is provided in Table 3.

Some commonalities with psychosocial theories are found. The body's response to stressors is influenced by how a person copes with stress (Demmink-Geertman & Dejonckere, 2002). Relative to MTD-1, overlaps between psychosocial and autonomic mechanisms include an emphasis on stress, life situations, and coping abilities (Emotion-Processing Deficits Model, COSO, MUS/CBT Model, and Perceived Control Model). Emphasis on high autonomic arousal in the impulse to freeze or flee is analogous to positions in the Emotion-Processing Deficits Model (Baker et al., 2013, 2014) and Trait Theory of MTD-1 (Roy & Bless, 2000; Roy et al., 2000a, 2000b). Additionally, neurovegetative complaints mirror those described in the MUS/CBT Model. Despite these commonalities with psychosocial mechanisms, the autonomic system reaction was included as its own mechanistic category because of the unique role it plays in our proposed framework, described in detail later. Briefly, the level of autonomic activation is thought to modulate different relationships within the model, in such a way that high levels of autonomic arousal are likely to induce heightened perception and reaction to all types of triggers. Although autonomic arousal varies based on the person and the situation and is therefore affected by psychosocial factors, the level of arousal then acts as a moderating variable that influences motor adaptation processes in response to triggers, by dictating the intensity of the response.

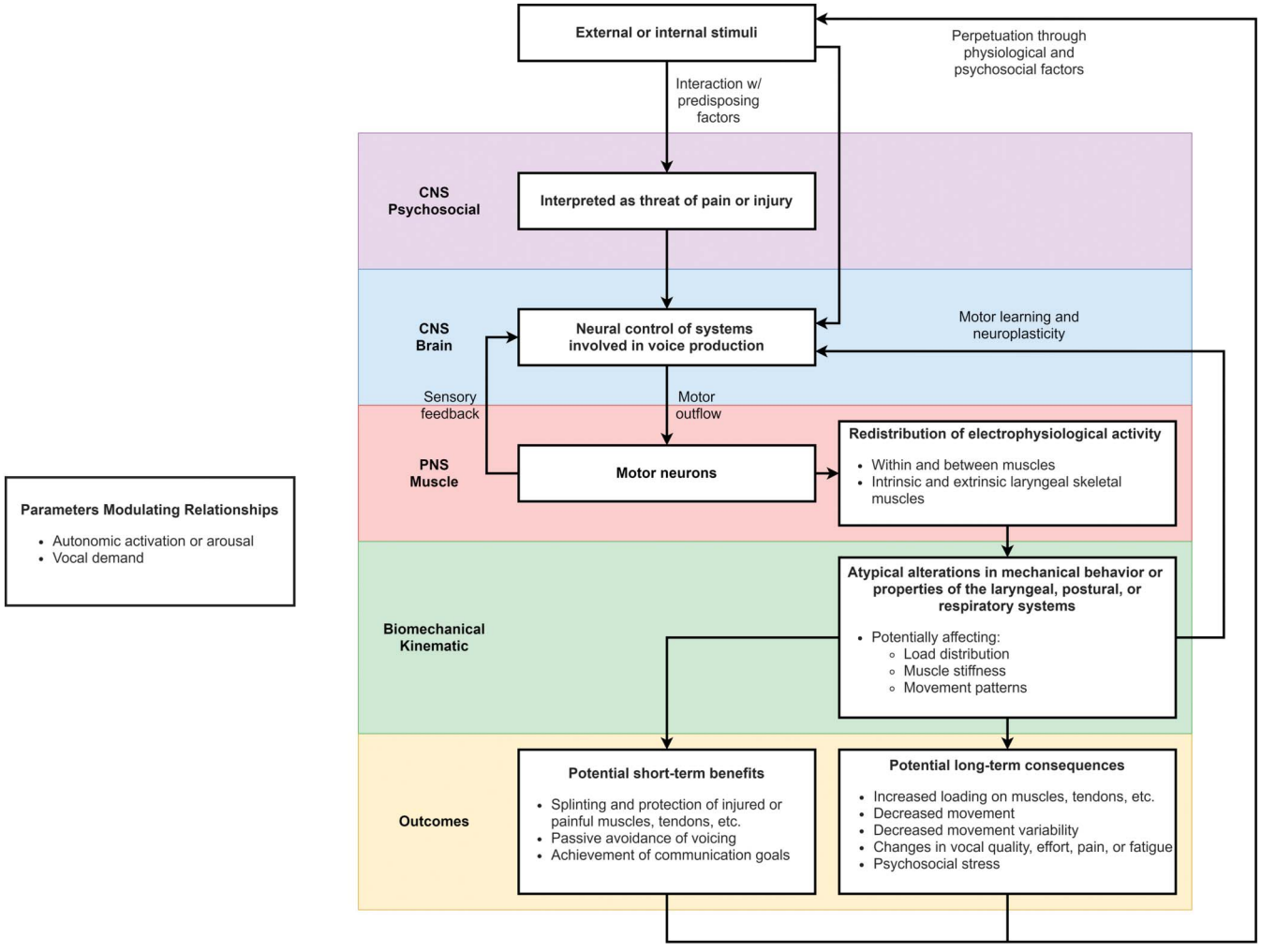
Sensorimotor Mechanisms

Disrupted sensorimotor integration and control. Stepp et al. (2017) hypothesized that patients with vocal hyperfunction have disordered auditory–motor integration systems and therefore that acute voice changes (e.g., as caused by an URI) can disrupt feedforward vocal motor planning, which relies in part on auditory control. Chronic changes in voice production may result (Stepp et al., 2017). Some evidence were provided consistent with this theory using a pitch shift paradigm in which many patients with hyperfunction exhibited an atypical adaptive response (their pitch followed the perturbation instead of opposing it) when compared to healthy controls (Stepp et al., 2017). Additionally, McKenna et al. (2020) found that patients with vocal hyperfunction had larger coefficients of variation in voice onset time when compared to healthy subjects, suggesting auditory discrimination deficits leading to larger "ranges of auditory targets" during voice production. Furthermore, Nagy et al. (2020) suggested that patients with hyperfunctional voice disorders (with or without vocal fold lesions) may have undiagnosed hearing impairments, causing them to speak loudly and effortfully because of disrupted sensorimotor mechanisms. However, studies including vocally healthy control

Table 3. Autonomic system reaction mechanism: subcategory, characteristics, and triggers.

Author(s), year	Subcategory	Associated characteristics	Potential triggers
Demmink-Geertman & Dejonckere (2002, 2008); Helou et al. (2013, 2018)	Autonomic system reaction (Fight or flight)	General neurovegetative complaints (e.g., cold hands/feet; excessive perspiration, tinnitus; lack of energy; etc.) Neurovegetative complaints with some relation to voice (e.g., allergy, sore throat, hyperventilation, neck pain while or after speaking, etc.) Laryngeal muscle response concomitant with autonomic nervous system activation	Internal or external "threat": Life situations, stress, anxiety, emotional problems, triggering memory, image, thought

Figure 1. Integrative model of the mechanisms underlying primary muscle tension dysphonia within the framework of motor adaptation to pain as suggested by Hodges (2011) and Hodges and Tucker (2011). CNS = central nervous system; PNS = peripheral nervous system. Figure adapted with permission from Hodges, 2011. Copyright © 2011 Elsevier.



groups would be necessary to further explore the latter hypothesis.

Further along the lines of disrupted sensorimotor control and integration, Ziethe et al. (2019) found differences in the response to a pitch shift in patients with MTD-1 and in healthy controls. Patients with MTD-1 demonstrated a larger change in fundamental frequency in comparison to healthy controls when the pitch feedback of their vocal productions was perturbed in a given direction. The authors interpreted these larger adaptive responses as symptomatic of disrupted kinesthetic feedback rather than impaired auditory–motor integration. This interpretation was supported by electroencephalogram signals showing normal auditory responses and shorter kinesthetic processing times in participants with MTD-1 when compared to healthy controls (Ziethe et al., 2019). Schultz-Coulon (1978) used auditory masking with white noise to compare kinesthetic control in vocally trained and untrained participants and in patients with vocal hyperfunction. During masking, all groups demonstrated similar increases in intensity, but patients with hyperfunction produced the largest shift in fundamental frequency, which the authors interpreted as evidence of "inefficient neuromuscular control." Last, the role of kinesthetic feedback was emphasized by Dworkin et al. (2000) in three case studies of patients with MTD-1 who did not benefit from behavioral voice therapy. All three patients experienced immediate vocal improvement following topical anesthesia of the tracheal, glottal, and supraglottal mucosa with injection of lidocaine through the cricothyroid membrane. The authors suggested that anesthesia may have interrupted an aberrant sensory feedback loop in those patients.

Theory of neural models/adaptive learning. Some authors have emphasized the role of the feedforward

system in the maintenance of MTD-1 without reference to an inherent sensorimotor disruption. Urberg-Carlson (2013) framed MTD-1 as the result of "adaptive learning." The hypothesis is that the error signal in the auditory and proprioceptive feedback, caused by the dysphonia, is habituated by the system and either (a) becomes part of the feedforward model, which starts to predict the motor pattern leading to dysphonia/fatigue or (b) is accepted by the system as a minimal error state and there is insufficient variability in vocal production to find a better state. Even though Urberg-Carlson's behavioral experiment was conducted on healthy speakers, there is evidence from brain imaging to support the role of disrupted sensory input in MTD-1. Kryshtopava et al. (2017) studied 10 patients with MTD-1 and 15 healthy speakers and found neurological signs of altered sensorimotor control in the patient group, as shown by reduced brain activity in sensory control–related areas and increased brain activity in motor control areas related to voice production. The authors suggested that sensory triggers (e.g., poor vocal quality, URI, stress, and vocal demands) may have altered the "neural models" in charge of voice production and induced disrupted patterns of motor activation. Results are in line with Urberg-Carlson's (2013) prediction that patients with MTD-1 would demonstrate less activation in the superior temporal gyrus compared to controls.

Laryngeal hypersensitivity. Morrison et al. (1999) proposed the irritable larynx syndrome hypothesis, suggesting a common etiology for MTD-1, chronic cough, globus pharyngeus, and episodic laryngospasm. The authors suggested that plasticity changes arising from injury or repetitive stimulation, viral infections, gastro-esophageal reflux disease (GERD), or even emotional states, can increase the sensitivity of laryngeal central nervous system neurons. Once the larynx becomes hyperexcitable, it is more likely to be triggered by external stimuli (e.g., chemicals, odors) than in its normally excitable state (Morrison et al., 1999). This neuropathological hypothesis is also central to Vertigan et al.'s (2008, 2013) research, which supported the laryngeal hypersensitivity syndrome proposition. In a study of 90 patients and 13 healthy controls, various tests compared sensory symptoms across diagnostic groups (MTD-1, chronic refractory cough, paradoxical vocal fold movement, globus pharyngeus, and healthy controls; Vertigan et al., 2013). "Cross stimulus responses" (occurring when a domain-specific stimulus causes a response in a different domain) were observed and were interpreted as evidence for a central sensitization common to these disorders. Main characteristics associated with the three sensorimotor mechanisms described in this section, as well as triggers that might elicit them, are summarized in Table 4.

Respiratory Mechanisms

CO_2-based (disruption in gas-exchange balance). Gillespie et al. (2013) proposed a theoretical framework based on dioxide-based (CO_2) metabolism to explain the etiology of MTD-1 in some patients. The hypothesis is that fluctuations in CO_2 levels may impact laryngeal activity because the larynx manages both respiratory homeostasis and phonation goals simultaneously. Gillespie et al. (2013, 2015) suggested that, in some patients, MTD-1 symptoms may reflect the larynx's attempt to regulate this homeostasis when confronted to internal perturbations—for example, as caused by an URI or by aberrant respiratory patterns. A "dysregulated voice motor control system" would have difficulty achieving this goal while at the same time regulating vocal production (Gillespie et al., 2015). Relevant to this hypothesis is the fact that abnormal CO_2 levels and atypical respiratory patterns have been reported in individuals with chronic anxiety (Gillespie et al., 2013; Han et al., 1998), a condition that has been cited as a predisposing and perpetuating factor in MTD-1 (Deary & Miller, 2011). To date, no direct evidence shows an association between disrupted CO_2 levels and aberrant laryngeal activity in patients with MTD-1. However, a variety of airflow patterns in patients with MTD-1 have corroborated the presence of atypical respiratory patterns (e.g., breath holding and longer inspiratory and expiratory airflow durations) in this population (Belsky et al., 2021; Gillespie et al., 2013; Gilman et al., 2019; Lu et al., 2021). Large interindividual variations in these studies, including diverse respiratory and laryngeal combinations affecting airflow data, emphasize the heterogeneity across MTD-1 patients.

