Auditory-Motor Function Pre- and Post-Therapy in Hyperfunctional Voice Disorders: A Case Series

[#,†,‡]Defne Abur, [§,¶,‖]Robert E. Hillman, and [#,**,††]Cara E. Stepp, #§¶‖**††*Boston, MA, and* †‡*Groningen, the Netherlands*

Summary: Objective/Hypothesis. Behavioral voice therapy is the most common treatment for hyperfunctional voice disorders (HVDs) but has limited long-term effectiveness since the comprehensive mechanisms underlying HVDs remain unclear. Recent work has implicated disordered sensorimotor integration during speech in some speakers with HVDs and suggests that auditory processing is a key factor to consider in HVD assessment and therapy. The purpose of this case-series study was to assess whether current voice therapy approaches for HVDs resulted in improvements to auditory-motor function.

Study Design. Longitudinal (pre-post) study.

Method. Pre and postvoice therapy for HVDs, 11 speakers underwent an assessment of auditory-motor function via auditory discrimination of vocal pitch, responses to unanticipated auditory perturbations, and responses to predictable auditory perturbations of vocal pitch.

Results. At the post-therapy session, 10 out of 11 participants demonstrated voice therapy success (via self-reported voice problems and/or auditory-perceptual judgements of voice by a clinician) and eight of the 11 participants demonstrated improvements in at least one measure of auditory discrimination and/or auditory-motor control. Specifically, three speakers demonstrated improvements in auditory discrimination, five speakers demonstrated improved (within typical cutoffs) responses to predictable perturbations, and two speakers demonstrated improvements in both auditory discrimination and auditory-motor measures.

Conclusions. Together, these findings support that voice therapy in individuals with HVDs may impact auditory-motor control and highlight the potential benefit of systematically addressing auditory function in voice therapy and assessment for HVDs.

Key Words: Auditory-motor control–Hyperfunctional voice disorders–Vocal nodules–Voice therapy–Auditory processing–Auditory perturbations.

INTRODUCTION

Approximately 30% of individuals will develop a voice disorder across their lifespan[1] and hyperfunctional voice disorders (HVDs) have been reported as the most common clinical diagnosis in voice clinics 45%.[2] Thus, HVDs are highly prevalent and can result in substantial declines in quality of life, including worse job performance, disrupted communicative ability, and increased isolation due to avoidance of social activities.[1,2] HVDs present with heterogenous symptoms, but they are typically associated with excessive peri-laryngeal muscle tension and improper muscle patterns. These inappropriate muscle patterns can occur with or without phonotraumatic lesions on the vocal folds.[3] HVDs have been extensively examined over decades of research (reviewed below), but all the factors that contribute to the onset and persistence of symptoms have yet to be clearly identified.

There are no universal criteria for clinical diagnosis and management of HVDs, but several factors have been attributed to their development. Primarily, HVDs are linked to voice use and are frequently reported by individuals with high voice use professions eg, singers and teachers.[4,5] Some have also postulated that there are links between HVDs and psychosocial behavior[6,7] and autonomic dysfunction.[8,9] The combination of risk factors in HVDs and the unclear underlying mechanisms are an obstacle for diagnosis, assessment, and therapeutic approaches.[10–15] There are also subtypes of HVDs: phonotraumatic vocal hyperfunction (PVH), which includes lesions on the vocal folds, and non-phonotraumatic vocal hyperfunction (NPVH), which occurs in the absence of any lesions. For the latter, NPVH is often a diagnosis of exclusion ie, individuals receive an HVD diagnosis if there are no other clear structural or neurological reasons for the voice changes.[16]

Although HVDs are clearly associated with impaired voice production, recent findings suggest that atypical auditory function may also be an additional factor in their presentation.[17–21] This possibility is theoretically motivated by notable similarities in speech subsystem disruptions between individuals with HVDs and individuals with hearing loss. In the respiratory subsystem, speakers with

Accepted for publication August 16, 2023.

This work was supported by grants P50 DC015446 (REH), T32 DC013017 (CAM and CES), and F31 DC019032 (DA) from the National Institute of Deafness and Other Communication Disorders. This work was also supported by a Graduate Fellow Award from the Rafik B. Hariri Institute for Computing and Computational Science and Engineering (DA) and an ASHFoundation New Century Doctoral Scholarship (DA).

From the #Department of Speech, Language, and Hearing Sciences, Boston University, Boston, MA; †Department of Computational Linguistics, Center for Language and Cognition Groningen, University of Groningen, Groningen, the Netherlands; ‡Research School of Behavioral and Cognitive Neurosciences, University of Groningen, Groningen, the Netherlands; §Center for Laryngeal Surgery and Voice Rehabilitation, Massachusetts General Hospital, Boston, MA; ¶Department of Surgery, Harvard Medical School, Boston, MA; ‖MGH Institute of Health Professions, Boston, MA; **Department of Biomedical Engineering, Boston University, Boston, MA; and the ††Department of Otolaryngology – Head and Neck Surgery, Boston University School of Medicine, Boston, MA.

Address correspondence and reprint requests to Defne Abur, University of Groningen, Department of Computational Linguistics, Center for Language and Cognition, Groningen, the Netherlands. E-mail: d.abur@rug.nl

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

0892-1997

https://doi.org/10.1016/j.jvoice.2023.08.017

hearing loss have demonstrated atypical speech breathing[22,23] that is also observed in HVDs.[24–26] In the laryngeal subsystem, both speakers with HVDs and speakers with hearing loss have shown increased subglottal pressure during voicing compared to speakers with typical voices.[27,28] Additionally, the voices of speakers with hearing loss are rated as perceptually breathy and strained, much like some speakers with HVDs.[22,27,29] In the articulatory subsystem, speakers with hearing loss show restricted tongue movements during vowel production compared to typical speakers,[30] which is in alignment with the reduced vowel space observed in HVDs.[31]

The role of auditory processing deficits in HVD symptoms is supported by current models of speech motor control: the generation of appropriate laryngeal muscle control patterns depends on the ability to detect sensory (somatosensory and auditory) changes and generate corrective output. Specifically, auditory-motor control of voice requires (1) the detection of auditory errors (the mismatch between expected and actual feedback), (2) generation of a corrective motor plan by the auditory feedback system, and (3) updating of the feedforward system to incorporate the corrective plan into future utterances auditory-motor integration.[32] Therefore, the persistent improper laryngeal control in individuals with HVDs may result from atypical auditory-motor function.

Auditory-motor control of voice can be examined experimentally with the use of pitch-shifting technology. The detection of auditory errors can be examined via comparisons of typical and pitch-shifted stimuli in a listening tasks to determine listeners' thresholds for discriminating auditory errors.[33] To examine components of the auditory feedback and feedforward control systems, unexpected and sustained (predictable) perturbations to speakers' auditory feedback can be applied during speech production. Commonly, these perturbations are applied to a speaker's fundamental frequency (f_o), the acoustic correlate of vocal pitch. Unexpected perturbations in feedback (yielding "reflexive" responses) allow for assessment of the feedback control system, whereas sustained perturbations in feedback (yielding "adaptive" responses) allow for assessment of auditory-motor integration. Both unexpected and sustained modifications to auditory feedback of vocal pitch yield robust responses in speakers with typical speech, in which they tend to compensate by opposing the direction of the perturbation.[20,34–39]

A few investigations to date have implicated disruptions to disparate aspects of central auditory processing in speakers with HVDs. Two studies have documented a difference in the detection of auditory errors in HVDs, reporting that speakers with HVDs show a reduced ability to discriminate the pitch of pure tones as well as their own vocal pitch compared to controls.[20,21] When examining reflexive f_o responses, Ziethe et al[17] found larger responses in speakers with HVDs (N = 10) compared to controls; yet a recent investigation in a larger sample of speakers with HVDs (N = 62) and a higher degree of experimental control reported no group differences (Abur et al.[20]). Two studies have also reported evidence of deficits in auditory-motor integration of vocal pitch in HVDs via atypical adaptive f_o responses.[18,20] In contrast to typical speakers, who demonstrate responses opposing the direction of the pitch shift, many speakers with HVDs show atypically large opposing responses, minimal responses (no pitch changes), or even "following" responses in the direction of the pitch shift. Of note, the one study that examined all three components of auditory-motor control in the same speakers with HVDs reported a relationship between worse auditory discrimination thresholds (reduced ability to detect auditory errors in vocal pitch) and atypical auditory-motor integration, directly linking auditory disruptions, and voice production in HVDs.[20] In addition, neuroimaging data support that HVDs may co-occur with overactivation of neural regions that regulate inhibition in the central nervous system, which could influence laryngeal motor activity.[21] Hence, previous work in differing samples of speakers with HVDs supports that HVDs may co-occur with auditory-motor control-based impairments.

