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## Characteristics of phonatory function in singers and non-singers with vocal fold nodules

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### Abstract

**Purpose**—The goal of this study was to determine if there were significant differences between singers and non-singers in the morphology of vocal nodules and associated impact on vocal function.

**Method**—Participants were 10 professionally trained singers with nodules, 8 non-singers with nodules, and 10 individuals with healthy normal voice (controls). Surface electromyography (sEMG) from three anterior neck locations and acoustic rise times for vowels /a/ and /i/ were measured in all participants. In individuals with nodules, dB SPL / cm H<sub>2</sub>O, glottal airflow, and nodule location and size were also measured.

**Results**—There were no significant differences between singers and non-singers with nodules in terms of airflow, dB SPL / cm H<sub>2</sub>O, nodule size, or nodule location. In non-singers with nodules, airflow and nodule size were significantly correlated, but were not significantly correlated in singers. Vowel rise times and sEMG during vocal tasks did not differentiate among nodule and control groups. Sternocleidomastoid sEMG during initiation of the vowel /a/ was statistically significantly stronger in non-singers with nodules relative to singers with nodules and controls.

**Conclusions**—Nodule morphology did not differ between singers and non-singers, although some behavioral aspects of phonation differed between the groups.

### Keywords

vocal hyperfunction; vocal nodules; neck surface electromyography

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## Introduction

A frequently occurring cause of hoarseness is vocal fold nodules (e.g., Colton, Casper, & Leonard, 2006). Vocal fold nodules are benign lesions, clinically defined as small protuberances located between the anterior and middle third of the vocal fold (Aronson, 1980; Dikkers & Schutte, 1991; Marcotullio, Magliulo, Pietrunti, & Suriano, 2002). They are described as being gray, white, or pearl in color and bilateral (Aronson, 1980; Marcotullio, et al., 2002), and can impede complete closure of the glottis leading to breathy voice production (e.g., Colton, et al., 2006).

While nodules occur in a variety of occupations, they are commonly found in singers (operationally defined here as individuals with professional training in singing; Herrington-Hall, Lee, Stemple, Niemi, & McHone, 1988). There has been some question as to whether nodules in singers and non-singers have a common etiology and/or presentation (e.g., Peppard, Bless, & Milenkovic, 1988). Previous studies have compared singer and non-singers with nodules with respect to nodule size and location, as well as acoustic and aerodynamic measures. Peppard et al. (1988) rated vocal fold mass on an equal-appearing interval scale from 1 to 6, finding that 10 non-singers had significantly larger nodules than 10 singers. In a retrospective qualitative review of 312 professional voice users (e.g., singers, teachers, and actors) reporting with voice defects, Sedláčková (1961) reported a trend toward differences in size, shape, and location of the nodules seen in singers and non-singers. Specifically, Sedláčková observed that singers' nodules tend to be small, pale/white, and located at the anterior third of the of the vocal fold, whereas nodules in non-singers tend to be larger, sitting on a larger base, having a gel-like appearance, and located further posterior. However, no study has confirmed these observations with scientific study.

In comparison to non-singers, singers are much more attuned to their voices, and thus they are more likely to seek treatment in the early stages of formation, when the nodules are relatively small. Further, singers are trained to adapt to day-to-day changes in their vocal mechanism to produce their best voice, making them likely to use differing compensatory mechanisms than non-singers. These confounding behavioral factors make it potentially difficult to elucidate possible anatomical differences between nodules in singers versus non-singers.

One possible difference between singers and non-singers is the role of vocal hyperfunction in both nodule formation and the resulting compensation. Vocal hyperfunction refers to "conditions of abuse and/or misuse of the vocal mechanism due to excessive and/or 'imbalanced' muscular forces" (Hillman, Holmberg, Perkell, Walsh, & Vaughan, 1989), characterized by excessive laryngeal and paralaryngeal tension (Aronson, 1980; Dworkin, Meleca, & Abkarian, 2000; Koufman & Blalock, 1991; Morrison, Rammage, Belisle, Pullan, & Nichol, 1983; Roy, Ford, & Bless, 1996), and commonly accompanies voice disorders. For many years, the development of most benign lesions on the vocal fold surface have been assumed to be related to hyperfunctional behavior or phonotrauma (Hillman, Gress, Hargrave, Walsh, & Bunting, 1990). For instance, in 1962, Godfrey Arnold wrote that "vocal nodules and polyps represent a local tissue reaction to the mental strain imposed by inappropriate emotional adjustment to the demands made by society" (Arnold, 1962). However, much is still unknown about the underlying mechanisms of vocal hyperfunction and its role in developing organic disorders (Hillman, et al., 1990). Given the extensive vocal demands of singers, it is possible that vocal hyperfunction plays a smaller role, and that nodule formation is more typically caused by greater vocal use.

The objective measures of airflow and vocal efficiency have been examined in singers and non-singers. Peppard et al. (1988) found that singers with nodules performed similarly to

non-singers without nodules on a selection of acoustic and aerodynamic measures, and even outperformed the non-singers on maximum performance tasks such as maximum phonation time and maximum frequency range. The glottal airflow magnitude (referred to simply as airflow and typically reported in units of L/s) represents the magnitude of airflow through the glottis during vocal fold oscillation. Airflow values have been reported for both singers and non-singers with nodules and healthy normal voice (Peppard, et al., 1988). Results indicated that mean airflow values did not differ between singers with nodules and those without, but that non-singers with nodules had significantly greater airflow values than non-singers with healthy normal voice. Airflow values for all participants were found to be significantly correlated ( $R^2 = 0.20$ ) with subjective ratings of nodule mass viewed during endoscopy; however, correlations were not calculated within singer and non-singer categories, leaving the question as to whether airflow values are more or less indicative of nodule size in the two populations.

Vocal efficiency is the ratio of acoustic power to aerodynamic power, calculated as the ratio of vocal intensity to the product of the airflow and subglottal pressure. Jiang et al. (2004) reported significant differences in the vocal efficiency measures of individuals with nodules and individuals with healthy normal voice at various vocal intensities. However, no comments were made regarding the singing training of these individuals, nor were measures of nodule size indicated. In an unpublished Master's thesis, Fulton (2007) explored the vocal characteristics of 10 vocally normal females who were trained singers relative to 10 vocally normal females who were non-singers, finding no significant differences between the vocal efficiency measures of the two groups when producing vowels at typical pitch and loudness levels. It is unclear, however, if this lack of difference in healthy normal speakers is relevant in a population of individuals with nodules. Further, measures of vocal efficiency have not been shown to be reliable indicators of vocal dysfunction in individuals with nodules (Hillman, et al., 1989). Hillman and colleagues noted that the sensitivity of this ratio to indicate vocal dysfunction was compromised by elevated levels of both transglottal pressure and airflow, leading to vocal efficiencies in the normal range.

Some possible objective indicators of vocal hyperfunction include the aero-acoustic measure of dB SPL / cm H<sub>2</sub>O, acoustic rise times and anterior neck surface electromyography (sEMG). Subglottal pressure is the driving force behind vocalization, and abnormally high values of subglottal pressure can indicate inefficient valving of the glottal airstream. However, given the inherent relationship between subglottal pressure and sound pressure level (e.g., Isshiki, 1964), using raw estimates of subglottal pressure is unlikely to be specific to vocal hyperfunction. Here we chose to investigate the ratio of sound pressure level (SPL) to subglottal pressure, often represented in units of dB SPL / cm H<sub>2</sub>O. This measure attempts to normalize the subglottal pressure used by the produced sound pressure level, and is associated with the level of effort necessary to achieve phonation.