Disruption in respiratory dynamics: phonatory lung volume. One of the most frequently studied respiratory parameters within the voice literature is lung volume during speech, specifically lung volume initiation (LVI) and termination. The relationship between vocal fold approximation and lung volume has been attributed by some authors to the mechanical relationship between the larynx and the diaphragm (referred to as the "tracheal pull"; Iwarsson & Sundberg, 1998; Iwarsson et al., 1998), as well as to the neurological coupling between laryngeal motoneurons and respiratory centers (Brancatisano et al., 1983). Other factors driving the relationship between lung volume and laryngeal activity include the requirement for conservation of airflow and respiratory effort as lung volume varies, which can promote laryngeal configurations consistent with vocal hyperfunction, including MTD-1 (Desjardins et al., 2021; Zhang, 2016).

Evidence supporting lung volume disruptions in MTD-1 remains scarce, but is gaining more and more attention. Koufman and Blalock (1988) mentioned overuse of the functional residual capacity, often associated with low tidal volumes ("shallow breathing"), in their observations of patients with *tension-fatigue*. The authors hypothesized that disruptions in speech breathing can result from various triggers such as URI or viral laryngitis. More recently, Lowell et al. (2008) showed that teachers with

Table 4. Sensorimotor mechanisms: subcategories, characteristics, and triggers.

Author(s), year	Subcategories	Associated characteristics	Potential triggers
Dworkin et al. (2000); McKenna et al. (2020); Nagy et al. (2020); Schultz-Coulon (1978); Stepp et al., (2017); Ziethe et al. (2019)	Disrupted sensorimotor integration/control	Abnormal response to pitch shifting and masking experiments Shorter voice onset time and greater coefficient of variation of voice onset time Reduced auditory discrimination abilities Potential hearing impairment	Disruptions in voice production pattern (e.g., URI; high voice-use), psychological factors (e.g., emotional stress), poor vocal quality
Kryshtopava et al. (2017); Urberg-Carlson (2013)	Theory of neural models/ adaptive learning	Neurological signs of altered sensorimotor control of phonation: reduced brain activity in sensory control-related areas; increased brain activity in motor control-related areas	Poor vocal quality, organic pathology (resolved), disrupted proprioceptive feedback, URI, stress, vocal demands
Morrison et al. (1999); Vertigan et al. (2006, 2008, 2013, 2014)	Irritable larynx syndrome/ laryngeal sensory dysfunction/ laryngeal hypersensitivity syndrome	Signs of extrathoracic hyperresponsiveness (coughing, throat clearing) Increased cough reflex sensitivity Episodic laryngospasms Possible associated symptom: globus pharyngeus Abnormal response to methacholine or hypertonic saline challenge Cross-stimulus responses (a domain-specific stimulus causing a response in a different domain)	Viral infection (including URI), Environmental stimuli (e.g., odor, chemicals, cold air), allergies, emotional distress, postural behavior, GERD/LPR, tissue injury, voice use, cough, foods/esophageal irritants, exertion, respiratory diseases (e.g., asthma), immune disorder

Note. URI = upper respiratory tract infection; GERD = gastroesophageal reflux disease; LPR = laryngopharyngeal reflux.

reported voice complaints (without vocal fold lesions) spoke at lower lung volumes when compared to vocally healthy teachers, especially when loudness was increased. As lung volume decreased, asymmetry between the contact-closing and opening phases of vocal fold vibration was amplified (as measured with electroglottography), which was interpreted by the authors as an indicator of increased muscle tone in the thyroarytenoid muscles. The presence of a relationship between vocal fatigue—a primary symptom in MTD-1—and lung volume was also reported by Hunter et al. (2019), who found an inverse linear relationship between raw pulmonary function (forced vital capacity, forced expiratory volume in one second, and peak expiratory flow) and perceived vocal fatigue in female teachers. Although participants with known structural alterations of the vocal folds were excluded from this study, no laryngeal examination was conducted. Together, these studies suggest that a lung volume that is not in line with speech goals could be involved in the development and/or the maintenance MTD-1. Additionally, Lowell et al. (2020) provided preliminary evidence to support the relevance of targeting LVI in patients with MTD-1 to improve voice outcomes using lung volume–based training, an intervention providing visual feedback of lung volume during speech.

Disruption in respiratory dynamics: control of the respiratory muscles. Morrison and Rammage (1993) suggested that insufficient control of airflow by the inspiratory muscles forces the vocal folds to act as a valve, thus avoiding uncontrolled airflow bursts in patients with MTD-1. This hypothesis parallels the proposition that musculoskeletal tension in MTD-1 could be the body's response to uncontrolled lung pressure (Rubin et al., 2011). Rubin et al. (2011) suggested that the reason for this protective reaction could lie in the mechanical disadvantage incurred by the diaphragm when the internal oblique muscles (IOM) are predominantly activated instead of the transverse abdominal muscles (TAM). This hypothesis stems from empirical observation of IOM-dominant patterns in a group of dysphonic patients with musculoskeletal tension, patterns different from those observed in vocally healthy individuals (Macdonald et al., 2012; Rubin et al., 2011). However, limited conclusions can be drawn from that study because the participant sample included patients with and without vocal fold lesions, and there was no control group. Moreover, results were partly contradicted by recent findings by Cryns et al. (2021) showing greater TAM recruitment (on ultrasound) during singing tasks in females with functional dysphonia (with diagnosis based solely on the Singing Voice Handicap Index score; Cohen et al., 2007) when compared to healthy females, while no difference was observed in terms of IOM activity. Importantly, participants were assessed in a supine position in the former study and in a standing position in the latter, which could partly explain the conflicting results. Nonetheless, Cryns et al. (2021) did find that female singers with functional dysphonia potentially had weaker TAM when compared to healthy singers, as evidenced by thinner muscles at rest and poorer results on the abdominal hollowing test evaluating recruitment of the TAM.

Table 5 displays the three respiratory mechanisms presented in this section, along with their specific characteristics and triggers.

Postural Mechanisms

Some authors have claimed that detrimental postural habits can lead to a high laryngeal position and decreased mobility of the cricothyroid joint, as observed in patients with MTD-1 (Kooijman et al., 2005; Rubin

Table 5. Respiratory mechanisms: subcategories, characteristics, and triggers.

Author(s), year	Subcategories	Associated characteristics	Potential triggers
Gillespie et al. (2013, 2015)	CO_2-based (disruption in gas-exchange balance)	Disrupted respiratory pattern (i.e., breath holding) Disruption in O_2/CO_2 homeostasis	Anxiety, perceived stress, lower airway disease (e.g., asthma, COPD), URI
Desjardins et al. (2021); Koufman & Blalock (1988); Lowell et al. (2008, 2020)	Disruption in respiratory dynamics: phonatory lung volume	Small tidal volume ("shallow breathing," "chest breathing") Speaking at low lung volume (use of functional residual capacity) Lack of increase in lung volume initiation during loud speaking	Viral laryngitis, URI, vocal demands (e.g., increased loudness), acute anxiety
Cryns et al. (2021); Morrison & Rammage (1993); Rubin et al. (2011)	Disruption in respiratory dynamics: control of respiratory muscles	Uncontrolled bursts of airflow Disrupted ratio of transverse abdominis and internal oblique muscles activity Sustained contraction of the SCM muscle	Body position, vocal genre (for singers), vocal technique

Note. COPD = chronic obstructive pulmonary disease; URI = upper respiratory infection; SCM = sternocleidomastoid.

et al., 2007). Rubin et al.'s theory describes mechanisms similar to a domino effect: A forward head posture strengthens the posterior deep neck extensors, leaving the anterior deep neck flexors lengthened and at a mechanical disadvantage. Next, the sternocleidomastoid muscle (SCM) takes over to stabilize the neck, thereby shortening the suprahyoid muscles and elevating the hyoid bone and larynx. The role of the SCM in voice problems was further highlighted in a study by Kooijman et al. (2005). In that study, a hypertonic SCM significantly predicted the worst Voice Handicap Index (VHI; Jacobson et al., 1997) score in 25 patients with a history of voice complaints along with a "posterior weight bearing" posture and an associated forward head position. The resulting cervical lordosis was hypothesized to disrupt the cricoid and thyroid cartilages' positions in relation to each other and to affect cricothyroid muscle function, essential to vocal fold tension adjustments (Kooijman et al., 2005; Rubin et al., 2000). Such limitations in thyroid tilt are sometimes referred to as "locked visor," especially by authors who emphasize the role of the cricothyroid space (or "cricothyroid visor") in the pathophysiology of musculoskeletal voice disorders (Dehqan & Ballard, 2021; Harris & Howard, 2018).

Together, observations from these studies warrant research regarding posture in patients with MTD-1 versus healthy subjects. While Franco et al. (2014) found a higher thoracic length curvature and kyphosis index in participants with dysphonia when compared to healthy speakers, the study included patients with or without lesions in their dysphonic group, thus limiting conclusions specific to MTD-1. Results from intervention studies may expand on the role of posture as a potential etiological and perpetuating mechanism in MTD-1. Tomlinson and Archer (2015)—who emphasized that patients with MTD-1 had muscle imbalances affecting the rhomboid, omohyoid, scalene, and transverse abdominis muscles, as well as the diaphragm and the anterior neck muscles—suggested addressing muscular imbalances using a "full-body approach," including postural work and stress and anxiety management. Nine women with MTD-1 received a 9-week intervention by a physical therapist (including manual therapy, stretches and exercises, relaxation techniques, and instructions on posture), after which most participants reported improvements in pain, functional status, voice-related quality of life, and cervical range of motion. Limitations of this study were the lack of a control group and concurrent SLP intervention in only a subset of participants. In a retrospective cohort study, Craig et al. (2015) found that patients with MTD-1 treated exclusively with physical therapy (addressing muscle imbalances, posture, cervical range of motion, and stress) experienced a decrease in VHI score (Jacobson et al., 1997) similar to that of patients who received voice therapy only. However, only those treated with voice therapy improved significantly more than the no-treatment control group.

There is an important lack of well-designed mechanistic studies assessing postural imbalances in patients with MTD-1 when compared to vocally healthy controls or to patients with other types of voice disorders. Due to this lack of detail on different postural mechanisms and their effect on voice production, only one mechanism is presented in Table 6, along with possible associated characteristics and triggers.

Inflammatory Mechanisms

"Primary" inflammatory mechanisms. Accounts of the mechanisms through which acid or nonacid reflux might create or sustain MTD-1 generally fall under two categories in the current review, here grouped into "primary" and "secondary" mechanisms. The "primary inflammatory" category argues that pepsin and other digestive enzymes may directly affect the cellular structure of the laryngeal and pharyngeal mucosa (Karkos et al., 2007). Bathing the laryngeal mucosa in refluxate may also have the potential to sensitize the adductory muscles of the larynx, such that the lateral cricoarytenoid, interarytenoid, thyroarytenoid, and even extrinsic laryngeal muscles respond aggressively to perceived threats to the airway,

Table 6. Postural mechanisms: subcategory, characteristics, and triggers.

Author(s), year	Subcategory	Associated characteristics	Potential triggers
Craig et al. (2015); Franco et al. (2014); Kooijman et al. (2005); Rubin et al. (2000, 2007); Tomlinson & Archer (2015)	Poor postural behavior	Decreased pitch regulation control Hypertonicity of the neck muscles Posterior weight bearing Anteroposition of the head Exaggerated thoracic kyphosis Exaggerated lordosis High and rounded shoulders Decreased jaw and cervical range of motion Short psoas muscle Neck stiffness Muscle cramping	Long periods of sitting in front of screens, intrinsic factors affecting posture: respiration (breathing pattern), neuromuscular fatigue, stress, anxiety, emotional triggers

(see, e.g., discussion of the "irritable larynx syndrome" and laryngeal hypersensitivity hypotheses of MTD-1; Morrison et al., 1999; Vertigan et al., 2008, 2013, 2014). Karkos et al. (2007) examined between-groups differences (MTD-1 vs. vocally healthy controls) in reflux episodes using 24-hr, dual-channel pH probe monitoring. Although the authors found statistically significant differences between vocally healthy and MTD-1 groups in two metrics (longest reflux episode at pharyngeal probe site; fraction of time with pH < 4 at pharyngeal probe site), they also acknowledged the limitations of pH probe monitoring as an experimental method. Their discussion of pepsin, carbonic anhydrase, and the potential tissue-damaging effects of refluxate suggests that Karkos et al. viewed cellular changes associated with long-term acid exposure as the main dangers of GERD/laryngopharyngeal reflux with respect to the development of MTD-1.