Behavioral voice therapy is currently the primary treatment approach for HVDs, but therapeutic effectiveness can vary greatly.[14,15,40] Despite high individual variability, voice therapy can effectively relieve voice symptoms for some individuals with HVDs, as quantified by acoustic, aerodynamic, perceptual, and patient self-report measures.[41–45] In the short-term, voice therapy typically results in improved vocal function; however, relapses and recurrences of voice symptoms are frequent a reported prevalence of 51–68%.[46,47] Thus, a clear understanding of HVD etiology would greatly improve global therapeutic effectiveness.

Current behavioral therapies for HVDs primarily target musculoskeletal symptoms, vocal function, and respiratory function. A review of the literature[48] determined that the majority of reported therapies for HVDs involved techniques to reduce elevated subglottal pressure during speech, change voice f_o, reduce perceived voice roughness, and increase maximum phonation time (the longest amount of time voice can be sustained on a single breath). These therapy goals are often achieved through semi-occluded vocal tract exercises,[49] resonant voice therapies,[50] flow phonation,[51] and manual laryngeal therapy.[52,53] Although strategies used in voice therapy for HVDs can include an auditory component, eg, use of altered auditory feedback (delayed auditory feedback, masking) or patients' auditory-perceptual evaluations of their own productions, such approaches are not universally or uniformly applied.[54]

Considering the evidence for auditory deviations in HVDs, the goal of the current work was to explore whether voice therapy for HVDs impacts auditory-motor control. Behavioral voice therapy is a common treatment option for speakers with HVDs, but often individuals may require several rounds of therapy because voice symptoms commonly persist or reoccur.[46,47] It is also not clear whether voice therapy induces neuroplasticity and alleviates the observed disruptions to auditory-motor control, or whether therapy results in temporary compensatory strategies that only benefit voice symptoms.

The purpose of the current work was to determine the effect of behavioral voice therapy on auditory-motor control of f_o in HVDs. To examine this question, comprehensive measures of auditory-motor control were assessed within the same speakers before and after voice therapy. These measures of auditory-motor control included (1) auditory discrimination of voice f_o, (2) reflexive f_o responses, and (3) adaptive f_o responses. Given the previous findings of disrupted auditory discrimination of f_o and concurrent atypical adaptive f_o responses in HVDs, it was expected that, relative to pretherapy, behavioral voice therapy would result in (1) better auditory discrimination (via reduced acuity thresholds), (2) no differences in reflexive f_o responses (as no disruptions were observed pretherapy in prior work; see Abur et al[20]), and (3) more typical adaptive f_o responses.

METHOD

Participants

A total of 11 individuals with HVDs participated pre and postvoice therapy. The group characteristics are listed in Table 1. Participants labeled "singers" had at least 5 years of formal training in vocal performance. All individuals with HVDs were diagnosed by a laryngologist with either NPVH or PVH based on a comprehensive voice evaluation including videolaryngoscopy at either the Boston Medical Center (Boston, MA) or the Massachusetts General Hospital Voice Center (Boston, MA). Per standard clinical practice, therapy was individualized based on clinical judgment and patient needs. Hence, participants underwent between 4 and 16 weekly sessions of voice therapy across 1–5 months (see Table 1). For the participants with PVH, two speakers included in the study underwent surgical interventions to remove the lesions followed by voice therapy and the post-therapy evaluations reported reduced evidence of phonotrauma via videolaryngoscopy. These participants were still included in the study since because it is standard practice to do postsurgical voice therapy to address any persistent patterns of hyperfunctional voice use that could result in recurrence of phonotrauma. None of the participants had a history of neurological disorders or other speech, language, and hearing disorders. Due to the impact of hearing on auditory processing,[55] all participants underwent hearing threshold testing with insert earphones or headphones on a GSI 17 or GSI 18 audiometer (Grason-Stadler, Littleton, MA). Participants were included if they had hearing thresholds of 25 dB HL or below at 250, 500, 1000, 2000, and 4000 Hz per pure-tone hearing screening guidelines by the.[56] All study participants provided informed consent prior to the study and all experiments were performed in accordance with the Boston University Institutional Review Board.

Experimental setup

All participants completed tasks to quantify auditory-motor control at two time points (pre and post-therapy) in a sound-attenuated booth at Boston University. The pre and post-therapy experimental session consisted of the same tasks. Each session lasted 2–3 hours and included the following experimental tasks: (1) auditory discrimination of voice f_o, (2) reflexive f_o responses, and (3) adaptive f_o responses.

For all tasks, an omni-directional ear-set microphone (Shure MX153) was positioned at approximately 45 degrees from the midline and 7 cm away from the corner of the mouth to record all voice signals. The microphone gain was adjusted with a preamplifier (RME Quadmic II) and was digitized with a soundcard (MOTU Ultralite-mk3 Hybrid or RME Fireface UCX). An Eventide Eclipse V4 Harmonizer was used to create experimental shifts in voice fundamental frequency (f_o) with a processing delay between 10 and 30 ms.[57] The processed signal was amplified with an earphone amplifier (Behringer Xenyx Q802) and auditory feedback was administered via Etymotic ER-2 insert earphones or Sennheiser HD 280 Pro headphones.

The software and hardware systems were calibrated prior to each experimental session to ensure the correct output intensity for auditory feedback (Figure 1). The experimental setup was

TABLE 1.
Group Characteristics

Case	Age (y)	Sex	Gender	Musicality	Diagnosis	Therapy Sessions	Therapy Duration (mo)
Case 01	43	Female	Woman	Singer	PVH	4	1
Case 02	35	Female	Woman	Non-singer	NPVH	5	2
Case 03	20	Female	Woman	Singer	PVH	6	1.5
Case 04	36	Female	Woman	Singer	NPVH	16	4
Case 05	19	Female	Woman	Singer	PVH	12	3
Case 06	48	Male	Man	Singer	PVH	10	5
Case 07	32	Female	Woman	Singer	NPVH	10	3
Case 08	18	Male	Man	Singer	NPVH	8	4
Case 09	21	Female	Woman	Singer	PVH	10	2.5
Case 10	44	Female	Woman	Non-singer	NPVH	8	2
Case 11	24	Male	Man	Non-singer	NPVH	5	5

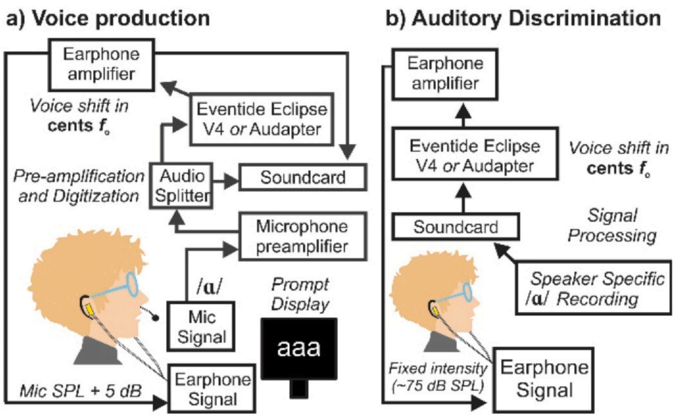

FIGURE 1. Experimental setup for shifts in auditory feedback of fundamental frequency (f_o). Voice production tasks (**a**) were calibrated such that auditory feedback was administered 5 dB above the microphone (mic) sound pressure level (SPL). The auditory discrimination task (**b**) was calibrated such that shifts in voice f_o were played back at approximately 75 dB SPL.

calibrated using a 2cc coupler (Type 4946, Bruel and Kjaer Inc) or an artificial ear headphone coupler (Type 4153, Bruel and Kjaer Inc), depending on whether insert earphones or headphones were used, respectively. The couplers were connected to a sound-level meter (Type 2250A with a Type 4947 ½″ Pressure Field Microphone, Bruel and Kjaer) to measure sound output. For the auditory discrimination task, the earphone or headphone output was calibrated to be 75-dB sound pressure level (SPL), regardless of the intensity of the input, using a 1-kHz tone played from a handheld Olympus LS-10 Linear PCM Recorder positioned 7 cm from the microphone. A level of 75-dB SPL was chosen to approximate the intensity of conversational speech.[58]

For the voice production (reflexive and adaptive) tasks, auditory feedback was amplified +5 dB relative to the microphone sound pressure level to mask bone-conducted feedback during speech.[59] Thus, the earphone or headphone output intensity was calibrated such that the 1-kHz tone played from a handheld Olympus LS-10 Linear PCM Recorder positioned 7 cm from the microphone yielded an amplification of +5 dB relative to the microphone signal.

