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Research Note

Volitional Control of Frequency and Intensity in Speakers With and Without Hyperfunctional Voice Disorders

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ABSTRACT

Purpose: Prior studies of vocal auditory–motor control in people with hyperfunctional voice disorders (HVDs) have found evidence of unusually large responses to auditory feedback perturbations of fundamental frequency (*F*0) and more variable voice onset times in unperturbed speech. However, it is unknown whether people with HVDs perform similarly to people with typical voices when asked to make small changes in vocal parameters in volitional tasks. The purpose of this study was to compare performance on minimal movement tasks for *F*0 and intensity in people with and without HVDs.
Method: Twenty-six people with HVDs and 26 matched controls participated in tasks to assess the smallest volitional increases and decreases they could make in vocal *F*0 and intensity. Measures included the mean smallest change, variability of change, and accuracy of the direction of change. Group differences were tested with general linear models.
Results: No significant differences were found between people with and without HVDs on any of the measures. Singers produced significantly smaller mean smallest changes of both *F*0 and intensity than nonsingers.
Conclusions: Our findings support the interpretation of prior studies of auditory–motor control in people with HVDs. Specifically, unusually large responses to perturbations of vocal auditory feedback cannot be explained by a broader impairment of the ability to make small changes in the vocal parameters *F*0 or intensity. The method devised to assess minimal movements for voice is sensitive to relevant group differences, such as singing experience.
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Hyperfunctional voice disorders (HVDs) are characterized by dysregulated laryngeal muscle activation, sensations of increased effort and vocal fatigue, and varying degrees of disrupted voice quality, with or without trauma to vocal fold tissues from phonation (Hillman et al., 2020). Recent evidence from multiple sources points to underlying auditory–motor impairment in at least some people with HVDs, including impaired fundamental frequency (*F*0) discrimination and atypical adaptive responses to altered auditory feedback for *F*0 (Abur et al., 2021; Nguyen et al., 2022; Stepp et al., 2017; Tam et al., 2018). Specifically, people with HVDs were more likely to produce extreme adaptive responses to altered *F*0 feedback than people with typical voices (Abur et al., 2021). These findings suggest an impaired ability to integrate auditory feedback for vocal learning in at least a subset of people with HVDs, in the setting of impaired auditory discrimination, as either an etiological factor or a result of having the disorder. However, without specific examination of

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volitional vocal control, it is unknown whether this truly represents an impairment of auditory–motor learning, or if people with HVDs were unable to make smaller *F*0 changes due to other limitations of the motor control system or the effector, that is, the peripheral vocal mechanism. Clarifying the interpretation of these findings is crucial for elucidating the pathophysiology of these disorders and, in turn, developing evidence-based assessment and treatment approaches that their root causes.

Speech motor control relies on combined feedforward and feedback control systems for fluent, accurate speech (Parrell et al., 2019; Tourville & Guenther, 2011). When a speaker produces an utterance, motor commands are sent to the involved musculature, but the control system also predicts the sensory outcomes of the intended actions, including auditory feedback. If a mismatch between actual and predicted auditory feedback occurs, this provides an error signal that can elicit involuntary, reflex-like corrections (Hain et al., 2000). Error signals also may be integrated into the feedforward planning for future productions, that is, adaptive sensorimotor learning. Adaptive sensorimotor learning for speech has been shown to be an implicit process, that is, it does not appear to be driven by explicit strategies (Kim & Max, 2021; Lametti et al., 2020). In fact, even when participants were instructed to ignore auditory feedback that had been shifted in *F*0, thus producing a feedback mismatch, they produced adaptive responses that were not significantly different to when they were instructed to compensate for the *F*0 shift (Keough et al., 2013).

Auditory–motor impairment may be one underlying factor in HVDs (Hillman et al., 2020). On average, people with HVDs have worse pitch discrimination (related to *F*0) both for pure tones (Nguyen et al., 2022; Tam et al., 2018) and for their own voices (Abur et al., 2021). This may suggest that people with HVDs have less well-defined (i.e., larger) auditory targets for vocal *F*0. People with HVDs also are more likely to produce atypical adaptive responses to *F*0-shifted auditory feedback. On average, typical responses oppose the *F*0 shift (often referred to as compensation), although the distribution of typical responses spans both opposing responses and responses in the same direction as the perturbation (often referred to as following; Miller et al., 2023). People with HVDs are more likely to have responses that fall outside a typical distribution, in either direction. Furthermore, these atypical adaptive responses were associated with worse auditory discrimination for *F*0 (Abur et al., 2021).

Evidence regarding reflexive feedback control in people with HVDs is mixed. In one study, participants with HVDs produced significantly larger reflex-like responses to *F*0 shifts than people with typical voices (n = 61 controls, n = 22 participants with HVDs; Ziethe et al., 2019). However, in a larger study, no significant differences were found (n = 62 controls, n = 62 participants with HVDs; Abur et al., 2021). In addition to sample size, an important methodological difference between these two studies was the size of the *F*0 shift, which was very large in Ziethe et al. (2019; 700 cents) and elicited larger reflexive responses (control group: M = 43.6 cents, SD = 24.0 cents; HVD group: M = 69.8 cents, SD = 33.1 cents), and relatively small in Abur et al. (2021; 100 cents) and elicited smaller responses (control group: M = 13 cents, SD = 13 cents; HVD group: M = 16 cents, SD = 14 cents). One interpretation of the results of these studies is that larger reflexive responses in people with HVDs may occur due to underlying auditory–motor impairment. Furthermore, when the results from discrimination, adaptive, and reflexive experiments are considered together, they suggest that people with HVDs may have impaired auditory discrimination and impaired involuntary auditory–motor processes for vocal control and learning. However, the current body of evidence is not yet sufficient to rule out a second possible interpretation, that is, larger responses to *F*0-shifted auditory feedback in people with HVDs might occur because of impairments of the peripheral vocal mechanism or of volitional control, rather than a specific impairment of sensorimotor learning.