"Secondary" inflammatory mechanisms. The second category of theories surrounding inflammatory contributions to MTD-1 describes what might be called a "coordinated aerodigestive response" to reflux (Angsuwarangsee & Morrison, 2002; Van Houtte et al., 2012). Because the inferior pharyngeal constrictor muscle attaches to both the thyroid and cricoid cartilages, and the medial pharyngeal constrictor muscle to the hyoid bone, tonic or phasic contraction of these muscles during reflux events may alter the position of laryngeal structures, including the laryngeal cartilages. Abrupt increases in upper esophageal sphincter (UES) pressure have been previously registered during reflux events, stemming in principle from contraction of the pharyngeal constrictors (Torrico et al., 2000). Frequent reflux events may therefore result in chronic overcontraction of the pharyngeal constrictors, contributing to the signs and symptoms associated with MTD-1. Additionally, both the pharyngeal constrictors and the intrinsic laryngeal muscles are innervated by branches of the vagus nerve; co-contraction of the pharyngeal and laryngeal muscles may be possible during reflux events.

Angsuwarangsee and Morrison (2002) attempted to test the coordinated aerodigestive response hypothesis by comparing manually palpated muscle tension ratings in patients with and without both GERD and MTD-1. This study was motivated by older animal models in which porcine esophagi were perfused with acid in order to monitor the response of intrinsic laryngeal muscles (Gill & Morrison, 1998). Gill and Morrison (1998) found that acid perfusion of the distal esophagus—with no spillover into the larynx—elicited a statistically significant electromyographic (EMG) response in the thyroarytenoid muscles. Angsuwarangsee and Morrison (2002) found significant differences in manually palpated thyrohyoid, cricothyroid, and pharyngeal constrictor muscle tension between the MTD-1 group and the vocally healthy control group, as well as significant differences in manually palpated thyrohyoid and cricothyroid muscle tension between the GERD/LPR group and the healthy control group.

Like Angsuwarangsee and Morrison (2002), Van Houtte et al. (2012) also theorized that the sudden pressure increase in the UES associated with inferior pharyngeal constrictor contraction during reflux events (Torrico et al., 2000; Vilkman et al., 1996) could affect the intrinsic and extrinsic muscles of the larynx. Despite robust between-groups differences (MTD-1 vs. vocally healthy controls) in acoustic, laryngoscopic, voice range profile, and quality of life measures, however, Van Houtte et al. did not find any between-groups differences in UES pressure during resting breathing and a variety of voicing conditions. As the authors noted, factors as divergent as probe manufacture to sample size to the phonation times used to assess UES pressure could have contributed to their null results.

The two main hypotheses (primary and secondary) suggesting a physiological explanation for the inflammatory mechanism are summarized in Table 7, along with the main characteristics and triggers reported in the studies described in this section. Note that the triggers are the same for both subtypes and consist of GERD/LPR. Other authors have also compared the prevalence of reflux in MTD-1 patients versus patients with other voice disorders, without specifying a mechanism by which reflux might cause MTD-1. Koufman et al. (2000) found an abnormal pH probe prevalence rate of 50% across all voice disorder groups, and a 78% prevalence rate in the 23-person MTD subgroup (presumably MTD-1). However, because the authors did not examine GERD/LPR prevalence in healthy controls and because of the lack of between-

Table 7. Inflammatory mechanisms: subcategories, characteristics, and triggers.

Author(s), year	Subcategories	Associated characteristics	Potential triggers
Karkos et al. (2007)	Primary inflammatory mechanism (cellular response)	Positive 24-hr pH monitoring	GERD/LPR
Angsuwarangsee & Morrison (2002); Gill & Morrison (1998); Van Houtte et al. (2012)	Secondary inflammatory mechanism (coordinated aerodigestive response)	High upper esophageal sphincter pressure on manometry (test measuring esophageal motility)	GERD/LPR

Note. GERD = gastroesophageal reflux disease; LPR = laryngopharyngeal reflux.

groups statistical testing, it is difficult to gauge to what extent the prevalence rates reported for the MTD-1 subgroup differ from the prevalence rates in the broader population.

Neuromuscular Mechanisms

MTD-1 is often characterized by vocal fatigue, pain during voicing, visible or palpable tightness in the extrinsic laryngeal muscles, elevated hyoid position, and a host of abnormal laryngoscopic findings. In many cases, the presumptive etiology for these signs and symptoms has been neuromuscular, with authors citing increased muscle tension/stiffness at rest or increased neuromuscular activity during speech as the proximal cause for MTD-1. Consequently, many researchers have used EMG techniques to examine whether the amplitude, timing, or frequency content of the EMG signal in different extrinsic or intrinsic laryngeal muscles effectively distinguishes patients with MTD-1 from those without. Note that although other methods have been used to indirectly measure muscle activation (e.g., Adleberg et al., 2020), the focus of this section is EMG.

The five EMG studies in the current review (Hocevar-Boltezar et al., 1998; Khoddami et al., 2017; Lu et al., 2021; Redenbaugh & Reich, 1989; Van Houtte et al., 2013) used surface electromyographic techniques (sEMG). In our review, these studies were screened for adherence to the methodological recommendations of the International Society of Electrophysiology and Kinesiology (ISEK; Merletti & Di Torino, 1999). Two studies (Hocevar-Boltezar et al., 1998; Lu et al., 2021) adhered to fewer than 30% of the ISEK-recommended reporting criteria, making interpretation of their results difficult, while three studies (Khoddami et al., 2017; Redenbaugh & Reich, 1989; Van Houtte et al., 2013) adhered to more than 70% of the ISEK-recommended reporting criteria. Given that methodological flaws can directly affect the validity and replicability of EMG findings, adherence to these standards of EMG practice and reporting is more than nominally important.

The results of the five sEMG studies are summarized in Table 8, which shows the general trends in EMG amplitude signals observed in individuals with MTD-1 when compared to controls. Although several studies showed between-groups differences in EMG amplitude, roughly half of them found no significant between-groups differences. In some cases, null results may represent flaws in EMG methods (e.g., poor skin cleaning or absent impedance testing can result in noisier EMG amplitude baselines that do not reflect electrophysiological activity; Konrad, 2005); it is equally possible, however, that the intrinsic and extrinsic laryngeal muscles of patients with MTD-1 do not consistently exhibit stereotypical increases or decreases in electrophysiological activity. The proposed integrative framework section will attempt to reconcile the diverse EMG findings.

Observable Manifestations: Biomechanical and Kinematic Repercussions

Kinematic Correlates of MTD-1

In addition to examining the patterns of electrophysiological stimulation underlying MTD-1, investigations have been conducted on vocal fold and supraglottal kinematics in MTD-1. Although changes in laryngeal and supraglottal movement theoretically stem from changes in muscle activation, kinematics as a discipline does not focus on the causes of movement, only the effects of movement.

Preliminary evidence based on kinematic estimators of muscle stiffness shows increased intrinsic and extrinsic laryngeal muscle stiffness in subjects with MTD-1, relative to subjects with healthy voices (Azizi Ata et al., 2020; Stepp et al., 2010). Heller Murray et al. (2017) also found that, in comparison with healthy controls, speakers with MTD-1 had significantly lower relative fundamental frequency values during the offset of phonation, suggesting a smaller range of vocal fold tension available when transitioning between voiced vowels and voiceless consonants. Considering that this transition requires an increase in longitudinal vocal fold tension to interrupt vibration (Stevens, 1977), Heller Murray et al. proposed that individuals with vocal hyperfunction (including MTD-1) have heightened vocal fold tension at baseline, which limits their ability to further increase tension when transitioning to a voiceless consonant. Using high-speed digital imaging of the vocal

Table 8. Vote counts for general trends in electromyographic amplitude signals.

Trend	Infrahyoid muscles	Suprahyoid muscles	Cricothyroid muscle	Sternocleidomastoid muscle	Facial muscles (Orbicularis oris)
Increased relative to controls	Lu et al. (2021); Redenbaugh and Reich (1989)	Hocevar-Boltezar et al. (1998); Lu et al. (2021)	Lu et al. (2021)	Lu et al. (2021; only for loud phonation)	Hocevar-Boltezar et al. (1998)
No significant between-groups differences	Hocevar-Boltezar et al. (1998); Khoddami et al. (2017)	Van Houtte et al. (2013)	Khoddami et al. (2017)	Hocevar-Boltezar et al. (1998); Lu et al. (2021); Van Houtte et al. (2013)	
Decreased relative to controls	Van Houtte et al. (2013)				

Table 9. Criteria for diagnosis of primary muscle tension dysphonia.

Criterion for diagnosis	*n*
Presence of a voice problem in the absence of vocal fold pathology	27
Perceptually abnormal voice quality	12
Not specified	11
Presence of self-reported voice complaints	7
Specific laryngeal features	7
Presence of laryngeal muscle tension, as detected visually or manually	7
History of "vocal abuse or misuse" associated with a specific context (e.g., high vocal demands, stressful situations, etc.)	5
Dysphonia Severity Index score (Wuyts et al., 2000)	2

folds, authors have also commented on unusual kinematic patterns in patients with MTD-1, associated with perceptual judgments of diplophonia, glottal fry, breathy phonation, and pressed phonation (Patel et al., 2011). Observations of elevated hyoid and laryngeal position during phonation were also reported when comparing patients with MTD-1 to vocally healthy subjects (Lowell et al., 2012).

In a series of studies aimed at differentiating patients with nonphonotraumatic vocal hyperfunction (associated with MTD-1) from those with phonotraumatic vocal hyperfunction and healthy controls, findings indicated a reduced abruptness of vocal fold closure in the achievement of a target vocal output (Van Stan et al., 2021). Vocal inefficiency was noted in those patients, manifesting as greater subglottal pressure and open quotient for the production of a target intensity (Espinoza et al., 2017, 2020; Hillman et al., 1989), consistent with previous results (Zheng et al., 2012) showing greater subglottal pressure but not greater sound pressure level in patients with MTD-1 relative to healthy controls. The open quotient and reduced vocal fold closure abruptness may explain why these patients do not typically develop vocal fold lesions despite exhibiting high subglottal pressure values, in contrast to patients with phonotraumatic vocal hyperfunction (Espinoza et al., 2017, 2020).

Laryngoscopic Patterns of MTD-1

Abnormal laryngoscopic findings are among the most common clinical diagnostic criteria for MTD-1 and have featured prominently in several different phenotypic classifications (Koufman & Blalock, 1991; Morrison et al., 1986; Van Lawrence, 1987). These classifications typically emphasize anterior–posterior or medial compression at the level of the vocal folds or ventricular folds, as well as unusual glottal closure patterns (e.g., posterior glottal gaps) that are thought to stem from abnormal muscular activation. Associations have been investigated between the various MTD-1 phenotypes described in early classification papers and acoustic, aerodynamic, and biomechanical outcome measures. Additionally, studies have examined whether the glottal and supraglottal "postures" identified in these classifications are unique to patients with suspected MTD-1.

Stager et al. (2000) found significant differences in both ventricular fold compression and anterior–posterior compression of the larynx between subjects with MTD-1 and subjects with healthy voices, and noted that ventricular fold compression tended to occur as a transient phenomenon during speech, as contrasted with anterior–posterior compression, which tended to remain static even at rest. Behrman et al. (2003) observed that while anterior–posterior compression was significantly greater in subjects with MTD-1 compared to those with healthy voices, medial compression did not reliably distinguish the two groups. Sama et al. (2001) found no significant between-groups differences (MTD-1 vs. vocally healthy controls) in the six diagnostic criteria from the Van Lawrence (1987) classification or six separate diagnostic criteria from the Morrison and Rammage (1993) classification. Furthermore, over 60% of healthy controls exhibited one or more signs identified in these classification schemes as presumably indicative of MTD-1. Dabirmoghaddam et al. (2021) likewise found that, although patients with MTD-1 were consistently rated as having more severe laryngoscopic findings than vocally healthy controls on the Koufman and Blalock (1991) criteria, there was some overlap between the MTD-1 and control groups (e.g., an open posterior commissure and mediolateral compression of the ventricular folds appeared common in both groups).

Garaycochea et al. (2019) examined associations between acoustic or aerodynamic measures and the MTD phenotypes proposed by Van Lawrence (1987), Koufman and Blalock (1991), and Morrison and Rammage (1993). Various features pertaining to anteroposterior compression of the larynx and lateral compression (both at the level of the glottis and the supraglottis) had statistically significant associations with mean peak subglottal pressure—a feature that has been shown to differentiate patients with MTD-1 from healthy controls (Zheng et al., 2012). However, Garaycochea et al.'s study revealed no significant associations between the MTD-1 phenotype and transglottal flow, aerodynamic resistance, maximum phonation time, s:z ratio, phonorespiratory quotient, jitter, shimmer, or harmonic-to-noise ratio.

