

Impact of Instructed Laryngeal Manipulation on Acoustic Measures of Voice—Preliminary Results

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Summary: Background. Control of laryngeal muscles is required to manipulate pitch, volume, and voice quality. False vocal fold activity (FVFA) refers to the constriction and release of constriction of the false vocal folds. True vocal fold mass (TVFM) represents the cross-sectional thickness of the vocal folds. Larynx height (LH) refers to the vertical position of the larynx in the neck. To date, studies of voice control have examined the effects of these parameters separately. No study has investigated the impact of instructed systematic manipulation of these parameters on acoustic voice measures in vocally healthy trained subjects.

Aims. This study examined the effects of systematically manipulating FVFA, TVFM, and LH on several acoustic voice measures.

Method. Twelve vocally trained speakers were instructed to use specific techniques to achieve experimental conditions of constriction and release of constriction of FVFA, thicker and thinner TVFM, and normal and low LH. Each condition was implemented in combination with manipulating the other parameters. Voice recordings of sustained vowel /a/ and Rainbow Passage were obtained for all laryngeal manipulation conditions and underwent acoustic analyses for fundamental frequency (F0), signal typing, harmonics-to-noise ratio (HNR), cepstral peak prominence (CPP), and vocal relative intensity.

Results. Constricted FVFA caused more aperiodicity in the signals, lower CPP, and lower vocal relative intensity than release of constriction. Thicker TVFM resulted in significantly higher CPP and vocal relative intensity than thinner TVFM. Modifying TVFM did not affect F0 and HNR. Low LH had significantly lower F0 but did not impact on HNR, CPP, and intensity.

Conclusions. The effects of systematic manipulation of each laryngeal parameter resulted in independent acoustic effects without measurable interaction. Release of constriction of FVFA, thicker TVFM, and low LH were configurations that resulted in more optimal acoustic signals.

Key Words: Vocal fold—Larynx—Acoustic analysis—Voice quality—Voice control—Laryngeal configuration.

INTRODUCTION

The human voice is generated by flow-induced vibration of the adducted true vocal folds under aerodynamic driving forces¹ characterized by mucosal waves that modulate the transglottal air stream into glottal pulses released into the vocal tract.² The vibratory characteristics of the vocal folds are dependent upon various factors, for example geometric, inertial, and elastic properties.¹ Physical and biomechanical forces at the glottic and supraglottic levels, and the vocal tract, function in anteroposterior (AP), lateromedial, and vertical dimensions, all interact to affect phonation.^{3,4} For example, movement of the larynx caudally exerts an abductory force on the arytenoids, widening the laryngeal inlet.⁵ This in turn impacts the amount of medial closure depending on actions of the thyroarytenoid and cricothyroid muscles.⁶ Different laryngeal configurations⁷ are associated with different vibratory patterns of the vocal folds in specific vocal registers or phonation types.⁸ Although voice

production is controlled by many factors,⁹ the false vocal folds, true vocal folds, and vertical larynx position are laryngeal parameters that influence voice quality.

False vocal fold activity (FVFA) involves constriction and release of constriction of the false vocal folds during voice production.¹⁰ FVFA has been documented as a normal articulatory phenomenon of the larynx in speech tasks with glottal stops¹¹ and in the production of phonation types such as vocal fry.¹² FVFA also occurs as compensation for glottic insufficiency in vocal fold weakness or paralysis.^{13,14} False vocal fold constriction has also been considered as playing a primary role in the clinical presentation in muscle tension voice disorders and has been included in classification schemes of abnormal muscle tension patterns e.g. in Morrison and Rammage¹⁵ and Koufman and Blalock.¹⁶ Studies on vocal hyperfunction have shown typical phonation onset i.e. hard glottal attack,¹⁷ unusual phonation patterns ie, plica ventricularis,¹⁵ pathological voice qualities,¹⁸ and abnormal acoustic measures.^{19,20} These have implied the involvement of FVFA in vocal dysfunction although no study has objectively isolated the impact of FVFA on the voice. Experimental and modeling studies have also shown the contribution of FVFA to the voice although results were inconsistent. Zhang et al²¹ demonstrated that the FVFA reduced the flow resistance of the glottis whilst Alipour et al²² found that the FVFA and epiglottis contributed to increasing glottal flow resistance. Medial and AP compressions of the FVFA were associated

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with an increase in sound intensity.²² Modeling has shown that FVFA contributes to supraglottic acoustic resonance with additional high frequency components in the 2500 Hz region.²¹ On the contrary, McGowan and Howe²³ did not observe any significant effect of the FVFA on acoustic measures. In addition, most of these studies on FVFA have used modeling and no study has examined the effects of manipulating FVFA on phonation in vocally healthy speakers despite their implication in voice disorders.

The vocal folds are the vibrators acting as the source of the acoustic signals released into the vocal tract. The vocal fold vibration is determined by its biomechanical characteristics and geometry eg, mass and stiffness³ in which stiffness plays an important role in determining mucosal wave.²⁴ Vocal fold stiffness is controlled primarily by the intrinsic laryngeal muscles eg, thyroarytenoid and cricothyroid muscles²⁵ in which the cricothyroid muscle stretches and thins the vocal folds, contributing to changes in vocal fold geometry associated with decreasing vibration wavelength and decreasing vibration amplitude.²⁶ Because the control of vocal fold stiffness and geometry is inter-related,²⁷ changes in vocal fold stiffness are associated with changes in geometry e.g. vertical thickness.²⁸ In the present paper, the cross-sectional thickness of the vocal fold was referred to as true vocal fold mass (TVFM).⁴ Mathematical models and *in vivo* experiments have suggested that the voice is affected by changes in thickness of both the cover and body of the vocal fold.²⁹ The cover-body layer thickness ratio significantly affects flow-tissue interactions influencing both fundamental frequency (F0) and vibration pattern.³⁰ Increased vocal fold thickness (thick TVFM) was believed to result in greater vertical phase difference and longer glottal closure time, leading to stronger harmonic structures.³¹ Currently, there is a lack of human-based experiments that have succeeded in simulating the changes in TVFM that take part in phonation. To date, no study has examined the effects of intentionally manipulated TVFM by vocally healthy trained speakers on voice quality.

Larynx height (LH) is the vertical position of the larynx in the neck. The effects of LH on phonation have also been extensively examined in the literature. LH contributes to the control of vocal pitch³² and vocal register.³³ In untrained speakers, LH correlates with vocal pitch³⁴ and is involved in pitch variation³⁵ and articulatory phenomena³⁶ in different linguistic contexts.³⁷ In patients with muscle tension dysphonia, LH increases during phonation¹⁸ and lowering LH is one of the goals in voice therapy for such populations.³⁸ There has been limited research on the effects of voluntarily modifying or manipulating this parameter in combination with other laryngeal parameters eg, TVFM and FVFA. In addition, it is unclear about the impact of manipulation FVFA and TVFM at different LHs.