For example, people with HVDs often demonstrate peripheral impairment of vocal production, such as vibratory impairment due to phonotrauma, which subsequently affects *F*0 and intensity (Bastian et al., 1990; Carroll et al., 2006; Toles et al., 2021). Furthermore, increased laryngeal tension during vocal production may also be present in people with HVDs, not specific to auditory–motor integration tasks (Stepp et al., 2010), which may also affect vocal parameters such as *F*0 even in the absence of phonotrauma (Van Stan et al., 2021). Thus, atypically large adaptive vocal responses to *F*0-shifted auditory feedback in the Ziethe et al. (2019) and Abur et al. (2021) studies in people with HVDs could simply have been due to an inability to make smaller vocal changes. However, the difference between findings for reflexive feedback control in people with HVDs in these two studies does not support this interpretation. Specifically, no group differences were found when participants made small responses to small auditory feedback perturbations in Abur et al. (2021), whereas people with HVDs produced larger responses than people with typical voices when the auditory feedback perturbations were large in the study by Ziethe et al. (2019).

To clarify the interpretation of the above findings, evidence regarding volitional control of small changes in vocal parameters in people with HVDs is needed. If previous findings of atypically large adaptive responses to *F*0-

shifted feedback in people with HVDs were driven by impairments such as phonotrauma or laryngeal tension affecting all vocalizations, we would expect to see larger mean volitional changes in vocal parameters in people with HVDs than people with typical voices. On the other hand, if the previous findings were driven by impaired sensorimotor learning, which is primarily an implicit,[1] involuntary process, we would expect to see no difference in mean volitional changes in vocal parameters between people with HVDs and people with typical voices. However, in this study, it was important to include not only measures of mean volitional changes in vocal parameters but also measures of accuracy and variability. Because there is evidence of increased variability of voice onset times during volitional speech in people with HVDs (McKenna et al., 2020), it also was possible that increased variability would be observed in volitional control of *F*0 and intensity, reflected in larger standard deviations for the volitional control tasks. This finding would be consistent with the suggestion that people with HVDs have larger sensory targets for vocal parameters.

To date, studies of sensorimotor control in people with HVDs have focused primarily on *F*0, although other vocal parameters such as intensity are also impacted in the vocal production of people with HVDs (Bastian et al., 1990; Carroll et al., 2006; Toles et al., 2021). There is conflicting evidence regarding the extent to which the vocal parameters *F*0 and intensity are controlled independently versus in an integrated fashion in speakers with typical voices (Patel et al., 2011, 2015). Future research should address sensorimotor control of vocal parameters other than *F*0 in this population, to determine whether atypical findings represent impairments of specific vocal parameters or a broader impairment of vocal sensorimotor control.

The purpose of this study was to assess volitional vocal sensorimotor control in individuals with and without HVDs. To study sensory and motor aspects of jaw control in people who stutter, De Nil and Abbs (1991) and Daliri et al. (2013) used a task to assess participants' volitional control of small jaw displacements relevant for speech, referred to as a minimal movement task. In the present study, we devised an adaptation of this task to assess participants' abilities to make small changes in vocal *F*0 and intensity, specifically by examining differences between two utterances, as occurs in adaptive auditory–motor learning paradigms. We assessed the smallest volitional changes participants were able to make (minimal movement), the percentage of trials with *F*0 or intensity changes in the correct direction (task accuracy), and variability. Thus, this study addressed the following questions: (a) Do people with HVDs produce larger mean changes than people with typical voices in tasks assessing minimal movement for vocal *F*0 and intensity? and (b) Are people with HVDs less accurate or more variable in these same minimal movement tasks? We hypothesized that people with and without HVDs would not differ significantly on mean minimal movements for *F*0 and intensity, reflecting grossly preserved volitional movement capacity for voice in people with HVDs. However, we hypothesized that people with HVDs would be less accurate and more variable in their performance of minimal movement tasks, consistent with prior findings of more variable voice onset times in people with HVDs (McKenna et al., 2020).

Method

All procedures were approved by the institutional review board (IRB) at the University of Washington (IRB Approval No. STUDY00006254). All participants provided informed consent prior to completing study procedures.

Participants

Twenty-six individuals with HVDs and 26 participants with typical voices were matched pairwise for relevant variables including age (±5 years), voice *F*0 range (typically masculine vs. typically feminine), and singing experience. Participant characteristics are included in Table 1. The participant age range was limited to 18–65 years to eliminate two potential confounding factors: (a) ongoing auditory–motor and laryngeal development in pediatric speakers (Kent, 1976; Scheerer et al., 2016) and (b) compensation for glottal insufficiency (Belafsky et al., 2002) in presbyphonia. Both participant groups included more individuals who were assigned female than male at birth (21 vs. five in each group). HVDs are more prevalent in female speakers, justifying skewed enrollment with respect to sex (Roy et al., 2005). In this study, *musician* was defined as an individual with musical training beyond secondary school. Nonsinger musicians were excluded due to confounding effects on *F*0 perception (Micheyl et al., 2006). An exception was made to include musicians who were singers due to the high prevalence of HVDs in this population (Pestana et al., 2017). *Singer* was defined as an individual with postsecondary singing training and/or performing experience. Speakers of tonal languages were excluded due to confounding effects on auditory–motor control of vocal *F*0 (Liu et al., 2010). Participants taking hormone treatments that affect vocal *F*0 were only included if they reported no changes in hormone treatment for at

[1]Although sensorimotor learning for speech is thought to be a primarily implicit process, evidence suggests a role for combined explicit and implicit cues for learning in a therapeutic context (Perta et al., 2023).

Table 1. Participant characteristics.

Group	Age	Sex assigned at birth	Gender	Singers
Control	*M* = 39.35 (*SD* = 14.09, range: 22–65)	21 AFAB, 5 AMAB	19 cis women, 4 cis men, 2 nonbinary, 1 trans man	13 singers, 13 nonsingers
HVD	*M* = 39.42 (*SD* = 12.68, range: 20–64)	21 AFAB, 5 AMAB	18 cis women, 5 cis men, 2 nonbinary, 1 trans man	13 singers, 13 nonsingers

Note. AFAB = assigned female at birth; AMAB = assigned male at birth; HVD = hyperfunctional voice disorder.

least 12 months (n = 2 control participants, n = 2 participants with HVDs).

