Purpose: The purpose of this study was to investigate the relation of respiratory forced oscillation to the acoustic characteristics of vocal tremor.

Method: Acoustical analyses were performed to determine the characteristics of the intensity and fundamental frequency (F₀) for speech samples obtained by Farinella, Hixon, Hoit, Story, and Jones (2006) using a respiratory forced oscillation paradigm with 5 healthy adult males to simulate vocal tremor involving respiratory pressure modulation. The analyzed conditions were sustained productions of /a/ with amplitudes of applied pressure of 0, 1, 2, and 4 cmH₂O and at a rate of 5 Hz.

Results: Forced oscillation of the respiratory system produced modulation of the intensity and F₀ for all participants. Variability was observed between participants and conditions in the change in intensity and F₀ per unit of pressure change, as well as in the mean intensity and F₀. However, the extent of modulation of intensity and F₀ generally increased as the applied pressure increased, as would be expected.

Conclusion: These findings suggest that individuals develop idiosyncratic adaptations to pressure modulations, which are important to understanding aspects of variability in vocal tremor, and highlight the need to assess all components of the speech mechanism that may be directly or indirectly affected by tremor.

Key Words: voice, tremor, respiratory

Vocal tremor is a voice disorder characterized by the perception of voice instability or shakiness (Brown & Simonson, 1963; Hachinski, Thomsen, & Buch, 1975). This perception is associated with atypical modulation of the intensity (i.e., the acoustic correlate of the perception of loudness) and frequency (i.e., the acoustic correlate of the perception of pitch) of the acoustic signal generated during voice production (Brown & Simonson, 1963; Ludlow, Bassoich, Connor, & Coulter, 1986). The acoustic modulations are a result of abnormal physiologic oscillations within the speech mechanism, which are caused by neurological disorders that produce involuntary, periodic contraction of antagonistic muscles that are activated during speech production. Muscles within the respiratory system, the larynx, and/or the vocal tract may be affected by such neurological disorders (Brown & Simonson, 1963; Hachinski et al., 1975; Lou & Jankovic, 1991).

Previous research has focused primarily on vocal tremor with a laryngeal source (Brown & Simonson, 1963; Finnegan, Luschei, Barkmeier, & Hoffman, 2003; Gamboa et al., 1998; Koda & Ludlow, 1992; Ludlow et al., 1986; Warrick, Dromey, Irish, & Durkin, 2000). However, tremor has been found to affect the respiratory musculature and to produce perceivable modulation of the voice (Hachinski et al., 1975; Tomoda, Shibaski, Kuroda, & Shin, 1987). Based on the role of the respiratory system in speech production, tremor affecting this component of the speech mechanism is expected to impose specific effects on the acoustic output. That is, periodic contractions of the muscles of the chest wall (i.e., rib cage wall, diaphragm, abdominal wall) would be expected to result in modulation of the pressure supplied to the vocal system. These pressure modulations would, in turn, modulate the amplitude of the glottal flow (i.e., the sound source for vowels). This would ultimately be transferred to the sound pressure radiated at the lips and be realized as modulations of the intensity of the acoustic output.

The fundamental frequency (F₀) of vocal fold vibration would also be modulated by pressure modulation (Hixon, Klatt, & Mead, 1971; Lieberman, Knudson, & Mead, 1969; Rothenberg & Mahshie, 1986). This is due to the amplitude-dependent tension in the vocal folds, where greater vibratory amplitude results in increased vocal fold tension (Titze, 1989). Additionally, because the harmonics in the vocal output are multiples of the F₀, all of the harmonics would increase and decrease with the F₀. The harmonics would, therefore, shift in and out of the resonances of the vocal tract, causing their amplitudes to cyclically increase and decrease. This resonance-harmonic interaction would result in additional modulation of the output pressure and, thus,
contribute to the overall modulation in vocal intensity (Horii, 1989).