Proposed Integrative Framework

Motor Adaptation to Aversive or Threatening Stimuli

This integrative review identified seven categories of etiological mechanisms that have been proposed in the MTD-1 literature and summarized evidence around each mechanism as well as the biomechanical and kinematic outcomes of these mechanisms. The multiplicity of

psychosocial and physiological mechanisms thought to be involved in MTD-1, the complex interactions among mechanisms, and the apparent idiosyncrasy of the body's response to these interactions could explain the barriers to identification of features specific to MTD-1. As a recent example, Shembel et al. (2021) attempted to characterize MTD-1 using 15 acoustic and aerodynamic measures but were unable to reliably differentiate patients diagnosed with MTD-1 from patients with other voice disorders. The authors emphasized that while current clinical measures can describe a patient's vocal output, they generally cannot reveal the processes underlying that output (Shembel et al., 2021). It is precisely these latent processes, however, that may provoke and sustain chronic MTD-1. Although MTD-1 may manifest differently from one patient to another, a feature common to most reviewed mechanisms is the presence of an initial stimulus, a psychosocial or physiological perturbation, that leads to aversive outcomes or is interpreted as aversive and is therefore threatening to the speaker, either from a physical or psychosocial standpoint. Additionally, the symptoms most consistently reported since the early MTD-1 classifications are those related to *fatigue*, *discomfort,* and *pain* in the laryngeal region (Koufman & Blalock, 1988, 1991; Morrison et al., 1986; Morrison & Rammage, 1993). The suggestion that *the threat of aversive outcomes arising from an initial perturbation* is a defining feature of MTD-1 allowed us to expand on a previously established model of motor adaptation to pain or threat of pain (Hodges, 2011; Hodges & Tucker, 2011). Integrating the findings of the current review with this previous model, we have created a conceptual model that frames MTD-1 as the result of motor adaptations in response to the threat posed by aversive outcomes. These threats often arise from a precipitating physiological or psychological perturbation and are sufficient to create a cascade of motor adaptations that ultimately yield the chronic clinical signs and symptoms we associate with MTD-1.

An emerging body of literature from the domains of physical therapy and kinesiology (reviews in the works of Hodges, 2011, and Hodges & Tucker, 2011) proposes that pain/threat of pain and movement are intrinsically linked. Physiological accounts generally sort pain into three categories: *nociceptive pain*, caused by mechanical, thermal, ischemic, or inflammatory damage; *neuropathic pain*, caused by damage or disease that affects the somatosensory nervous system; and *nociplastic pain*, which (despite a more limited history of research) is thought to be caused by dysregulation of the central nervous system, causing distortion or amplification of nociceptive signals (Fitzcharles et al., 2021; Shraim et al., 2021). Readers may note that many of the putative causes of MTD-1 discussed in the current review fall into one or more of the three pain categories. However, because of limited research specifically addressing pain in patients with MTD-1, and because not all authors have explicitly considered pain as a central defining feature of MTD-1, the term *perturbation* will be used in the following section to be inclusive of a broader range of physiological and psychosocial stimuli that, while not strictly "painful," may be interpreted as aversive or threatening.

Presentation of the Framework

The following sections describe the suggested framework for chronic voice symptoms in MTD-1. The model, illustrated in Figure 1, was adapted from Hodges (2011) and Hodges and Tucker (2011) and integrates the evidence and theories specific to the mechanisms underlying MTD-1. It is separated into five main sections, based on the main level(s) at which the suggested mechanisms take place: central nervous system (psychosocial and sensorimotor mechanisms); peripheral nervous system (neuromuscular and some aspects of sensorimotor mechanisms); biomechanical and kinematic manifestations (respiratory, postural, and laryngeal); and concrete outcomes of these manifestations for the speaker. Note that there is not a perfect one-to-one mapping between the levels of the framework and the categories of mechanisms documented in this review because of the complex interactions within the framework and the fact that most of the mechanisms suggest coexisting changes at various levels of the model. Additionally, almost all categories of mechanisms were associated with initial triggers (such as GERD, URI, stress, postural habits, etc.), which are represented in the "External or internal stimuli" box at the very top of the model (see Figure 1) and can go on to impact the control of voice production. A description of each level of the framework follows.

From an Initial Stimulus to the Central Nervous System

Both internal stimuli (e.g., respiratory infection, reflux) and external stimuli (e.g., interpersonal situations, environmental irritants) represent perturbations that a speaker may perceive as threatening, especially in the context of high emotional reactivity. As suggested by multiple psychosocial hypotheses surrounding MTD-1, high trait introversion and neuroticism increase the likelihood of perception of threat, which in turn may trigger autonomic nervous system responses out of proportion with the frequency or intensity of stimuli. Autonomic nervous system response may then moderate other processes in the body germane to MTD-1, such as the perception of aversive sensations (Schlereth & Birklein, 2008). Psychosocially, high autonomic nervous system activity can reinforce extant overactive perceived threat (Roy et al., 2000a).

Even when not initially threatening, triggers can modify the neural control of voice production, as suggested by the sensorimotor theories in this review and

shown by the arrow going directly from the "External or internal stimuli box" to the "Neural control of systems involved in voice production" box in Figure 1. For example, disrupted sensory feedback (auditory or proprioceptive) caused by an acute voice disturbance during an URI may alter the neural control of voice production and reinforce a pattern of disordered motor activation (Kryshtopava et al., 2017; Urberg-Carlson, 2013). This loop is represented by the "Motor outflow" and "Sensory feedback" arrows in Figure 1. When sustained, these motor patterns are capable of generating aversive or threatening outcomes per se, such as physical discomfort, for example, due to an inefficient vocal production requiring greater effort to meet phonation goals (Espinoza et al., 2017, 2020). As suggested by Hillman et al. (2020) in their recently revisited framework, MTD-1 appears to be a disorder that primarily affects the *vocal demand response,* or the way that voice users modify their respiratory, phonatory, and articulatory strategies to meet a given level of vocal demand (Hunter et al., 2020) or respond to a triggering stimulus. Regardless of which specific precipitating factors initiate the associated motor adaptations, the pathological core of MTD-1 appears to be that the response persists even after the precipitating stimuli are no longer present (Hillman et al., 2020). Until now, the physiological explanation for this sustained response has remained largely undefined. We propose that the mechanisms underlying this perpetuation cycle may relate to how the body adapts to aversive or threatening perturbations, as described next.

The Peripheral Nervous System

The model created by Hodges (2011) and Hodges and Tucker (2011) and adapted in the current review (see Figure 1) proposes that muscle activity during pain and potentially during other forms of perturbation (Hodges et al., 2001) is redistributed *within and between muscles in ways that may be unique to the patient, task, or time*. In other words, there are many ways in which an individual may obtain temporary protection against a threatening perturbation, especially in systems with high redundancy (where the same outcome can be achieved by different muscles; Hodges, 2011). Based on this model, redistribution of muscle activity cannot be explained through simple increases or decreases in cortical excitability in response to an aversive stimulus, but occurs as a result of input from many points along the sensorimotor pathway and may increase responsiveness in one muscle or motor unit while decreasing responsiveness in another (Hodges, 2011). The idiosyncratic nature of the motor adaptation process could partly account for the lack of clear, consistent patterns of motor unit activity in MTD-1 in EMG studies of the extrinsic and intrinsic laryngeal muscles reported (as described in the Neuromuscular Mechanisms section), with the caveat that surface electromyography reveals only a composite electrophysiological signal from many motor units underlying the electrodes and cannot easily detect changes in the activity of individual motor units. With specific reference to MTD-1, redistribution of electrophysiological activity may occur between or within the intrinsic or extrinsic laryngeal muscles, respiratory muscles, or even postural muscles, and may be exacerbated by autonomic nervous system upregulation. As described previously, autonomic arousal moderates many different relationships within the proposed model and may therefore affect descending cortical drive to muscles (Amann et al., 2015) as well as muscle contractility (Ball, 2015). One of the possible implications of this idiosyncratic process is that changes in (para)laryngeal, postural, and respiratory muscle activation patterns, even when not initially present, could arise at a later stage of the voice disorder as a result of the motor adaptation process, with the associated biomechanical and kinematic effects described in the next section.

Biomechanical and Kinematic Effects

Based on Hodges and Tucker (2011), redistributed electrophysiological activity in response to painful or threatening stimuli may produce increased, decreased, or competing patterns of muscle contraction that ultimately lead to atypical alterations in the mechanical behavior of the affected systems. When sustained, these mechanical alterations may increasingly affect load distribution, muscle stiffness, and movement patterns in the (para)laryngeal, respiratory, or even postural systems, thus further disrupting vocal production, as shown in the "Biomechanical/Kinematic" section of the model in Figure 1. In fact, the present review reported suggested differences in respiratory and postural patterns in patients with MTD-1 when compared to healthy controls—although the evidence remains sparse. Additionally, while it is not clear from the reviewed publications whether the reported differences were a cause (initial stimulus) or biomechanical/kinematic consequence of the voice problem, Figure 1 shows that such changes are likely to impact voice production regardless. The final results of this cycle are unpredictable, with high variability between individuals. Moreover, most authors appear to agree that disturbances in one system (e.g., respiration, posture, intrinsic or extrinsic laryngeal muscle contraction, threat evaluation, sensory feedback) can quickly yield disturbances in other systems. For example, postural habits may impact voice indirectly through their effects on the respiratory musculature: strong kyphosis with rounded shoulders can shorten the abdominal and intercostal muscles and affect respiratory biomechanics for speech by limiting diaphragm and ribcage movement, thereby reducing inspiratory lung volume for speech (Wilson Arboleda & Frederick, 2008). Authors have also suggested a relationship between postural and respiratory mechanisms by

highlighting that breathing patterns can impact posture (Franco et al., 2014; Kooijman et al., 2005), and between posture and laryngeal muscle activation by suggesting that MTD-1 symptoms could hinder cervical proprioception coming from the neck (Faralli et al., 2017).

Short-Term and Long-Term Outcomes for the Speaker

Although the motor adaptations described in this proposed model may lead to some short-term benefits (e.g., protection of injured or painful muscles or tendons, passive avoidance of communication, or achievement of communication goals despite adverse psychosocial or physiological conditions), they may also lead to detrimental long-term consequences (e.g., increased muscle, tendon, or joint loading, decreased movement and movement variability, negative changes in vocal quality, fatigue, effort, or pain, increased loneliness or powerlessness, etc.; Deary & Miller, 2011; Hodges, 2011; Miller et al., 2014). Both short-term benefits and long-term consequences are shown in the bottom section of the model, named "Outcomes," in Figure 1. As motor adaptations become solidified through motor learning and neuroplasticity, they may eventually become "all consequence, no benefit," in that the conditions originally addressed by the adaptation no longer exist or the adaptation itself has become an impediment to effective voice production or communication. In fact, new aversive sensations resulting from mal(adaptive) behaviors that do not resolve with time may replace initial triggers and perpetuate the cycle of motor adaptation. From a psychosocial perspective, perception of the voice problem itself can precipitate further episodes of MTD-1 by making a person more sensitive to certain stimuli (Misono et al., 2019; Rammage et al., 1987), thus triggering one or more of the previously discussed physiological mechanisms. In Figure 1, this loop of perpetuation through physiological and psychosocial factors is represented by an arrow linking the "Outcomes" section of the model to the "External or internal stimuli" box: The physiological and psychosocial outcomes of the adaptation cascade become new threatening stimuli, leading to the perpetuation of the motor adaptation cycle.

In addition to autonomic activation/arousal, vocal demand may be seen as a moderating variable in the proposed model as it may accelerate movement through all of its stages: As demand increases, individuals have more opportunities to experience physical discomfort, threat of discomfort, or sensory disruption, prompting more neuromuscular and biomechanical adaptations, which are then consolidated via motor learning into motor plans (as shown by the "Motor learning and neuroplasticity" arrow in Figure 1). This is consistent with the parallel that has been made between MTD-1 and repetitive strain injuries, for example, in individuals using speech recognition systems (Olson et al., 2004).

Clinical Implications

Finally, some of the treatment modalities or principles currently used for MTD-1 bear a striking resemblance to those used for pain management in kinesiology and physical therapy, including EMG biofeedback (Yiu et al., 2005), Kinesio Taping (Mezzedimi et al., 2020), manual therapy (Aghadoost et al., 2020; D'haeseleer et al., 2013; Dehqan & Scherer, 2019; Dromey et al., 2008; Roy, 2008; Roy, Bless, et al., 1997; Roy et al., 2009; Van Lierde et al., 2010), and functional movement analysis with an emphasis on learning new, biomechanically efficient movement patterns, such as variations of resonant voice therapy (Watts et al., 2019), flow phonation (Watts et al., 2015, 2019), and semi-occluded vocal tract exercises including vocal function exercises (Guzmán et al., 2016; Nguyen & Kenny, 2009; Stemple et al., 1994). CBT or other counseling treatments for pain or psychosocial distress are also gaining more attention in the field of voice (e.g., Daniilidou et al., 2007; Deary et al., 2018; Miller et al., 2014). Although a review of the exact mechanisms of each of these treatment modalities in relation to the proposed model is beyond the scope of this review article, it is reasonable to assume that each treatment acts at one or more levels of the model: Circumlaryngeal manual therapy, for example, might alleviate pain directly while also reducing muscle stiffness, and resonant voice therapy might alter motor learning while also minimizing the negative effects of high vocal demand. Additionally, counseling strategies may lessen the impact of psychosocial factors on the perpetuation of the cycle. The current model argues for a complementary approach—rather than a competitive approach—to MTD-1 treatment. Indeed, nuanced, individualized treatment approaches may be necessary to address the individualized motor adaptations to noxious stimuli that this model of MTD-1 proposes.