DATA COLLECTION

Auditory discrimination

All participants completed an auditory discrimination task at both experimental sessions. At each session, participants were asked to produce a steady /ɑ/ vowel for 2–3 seconds and their voices were recorded with Praat software.[60] A steady 500-ms portion of each produced vowel was used as speaker-specific stimuli for the experiment. The auditory discrimination task consisted of a just-noticeable-difference experiment with a one-up, two-down staircase paradigm that used a 1:1 up-down step-size ratio. This type of paradigm results in a threshold of discrimination (ie, the point at which a speaker can no longer detect differences between stimuli) at 70.71% accuracy.[61] During the listening task, participants were presented with pairs of their own vowel recordings and asked to judge whether the two stimuli sounded the “same” or “different” in terms of their pitch. Each trial consisted of one stimulus that was a reference (the original recording) and one stimulus with a shift in f_o, which was applied based on the staircase procedure logic. The original and the pitch-shifted recordings were presented in randomized order for each trial; thus, each trial contained two recordings for comparison. The initial f_o change applied to the shifted stimulus was +50 cents (100 cents are equivalent to one semitone), with a 4-cent change in direction following two correct responses (decreasing) or one incorrect response (increasing). The 4-cent change in direction was based on prior studies, which determined the step size best suited to reach the threshold in the shortest amount of time.[33] In 20% of trials (“catch trials”), the reference stimulus was played twice to ensure attention to the task. Catch trial responses were not used in the staircase logic, but all participants had above chance catch trial accuracy (> 50%). The experiment ended after 10 “reversals” (ie, changes in the direction), which require two correct responses followed by an incorrect response or one incorrect response followed by two correct responses.

Reflexive and adaptive responses

During the tasks to elicit reflexive and adaptive f_o responses, participants were actively voicing while their auditory feedback was experimentally manipulated (Figure 1). For both tasks, participants were instructed to sustain a steady /ɑ/ vowel for 2–3 seconds for 108 trials per condition. The inter-trial interval was randomly jittered between 1 and 3 seconds to prevent the participant from using rhythmic cues. For both reflexive and adaptive tasks, each condition lasted 10 minutes.

The reflexive response task consisted of two conditions: shift-up and shift-down. Each condition had 84 trials with typical feedback (amplified +5 dB relative to the microphone signal with no pitch-shifting) and 24 trials with the addition of a sudden pitch shift of either +100 cents (shift-up) or −100 cents (shift-down) in voice f_o. To prevent habituations to the shifted feedback, there were always at least three typical feedback trials between each trial that had a pitch shift. During the pitch-shifted trials, voice shifts occurred randomly between 0.5 and 1 second after voicing onset to allow the voice to stabilize before the unexpected feedback shift, and remained for the duration of the trial as in prior work [20,35,59,62,63]. The two conditions were completed in counterbalanced order across participants.

The adaptive response task consisted of three conditions: shift-up, shift-down, and control. The conditions with pitch shifts (shift-up and shift-down) were completed first or third, in counterbalanced order across participants. The control condition was always completed second. The pitch-shifted conditions involved four ordered phases: “baseline”: 24 trials of typical feedback; “ramp”: 30 trials with gradual increases (shift-up) or decreases (shift-down) of 3.3 cents in the f_o of the auditory feedback each trial; “hold”: 30 trials with the pitch shifts maintained at +100 cents (shift-up) or −100 cents (shift-down); and "after-effect": 24 trials of typical feedback. For all shifted trials, the pitch shifts were applied at the beginning of each trial and were maintained for the full period of vocalization.

All participants completed the auditory discrimination task and both the shift-up and shift-down conditions for the adaptive f_o response task. For the reflexive response task, data were collected for nine participants in the shift-up condition and eight participants in the shift-down condition. More specifically, eight participants completed both shift-up and shift-down at both sessions, one additional participant completed only the shift-up condition at both sessions, and two participants were not able to complete either condition due to time constraints in the initial session.

Self-reported voice complaints

Self-reported voice complaints were documented for each speaker at the pre and post-therapy experimental session using the Voice-Related Quality of Life scale V-RQOL.[64] Speakers rated their voice-related problems on a scale of 1 ("not a problem at all") to 5 ("as bad as it can be") on 10 voice-related questions, which resulted in an overall score that could range from 10 (no problems) to 50 (all problems are as bad as they can be). The raw scores were standardized to values between 0 and 100, Table 1,[64] with higher standardized scores reflecting a better voice-related quality of life.

Reading task

All participants also completed three speech tasks at the two time points (pre and post-therapy): three prolonged /ɑ/ vowels, three prolonged /i/ vowels, "The Rainbow Passage,"[65] and a 1-minute natural speech sample in response to one of the following prompts: "How did you get here today?"; "Do you have any vacation or travel plans?"; "Tell me how the weather has been lately"; "What did you do last weekend?". Speech was recorded using an omnidirectional ear-set microphone (Shure MX153) at approximately 45 degrees from the midline and 7 cm away from the corner of the mouth. The microphone gain was adjusted with a preamplifier (RME Quadmic II) and was digitized with a soundcard (MOTU Ultralite-mk3 Hybrid or RME Fireface UCX). Audio recordings were collected using SONAR Artist (Cakewalk, Inc.) software at a sampling rate of 44.1 kHz and resolution of 16 bits.

DATA PROCESSING

Auditory-motor measures

To quantify auditory discrimination, a threshold was calculated for each participant as the average f_o shift values in cents across the last six reversals for each participant.[33] To examine reflexive and adaptive f_o responses, each participant's voice f_o was calculated for each trial and condition using an autocorrelation method via Praat[60] and custom MATLAB[66] scripts.

For the reflexive responses, the 120–240 ms portion (with 0 ms at the onset of the pitch shift) of each shifted trial was used for analyses, in line with prior work.[20,35,59] Each 120–240 ms portion contained voice f_o values sampled every 10 ms and this time range was selected to capture the feedback control system response.[35] Each f_o trace in Hz was converted to cents by using the average across the 100 ms immediately preceding the pitch shift as the reference frequency for each trial. The resulting voice f_o in cents was averaged across all pitch-shifted trials into a single trace for each speaker for each condition (shift-up and shift-down). The average across the f_o values in the 120–240 ms trace was termed as the "reflexive response."

For the adaptive responses, the 40–120 ms portion of each trial was extracted (with 0 ms corresponding to the start of vocalization) and used for analyses. This region of vocalization was used to capture the feedforward control system response.[67] For all conditions, the average voice f_o across the 40–120 ms region of each trial was extracted in Hz. Then, the average f_o values across the baseline trials in each condition were used as reference frequencies to convert the average f_o for each trial in Hz to cents for each associated condition (shift-up, shift-down, and control). To account for natural variability in voice f_o, the control condition was then subtracted from the two pitch-shifted conditions.[68] Given that prior work has observed variable adaptive responses in speakers with HVDs (atypically large opposing responses, no response, and following responses), adaptive responses were examined by a z-score method.[20] z-Scores were computed by comparing each participant's adaptive f_o response pre and post-therapy to a previously reported adaptive f_o response mean from a control group (N = 62) using a 90th percentile cutoff value for classification. Using the z-score cutoff values for the shift-up (z = 1.46) and shift-down (z = 1.52) conditions, all participant responses were classified as either "typical" (below the 90th percentile cutoff) or "atypical" (greater than the 90th percentile cutoff).