In summary, vocal tremor with a respiratory source could be characterized by both intensity and frequency modulation. Intensity modulation could be a direct result of the changes in pressure supplied to the respiratory system, and frequency modulation could be induced by the change in vocal fold tension associated with the pressure changes. These predicted acoustic characteristics of respiratory-induced vocal tremor have not been thoroughly studied in humans.

Because little is known about respiratory-induced vocal tremor, current clinical practice does not consistently include assessment of the contribution of the respiratory system to vocal tremor. However, it is essential to identify when clients with vocal tremor have respiratory involvement because this could significantly affect the treatment plan. Furthermore, understanding the characteristics of vocal tremor with a respiratory source may aid in determining the prevalence of this source of vocal tremor, which is currently unknown.

As a first step toward understanding the nature of respiratory-induced vocal tremor, Farinella, Hixon, Hoit, Story, and Jones (2006) conducted a study of the perceptual characteristics of vocal tremor with a respiratory source. These authors used a respiratory forced oscillation paradigm with five healthy adult male speakers to simulate respiratory-induced vocal tremor. The purpose of the study was to determine the detection threshold of vocal tremor with different amplitudes and rates, as well as the contexts in which respiratory-induced tremor was most easily perceived. The results indicated that, as the amplitude of the applied pressure increased, so did the magnitude of perceived vocal tremor. Furthermore, soft utterances typically had a greater magnitude of perceived vocal tremor than loud utterances, and breathy utterances typically had a greater magnitude of perceived vocal tremor than pressed productions. However, there was variability in the perceived magnitude across the five speakers. These differences were attributed to differences in the speaker’s voice characteristics, including loudness, pitch, and quality, and to possible differences in the extent to which the pressure applied to the surface of the respiratory system was reflected in tracheal pressure change for each speaker.

The current study was designed to investigate the acoustic characteristics of respiratory-induced vocal tremor for the speakers who had been studied by Farinella et al. (2006). It was expected that these findings would contribute to the understanding of the acoustic characteristics of respiratory-induced vocal tremor that influence the perception of vocal tremor. It was also expected that an understanding of the acoustic characteristics of vocal tremor with a respiratory source would aid in the clinical assessment of tremor affecting the respiratory system.

Method

Participants

Farinella et al. (2006) used a respiratory forced oscillation paradigm with five males ages 50 to 65 years. This age range was selected to include the typical age of onset of respiratory tremor resulting from neurological disease. The participants were reportedly in good health and denied a history of laryngeal, respiratory, cardiovascular, and/or neuromotor disease. All participants were native English speakers with normal speech, language, and hearing. Information about the participants’ race, ethnicity, and socioeconomic status was not available.

Procedure

The participants were seated in a full-body plethysmograph with four 12-in loudspeakers attached (see Figure 1 in Farinella et al., 2006, and also Hixon, 1972). The loudspeakers were controlled by an oscillator and a power amplifier, which created pressure changes within the plethysmograph that modulated the surface of the respiratory system and, thus, the pressure supplied to the vocal system. The larynx and vocal tract were not directly modulated by the applied pressure changes, as each participant’s head and neck remained outside of the chamber of the plethysmograph. For a more detailed description of this configuration, see Hixon (1972).

The participants were asked to produce the vowels /a, i, u, æ, ð/, and the sentence “People talk back and forth by using acoustic signals.” The participants produced the vowel /a/ with three different vocal qualities (i.e., breathy, normal, and pressed), three different levels of loudness (i.e., soft, normal, and loud), and three different pitches (i.e., habitual pitch, a pitch ½ of an octave higher than the habitual pitch, and a pitch ¾ of an octave higher than the habitual pitch). The amplitude of the applied pressure was changed from 4 cmH₂O to 0 cmH₂O in 0.5 cmH₂O decrements, and the rate was maintained at 5 Hz. In the subsequent condition, the rate of applied pressure was changed to 5, 6, 8, 10, and 12 Hz, and the amplitude of applied pressure was maintained at 4 cmH₂O. The participants’ utterances were recorded using a head-mounted unidirectional microphone and were routed to an oscilloscope and instrumentation recorder.