Limitations

Limitations of the Studies and the Literature

Significant advances have been made in the field of voice science to disentangle the specific mechanisms underlying MTD-1. To help move research forward on this topic, we have identified limitations in the reviewed studies and in the broader literature, as described next.

A considerable number of studies were excluded from this review on the grounds that participants with different voice disorders were grouped together, most commonly MTD-1 and MTD-2. Although Morrison et al. (1986) and others have suggested that MTD-1 can

lead to the mucosal changes aligned with MTD-2 (e.g., vocal fold nodules), it is generally accepted that chronic fibrous nodules and MTD-1 have different pathophysiologies (Hillman et al., 2020). Averaging the experimental results of both disorders may obscure otherwise potentially significant findings for one of the groups and limit inference. One solution would be to conduct post hoc analyses to compare the two subgroups, as in some studies in the current review (McKenna et al., 2020; Stepp et al., 2017). Another identified limitation in some of the reviewed studies was the lack of a vocally healthy control group or other voice disorder group when assessing the features characteristic of MTD-1. Widespread use of control groups would help to clarify which signs, symptoms, or mechanisms of action are unique to MTD-1, and which are shared by vocally healthy speakers and/or speakers with other voice disorders.

One of the main limitations of the MTD-1 literature is inconsistency in the inclusion criteria for individuals with MTD-1, a limitation largely driven by MTD-1's current status as a diagnosis of exclusion. As others have noted (Samargia et al., 2016), studies that aim to define MTD-1 depend on which participants are included in the study; which participants are included in turn depends on diagnosis criteria, creating a loop of circular logic. The issue of inclusion is further complicated by discrepancies in terminology that have evolved over the years. The term *functional dysphonia*, for example, is still widely used as a proxy for MTD-1 despite concerns about its ambiguity (Hacki et al., 2022).

Overall, this review revealed a lack of strong empirical data to support most of the theoretical claims regarding underlying mechanisms in MTD-1: On a total of 100 included papers, only 42 met our criteria to be considered as "empirical." In addition, because of the cross-sectional nature of most empirical studies, their ability to speak to *precipitating* etiological mechanisms remains limited. Further high-quality studies are needed to support or challenge the etiological hypotheses of MTD-1 and guide diagnosis and treatment of patients experiencing this disorder.

Limitations of the Review and Future Studies

The framework that we propose is built in large part on evidence from the pain literature and was adapted to integrate the different etiologies and triggers suggested for MTD-1. However, the framework should be re-evaluated and updated as more evidence is gathered in the voice literature. In the meantime, we hope that the proposed model, within the paradigm of motor adaptation to pain (Hodges, 2011; Hodges & Tucker, 2011), provides a unifying framework allowing us to reconcile the various mechanisms that have been proposed to underlie MTD-1, to help identify gaps in our understanding of this disorder, and to direct research in constructive ways. For example, future studies could explore motor adaptations in the extralaryngeal muscles (and potentially in other muscles involved in voice production) in response to the delivery of aversive stimuli to the laryngeal receptors in speakers with and without MTD-1.

Another limitation of the framework stems from the limitations of the review process itself. First, we included only publications in English and in French, and consequently, it is possible that relevant articles were excluded. Similarly, the search strategy may have been too narrow to detect all publications suggesting or providing empirical support for mechanisms underlying MTD-1. Nonetheless, we were able to identify seven categories and 19 subcategories of mechanisms hypothesized to play a role in the development and/or maintenance of MTD-1. Note that the data extraction process to identify and classify the categories of mechanisms and their subcategories, although performed using a consensus approach, has limitations in terms of reliability as it is subject to the authors' interpretation. It is possible that a different group of authors would have come up with a slightly different classification to organize the data.

Conclusions

The current integrative review surveyed the literature surrounding MTD-1 and its contributing factors in an attempt to thoroughly inventory—within the bounds of the study's inclusion criteria—the disorder's proposed underlying mechanisms. Mechanisms were organized into seven categories (psychosocial, autonomic, sensorimotor, respiratory, postural, inflammatory, and neuromuscular), and the evidence supporting each was critically appraised, highlighting gaps to be addressed in future studies. Synthesis of the existing literature generated an integrative model of MTD-1 inspired by a theoretical framework on motor adaptation to pain (Hodges, 2011; Hodges & Tucker, 2011), particularly due to MTD-1's apparent function as a motor adaptation to various internal or external stimuli that may be perceived as threatening, painful, or otherwise aversive. Within this paradigm, physiological responses (e.g., redistributed electrophysiological activity) to perturbations (e.g., threatening stimuli, pain, etc.) are compounded and maintained through motor learning, even after the original perturbations are removed—resulting in a chronic voice disorder. Finally, we emphasized the role of individualized assessment and intervention at multiple levels of the framework in the interest of optimal treatment outcomes.

Acknowledgments

This study was supported in part by the R01 DC017923 from the National Institute on Deafness and Other

Communication Disorders (PI: Katherine Verdolini Abbott). The authors would like to acknowledge the contribution of Lauren Ondrejack, who provided valuable help with the compilation of data and manuscript formatting and editing.

References

Adleberg, J., O'Connell Ferster, A. P., Benito, D. A., & Sataloff, R. T. (2020). Detection of muscle tension dysphonia using Eulerian video magnification: A pilot study. *Journal of Voice, 34*(4), 622–628. https://doi.org/10.1016/j.jvoice.2019.02.006

Aghadoost, S., Jalaie, S., Khatoonabadi, A. R., Dabirmoghaddam, P., & Khoddami, S. M. (2020). A study of vocal facilitating techniques compared to manual circumlaryngeal therapy in teachers with muscle tension dysphonia. *Journal of Voice, 34*(6), 936.e11–963.e21. https://doi.org/10.1016/j.jvoice.2019.06.002

Amann, M., Sidhu, S. K., Weavil, J. C., Mangum, T. S., & Venturelli, M. (2015). Autonomic responses to exercise: Group III/IV muscle afferents and fatigue. *Autonomic Neuroscience, 188,* 19–23. https://doi.org/10.1016/j.autneu.2014.10.018

Andrea, M., Dias, Ó., Andrea, M., & Figueira, M. L. (2017). Functional voice disorders: The importance of the psychologist in clinical voice assessment. *Journal of Voice, 31*(4), 507.e13–507.e22. https://doi.org/10.1016/j.jvoice.2016.10.013

Angsuwarangsee, T., & Morrison, M. (2002). Extrinsic laryngeal muscular tension in patients with voice disorders. *Journal of Voice, 16*(3), 333–343. https://doi.org/10.1016/S0892-1997(02)00105-4

Aronson, A. E. (1990). *Clinical voice disorders: An interdisciplinary approach* (3rd ed.). Thieme.

Azizi Ata, N., Khoddami, S. M., Babaei-Ghazani, A., Izadi, F., & Maroufizadeh, S. (2020). Strain elastosonography measurement in patients with primary muscle tension dysphonia compared with healthy speakers: A pilot study. *Journal of Voice*. https://doi.org/10.1016/j.jvoice.2020.05.026

Baker, J. (2008). The role of psychogenic and psychosocial factors in the development of functional voice disorders. *International Journal of Speech-Language Pathology, 10*(4), 210–230. https://doi.org/10.1080/17549500701879661

Baker, J., Ben-Tovim, D., Butcher, A., Esterman, A., & McLaughlin, K. (2007). Development of a modified diagnostic classification system for voice disorders with inter-rater reliability study. *Logopedics, Phoniatrics, Vocology, 32*(3), 99–112. https://doi.org/10.1080/14015430701431192

Baker, J., Ben-Tovim, D., Butcher, A., Esterman, A., & McLaughlin, K. (2013). Psychosocial risk factors which may differentiate between women with functional voice disorder, organic voice disorder and a control group. *International Journal of Speech-Language Pathology, 15*(6), 547–563. https://doi.org/10.3109/17549507.2012.721397

Baker, J., Oates, J. M., Leeson, E., Woodford, H., & Bond, M. J. (2014). Patterns of emotional expression and responses to health and illness in women with functional voice disorders (MTVD) and a comparison group. *Journal of Voice, 28*(6), 762–769. https://doi.org/10.1016/j.jvoice.2014.03.005

Ball, D. (2015). Metabolic and endocrine response to exercise: Sympathoadrenal integration with skeletal muscle. *The Journal of Endocrinology, 224*(2), R79–R95. https://doi.org/10.1530/JOE-14-0408

Behrman, A., Dahl, L. D., Abramson, A. L., & Schutte, H. K. (2003). Anterior–posterior and medial compression of the supraglottis: Signs of nonorganic dysphonia or normal postures. *Journal of Voice, 17*(3), 403–410. https://doi.org/10.1067/S0892-1997(03)00018-3

Belsky, M. A., Rothenberger, S. D., Gillespie, A. I., & Gartner-Schmidt, J. L. (2021). Do phonatory aerodynamic and acoustic measures in connected speech differ between vocally healthy adults and patients diagnosed with muscle tension dysphonia. *Journal of Voice, 35*(4), 663.e1–663.e7. https://doi.org/10.1016/j.jvoice.2019.12.019

Brancatisano, T., Collett, P. W., & Engel, L. A. (1983). Respiratory movements of the vocal cords. *Journal of Applied Physiology, 54*(5), 1269–1276. https://doi.org/10.1152/jappl.1983.54.5.1269

Buck, F., Drinnan, M., Wilson, J., & Barnard, I. S. (2007). What are the illness perceptions of people with dysphonia: A pilot study. *The Journal of Laryngology and Otology, 121*(1), 31–39. https://doi.org/10.1017/S0022215106004002

Cohen, S. M., Jacobson, B. H., Garrett, C. G., Noordzij, J. P., Stewart, M. G., Attia, A., Ossoff, R. H., & Cleveland, T. F. (2007). Creation and validation of the singing voice handicap index. *Annals of Otology, Rhinology & Laryngology, 116*(6), 402–406. https://doi.org/10.1177/000348940711600602

Craig, J., Tomlinson, C., Stevens, K., Kotagal, K., Fornadley, J., Jacobson, B., Garrett, C. G., & Francis, D. O. (2015). Combining voice therapy and physical therapy: A novel approach to treating muscle tension dysphonia. *Journal of Communication Disorders, 58,* 169–178. https://doi.org/10.1016/j.jcomdis.2015.05.001

Cryns, N., Schemmann, H., Zalpour, C., & von Piekartz, H. (2021). Are there differences in abdominal muscle function in female singers with and without voice disorders? An observational study. *Journal of Voice*. https://doi.org/10.1016/j.jvoice.2021.03.015

D'haeseleer, E., Claeys, S., & Van Lierde, K. (2013). The effectiveness of manual circumlaryngeal therapy in future elite vocal performers: A pilot study. *The Laryngoscope, 123*(8), 1937–1941. https://doi.org/10.1002/lary.24050

Dabirmoghaddam, P., Aghajanzadeh, M., Erfanian, R., Aghazadeh, K., Sohrabpour, S., Firouzifar, M., Maroufizadeh, S., & Nikravesh, M. (2021). Comparative study of increased supraglottic activity in normal individuals and those with muscle tension dysphonia (MTD). *Journal of Voice, 35*(4), 554–558. https://doi.org/10.1016/j.jvoice.2019.12.003

Daniilidou, P., Carding, P., Wilson, J., Drinnan, M., & Deary, V. (2007). Cognitive behavioral therapy for functional dysphonia: A pilot study. *Annals of Otology, Rhinology & Laryngology, 116*(10), 717–722. https://doi.org/10.1177/000348940711601002

Deary, V., McColl, E., Carding, P., Miller, T., & Wilson, J. (2018). A psychosocial intervention for the management of functional dysphonia: Complex intervention development and pilot randomised trial. *Pilot and Feasibility Studies, 4*(1), 46. https://doi.org/10.1186/s40814-018-0240-5

Deary, V., & Miller, T. (2011). Reconsidering the role of psychosocial factors in functional dysphonia. *Current Opinion in Otolaryngology and Head and Neck Surgery, 19*(3), 150–154. https://doi.org/10.1097/MOO.0b013e328346494d

Dehqan, A., & Ballard, K. J. (2021). An evaluation of short-term treatment outcomes of cricothyroid visor maneuver: A proof-of-concept pilot study. *Journal of Voice, 35*(2), 330.e1–330.e7. https://doi.org/10.1016/j.jvoice.2019.09.016