Consensus Auditory-perceptual Evaluation of Voice

Recorded speech from the reading task was used as stimuli for the Consensus Auditory-Perceptual Evaluation of Voice (CAPE-V) rating protocol.[69] For each time point (pre and post-therapy), the set of three /ɑ/ and three /i/ vowel productions, six sentences from "The Rainbow Passage," and 30 seconds of natural speech were combined into a single recording for each speaker. This yielded two recordings for each speaker. A blinded voice-specializing speech-language pathologist completed the full CAPE-V rating protocol on all types of stimuli, which were presented randomly from the full set of pre and post-therapy recordings (N = 22). Approximately 15% of samples (N = 3/22) were repeated for reliability. The rater showed excellent intra-rater reliability via an interclass correlation coefficient > 0.9[70]; for the four rated percepts (overall severity of dysphonia: 0.96 roughness: 0.97, breathiness: 0.96, and strain: 0.99).

RESULTS

The results of the study are reported descriptively for the pre and post-therapy sessions. For auditory discrimination and adaptive response tasks, results are reported for all 11 speakers. For the reflexive responses, two speakers were not able to complete the task and one speaker completed only the shift-up condition due to time constraints (see Methods), so pre and post-therapy data are reported for nine speakers in the shift-up condition and eight speakers in the shift-down condition.

Auditory discrimination

Average auditory discrimination thresholds were 40.1 cents (SD = 19.0 cents) pretherapy and 38.5 cents (SD = 19.4 cents) post-therapy, but there was substantial variability at an individual level (Figure 2 and Table 2). Given differences in auditory discrimination in speakers with HVDs and typical speakers by singing experience,[20] results are discussed separately for singers (N = 8) and non-singers (N = 3). Of the eight singers in the study, two singers displayed typical discrimination thresholds at both the pretherapy and post-therapy sessions (Cases 08 and 09; Figure 2) and the remaining six singers displayed atypical discrimination in the pretherapy session (ie, outside of the 95% confidence interval for singers with typical voices who completed the same paradigm in prior work; Abur et al., 2021). Three of the six singers with atypical discrimination pretherapy (Cases 03, 06, and 07) displayed discrimination thresholds within typical range at the post-therapy session, whereas the other three (Cases 01, 04, and 05) still had discrimination thresholds within the atypical range post-therapy (Figure 2). Cases 04 and 05 still demonstrated reduced

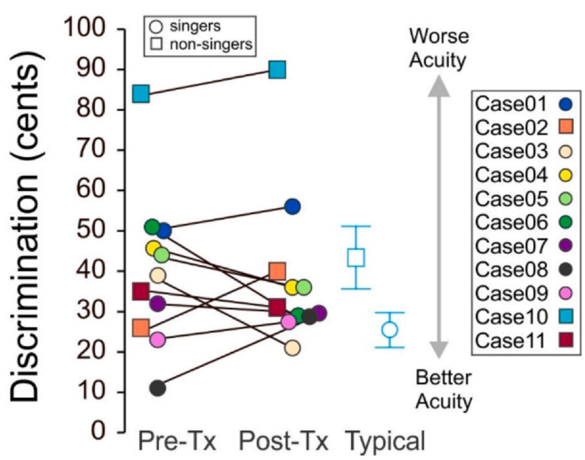

FIGURE 2. Auditory discrimination in cents is shown for each speaker pretherapy (Pre-Tx) and post-therapy (Post-Tx). The average discrimination values and 95% confidence intervals are shown for 33 typical speakers who were non-singers (square) and 28 typical speakers who were singers (circle) for reference[20]).

TABLE 2.
Summary of Changes From Pretherapy to Post-Therapy

Case	Musicality	V-RQOL	CAPE-V OS (mm)	Auditory Discrimination	Adaptive Responses	Reflexive Responses
Case 01	Singer	95.0 → 95.0	24.6 → 28.7	Atypical → Atypical	No changes (typical)	No changes (typical)
Case 02	**Non-singer**	**80.0 → 90.0**	9.3 → 14.5	Typical → Typical	No changes (typical)	No changes (typical)
Case 03	**Singer**	**70.0 → 92.5**	3.4 → 8.5	**Atypical → Typical**	**1 Atypical → 2 Typical**	No changes (typical)
Case 04	**Singer**	**90.0 → 95.0**	10.8 → 11.2	**Atypical → Atypical (improved)**	No changes (typical)	No changes (typical)
Case 05	**Singer**	**67.5 → 92.5**	11.4 → 11.7	**Atypical → Atypical (improved)**	No changes (typical)	No changes (typical)
Case 06	**Singer**	**75.0 → 97.5**	**45.9 → 37.0**	**Atypical → Typical**	**1 Atypical → 2 Typical**	No changes (typical)
Case 07	**Singer**	**52.5 → 90.0**	**7.0 → 2.7**	**Atypical → Typical**	No changes (typical)	No changes (typical)
Case 08	**Singer**	**80.0 → 95.0**	**17.7 → 8.8**	Typical → Typical	**2 Atypical → 1 Typical, 1 improved**	—
Case 09	**Singer**	**60.0 → 82.5**	**25.0 → 13.4**	Typical → Typical	No changes (typical)	No changes (typical)
Case 10	**Non-singer**	**60.0 → 87.5**	**20.4 → 9.7**	Atypical → Atypical	**1 Atypical → 2 Typical**	—
Case 11	**Non-singer**	**70.0 → 92.5**	**12.5 → 9.7**	Typical → Typical	**1 Atypical → 2 Typical**	No changes (typical) *only shift-up condition collected

Notes: Bolded cells denote improvement. "—" denotes no data for the task.
Abbreviations: V-RQOL, standardized voice-related quality of life ratings ranging from 0 (worst) to 100 (best). CAPE-V OS, Consensus Auditory-Perceptual Evaluation of Voice Overall Severity ratings.

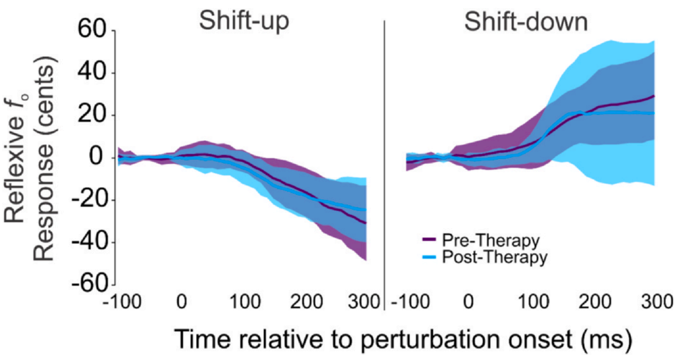

FIGURE 3. Reflexive responses in cents voice fundamental frequency (f_o) are shown for the pretherapy (light blue) and post-therapy (dark blue) sessions. The shift-up condition is shown on the right and the shift-down condition is shown on the left. Shaded regions, standard deviations.