Analysis

The recordings collected by Farinella et al. (2006) were converted into two-channel digital files that contained the microphone signal and the low-bandwidth pressure signal from within the plethysmograph using the Computerized Speech Lab Model 4400 (KayPentax, Montvale, NJ). An example is shown in Figure 1, where the upper panel is the microphone signal and the lower panel is the corresponding plethysmograph pressure. The sampling rate was set to 44100 Hz and, to avoid clipping of the waveform, the gain was set individually for 300-s segments of the acoustic signal. The conditions that were chosen for analysis were the second trial of a sustained production of /a/ with amplitudes of applied pressure of 0, 1, 2, and 4 cmH₂O and a rate of 5 Hz for each of the five participants. The rate of 5 Hz was selected because this is within the range of modulation rates exhibited by individuals with vocal tremor (Brown & Simonson, 1963). Sustained vowels were selected over
sentences because these samples allowed for measurement of intensity and F0 across a greater number of cycles of modulation. In addition, Farinella et al. (2006) found that modulation of the voice was more easily perceived by listeners for sustained vowel productions, which suggested greater modulation within these signals.

Using custom written MATLAB functions, 1.5-s samples were taken to exclude the onset and offset of each of the vowel productions for the four pressure conditions. Measures of the intensity and F0 for each of these segments were obtained. The change in intensity and change in F0 related to the change in the amplitude of the applied pressure were calculated. In addition, the mean intensity, mean F0, extent of overall intensity modulation, and extent of F0 modulation were determined.

Results

Acoustical analyses are shown in Figure 2 for the four conditions and the five participants. Each row corresponds to one participant, with the plot on the left representing the intensity contours for each pressure oscillation condition over the time course of the vowel, and the plot on the right representing the corresponding F0 contours. Because the microphone signal had not been calibrated during the original data collection, the intensity contours are plotted as deviations from the mean intensity for each individual vowel production. For all five participants, the intensity and the F0 modulated cyclically across time at a rate of 5 Hz during the 1, 2, and 4 cmH2O conditions.

The change in intensity and change in F0 did not vary systematically with the amplitude of the applied pressure. The variability between participants and conditions can be seen in Tables 1 and 2, respectively. The range of change in intensity per unit of pressure was 0.2–1.5 dB, and the range in change of frequency per unit of pressure was 0.8–2.1 Hz.

Although the participants were instructed to maintain a constant loudness and pitch during all pressure conditions to attempt to eliminate the potential effect of these characteristics on the perceived magnitude of vocal tremor, there was variability in the participants’ mean intensities and mean F0 across conditions. This can be seen in Figures 3 and 4, respectively. The difference in mean intensity across conditions ranged from 1.7 dB to 17.5 dB across the participants, and the difference in mean F0 across conditions ranged from 5 Hz to 10 Hz across the participants. A systematic change was not observed in the mean intensity or mean F0 as the amplitude of the applied pressure increased.

Because of these differences in mean intensity and F0 and their potential impact on the change in intensity and F0, the extent of intensity modulation and the extent of F0 modulation were determined. The extent of F0 modulation was calculated by dividing the range of modulation (i.e., the difference between the maximum and minimum F0) by the sum of the maximum and minimum F0, and then multiplying this number by 100 to obtain a percentage. The same method was used to calculate the extent of intensity modulation, except the intensity in decibels was converted to Pascals before making the calculations. It is noted that in each case there is a nonzero value for the 0 cmH2O condition. Although this may seem counterintuitive, there is normally some variability over the time course of a sustained vowel production. These calculated values of extent of intensity and F0 are plotted for each participant in Figures 5 and 6, respectively.