A common perceptual correlate of vocal hyperfunction is hard glottal attack, which is a method of vowel initiation perceptually “characterized by rapid and complete adduction of the vocal folds before the initiation of phonation” (Colton, et al., 2006, pg 78). A retrospective study by Morrison and colleagues found that 65% of patients with hyperfunction presented with hard glottal attack (Morrison, Nichol, & Rammage, 1986). Further, comparison of the frequency of hard glottal attack in vocal disorders thought to be related to vocal hyperfunction found higher frequencies of hard glottal attack in all groups with voice disorder when compared with a control group of individuals with healthy normal voice (Andrade, et al., 2000). While clinical practice continues to rely on broad auditory perception to characterize glottal attack, more objective methods do exist (see Orlikoff, Watson, Baken, & Deliyski, 2006 for review). Peters et al. (1986) calculated the rise time of the acoustic signals of vowel production and found a correlation of  $-0.69$  with auditory

perceptual ratings of voice onset abruptness, indicating the usefulness of vowel rise time as an objective measure of glottal attack.

Surface electromyography (sEMG) has been studied in attempts to objectively quantify neck muscle tension. Redenbaugh and Reich (1989) measured mean neck sEMG of 7 individuals with healthy normal voice and 7 “hyperfunctional” individuals, finding that the individuals with disordered voice had significantly greater mean normalized neck sEMG during phonation than individuals with healthy normal voice. The sEMG signal corresponded with a single electrode position, centered over the thyrohyoid membrane, a recording position likely to sample electrical activity from the sternohyoid and possibly cricothyroid muscles during production of vowels and a reading passage. The disordered population in their study was varied, consisting of seven individuals with very different clinical presentation, history, and even sex (5 women, 2 men), and the data collection method was relatively rudimentary. The sEMG signals were amplified, filtered, and integrated (2-sec interval) in real-time, with the integrated values displayed on-screen only and recorded by hand. Moreover, their study did not comment on the past singing training of participants, which could have significant effects on the use of the extrinsic musculature for speech. For instance, in a study of four professional, classically trained singers (baritones) and four non-singers producing the vowel /a/ at seven frequency points between 90 and 350 Hz, qualitative differences between frequency and laryngeal height were found (Shipp & Izdebski, 1975). In this study, non-singers tended to increase laryngeal height with increases in fundamental frequency, whereas singers tended to keep their larynges below their resting heights for all fundamental frequencies. While this study may largely account for technical differences through classical training in respect to laryngeal position, it is possible that singers use their extrinsic laryngeal musculature in very different ways than non-singers to attempt to compensate for their vocal pathology.

The purpose of the present study was to determine if there are significant differences in phonatory function between singers and non-singers with vocal nodules. Particular attention was paid to the possible role of vocal hyperfunction by collecting measures of airflow, dB SPL / cm H<sub>2</sub>O, normalized nodule size and location, acoustic vowel rise times, and anterior neck sEMG measures.

## Method

### Participants

Participants were 10 adult females with a history of professional singing training and active singing engagement diagnosed with vocal fold nodules prior to any therapeutic intervention (mean age = 19.7 years, SD = 0.8 years), 8 adult females diagnosed with vocal fold nodules prior to any therapeutic intervention with no history of singing training (mean age = 34.1 years, SD = 12.1 years), and 10 adult females with healthy normal voice (mean age = 23.8 years, SD = 2.0 years) to act as experimental controls. The group of singers with nodules was comprised of working professional singers and full-time college or graduate students enrolled in voice training/singing programs. Based on self-report, their primary styles of singing were: musical theater (N = 6), pop (N = 2), gospel (N = 1), and opera (N = 1). Individuals recruited with healthy normal voice were volunteers with no voice-related complaints and were screened for abnormal pathology of the larynx using trans-oral or trans-nasal endoscopy with stroboscopy. While 2 of these 10 individuals sang for pleasure, none were professionally trained. Disordered participants were diagnosed based on comprehensive voice evaluation procedures that included endoscopic, acoustic, aerodynamic, and perceptual assessment by a team comprised of a laryngologist and one or more certified speech-language pathologists, and images of their vocal folds were collected via standard trans-oral endoscopy with stroboscopy.

## Recording Procedure

Recordings consisted of a brief vocal assessment of each participant including three trials of the vowels /a/ and /i/, read speech (The Rainbow Passage; Fairbanks, 1960), six read CAPE-V sentences (i.e., Kempster, Gerratt, Verdolini Abbott, Barkmeier-Kraemer, & Hillman, 2009), and spontaneous running speech. Spontaneous speech was elicited by questions from the investigator or a speech-language pathologist, asking the participant to describe their voice issues (if relevant), or describe what they did the previous weekend (or similar). After completion of these speech tasks, maximal voluntary contraction (MVC) maneuvers were performed. These consisted of asking the participants to perform neck contraction against manual resistance for the purpose of normalizing sEMG data (see Data Analysis section). Each participant provided 2 – 3 maximum contractions, each lasting 2 – 3 seconds.

Simultaneous anterior neck sEMG and acoustic signals from a lavalier microphone (Sennheiser MKE2-P-K, Wedemark, Germany) were recorded digitally with Delsys™ (Boston, Massachusetts) hardware (Bagnoli Desktop System) and software (EMGworks 3.3) at 20 kHz. The sEMG was recorded and analyzed in view of current European standards (Hermens, et al., 1999). Participants' necks were prepared for electrode placement by cleaning the neck surface with an alcohol pad and "peeling" with tape (exfoliation) to reduce electrode-skin impedance, noise, DC voltages, and motion artifacts. The anterior neck sEMG was recorded with three Delsys™ 3.1 double-differential surface electrodes placed parallel to the underlying muscle fibers of the 1) thyrohyoid, omohyoid, and sternohyoid muscles, 2) cricothyroid and sternohyoid muscles, and 3) sternocleidomastoid muscle. The Delsys™ 3.1 double-differential surface electrodes consisted of three 10 mm long and 1 mm wide silver bars with inter-electrode distances of 10 mm.

Electrode 1 was centered about 1 cm lateral to the anterior neck midline, as far superior as was possible without impeding jaw opening of the participant. Electrode 2 was centered on the gap between the cricoid and thyroid cartilages of the larynx, and centered at 1 cm lateral to the midline, contralateral to Electrode 1. Electrode 3 was centered one-third of the distance from the sternal notch of each participant to his or her mastoid process (following Falla et al. 2002). See Panel B of Figure 1 for a schematic of electrode placements. A ground electrode was placed on the superior aspect of the participant's left shoulder. The sEMG signals were pre-amplified and filtered using Delsys™ Bagnoli systems set to a gain of 1000 and a band-pass filter with roll-off frequencies of 20 Hz and 450 Hz.

Individuals with nodules underwent aerodynamic voice assessment with the Phonatory Aerodynamic System (KayPENTAX, Lincoln Park, NJ). The participants produced a series of /pæ/ vocalizations at a comfortable pitch and loudness while the oral airflow was measured with a face mask and pneumotachograph, intra-oral air pressure was sensed with a translabially placed catheter, and the acoustic signal was recorded with a microphone.

## Data Analysis

The mean of the root-mean-squared (RMS) values of anterior neck sEMG data computed in 1 s windows (no overlap) was calculated for the entire length of all completed vocal tasks using custom MATLAB® (Mathworks Inc., Natick, MA) software. Intrinsic laryngeal musculature is most active during vowel initiation and cessation relative to the steady state portion of the vowel (e.g., Gallena, Smith, Zeffiro, & Ludlow, 2001; Hillel, 2001). Thus, for the production of the vowels /a/ and /i/, the RMS was also calculated for 100 ms windows (no overlap) during the 500 ms before and 300 ms after vowel initiation, as well as during the 300 ms before and 500 ms after vowel termination. In order to compare anterior neck sEMG gathered among participants, the variability associated with neck surface electrode contact and placement was minimized by normalizing the sEMG to the MVC reference



contraction (calculated as the maximum RMS in a 1 s window). It has been shown that for anterior neck musculature, the MVC reference is more reliable than submaximal reference contractions (Netto & Burnett, 2006). For this reason, all of the sEMG data presented here are in terms of % MVC.

