Research Article

Effects of Cognitive Stress on Voice Acoustics in Individuals With Hyperfunctional Voice Disorders

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ABSTRACT

Purpose: Autonomic nervous system dysfunction has been implicated in the development and persistence of hyperfunctional voice disorders (HVDs). The purpose of this study was to determine the effects of cognitive stress, which is known to arouse the autonomic nervous system, on voice acoustics in female speakers with and without HVDs.

Method: Adult female speakers—66 with HVDs, 66 without—were recorded while speaking with and without a cognitive stressor. Root-mean-square (RMS) of amplitude, fundamental frequency \( (f_0) \), low-to-high spectral energy ratio (L/H ratio), cepstral peak prominence (CPP), and relative \( f_0 \) (RFF) were measured for each speaker and cognitive stress condition. Mixed-model analyses of variance and post hoc \( t \) tests were conducted to determine if cognitive stress affected voice acoustics and whether voice changes were greater for those with HVDs.

Results: All measures differed significantly under cognitive stress for speakers with and without HVDs. RMS and CPP increased whereas \( f_0 \), CPP, and RFF decreased under cognitive stress. Changes in these measures were not greater in those with HVDs.

Conclusion: Cognitive stress and presumed autonomic arousal affect voice similarly in female speakers with and without HVDs.

Hyperfunctional voice disorders (HVDs) are characterized by excessive or dysregulated laryngeal and perilaryngeal muscle tension. HVDs are more prevalent among female speakers (Roy et al., 2005) and include both phonotraumatic (e.g., vocal fold nodules) and nonphonotraumatic (e.g., muscle tension dysphonia) types (Hillman et al., 2020). Dysphonia, supraglottic compression, vocal fatigue, and atypical speech breathing are common signs and symptoms of HVDs (Gillespie et al., 2013; Morrison et al., 1983; Sama et al., 2001; Shim et al., 2016; Solomon, 2008).

Although HVDs are exceptionally common (Bhattacharyya, 2014), their etiology remains poorly understood. Several factors have been implicated in the development and persistence of HVDs, including psychosocial factors (Ng et al., 2013; Roy & Bless, 2000; Roy et al., 2000; Toles et al., 2021), voice use factors (Altman et al., 2005; Van Houtte et al., 2011; Van Stan et al., 2021), auditory–motor impairment (Abur, Subaciute, et al., 2021; Stepp et al., 2017), and autonomic dysfunction (Demmink-Geertman & Dejonckere, 2002; Park & Behlau, 2011).

The autonomic nervous system regulates involuntary body functions, like respiration and heart rate. A person with autonomic dysfunction may experience more frequently neurovegetative symptoms like dizziness, sweating, and palpitations (Demmink-Geertman & Dejonckere, 2002; Park & Behlau, 2011), symptoms that are also associated with autonomic arousal. The autonomic nervous system is aroused under stress conditions, such as increased cognitive load (Bear et al., 2007). This arousal is instigated by activation of the sympathetic division of the autonomic nervous system, which regulates stress responses,
and inhibition of the parasympathetic division, which regulates “rest and digest” behaviors (Ziegler, 2012). Along with cardiovascular, respiratory, and electrodermal symptoms, autonomic arousal may induce excessive muscle tension, including that of the laryngeal muscles (Ellaway et al., 2010; Helou et al., 2013; Kyle & McNeil, 2014).