It has been shown that specific manipulation techniques in can be utilized to differentially manipulate the above-mentioned laryngeal parameters to achieve desirable voice targets.^{39,40} A previous study⁴¹ has shown that manipulation of FVFA, TVFM, and LH in vocally healthy trained

speakers was correlated with auditory-perceptual judgments of voice quality. For example, ratings of strain and glottal fry were associated with constriction of the false vocal folds; a thinner TVFM was strongly associated with increased softness, softer tone onset, and increased breathiness. It is unclear how acoustic measures of voice quality would change as a result of the differential manipulation of these laryngeal parameters in vocally healthy trained speakers and whether any changes are consistent across speakers. An understanding of these effects would provide information to infer the patterns of underlying dysfunction based on the output signals in people with an anatomically “normal” larynx. Additionally, simulating voice production in different conditions related to laryngeal parameters (FVFA, TVFM, LH) by vocally healthy speakers can provide useful data about the voice in systematically controlled conditions that are less affected by confounding factors given the large between-speaker variability in phonation.⁴²

Voice quality can be effectively examined using acoustic analyses, which provide useful information about the function of both the voice source (larynx) and the vocal tract (resonant space from the laryngeal outlet to the mouth).⁴³ Currently there are two types of acoustic measurements. The first involves frequency-based measurements which are based on F0 tracking eg, harmonics-to-noise ratio (HNR).⁴⁴ These measures are applicable to sustained vowels and are reliable only in Type 1 and some Type 2 signals⁴⁵ (ie, nearly periodic signals). F0 remains one of the most important frequency-based measures that has been used extensively to reflect voice changes associated with different laryngeal configurations eg, vocal fold dimension,⁴⁶ stiffness,⁴⁷ and LH.⁴⁸ HNR has also been a commonly used measure in acoustic assessment of voice quality. It has been found that HNR is correlated with perceptual assessment of hoarseness⁴⁴ and vocal clarity.⁴⁹ Given the possible impact of TVFM on vocal harmonics,³¹ it seems reasonable to explore how manipulating laryngeal configuration affects this measure.

The voice can also be analyzed using spectral analysis which is applicable in both vowels and connected speech without depending upon accurate F0 tracking. The voice cepstrum is obtained by a Fourier transform of the logarithm power spectrum.⁵⁰ A cepstral peak is identified within the dominant “harmonic” corresponding to the fundamental period from which the cepstral peak prominence (CPP) is calculated as the amplitude between the peak and the regression line directly below it.⁵¹ Being a measure of periodicity and strength of the harmonics in the signal, a signal with a clear harmonic structure would have a higher cepstral peak than aperiodic signals with weak harmonics.⁵¹ Given the assumption about the impact of vocal fold thickness/TVFM on vocal contact, vertical phase difference, and closure duration on harmonic structure,³¹ CPP would be a reasonable measure to examine the impact of manipulating this laryngeal parameter. Additionally, CPP has been also shown to have stronger weighted correlations with overall voice quality than any other acoustic measures.⁵² CPP has

also been considered a significant predictor of dysphonic severity.⁵³ It is however also influenced by intensity⁵⁴ and nasal resonance.⁵⁵

Although a physiological link between differential laryngeal manipulation and the above-mentioned acoustic measures particularly HNR and CPP has not been established experimentally, it is useful to use those measures to explore how the voice changes as a result of laryngeal manipulation in vocally healthy speakers as these are common acoustic voice outcome measures. This study aimed to examine the immediate effects of manipulating FVFA, TVFM, and LH on frequency- and spectral-based acoustic voice measures.

MATERIALS AND METHODS

Ethical approval

The study protocol was approved by The University of Sydney Human Research Ethics Committee (protocol number: 2019/281). Written informed consent was obtained from all participants to participate in this study. The study was implemented in accordance with relevant ethical guidelines and regulations.

Participants

This study involved 12 participants (8 female and 4 male) aged 19–36 years who were undergraduate students in a bachelor speech-language pathology program. Participants were required to satisfy the following inclusion criteria: (1) no self-reported voice problems; (2) hearing within normal limits; (3) English as the first language; (4) nonsmokers; (5) no history of voice disorder; (6) no other disorders that might interfere with phonation (eg, asthma, gastroesophageal reflux disease); and (7) had not completed a Voice and Voice Disorders course or training in differentiated vocal tract control (Voicecraft³⁹ or Estill Voice Training⁴⁰). Five participants had received prior vocal training.

Voice training

All participants took part in training to manipulate the three laryngeal parameters in isolation and combination. Manipulation of each muscular parameter was taught by the

instructor providing cues to activate the muscle group in a gross movement (eg, yawning, coughing, laughing) which was then refined and shaped with practice, feedback with focus of attention on kinaesthetic and auditory features. The three skills were initially taught in isolation until consistent mastery of the maneuver was demonstrated after which training to combine specific maneuvers was undertaken.⁵⁶ In summary, all participants were trained in three parameters of vocal tract control including FVFA (constriction [CONST] and release of constriction [ROC] of the false vocal folds), TVFM (manipulation of TVFM into a thin versus thick vocal fold setting), and LH (lowering the larynx compared to a habitual/normal larynx position) using the Voicecraft³⁹ system of training. Training was provided by the first author over a 12-week period and consisted of six 30-minute group instruction sessions, conducted once per week. Additional sessions were provided to individuals as requested to seek additional support in the training and practice. All participants reported to have practice for a minimum of 10 minutes per day during the training period. Total training time ranged from 3 to 12 hours with an average of 6 hours per speaker reported over a 12-week training period.⁴⁶ At the end of the training period, flexible laryngoscopy confirmed that all three laryngeal configurations were successfully manipulated by the speakers.⁵⁶ Table 1 shows all experimental conditions.

Voice recording

Voice recordings took place in a sound-proof booth using an AKG C420 series II condenser cardioid ear-mounted microphone positioned 5 cm and 45° off the mouth axis. Voice signals were recorded to a Tascam DA20MkII digital audiotape recorder via a Behringer Eurotrack MX802A –ULN 8 Channel 2-Bus Mixing Amplifier at 48 kHz/16-bit. Voice samples were transferred to a personal computer via an Edirol UA-5 96 kHz/24-bit USB Audio Interface⁵⁷ with the same sampling rate and resolution and saved in *.wav file format. To obtain baseline measurements, participants were required to sustain the vowel /a/ (v) and read the Rainbow Passage (rp),⁵⁸ once only, in their habitual voice quality (HVQ) using the most comfortable pitch and intensity levels

TABLE 1.
Experimental Conditions Examined in Study Participant

Conditions	FVFA	TVFM	LH	Abbreviation
1	Habitual	Habitual	Habitual	HVQ
2	Constricted	Thick	Low	CONST.TCK.LL
3	Constricted	Thick	Normal	CONST.TCK.NL
4	Constricted	Thin	Low	CONST.THN.LL
5	Constricted	Thin	Normal	CONST.THN.NL
6	Released	Thick	Low	ROC.TCK.LL
7	Released	Thick	Normal	ROC.TCK.NL
8	Released	Thin	Low	ROC.THN.LL
9	Released	Thin	Normal	ROC.THN.NL

Abbreviations: FVFA, false vocal fold activity; TVFM, true vocal fold mass; LH, larynx height.

for each individual. They were then required to repeat both tasks, once only, using each of the three experimental laryngeal configurations ($n = 8$ experimental trials per participant \times 12 participants = 96) (Table 1). Only one trial of each laryngeal configuration was attempted; requiring the participants to repeat the tasks in all experimental conditions was deemed unsuitable due to the possibility of causing significant fatigue and changes in laryngeal function/vocal fold mucosa/laryngeal tension that could affect inference about the conditions.