Participants with HVDs were recruited from the Laryngology Clinic at the University of Washington Medical Center and from the University of Washington Speech and Hearing Clinic in the Department of Speech and Hearing Sciences. Participants with HVDs had a confirmed diagnosis of an HVD, with or without phonotrauma (e.g., benign vocal fold lesions, muscle tension dysphonia), provided by a board-certified otolaryngologist (see Table 2). Individuals with voice diagnoses related to neoplasm, neurological conditions, and/or presbyphonia were excluded. Control participants reported no history of voice disorders and completed an auditory-perceptual and vocal health history screening by the first author (M.R.K.-S., a certified speech-language pathologist with experience assessing and treating individuals with voice disorders). All participants reported no other known neurological, speech, language, or hearing disorders. Participants were screened to ensure typical hearing using pure-tone air conduction at octave frequencies from 125 to 8000 Hz, with a pass criterion of 25 dB HL at all frequencies for individuals under 50 years of age (American Speech-Language-Hearing Association, 2018), or 25 dB HL at 125–1000 Hz and 40 dB HL at 2000–8000 Hz for individuals aged 50 years and older (Schow, 1991). Overall severity of dysphonia was rated on a visual analog scale (0–100, where 0 = *typical voice for age/gender/culture*; 100 = *severe*) by an experienced speech-language pathologist who was blinded to group assignment. The mean overall severity rating for control participants was 4.0 (*SD* = 3.1, range: 0.6–10.5). The mean overall severity rating for participants with HVDs was 7.7 (*SD* = 8.7, range: 0.9–35). Intrarater reliability was tested by randomly repeating 20% of samples and was good (r = .87). Participants completed the Voice Handicap Index–10 (VHI-10; Rosen et al., 2004). The mean VHI-10 score for control participants was 2.5 out of a possible 40 points (*SD* = 2.5, range: 0–8). The mean VHI-10 score for participants with HVDs was 11.2 (*SD* = 2.5, range: 1–33).

Table 2. Diagnoses in the hyperfunctional voice disorder group (n = 26).

Diagnosis	Number of participants
Primary muscle tension dysphonia	13
Bilateral benign vocal fold lesions and vocal hyperfunction	9
Unilateral benign vocal fold lesion and vocal hyperfunction	2
Bilateral vocal fold edema and vocal hyperfunction	1
Unilateral sulcus vocalis status post excision of benign vocal fold lesion and vocal hyperfunction	1

Procedure

Setup

All participants underwent the same procedures. They were seated in a sound-attenuated booth in front of a computer monitor used to display stimulus prompts. These data were collected as part of a larger study that included measures of auditory discrimination and adaptive auditory–motor vocal learning. Voice signals were captured with an omnidirectional ear-set microphone (Shure MX153) placed 7 cm from the center of the mouth at a 45° angle. Auditory feedback was presented to the participant through insert earphones (Etymotic ER-2). Microphone gain was adjusted with a preamplifier (RME Quadmic II), and the signal was digitized at 44.1 kHz by a soundcard (RME Fireface UCX). A microphone intensity reference tone was recorded for calibration using a monotone electrolarynx (TruTone, Griffin Labs). Sound pressure level of the reference tone (Bruel and Kjaer Type 2250 SLM) was recorded to enable calibration of recordings to SPL in postprocessing as needed. Participants heard near real-time auditory feedback of their own voice (10- to 30-ms delay; Weerathunge et al., 2020). Earphone levels were adjusted with an amplifier (Behringer Xenyx Q802) to provide 0-dB amplification relative to the microphone signal. Software and hardware systems were calibrated using a 2-cc coupler (Type 4946, Bruel and Kjaer) connected to a sound-level meter (Type 2250A with a Type 4947 ½" Pressure Field Microphone, Bruel and Kjaer).

Task

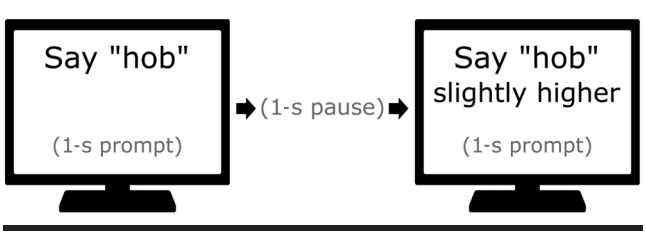
The minimal movement task, adapted from Daliri et al. (2013), assessed the smallest producible change in vocal frequency ($F0$; Brennan, 1926; Seashore & Jenner, 1910) and intensity (Jacobsen, 1936) that each participant

was able to voluntarily produce in both directions (i.e., mean smallest change), comprising four experimental conditions: higher (*F*0 up), lower (*F*0 down), louder (intensity up), and softer (intensity down). During this task, participants produced a series of pairs of syllables with a 1-s interstimulus interval (Seashore & Jenner, 1910) in response to prompts on a computer monitor (see Figure 1). The syllable "hob" was chosen to elicit a vowel similar to the sustained /ɑ/ used in prior studies of auditory–motor control in this population but in a speech task. The initial /h/ discouraged creaky onsets and the final voiced consonant encouraged a vowel that was long enough for analysis. Participants were given verbal instructions explaining the task and the condition before each block of trials. They were instructed to produce the first token at a comfortable pitch and loudness, followed by a second production that was slightly higher in pitch, slightly lower in pitch, slightly louder, or slightly softer than their first production within the syllable pair. They were instructed to produce the smallest change possible. Before each new condition (higher, lower, louder, softer), a single presentation of a prerecorded model was played to the participant via the headphones. Models differed by 3 dB or 1 semitone (ST). A single model was used for all participants, with a mean *F*0 on the starting token of 196 Hz (range: 190–206). The examiner conversed with the participant between presentation of the model and subsequent trials to discourage direct imitation of the model. Participants were then verbally instructed to produce smaller changes than the model if possible. Auditory targets other than the initial model were not provided to avoid confounding influences of *F*0 and intensity discrimination and matching abilities. After five practice trials, two sets of 10 trials were performed in each of the four conditions, with direction (up/down) of sets interleaved within *F*0 and intensity blocks. The order of *F*0 and intensity experiments and direction (up/down) was counterbalanced across participants. The entire experiment took approximately 15 min.