Dehqan, A., & Scherer, R. C. (2019). Positive effects of manual circumlaryngeal therapy in the treatment of muscle tension dysphonia (MTD): Long term treatment outcomes. *Journal of Voice, 33*(6), 866–871. https://doi.org/10.1016/j.jvoice.2018.07.010

Demmink-Geertman, L., & Dejonckere, P. H. (2002). Nonorganic habitual dysphonia and autonomic dysfunction. *Journal of Voice, 16*(4), 549–559. https://doi.org/10.1016/S0892-1997(02)00130-3

Demmink-Geertman, L., & Dejonckere, P. H. (2008). Neurovegetative symptoms and complaints before and after voice therapy for nonorganic habitual dysphonia. *Journal of Voice, 22*(3), 315–325. https://doi.org/10.1016/j.jvoice.2006.10.011

Desjardins, M., Verdolini Abbott, K., & Zhang, Z. (2021). Computational simulations of respiratory-laryngeal interactions and their effects on lung volume termination during phonation: Considerations for hyperfunctional voice disorders. *The Journal of the Acoustical Society of America, 149*(6), 3988–3999. https://doi.org/10.1121/10.0005063

Dietrich, M., & Verdolini Abbott, K. (2012). Vocal function in introverts and extraverts during a psychological stress reactivity protocol. *Journal of Speech, Language, and Hearing Research, 55*(3), 973–987. https://doi.org/10.1044/1092-4388(2011/10-0344)

Dietrich, M., Verdolini Abbott, K., Gartner-Schmidt, J., & Rosen, C. A. (2008). The frequency of perceived stress, anxiety, and depression in patients with common pathologies affecting voice. *Journal of Voice, 22*(4), 472–488. https://doi.org/10.1016/j.jvoice.2006.08.007

Dietrich, M., Andreatta, R. D., Jiang, Y., & Stemple, J. C. (2020). Limbic and cortical control of phonation for speech in response to a public speech preparation stressor. *Brain Imaging and Behavior, 14*(5), 1696–1713. https://doi.org/10.1007/s11682-019-00102-x

Dromey, C., Nissen, S. L., Roy, N., & Merrill, R. M. (2008). Articulatory changes following treatment of muscle tension dysphonia: Preliminary acoustic evidence. *Journal of Speech, Language, and Hearing Research, 51*(1), 196–208. https://doi.org/10.1044/1092-4388(2008/015)

Dworkin, J. P., Meleca, R. J., Simpson, M. L., & Garfield, I. (2000). Use of topical lidocaine in the treatment of muscle tension dysphonia. *Journal of Voice, 14*(4), 567–574. https://doi.org/10.1016/S0892-1997(00)80012-0

Eastwood, C., Madill, C., & McCabe, P. (2015). The behavioural treatment of muscle tension voice disorders: A systematic review. *International Journal of Speech-Language Pathology, 17*(3), 287–303. https://doi.org/10.3109/17549507.2015.1024169

Espinoza, V. M., Mehta, D. D., Van Stan, J. H., Hillman, R. E., & Zañartu, M. (2020). Glottal aerodynamics estimated from neck-surface vibration in women with phonotraumatic and nonphonotraumatic vocal hyperfunction. *Journal of Speech, Language, and Hearing Research, 63*(9), 2861–2869. https://doi.org/10.1044/2020_JSLHR-20-00189

Espinoza, V. M., Zañartu, M., Van Stan, J. H., Mehta, D. D., & Hillman, R. E. (2017). Glottal aerodynamic measures in women with phonotraumatic and nonphonotraumatic vocal hyperfunction. *Journal of Speech, Language, and Hearing Research, 60*(8), 2159–2169. https://doi.org/10.1044/2017_JSLHR-S-16-0337

Eysenck, H. J., & Eysenck, S. B. G. (1994). *Manual of the Eysenck Personality Questionnaire: (EPQ-R Adult)*. Educational and Industrial Testing Service.

Falanga, C., Costa, G., Volpe, U., Saracino, D., Lechien, J. R., Estomba, C. M. C., Tripodi, M., Cammaroto, G., Angelillo, N., Nacci, A., & Barillari, M. R. (2020). Psychological distress in a sample of adult Italian patients affected by vocal nodules and muscle-tension dysphonia: Preliminary results. *Journal of Voice*. https://doi.org/10.1016/j.jvoice.2020.12.011

Faralli, M., Lapenna, R., Pagliari, J., Longari, F., D'Ascanio, L., & Ricci, G. (2017). The effect of speech rehabilitation therapy for muscle tension dysphonia on global postural strategy. *Hearing, Balance and Communication, 15*(4), 271–281. https://doi.org/10.1080/21695717.2017.1379677

Fitzcharles, M.-A., Cohen, S. P., Clauw, D. J., Littlejohn, G., Usui, C., & Häuser, W. (2021). Nociplastic pain: Towards an understanding of prevalent pain conditions. *Lancet (London, England), 397*(10289), 2098–2110. https://doi.org/10.1016/S0140-6736(21)00392-5

Franco, D., Martins, F., Andrea, M., Fragoso, I., Carrão, L., & Teles, J. (2014). Is the sagittal postural alignment different in normal and dysphonic adult speakers. *Journal of Voice, 28*(4), 523.e1–523.e5238. https://doi.org/10.1016/j.jvoice.2014.01.002

Garaycochea, O., Navarrete, J. M. A., Del Río, B., & Fernández, S. (2019). Muscle tension dysphonia: Which laryngoscopic features can we rely on for diagnosis. *Journal of Voice, 33*(5), 812.e15–812.e18. https://doi.org/10.1016/j.jvoice.2018.04.015

Gill, C., & Morrison, M. D. (1998). Esophagolaryngeal reflex in a porcine animal model. *The Journal of Otolaryngology, 27*(2), 76–80.

Gillespie, A. I., Gartner-Schmidt, J., Rubinstein, E. N., & Abbott, K. V. (2013). Aerodynamic profiles of women with muscle tension dysphonia/aphonia. *Journal of Speech, Language, and Hearing Research, 56*(2), 481–488. https://doi.org/10.1044/1092-4388(2012/11-0217)

Gillespie, A. I., Slivka, W., Atwood, C. W., Jr., & Verdolini Abbott, K. (2015). The effects of hyper- and hypocapnia on phonatory laryngeal airway resistance in women. *Journal of Speech, Language, and Hearing Research, 58*(3), 638–652. https://doi.org/10.1044/2015_JSLHR-S-13-0270

Gilman, M., Maira, C., & Hapner, E. R. (2019). Airflow patterns of running speech in patients with voice disorders. *Journal of Voice, 33*(3), 277–283. https://doi.org/10.1016/j.jvoice.2017.12.004

Gray, J. A. (1970). The psychophysiological basis of introversion-extraversion. *Behaviour Research and Therapy, 8*(3), 249–266. https://doi.org/10.1016/0005-7967(70)90069-0

Guzmán, M., Castro, C., Madrid, S., Olavarria, C., Leiva, M., Muñoz, D., Jaramillo, E., & Laukkanen, A. M. (2016). Air pressure and contact quotient measures during different semi-occluded postures in subjects with different voice conditions. *Journal of Voice, 30*(6), 759.e1–759.e10. https://doi.org/10.1016/j.jvoice.2015.09.010

Hacki, T., Moerman, M., & Rubin, J. S. (2022). 'Malregulative' rather than 'functional' dysphonia: A new etiological terminology framework for phonation disorders- A position paper by the Union of European Phoniatricians (UEP). *Journal of Voice, 36*(1), 50–53. https://doi.org/10.1016/j.jvoice.2020.04.032

Han, J. N., Stegen, K., Schepers, R., Van den Bergh, O., & Van de Woestijne, K. P. (1998). Subjective symptoms and breathing pattern at rest and following hyperventilation in anxiety and somatoform disorders. *Journal of Psychosomatic Research, 45*(6), 519–532. https://doi.org/10.1016/s0022-3999(98)00044-0

Harris, T., & Howard, D. M. (2018). *The voice clinic handbook*. Compton Publishing Limited.

Haselden, K., Powell, T., Drinnan, M., & Carding, P. (2009). Comparing health locus of control in patients with spasmodic dysphonia, functional dysphonia and nonlaryngeal dystonia. *Journal of Voice, 23*(6), 699–706. https://doi.org/10.1016/j.jvoice.2008.03.011

Heller Murray, E. S., Lien, Y.-A. S., Van Stan, J. H., Mehta, D. D., Hillman, R. E., Pieter Noordzij, J., & Stepp, C. E. (2017). Relative fundamental frequency distinguishes between phonotraumatic and non-phonotraumatic vocal hyperfunction. *Journal of Speech, Language, and Hearing Research, 60*(6), 1507–1515. https://doi.org/10.1044/2016_JSLHR-S-16-0262

Helou, L. B., Rosen, C. A., Wang, W., & Verdolini Abbott, K. (2018). Intrinsic laryngeal muscle response to a public speech preparation stressor. *Journal of Speech, Language, and Hearing Research, 61*(7), 1525–1543. https://doi.org/10.1044/2018_JSLHR-S-17-0153

Helou, L. B., Wang, W., Ashmore, R. C., Rosen, C. A., & Abbott, K. V. (2013). Intrinsic laryngeal muscle activity in

response to autonomic nervous system activation. *The Laryngoscope, 123*(11), 2756–2765. https://doi.org/10.1002/lary.24109

Hillman, R. E., Holmberg, E. B., Perkell, J. S., Walsh, M., & Vaughan, C. (1989). Objective assessment of vocal hyperfunction: An experimental framework and initial results. *Journal of Speech and Hearing Research, 32*(2), 373–392. https://doi.org/10.1044/jshr.3202.373

Hillman, R. E., Stepp, C. E., Van Stan, J. H., Zañartu, M., & Mehta, D. D. (2020). An updated theoretical framework for vocal hyperfunction. *American Journal of Speech-Language Pathology, 29*(4), 2254–2260. https://doi.org/10.1044/2020_AJSLP-20-00104

Hocevar-Boltezar, I., Janko, M., & Zargi, M. (1998). Role of surface EMG in diagnostics and treatment of muscle tension dysphonia. *Acta Oto-Laryngologica, 118*(5), 739–743. https://doi.org/10.1080/00016489850183287

Hodges, P. W. (2011). Pain and motor control: From the laboratory to rehabilitation. *Journal of Electromyography and Kinesiology, 21*(2), 220–228. https://doi.org/10.1016/j.jelekin.2011.01.002

Hodges, P. W., Heijnen, I., & Gandevia, S. C. (2001). Postural activity of the diaphragm is reduced in humans when respiratory demand increases. *The Journal of Physiology, 537*(Pt. 3), 999–1008. https://doi.org/10.1111/j.1469-7793.2001.00999.x

Hodges, P. W., & Tucker, K. (2011). Moving differently in pain: A new theory to explain the adaptation to pain. *Pain, 152*(3), S90–S98. https://doi.org/10.1016/j.pain.2010.10.020

House, A. O., & Andrews, H. B. (1988). Life events and difficulties preceding the onset of functional dysphonia. *Journal of Psychosomatic Research, 32*(3), 311–319. https://doi.org/10.1016/0022-3999(88)90073-6

Hunter, E. J., Cantor-Cutiva, L. C., Van Leer, E., van Mersbergen, M., Nanjundeswaran, C. D., Bottalico, P., Sandage, M. J., & Whitling, S. (2020). Toward a consensus description of vocal effort, vocal load, vocal loading, and vocal fatigue. *Journal of Speech, Language, and Hearing Research, 63*(2), 509–532. https://doi.org/10.1044/2019_JSLHR-19-00057

Hunter, E. J., Maxfield, L., & Graetzer, S. (2019). The effect of pulmonary function on the incidence of vocal fatigue among teachers. *Journal of Voice, 34*(4), 539–546. https://doi.org/10.1016/j.jvoice.2018.12.011

Iwarsson, J., & Sundberg, J. (1998). Effects of lung volume on vertical larynx position during phonation. *Journal of Voice, 12*(2), 159–165. https://doi.org/https://doi.org/10.1016/S0892-1997(98)80035-0

Iwarsson, J., Thomasson, M., & Sundberg, J. (1998). Effects of lung volume on the glottal voice source. *Journal of Voice, 12*(4), 424–433. https://doi.org/https://doi.org/10.1016/S0892-1997(98)80051-9

Jacobson, B. H., Johnson, A., Grywalski, C., Silbergleit, A., Jacobson, G., Benninger, M. S., & Newman, C. W. (1997). The Voice Handicap Index (VHI): Development and validation. *American Journal of Speech-Language Pathology, 6*(3), 66–70. http://www.scopus.com/inward/record.url?eid=2-s2.0-0642374983&partnerID=40&md5=f5c0ba58471a4cc143fe08e251372ab6, https://doi.org/10.1044/1058-0360.0603.66

Karkos, P. D., Yates, P. D., Carding, P. N., & Wilson, J. A. (2007). Is laryngopharyngeal reflux related to functional dysphonia. *Annals of Otology, Rhinology & Laryngology, 116*(1), 24–29. https://doi.org/10.1177/000348940711600105

Khoddami, S. M., Talebian, S., Izadi, F., & Ansari, N. N. (2017). Validity and reliability of surface electromyography in the assessment of primary muscle tension dysphonia. *Journal of Voice, 31*(3), 386.e9–386.e17. https://doi.org/10.1016/j.jvoice.2016.09.010

Kollbrunner, J., & Seifert, E. (2017). Encouragement to increase the use of psychosocial skills in the diagnosis and therapy of patients with functional dysphonia. *Journal of Voice, 31*(1), 132.e1–132.e7. https://doi.org/10.1016/j.jvoice.2015.11.021

Konrad, P. (2005). *The ABC of EMG: A practical introduction to kinesiological electromyography*. Noraxon Inc.