Task performance was verified after training using flexible laryngoscopy and the results of the visual verification have been published elsewhere.⁵⁶ Nasendoscopy was deemed inappropriate and invasive to conduct at the time of voice recording for acoustic analysis as previous attempts were reported by participants to interfere with the desired differential laryngeal manipulation during data collection, confounding the data. The first author (CM) also verified task performance during data collection to ensure that the participants correctly manipulated the laryngeal parameters as trained.

Acoustic analyses

The sustained /a/ vowel samples (v) were edited by Audacity version 2.1.0 for Windows⁵⁹ to extract the middle 3 seconds where the signal was the most stable. The Rainbow Passage⁵⁸ voice recordings (rp) were edited to include only the second and third sentences “*The rainbow is a division of white light into many beautiful colours. These take the shape of a long round arch, with its path high above, and its two ends apparently beyond the horizon*” (p. 127). This was to allow comparisons with previous studies on cepstral/spectral measures.⁶⁰

Signal typing

All edited vowel samples were signal typed by the last author (D.D.N.) and an assistant using criteria recommended by Titze⁴⁵ and Sprecher et al⁶¹ using narrow-band spectrograms created in Praat version 6.0.39.⁶² Praat settings for signal typing used by Sprecher et al⁶¹ were as follows: duration = 0.5 s, hamming window, window length = 50 ms, time step = 0.002 s, frequency step = 5 Hz, and dynamic range = 40 dB. Signal typing was performed visually by comparing each spectrogram picture with the exemplar signal types.⁶¹ All of the samples ($n = 108$) were signal typed a second time to test intra-rater reliability of signal typing. The frequency of absolute agreement between the first and second attempt was 101 (93.5%). Signal types in all samples were also compared between the two raters. The frequency of exact agreement was 95.0%.

Frequency-based measurements

Praat (version 6.0.39) was also used to measure F0 for vowel (F0v) and Rainbow Passage (F0rp) and HNR. All voice data with signal Types 3 were excluded from F0 and HNR

measurements. F0 settings in Praat were: range = 75–500 Hz, cross-correlation method, max number of candidates = 15, silence threshold = 0.03, voicing threshold = 0.45, octave cost = 0.01, octave-jump cost = 0.35, and voice/unvoiced cost = 0.14. Additionally, a check box of “very accurate” was ticked.

Cepstral analysis

The acoustic analysis program Analysis of Dysphonia in Speech and Voice (ADSV)⁶³ was used to measure CPP in decibels (dB) for both vowel (CPPv) and Rainbow Passage (CPPrp) samples. CPP settings in ADSV were as follows: resampling rate = 25 kHz, spectral window size (pts) = 1024, max frequency for regression line calculation = 10,000, frame overlap = 75%, cepstral time averaging (frames) = 7, CPP threshold (dB) = 0, cepstral peak extraction range min-max = 60–300 Hz. Low/High spectral ratio cut-off = 4000 Hz. For intra-rater reliability analyses, 30% of the vowel and Rainbow Passage samples were acoustically re-analyzed a second time (by D.D.N.) for CPP using ADSV. A paired *t*-test showed no statistically significant difference between the first and second analyses ($P > 0.05$). To test inter-rater reliability, 30% of the samples were also analyzed by a research assistant who was blind to the aims of the study. Intraclass correlation coefficient (ICC) = 1 (mixed model, absolute agreement) for both single and average measures ($P < 0.001$).

Vocal relative intensity

Intensity was also measured in Praat. The purpose of measuring vocal relative intensity was to provide supportive data to explain CPP findings given the dependence of this measure upon vocal intensity.⁵⁴ Pearson's correlation coefficient showed significant correlation between relative intensity and CPP ($r = 0.582$, $P = 0.005$, $n = 12 \times 8$ conditions \times 2 tasks = 192).

Statistical analyses

Data were analyzed using IBM SPSS 25.0⁶⁴ and GraphPad Prism 8.1.2.⁶⁵ Chi-square tests were used to examine the effects of different laryngeal configurations on frequency of signal types. Independent sample *t* test was used to compare two conditions in a laryngeal parameter. Two-way repeated-measures analysis of variance (ANOVA) was used to examine the effects of FVFA, TVFM, and LH on acoustic voice measures. Significant main effects were evaluated with Bonferroni-adjusted tests. Prior to analyses normal distribution of the data was examined using Kolmogorov-Smirnov tests.⁶⁶ Mauchly's test of sphericity was performed before ANOVA and, if sphericity assumptions were not met, a Greenhouse-Geisser adjustment was used. Effect size was calculated using partial Eta squared (η^2). Effect size of 0.01, 0.1, and 0.25 indicated small, medium, and large effects, respectively.⁶⁷ Significance level of 0.05 was used.

RESULTS

There were three laryngeal parameters each of which was manipulated into two distinct conditions: FVFA (CONST, ROC), TVFM (thick, thin), and LH (low, normal). To examine the effects of each parameter, a two-way ANOVA (2 [parameter] x 2 [vocal task] or (2 [parameter] x 2 [gender]) with repeated-measures on the parameter (FVFA, TVFM, LH) was calculated. Where task was not factored in the ANOVA model, data from both vowel and Rainbow Passage were combined. Where gender was not factored in the ANOVA model, data from both female and male were combined. Where a significant main effect was observed post hoc test using Bonferroni adjustment was used. For comparison between two conditions in the same parameter without factoring task or gender, an independent sample *t* test was used.

Signal type

Figure 1 shows the distribution of signal types for both CONST and ROC conditions associated with TVFM and LH modifications. The majority of signal types in CONST were Types 2 and 3 while ROC and HVQ showed both Type 1 and Type 2 signals. There was significant association between signal type and FVFA phenomena in which CONST resulted in significantly higher incidence of signal Type 3 than ROC ($\chi^2 = 29.471$, $df = 2$, $P = 0.005$). Additionally, CONST Type 3 signals occurred significantly more often in thin than in thick TVFM ($\chi^2 = 7.056$, $df = 1$, $P = 0.008$).

Frequency-based measures such as F0 and HNR were not obtained from samples that showed Type 3 signals.

Impact of manipulating FVFA

Because of the aperiodicity of the signals in constricted FVFA, F0, and HNR were not analyzed for this parameter. Acoustic measures of CPP and vocal relative intensity were obtained.

CPP

Figure 2 shows CPP data across FVFA conditions. A significant main effect of FVFA on CPP was observed ($F_{1,188} = 44.844$, $P = 0.005$, partial $\eta^2 = 0.193$). Constricted FVFA caused CPP to decrease by 2.8 dB compared with ROC ($P = 0.005$). There were also significant main effects of tasks ($F_{1,188} = 138.226$, $P = 0.005$, partial $\eta^2 = 0.424$) in which vowel CPP was 4.8 dB higher than connected speech CPP ($P = 0.005$). Significant interactions between FVFA and tasks were observed ($F_{1,188} = 5.548$, $P = 0.02$, partial $\eta^2 = 0.029$), suggesting that the effects of FVFA depended upon whether the production was vowel or connected speech ie, the magnitude of vowel CPP drop in CONST was greater than that of Rainbow Passage CPP.

Post hoc analyses were performed to compare CPP between CONST and ROC in specific TVFM and LH conditions. CPP dropped in CONST in all TVFM and LH conditions (Table A2 in Appendix).