Autonomic arousal is also associated with changes in the voice. For example, fundamental frequency ($f_0$) and amplitude have been shown to respond to cognitive stress (Boyer et al., 2018; Dietrich, 2008; Lively et al., 1993; Mendoza & Carballo, 1998; Perrine & Scherer, 2020; Ruiz et al., 1996; Scherer et al., 2002; van Lierde et al., 2009; van Mersbergen & Payne, 2020), although the presence and direction of changes in these measures is inconsistent across studies. Cognitive stress-induced changes in measures of voice quality, including low-to-high spectral energy ratio (L/H ratio) and cepstral peak prominence (CPP), have also been documented (MacPherson et al., 2017; van Mersbergen & Payne, 2020), although not in older adults (Abur, MacPherson, et al., 2021). The nature of observed changes in L/H ratio and CPP—L/H ratio decreased while CPP increased—suggests that speakers may use a more pressed voice when speaking under cognitive stress (MacPherson et al., 2017). This interpretation is consistent with evidence of increased laryngeal muscle activation during autonomic arousal (Helou et al., 2013). It is also consistent with a finding of decreased relative $f_0$ (RFF) at voicing offset (Dahl & Stepp, 2021), which suggests elevated laryngeal muscle tension under cognitive stress.

The evidence for voice changes under cognitive stress, however, is limited to studies of speakers with typical voices. There is reason to believe the effects of cognitive stress on voice may differ in speakers with HVDs. First, the mechanism by which cognitive stress is believed to affect voice is through arousal of the autonomic nervous system (Bear et al., 2007; MacPherson et al., 2017). Autonomic dysfunction is more prevalent among individuals with HVDs, in both the phonotraumatic and non-phonotraumatic types (Demmink-Geertman & Dejonckere, 2002; Park & Behlau, 2011). Thus, autonomic arousal induced by cognitive stress may have an exaggerated effect on voice in this population. Second, there are notable similarities in voice acoustics and laryngeal muscle activity between speakers with typical voices under high cognitive load and speakers with HVDs under usual cognitive load. Although there is no established acoustic profile of HVDs, it may include reduced amplitude (Belsky et al., 2021), $f_0$ (Cooper, 1974; Gillespie et al., 2019; Shim et al., 2016), L/H ratio (Awan & Roy, 2005; Belsky et al., 2021), CPP (Awan & Roy, 2005; Belsky et al., 2021; Shim et al., 2016; Van Stan et al., 2021), and RFF (Heller Murray et al., 2017; Roy et al., 2016; Stepp et al., 2010, 2011). Many of these same changes have been observed in speakers with typical voices when speaking under cognitive stress. Thus, the acoustic and electromyographic evidence suggests that cognitive stress induces hyperfunctional voice behaviors. Cognitive stress may, therefore, exacerbate symptoms of vocal hyperfunction in individuals with HVDs.

The purpose of this study was to determine the effects of cognitive stress on acoustic measures of voice in female speakers with and without HVDs. We manipulated cognitive load with a sentence-level Stroop task (Stroop, 1935) and measured root-mean-square (RMS) of amplitude, $f_0$, L/H ratio, CPP, and RFF in both groups under typical and increased cognitive load. These acoustic measures were selected because of their associations with vocal hyperfunction and their use in previous research on voice under cognitive stress. We hypothesized that, in both groups, RMS and CPP would increase, and $f_0$, L/H ratio, and RFF would decrease under cognitive stress. We also hypothesized that stress-induced changes in all of these acoustic measures would be greater in individuals with HVDs than in those without.

**Method**

**Participants**

Participants were 132 female speakers—66 with HVDs ($M = 31.8$ years, $SD = 13.5$ years, range: 18–66 years) and 66 without HVDs, age-matched within 5 years ($M = 31.8$ years, $SD = 13.8$ years, range: 18–68 years). Participants with HVDs were evaluated by a laryngologist and subsequently diagnosed with muscle tension dysphonia ($n = 41$), vocal fold nodules ($n = 18$), unspecified phonotraumatic HVD ($n = 6$), or vocal fold polyp ($n = 1$). Participants reported no other history of speech, language, or hearing disorders, nor any color blindness. Most participants (109/132) passed a hearing screening with age-appropriate thresholds. All participants provided written informed consent, in compliance with the Boston University Institutional Review Board.