Data Analysis

Onsets and offsets of vowels produced by the participants were selected manually for each trial, and *F*0 and intensity were extracted using *Praat* (Boersma & Weenink, 2016) scripts. Mean *F*0 was calculated using an autocorrelation method and mean intensity using a root-mean-square method. To isolate the response of the feedforward motor control system during steady-state phonation, the 40- to 120-ms portion of each vowel within each paired syllable trial was analyzed. The difference in *F*0 of the second vowel relative to the first was measured in cents for the *F*0 experiment in both the high/lower conditions. The difference in intensity of the second vowel relative to the first was measured in decibels (dB) for the intensity experiment in both the louder/softer conditions. Because averaging all trials could cause trials that are produced in the wrong direction (e.g., a decrease in *F*0 when instructed to increase pitch) to "improve" a participant's score, only the 10 best trials (i.e., smallest change in ST or dB in the correct direction) were averaged for each condition, resulting in a measure that was called *mean smallest change* (Brennan, 1926; Jacobsen, 1936; Seashore & Jenner, 1910). The standard deviation of all trials also was calculated as a measure of *variability*. Prior work shows increased variability in voice onset times in people with HVDs (McKenna et al., 2020), suggesting this was a dependent measure of interest. Finally, to account for accuracy in the direction of change, a third measure was calculated: the percentage of trials with change produced in the correct direction (i.e., *percent correct*). This accuracy measure was included because atypical auditory–motor integration in response to *F*0 perturbation has been observed in individuals with HVDs both in terms of degree and direction of adaptation (Abur et al., 2021). All three measures (mean smallest change, variability, and percent correct) were calculated for each of the four conditions for each participant.

Figure 1. Structure of a single trial in the minimal movement task (example shown for the fundamental frequency–up condition).



Statistical Analysis

Statistical analyses were conducted using SPSS Statistics (Version 26; IBM, 2019). Significance level was defined a priori as $\alpha = .05$. For the purposes of statistical analysis, the mean smallest change scores for the minimal movement tasks were converted to absolute values (i.e., decreases in *F*0 or intensity were analyzed as positive values). To examine the relationship between voice disorder status and voluntary control of vocal *F*0 and intensity, repeated-measures general linear models were conducted (function GLM) to examine mean smallest change and variability with between-subjects factors of HVD status (HVD vs. control) and singing experience (nonsinger vs. singer), and the within-subject factor direction of change (up vs. down), and all two- and three-way interactions. Effect sizes were measured using the squared partial curvilinear correlation η^2 ($\eta^2 = .01$ indicates a small effect, $\eta^2 = .06$ indicates a medium effect, $\eta^2 = .14$ indicates a large effect). The sample size in the present study allowed

Table 3. Mean starting values by group for minimal movement tasks.

Group	*F*0 increase	*F*0 decrease	Intensity increase	Intensity decrease
Control	*M* = 173 Hz, *SD* = 43	*M* = 181 Hz, *SD* = 46	*M* = 65.1 dB, *SD* = 3.7	*M* = 66.5 dB, *SD* = 3.2
HVD	*M* = 174 Hz, *SD* = 40.5	*M* = 179 Hz, *SD* = 41	*M* = 65.1 dB, *SD* = 3.5	*M* = 65.5 dB, *SD* = 3.9

Note. *F*0 = fundamental frequency; HVD = hyperfunctional voice disorder.

for 80% power to detect small effects ($\eta^2 = .03$). These analyses were conducted for the vocal parameters *F*0 and intensity (two outcome variables × two vocal parameters = four GLMs). Because percent correct data were not normally distributed due to ceiling effects, a categorical analysis was employed, motivated by the findings of Abur et al. (2021). The 10th percentile was calculated for the control group, and this was used as a criterion to categorize accuracy for each participant and each condition into typical (≥ 10 percentile) and atypical (< 10 percentile). A chi-square test for association was used to assess whether there was a difference in the number of participants with atypical accuracy scores between groups. Effect size *w* was measured for chi-square tests. The sample size in the present study allowed for 80% power to detect medium effects ($w = 0.39$). These analyses were conducted for the vocal parameters *F*0 and intensity (one outcome variable × two vocal parameters × two directions = four chi-square tests). No familywise error corrections were employed. Because the study hypotheses were primarily null hypotheses, the omission of Type I error corrections provides a more rigorous test.

Results

The mean starting values (i.e., values in the first token of each trial) and standard deviations are provided by group for each task in Table 3. This information is provided for context because starting *F*0 and intensity were not prescribed. Descriptive statistics for HVD and control group results for the three variables (mean smallest change, variability, and percent correct) and the two vocal parameters (*F*0 and intensity) are provided in Tables 4 and 5.

Mean Smallest Change

Group data are shown in Figure 2. Repeated-measures GLMs were conducted to examine mean smallest change with between-subjects factors of HVD status (HVD vs. control) and singing experience (nonsinger vs. singer) and the within-subject factor direction of change (up vs. down), and their interactions (see Tables 6 and 7).

There was no significant main effect of HVD status (control/HVD) for mean smallest *F*0 change. There were significant main effects of singing experience ($p = .001$) and direction ($p = .002$), both with large effect sizes. Singers produced significantly smaller *F*0 changes than nonsingers. Changes in *F*0 were significantly smaller when decreasing *F*0 than when increasing *F*0. There was a significant interaction of direction and singing experience ($p = .014$), with a medium effect size. The difference between singers and nonsingers was greater for *F*0 increases than *F*0 decreases. There were no other significant interactions.

There was no significant main effect of HVD status (control/HVD) for mean smallest intensity change. There were significant main effects of singing experience ($p = .009$) and direction ($p < .001$), again both with large effect sizes. Singers produced significantly smaller changes in intensity than nonsingers. Intensity changes were significantly smaller when increasing intensity than when decreasing intensity. There were no significant interactions.

Variability

Group data for variability of *F*0 and intensity changes are shown in Figure 3. Repeated-measures GLMs were conducted to examine variability with between-subjects factors of HVD status (HVD vs. control) and singing experience (nonsinger vs. singer) and the within-subject factor direction of change (up vs. down), and their interactions (see Tables 8 and 9).

Table 4. Mean results for hyperfunctional voice disorder (HVD) and control groups for fundamental frequency tasks.

Group	Smallest increase	Smallest decrease	Increase variability	Decrease variability	Increase accuracy	Decrease accuracy
Control	*M* = 133 cents, *SD* = 107	*M* = 86 cents, *SD* = 45	*M* = 92 cents, *SD* = 59	*M* = 77 cents, *SD* = 44	*M* = 90%, *SD* = 14	*M* = 89%, *SD* = 12
HVD	*M* = 105 cents, *SD* = 75	*M* = 87 cents, *SD* = 59	*M* = 86 cents, *SD* = 50	*M* = 76 cents, *SD* = 41	*M* = 88%, *SD* = 13	*M* = 88%, *SD* = 13

Table 5. Mean results for hyperfunctional voice disorder (HVD) and control groups for intensity tasks.