Vocal relative intensity

Two-way ANOVA (2[FVFA] x 2[tasks], repeated-measures for FVFA) showed significant main effects of FVFA ($F_{1,188} = 11.298$, $P = 0.001$, partial $\eta^2 = 0.057$) and task ($F_{1,188} = 24.644$, $P = 0.005$, partial $\eta^2 = 0.116$). No interaction effect was found between FVFA and task ($P = 0.633$). From both tasks combined, relative intensity in CONST was 3.5 dB lower than that in ROC ($P = 0.001$). Relative intensity of RP was 5.2 dB lower than that of vowel ($P = 0.005$).

Impact of manipulating TVFM

Fundamental frequency (F0)

Figure 3 shows F0 data across experimental conditions. Because of aperiodicity of the signals in constricted FVFA, F0 was only measured in ROC. Manipulating TVFM did not impact on F0. A two-way ANOVA (2[TVFM] x 2[gender]) did not show any significant main effects of TVFM on

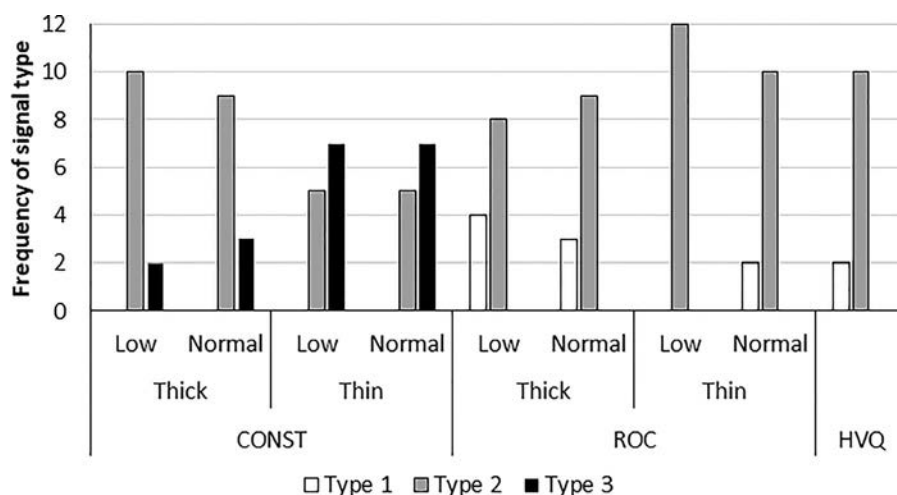


FIGURE 1. Distribution of signal types in different laryngeal conditions. CONST, constriction of false vocal folds; ROC, release of constriction of false vocal folds; HVQ, habitual voice quality.

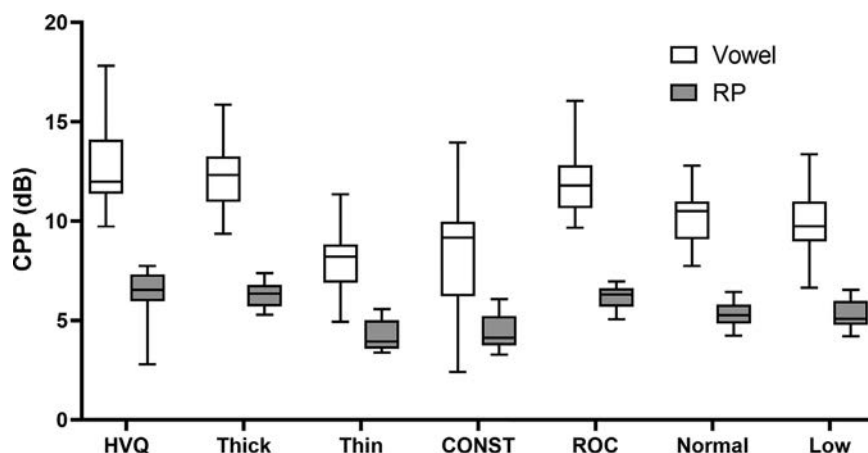


FIGURE 2. Cepstral peak prominence (CPP) in all laryngeal conditions. RP, Rainbow Passage; dB, decibel; CONST, Constriction of false vocal folds; ROC, release of constriction; HVQ, habitual voice quality.

F0 from both vowel and RP combined ($P = 0.918$) but there was significant main effect of gender ($F_{1,92} = 439.408$, $P = 0.005$, partial $\eta^2 = 0.827$) with F0 in male being 97.0 Hz lower than that in female (Figure 3).

Harmonics-to-noise ratio (HNR)

HNR was also measured in ROC conditions given the aperiodicity of the acoustic signals in CONST. Mean HNR in thick and thin TVFM were 23.5 dB and 21.7 dB, respectively. t Test showed no significant differences in HNR between thick and thin TVFM ($P = 0.084$).

CPP

Figure 2 shows CPP data across TVFM conditions for all three parameters. When TVFM changed from thick to thin configuration, CPP dropped significantly. Two-way ANOVA (2[TVFM] x 2[task], repeated-measures for TVFM) revealed significant main effects of TVFM ($F_{1,188} = 67.652$, $P = 0.005$, partial $\eta^2 = 0.265$). CPP produced in thick TVFM was 3.2 dB higher than that in thin TVFM ($P = 0.005$). Vocal

tasks also had significant effects on CPP ($F_{1,188} = 153.074$, $P = 0.005$, partial $\eta^2 = 0.449$) in which vowel CPP was 4.8 dB higher than connected speech CPP ($P = 0.005$). There was also a significant interaction effect between TVFM and tasks ($F_{1,188} = 8.347$, $P = 0.004$, partial $\eta^2 = 0.043$): the effects of TVFM on CPP depended upon vocal tasks ie, CPP of both vowel and Rainbow Passage was decreased in thin TVFM than in thick TVFM but vowel CPP showed a larger magnitude of decrease than Rainbow Passage CPP.

Post hoc tests comparing thick and thin TVFM across different FVFA and LH conditions also showed significantly higher CPP in thick TVFM than in thin TVFM (Table A1 in Appendix).

Vocal relative intensity

Two-way ANOVA (2[TVFM] x 2[tasks], repeated-measures for TVFM) showed significant main effects of TVFM ($F_{1,188} = 116.466$, $P = 0.005$, partial $\eta^2 = 0.383$) and task ($F_{1,188} = 37.829$, $P = 0.005$, partial $\eta^2 = 0.168$). No interaction effect was present ($P = 0.18$). From both tasks combined, relative intensity in thick TVFM was 9.2 dB higher

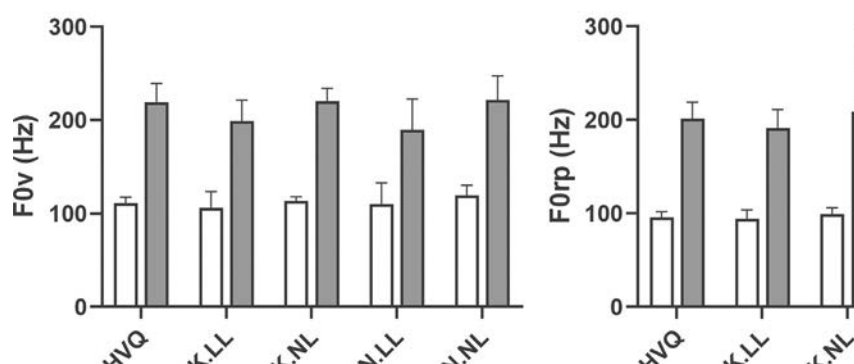


FIGURE 3. Fundamental frequency (F0) in 3 laryngeal conditions. In FVFA, only release of constriction was included as constriction resulted in a large frequency of Type 3 signals. v, vowel; rp, rainbow passage; Hz, Hertz; HVQ, habitual voice quality; ROC, release of constriction; TCK, thick; THN, thin; LL, low larynx height; NL, normal larynx height.

than that in thin TVFM ($P= 0.005$). Relative intensity of RP was 5.2 dB lower than that of vowel ($P= 0.005$).