To characterize the overall severity of voice impairment in the sample, a voice-specializing speech-language pathologist (SLP) blinded to the study purpose and participant identities completed an auditory-perceptual evaluation of all participants. These evaluations were completed on a

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1 Only speaker sex is reported, as gender information was not available.

2 Screening criteria were maximum thresholds of 25 dB HL at octaves from 125 Hz to 4 kHz for younger adults. For those over 40, the maximum threshold at octaves from 1 to 4 kHz was 40 dB HL. Hearing screening results were not available for 19 participants. Three others had unilaterally elevated thresholds (30–55 dB HL) at a single frequency. One participant had elevated thresholds (50 dB HL) at 2 and 4 kHz in the right ear and 4 kHz in the left.
Participants were recorded as they read aloud 12 sentences with an embedded Stroop task. All acoustic recordings were collected in either a sound-treated booth at Boston University with a condenser headset microphone (Model SM35XLR; Shure) or in a quiet room at Boston Medical Center with a dynamic headset microphone (Model WH20XLR; Shure) sampled at 44.1 kHz with a 16-bit resolution. In both settings, the microphone was placed at a fixed distance of 7 cm from the mouth at a 45° angle.

The sentence-level Stroop task allowed for manipulation of cognitive load during voice production in running speech. Each sentence contained four color terms printed in colored ink (e.g., “Then our pal gave blue, purple, brown, and red new posters to us”). All other words were printed in black ink. See the work of Dahl and Stepp (2021) for the full set of sentences. Participants were instructed to say the color of the ink in which a color term was printed, rather than the word itself. They were asked to use their usual speaking voice while reading the sentences, with no further instructions regarding speaking rate. In the first six sentences, the color terms and ink matched (e.g., “blue” printed in blue ink). These sentences comprised the congruent condition, which entailed a typical cognitive load. In the remaining six sentences, the color terms and colored ink differed (e.g., “red” printed in blue ink). These sentences comprised the incongruent condition, in which cognitive load was increased and thus participants were speaking under cognitive stress.

The congruent condition always preceded incongruent due to the duration of laryngeal responses to autonomic arousal. This response may last for several minutes (Helou et al., 2013), and so a fixed order ensured that acoustic measures from the congruent condition were not contaminated by autonomic arousal induced during the incongruent condition.

Within each sentence were sequences of voiced–voiceless–voiced phonemes, which allow RFF to be measured at voicing offset and onset. Each sentence contained at least four such sequences, such as /ụpi/ in “blue,” “pink,” and /ụpọọ/ in “new posters.” There were 34 possible RFF tokens per condition.

### Voice Analysis

Before analyzing voice acoustics, samples were evaluated for substantial slowing of speech rate in the incongruent condition. Cognitive load may result in either slower or faster speech, depending in part on the time demands of the task (Jameson et al., 2010; Lively et al., 1993). Participants in this study were given no time limit to complete the task and so had the option of using markedly slower speech (i.e., long pauses within the sentence) as a strategy to minimize cognitive load (Berthold & Jameson, 1999). A substantial slowing of speech could, therefore, indicate that the intended stressor was not achieved, and so sentences in the incongruent condition that were longer than the corresponding sentence in the congruent condition by more than 4 s were excluded. This cutoff corresponded to the authors’ perception of substantial slowing and is consistent with our prior work (Dahl & Stepp, 2021). We excluded a total of 35 sentences from 28 participants (2% of the data set). The remaining data set, used for all analyses described below, included 372 sentences from participants with HVDs (an average of 5.6 per participant) and 385 sentences from control participants (an average of 5.8 per participant).

### Amplitude

Each sentence in the Stroop task was analyzed individually for RMS of amplitude. RMS was calculated on speech samples from which silence between words had been removed using a custom script in MATLAB (MathWorks). This script applied a lowpass Butterworth filter with a frequency cutoff of 400 Hz and created an envelope of the resulting signal. A trained technician visually inspected the envelope and selected an amplitude threshold that best separated speech from silence. All parts of the signal with an amplitude below this threshold were removed by the custom script. The resulting waveform was again visually inspected and the resulting sound file played to ensure that silence was removed without loss of the speech signal. Because RMS is a measure of average amplitude, periods of silence may bias the measure toward an artificially low value. RMS was extracted from speech-only samples to reduce such bias.