Kooijman, P. G., de Jong, F. I., Oudes, M. J., Huinck, W., van Acht, H., & Graamans, K. (2005). Muscular tension and body posture in relation to voice handicap and voice quality in teachers with persistent voice complaints. *Folia Phoniatrica et Logopaedica, 57*(3), 134–147. https://doi.org/10.1159/000084134

Koufman, J. A., Amin, M. R., & Panetti, M. (2000). Prevalence of reflux in 113 consecutive patients with laryngeal and voice disorders. *Otolaryngology—Head & Neck Surgery, 123*(4), 385–388. https://doi.org/10.1067/mhn.2000.109935

Koufman, J. A., & Blalock, P. D. (1982). Classification and approach to patients with functional voice disorders. *Annals of Otology, Rhinology & Laryngology, 91*(4 Pt. 1), 372–377. https://doi.org/10.1177/000348948209100409

Koufman, J. A., & Blalock, P. D. (1988). Vocal fatigue and dysphonia in the professional voice user: Bogart-Bacall syndrome. *The Laryngoscope, 98*(5), 493–498. https://doi.org/10.1288/00005537-198805000-00003

Koufman, J. A., & Blalock, P. D. (1991). Functional voice disorders. *Otolaryngologic Clinics of North America, 24*(5), 1059–1073. https://www.scopus.com/inward/record.uri?eid=2-s2.0-0025940145&partnerID=40&md5=020bae56ddda368a620f3fce82279939

Kridgen, S., Hillman, R. E., Stadelman-Cohen, T., Zeitels, S., Burns, J. A., Hron, T., Krusemark, C., Muise, J., & Van Stan, J. H. (2021). Patient-reported factors associated with the onset of hyperfunctional voice disorders. *Annals of Otology, Rhinology & Laryngology, 130*(4), 389–394. https://doi.org/10.1177/0003489420956379

Kryshtopava, M., Van Lierde, K., Meerschman, I., D'haeseleer, E., Vandemaele, P., Vingerhoets, G., & Claeys, S. (2017). Brain activity during phonation in women with muscle tension dysphonia: An fMRI study. *Journal of Voice, 31*(6), 675–690. https://doi.org/10.1016/j.jvoice.2017.03.010

Lowell, S. Y., Barkmeier-Kraemer, J. M., Hoit, J. D., & Story, B. H. (2008). Respiratory and laryngeal function during spontaneous speaking in teachers with voice disorders. *Journal of Speech, Language, and Hearing Research, 51*(2), 333–349. https://doi.org/10.1044/1092-4388(2008/025)

Lowell, S. Y., Colton, R. H., Kelley, R. T., Auld, M., & Schmitz, H. (2020). Isolated and combined respiratory training for muscle tension dysphonia: Preliminary findings. *Journal of Voice*. https://doi.org/https://doi.org/10.1016/j.jvoice.2020.06.013

Lowell, S. Y., Kelley, R. T., Colton, R. H., Smith, P. B., & Portnoy, J. E. (2012). Position of the hyoid and larynx in people with muscle tension dysphonia. *The Laryngoscope, 122*(2), 370–377. https://doi.org/10.1002/lary.22482

Lu, J., Fang, Q., Cheng, L., & Xu, W. (2021). Exploring the characteristics of functional dysphonia by multimodal methods. *Journal of Voice*. https://doi.org/10.1016/j.jvoice.2020.12.020

Macdonald, I., Rubin, J. S., Blake, E., Hirani, S., & Epstein, R. (2012). An investigation of abdominal muscle recruitment for sustained phonation in 25 healthy singers. *Journal of Voice, 26*(6), 815.e9–815.e8.15E16. https://doi.org/10.1016/j.jvoice.2012.04.006

McKenna, V. S., Hylkema, J. A., Tardif, M. C., & Stepp, C. E. (2020). Voice onset time in individuals with hyperfunctional voice disorders: Evidence for disordered vocal motor control. *Journal of Speech, Language, and Hearing Research, 63*(2), 405–420. https://doi.org/10.1044/2019_JSLHR-19-00135

McNaughton, N., & Corr, P. J. (2004). A two-dimensional neuropsychology of defense: Fear/anxiety and defensive distance. *Neuroscience and Biobehavioral Reviews, 28*(3), 285–305. https://doi.org/10.1016/j.neubiorev.2004.03.005

McNaughton, N., & Gray, J. A. (2000). Anxiolytic action on the behavioural inhibition system implies multiple types of arousal contribute to anxiety. *Journal of Affective Disorders, 61*(3), 161–176. https://doi.org/10.1016/s0165-0327(00)00344-x

Merletti, R., & Di Torino, P. (1999). Standards for reporting EMG data. *Journal of Electromyography and Kinesiology, 9*(1), 3–4.

Mezzedimi, C., Spinosi, M., Mannino, V., Ferretti, F., & Al-Balas, H. (2020). Kinesio taping application in dysphonic singers. *Journal of Voice, 34*(3), 487.e11–487.e20. https://doi.org/10.1016/j.jvoice.2018.11.001

Miller, T., Deary, V., & Patterson, J. (2014). Improving access to psychological therapies in voice disorders: A cognitive behavioural therapy model. *Current Opinion in Otolaryngology & Head and Neck Surgery, 22*(3), 201–205. https://doi.org/10.1097/moo.0000000000000056

Mirza, N., Ruiz, C., Baum, E. D., & Staab, J. P. (2003). The prevalence of major psychiatric pathologies in patients with voice disorders. *Ear, Nose, & Throat Journal, 82*(10), 808–814.

Misono, S., Dietrich, M., & Piccirillo, J. F. (2020). The puzzle of medically unexplained symptoms—A holistic view of the patient with laryngeal symptoms. *JAMA Otolaryngology–Head & Neck Surgery, 146*(6), 550–551. https://doi.org/10.1001/jamaoto.2020.0559

Misono, S., Haut, C., Meredith, L., Frazier, P. A., Stockness, A., Michael, D. D., Butcher, L., & Harwood, E. M. (2019). Dysphonia, perceived control, and psychosocial distress: A qualitative study. *Journal of Voice, 33*(5), 682–690. https://doi.org/10.1016/j.jvoice.2018.04.003

Montgomery, J., Hendry, J., Wilson, J. A., Deary, I. J., & MacKenzie, K. (2016). Pragmatic detection of anxiety and depression in a prospective cohort of voice outpatient clinic attenders. *Clinical Otolaryngology, 41*(1), 2–7. https://doi.org/10.1111/coa.12459

Morrison, M. D., Nichol, H., & Rammage, L. A. (1986). Diagnostic criteria in functional dysphonia. *The Laryngoscope, 96*(1), 1–8. https://doi.org/10.1288/00005537-198601000-00001

Morrison, M. D., & Rammage, L. A. (1993). Muscle misuse voice disorders: Description and classification. *Acta Oto-Laryngologica, 113*(3), 428–434. https://doi.org/10.3109/00016489309135839

Morrison, M. D., Rammage, L. A., Belisle, G. M., Pullan, C. B., & Nichol, H. (1983). Muscular tension dysphonia. *The Journal of Otolaryngology, 12*(5), 302–306.

Morrison, M., Rammage, L., & Emami, A. J. (1999). The irritable larynx syndrome. *Journal of Voice, 13*(3), 447–455. https://doi.org/10.1016/s0892-1997(99)80049-6

Nagy, A., Elshafei, R., & Mahmoud, S. (2020). Correlating undiagnosed hearing impairment with hyperfunctional dysphonia. *Journal of Voice, 34*(4), 616–621. https://doi.org/10.1016/j.jvoice.2019.02.002

Nguyen, D. D. K., & Kenny, D. T. (2009). Randomized controlled trial of vocal function exercises on muscle tension dysphonia in Vietnamese female teachers. *Journal of Otolaryngology—Head & Neck Surgery, 38*(2), 261–278.

Oates, J., & Winkworth, A. (2008). Current knowledge, controversies and future directions in hyperfunctional voice disorders. *International Journal of Speech-Language Pathology, 10*(4), 267–277. https://doi.org/10.1080/17549500802140153

O'Hara, J., Miller, T., Carding, P., Wilson, J., & Deary, V. (2011). Relationship between fatigue, perfectionism, and functional dysphonia. *Otolaryngology—Head & Neck Surgery, 144*(6), 921–926. https://doi.org/10.1177/0194599811401236

Olson, D. E., Cruz, R. M., Izdebski, K., & Baldwin, T. (2004). Muscle tension dysphonia in patients who use computerized speech recognition systems. *Ear, Nose, & Throat Journal, 83*(3), 195–198. https://doi.org/10.1177/014556130408300317

Patel, R. R., Liu, L., Galatsanos, N., & Bless, D. M. (2011). Differential vibratory characteristics of adductor spasmodic dysphonia and muscle tension dysphonia on high-speed digital imaging. *The Annals of Otology, Rhinology & Laryngology, 120*(1), 21–32. https://doi.org/10.1177/000348941112000104

Piersiala, K., Akst, L. M., Hillel, A. T., & Best, S. R. (2020). Chronic pain syndromes and their laryngeal manifestations. *JAMA Otolaryngology–Head & Neck Surgery, 146*(6), 543–549. https://doi.org/10.1001/jamaoto.2020.0530

Piersiala, K., Akst, L. M., Hillel, A. T., & Best, S. R. (2021). Laryngeal pathologies and their associations with mental health disorders. *The Laryngoscope, 131*(1), E231–e239. https://doi.org/10.1002/lary.28755

Rammage, L., Nichol, H., & Morrison, M. D. (1987). The psychopathology of voice disorders. *Human Communication Canada, 11*(4), 21–25.

Redenbaugh, M. A., & Reich, A. R. (1989). Surface EMG and related measures in normal and vocally hyperfunctional speakers. *Journal of Speech and Hearing Disorders, 54*(1), 68–73. https://doi.org/10.1044/jshd.5401.68

Roy, N. (2008). Assessment and treatment of musculoskeletal tension in hyperfunctional voice disorders. *International Journal of Speech-Language Pathology, 10*(4), 195–209. https://doi.org/10.1080/17549500701885577

Roy, N. (2011). Personality and voice disorders. *SIG 3 Perspectives on Voice and Voice Disorders, 21*(1), 17–23. https://doi.org/10.1044/vvd21.1.17

Roy, N., & Bless, D. M. (2000). Personality traits and psychological factors in voice pathology: A foundation for future research. *Journal of Speech, Language, and Hearing Research, 43*(3), 737–748. https://doi.org/10.1044/jslhr.4303.737

Roy, N., Bless, D. M., & Heisey, D. (2000a). Personality and voice disorders: A multitrait–multidisorder analysis. *Journal of Voice, 14*(4), 521–548. https://doi.org/10.1016/s0892-1997(00)8009-0

Roy, N., Bless, D. M., & Heisey, D. (2000b). Personality and voice disorders: A superfactor trait analysis. *Journal of Speech, Language, and Hearing Research, 43*(3), 749–768. https://doi.org/10.1044/jslhr.4303.749

Roy, N., Bless, D. M., Heisey, D., & Ford, C. N. (1997). Manual circumlaryngeal therapy for functional dysphonia: An evaluation of short- and long-term treatment outcomes. *Journal of Voice, 11*(3), 321–331. https://doi.org/10.1016/s0892-1997(97)80011-2

Roy, N., Dietrich, M., Blomgren, M., Heller, A., Houtz, D. R., & Lee, J. (2019). Exploring the neural bases of primary muscle tension dysphonia: A case study using functional magnetic resonance imaging. *Journal of Voice, 33*(2), 183–194. https://doi.org/10.1016/j.jvoice.2017.11.009

Roy, N., McGrory, J. J., Tasko, S. M., Bless, D. M., Heisey, D., & Ford, C. N. (1997). Psychological correlates of functional dysphonia: An investigation using the Minnesota Multiphasic Personality Inventory. *Journal of Voice, 11*(4), 443–451. https://doi.org/10.1016/s0892-1997(97)80041-0