Impact of manipulating LH

Fundamental frequency

A two-way ANOVA (2[LH] x 2[gender]) showed significant main effects of LH on vocal F0 ($F_{1,92} = 13.496$, $P= 0.005$, partial $\eta^2 = 0.128$): F0 in low LH was 15.1 Hz lower than that in normal LH ($P= 0.005$). Significant gender effect was also found ($F_{1,92} = 557.157$, $P= 0.005$, partial $\eta^2 = 0.858$): Male F0 was 97.0Hz lower than female F0 ($P= 0.005$). Weak but significant interaction effects between LH and gender were also present: ($F_{1,92} = 4.445$, $P= 0.038$, partial $\eta^2 = 0.046$) in which female showed a larger magnitude of F0 decrease in low LH compared to male.

Harmonics-to-noise ratio (HNR)

Mean HNR in low and normal LH was 22.5 dB and 22.7 dB, respectively. *t* Test showed no statistically significant differences in this measure between low and normal LH ($P= 0.839$).

CPP

CPP data for LH conditions is shown in Figure 2. Two-way ANOVA (2[LH] x 2[task]) showed that LH did not affect CPP ($P= 0.637$). However, there was a significant main effect of task ($F_{1,188} = 109.212$, $P= 0.005$, partial $\eta^2 = 0.367$) in which vowel CPP was 4.8 dB higher than connected speech CPP ($P= 0.005$). There was no significant interaction effect between LH and task ($P= 0.719$). Post hoc tests were not calculated for LH conditions.

Vocal relative intensity

There was no significant main effect of LH on relative intensity ($P= 0.223$). However, there was significant main effect of task ($F_{1,188} = 23.406$, $P= 0.005$, partial $\eta^2 = 0.111$). Relative intensity in RP was 5.2 dB lower than that of vowel ($P= 0.005$). No interaction between LH and task was found ($P= 0.964$).

DISCUSSION

Investigation of the impact of manipulating laryngeal parameters FVFA, TVFM, and LH on the voice are necessary but challenging as sophisticated vocal techniques must be mastered to achieve the intended differentiated laryngeal postures. It is important to note that previous studies have addressed the impact of these parameters on the voice eg, studies on the effects of modifying false vocal folds,²² vocal fold mass,³¹ and LH.^{68,69} However, no study has examined the effects of systematic manipulation of each parameter ie, a laryngeal parameter was modified under the impact of the other two parameters also manipulated concomitantly. In this study, the impact of constricted FVFA and ROC of FVFA was investigated in both thick and thin TVFM

condition at both low and normal LH. Several combinations of these parameters were difficult to perform eg, constricted FVFA + thin TVFM + low LH. The design of these experimental conditions allowed for investigation of specific laryngeal muscular conditions to understand the resultant voice outcome. In a small number of vocally healthy, trained speakers, this study attempted to demonstrate how the vocal signals changed when these three components of the phonation mechanism were modified. The findings have implications for understanding laryngeal biomechanics in vocal pedagogy, speech pathology, and laryngology.

FVFA

The findings on the manipulation of FVFA was as expected; CONST resulted in significantly poorer voice quality reflected by the occurrence of aperiodic signal types and lower CPP values; HVQ and ROC were associated with the occurrence of more periodic signal types (1 and 2), higher CPP values and HNR values that were within the normal range. These findings support previous assumptions that constricted FVFA may interact to interfere with true vocal fold vibration.⁷⁰ Our results further confirmed the impact of constricted FVFA on the voice, a condition frequently observed in muscle tension voice disorders.¹⁵ These findings can be explained given the mechanical and aerodynamic interactions between the false and true vocal folds (illustrated in Figure 4) which can disrupt vocal fold vibrations in some cases.¹⁰ Numerous researchers have found that there are variable effects of false or ventricular fold activity in the larynx. These effects range from increased glottal resistance and sound intensity in canine larynx studies⁸ to the alteration, suppression or enhancement of vocal fold vibrations in high speed video *in vivo* studies.⁷ Others have observed the occurrence of rough voice quality in the presence of ventricular fold activation but in some cases no impact on voice quality was detected when the ventricular folds were observed to vibrate in phase with the true vocal folds.^{71,72} This variation in the effect of the false vocal folds on vibration of the true vocal folds would and explain the presence of Type 2 and 3 signal type across constricted conditions in this study as well as the variation in effect on CPP across vowel and connected speech samples. The greater

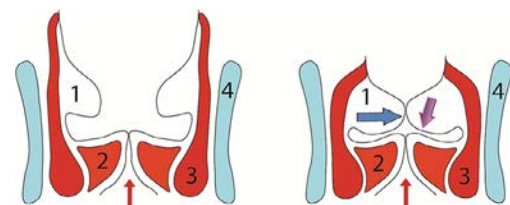


FIGURE 4. Schematic diagram of the action of laryngeal muscles on the false vocal folds. Left: Coronal view of the larynx in non-constricted condition. Right: Muscle constriction results in both medial compression (blue arrow) and inferior compression (purple arrow) of the false vocal folds. (1) False vocal fold; (2) True vocal fold; (3) Aryepiglottic muscle; (4) Thyroid cartilage.

reduction of vowel CPP in CONST compared to connected speech is likely explained by the maintenance of a static state of FVF constriction, whereas, the smaller reduction of CPP in connected speech suggests that FVF constriction varied in response to articulatory movements, having less impact on overall voice quality. It should be noted that only lateral false vocal fold constriction was identified in the speakers⁵⁶ but was not quantifiable using acoustic measurements. Therefore, it is impossible to understand whether the effects of FVFA resulted from only lateral rather than AP or sphincteric constriction, and whether the relationship between FVFA and the acoustic measures was linear. Future studies may incorporate detailed visualization examination and analysis with supraglottic ratings in different vocal tasks to give more insight into the link between the magnitude and dimension of FVFA and the vocal output.

TVFM

TVFM in this study represented the cross-sectional thickness of the vocal fold⁴ with two conditions ie, thick and thin. The manipulation of TVFM involves voluntary changes in vocal fold geometry which is mainly implemented via the synergistic action of the thyroarytenoid and cricothyroid muscles.²⁵ Hirano⁷³ has shown that vocal fold thickness is controlled by the following muscles: cricothyroid (thinning), thyroarytenoid (thickening), and interarytenoid (thickening). Previous analysis concluded from perceptual evaluation of laryngoscopic footage that speakers were achieving the target TVFM condition in the experiment.⁵⁶ Our findings showed that recordings when speakers were targeting a thin TVFM mass condition was associated with significantly lower CPP and lower intensity compared with thick TVFM. This implies that thick TVFM represents a more optimal vocal fold mass configuration than thin vocal folds, as the thick TVFM condition produced more acoustic energy without explicit instruction to raise volume.

