The Prevalence of Creak Across Breath Groups in Adductor Laryngeal Dystonia

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Abstract: Objective. Creak is an acoustic feature found to discriminate speakers with adductor laryngeal dystonia (AdLD) from typical speakers with outstanding diagnostic accuracy. Yet creak is also used by typical speakers as a phrase-boundary marker. This study aims to compare the prevalence of creak across estimated breath groups in speakers with AdLD and controls to delineate physiological mechanisms underlying creak in AdLD.

Methods. Thirty-four speakers read aloud the first paragraph of the Rainbow Passage (17 diagnosed with AdLD and 17 with no history of voice complaints). "Breath-like" pauses were defined as any in which technicians audibly heard a pause and all pauses > 500 ms. For each phoneme, the time preceding the next breath-like pause was calculated, and the probability of creak occurrence was calculated. A generalized linear mixed-effects model was performed to determine the relationship between creak and time preceding a breath-like pause.

Results. Inter-rater and intrarater reliability of technicians were excellent. There was a statistically greater probability of creak in the AdLD group compared to controls (22% vs. 5%) and a statistically greater probability of creak as speakers approached a breath-like pause in both groups. The interaction between the time preceding a breath-like pause and group was significant, with a stronger relationship between the time preceding a breath-like pause and creak for control speakers ($P < 0.001$).

Conclusions. Creak is more prevalent in speakers with AdLD and may not only be related to respiratory phrasing but possibly in response to or because of the hyperadduction of the vocal folds during a laryngeal spasm.

Lay Summary. The probability of creak occurring was greater towards the end of estimated breath groups in speakers with and without AdLD; however, for speakers with AdLD, creak was more prominent across the entire breath group.

Level of Evidence. 3

Key Words: Laryngeal dystonia–Voice disorders–Acoustics–Speech-language pathology–Creak.

INTRODUCTION

Laryngeal dystonia (LD) is a rare neurological focal dystonia that causes spasms of the laryngeal muscles during speech. Its prevalence is estimated to be 1 in every 100 000 people.[1] The most common subtype is adductor laryngeal dystonia (AdLD), in which the spasms occur in the adductor laryngeal muscles, typically during voiced phonemes.[2] LD is task-specific; in AdLD, spasms occur during speech but are less likely to occur during whispering or innate vocal tasks, such as crying or laughing.[3] The laryngeal spasms disrupt the speech signal, resulting in what listeners perceive as a strained or strangled voice quality.[4–6] Some individuals with AdLD report that people often assume they are sick or have been crying.[7] Many report having greater difficulty when speaking over the phone or during stressful events.[7] There are far-reaching negative consequences of AdLD on a person's quality of life, including social, emotional, and even financial constraints.[7–10]

AdLD is difficult to diagnose, in part because it can present similarly to a common behavioral voice disorder, muscle tension dysphonia (MTD), and there are no diagnostic measures that are specific to the primary signs of AdLD. Consequently, patients require, on average, a reported four office visits over five-to-six years to receive an accurate diagnosis.[11,12] Clinical assessment typically involves laryngeal imaging and acoustic and aerodynamic recordings, often with the use of specialized stimuli, such as sentences loaded with voiced or voiceless phonemes.[3] Based on simultaneous laryngeal electromyography and acoustic recordings, spasm durations are often less than 20 ms.[13] Laryngeal imaging can include endoscopy with or without stroboscopy, but subtle or fine-grained movements are

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often not appreciable due to the frame-rate limitations of clinical systems, which are confined to 30 frames per second (33 ms between each frame). Acoustic and aerodynamic measures are used to quantify symptoms of dysphonia, but these measures are used to describe a variety of voice disorders and are not specific or sensitive to the primary signs of AdLD without the use of specific stimuli. Therefore, auditory-perceptual evaluation of voice remains the gold standard for a differential diagnosis despite poor clinician reliability.[14]

Several acoustic measures have been investigated as potential discriminative measures for AdLD and MTD. Sapienza et al found that manual labels of phonatory breaks, frequency shifts, and aperiodicity differentiated the two disorders.[15] However, manual labeling is extremely time-intensive, so practically infeasible in clinical practice. Roy and Mazin[16] investigated the cepstral spectral index of dysphonia (CSID) in speakers with AdLD and speakers with MTD. The CSID is thought to be an objective estimate of overall severity of dysphonia that is calculated via a multiple regression model that incorporates both spectral and cepstral measures.[16] The difference in CSID between connected speech and vowels differentiated speakers with AdLD from speakers with MTD with acceptable discrimination,[16] comparable to that of auditory-perceptual judgments in their previous work.[17] Houtz et al[18] investigated the discrimination of AdLD and MTD using the long-term average spectrum (LTAS), which is the average amplitude across a selected frequency range. It is a measure that is also thought to reflect the severity of voice quality. The LTAS also demonstrated acceptable discrimination between AdLD and MTD, as measured on an all-voiced phoneme stimulus.[18]