The air pressure, air flow, and acoustic signals acquired during aerodynamic voice assessment were digitized and analyzed with the Phonatory Aerodynamic System to produce indirect estimates of subglottal air pressure (cm H<sub>2</sub>O), glottal air flow (L / sec) and sound pressure level (dB). The ratio of the sound pressure level in dB SPL to the subglottal air pressure was also calculated as a first order estimate of vocal efficiency (Hillman, Montgomery, & Zeitels, 1997).

In order to find an acoustic correlate for abruptness of attack, the acoustic rise times of the vowels /a/ and /i/ were measured by analyzing the acoustic signals similarly to the method utilized in Peters et al. (1986). Peters et al. (1986) defined the rise time of the acoustic signal as the time needed for an envelope of the acoustic signal to go from 10% to 90% of the maximum amplitude. To implement this method, the RMS of the acoustic signal in 80 ms rectangular windows was calculated in intervals of 2.5 ms (97% overlap).

Due to the disordered nature of the voice signals being analyzed and the irregularity of the acoustic signals, the window size employed here was modified from the method employed by Peters et al. (1986) to be 80 ms rather than 40 ms, the slope initiation was defined as 20% of the maximum amplitude rather than 10%, and the slope termination was defined as 80% of the maximum amplitude rather than 90%. An example of the rise time for a vowel production is shown in Panel A of Figure 1.

For the individuals with nodules, a representative digital still was taken from their trans-oral stroboscopy exam. Stills were chosen to maximize image quality, while showing the full extent of the vocal folds during abduction. The vocal fold stills were analyzed using custom MATLAB<sup>®</sup> software that allowed for the following landmarks to be marked (in pixels): the length of each vocal fold (right and left) from the anterior commissure to and including the vocal process of the arytenoid cartilage, the distance from the center of each nodule (right and left) to the anterior commissure, and the visible extent (area) of each nodule (right and left). The marked extent of each nodule was based on the visible changes to the vocal fold surface. In some more mild cases, the area defined as the nodule consisted of only the raised protrusion above the smooth edge of the vocal fold. In more extreme cases, distinct fibrovascular changes to the vocal fold could be seen lateral to the raised surface of the nodule, and these changes were included as part of the extent of the nodule. The nodule location (anterior-posterior) for each participant was defined as the mean of the ratios of the distance from each nodule to the anterior commissure to the total length of the vocal fold. The nodule size was calculated as the mean of the two nodule extents normalized by the mean of the length of the two vocal fold.

Minitab<sup>®</sup> Statistical Software (Minitab Inc., State College, PA) was used to calculate Pearson's correlations, analysis of variance (ANOVA), Tukey simultaneous *t*-tests, and Student's *t*-tests. Effects showing  $p < 0.05$  were considered statistically significant. Statistical testing of differences between singers and non-singers with vocal nodules using Student's *t*-tests was not adjusted for alpha inflation due to the exploratory nature of this study.

## Results

### Mean anterior neck sEMG and acoustic rise time in all participants

The mean anterior neck sEMG at recording positions 1, 2, and 3 during the production of all vocal tasks was analyzed with a two-factor ANOVA by group and vocal task, which failed to find a statistically significant effect of group (control, singer nodules, non-singer nodules) for any anterior neck sEMG location, but did find a statistically significant effect of vocal task (rest, /a/, /i/, high /a/, low /a/, read sentences, read paragraph, and spontaneous speech) for all three recording locations (EMG1; EMG2; EMG3). The mean anterior neck sEMG at all three recording positions tended to be lower during rest and production of the vowels /a/ and /i/, and higher during both read and spontaneous speech production.

The anterior neck sEMG recorded from positions 1, 2, and 3 was averaged in 100 ms segments during the 500 ms before and 300 ms after vowel initiation, as well as the 300 ms before and 500 ms after vowel termination. The results of this analysis are shown as a function of group and vowel (/a/ and /i/) in Figure 2. Based on the trends seen in the vowel initiation, a one-factor ANOVA of the mean anterior neck sEMG at recording positions 1, 2, and 3 during the 500 ms prior to vowel production was performed; however, it failed to find a statistically significant effect of group (control, singer nodules, non-singer nodules) for both vowels (/a/ and /i/) and all recording positions (1, 2, 3), with the exception of recording position 3 prior to the vowel /a/, which showed a statistically significant effect of group. Tukey simultaneous *t*-tests showed a statistically significant difference between the anterior neck sEMG at recording position 3 prior to /a/ between the controls (MEAN = 0.015 % MVC) and non-singers with nodules (MEAN = 0.037 % MVC) and between singers with nodules (MEAN = 0.015 % MVC) and non-singers with nodules. No statistically significant difference was found between controls and singers with nodules. Example traces of the raw voice and anterior neck sEMG data for one control participant and one non-singer with nodules are shown in Figure 3.

A one-factor ANOVA of the acoustic rise time during vowel production (/a/ and /i/) failed to find a statistically significant effect of group (control, singers with nodules, non-singers with nodules). However, a general trend was seen in the acoustic rise time measure for both vowels, suggesting smaller rise times (more abrupt voice onset) for non-singers with nodules relative to the singers with nodules and controls. Boxplots of the acoustic rise time measures in the three groups are shown in Figure 4.

Correlations between acoustic rise time and the anterior neck sEMG at recording positions 1, 2, and 3 500 ms prior to vowel production are shown for all groups in Table 1. No statistically significant correlations were found between any of the rise time measures and anterior neck sEMG measures, and correlations were generally weak with  $R^2$  values ranging from 0.01 – 0.11.

### Airflow, dB SPL / cm H<sub>2</sub>O, and measures of nodule size and location in singers and non-singers with vocal nodules

Student's *t*-tests on airflow, dB SPL / cm H<sub>2</sub>O, and nodule size and location did not show statistically significant (two-sided) differences between singers and non-singers with nodules. There was a trend, however, for larger nodule size and greater airflow in non-singers relative to singers. Boxplots of the airflow, vocal dB SPL / cm H<sub>2</sub>O, and nodule size and location for the two groups are shown in Figure 5.

## Correlations between measures in singers and non-singers with vocal nodules

Correlations among vowel rise time measures, anterior neck sEMG prior to vowel production, airflow, dB SPL / cm H<sub>2</sub>O, nodule location, and nodule size in all individuals with nodules (singers and non-singers) are shown in Table 2. Statistically significant correlations were found between a number of the anterior neck sEMG measures, as well as between airflow and nodule size.

Correlations among vowel rise time measures, anterior neck sEMG prior to vowel production, airflow, vocal dB SPL / cm H<sub>2</sub>O, nodule location, and nodule size were calculated separately in non-singers and singers with nodules and are shown in Table 3 and Table 4, respectively. In non-singers, statistically significant correlations were found between a number of the anterior neck sEMG measures, as well as between: dB SPL / cm H<sub>2</sub>O and EMG 3 prior to /a/, airflow and EMG 3 prior to /a/, airflow and nodule size, and airflow and vocal dB SPL / cm H<sub>2</sub>O. In singers, statistically significant correlations were found between a number of the anterior neck sEMG measures, as well as between: airflow and linear rise time for /i/, nodule size and EMG3 prior to /i/, and dB SPL / cm H<sub>2</sub>O and EMG3 prior to /a/.

## Discussion

Contrary to previous speculation (i.e., Sedláčková, 1961) and findings (i.e., Peppard, et al., 1988), quantitative measures of nodule morphology did not differ between singers and non-singers. However, there were differences seen between singers and non-singers with nodules with respect to some behavioral descriptors of phonation. Specifically, short-term trends in sEMG differed between singers and non-singers with nodules, as did the relationship between airflow and nodule size. These results suggest possible differences in compensatory behaviors between singers and non-singers with nodules, and suggest future directions for research into sEMG measures in these populations.