Group	Smallest increase	Smallest decrease	Increase variability	Decrease variability	Increase accuracy	Decrease accuracy
Control	*M* = 2.36 dB, *SD* = 1.24	*M* = 3.34 dB, *SD* = 1.50	*M* = 2.12 dB, *SD* = 0.63	*M* = 2.20 dB, *SD* = 0.62	*M* = 89%, *SD* = 10	*M* = 96%, *SD* = 8
HVD	*M* = 1.91 dB, *SD* = 0.83	*M* = 3.08 dB, *SD* = 1.30	*M* = 2.17 dB, *SD* = 0.65	*M* = 2.33 dB, *SD* = 0.64	*M* = 86%, *SD* = 13	*M* = 96%, *SD* = 7

There were no significant main effects of HVD status (control/HVD) or singing experience for *F*0 change variability. There was a significant main effect of direction (p = .032) with a medium effect size. Change variability was significantly less when decreasing *F*0 than when increasing *F*0. There was a significant three-way interaction of HVD status, singing experience, and direction of change (p = .011) with a medium effect size.

Figure 2. Mean smallest changes by hyperfunctional voice disorder (HVD) status and singing experience for (a) fundamental frequency (*F*0) increase, (b) *F*0 decrease, (c) intensity increase, and (d) intensity decrease.

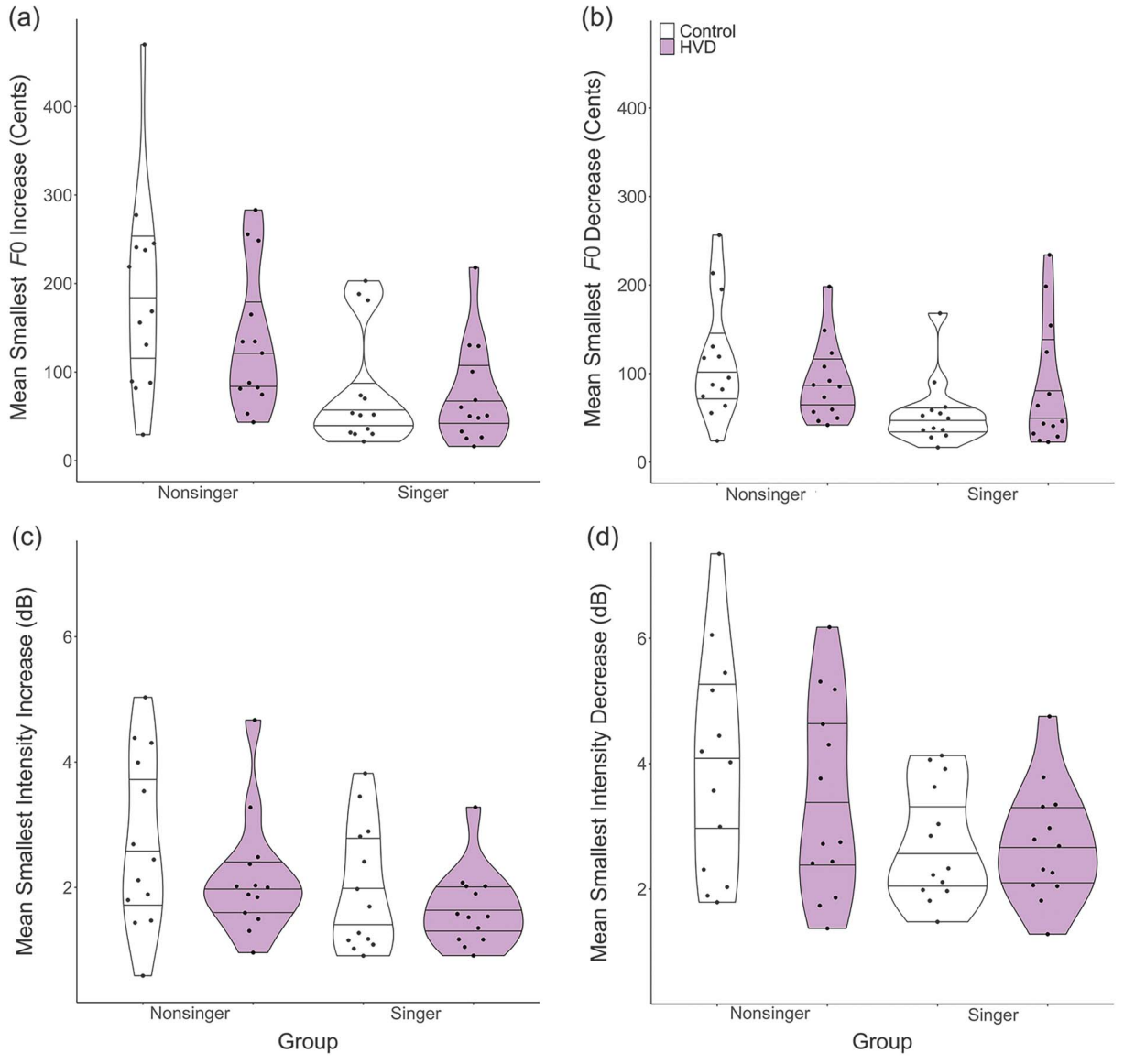


Table 6. Statistical results for mean smallest fundamental frequency changes.

Effect	*df*	η_p^2	*F*	*p*
HVD status (control, HVD)	1	.01	0.65	.423
Singing experience (singer, nonsinger)	1	.21	12.38	.001*
Direction (up, down)	1	.18	10.20	.002*
Direction × HVD Status	1	.04	2.08	.156
Direction × Singing Experience	1	.12	6.57	.014*
HVD Status × Singing Experience	1	.05	2.25	.141
Direction × HVD Status × Singing Experience	1	< .01	0.04	.835

Note. HVD = hyperfunctional voice disorder.
*Significant at $p < .05$.

Control participants had less variability in pitch increases if they were singers. Put another way, singers were significantly less variable in pitch increases than nonsingers, unless they had HVDs.