RMS in volts was extracted from each of these samples using a custom MATLAB script. RMS was converted to dB using Equation 1, in which RMS₂ was the RMS of a given sentence, and RMS₁ was the mean RMS of all sentences produced by the speaker in the congruent condition.

\[
\text{dB} = 20 \times \log_{10} \frac{\text{RMS}_2}{\text{RMS}_1}. \tag{1}
\]

This approach to dB conversion—in which each participant’s RMS was normalized to their mean in the
RFF tokens were excluded due to the presence of any of the following issues, each of which would prevent valid RFF estimation (number [percent] rejected): voicing of the voiceless segment (6,673 [38.0%]), pauses > 250 ms between phonemes (1,078 [6.1%]), aperiodicity during the voice segment (964 [5.5%]), failure to reach steady state during the voiced segment (325 [1.9%]), word or sound errors (262 [1.5%]), voiced segment with < 10 voicing cycles (105 [0.6%]), unresolvable pitch tracking error (91 [0.5%]), or excessive background noise (26 [0.1%]). The final data set included 4,335 RFF tokens in the congruent condition and 3,675 RFF tokens in the incongruent condition. There was an average of 32.8 tokens per participant in the congruent condition (30.1 for those with HVDs, 34.7 for controls) and 27.8 in the incongruent condition (25.7 for participants with HVDs, 30.0 for controls). There were at least six tokens per participant in each condition (range: 6–59), in accordance with the recommended minimum for reliable RFF measurement (Eadie & Stepp, 2013).

RFF was calculated by six trained technicians, each of whom measured RFF for 22 participants on average (range: 9–36). All technicians first analyzed RFF from a shared set of 10 participants for calculation of interrater reliability. Interrater reliability was assessed with an intraclass correlation coefficient for consistency (ICC[C, k]). To measure intrarater reliability, each technician reanalyzed data from three participants randomly selected from that technician’s completed set. Intrarater reliability was assessed with Pearson product–moment correlations. Both intrarater (r = .94) and intrarater (mean r = .95; range: .92–.97) reliability were excellent for all technicians.
Statistical Analysis

We tested our hypotheses that cognitive stress would affect voice acoustics in speakers with and without HVDs with mixed-model analyses of variance (ANOVAs). Specifically, four mixed-model ANOVAs were constructed to measure the main effect of group (HVD, control), condition (congruent, incongruent), and their interactions on RMS (dB), \( f_o \) (Hz), L/H ratio (dB), and CPP (dB). Two additional mixed-model ANOVAs were constructed to measure the main effects of group, condition, voicing cycle (1–10), and all interactions on mean RFF offset and onset. Speaker was entered as a random factor for all models. Effect sizes for significant effects and interactions were calculated as partial curvilinear correlations (\( \eta^2_p \)) and designated as small at approximately .01, medium at approximately .09, and large at > .25 (Witte & Witte, 2009).

For measures that showed statistically significant interactions of group and condition, we tested our hypothesis that speakers with HVDs would experience greater acoustic changes under cognitive stress by conducting post hoc one-tailed, independent-samples \( t \) tests. These \( t \) tests compared the difference in acoustic measures between conditions (i.e., incongruent – congruent) for each group.

To best account for the nonlinear relationship between \( f_o \) in Hz and the perception of pitch, the \( t \) test for \( f_o \) was conducted using values that had been converted to ST. ST were calculated using Equation 2 above, where \( f_2 \) was the \( f_o \) of a given sentence in Hz, and \( f_1 \) was the mean \( f_o \) in Hz of all sentences produced by the speaker in the congruent condition. These values were then averaged to generate a mean \( f_o \) (ST) for each speaker in each condition.