Thin TVFM might be associated with a decrease in the vertical depth of the medial edge and this might have an effect on vertical phase difference⁷⁴ and glottal flow characteristics.³¹ Zhang³¹ showed distinct differences between thin and thick TVFM in glottal flow waveform and the corresponding sound spectra. Compared with a thin vocal fold medial surface, increased medial edge in the thick TVFM condition resulted in larger contact areas, which affected the glottal flow waveform and its time derivative, creating stronger excitation of higher harmonics. Zhang³¹ also showed that glottal closure duration was also increased, introducing more spectral peaks in the lower frequency range; ie, the harmonic structure was stronger in the thick than in the thin TVFM condition. Because CPP is measured from spectral parameters, modification of the voice spectra would change these values. Our findings of the effects of TVFM on this acoustic voice measure implies that the thicker vocal fold configuration creates more acoustic energy across the spectrum.

We also found that changes in TVFM toward thin or thick did not significantly affect F0 in both vowel and connected speech, which contradicts previous observations. Hollien⁴⁶ showed a high correlation between F0 and vocal fold thickness in which F0 was lower for thick vocal folds and vice versa. Using computational modeling, Zhang³¹ showed that F0 decreased with an increase in medial surface thickness. Titze,⁷⁵ however, states that the relationship between vocal fold mass and F0 should be considered with respect to factors such as thickness, length, and depth. A possible explanation for this finding is to consider the role that mass, length and tension have on the vibration rate of the true vocal folds. It has been considered that these three characteristics are interdependent on each other; that is, changing one changes the other to the same degree. Our results provide support for the claim that “regulation of the vocal ligament tension is the primary means of controlling pitch and is achieved by the distraction of the ends of the ligament” (p. 68).⁶ That is, the manipulation of vocal fold mass may not have a primary effect on F0 in the absence of changing length and tension of the true vocal folds (ie, where a higher pitch is required, or at a higher lung volume where tracheal pull is invoked). In the context where length and tension are increased, the manipulation of mass may regulate the impact of lengthening on the vibration of the free edge of the true vocal fold in the vertical dimension, creating the conditions for a longer closed phase at the specified vibration rate. This effect has been observed in a previous study⁷⁶ where closed quotient (CQ) was not affected by lung volume condition at voice onset on a vowel. However, F0 was significantly increased in a high lung volume condition, theoretically when the vocal ligament is lengthened due to the effect of tracheal pull.³⁴ Biomechanically this phenomenon can be explained by the Body-Cover theory,⁷⁷ as manipulation of the thyroarytenoid muscle would change the cross-sectional area of the muscle (body), whilst the vibrating edge (cover) would be passively controlled by lengthening the true vocal fold and increasing tension. Changing the length of the true vocal folds may have a greater effect on F0, most likely by influencing tension of the vibrating edge. More experiments in which both CQ and F0 are measured, are needed to clarify the effect of TVFM manipulation on F0.

LH

This is the first study to our knowledge to examine the effects of LH on cepstral measures and we have showed that low LH had lower CPP in both vowel and connected speech. This finding was unexpected given the use of larynx lowering as a therapeutic intervention in voice therapy^{78,79} and the use of a lowered larynx position in classical singing training.⁸⁰ The lower cepstral values in low LH does not necessarily imply an unfavorable larynx position in phonation. Instead, it reflects the fact that the cepstral measurement was highly dependent upon vocal tract effects, as suggested in previous research.⁵⁵ Alternatively, the

mechanical coupling effect described by Fink and Dumarrest⁶ and Iwarsson and Sundberg³⁴ would result in a reduced CQ at a lower larynx due to the abductory forces on the arytenoids when the larynx is pulled caudally by either sternothyroid and/or accessory extrinsic muscles (Figure 5). A reduced CQ produces less spectral energy in the vocal signal, which would result in a lower CPP value. A lower intensity may also result depending on vocal tract configuration which would also result in a lower CPP value, as found in our results. Although some authors have maintained that the effects of the vocal tract can be isolated from those of the voice source,⁸¹ our findings do not support those claims. This characteristic of cepstral measurements must be taken into consideration if this analysis is to be used to assess vocal function. If LH is to be considered as a factor that represents satisfactory response to voice therapy in the treatment of muscle tension voice disorders, using cepstral analysis as an outcome measure may be confounded by a reduction in intensity.

We also found that low LH was associated with lower F0 in both vocal tasks. These findings are in agreement with previous observations of the effects of laryngeal vertical position on F0.⁴⁸

Our results show that there was a dominant effect on voice quality of FVFA constriction across all speakers. Whilst effects of TVFM and LH were also evident, these did not offset the significant effect of FVFA on the acoustic signal. This suggests that activity of the FVFA should be the primary focus in voice therapy and training to improve voice quality and control. Given the improved voice quality in the thick TVFM condition, regardless of the activity of the FVFA, manipulation of TVFM should also be considered as a training focus if voice quality is to be optimized. Manipulation of these two muscular parameters, however, requires differentiated control of the true and false vocal folds, ie, selective activation of the thyroarytenoid muscle to increase cross-sectional mass of the vibrating edge without over-activation that would recruit FVFA constriction. Whilst the effect of a low LH in moderating medial compression via trachea pull was not evident in our results, there may also be a role of manipulating LH in balancing control of FVFA and TVFM. Further research is required to investigate the role of LH in overall voice quality control.

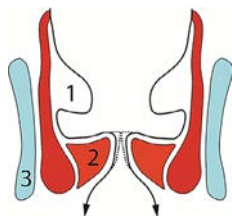


FIGURE 5. Schematic illustration of the glottal opening effects of tracheal pull (arrows). Dashed line indicates vocal fold position before the pull. (1) False vocal fold; (2) True vocal fold; (3) Thyroid cartilage.

CONCLUSIONS

Differentiated manipulation of three laryngeal parameters demonstrated consistent acoustic effects in 12 vocally healthy trained speakers. The major findings from our study are as follows:

- (1) Constriction of the FVFA modified periodicity of the acoustic signals manifested by the occurrence of Type 3 signals and a significant reduction in CPP for both vowel and connected speech. The effects of FVFA on CPP depended upon vocal tasks and the combination of TVFM/LH configuration in which FVFA constriction appeared to affect this measure less in thin TVFM and low LH.
- (2) Modification of TVFM regardless of LH resulted in changes in cepstral and hence spectral characteristics of phonation but did not affect frequency-based measures eg, F0 and HNR.
- (3) Change in larynx vertical position led to changes in F0 in both thick and thin TVFM. This suggests LH was an important factor in controlling vocal fold vibration. However, LH did not affect CPP, which suggests that this measure was not sensitive to changes in vocal tract length.

These preliminary findings further confirm that the vocal mechanism is biomechanically complex and works in multi-dimensional interaction, even in the context of attempting to differentially control specific muscular parameters of the larynx and vocal tract. Deeper understanding of these interactions, and how they can be manipulated via instruction, will inform targeted vocal training, interpreting pathophysiological phenomena, and advance the development of suitable treatment strategies to address those phenomena in voice disorders.