An additional acoustic measure, spectral aggregate of the fundamental frequency contour (SAHf_o) has been investigated as a measure specific to the primary signs of AdLD. SAHf_o is an automatic acoustic measure that was designed to detect fast transitions in f_o that co-occur with laryngeal spasms.[19] In a validation study,[20] SAHf_o was compared to manual labels of LD discontinuities for a voiced phoneme-loaded sentence and a voiceless phoneme-loaded sentence. Manual labels of LD included phonatory breaks and frequency shifts,[21] as well as creak, following descriptions of Keating et al,[22] in which creak was used as an umbrella term for irregularities in f_o, including low and irregular f_o, multiply pulsed voice, aperiodic voice, or tense/pressed voice.[20] A statistically significant relationship was found between SAHf_o and LD discontinuity labels; however, the results were dependent on phonemic context. An additional finding was that creak was the most commonly occurring type of discontinuity in speakers with AdLD, regardless of sentence type.[20] Thus, although SAHf_o, the CSID, and the LTAS are promising quantitative measures that may aid a differential diagnosis, the problem remains that the specialized stimuli that these measures rely on are not consistently recorded in clinical contexts. Rather, they are only used when LD is already suspected.

The prevalence of manually labeled creak in prior work[20] motivated the investigation of creak as estimated automatically,[23] using a unique dataset of speakers with AdLD reading aloud the first paragraph of the Rainbow Passage.[24,25] An open-source, automated creak detector[23] was implemented in MATLAB to calculate % creak of the first paragraph of the rainbow passage. Receiver-operating curve analyses were used to determine diagnostic accuracy, plotted as sensitivity over 1–specificity. Area under the curve (AUC) measures were calculated as measures of diagnostic accuracy between AdLD and controls, as well as between AdLD and MTD. Automated estimates of creak differentiated speakers with AdLD from control speakers with outstanding diagnostic accuracy (AUC = 0.94), and more importantly, creak differentiated speakers with AdLD from speakers with MTD with excellent diagnostic accuracy (AUC = 0.86).[25] The study provided preliminary evidence that automated estimates of creak could be used as a screening tool during a typical voice evaluation, using stimuli commonly used clinically for auditory-perceptual judgments as well as acoustic measures to indicate when further workup is needed for a differential diagnosis of AdLD.

Yet, creak is not unique to speakers with AdLD. Many speakers, regardless of vocal status or the presence of a voice disorder, use creaky voice, particularly at the ends of phrases[26] and prosodic boundaries.[27] It has been theorized that prototypical creak (declination of f_o) in English speakers is a passive result of gradually decreasing subglottal pressure during connected speech across a breath group; at the end of the breath group, the subglottal pressure rapidly decreases, causing the vibration of the vocal folds to also rapidly decrease.[28] To offset the falling subglottal pressure, the laryngeal muscles, particularly the cricothyroid muscle, may tense to prevent the decrease in f_o, resulting in phrases without creak. Presumably, when the laryngeal muscles do not tense in this way, the result is creak.[28] Expiratory muscles of respiration may become engaged when the volume of air in the lungs falls to the point at which the elastic recoil force alone is insufficient to maintain the intended subglottal pressure.[28] Creaky voice is produced with reduced airflow and reduced subglottal pressure but higher average laryngeal resistance due to increased vocal fold thickness.[29] From a respiratory perspective, creak has been found more likely to occur during longer exhalations and when accompanied by a slightly lower inhalation amplitude before speech initiation.[30] As such, researchers postulate that creak can be employed as an air preservation strategy over the course of longer sentences that extend below relaxation pressure lung volume level.[30]

Although creaky voice in typical speakers has been well-studied, there is a gap in evidence for creaky voice in speakers with AdLD. Given the discriminative validity of creak to differentiate speakers with AdLD from typical speakers,[25] a question remains as to whether breathing patterns lead to or contribute to the symptomatology of