### Nodule Size and Location

Surprisingly, no significant differences in nodule size or location were seen between singers and non-singers with nodules (see Figure 5). There were, however, notable trends for non-singers with nodules to have larger nodule size relative to singers. This trend is consistent with the work of Peppard et al. (1988) who found significantly larger nodules in non-singers relative to singers (as measured with a qualitative 6-point scale). Nodule location in the two groups ranged between 38% – 48% of vocal fold length (more anterior), with no obvious trend for a difference between singers and non-singers. These findings do not support the qualitative observations of Sedláčková (1961), who postulated that the nodules of singers were located more anteriorly than those of non-singers.

### Aerodynamic and Acoustic Measures

Although not significant, non-singers tended to have greater airflow relative to singers, similar to Peppard et al. (1988) who found significantly higher airflow in non-singers with nodules relative to singers with nodules. Measures of glottal airflow were consistently higher than values seen in a normal population (RANGE = 0.09 – 0.2 L/s; Holmberg, Hillman, & Perkell, 1988); however, most values for glottal airflow were somewhat lower than those seen previously in two individuals with nodules, who reported with airflow values of 0.41 L/s and 0.49 L/s (Hillman, et al., 1989). Measures of dB SPL / cm H<sub>2</sub>O can be estimated for the two individuals with nodules studied by Hillman et al. (1989) as 8.5 dB SPL / cm H<sub>2</sub>O and 8.6 dB SPL / cm H<sub>2</sub>O, based on their reported sound pressure levels and subglottic pressures. These values compare well with the values seen in our participants.



Although no statistically significant differences were seen among the groups in the acoustic rise times, the acoustic rise time measure for both vowels trended toward smaller values for non-singers with nodules relative to the singers with nodules and to controls (see Figure 4). This measure has not been previously explored in these populations. Future work with more repetitions of this measure in individuals should be performed to follow up on the trend found here.

### Anterior Neck sEMG

Although no differences were seen in the mean anterior neck sEMG at any of the three recording positions during the entire extent of all vocal tasks, some interesting trends were noted during the shorter time-scale surrounding vowel initiation and termination. These trends were observed in the anterior neck sEMG activity at all 3 recording locations (see Figure 2) indicating that perhaps some non-singers with nodules are recruiting neck musculature during vowel production to a greater degree than controls. However, only the anterior neck sEMG at recording position 3 prior to the vowel /a/ showed a statistically significant effect of group, with non-singers with nodules showing higher anterior neck sEMG than controls and singers with nodules. Thus, RMS sEMG measures did not show specificity for the presence of nodules in singers or non-singers, which is consistent with the large degree of variability seen here in both individuals with nodules and controls (cf. Figure 2).

Our sEMG results are inconsistent with those of Redenbaugh & Reich (1989). Their study examined 7 individuals with hyperfunctionally-related voice disorders (2 individuals with vocal contact ulcers, 2 individuals with vocal nodules, and 3 individuals with vocal fold erythema) relative to 7 controls, finding increased normalized anterior neck sEMG during speech in individuals with vocal hyperfunction relative to controls (Redenbaugh & Reich, 1989). Apart from the previous issues regarding out-dated methodology raised in the introduction, our only explanation for this discrepancy is a difference in the methodology utilized during the elicitation of MVC used for normalization. Here, each participant was asked to provide 2 – 3 maximum contractions lasting 2 – 3 seconds each. The maximum RMS sEMG for any 1 second window was used for sEMG normalization. Redenbaugh & Reich (1989) elicited the MVC by having participants exert a “maximal” force over a 15 seconds, the mean of which was used for normalization. This methodology should result in lowered MVC values, but the degree to which a 15 sec “maximal” task could differentially affect a disordered group relative to controls is unknown.

### Correlations among Measures

Correlations among vowel rise time measures, anterior neck sEMG prior to vowel production, airflow, dB SPL / cm H<sub>2</sub>O, nodule location, and nodule size in all individuals with nodules (singers and non-singers) showed significant correlations between anterior neck sEMG measures, and also between airflow and nodule size. The correlations among anterior neck sEMG measures indicate that individuals who use extrinsic laryngeal musculature and SCM prior to voicing tend to do so non-specifically. Their tendency appears to be to employ muscles beneath all of the recording locations during voicing, rather than just particular areas. The significant correlation between airflow and nodule size ( $R = 0.62$ ) is consistent with the previous finding of Peppard et al. (1988), that airflow values were significantly correlated with subjective ratings of nodule mass viewed during endoscopy ( $R = 0.45$ ).

When correlations among measures were examined separately in non-singers and singers, there were a few interesting differences. In non-singers, the anterior neck sEMG at electrode 3 (SCM) prior to the vowel /a/ was significantly correlated with dB SPL / cm H<sub>2</sub>O ( $R =$

0.82) and airflow ( $R = -0.94$ ). Further, airflow was significantly correlated with nodule size ( $R = 0.71$ ) and dB SPL / cm H<sub>2</sub>O ( $R = -0.79$ ). One interpretation of this set of correlations is that in non-singers, there is less efficient compensation for glottal insufficiency. Specifically, non-singers show a high positive correlation between airflow and nodule size, indicating that the size of the nodule is a major factor in glottal closure. Further, airflow is negatively correlated with dB SPL / cm H<sub>2</sub>O, suggesting that non-singers may be using increasingly inappropriately high subglottal pressures to achieve conversational sound pressure levels in cases of increased airflow. The fact that the anterior neck sEMG at electrode 3 (SCM) prior to the vowel /a/ was significantly correlated with dB SPL / cm H<sub>2</sub>O ( $R = 0.82$ ) and airflow ( $R = -0.94$ ) suggests that less vocally impaired individuals may use more anterior neck sEMG prior to production of /a/. Without further study, it is difficult to predict whether this is a result of compensatory techniques or causative behaviors.

In singers, significant correlations were found between the anterior neck sEMG at electrode 3 (SCM) prior to the vowel /i/ and nodule size, sEMG at electrode 3 (SCM) prior to the vowels /a/ and /i/ and dB SPL / cm H<sub>2</sub>O, and airflow and the linear rise time for /i/. Some of the differences in correlations between singers and non-singers indicate that there may be more efficient compensation for glottal insufficiency in singers. Specifically, unlike non-singers, singers showed no significant correlation between airflow and nodule size. Interestingly, a significant correlation was seen between sEMG at electrode 3 (SCM) prior to the vowels /a/ and /i/ with dB SPL / cm H<sub>2</sub>O ( $R = -0.67$  and  $R = -0.75$ , respectively), possibly suggesting that individuals with decreased dB SPL / cm H<sub>2</sub>O are using increased SCM, contrary to the pattern seen in non-singers. Further, the sEMG at electrode 3 (SCM) during the vowel /i/ was significantly correlated with nodule size ( $R = 0.78$ ), suggesting that individuals with larger nodules were more likely to attempt to compensate with the SCM. Another interesting correlation seen in singers (and not in non-singers) was the significant correlation between the linear rise time prior to the vowel /i/ and airflow ( $R = 0.75$ ). This correlation suggests that singers who use “soft” or “easy” onset (associated with large rise times) may adapt a learned breathy style during sustained phonation, leading to increased steady-state airflow values.

One further factor that could affect correlations in singers with nodules is their typical style of singing. While singing, the classical or operatic style is associated with lower subglottal pressures and a lower closed quotient than is the musical theater style (e.g., Bjorkner, 2008; Stone, Cleveland, Sundberg, & Prokop, 2003). Assuming that these singing tendencies carry over into speech, they could affect some of the correlations noted in this group. Most obviously, style of singing offers an alternative explanation for the lack of correlation in singers between airflow and size of nodules. Many of the singers with nodules who were musical theater singers presented with the largest nodules. Given that musical theater singers typically phonate with a higher closed quotient during singing (thereby reducing flow), this underlying relationship could have offset the expected correspondence between airflow and size of nodules, and could explain the lack of correlation between the two seen here in singers.