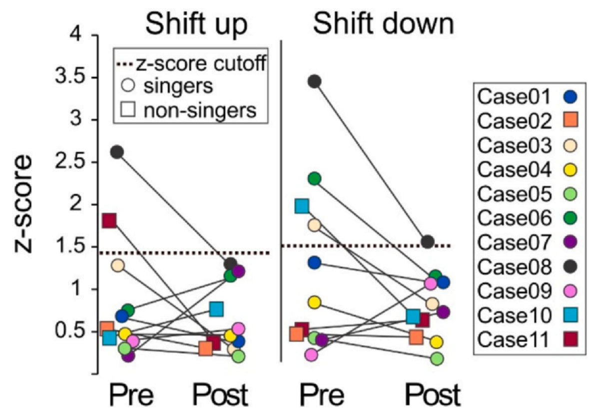

FIGURE 4. z-Scores for adaptive responses in the shift-up (left panel) and shift-down (right panel) condition by session is shown. *Dotted lines* indicate z-score cutoff for a typical response.

discrimination thresholds at post-therapy, demonstrating that auditory discrimination improved even though it did not change to be within the typical range. Thus, five of the six singers with atypical discrimination pretherapy demonstrated improved thresholds post-therapy, but only three of the six singers with atypical discrimination pretherapy demonstrated post-therapy discrimination thresholds within the typical range. Of the three non-singers in the current work, one speaker (Case 10) demonstrated atypical discrimination thresholds ie, outside the 95% confidence interval for non-singers with typical voices who completed the same paradigm in prior work[20] at both sessions and the other two speakers demonstrated discrimination thresholds within the typical range at both sessions (Cases 02 and 11; Figure 2).

Reflexive f_o responses

Given that the reflexive f_o responses pretherapy did not show statistical differences compared to typical speakers,[20] no changes were expected in reflexive f_o responses. On average, reflexive f_o response magnitudes in the 120–240 ms analysis range did not show descriptive differences due to therapy (ie, the individual responses remained within the bounds of mean and standard deviation interval previously reported in typical speakers for the shift-up condition (M = −13 cents, SD = 13 cents) and shift-down condition M = 21 cents, SD = 18 cents.[20] The group-level shift-up response for the nine speakers pretherapy (M = −20.7 cents, SD = 13.3 cents), shift-up response for the nine speakers post-therapy (M = −19.2 cents, SD = 12.3 cents), shift-down response for the eight speakers pretherapy (M = 23.6 cents, SD = 17.7 cents), and shift-down response for the eight speakers post-therapy (M = 20.1 cents, SD = 29.4 cents) are all displayed in Figure 4.

Adaptive f_o responses

The adaptive f_o responses for each speaker were classified as "typical" or "atypical" using a z-score method; responses within the 90th percentile of a 62-speaker control group were considered "typical" see methods in Abur et al.[20] In the pretherapy session, five speakers demonstrated atypical adaptive responses (outside the z-score cutoff value; see Methods). Three speakers had atypical responses for the shift-down direction (two singers, one non-singer), one speaker (non-singer) had an atypical response for the shift-up direction, and one speaker (singer) had an atypical response in both the shift-up and shift-down directions. The remaining six speakers had typical responses in both shift directions. At the post-therapy session, all five speakers with atypical adaptive responses pre-therapy demonstrated improvements post-therapy (Figure 4 and Table 2). For four of the five speakers with atypical adaptive f_o responses pre-therapy, the post-therapy adaptive responses were below the cutoff for a typical response. One speaker with an atypical response to both shift-up and shift-down conditions pre-therapy demonstrated a typical response for the shift-up post-therapy, but still displayed an atypical response the shift-down post-therapy; however, the shift-down response post-therapy showed improvement compared to the pretherapy session as evidenced by a z-score closer to the cutoff value.

Self-reported voice complaints

On average, the group demonstrated improved standardized V-RQOL scores from pre-therapy (M = 72.7, SD = 13.0) to post-therapy (M = 91.8, SD = 4.2). The group-level improvement was greater than the minimally clinically important difference (ie, reflecting improved vocal function) reported in prior work M = 18.6 with an SD = 12.7, based on patient's self-report of voice quality pre and post-therapy.[64] The range of standardized scores pretherapy was 52.5–95.0 and the range of scores post-therapy was 82.5–97.5. At an individual level, 10 of the 11 speakers demonstrated decreases in V-RQOL score post-therapy, indicating an improvement in self-reported voice problems (Figure 5 and Table 2). One speaker with minimal self-reported voice problems pretherapy (standardized V-RQOL score of 95) demonstrated no changes in

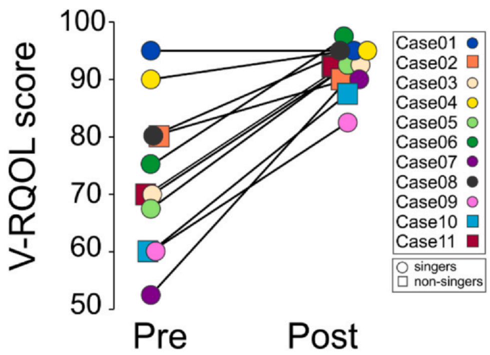

FIGURE 5. Self-reported voice-related quality of life scores pre and post-therapy.

V-RQOL score pre- compared to post-therapy (dark blue square in Figure 5).

Consensus Auditory-Perceptual Evaluation of Voice

The group-level CAPE-V ratings decreased for all percepts. On average, overall severity of voice ratings decreased from 17.1 mm pretherapy to 14.2 mm post-therapy, roughness ratings decreased from 14.5 mm pretherapy to 10.8 mm post-therapy, breathiness ratings decreased from 11.8 to 8.2 mm, and strain ratings decreased from 8.1 to 7.5 mm. Overall, CAPE-V ratings were in the lower range at both pre and post-therapy sessions (< 30/100 mm for 10 speakers and < 50/100 mm for one speaker), which indicates mild perceptual dysphonia at a group level. The pre and post-therapy session CAPE-V ratings ranged from 0.3 to 11.6 mm. Six of the 11 speakers demonstrated decreased overall severity of dysphonia ratings (ie, improved) post-therapy and five speakers, who had low overall severity ratings pretherapy (< 25 mm), demonstrated minimal changes post-therapy (< 5 mm in either direction; Figure 6 and Table 2).

DISCUSSION

The goal of this case-series study was to assess whether voice therapy resulted in improvements to auditory discrimination and auditory-motor control in speakers with HVDs. This is the first report of a comprehensive assessment of the auditory-motor control system (ie, auditory discrimination, reflexive responses, and adaptive responses) in any speech disorder before and after therapeutic intervention. Additionally, this work examined auditory-motor measures together with patient self-reported voice problems and expert auditory-perceptual judgements of overall voice severity to provide a multi-factorial assessment of voice changes post-therapy.

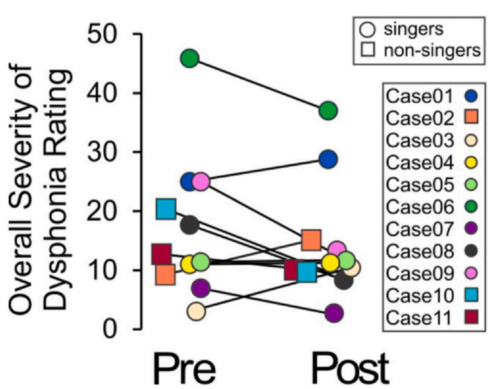

FIGURE 6. Overall severity of dysphonia ratings from 0 (no dysphonia) to 100 (maximum severity) for the pre and post-therapy sessions.

The self-reported voice problems via the V-RQOL and expert clinical judgements of voice (CAPE-V overall severity of dysphonia) support that vocal function improved for participants in the study. Ten of the 11 speakers demonstrated improvements in either VRQOL or CAPE-V ratings. The remaining speaker had minimal voice complaints (standardized VRQOL score = 95) and mild perceptual voice severity at both sessions (24.6–28.7 mm on the CAPE-V).

Previous work has examined one component of auditory-motor control (reflexive f_o responses) pre and post-therapy in a similarly small sample (N = 12) of speakers with Parkinson's disease.[71] Speakers with Parkinson's disease demonstrated an atypically large response, on average, to reflexive perturbations of f_o compared to controls at a pretherapy session. Following speech therapy focused on improving vocal intensity (LSVT LOUD,[72] the speakers with Parkinson's disease demonstrated improvements in reflexive f_o responses (reduced magnitudes). This finding supports the possibility that disrupted auditory-motor measures involving speech production could become more typical following voice therapy. However, Li et al[71] did not investigate measures of auditory discrimination, auditory feedback integration (adaptive f_o responses), self-perceived voice symptoms, or auditory-perceptual judgments of voice by a clinician. The lack of these additional measures limits the interpretation of which specific mechanisms might have benefited from the therapeutic intervention in Li et al.[71] As posited by the DIVA model and supported by neurophysiological studies,[32] measures of auditory discrimination reflect error sensitivity, reflexive responses to sudden changes reflect feedback control of speech, and adaptive responses to predictable change reflect feedforward control of speech (incorporation of errors and motor learning). Thus, the inclusion of these three measures in the current study, together with self-reported voice complaints and clinical judgements, yields a comprehensive assessment of speech motor control in the auditory domain to more fully interpret the impact of therapy.