TABLE 1.
Demographics

Group	AdLD	Control
Age, mean (SD), years	55 (17)	55 (17)
Age, min-max, years	20-75	19-76
Sex (*n*)		
Female	13	13
Male	4	4
Race (*n*)		
Asian	1	0
Black	0	1
White	14	14
Native American/Alaskan	0	0
More than one race	0	1
Unknown/not reported	2	1
Ethnicity (*n*)		
Hispanic or Latino	1	0
Not Hispanic or Latino	14	16
Not reported	2	1

creak in speakers with AdLD. Therefore, the purpose of this study was to further investigate creak in speakers with AdLD and controls by comparing the prevalence of creak across the sentences and estimated breath groups between each group. Specifically, we hypothesized that creak would be consistent across estimated breath groups in speakers with AdLD, whereas creak would be primarily located at the end of phrases/breath groups in speakers without voice disorders.

METHODS

Participants included 17 speakers with AdLD and 17 sex- and age-matched control speakers without voice disorders, as approved by the Boston University Institutional Review Board (# 2625). Demographic information is listed in Table 1. The mean overall severity, as rated by one voice-specialized speech-language pathologist, for the AdLD group was 33.48 (SD = 14.27, min = 9.4, max = 53.8). A subset of these speakers were used in an earlier study,[25] and the procedures from the study were replicated for three additional speakers with AdLD who were in a symptomatic state and three additional controls. Each speaker read aloud the first paragraph of the Rainbow Passage[24] in a quiet clinical environment. Speech was recorded using a head-mounted omnidirectional microphone that was placed approximately 7 cm off-center from the lips. Files were digitized at 44.1 kHz.

Boundary marking

As in previous work,[31] phoneme boundaries were identified using P2FA, an open-source forced alignment toolkit, which matches predefined text strings to the recorded speech based on acoustic models of American English.[32] Data analysis was split between two trained technicians. Each technician modified the text string for each participant to exactly match what was said (eg, accounting for reading errors or repetitions) for each recording. The outputs of P2FA were imported as textgrids into Praat (Version 6.2.09), and a technician manually adjusted the timing of phoneme and silent pause boundaries based on waveform, spectrogram, and auditory-perceptual judgment of silence. Consistent with prior literature,[33,34] any silent pause > 500 ms was assumed to include an inhalation and was thus automatically marked as a breath-like pause. Additionally, a technician listened to all pauses < 500 ms to determine whether a breath was audible, in which case it was manually labeled accordingly. Each technician realigned phoneme boundaries and marked pauses for 20% of the recordings that overlapped for the purposes of calculating intrarater and inter-rater reliability. Figure 1 illustrates an example of the textgrids that were manually adjusted by the technicians beneath the corresponding waveform and spectrogram. Breaths and P2FA phonemes were marked and adjusted in the first text grid tier, and P2FA words were in the second textgrid tier.

Analysis

The presence of creak was estimated from the audio recordings in a time-varying way (10-ms window) using a creak detection algorithm,[23] implemented in a custom MATLAB[35] script, as in Marks et al.[25] A Praat textgrid

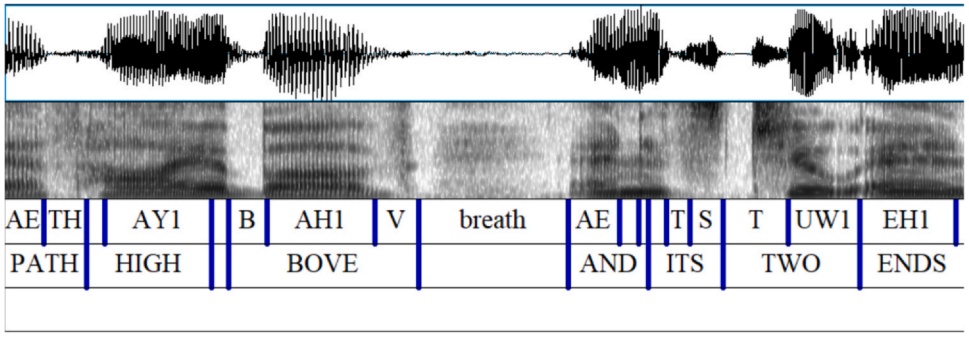

FIGURE 1. An example of labels that were manually adjusted by the technicians. The top panel displays the waveform, with its corresponding spectrogram below. The first text grid tier included P2FA phonemes and "breath" labels to indicate a breathlike pause, which was defined as an absence of voicing > 500 ms or an audible breath. The second text grid tier displays P2FA words.

was generated for each audio recording, indicating intervals of "creak" or "no creak." Creak occurrence on each phoneme was coded binarily depending on whether creak occurred within the time boundaries of the phoneme. Consistent with prior work,[31] for creak to have occurred during a given phoneme, an interval of creak had to either occur entirely within the time boundaries of the phoneme or overlap in time with the phoneme by at least 15 ms. For each phoneme, the time between the phoneme midpoint and the onset of the next breath-like pause was calculated.