## Conclusions

Nodule morphology did not differ between singers and non-singers, although some behavioral aspects of phonation differed between the groups. Specifically, no significant differences in airflow, dB SPL / cm H<sub>2</sub>O, nodule size, or nodule location were seen between singers and non-singers with nodules. Correlations among vowel rise time measures, anterior neck sEMG prior to vowel production, airflow, dB SPL / cm H<sub>2</sub>O, nodule location, and nodule size in all individuals with nodules (singers and non-singers) showed significant correlations between anterior neck sEMG measures, and also between airflow and nodule

size. When correlations among measures were examined separately in non-singers and singers, some differences were seen. Some of the differences in correlations between singers and non-singers indicate that there may be more efficient compensation for glottal insufficiency in singers. In singers, no significant correlation was seen between airflow and nodule size, whereas non-singers showed a high correlation between airflow and nodule size ( $R = 0.71$ ), indicating that the size of the nodule was a major factor in glottal closure.

Overall, anterior neck sEMG during vocal tasks and acoustic rise time measures did not differentiate singers or non-singers with nodules from healthy controls, indicating that neither of these objective measures show specificity for the presence of nodules in singers or non-singers. However, these objective measures could be useful for assessing inappropriate phonatory behaviors noted in some individuals with nodules. Future work should be performed to assess the utility of biofeedback based on these measures over the course of voice therapy through rehabilitation.

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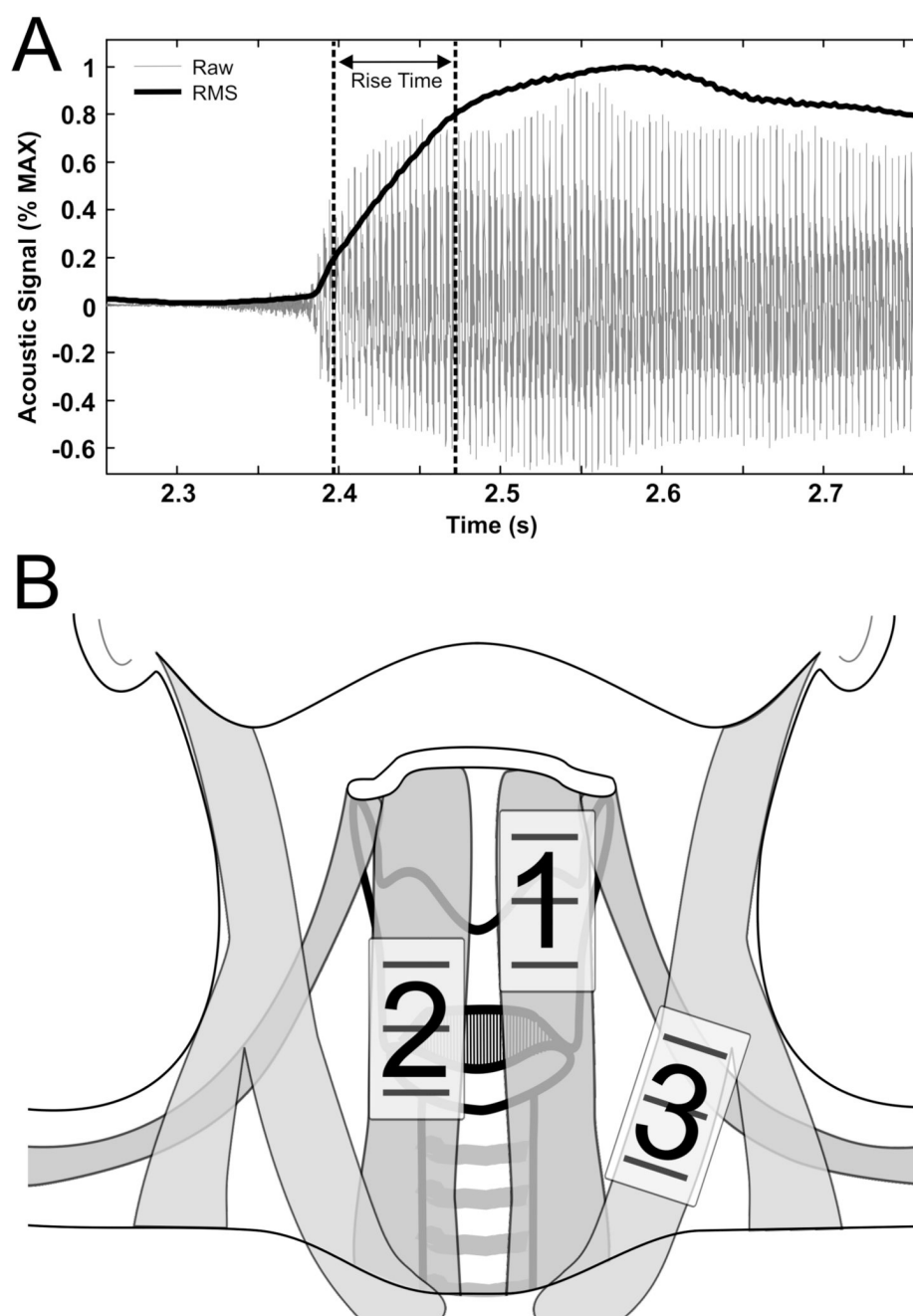
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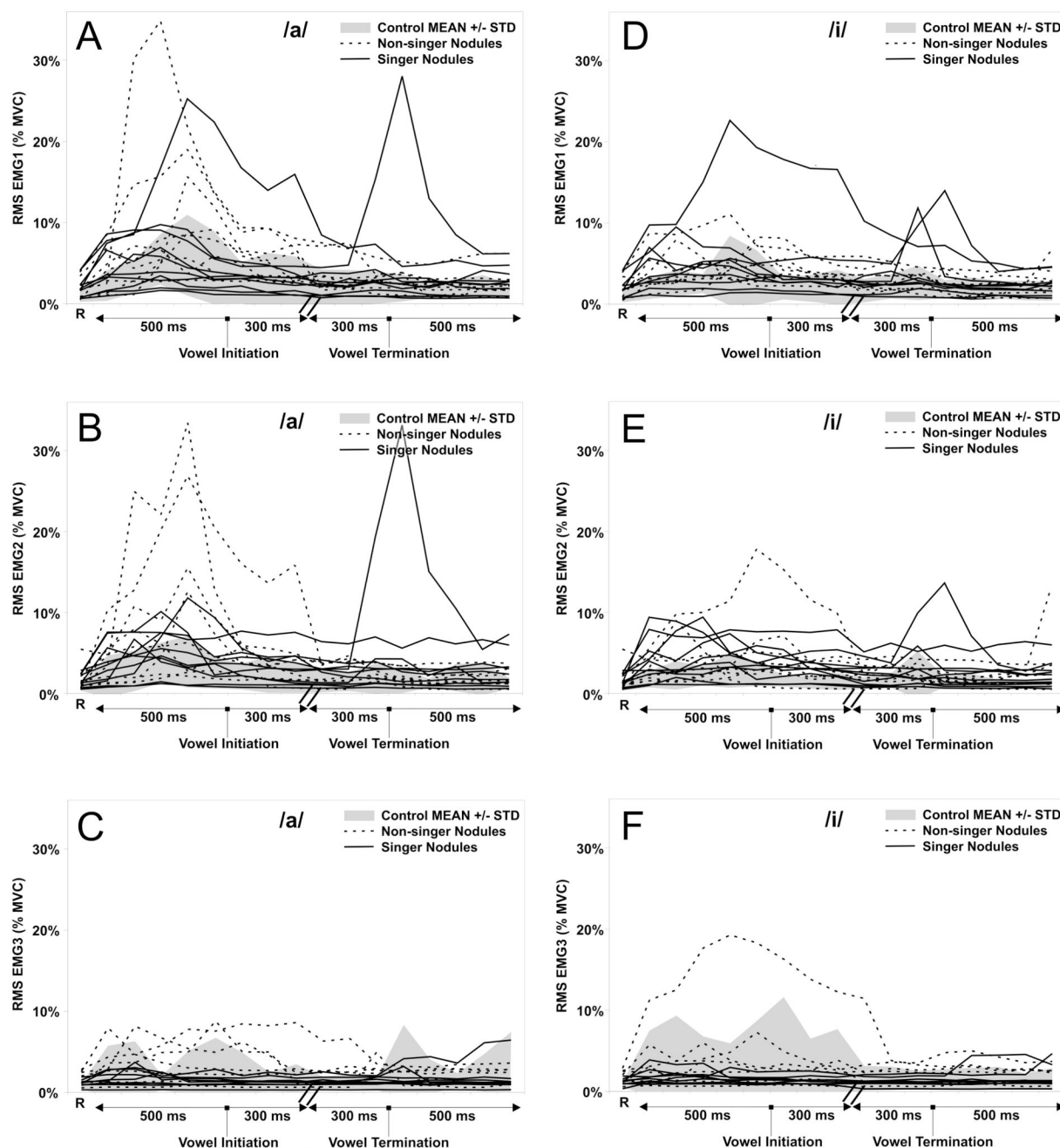
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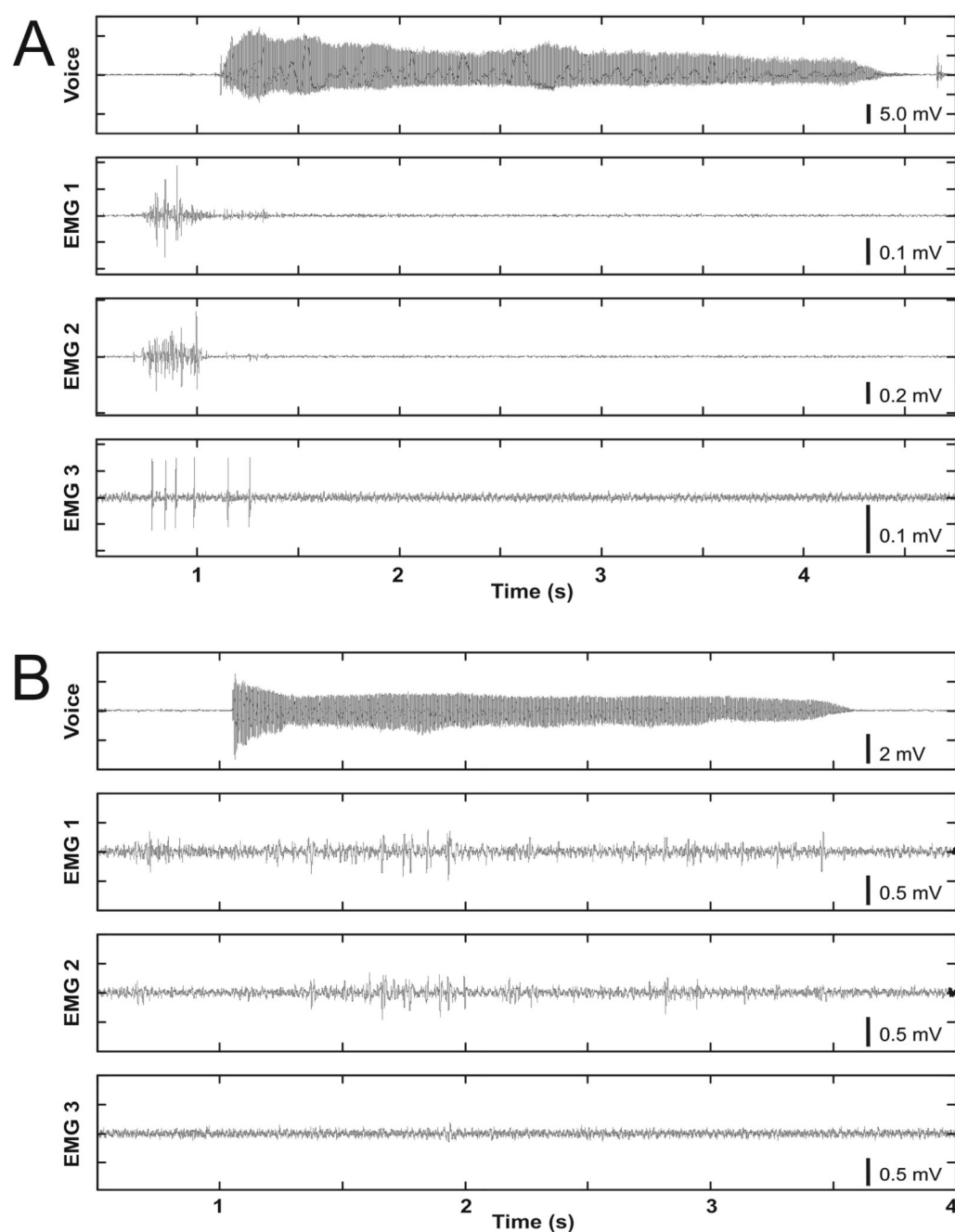
**Figure 1.**