Based on the results from Abur et al,[20] which demonstrated disruptions to auditory discrimination that were associated with impaired auditory-motor control in HVDs, successful voice therapy was expected to result in (1) improved f_o discrimination post-therapy (via reduced acuity thresholds), (2) no differences to reflexive f_o responses (as no disruptions were observed pretherapy), and (3) more typical adaptive f_o responses post-therapy (via z-score values relative to a control group distribution). The results varied at an individual level for the 11 speakers examined pre and post-therapy (Figure 6). Given the heterogeneity in HVDs,[4] the variability in the degree of improvement post-therapy in the current sample is not surprising. Findings are discussed for each experimental assessment below.

Auditory-motor improvements following voice therapy

The results for auditory discrimination were variable across speakers in the current work, but a subset of speakers who had disruptions to auditory discrimination demonstrated improvements post-therapy. Four of the 11 speakers did not show atypical discrimination thresholds either pre or post-therapy, whereas seven speakers showed atypical discrimination thresholds at the pretherapy session (based on previous data on typical speakers by singing experience; Figure 2). Of the seven speakers with atypical discrimination thresholds pretherapy, three speakers demonstrated typical responses post-therapy and two speakers demonstrated some improvements (thresholds closer to the typical range). The variability in individual improvements in auditory discrimination is not entirely unexpected since current voice therapy techniques for HVDs do not uniformly employ an auditory component.[54] Prior work has demonstrated that an auditory discrimination training task together with passive stimuli exposure is an effective method to induce auditory learning in speakers without voice disorders;[73] thus, future work could explore whether similar paradigms could benefit auditory function in HVDs. For two of the five speakers who demonstrated improved auditory discrimination post-therapy, adaptive responses also improved (atypical adaptive responses became typical adaptive responses), which suggests that auditory-motor control changes may relate to changes in auditory mechanisms for some speakers.

Reflexive f_o responses at the pretherapy sessions were within the typical range for the nine speakers who completed the shift-up condition and the eight speakers who completed the shift-down condition; hence, no changes were expected for this measure post-therapy. Descriptively, the group averages demonstrated minimal differences between the pre and post-therapy sessions. Previous work has observed larger reflexive f_o response magnitudes in individuals with HVDs compared to typical speakers,[17] but substantial differences in methodology prevent a direct comparison to the current work see Abur et al[20] for a review. Although there were no qualitative differences noted after therapy in the current work, both the pre and post-therapy reflexive f_o responses for the shift-down direction were qualitatively more variable compared to the shift-up direction, as shown by the larger standard deviation bars (Figure 3). This lends further support to the notion that shift direction is an important consideration for measures of reflexive f_o response, as observed in prior work.[20,74] The similar results for reflexive f_o responses across sessions in the current speaker sample also provide support for the replicability of reflexive response measures over time.

The adaptive f_o responses, which are thought to reflect the incorporation of changes in auditory feedback into voice production over time, showed improvements post-therapy for all five of the speakers who demonstrated atypical responses pretherapy (Figure 4 and Table 2). For the shift-up condition, 2/11 (18%) of speakers demonstrated atypical adaptive responses pretherapy and for the shift-down condition, 4/11 (36%) of speakers demonstrated atypical responses pretherapy. These incidence rates of atypical adaptive responses are in line with those previously reported in the larger group of 62 speakers with HVDs (17% for shift-up and 30% for shift-down). The overall improvement in adaptive responses in the current work provides promising evidence that auditory-motor control (ie, the integration of auditory feedback into voice production) can improve with voice therapy, adding to prior work that found evidence of improved auditory feedback processing (via reflexive f_o responses) in Parkinson's disease following voice therapy.[71] It is important to note that improved adaptive f_o responses may result from several mechanisms, and the current work did not examine detailed laryngeal-level information (eg, vocal fold kinematics). Therefore, the contributions of sensory changes (ie, auditory, somatosensory) compared to motor improvements following voice therapy in HVDs cannot be determined here and should be explored in future studies.

Summary of auditory-motor findings

The auditory-motor findings provide preliminary evidence that therapeutic protocols may improve auditory-motor control for some speakers while also supporting the systematic inclusion of auditory processing-related strategies in voice therapy. The subset of post-therapy improvements in auditory discrimination as well as adaptive responses suggest that current strategies for voice therapy do not universally improve auditory-motor function. Hence, inclusion of a more formal auditory training component for individuals with HVDs who display atypical pretreatment auditory responses could be beneficial. Specifically, employing auditory discrimination tasks and passive stimuli exposure (ie, speakers passively listening to the recordings of their own voice from the discrimination task), separately, may be effective methods to induce auditory learning based on evidence from prior work.[73,75] Although post-therapy improvements in auditory-motor control were found in a subset of the current sample, it remains unclear how long the observed improvements may last. This possibility should be explored in additional studies by examining the degree to which improvements in auditory-motor measures post-therapy remain at follow-up sessions (eg, after 6 months).

Relation of auditory-motor findings to self-reported voice complaints and clinical voice ratings

At a group-level, the auditory-motor improvements were accompanied by changes in self-reported voice complaints (via V-RQoL ratings) or CAPE-V ratings of overall voice severity. For the auditory discrimination task, two of the three speakers with discrimination thresholds that changed from atypical to typical post-therapy showed concurrent improvements to CAPE-V ratings. This finding suggests that better discrimination thresholds may relate to improved vocal quality. For the adaptive responses, four of the five speakers who had atypical responses pre-therapy that improved in the post-therapy session demonstrated concurrent improvements in CAPE-V ratings (Figure 6). This result strengthens previous evidence that adaptive responses are associated with auditory-perceptual measures of speech function.[63]

Limitations

This case series is a descriptive report of auditory-motor measures pre and post-therapy in HVDs that provides important preliminary data, but there are limitations. Notably, this work only included 11 speakers with HVDs pre and post-voice therapy. Hence, the results from this case-series report can be used as a basis to inform future investigations in a larger sample of speakers with HVDs that can yield statistical support for the relationships between the current experimental variables. Further, a control group was not included, so this work cannot fully rule out the possibility of additional variables that may contribute to changes in auditory-motor control over time.

In terms of the sample, the 11 speakers included here may not be generalizable to the larger population of individuals with HVDs for several reasons. All speakers in this case series had voice complaints pretherapy and were diagnosed with an HVD. However, the pretherapy CAPE-V ratings of overall severity of dysphonia were mild (group average of 17.1/100 mm), indicating more mild dysphonia compared to prior investigations in speakers with HVDs (eg, average severity of dysphonia ratings from 10 speakers with MTD = 79.3/100 mm,[76] and from 111 speakers with MTD = 67.9/100 mm.[77] This may be a result of the case-series sample consisting of mostly singers (N = 9), who are at higher risk of developing HVDs[5] and who show greater sensitivity to changes in their vocal pitch compared to non-singers, ie, singers have better discrimination thresholds of self-produced vocal pitch compared to non-singers.[20]

There are also limitations related to the components of voice therapy. The speakers in the current study did not complete the exact same therapy protocol due to the need to personalize therapy based on specific voice symptoms in line with prior research in HVDs.[78] However, comparisons of detailed therapeutic techniques and their impact on auditory-motor control improvements in HVDs could be important considerations for subsequent studies. In addition to possible confounds of individualized therapy, speakers also did not undergo the same number of therapy sessions since symptom resolution naturally occurred at different time points. Descriptively, the speakers with more therapy sessions did not appear to demonstrate better voice improvements in this case-series report, in line with prior findings that higher therapy dosage does not always correspond to therapeutic effectiveness.[42]

Another source of variability in speakers with HVDs is related to differences in laryngeal kinematics, somatosensation, and aerodynamic function, none of which were included in the current investigation. Future work should examine these measures together with auditory-motor measures in speakers with HVDs.