There were no significant main effects of HVD status (control/HVD) or direction for intensity change variability. There was a significant main effect of singing experience ($p = .048$) with a medium effect size. Singers were significantly less variable when changing intensity than nonsingers. There were no significant interactions.

Percent Correct

For statistical analyses, percent correct cutoff scores were derived from the 10th percentile of scores in the control group. Because significant effects of singing experience were observed in the mean smallest change and variability results, separate cutoffs were calculated for singers and nonsingers. Scores were categorized as atypical if they fell below the following thresholds: *F*0 increase, 56.00% singer/69.00% nonsinger; *F*0 decrease, 62.00% singer/79 .58% nonsinger; intensity increase, 82.00% singer/72.00% nonsinger; intensity decrease, 89.00% singer/72.00% nonsinger (see Figure 4).

Table 7. Statistical results for mean smallest intensity changes.

Effect	*df*	η_p^2	*F*	*p*
HVD status (control, HVD)	1	.03	1.51	.225
Singing experience (singer, nonsinger)	1	.14	7.48	.009*
Direction (up, down)	1	.49	48.89	< .001*
Direction × HVD Status	1	.01	0.39	.535
Direction × Singing Experience	1	.02	1.12	.296
HVD Status × Singing Experience	1	.01	0.45	.505
Direction × HVD Status × Singing Experience	1	< .01	0.12	.727

Note. HVD = hyperfunctional voice disorder.
*Significant at $p < .05$.

A chi-square test of association was performed on the categorical accuracy scores for the two vocal parameters and two directions. Results are shown in Table 10. There were no significant effects of HVD status (control/HVD) on accuracy in any of the four tasks.

Post Hoc Tests

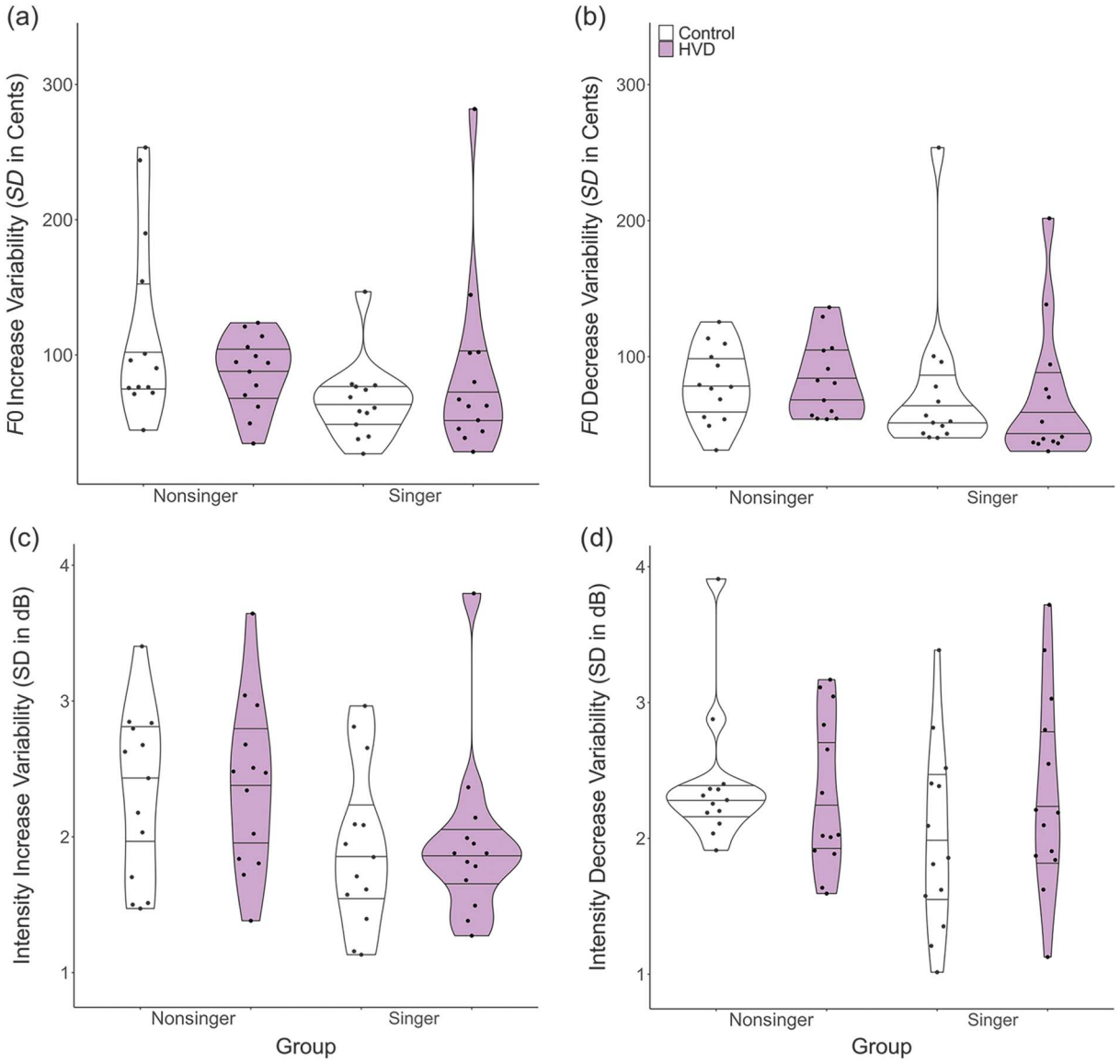
As a post hoc analysis, peak values of *F*0 and intensity were extracted and analyzed in the same way as the mean values of the 40- to 120-ms segment of phonation. This was performed to determine whether the peak value would be more sensitive to group differences than the mean. The peak was extracted from the portion of each vowel from 40 ms after the onset to 40 ms before the offset, to exclude instabilities that may occur due to phonation onset or phoneme transitions. The same statistical analyses were performed. Results are given in Supplemental Material S1. Similar patterns of statistical results were found; however, relationships were generally weaker, and in some cases, relationships that were significant in the main analysis were not significant in the post hoc analysis.