Intraclass correlation coefficients (ICC) were calculated in MATLAB to assess inter-rater (type C,1) and intrarater (type A,1) reliability[36] for the durations of individual phonemes. All other statistical analyses were conducted in R (version 4.1.2).[37] To determine the relationship between the time preceding a breath-like pause and whether creak occurred, a generalized linear mixed-effects model with a binomial outcome variable (creak vs. no creak) was performed using the *glmer* function (package lme4).[38] In this model, group (AdLD, control), time preceding the next breath-like pause, and their interaction were modeled as fixed effects. Phoneme duration was included as a fixed effect control variable to account for varying opportunities for creak to occur depending on the duration of the phoneme. Participants were modeled as random intercepts. Continuous independent variables were centered prior to being entered into the model. Significant fixed effects were followed up by estimating marginal means and slopes using the *emmeans* and *emtrends* functions (package emmeans).[39] Breath count and duration were also compared between groups using two-sample *t* tests to further examine whether these factors affect the prevalence of creak in AdLD.

RESULTS

Intrarater and inter-rater reliability for the two technicians were excellent (ICC(A,1) = 0.91 and 0.92; ICC(C,1) = 0.90, respectively) for marking phoneme boundaries. Results of the generalized linear mixed-effects model revealed there was a greater probability of creak occurring in the AdLD group compared to controls (22% vs. 5%, $P < 0.001$, odds ratio [OR] = 5.0, 95% CI 2.7-9.2) and a greater probability of creak occurring as speakers approached a breath-like pause in both groups (control: $P < 0.001$, OR = 0.67, 95% CI 0.62-0.73; AdLD: $P < 0.001$, OR = 0.84, 95% CI 0.80-0.87). Moreover, the interaction between time preceding a breath-like pause and group (AdLD and controls) was statistically significant ($P < 0.001$, OR = 0.80, 95% CI 0.73-0.88), such that there was a stronger relationship between the time preceding a breath-like pause and creak for control speakers. Figure 2 displays the proportion of all phonemes that did and did not contain creak across the breath group for controls, and Figure 3 displays the same for speakers with AdLD. Figure 4 illustrates the proportion of phonemes that contain creak as a function of time (s) preceding a breath group for both AdLD and control groups.

No statistical difference was found in the number of breaths taken between control speakers (mean = 7.2, SD = 2.1) and speakers with AdLD (mean = 8.6, SD = 5.3; ($t(32) = -1.02$, $P = 0.32$). Interestingly, speakers with AdLD took significantly longer breath-like pauses (mean = 617 ms, SD = 96 ms) than control speakers (M = 510 ms, SD = 83 ms; $t(32) = -3.46$, $P = 0.002$). To determine whether this difference could be accounted for by differences in speaking rate, a *post hoc* linear model with