Significance for all statistical testing was set \( a \) priori at \( \alpha = .05 \). Statistical analyses were conducted in Minitab (Version 21, Minitab, Inc.).

Results

Cognitive stress had a statistically significant effect on all acoustic outcomes—RMS, \( f_o \), L/H ratio, CPP, and RFF. Figure 2 shows the change in RMS, \( f_o \), L/H ratio, and CPP under cognitive stress (i.e., incongruent – congruent) for speakers with and without HVDs. Figure 3 shows mean RFF values for each group and condition. Statistical results from all mixed effects models are presented in Table 1.

Amplitude

RMS increased under cognitive stress for speakers with and without HVDs. There was a significant main effect of cognitive load on RMS with a medium effect size and a small but significant interaction between group and condition. There was no statistically significant main effect of group on RMS. A post hoc one-tailed \( t \) test showed that the increase in RMS under cognitive stress for speakers with HVDs (1.10 dB) was not significantly
greater than for control participants (0.80 dB; $t = -1.45, p = .075$).

$f_o$

Cognitive stress was associated with a decrease in $f_o$ for speakers with and without HVDs. There was a significant main effect of cognitive load on $f_o$ with a medium effect size and a small but significant interaction between group and condition. There was no statistically significant main effect of group on $f_o$. A post hoc one-tailed $t$ test showed that the decrease in $f_o$ for speakers with HVDs ($-0.66$ ST) was not significantly greater than for control participants ($-0.42$ ST; $t = 1.37, p = .914$).

**Voice Quality**

Acoustic correlates of voice quality were affected by cognitive stress in speakers with and without HVDs, with L/H ratio decreasing and CPP increasing under cognitive stress. There was a significant main effect of cognitive load on L/H ratio and CPP with small and medium effect sizes, respectively. There was no significant effect of group nor interaction of group and condition for either measure.

**RFF**

RFF decreased under cognitive stress for speakers with and without HVDs. For RFF offset, there were small but significant main effects of group and condition. There was also a significant, large effect of cycle and a small but