Finally, a single rater completed clinical ratings in this work on a set of speech stimuli that did not include CAPE-V sentences, which could limit the generalizability of the auditory-perceptual assessment. Having a single rater was a deliberate choice to mirror a clinical setting, wherein one clinician makes a pre and post-therapy assessment of a patient's voice. However, this design choice, together with the stimuli, may not be fully representative of a larger population of clinical raters.

Conclusion

In sum, the case series reported here is the first investigation of the impact of voice therapy on all components of auditory-motor control; specifically, in speakers with HVDs. Ten of the 11 patients with HVDs included in this case series demonstrated improvements in voice via either self-reported voice complaints or expert clinical judgements of overall severity of dysphonia. Eight of the 11 patients demonstrated improvements in either auditory discrimination, adaptive f_o responses, or both. These findings support the benefits of voice therapy in individuals with HVDs and highlight the benefit of considering auditory discrimination and auditory-motor control measures in assessment and therapy for HVDs.

Declaration of Competing Interest

Robert E. Hillman has financial interests in InnoVoyce LLC (board membership), a company focused on developing and commercializing technologies for the prevention, diagnosis, and treatment of voice-related disorders. His interests were reviewed and are managed by Massachusetts General Hospital and Mass General Brigham in accordance with their conflict of interest policies. The other authors have no relevant conflicts of interest to declare.

Acknowledgments

The authors thank Lauren Tracy, J. Pieter Noordzij, Katherine Brown, Anton Dolling, Austeja Subaciute, and Roxanne Segina for help with recruitment and data collection.

References

1. Roy N, Merrill RM, Gray SD, et al. Voice disorders in the general population: prevalence, risk factors, and occupational impact. *Laryngoscope.* 2005;115:1988–1995.
2. Van Houtte E, Van Lierde K, D'Haeseleer E, et al. The prevalence of laryngeal pathology in a treatment-seeking population with dysphonia. *Laryngoscope.* 2010;120:306–312.

3. Hillman RE, Stepp CE, Van Stan JH, et al. An updated theoretical framework for vocal hyperfunction. *Am J Speech-Lang Pathol.* 2020;29:2254–2260.
4. Altman KW, Atkinson C, Lazarus C. Current and emerging concepts in muscle tension dysphonia: a 30-month review. *J Voice.* 2005;19:261–267.
5. Roy N, Merrill RM, Thibeault S, et al. Prevalence of voice disorders in teachers and the general population. *J Speech Language Hear Res.* 2004.
6. Van Houtte E, Van Lierde K, Claeys S. Pathophysiology and treatment of muscle tension dysphonia: a review of the current knowledge. *J Voice.* 2011;25:202–207. https://doi.org/10.1016/j.jvoice.2009.10.009.
7. Roy N, Bless DM. Personality traits and psychological factors in voice pathology-a foundation for future research. *J Speech Lang Hear Res.* 2000;43:737–748.
8. Van Mersbergen M, Patrick C, Glaze L. Functional dysphonia during mental imagery: testing the trait theory of voice disorders. *J Speech Lang Hear Res.* 2008;51:1405–1423.
9. Park K, Behlau M. Signs and symptoms of autonomic dysfunction in dysphonia individuals. *J Soc Bras Fonoaudiol.* 2011;23:164–169.
10. Aronson AE. *Clinical Voice Disorders: An Interdisciplinary Approach*. New York: Thieme,; 1990.
11. Morrison M, Rammage LA, Belisle GM, et al. Muscular tension dysphonia. *J Otolaryngol.* 1983;12:302–306.
12. Desjardins M, Halstead L, Cooke M, et al. A systematic review of voice therapy: what “effectiveness” really implies. *J Voice.* 2017;31:392.e13–392.e32.
13. Van Houtte E, Claeys S, D’haeseleer E, et al. An examination of surface EMG for the assessment of muscle tension dysphonia. *J Voice.* 2013;27:177–186.
14. Verdolini-Marston K, Burke MK, Lessac A, et al. Preliminary study of two methods of treatment for laryngeal nodules. *J Voice.* 1995;9:74–85.
15. Ogawa M, Inohara H. Is voice therapy effective for the treatment of dysphonic patients with benign vocal fold lesions? *Auris Nasus Larynx.* 2018;45:661–666.
16. Tanner K, Milstein CF, Smith ME. Assessment and management of muscle tension dysphonia: a multidisciplinary approach. *Perspect ASHA Spec Interest Groups.* 2018;3:77–81.
17. Ziethe A, Petermann S, Hoppe U, et al. Control of fundamental frequency in dysphonic patients during phonation and speech. *J Voice.* 2019;33:851–859.
18. Stepp CE, Lester-Smith RA, Abur D, et al. Evidence for auditory-motor impairment in individuals with hyperfunctional voice disorders. *J Speech Lang Hear Res.* 2017;60:1545–1550.
19. Nagy A, Elshafei R, Mahmoud S. Correlating undiagnosed hearing impairment with hyperfunctional dysphonia. *J Voice.* 2020;34:616–621.
20. Abur D, Subaciute A, Kapsner-Smith M, et al. Impaired auditory discrimination and auditory-motor integration in hyperfunctional voice disorders. *Sci Rep.* 2021;11:13123. https://doi.org/10.1038/s41598-021-92250-8.
21. Nguyen DD, Chacon AM, Novakovic D, et al. Pitch discrimination testing in patients with a voice disorder. *J Clin Med.* 2022;11:584.
22. Forner LL, Hixon TJ. Respiratory kinematics in profoundly hearing-impaired speakers. *J Speech Hear Res.* 1977;20:373–408.
23. Perkell JS, Guenther FH, Lane H, et al. A theory of speech motor control and supporting data from speakers with normal hearing and with profound hearing loss. *J Phon.* 2000;28:233–272.
24. Sundberg I. Breathing behaviors during speech in healthy females and patients with vocal fold nodules. *Logop Phoniatr Vocol.* 1999;24:154–169.
25. Hixon T, Putnam A. Voice Disorders in Relation to Respiratory Kinematics. *Semin. Speech Lang.* 1983;4(3):217–231.
26. Lowell S.Y., Hoit J.D., Barkmeier J.M.Speech breathing behavior in muscle tension dysphonia: preliminary findings. *American Speech-Language-Hearing Association Convention*, 2004.
27. Higgins MB, Carney AE, Schulte L. Physiological assessment of speech and voice production of adults with hearing loss. *J Speech Lang Hear Res.* 1994;37:510–521.
28. Espinoza VM, Zanartu M, Van Stan JH, et al. Glottal aerodynamic measures in women with phonotraumatic and nonphonotraumatic vocal hyperfunction. *J Speech Lang Hear Res.* 2017;60:2159–2169. https://doi.org/10.1044/2017_JSLHR-S-16-0337.
29. Arends N, Povel D-J, Os EV, et al. Predicting voice quality of deaf speakers on the basis of glottal characteristics. *J Speech Lang Hear Res.* 1990;33:116–122.
30. Xue P, Zhang X, Bai J, et al. Acoustic and kinematic analyses of Mandarin vowels in speakers with hearing impairment. *Clin Linguist Phon.* 2018;32:622–639.
31. Roy N, Ferguson NA. Formant frequency changes following manual circumlaryngeal therapy for functional dysphonia: evidence of laryngeal lowering? *J Med Speech-Lang Pathol.* 2001;9:169–175.
32. Tourville JA, Guenther FH. The DIVA model: a neural theory of speech acquisition and production. *Lang Cogn Process.* 2011;26:952–981. https://doi.org/10.1080/01690960903498424.
33. Abur D, Stepp CE. Acuity to changes in self-generated vocal pitch in Parkinson's disease. *J Speech Lang Hear Res.* 2020;63:1–7.
34. Burnett TA, Freedland MB, Larson CR, et al. Voice F0 responses to manipulations in pitch feedback. *J Acoust Soc Am.* 1998;103:3153–3161.
35. Lester-Smith RA, Daliri A, Enos N, et al. The relation of articulatory and vocal auditory–motor control in typical speakers. *J Speech Lang Hear Res.* 2020;63:1–15.
36. Larson CR, Sun J, Hain TC. Effects of simultaneous perturbations of voice pitch and loudness feedback on voice F0 and amplitude control. *J Acoust Soc Am.* 2007;121:2862–2872.
37. Houde, Jordan MI. Sensorimotor adaptation in speech production. *Science.* 1998;279:1213–1216.
38. Chen SH, Liu H, Xu Y, et al. Voice F0 responses to pitch-shifted voice feedback during English speech. *J Acoust Soc Am.* 2007;121:1157–1163.
39. Jones JA, Keough D. Auditory-motor mapping for pitch control in singers and nonsingers. *Exp Brain Res.* 2008;190:279–287.
40. Gillespie AI, Dastolfo C, Magid N, et al. Acoustic analysis of four common voice diagnoses: moving toward disorder-specific assessment. *J Voice.* 2014;28:582–588.
41. Roy N, Leeper HA. Effects of the manual laryngeal musculoskeletal tension reduction technique as a treatment for functional voice disorders: perceptual and acoustic measures. *J Voice.* 1993;7:242–249.
42. Ramig LO, Verdolini K. Treatment efficacy: voice disorders. *J Speech Lang Hear Res.* 1998;41:S101–S116.
43. Holmberg EB, Doyle P, Perkell JS, et al. Aerodynamic and acoustic voice measurements of patients with vocal nodules: variation in baseline and changes across voice therapy. *J Voice.* 2003;17:269–282.
44. Holmberg EB, Hillman RE, Hammarberg B, et al. Efficacy of a behaviorally based voice therapy protocol for vocal nodules. *J Voice.* 2001;15:395–412.
45. Dastolfo C, Gartner-Schmidt J, Yu L, et al. Aerodynamic outcomes of four common voice disorders: moving toward disorder-specific assessment. *J Voice.* 2016;30:301–307.
46. Roy N, Bless DM, Heisey D. Manual circumlaryngeal therapy for functionaldysphonia: an evaluation of short-and long-term treatment outcomes. *J Voice.* 1997;11:321–331.
47. Van Lierde K, Claeys S, De Bodt M, et al. Long-term outcome of hyperfunctional voice disorders based on a multiparameter approach. *J Voice.* 2007;21:179–188.
48. da Cunha Pereira G, de Oliveira Lemos I, Gadenz CD, et al. Effects of voice therapy on muscle tension dysphonia: a systematic literature review. *J Voice.* 2018;32:546–552.
49. Titze IR. Voice training and therapy with a semi-occluded vocal tract: rationale and scientific underpinnings. *J Speech Lang Hear Res.* 2006;49:448–459.
50. Behrman A, Haskell J. *Exercises for Voice Therapy*. San Diego, CA, USA: Plural Publishing; 2019.
51. McCullough GH, Zraick RI, Balou S, et al. Treatment of laryngeal hyperfunction with flow phonation: a pilot study. *J Laryngol Voice.* 2012;2:64.
52. Mathieson L, Hirani S, Epstein R, et al. Laryngeal manual therapy: a preliminary study to examine its treatment effects in the management of muscle tension dysphonia. *J Voice.* 2009;23:353–366.