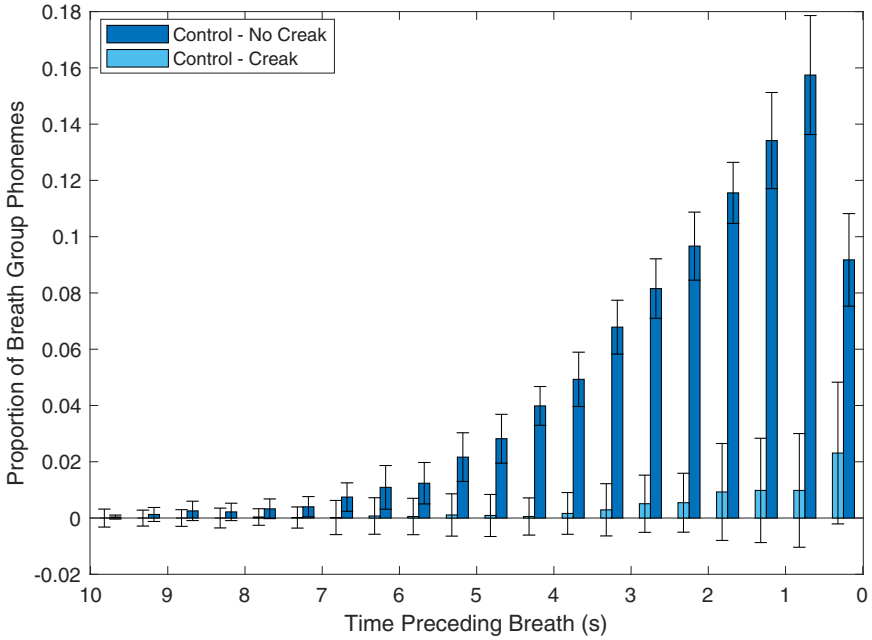

FIGURE 2. The proportion of phonemes within a breath group that contained (light shaded bars) or did not contain (dark shaded bars) creak as a function of time preceding the breath (s) in speakers without voice disorders. Error bars indicate 95% confidence intervals.

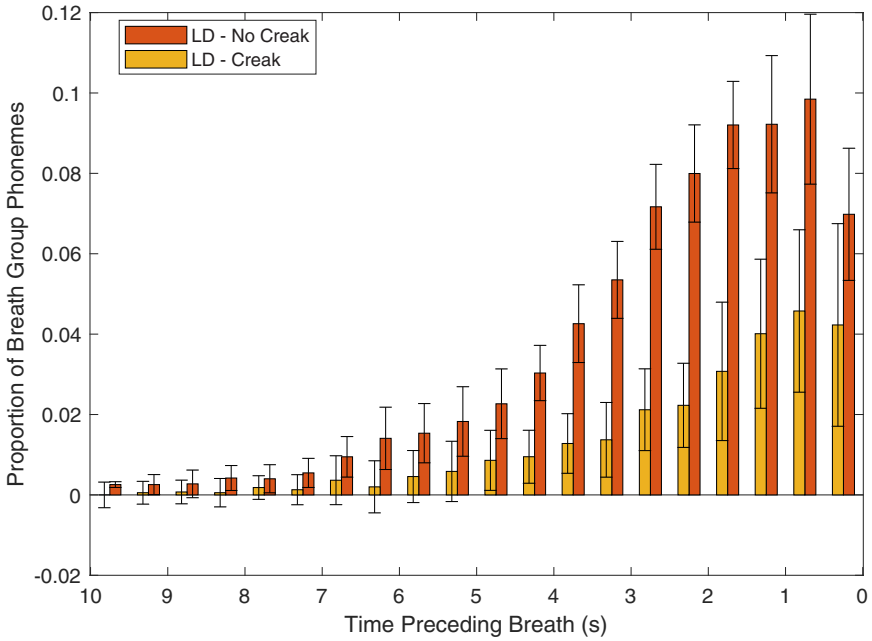

FIGURE 3. The proportion of phonemes within a breath group that contained (light shaded bars) or did not contain (dark shaded bars) creak as a function of time preceding the breath (s) in speakers with AdLD. Error bars indicate 95% confidence intervals.

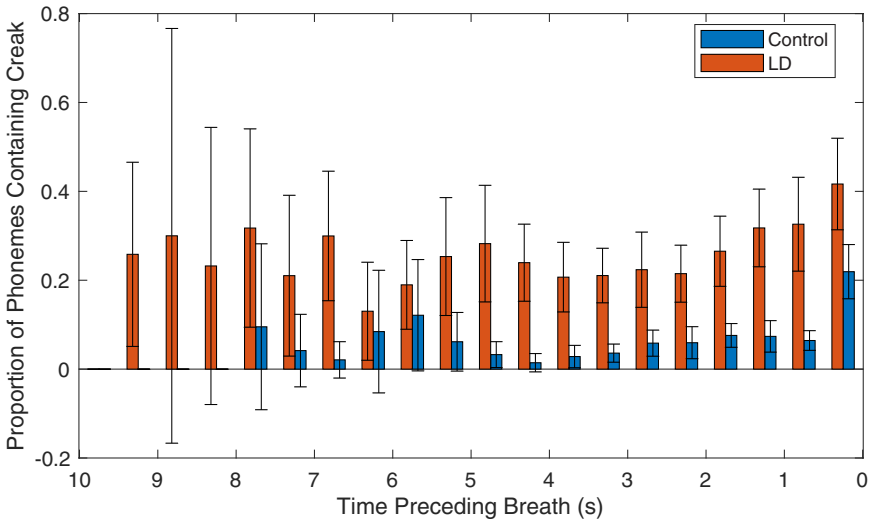

FIGURE 4. The proportion of phonemes containing creak as a function of time preceding breath (s) in speakers with AdLD (medium shaded bars) and controls (dark shaded bars). Error bars indicate 95% confidence intervals.