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### Table 1. Results of mixed-effects analyses of variance for acoustic outcomes.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Effect</th>
<th>df</th>
<th>$F$</th>
<th>$p$</th>
<th>$\eta^2_p$</th>
<th>Effect size</th>
</tr>
</thead>
<tbody>
<tr>
<td>RMS (dB)</td>
<td>Group</td>
<td>1</td>
<td>1.65</td>
<td>.201</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>Condition</td>
<td>1</td>
<td>223.12</td>
<td>&lt; .001*</td>
<td>.136</td>
<td>Medium</td>
</tr>
<tr>
<td></td>
<td>Group $\times$ Condition</td>
<td>1</td>
<td>4.19</td>
<td>.041*</td>
<td>.003</td>
<td>Small</td>
</tr>
<tr>
<td>$f_o$ (Hz)</td>
<td>Group</td>
<td>1</td>
<td>0.04</td>
<td>.838</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>Condition</td>
<td>1</td>
<td>91.45</td>
<td>&lt; .001*</td>
<td>.061</td>
<td>Medium</td>
</tr>
<tr>
<td></td>
<td>Group $\times$ Condition</td>
<td>1</td>
<td>6.76</td>
<td>.009*</td>
<td>.005</td>
<td>Small</td>
</tr>
<tr>
<td>L/H ratio (dB)</td>
<td>Group</td>
<td>1</td>
<td>0.23</td>
<td>.636</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>Condition</td>
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<td>8.69</td>
<td>.003*</td>
<td>.006</td>
<td>Small</td>
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<td></td>
<td>Group $\times$ Condition</td>
<td>1</td>
<td>0.20</td>
<td>.652</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>CPP (dB)</td>
<td>Group</td>
<td>1</td>
<td>0.13</td>
<td>.717</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>Condition</td>
<td>1</td>
<td>200.40</td>
<td>&lt; .001*</td>
<td>.124</td>
<td>Medium</td>
</tr>
<tr>
<td></td>
<td>Group $\times$ Condition</td>
<td>1</td>
<td>3.69</td>
<td>.055</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>RFF offset (ST)</td>
<td>Group</td>
<td>1</td>
<td>5.15</td>
<td>.025*</td>
<td>.025</td>
<td>Small</td>
</tr>
<tr>
<td></td>
<td>Condition</td>
<td>1</td>
<td>10.63</td>
<td>.001*</td>
<td>.004</td>
<td>Small</td>
</tr>
<tr>
<td></td>
<td>Cycle</td>
<td>9</td>
<td>188.43</td>
<td>&lt; .001*</td>
<td>.407</td>
<td>Large</td>
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<tr>
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<td>Group $\times$ Condition</td>
<td>1</td>
<td>0.05</td>
<td>.822</td>
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<td>Group $\times$ Cycle</td>
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<td>2.13</td>
<td>.024*</td>
<td>.008</td>
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<td>Condition $\times$ Cycle</td>
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<td>0.90</td>
<td>.523</td>
<td>—</td>
<td>—</td>
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<td>Group $\times$ Condition $\times$ Cycle</td>
<td>9</td>
<td>0.26</td>
<td>.986</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>RFF onset (ST)</td>
<td>Group</td>
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<td>6.90</td>
<td>.010*</td>
<td>.028</td>
<td>Small</td>
</tr>
<tr>
<td></td>
<td>Condition</td>
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<td>5.54</td>
<td>.019*</td>
<td>.002</td>
<td>Small</td>
</tr>
<tr>
<td></td>
<td>Cycle</td>
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<td>646.46</td>
<td>&lt; .001*</td>
<td>.702</td>
<td>Large</td>
</tr>
<tr>
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<td>Group $\times$ Condition</td>
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<td>0.40</td>
<td>.526</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>Group $\times$ Cycle</td>
<td>9</td>
<td>3.67</td>
<td>&lt; .001*</td>
<td>.013</td>
<td>Small</td>
</tr>
<tr>
<td></td>
<td>Condition $\times$ Cycle</td>
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<td>0.47</td>
<td>.898</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>Group $\times$ Condition $\times$ Cycle</td>
<td>9</td>
<td>0.15</td>
<td>.998</td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>

*Note. RMS = root-mean-square; dB = decibels; $f_o$ = fundamental frequency; Hz = hertz; L/H ratio = low-to-high spectral energy ratio; CPP = cepstral peak prominence; RFF = relative fundamental frequency; ST = semitones; — = not applicable for nonsignificant findings.

$^*$Significant at $p < .05$. 

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significant interaction between group and cycle on RFF offset. Results were similar for RFF onset, for which there were small but significant effects of group, condition, and Group × Cycle interaction, as well as a significant, large effect of cycle. There was no significant interaction between group and condition or between group, condition, and cycle for RFF offset nor onset, indicating that cognitive stress did not affect this measure differently in speakers with HVDs than speakers without.

Discussion

The purpose of this study was to determine if increased cognitive load—a stressor that arouses the autonomic nervous system (Bear et al., 2007)—affects voice in female speakers with HVDs differently than those without HVDs. Although cognitive stress was indeed associated with significant changes in RMS, $f_o$, L/H ratio, CPP, and RFF for both groups, none of these changes were greater in speakers with HVDs than those without.