Panel A: An example of the calculation of the acoustic rise time for a production of the vowel /a/. The thin line shows the raw acoustic signal, and the thick line shows the RMS of the acoustic signal. The acoustic signals are plotted as a function of the maximum values for clear display. Panel B: Schematic of sEMG electrode recording locations.



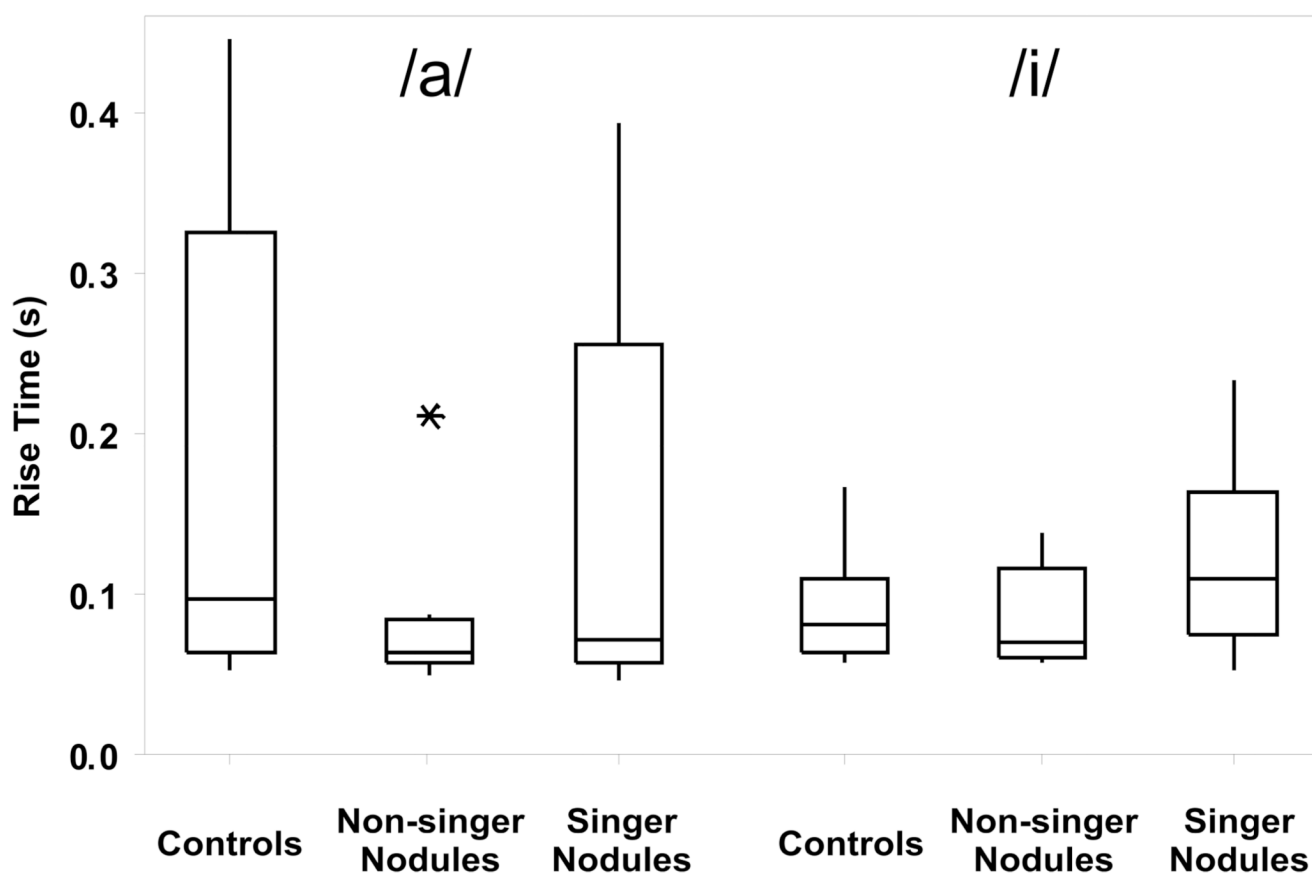
**Figure 2.**

The anterior neck sEMG recorded from positions 1, 2, and 3, averaged in 100 ms segments during the 500 ms before and 300 ms after vowel initiation, as well as the 300 ms before and 500 ms after vowel termination for the vowels /a/ and /i/. The shaded area indicates  $\pm$  one standard deviation from the mean of the participants with healthy normal voice. Individual data for individuals with nodules are shown. Singers are shown in the solid line, whereas non-singers are shown in the broken line. The initial datapoint labeled with an 'R' indicates sEMG at rest for reference.



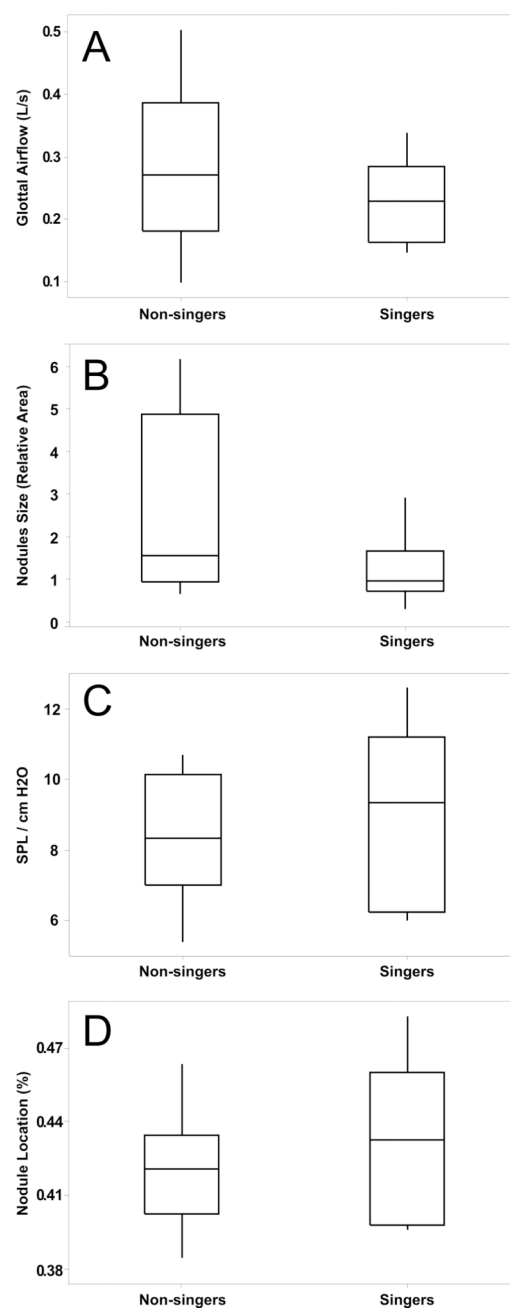
**Figure 3.**

Two examples of the raw acoustic signals and anterior neck sEMG leading up to the production of the vowel /a/. Panel A shows the signals of a non-singer with nodules. Panel B shows the signals of a participant with healthy normal voice.



**Figure 4.**

Boxplots of the acoustic rise time for vowels /a/ and /i/. Horizontal box lines indicate the lower and upper quartiles of the data, with the center line marking the data median. Vertical whiskers extend from the boxes to the minimum and maximum values of each dataset. The asterisk marks a data observation falling more than 1.5 times the interquartile range higher than the third quartile.



**Figure 5.**

Boxplots of the airflow (Panel A), nodule size (Panel B), vocal dB SPL / cm H<sub>2</sub>O (Panel C), and nodule location (Panel D) in individuals with vocal fold nodules. Horizontal box lines indicate the lower and upper quartiles of the data, with the center line marking the data median. Vertical whiskers extend from the boxes to the minimum and maximum values of each dataset. “Relative area” refers to the approximation of the nodule size, which was calculated as the mean of the two hand-marked nodule extents normalized by the mean of the length of the two vocal folds (pixels<sup>2</sup> / pixels).



Correlations between acoustic rise time and electromyographic measures in all participants (controls with healthy normal voice, singers with nodules, and non-singers with nodules). Associated *p*-values for the Pearson's correlations are shown in parentheses. The linear rise time (s) is abbreviated as *T<sub>r</sub>* during vowels /a/ and /i/.

Table 1

	<i>T<sub>r</sub></i> /a/		<i>T<sub>r</sub></i> /i/	
	EMG1	sEMG 500 msec prior to /a/ initiation	EMG1	EMG2
sEMG 500 msec prior to /a/ initiation	-0.084 (0.582)		0.331 (0.086)	-0.085 (0.668)
	EMG2		EMG3	
	-0.240 (0.218)		-0.112 (0.587)	

Table 2

Correlations between acoustic, aerodynamic, and electromyographic measures in individuals with nodules (both singers and non-singers). Associated  $p$ -values for the Pearson's correlations are shown in parentheses. Abbreviations are as follows:  $T_r$ , rise time (s) during vowels /a/ and /i/;  $E_{1-3}$ , sEMG at recording locations 1 – 3 in the 500 ms prior to vowel initiation (% MVC);  $N_{loc}$ , nodule location (% vocal fold length);  $N_{size}$ , nodule size (square pixels / pixels of vocal fold length);  $SPL/P_s$ , dB SPL / cm  $H_2O$ ; Flow, glottal airflow magnitude (L / s). Bold text highlights the parameters for which a correlation significant at the  $p \leq 0.05$  level was found.