Discussion

The purpose of this study was to determine whether differences exist between people with and without HVDs in volitional control of small changes in vocal *F*0 (perceived as pitch) and intensity (perceived as loudness). We developed a protocol to elicit these changes modeled after Daliri et al. (2013), with some key alterations to increase the similarity to speech tasks found in studies of vocal auditory–motor integration. We found no significant differences related to voice disorder status in the magnitude or accuracy of changes when individuals were instructed to make the smallest possible changes in these parameters during a speech task. However, we did find significant effects of singing experience and of the direction of the task (increase vs. decrease), which suggests this protocol is able to capture differences between speakers with different characteristics. We also found a three-way interaction of HVD status, singing experience, and *F*0 change variability. Specifically, singers showed significantly less variability in pitch increases than nonsingers, unless they had HVDs.

Consistent with our first hypothesis, we did not find evidence of differences in volitional production of minimal changes in *F*0 and intensity between people with and without HVDs. This suggests volitional control of these parameters is generally preserved in this population. It also lends support to interpretation of prior findings of unusually large adaptive responses to *F*0 feedback

Figure 3. Variability by hyperfunctional voice disorder (HVD) status and singing experience for (a) fundamental frequency (*F*0) increase, (b) *F*0 decrease, (c) intensity increase, and (d) intensity decrease.



alteration in people with HVDs as reflecting underlying impairment in auditory–motor integration (Abur et al., 2021), rather than an inability to make smaller changes due to either central or peripheral impairments. Peripheral impairment would not be expected in people with nonphonotraumatic vocal hyperfunction (i.e., primary muscle tension dysphonia), as the peripheral vocal mechanism is normal upon examination in this population. However, half of the patient sample in the present study comprised people with phonotraumatic vocal hyperfunction (including diagnoses like benign vocal fold lesions), yet there were no group differences in the ability to make small volitional vocal changes. This is also consistent with findings in Abur et al. (2021) of preserved reflex-like responses to unexpected *F*0 feedback alterations, which are small in magnitude.

We found limited evidence in support of our second hypothesis. Specifically, we found that variability of *F*0 increases is reduced in people with singing training but that this difference is not significant in people with HVDs. This suggests some subtle impact of HVDs on the consistency of volitional *F*0 control in trained vocalists. We did not find any other significant differences in the variability or accuracy of volitional production of minimal differences in *F*0 and intensity between people with and without

Table 8. Statistical results for fundamental frequency change variability.

Effect	*df*	η_p^2	*F*	*p*
Group (control, HVD)	1	< .01	.09	.760
Singing experience (singer, nonsinger)	1	.05	2.41	.127
Direction (up, down)	1	.09	4.87	.032*
Direction × HVD Status	1	< .01	0.15	.702
Direction × Singing Experience	1	.05	2.43	.126
HVD Status × Singing Experience	1	.02	0.76	.388
Direction × HVD Status × Singing Experience	1	.13	6.99	.011*

Note. HVD = hyperfunctional voice disorder.
*Significant at $p < .05$.

HVDs. In a prior study, McKenna et al. (2020) found that although mean voice onset times did not vary between people with and without HVDs, voice onset times were more variable in people with HVDs, suggesting subtle underlying sensorimotor impairment in control of voicing for speech. There are key differences between the tasks that were produced in the present study versus McKenna et al. (2020). Whereas voice onset time requires fine coordination of the timing of phonation onset, the task in the present study involves control of the spectro-temporal parameters *F*0 and intensity through small adjustments in vocal fold length, tension, adduction, and subglottal pressure. Furthermore, voice onset time is a linguistic parameter, whereas changes in *F*0 and intensity are paralinguistic parameters in English. It is noteworthy that the result for intensity increase accuracy was substantially closer to significance than any of the other findings ($p = .067$), with a small effect size ($w = 0.25$). It is possible that the study was underpowered for this parameter, given that our sample size provided 80% power to detect medium effect sizes for accuracy analyses ($w = 0.39$). Of the seven participants with HVDs who had atypical intensity increase accuracy scores, six (86%) had diagnoses of phonotraumatic vocal hyperfunction (vs. 54% of the entire HVD sample). Phonotrauma can cause increased stiffness of the vocal folds and create difficulty increasing intensity. It is possible that vibratory impairment affected these participants' accuracy in the intensity increase task. However, these results should be interpreted with caution, given the lack of statistical significance.

Table 9. Statistical results for intensity change variability.

Effect	*df*	η_p^2	*F*	*p*
HVD status (control, HVD)	1	.01	0.37	.547
Singing experience (singer, nonsinger)	1	.08	4.13	.048*
Direction (up, down)	1	.04	1.90	.175
Direction × HVD Status	1	< .01	0.18	.673
Direction × Singing Experience	1	.03	1.34	.253
HVD Status × Singing Experience	1	.01	0.40	.531
Direction × HVD Status × Singing Experience	1	.03	1.48	.229

Note. HVD = hyperfunctional voice disorder.
*Significant at $p < .05$.

We found a significant effect of singing experience on smallest changes in *F*0 and intensity, and the variability of intensity changes. It is not surprising that singers would perform better than nonsingers in these tasks, likely due to the effects of prior training in volitional control of vocal *F*0 and intensity, two parameters that are of substantial importance in musical dynamics. Trained musicians are also better at auditory discrimination of parameters such as *F*0 (Micheyl et al., 2006), which could play a role in auditory–motor control (Tourville & Guenther, 2011).

This study has some limitations. We did not control the *F*0 or intensity range produced by participants, for example, by requiring them to match a target for their first production. We chose instead to ask participants to begin with a comfortable pitch and loudness. This choice was to avoid the confound of auditory discrimination ability, which is affected in this population (Abur et al., 2021; Nguyen et al., 2022; Tam et al., 2018), and demands on *F*0 matching abilities. Our sample size was relatively small, and it is possible that the result for intensity increase accuracy in particular was underpowered. However, performance between the control and HVD groups was remarkably similar on all other variables. In addition, the average rating of overall severity of dysphonia in our sample was low ($M = 7.7$, $SD = 8.7$, range: 0.9–35). It is possible that participants with more severe dysphonia would perform differently. However, some of the hallmark characteristics of HVDs include complaints such as discomfort when speaking and vocal fatigue (Hillman et al., 2020; Solomon, 2008), which can occur in the absence of voice quality disturbance. On average, participants with HVDs in this study had VHI-10 scores indicative of clinically significant voice-related handicap (Arffa et al., 2012), with scores ranging as high as 33/40 points. This suggests a substantial impact of participants' vocal function on their daily communication even in the absence of impaired voice quality, likely due to factors other than vibratory function of the vocal folds, and may be representative of the clinical profile of many people with HVDs.