In most cases, the extent of both intensity and F0 modulation increased as the amplitude of the applied pressure increased, although the amount of increase was speaker dependent. For the extent of intensity modulation, there were two instances that did not follow the pattern of increasing extent with increasing amplitude of the applied pressure. Participant 1 had a decrease in the extent of intensity modulation from the 0 cmH2O condition to the 1 cmH2O condition, and Participant 3 had no change in the extent of intensity modulation from the 1 cmH2O condition to the 2 cmH2O condition. An exception to the pattern for F0 was Participant 5, who had no change in the extent of F0 modulation from the 0 cmH2O condition to the 1 cmH2O condition.

Discussion

The results of this study indicate that respiratory forced oscillation produces modulation of the intensity and F0 of the acoustic signal that is generated during voice production. The amount of change in intensity or F0 was not consistent in relation to the absolute pressure change. This finding may be due to the variability in the mean intensity and mean F0 across participants and across conditions. Nevertheless, the extent of modulation of the intensity and F0 as expressed in normalized form (i.e., percentage of the range of intensity or F0) did reveal a predictable pattern.
Figure 2. Intensity modulation and fundamental frequency (F0) modulation across 1.5 s for each of the five participants. Each row represents the intensity contour on the left and the F0 contour on the right for one participant. The intensity and F0 contours are provided for the four pressure conditions (i.e., 0, 1, 2, 4 cmH2O).
That is, the modulation extent generally increased as the amplitude of the applied pressure increased, although the range of modulation extents differed among participants. These findings have implications for clinical practice and future research on vocal tremor.

Before discussing the implications of these results, it is important to note one methodological consideration. Known pressure changes were applied to the surface of the chest wall by Farinella et al. (2006), which produced modulations of the tracheal pressure that caused modulation of the intensity and F0 of the voice. The absolute amplitudes of the tracheal pressure modulations were unknown because direct measurement of the tracheal pressure would have required use of an invasive procedure such as tracheal puncture, which was not included in the original protocol. The modulation of tracheal pressure was almost certainly lower in amplitude.

Table 1. Change in intensity per unit of pressure for each of the three oscillated pressure conditions for the five participants (P1–P5).

<table>
<thead>
<tr>
<th></th>
<th>1 cmH2O</th>
<th>2 cmH2O</th>
<th>4 cmH2O</th>
</tr>
</thead>
<tbody>
<tr>
<td>P1</td>
<td>0.21</td>
<td>0.41</td>
<td>0.31</td>
</tr>
<tr>
<td>P2</td>
<td>1.52</td>
<td>1.28</td>
<td>1.08</td>
</tr>
<tr>
<td>P3</td>
<td>0.58</td>
<td>0.29</td>
<td>0.45</td>
</tr>
<tr>
<td>P4</td>
<td>1.45</td>
<td>0.94</td>
<td>0.94</td>
</tr>
<tr>
<td>P5</td>
<td>0.26</td>
<td>0.42</td>
<td>0.29</td>
</tr>
</tbody>
</table>

Table 2. Change in fundamental frequency (F0) per unit of pressure for each of the three oscillated pressure conditions for the five participants.

<table>
<thead>
<tr>
<th></th>
<th>1 cmH2O</th>
<th>2 cmH2O</th>
<th>4 cmH2O</th>
</tr>
</thead>
<tbody>
<tr>
<td>P1</td>
<td>2.11</td>
<td>1.88</td>
<td>1.55</td>
</tr>
<tr>
<td>P2</td>
<td>1.72</td>
<td>1.3</td>
<td>1.15</td>
</tr>
<tr>
<td>P3</td>
<td>1.00</td>
<td>0.75</td>
<td>0.86</td>
</tr>
<tr>
<td>P4</td>
<td>1.76</td>
<td>2.12</td>
<td>1.95</td>
</tr>
<tr>
<td>P5</td>
<td>1.11</td>
<td>0.79</td>
<td>1.02</td>
</tr>
</tbody>
</table>
than the modulation of pressure at the body surface due to pressure loss across the chest wall, and probably differed across participants (Hixon, 1972; Hixon et al., 1971). In addition, the amplitude of tracheal pressure modulation may have varied across participants because of differences in their average tracheal pressures. For example, an applied pressure of 4 cmH\textsubscript{2}O might affect the tracheal pressure modulation of a participant with an average tracheal pressure of 5 cmH\textsubscript{2}O to a greater extent than one with an average tracheal pressure of 10 cmH\textsubscript{2}O.