Comparison to Prior Work

The specific effects of cognitive stress on voice acoustics seen in this study are consistent with some previous work with younger adults with typical voices, yet not with others. This is as expected for those acoustic measures—namely, $f_o$ and RMS—for which variable findings have been reported. That is, some studies have also shown that $f_o$ decreases under cognitive stress (Dietrich, 2008; Streeter et al., 1983; van Lierde et al., 2009), but many others have found $f_o$ to increase (Boyer et al., 2018; Mendoza & Carballo, 1998; Perrine & Scherer, 2020; Ruiz et al., 1996; Scherer et al., 2002; van Mersbergen & Payne, 2020). Similarly, both a decrease (Dietrich, 2008; van Lierde et al., 2009) and an increase (Lively et al., 1993) in measures of vocal amplitude have been reported. Thus, though our hypotheses for $f_o$ and RMS proved true in our sample, the directionality of these hypotheses were selected somewhat arbitrarily. Alternative hypotheses predicting a change in the opposite direction would have been equally supported by the literature. Our findings do not resolve the contradictions in prior research. Rather, the persistence of these variable findings suggests other methodological and individual factors, beyond just cognitive stress, may influence these measures.

Cognitive stress may have a more specific and predictable effect on voice quality than on $f_o$ and amplitude. As reported here, others have found L/H ratio to decrease (MacPherson et al., 2017) and CPP to increase (MacPherson et al., 2017; van Mersbergen & Payne, 2020) under cognitive stress for younger adults with typical voices. Together, these two changes suggest that speakers use a more pressed voice when speaking under stress. This study extends these findings to speakers with HVDs. Even though these speakers may have had deviations in voice quality at baseline, cognitive stress had a similar effect on their voices. The relatively mild severity of dysphonia among participants with HVDs in this sample may have influenced these results. Changes in voice quality under cognitive stress may differ for speakers with more severe dysphonia, particularly if that dysphonia is marked by excessive strain.

Autonomic Dysfunction and Hyperfunctional Voice Disorders

This study was motivated by evidence of possible autonomic dysfunction among individuals with HVDs. Specifically, people with HVDs report experiencing more symptoms of autonomic arousal more frequently than those without voice complaints (Demmink-Geertman & Dejonckere, 2002; Park & Behlau, 2011). This excess of symptoms may reflect a hyperactive autonomic nervous system in this population. We, therefore, expected a cognitive stressor to elicit greater autonomic arousal and, thus, greater changes in voice among those with HVDs. That was not the case. This finding can be interpreted in a few different ways.

First, the evidence for autonomic dysfunction among those with HVDs consists primarily of symptom self-reports. Though differences in symptom occurrence have been found in robust samples (Demmink-Geertman & Dejonckere, 2002; Park & Behlau, 2011), autonomic dysfunction among those with HVDs has not been confirmed through more objective means. It, therefore, remains possible that no such dysfunction distinguishes those with HVDs from those without. Rather, autonomic arousal may play a moderating, but not directly etiologic, role in the development and persistence of HVDs (Desjardins et al., 2022). Discerning this role is complicated by the overlap between autonomic and psychosocial theories of HVDs, both of which involve stress responses—physiological and emotional responses, respectively—that can be difficult to objectively differentiate (Demmink-Geertman & Dejonckere, 2002; Desjardins et al., 2022). Furthermore, though evidence of autonomic dysfunction was present in a sample of individuals with both

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3Abur, MacPherson, et al. (2021), however, found LHR and CPP to remain stable across cognitive load conditions in a small sample of adults aged 68 years and older, which could suggest that the effects of cognitive stress interact with those of typical aging. Speech kinematic findings also show that cognitive stress affects older adults differently than younger adults (MacPherson, 2019). Given the ages of participants in the present study (≤ 66 years), younger adults provide a clearer comparison.
phonotraumatic and nonphonotraumatic types (Park & Behlau, 2011), a future study is warranted to systematically determine the prevalence of such dysfunction separately by subtype.