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SUPPLEMENTARY DATA

Supplementary data related to this article can be found online at [doi:10.1016/j.jvoice.2020.11.004](https://doi.org/10.1016/j.jvoice.2020.11.004).

REFERENCES

1. Titze IR. The physics of small-amplitude oscillation of the vocal folds. *J Acoust Soc Am*. 1988;83(4):1536–1552.
2. Berke GS, Gerratt BR. Laryngeal biomechanics: an overview of mucosal wave mechanics. *J Voice*. 1993;7(2):123–128.
3. Jiang J, Lin E, Hanson DG. Vocal fold physiology. *Otolaryngol Clin North Am*. 2000;33(4):699–718.
4. Titze IR. *Principles of Voice Production*. Englewood Cliffs, NJ: Prentice-Hall, Inc.; 1994.

5. Kotby MN, Haugen LK. Attempts at evaluation of the function of various laryngeal muscles in the light of muscle and nerve stimulation experiments in man. *Acta Otolaryngol.* 1970;70(5):419–427.
6. Fink BR. *Laryngeal Biomechanics ed., R.J. Demarest.* Cambridge, MA: Harvard University Press; 1978.
7. Titze IR, Talkin DT. A theoretical study of the effects of various laryngeal configurations on the acoustics of phonation. *J Acoust Soc Am.* 1979;66(1):60–74.
8. Murry T, Xu JJ, Woodson GE. Glottal configuration associated with fundamental frequency and vocal register. *J Voice.* 1998;12(1):44–49.
9. Jiang J, Lin E, Hanson DG. Vocal fold physiology. *Otolaryngol Clin North Am.* 2000;33(4):699–718.
10. Bailly L, Bernardoni NH, Muller F, et al. Ventricular-fold dynamics in human phonation. *J Speech Lang Hear Res.* 2014;57(4):1219–1242.
11. Stager SV, Bielamowicz SA, Regnell JR, et al. Supraglottic activity: evidence of vocal hyperfunction or laryngeal articulation? *J Speech Lang Hear Res.* 2000;43(1):229–238.
12. Allen EL, Hollien H. A laminagraphic study of pulse (vocal fry) register phonation. *Folia Phoniatr (Basel).* 1973;25(4):241–250.
13. Belafsky PC, Postma GN, Reulbach TR, et al. Muscle tension dysphonia as a sign of underlying glottal insufficiency. *Otolaryngol Head Neck Surg.* 2002;127(5):448–451.
14. Bielamowicz S, Kapoor R, Schwartz J, et al. Relationship among glottal area, static supraglottic compression, and laryngeal function studies in unilateral vocal fold paresis and paralysis. *J Voice.* 2004;18(1):138–145.
15. Morrison MD, Rammage LA. Muscle misuse voice disorders: description and classification. *Acta Otolaryngol.* 1993;113(3):428–434.
16. Koufman JA, Blalock PD. Functional voice disorders. *Otolaryngol Clin North Am.* 1991;24(5):1059–1073.
17. Andrade DF, Heuer R, Hockstein NE, et al. The frequency of hard glottal attacks in patients with muscle tension dysphonia, unilateral benign masses and bilateral benign masses. *J Voice.* 2000;14(2):240–246.
18. Morrison MD, Nichol H, Rammage LA. Diagnostic criteria in functional dysphonia. *Laryngoscope.* 1986;96(1):1–8.
19. Klingholz F, Martin F. Speech wave aperiodicities at sustained phonation in functional dysphonia. *Folia Phoniatrica.* 1983;35(6):322–327.
20. Kotby MN, Titze IR, Saleh MM, et al. Fundamental frequency stability in functional dysphonia. *Acta Oto-Laryngologica.* 1993;113(3):439–444.
21. Zhang C, Zhao W, Frankel SH, et al. Computational aeroacoustics of phonation, part II: Effects of flow parameters and ventricular folds. *J Acoust Soc Am.* 2002;112(5 Pt 1):2147–2154.
22. Alipour F, Jaiswal S, Finnegan E. Aerodynamic and acoustic effects of false vocal folds and epiglottis in excised larynx models. *Ann Otol Rhinol Laryngol.* 2007;116(2):135–144.
23. McGowan RS, Howe MS. Influence of the ventricular folds on a voice source with specified vocal fold motion. *J Acoust Soc Am.* 2010;127(3):1519–1527.
24. Berke GS, Gerratt BR. Laryngeal biomechanics: an overview of mucosal wave mechanics. *J Voice.* 1993;7(2):123–128.
25. Vahabzadeh-Hagh AM, Zhang Z, Chhetri DK. Three-dimensional posture changes of the vocal fold from paired intrinsic laryngeal muscles. *Laryngoscope.* 2017;127(3):656–664.
26. Moore DM, Berke GS. The effect of laryngeal nerve stimulation on phonation: a glottographic study using an in vivo canine model. *J Acoust Soc Am.* 1988;83(2):705–715.
27. Johns MM, Urbanchek M, Chepeha DB, et al. Length-tension relationship of the feline thyroarytenoid muscle. *J Voice.* 2004;18(3):285–291.
28. Zhang Z. Effect of vocal fold stiffness on voice production in a three-dimensional body-cover phonation model. *J Acoust Soc Am.* 2017;142(4):2311–2321.
29. Jiang W, Xue Q, Zheng X. Effect of Longitudinal Variation of Vocal Fold Inner Layer Thickness on Fluid-Structure Interaction During Voice Production. *J Biomech Eng.* 2018;140(12).
30. Jiang W, Zheng X, Xue Q. Influence of vocal fold cover layer thickness on its vibratory dynamics during voice production. *J Acoust Soc Am.* 2019;146(1):369.
31. Zhang Z. Cause-effect relationship between vocal fold physiology and voice production in a three-dimensional phonation model. *J Acoust Soc Am.* 2016;139(4):1493.
32. Shipp T. Vertical laryngeal position during continuous and discrete vocal frequency change. *J Speech Hear Res.* 1975;18(4):707–718.
33. Echternach M, Traser L, Markl M, et al. Vocal tract configurations in male alto register functions. *J Voice.* 2011;25(6):670–677.
34. Iwarsson J, Sundberg J. Effects of lung volume on vertical larynx position during phonation. *J Voice.* 1998;12(2):159–165.
35. Ohde RN. Fundamental frequency as an acoustic correlate of stop consonant voicing. *J Acoust Soc Am.* 1984;75(1):224–230.
36. Esling JH. The IPA categories "pharyngeal" and "epiglottal": laryngoscopic observations of pharyngeal articulations and larynx height. *Lang Speech.* 1999;42(Pt 4):349–372.
37. Brunelle M, Nguyen DD, Nguyen KH. A laryngographic and laryngoscopic study of Northern Vietnamese tones. *Phonetica.* 2010;67(3):147–169.
38. Elliot N, Sundberg J, Gramming P. Physiological aspects of a vocal exercise. *J Voice.* 1997;11(2):171–177.
39. Bagnall AD. *Voicecraft Workshop Manual.* Adelaide, SA: Voicecraft International; 1997.
40. Estill J. *Primer of Compulsory Figures: Level One.* Santa Rosa, CA: Estill Voice Training Systems; 1996.
41. Madill CJ, Sheard C, Heard R. Are Instructions to Manipulate Specific Parameters of Laryngeal Function Associated with Auditory-Perceptual Ratings of Voice Quality in Nondisordered Speakers? *J Voice.* 2017;31(4):504.e21–504.e33.
42. Castellana A, Carullo A, Astolfi A, et al. Intra-speaker and inter-speaker variability in speech sound pressure level across repeated readings. *J Acoust Soc Am.* 2017;141(4):2353.
43. Baken RJ, Orlikoff RF. *Clinical measurement of speech and voice.* 2nd ed. San Diego: Singular Thomson Learning; 2000.
44. Yumoto E, Gould WJ, Baer T. Harmonics-to-noise ratio as an index of the degree of hoarseness. *J Acoust Soc Am.* 1982;71(6):1544–1550.
45. Titze IR. *Workshop on Acoustic Voice Analysis: Summary Statement.* Iowa City: National Center for Voice and Speech; 1995.
46. Hollien H. Vocal fold dynamics for frequency change. *J Voice.* 2014;28(4):395–405.
47. McKenna VS, Murray ESH, Lien YAS, et al. The Relationship Between Relative Fundamental Frequency and a Kinematic Estimate of Laryngeal Stiffness in Healthy Adults. *J Speech Lang Hear Res.* 2016;59(6):1283–1294.
48. Honda K, Hirai H, Masaki S, et al. Role of vertical larynx movement and cervical lordosis in F0 control. *Lang Speech.* 1999;42:401–411.
49. Warhurst S, Madill C, McCabe P, et al. The vocal clarity of female speech-language pathology students: an exploratory study. *J Voice.* 2012;26(1):63–68.
50. Noll AM. Cepstrum pitch determination. *J Acoust Soc Am.* 1967;41(2):293–309.
51. Hillenbrand J, Cleveland RA, Erickson RL. Acoustic correlates of breathy vocal quality. *J Speech Hear Res.* 1994;37(4):769–778.
52. Maryn Y, Roy N, De Bodt M, et al. Acoustic measurement of overall voice quality: a meta-analysis. *J Acoust Soc Am.* 2009;126(5):2619–2634.
53. Awan SN, Roy N. Toward the development of an objective index of dysphonia severity: a four-factor acoustic model. *Clin Linguist Phon.* 2006;20(1):35–49.
54. Awan SN, Giovinco A, Owens J. Effects of vocal intensity and vowel type on cepstral analysis of voice. *J Voice.* 2012;26(5). 670 e15-20.
55. Madill C, Nguyen DD, Yick-Ning Cham K, et al. The Impact of Nasalance on Cepstral Peak Prominence and Harmonics-to-Noise Ratio. *Laryngoscope.* 2019;129(8):E299–E304.
56. Madill C, Sheard C, Heard R. Differentiated vocal tract control and the reliability of interpretations of nasendoscopic assessment. *J Voice.* 2010;24(3):337–345.
57. Roland Corp. *UA-5, USB Digital Audio Capture.* [23/10/2019]; Available from: <https://www.roland.com/au/products/ua-5/>.
58. Fairbanks G. *Voice and articulation drillbook.* 2nd ed. New York: Harper & Row; 1960.