Roy, N., Nissen, S. L., Dromey, C., & Sapir, S. (2009). Articulatory changes in muscle tension dysphonia: Evidence of vowel space expansion following manual circumlaryngeal therapy. *Journal of Communication Disorders, 42*(2), 124–135. https://doi.org/10.1016/j.jcomdis.2008.10.001

Roy, N., Smith, M. E., Allen, B., & Merrill, R. M. (2007). Adductor spasmodic dysphonia versus muscle tension dysphonia: Examining the diagnostic value of recurrent laryngeal nerve lidocaine block. *The Annals of Otology, Rhinology, and Laryngology, 116*(3), 161–168. https://doi.org/10.1177/000348940711600301

Rubin, J. S., Blake, E., & Mathieson, L. (2007). Musculoskeletal patterns in patients with voice disorders. *Journal of Voice, 21*(4), 477–484. https://doi.org/10.1016/j.jvoice.2005.02.001

Rubin, J. S., Lieberman, J., & Harris, T. M. (2000). Laryngeal manipulation. *Otolaryngologic Clinics of North America, 33*(5), 1017–1034. https://doi.org/10.1016/S0030-6665(05)70261-9

Rubin, J. S., Macdonald, I., & Blake, E. (2011). The putative involvement of the transabdominal muscles in dysphonia: A preliminary study and thoughts. *Journal of Voice, 25*(2), 218–222. https://doi.org/10.1016/j.jvoice.2009.09.001

Sama, A., Carding, P. N., Price, S., Kelly, P., & Wilson, J. A. (2001). The clinical features of functional dysphonia. *The Laryngoscope, 111*(3), 458–463. https://doi.org/10.1097/00005537-200103000-00015

Samargia, S., Schmidt, R., & Kimberley, T. J. (2016). Cortical silent period reveals differences between adductor spasmodic dysphonia and muscle tension dysphonia. *Neurorehabilitation and Neural Repair, 30*(3), 221–232. https://doi.org/10.1177/1545968315591705

Schlereth, T., & Birklein, F. (2008). The sympathetic nervous system and pain. *Neuromolecular Medicine, 10*(3), 141–147. https://doi.org/10.1007/s12017-007-8018-6

Schultz-Coulon, H. J. (1978). The neuromuscular phonatory control system and vocal function. *Acta Oto-Laryngologica, 86*(1–6), 142–153. https://doi.org/10.3109/00016487809124731

Shembel, A. C., Lee, J., Sacher, J. R., & Johnson, A. M. (2021). Characterization of primary muscle tension dysphonia using acoustic and aerodynamic voice metrics. *Journal of Voice*. https://doi.org/10.1016/j.jvoice.2021.05.019

Shraim, M. A., Massé-Alarie, H., & Hodges, P. W. (2021). Methods to discriminate between mechanism-based categories of pain experienced in the musculoskeletal system: A systematic review. *Pain, 162*(4), 1007–1037. https://doi.org/10.1097/j.pain.0000000000002113

Siupsinskiene, N., Razbadauskas, A., & Dubosas, L. (2011). Psychological distress in patients with benign voice disorders. *Folia Phoniatrica et Logopaedica, 63*(6), 281–288. https://doi.org/10.1159/000324641

Spencer, M. L. (2015). Muscle tension dysphonia: A rationale for symptomatic subtypes, expedited treatment, and increased therapy compliance. *SIG 3 Perspectives on Voice and Voice Disorders, 25*(1), 5–15. https://doi.org/10.1044/vvd25.1.5

Stager, S. V., Bielamowicz, S. A., Regnell, J. R., Gupta, A., & Barkmeier, J. M. (2000). Supraglottic activity: Evidence of vocal hyperfunction or laryngeal articulation. *Journal of Speech, Language, and Hearing Research, 43*(1), 229–238. https://doi.org/10.1044/jslhr.4301.229

Stemple, J. C., Lee, L., D'Amico, B., & Pickup, B. (1994). Efficacy of vocal function exercises as a method of improving voice production. *Journal of Voice, 8*(3), 271–278. https://doi.org/10.1016/S0892-1997(05)80299-1

Stepp, C. E., Hillman, R. E., & Heaton, J. T. (2010). A virtual trajectory model predicts differences in vocal fold kinematics in individuals with vocal hyperfunction. *The Journal of the Acoustical Society of America, 127*(5), 3166–3176. https://doi.org/10.1121/1.3365257

Stepp, C. E., Lester-Smith, R. A., Abur, D., Daliri, A., Pieter Noordzij, J., & Lupiani, A. A. (2017). Evidence for auditory-motor impairment in individuals with hyperfunctional voice disorders. *Journal of Speech, Language, and Hearing Research, 60*(6), 1545–1550. https://doi.org/10.1044/2017_JSLHR-S-16-0282

Stevens, K. N. (1977). Physics of laryngeal behavior and larynx modes. *Phonetica, 34*(4), 264–279. https://doi.org/10.1159/000259885

Tomlinson, C. A., & Archer, K. R. (2015). Manual therapy and exercise to improve outcomes in patients with muscle tension dysphonia: A case series. *Physical Therapy, 95*(1), 117–128. https://doi.org/10.2522/ptj.20130547

Torrico, S., Kern, M., Aslam, M., Narayanan, S., Kannappan, A., Ren, J., Sui, Z., Hofmann, C., & Shaker, R. (2000). Upper esophageal sphincter function during gastroesophageal reflux events revisited. *American Journal of Physiology-Gastrointestinal and Liver Physiology, 279*(2), G262–G267. https://doi.org/10.1152/ajpgi.2000.279.2.G262

Urberg-Carlson, K. E. (2013). *Muscle tension dysphonia as a disorder of motor learning* (Publication No. 3560601) [Doctoral dissertation, The University of Minnesota]. ProQuest Dissertations Publishing.

Van Houtte, E., Claeys, S., D'haeseleer, E., Wuyts, F., & Van Lierde, K. (2013). An examination of surface EMG for the assessment of muscle tension dysphonia. *Journal of Voice, 27*(2), 177–186. https://doi.org/10.1016/j.jvoice.2011.06.006

Van Houtte, E., Van Lierde, K., & Claeys, S. (2011). Pathophysiology and treatment of muscle tension dysphonia: A review of the current knowledge. *Journal of Voice, 25*(2), 202–207. https://doi.org/10.1016/j.jvoice.2009.10.009

Van Houtte, E., Van Lierde, K., D'haeseleer, E., & Claeys, S. (2010). The prevalence of laryngeal pathology in a treatment-seeking population with dysphonia. *The Laryngoscope, 120*(2), 306–312. https://doi.org/10.1002/lary.20696

Van Houtte, E., Van Lierde, K., D'haeseleer, E., Van Imschoot, B., & Claeys, S. (2012). UES pressure during phonation using high-resolution manometry and 24-h dual-probe pH-metry in patients with muscle tension dysphonia. *Dysphagia, 27*(2), 198–209. https://doi.org/10.1007/s00455-011-9354-0

Van Lawrence, L. (1987). *Suggested criteria for fibre-optic diagnosis of vocal hyperfunction*. Presentation at Care of the Professional Voice Symposium; the British Voice Association, London, United Kingdom.

Van Lierde, K. M., Claeys, S., De Bodt, M., & van Cauwenberge, P. (2007). Long-term outcome of hyperfunctional voice disorders based on a multiparameter approach. *Journal of Voice, 21*(2), 179–188. https://doi.org/10.1016/j.jvoice.2005.11.002

Van Lierde, K. M., De Bodt, M., D'haeseleer, E., Wuyts, F., & Claeys, S. (2010). The treatment of muscle tension dysphonia: A comparison of two treatment techniques by means of an objective multiparameter approach. *Journal of Voice, 24*(3), 294–301. https://doi.org/10.1016/j.jvoice.2008.09.003

van Mersbergen, M., Patrick, C., & Glaze, L. (2008). Functional dysphonia during mental imagery: Testing the trait theory of voice disorders. *Journal of Speech, Language, and Hearing Research, 51*(6), 1405–1423. https://doi.org/10.1044/1092-4388(2008/06-0216)

Van Stan, J. H., Ortiz, A. J., Cortes, J. P., Marks, K. L., Toles, L. E., Mehta, D. D., Burns, J. A., Hron, T., Stadelman-Cohen, T., Krusemark, C., Muise, J., Fox-Galalis, A. B., Nudelman, C., Zeitels, S., & Hillman, R. E. (2021). Differences in daily voice use measures between female patients with nonphonotraumatic vocal hyperfunction and matched controls. *Journal of Speech, Language, and Hearing Research, 64*(5), 1457–1470. https://doi.org/10.1044/2021_JSLHR-20-00538

Verdolini, K., Rosen, C. A., & Branski, R. C. (2014). *Classification manual for voice disorders-I*. Psychology Press. https://doi.org/10.4324/9781410617293

Vertigan, A. E., Bone, S. L., & Gibson, P. G. (2013). Laryngeal sensory dysfunction in laryngeal hypersensitivity syndrome. *Respirology, 18*(6), 948–956. https://doi.org/10.1111/resp.12103

Vertigan, A. E., Bone, S. L., & Gibson, P. G. (2014). Development and validation of the Newcastle Laryngeal Hypersensitivity Questionnaire. *Cough, 10*(1), 1. https://doi.org/10.1186/1745-9974-10-1

Vertigan, A. E., Gibson, P. G., Theodoros, D. G., & Winkworth, A. L. (2008). The role of sensory dysfunction in the development of voice disorders, chronic cough and paradoxical vocal fold movement. *International Journal of Speech-Language Pathology, 10*(4), 231–244. https://doi.org/10.1080/17549500801932089

Vertigan, A. E., Gibson, P. G., Theodoros, D. G., Winkworth, A. L., Borgas, T., & Reid, C. (2006). Involuntary glottal closure during inspiration in muscle tension dysphonia. *The Laryngoscope, 116*(4), 643–649. https://doi.org/10.1097/01.Mlg.0000201906.41316.Fc

Vilkman, E., Sonninen, A., Hurme, P., & Körkkö, P. (1996). External laryngeal frame function in voice production revisited: A review. *Journal of Voice, 10*(1), 78–92. https://doi.org/10.1016/s0892-1997(96)80021-x

Watts, C. R., Hamilton, A., Toles, L., Childs, L., & Mau, T. (2015). A randomized controlled trial of stretch-and-flow voice therapy for muscle tension dysphonia. *The Laryngoscope, 125*(6), 1420–1425. https://doi.org/10.1002/lary.25155

Watts, C. R., Hamilton, A., Toles, L., Childs, L., & Mau, T. (2019). Intervention outcomes of two treatments for muscle tension dysphonia: A randomized controlled trial. *Journal of Speech, Language, and Hearing Research, 62*(2), 272–282. https://doi.org/10.1044/2018_JSLHR-S-18-0118

Whittemore, R., & Knafl, K. (2005). The integrative review: Updated methodology. *Journal of Advanced Nursing, 52*(5), 546–553. https://doi.org/10.1111/j.1365-2648.2005.03621.x

Wilson Arboleda, B. M., & Frederick, A. L. (2008). Considerations for maintenance of postural alignment for voice production. *Journal of Voice, 22*(1), 90–99. https://doi.org/10.1016/j.jvoice.2006.08.001

Wuyts, F. L., De Bodt, M. S., Molenberghs, G., Remacle, M., Heylen, L., Millet, B., Van Lierde, K., Raes, J., & Van de Heyning, P. H. (2000). The dysphonia severity index: An objective measure of vocal quality based on a multiparameter approach. *Journal of Speech, Language, and Hearing Research, 43*(3), 796–809. https://doi.org/10.1044/jslhr.4303.796

Yiu, E. M.-L., Verdolini, K., & Chow, L. P. Y. (2005). Electromyographic study of motor learning for a voice production task. *Journal of Speech, Language, and Hearing Research, 48*(6), 1254–1268. https://doi.org/10.1044/1092-4388(2005/087a)

Zhang, Z. (2016). Respiratory laryngeal coordination in airflow conservation and reduction of respiratory effort of phonation. *Journal of Voice, 30*(6), 760.e7–760.e13. https://doi.org/10.1016/j.jvoice.2015.09.015

Zheng, Y.-Q., Zhang, B.-R., Su, W.-Y., Gong, J., Yuan, M.-Q., Ding, Y.-L., & Rao, S.-Q. (2012). Laryngeal aerodynamic analysis in assisting with the diagnosis of muscle tension dysphonia. *Journal of Voice, 26*(2), 177–181. https://doi.org/10.1016/j.jvoice.2010.12.001

Ziethe, A., Petermann, S., Hoppe, U., Greiner, N., Brüning, M., Bohr, C., & Döllinger, M. (2019). Control of fundamental frequency in dysphonic patients during phonation and speech. *Journal of Voice, 33*(6), 851–859. https://doi.org/10.1016/j.jvoice.2018.07.001