Second, autonomic dysfunction may indeed be more prevalent among those with HVDs but characterized by a difference in the time course of arousal rather than its magnitude. That is, the autonomic nervous system may be aroused by stress to an equal degree in those with and without HVDs, but this arousal may last longer in the former. If so, instead of greater changes in voice acoustics under cognitive stress among those with HVDs, we would expect such changes to outlast those observed in speakers without HVDs. Neither this study nor existing evidence of autonomic dysfunction among those with HVDs can distinguish between these possibilities. Measuring physiological responses before, during, and after a stressor is introduced—has been done in speakers with typical voices (Helou et al., 2013)—could reveal the specific ways in which such dysfunction presents. Physiological measures could help characterize both magnitude and duration of autonomic arousal under stress and allow for more specific hypotheses regarding the effects of cognitive stress on the voice.

Finally, the stressor of this study may not have captured the specific autonomic differences between those with and without HVDs. Both level and type of stress affect the degree of autonomic arousal (Wallbott & Scherer, 1991). So, a task that imposes a lower level of stress may arouse the autonomic nervous system of speakers with HVDs but not those with typical voices. This would suggest that a hypersensitivity to mild stressors is what differentiates autonomic function of those with HVDs. Alternatively, differences may emerge in responses to a more stressful condition or to a different type of stressor (e.g., emotional). Measuring speakers’ physiological and voice responses under various types and levels of stress could determine if any of these possibilities is likely. Future study of the specific effects of different stress types may also shed light on the variability in RMS and $f_0$ outcomes seen across studies.

**Clinical Implications**

A clearer understanding of the relationship between autonomic dysfunction and HVDs must be reached before this research will have a substantial impact on clinical practice. Discerning whether such dysfunction plays an etiological or mediating role in the development or persistence of HVDs could inform targeted interventions and preventative care for those at risk of these disorders. Despite the clear need for future work, the present findings do shed some light on the utility of cognitive stressors in voice therapy. Voice clinicians often incorporate challenging conversational tasks into therapy to facilitate the transfer of therapeutic gains to daily communication (Gartner-Schmidt et al., 2013; Iwarsson et al., 2017). A cognitive stressor may indeed challenge a speaker to maintain efficient voicing even under autonomic arousal, when laryngeal muscle tension may be increasing (Dahl & Stepp, 2021; Helou et al., 2018, 2013). Whether this is the most appropriate stressor for people with HVDs, however, cannot be determined without a fuller understanding of autonomic function in this population.

**Limitations**

Certain limitations of this study should be considered when interpreting its findings. First, autonomic arousal was not confirmed with physiological measures. However, others have used such measures to establish a robust relationship between cognitive stress and autonomic arousal (Abur, MacPherson, et al., 2021; Boyer et al., 2018; Heaton et al., 2020; Kleinow & Smith, 2006; MacPherson et al., 2017). This prior research includes two studies with a sentence-level Stroop task nearly identical to the one used here (Abur, MacPherson, et al., 2021; MacPherson et al., 2017). We are, thus, reasonably confident that the cognitive stressor had the intended effect of arousing the autonomic nervous system. Second, all participants were female. Though there is a relatively high prevalence of HVDs among female speakers (Roy et al., 2005), there is also evidence of sex differences in autonomic responses to stressors (Wallbott & Scherer, 1991). So these findings may not extend to speakers of other sexes.

**Conclusions**

Cognitive stress and presumed autonomic arousal affect voice similarly in female speakers with and without HVDs. Both groups experienced decreases in $f_0$, L/H ratio, and RFF and increases in RMS and CPP when speaking during a cognitively demanding task. This unexpected similarity between groups contradicted the expectation that underlying autonomic dysfunction among those with HVDs (Demmink-Geertman & Dejonckere, 2002; Park & Behlau, 2011) would be associated with greater changes in voice acoustics. More objective assessments of autonomic function in this population are needed to confirm the prevalence of any dysfunction and, if confirmed, to better characterize the nature of that dysfunction.

**Data Availability Statement**

The data sets generated during and/or analyzed during this study are not publicly available due to

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commitments to protect participant confidentiality but are available from the corresponding author on reasonable request.

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