	$T_r$ /a/	$T_r$ /i/	$E_1$ /a/	$E_1$ /i/	$E_2$ /a/	$E_2$ /i/	$E_3$ /a/	$E_3$ /i/	$N_{loc}$	$N_{size}$	$SPL/P_s$	Flow
$T_r$ /a/												
$T_r$ /i/	0.214 (0.394)											
$E_1$ /a/	-0.139 (0.582)	0.288 (0.246)										
$E_1$ /i/	-0.175 (0.487)	0.400 (0.100)	0.622 (0.006)									
$E_2$ /a/	-0.271 (0.276)	-0.034 (0.895)	0.683 (0.002)	0.183 (0.467)								
$E_2$ /i/	-0.211 (0.402)	-0.204 (0.417)	-0.028 (0.911)	0.131 (0.603)	0.506 (0.032)							
$E_3$ /a/	-0.094 (0.728)	-0.033 (0.904)	0.522 (0.038)	0.062 (0.821)	0.768 (0.001)	0.363 (0.167)						
$E_3$ /i/	-0.150 (0.578)	-0.203 (0.451)	0.328 (0.215)	0.265 (0.320)	0.288 (0.280)	0.167 (0.537)	0.697 (0.003)					
$N_{loc}$	-0.392 (0.108)	0.213 (0.395)	0.045 (0.858)	0.076 (0.766)	0.107 (0.672)	0.263 (0.291)	-0.217 (0.419)	-0.218 (0.417)				
$N_{size}$	-0.289 (0.245)	-0.105 (0.680)	-0.221 (0.379)	-0.216 (0.388)	-0.277 (0.265)	-0.247 (0.323)	-0.145 (0.592)	-0.026 (0.925)	0.108 (0.669)			
$SPL/P_s$	-0.128 (0.613)	-0.086 (0.733)	0.036 (0.887)	0.216 (0.390)	0.131 (0.606)	0.088 (0.728)	-0.013 (0.961)	0.047 (0.864)	-0.264 (0.290)	-0.439 (0.068)		
Flow	-0.043 (0.867)	0.244 (0.328)	-0.123 (0.625)	0.035 (0.891)	-0.335 (0.174)	-0.292 (0.240)	-0.373 (0.154)	-0.416 (0.109)	0.173 (0.493)	0.624 (0.006)	-0.465 (0.052)	

Table 3

Correlations between acoustic, aerodynamic, and electromyographic measures in individuals with nodules who are non-singers. Associated *p*-values for the Pearson's correlations are shown in parentheses. Abbreviations are as follows:  $T_r$ , rise time (s) during vowels /a/ and /i/;  $E_{1-3}$ , sEMG at recording locations 1 – 3 in the 500 ms prior to vowel initiation (% MVC);  $N_{loc}$ , nodule location (% vocal fold length);  $N_{size}$ , nodule size (square pixels / pixels of vocal fold length);  $SPL/P_s$ , dB SPL / cm  $H_2O$ ; Flow, glottal airflow magnitude (L / s). Bold text highlights the parameters for which a correlation significant at the  $p \leq 0.05$  level was found.

	$T_r$ /a/	$T_r$ /i/	$E_1$ /a/	$E_1$ /i/	$E_2$ /a/	$E_2$ /i/	$E_3$ /a/	$E_3$ /i/	$N_{loc}$	$N_{size}$	$SPL/P_s$	Flow
$T_r$ /a/												
$T_r$ /i/	0.441 (0.274)											
$E_1$ /a/	-0.181 (0.668)	0.372 (0.363)										
$E_1$ /i/	-0.231 (0.582)	-0.423 (0.296)	0.336 (0.416)									
$E_2$ /a/	-0.240 (0.567)	0.243 (0.562)	0.716 (0.046)	0.105 (0.805)								
$E_2$ /i/	-0.099 (0.815)	-0.299 (0.472)	-0.137 (0.746)	0.127 (0.764)	0.532 (0.174)							
$E_3$ /a/	0.078 (0.868)	0.321 (0.483)	0.637 (0.124)	0.424 (0.343)	0.758 (0.048)	0.463 (0.295)						
$E_3$ /i/	-0.099 (0.832)	-0.188 (0.687)	0.326 (0.475)	.893 (0.007)	0.127 (0.785)	0.159 (0.733)	0.617 (0.140)					
$N_{loc}$	-0.614 (0.105)	-0.147 (0.728)	0.267 (0.523)	-0.181 (0.668)	0.201 (0.633)	-0.174 (0.680)	-0.385 (0.393)	-0.359 (0.429)				
$N_{size}$	-0.185 (0.660)	0.080 (0.851)	-0.369 (0.369)	-0.160 (0.705)	-0.579 (0.132)	-0.517 (0.190)	-0.671 (0.099)	-0.285 (0.535)	0.096 (0.822)			
$SPL/P_s$	0.140 (0.741)	0.164 (0.699)	0.272 (0.515)	0.465 (0.246)	0.505 (0.202)	0.538 (0.169)	0.824 (0.023)	0.531 (0.220)	0.526 (0.181)	-0.396 (0.331)		
Flow	0.189 (0.654)	0.103 (0.807)	-0.501 (0.206)	-0.565 (0.145)	-0.607 (0.110)	-0.480 (0.229)	-0.935 (0.002)	-0.749 (0.053)	0.281 (0.499)	0.714 (0.047)	-0.786 (0.021)	

Table 4

Correlations between acoustic, aerodynamic, and electromyographic measures in individuals with nodules who are singers. Associated  $p$ -values for the Pearson's correlations are shown in parentheses. Abbreviations are as follows:  $T_r$ , rise time (s) during vowels /a/ and /i/;  $E_{1-3}$ , sEMG at recording locations 1 – 3 in the 500 ms prior to vowel initiation (% MVC);  $N_{loc}$ , nodule location (% vocal fold length);  $N_{size}$ , nodule size (square pixels / pixels of vocal fold length);  $SPL/P_s$ , dB SPL / cm  $H_2O$ ; Flow, glottal airflow magnitude (L / s). Bold text highlights the parameters for which a correlation significant at the  $p \leq 0.05$  level was found.

	$T_r$ /a/ /i/	$E_1$ /a/ /i/	$E_2$ /a/ /i/	$E_3$ /a/ /i/	$E_3$ /i/	$N_{loc}$	$N_{size}$	$SPL / P_s$	Flow
$T_r$ /a/ /i/	0.060 (0.869)								
$E_1$ /a/ /i/	-0.008 (0.983)	0.538 (0.109)							
$E_2$ /a/ /i/	-0.188 (0.603)	0.588 (0.074)	0.941 ( <b>&lt;0.001</b> )						
$E_3$ /a/ /i/	-0.277 (0.438)	0.079 (0.828)	0.591 (0.072)	0.549 (0.100)					
$E_2$ /i/	-0.306 (0.389)	-0.194 (0.591)	0.104 (0.775)	0.154 (0.671)	0.771 ( <b>0.009</b> )				
$E_3$ /a/	0.306 (0.424)	0.296 (0.440)	-0.132 (0.734)	-0.241 (0.532)	0.252 (0.513)	0.370 (0.327)			
$E_3$ /i/	0.140 (0.720)	0.057 (0.884)	-0.332 (0.382)	-0.413 (0.270)	0.074 (0.849)	0.440 (0.236)	0.859 ( <b>0.003</b> )		
$N_{loc}$	-0.500 (0.141)	0.234 (0.515)	0.031 (0.932)	0.142 (0.695)	0.453 (0.188)	0.623 (0.054)	0.255 (0.508)	0.388 (0.302)	
$N_{size}$	-0.375 (0.286)	-0.005 (0.989)	-0.455 (0.186)	-0.453 (0.189)	-0.037 (0.920)	0.311 (0.381)	0.557 (0.119)	0.783 ( <b>0.013</b> )	0.544 (0.104)
$SPL / P_s$	-0.216 (0.549)	-0.164 (0.650)	-0.038 (0.917)	0.149 (0.681)	-0.137 (0.705)	-0.238 (0.508)	-0.667 ( <b>0.050</b> )	-0.753 ( <b>0.019</b> )	-0.625 (0.053)
Flow	-0.026 (0.943)	0.752 ( <b>0.012</b> )	0.323 (0.363)	0.497 (0.143)	-0.095 (0.794)	0.002 (0.996)	0.095 (0.807)	0.095 (0.807)	-0.140 (0.699)