The protocol used in the present study was shown to be sensitive to group differences in volitional vocal control (e.g., singing training) and may be useful for future research in HVDs and other populations with vocal impairments, as well as for measurement of vocal control in nonclinical populations such as singers. Given recent evidence of impaired auditory discrimination and auditory–motor

Figure 4. Accuracy of change direction for (a) fundamental frequency (*F*0) increase, (b) *F*0 decrease, (c) intensity increase, and (d) intensity decrease. Dashed lines are 10th percentile, used as cutoffs for typical (above) versus atypical (below) scores. HVD = hyperfunctional voice disorder.

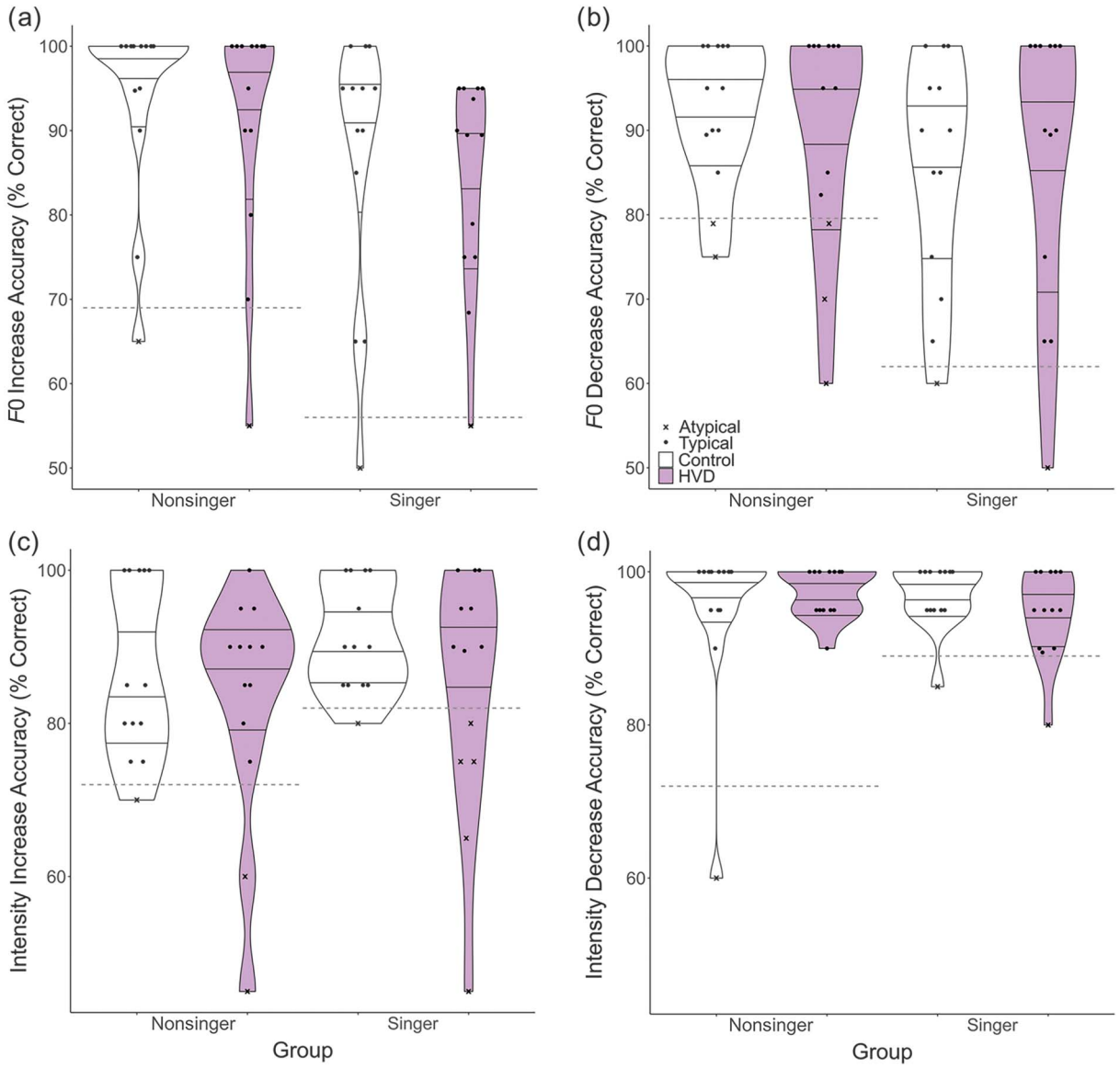


Table 10. Statistical results for change accuracy (typical vs. atypical).

Task	*df*	*w*	χ^2	*p*
*F*0 increase	1	< 0.01	< 0.01	1.000
*F*0 decrease	1	0.06	0.17	.685
Intensity increase	1	0.25	3.36	.067
Intensity decrease	1	0.08	0.35	.552

Note. *F*0 = fundamental frequency. Significance level at $p < .05$.

integration across HVDs (Abur et al., 2021; Nguyen et al., 2022), and differences in (para)laryngeal somatosensation in people with nonphonotraumatic HVDs (Shembel et al., 2024), integration of assays of both domains of sensation (auditory, somatosensory) and volitional and nonvolitional motor control mechanisms may provide unique insights into the pathophysiology of these disorders. Continued investigation of auditory perception and auditory–motor control in people with HVDs will aid in understanding their causes and refining assessment and treatment protocols accordingly.

Conclusions

This study provides preliminary evidence for preserved volitional sensorimotor control of *F*0 and intensity in individuals with HVDs. Our results lend support to the interpretation of prior findings of impaired auditory–motor integration for vocal learning in this population (Abur et al., 2021). Furthermore, we present a behavioral assay for measurement of volitional control of *F*0 and intensity that is sensitive to differences in participant characteristics such as singing experience. Further characterization of vocal sensorimotor control in people with HVDs is necessary to elucidate their pathophysiology and to facilitate improved diagnosis and treatment.

Data Availability Statement

The data sets generated are available from the corresponding author upon reasonable request.

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