53. Gartner-Schmidt JL, Roth DF, Zullo TG, et al. Quantifying component parts of indirect and direct voice therapy related to different voice disorders. *J Voice.* 2013;27:210–216.
54. Van Stan JH, Roy N, Awan S, et al. A taxonomy of voice therapy. *Am J Speech-Lang Pathol.* 2015;24:101–125.
55. Pichora-Fuller MK, Souza PE. Effects of aging on auditory processing of speech. *Int J Audiol.* 2003;42:11–14.
56. American Speech-Language-Hearing Association. *Guidelines for Manual Pure-Tone Threshold Audiometry*. Rockville, MD, USA: ASHA; 2005.
57. Heller Murray ES, Lupiani AA, Kolin KR, et al. Pitch shifting with the commercially available eventide eclipse: intended and unintended changes to the speech signal. *J Speech Lang Hear Res.* 2019;62:2270–2279.
58. Olsen WO. Average speech levels and spectra in various speaking/listening conditions: a summary of the Pearson, Bennett, & Fidell (1977) report. *Am J Audiol.* 1998;7:21–25.
59. Weerathunge HR, Abur D, Enos NM. Auditory-motor perturbations of voice fundamental frequency: feedback delay and amplification. *J Speech Lang Hear Res.* 2020;63:1–15.
60. Boersma P., Weenink D. Praat: doing phonetics by computer [Computer program]. Version 5214; 2011; Version 5.3-6.0. 2016. ⟨http://www.praat.org⟩.
61. García-Pérez MA. Forced-choice staircases with fixed step sizes: asymptotic and small-sample properties. *Vision Res.* 1998;38:1861–1881.
62. Burnett TA, Senner JE, Larson CR. Voice F0 responses to pitch-shifted auditory feedback: a preliminary study. *J Voice.* 1997;11:202–211.
63. Abur D, Subaciute A, Daliri A, et al. Feedback and feedforward auditory-motor processes for voice and articulation in Parkinson's disease. *J Speech Lang Hear Res.* 2021;64:1–13.
64. Hogikyan ND, Sethuraman G. Validation of an instrument to measure voice-related quality of life (V-RQOL). *J Voice.* 1999;13:557–569.
65. Fairbanks G. *Voice and Articulation Drillbook.* . New York: Harper; 1960.
66. MathWorks. MATLAB 2016 (Version b). 2016; Natick, MA, United States.
67. Tourville JA, Reilly KJ, Guenther FH. Neural mechanisms underlying auditory feedback control of speech. *Neuroimage.* 2008;39:1429–1443.
68. Abur D, Lester-Smith RA, Daliri A, et al. Sensorimotor adaptation of voice fundamental frequency in Parkinson's disease. *PLoS ONE.* 2018;13:e0191839.
69. Kempster GB, Gerratt BR, Abbott KV, et al. Consensus auditory-perceptual evaluation of voice: development of a standardized clinical protocol. *Am J Speech-Lang Pathol.* 2009;18:124–132.
70. Koo TK, Li MY. A guideline of selecting and reporting intraclass correlation coefficients for reliability research. *J Chiropr Med.* 2016;15:155–163.
71. Li Y, Tan M, Fan H, et al. Neurobehavioral effects of LSVT® LOUD on auditory-vocal integration in Parkinson's disease: a preliminary study. *Front Neurosci.* 2021;15:181.
72. Ramig LO, Sapir S, Countryman S, et al. Intensive voice treatment (LSVT®) for patients with Parkinson's disease: a 2 year follow up. *J Neurol Neurosurg Psychiatry.* 2001;71:493–498.
73. Wright BA, Baese-Berk MM, Marrone N, et al. Enhancing speech learning by combining task practice with periods of stimulus exposure without practice. *J Acoust Soc Am.* 2015;138:928–937.
74. Liu H, Meshman M, Behroozmand R, et al. Differential effects of perturbation direction and magnitude on the neural processing of voice pitch feedback. *Clin Neurophysiol.* 2011;122:951–957.
75. Wright BA, Sabin AT, Zhang Y, et al. Enhancing perceptual learning by combining practice with periods of additional sensory stimulation. *J Neurosci.* 2010;30:12868–12877.
76. Lowell SY, Kelley RT, Colton RH, et al. Position of the hyoid and larynx in people with muscle tension dysphonia. *Laryngoscope.* 2012;122:370–377.
77. Dromey C, Nissen SL, Roy N, et al. Articulatory changes following treatment of muscle tension dysphonia: preliminary acoustic evidence. *J Speech Lang Hear Res.* 2008;51:196–208.
78. Thijs Z, Knickerbocker K, Watts CR. Epidemiological patterns and treatment outcomes in a private practice community voice clinic. *J Voice.* 2020;36:437.e11–437.e20.