59. Audacity Team. Audacity(R): Free Audio Editor and Recorder [Computer application] 2019. Available from: <https://www.audacityteam.org/>.
60. Awan SN, Roy N, Zhang D, et al. Validation of the Cepstral Spectral Index of Dysphonia (CSID) as a Screening Tool for Voice Disorders: Development of Clinical Cutoff Scores. *J Voice*. 2016;30(2):130–144.
61. Sprecher A, Olszewski A, Jiang JJ, et al. Updating signal typing in voice: addition of type 4 signals. *J Acoust Soc Am*. 2010;127(6):3710–3716.
62. Boersma P., Weenink D. Praat: doing phonetics by computer 2018. Available from: <http://www.fon.hum.uva.nl/praat/>.
63. PentaxMedical. Analysis of Dysphonia in Speech and Voice - ADSV [Computer application]. 2018. Available from: <https://www.pentaxmedical.com/pentax/en/99/1/Analysis-of-Dysphonia-in-Speech-and-Voice-ADSV>.
64. IBM Corp. IBM SPSS Software. 2018. Available from: <https://www.ibm.com/analytics/data-science/predictive-analytics/spss-statistical-software>.
65. GraphPad Software. Prism 8. 2018. Available from: <https://www.graphpad.com/scientific-software/prism/>.
66. Massey FJ. The Kolmogorov-Smirnov Test for Goodness of Fit. *J Am Statist Assoc*. 1951;46(253):68–78.
67. Murphy KR. Statistical power analysis : a simple and general model for traditional and modern hypothesis tests. Fourth ed. Myors B, Wolach AH, editors. New York: Routledge; 2014.
68. Sundberg J, Askenfelt A. Larynx height and voice source. A relationship? *Quarterly Prog Status Rep.* 1981;22(2-3):023–036.
69. Elliott TM, Popeil L. Laryngeal height, modal registers, and modes of phonation contribute jointly to vocal timbre. *J Acoust Soc Am*. 2018;143(3). 1907-.
70. Agarwal M, Scherer RC. Effects of false vocal fold width on translaryngeal flow resistance. *J Acoust Soc Am*. 2001;110(5). 2762-.
71. Lindestad PA, Blixt V, Pahlberg-Olsson J, et al. Ventricular fold vibration in voice production: a high-speed imaging study with kymographic, acoustic and perceptual analyses of a voice patient and a vocally healthy subject. *Logoped Phoniatr Vocol*. 2004;29(4):162–170.
72. Lindestad PA, Hammarberg B, Larsson H, et al. Ventricular fold co-vibration in chronic laryngitis studied with high-speed imaging and acoustic analysis. In: *Proceedings of the 24th World Congress of the International Association of Logopedics and Phoniatrics (IALP)*. Amsterdam; 1998.
73. Hirano M. Vocal mechanisms in singing: Laryngological and phoniatric aspects. *J Voice*. 1988;2(1):51–69.
74. Zhang Z. Mechanics of human voice production and control. *J Acoust Soc Am*. 2016;140(4):2614.
75. Titze IR. Vocal fold mass is not a useful quantity for describing F0 in vocalization. *J Speech Lang Hear Res*. 2011;54(2):520–522.
76. Yeo S, Lee R, McCabe P, et al. Effects of Different Lung Volume Conditions on Closed Quotient, Vocal Fundamental Frequency and Relative Intensity in Vocally Untrained Female Speakers. *Acoust Austr.* 2018;46(3):339–347.
77. Hirano M. Morphological structure of the vocal cord as a vibrator and its variations. *Folia Phoniatr Logop*. 1974;26(2):89–94.
78. Boone DR, McFarlane SC. *The voice and voice therapy*. 6th ed. Boston, MA: Allyn and Bacon; 2000.
79. Boone DR, McFarlane SC. A critical view of the yawn-sigh as a voice therapy technique. *J Voice*. 1993;7(1):75–80.
80. Echternach M, Burk F, Burdumy M, et al. Morphometric Differences of Vocal Tract Articulators in Different Loudness Conditions in Singing. *PLoS One*. 2016;11(4) e0153792.
81. Skowronski MD, Shrivastav R, Sensitivity Hunter EJ. Cepstral Peak. A Theoretic Analysis and Comparison of Several Implementations. *J Voice*. 2015;29(6):670–681.