group and total speaking duration as independent variables and duration of breath-like pauses as the dependent variable was carried out. There was a statistically significant effect of total speaking duration on the duration of breath-like pauses ($\beta = 0.47$, $t(31) = 6.53$, $P < .001$), such that speakers who spoke at slower rates had longer breath-like pauses. Moreover, controlling for speaking duration, there was no statistically significant difference found in the duration of breath-like pauses between speakers with AdLD and controls.

DISCUSSION

Overall, the results of this study indicate that, within an estimated breath group, creak is more prevalent for speakers with AdLD compared to controls, despite there being no difference in the number of breaths. For both groups, the probability of creak occurring was greater when the phoneme was closer to the end of the estimated breath group (ie, closer to the subsequent breath-like pause); however, differences in the slopes suggest that typical speakers have a greater increase in the probability of

creak occurring at the end of breath groups compared to the increase in speakers with AdLD. The shallower slope in speakers with AdLD indicates creak was more prevalent throughout the breath group relative to controls.

In a previous study that used a subset of the same data, we found that creak differentiated speakers with AdLD and controls with outstanding diagnostic accuracy.[25] The findings from the current study suggest that creak that occurs at the end of a breath group may be less discriminative than creak that occurs at the beginning of a breath group. These interpretations are made with caution; further investigation into the discriminative validity of creak at various points of exhalation is warranted. Although our results indicate that speakers with AdLD have a greater likelihood of producing creak at the end of phrases, creak was prevalent throughout the breath group, presumably even when speakers' lung volumes were well above relaxation lung volume level. A remaining question is whether creak in AdLD is caused directly by laryngeal spasms or if it could be compensatory in anticipation of a spasm. Creak may occur due to the laryngeal spasms, in which the vocal folds are over-adducted, consistent with endoscopic descriptions of creaky voice in typical speakers, wherein the vocal folds are lax, short, and thick, albeit compressed, and the ventricular folds are often adducted and may load the vocal folds.[40,41] Alternatively, creak in AdLD could be the result of compensatory adjustments to subglottal pressure, decreased momentarily in anticipation of a laryngeal spasm or breath-holding to conserve air. These theories may be tested in future work to inform our understanding of the physiological mechanisms of creak in AdLD. A question remains as to the underlying cause of creak in speakers with AdLD, particularly in instances of creak that are not at the end of a breath group. Additional work is necessary to determine how creak may be useful as an acoustic outcome measure.

Limitations

This study was a secondary analysis on a dataset that contained a subset of recordings used to study the discriminant validity of creak in differentiating speakers with AdLD from speakers with MTD and controls. Thus, the generalizability of the results is limited. Moreover, respiratory kinematics were not measured at the time of data collection, so there is likely error in the estimated breath groups. Prospective studies with larger datasets are warranted to study creak and its underlying physiology in speakers with AdLD and its discriminative validity in differentiating speakers with AdLD from speakers with MTD and controls. However, this work provides essential preliminary evidence for future work to build upon that differences in creak prevalence exist across breath groups in speakers with AdLD and controls. Future work is warranted to include speakers with MTD to further elucidate the physiological mechanisms underlying creak.

CONCLUSION

This study serves as a preliminary investigation into the prevalence of creak across estimated breath groups for speakers with AdLD and controls. Results indicated that within an estimated breath group, creak was more prevalent for speakers with AdLD compared to controls, despite the lack of difference in the number of breaths. As both typical speakers and those with AdLD approached the end of the breath group, there was a greater probability of the occurrence of creak; however, this increase in probability was greater in controls than those with AdLD. This is preliminary evidence that creak in AdLD may not only be related to breath control but possibly in response to or because of the hyperadduction of the vocal folds during a laryngeal spasm. Further investigation into the underlying mechanisms of creak in speakers with AdLD is warranted.

Declaration of Competing Interest

Cara E. Stepp has received consulting fees from Altec, Inc./Delsys, Inc., companies focused on developing and commercializing technologies related to human movement. Stepp's interests were reviewed and are managed by Boston University in accordance with their conflict-of-interest policies. The other authors have declared that no other competing interests existed at the time of submission.

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