Previous research has demonstrated that direct changes in tracheal pressure produce small changes in intensity in the range of 0.5–3.0 dB per cmH\textsubscript{2}O (Hixon & Minifie, 1972). The range of change in intensity in this study was 0.2–1.5 dB per cmH\textsubscript{2}O. These smaller changes in intensity compared with previous studies may have been due to differences in the extent to which the applied pressure affected the tracheal pressure. Further, intensity range may have been influenced by participants’ average intensities. Hixon and Minifie (1972) reported that there was 0.5 dB per cmH\textsubscript{2}O during production of a soft voice and 3.0 dB per cmH\textsubscript{2}O for production of a loud voice. Therefore, participants’ starting mean intensities may have affected the amount of change in intensity per unit of pressure change.

Previous research has also demonstrated that direct changes in tracheal pressure produce small changes in F\textsubscript{0} in the range of 2–4 Hz per cmH\textsubscript{2}O (Hixon et al., 1971). For the current study, the range of F\textsubscript{0} change was 0.8–2.1 Hz per cmH\textsubscript{2}O. These smaller changes in F\textsubscript{0} compared with previous studies may have, again, been due to differences in the extent to which the applied pressure affected the tracheal pressure. These results also may have been affected by the speakers’ starting mean F\textsubscript{0}. Previous studies have indicated that the effect of pressure change supplied to the larynx on F\textsubscript{0} is most prominent at low F\textsubscript{0} (Titze, 1989).

These findings support the hypothesis of Farinella et al. (2006) that the effect of speaker may have been related to the extent to which the pressure that was applied to the surface of the respiratory system was reflected in tracheal pressure change for each speaker, as well as to the differences in the speakers’ voice characteristics (i.e., mean intensity and F\textsubscript{0}). The variability that was reported by Farinella et al. in the perceptual findings and that was observed in the acoustic measures between participants and between conditions may have also been due to the participants’ individual physiological adaptations to respiratory oscillations. It is possible that, because the applied pressure decreased systematically across conditions from 4 cmH\textsubscript{2}O to 0 cmH\textsubscript{2}O in 0.5 cmH\textsubscript{2}O decrements, the participants may have developed physiological strategies in response to the perturbations to minimize the effect on the acoustic output. These strategies may have been adjusted as the applied pressure decreased. For example, Participant 2 and Participant 3 may have either increased the degree of adduction of the vocal folds or increased the tension of the vocal folds in an attempt to control intensity modulation, which would have increased their mean F\textsubscript{0}. However, none of the participants was completely able to compensate for the applied pressure change. This is consistent with previously reported evidence that individuals cannot compensate for respiratory forced oscillation, even when the oscillations are applied at lower rates than the 5 Hz rate that was used in the present study (Hixon et al., 1971).

**Clinical Implications**

Because this was a study of simulated respiratory-induced vocal tremor using forced oscillation of the respiratory system, the measured acoustic characteristics may differ from those obtained in clinical populations. For example, the regularity in rate and extent of modulation in this study is not likely to be seen in individuals with vocal tremor related to neurological disorders (Brown & Simonsen, 1963; Ludlow et al., 1986). In addition, the physiologic adaptations that individuals develop in response to neurologically produced oscillation within the speech mechanism are likely to differ from those produced in response to forced oscillation of a normal respiratory system.

Despite these limitations, the results of this study indicate the importance of assessing the contribution of each component of the speech mechanism whether it is directly or indirectly affected by tremor. During current clinical assessment of vocal tremor, evaluation may focus on laryngeal involvement using endoscopic imaging and acoustical analyses. When modulation of the F\textsubscript{0} is detected in the acoustic signal, it is often assumed that this indicates that the larynx is directly affected by tremor, even when oscillation within the larynx is not visible during endoscopic imaging. As a result, intervention for vocal tremor is focused on reducing laryngeal involvement, generally through injection of botulinum toxin (i.e., Botox) into the laryngeal musculature.

However, the results of this study demonstrate that modulation of the F\textsubscript{0} may be induced by oscillation of the respiratory structures alone. For clients with isolated respiratory-induced vocal tremor, laryngeal Botox injections would be expected to have little or no beneficial effect on the voice. In fact, this intervention might be detrimental to the vocal quality because increasing vocal fold tension could help control intensity modulation. By using the acoustical analyses described earlier in conjunction with airflow measurements made at the airway opening, as described by Hixon and Hoit (2006), clinicians may effectively assess the contribution of the respiratory system to vocal tremor and make appropriate therapeutic recommendations. In addition to using these techniques, assessment of vocal tremor should investigate the individualized adaptations that clients may have developed in response to the physiologic oscillations associated with tremor to determine if these are beneficial in reducing the magnitude of modulation of the voice. Based on the results of the Farinella et al. (2006) study, some adaptations were more effective than others in reducing the magnitude of the perceived tremor.

**Future Directions**

Although the findings of this study demonstrate that simulated respiratory-induced vocal tremor is characterized
by modulation of speakers’ intensity and $F_0$, the acoustic characteristics of isolated respiratory-induced vocal tremor in clinical populations are unknown. This knowledge is essential for evaluating individuals with this voice disorder and providing appropriate treatment. However, the results of this study indicate that respiratory-induced vocal tremor cannot be studied in complete isolation in humans because of the interaction of the respiratory system and larynx.

The participants in this study developed idiosyncratic adaptations to pressure modulations that affected the characteristics of the acoustic output and could not be completely controlled. Therefore, future research requires a method of isolating tremor to the respiratory system and controlling for potential adaptations at the level of the larynx or vocal tract. This can be accomplished using a computational modeling approach (cf. Samlan & Story, 2011) that allows for control of the respiratory pressure, $F_0$, degree of vocal fold adduction, and configuration of the vocal tract. Experiments are planned that will isolate modulation of each of these components. Speech output will be produced and the associated acoustical and perceptual characteristics of vocal tremor will be investigated to better understand the nature of vocal tremor and the contribution of each component of the speech mechanism to this disorder. In addition, this model will be used to determine the effects of adjustment of the mean intensity and/or $F_0$ on the magnitude of the perceived tremor, as these modifications could potentially be useful in behavioral management of vocal tremor.

**Acknowledgments**

This research was funded by the Thomas J. Hixon Doctoral Fellowship. We would like to thank Kimberly Farinella for sharing these data and the associated laboratory documentation. We would also like to thank Jeannette Hoit for her contributions to the interpretation of these results in light of results from previous unpublished studies within the Speech Research Laboratory at the University of Arizona.

**References**


Acoustic Characteristics of Simulated Respiratory-Induced Vocal Tremor

Rosemary A. Lester, and Brad H. Story

*Am J Speech Lang Pathol* 2013;22;205-211; originally published online Nov 26, 2012;
DOI: 10.1044/1058-0360(2012/12-0043)

The references for this article include 3 HighWire-hosted articles which you can access for free at: http://ajslp.asha.org/cgi/content/full/22/2/205#BIBL

This information is current as of December 12, 2013

This article, along with updated information and services, is located on the World Wide Web at:
http://ajslp.asha.org/cgi/content/full/22/2/205