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# Use of Neck Strap Muscle Intermuscular Coherence as an Indicator of Vocal Hyperfunction

Cara E. Stepp, Robert E. Hillman, and James T. Heaton

**Abstract**—Intermuscular coherence in the beta band was explored as a possible indicator of vocal hyperfunction, a common condition associated with many voice disorders. Surface electromyography (sEMG) was measured from two electrodes on the anterior neck surface of 18 individuals with vocal nodules and 18 individuals with healthy normal voice. Coherence was calculated from sEMG activity gathered while participants produced both read and spontaneous speech. There was no significant effect of speech type on average coherence. Individuals with vocal nodules showed significantly lower mean coherence in the beta band (15–35 Hz) when compared to controls. Results suggest that bilateral EMG–EMG beta coherence in neck strap muscle during speech production shows promise as an indicator of vocal hyperfunction.

**Index Terms**—Motor drives, surface electromyography, vocal system.

## I. INTRODUCTION

**D**ISORDERS of the glottis (the area of the vocal folds) are often caused by or accompanied by maladaptive behaviors referred to collectively as vocal hyperfunction. Vocal hyperfunction has been defined as “conditions of abuse and/or misuse of the vocal mechanism due to excessive and/or ‘imbalanced’ muscular forces” [1], characterized by excessive laryngeal and paralaryngeal tension [2]–[6]. Despite the widespread use of the vocal hyperfunction designation, diagnosis and assessment in current clinical practice is dependent upon subjective interpretation of patient history and physical examination. There is currently no established objective measure for the detection of vocal hyperfunction.

Past attempts to develop such measures have included the investigation of acoustic and aerodynamic parameters, both individually and in combination. Although strain is likely the most

common auditory-perceptual quality attributed to vocal hyperfunction, there is no known good acoustic correlate (e.g., [7]). In healthy normal speakers, aerodynamic-acoustic measures have been shown to be correlated with categorical perceptual ratings of “pressed” voice, a voice quality modality associated with vocal hyperfunction [8]. Further, Hillman *et al.* [1] investigated aerodynamic and aerodynamic-acoustic measures (ratios of glottal resistance, vocal efficiency, and the ratio of the alternating airflow to the constant airflow through the glottis), finding that they could discriminate between individuals with different manifestations of vocal hyperfunction and individuals with healthy normal voice. However, the presence of laryngeal pathology in some of the patients studied (e.g., vocal fold nodules) causes glottal insufficiency that can impact aerodynamic measures, regardless of the presence of vocal hyperfunction, making it impossible to differentiate such effects from the separate influence of vocal hyperfunction.

Common benign organic pathologies that arise on the vocal fold surface such as vocal nodules, diffuse erythema and edema, and polyps are assumed to be related to hyperfunctional behavior (vocal hyperfunction) or phonotrauma [9]. However, in most cases seen clinically, it is unclear to what degree the organic pathology is a result of learned hyperfunctional behaviors and to what degree the vocal hyperfunction is a compensatory result of the glottal insufficiency caused by the organic pathology. Regardless, these organic pathologies are associated with vocal hyperfunction. A common manifestation of these organic pathologies is the vocal fold nodule. Clinically, a nodule is defined as a small protuberance located between the anterior and middle third of the vocal fold [2], [10]. It is a buildup of fibrotic tissue on the surface of the vocal fold. Vocal nodules usually occur in young to mid-aged females [4], [11], and also seem to be more common in young larynges [12]. Vocal hyperfunction is a common feature of vocal nodules, with one voice clinic reporting that 92% of cases of vocal nodules were coincident with vocal hyperfunction [4].

Commonly associated symptoms of vocal hyperfunction are not limited to the larynx. Many muscles in the neck that attach to the larynx and/or hyoid bone have voice and speech-related contractions due to their role in controlling the vertical position of the larynx in the neck and, to some degree, the position of the tongue. When individuals demonstrate an inappropriate degree of intrinsic laryngeal muscle contraction (hyperfunction), it is thought that they often simultaneously contract the extrinsic laryngeal muscles and other superficial neck muscles in a similar hyperfunctional or imbalanced manner [2]. However, with the exception of palpation-based measures (e.g., [13], [14]), little has been done to attempt to derive a measure of vocal hyperfunction from these known symptoms.

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Two past studies have attempted to use surface electromyography (sEMG) to objectively quantify neck muscle tension. Redenbaugh and Reich [15] measured mean neck sEMG of a single anterior neck electrode in seven individuals with healthy normal voice and seven “hyperfunctional” individuals, finding that the individuals with disordered voice had significantly greater mean normalized neck sEMG during phonation than individuals with healthy normal voice. However, the disordered population was varied in age, sex, and clinical presentation, and data collection method was relatively rudimentary; sEMG signals were amplified, filtered, and integrated in real-time, with the integrated values displayed on-screen and recorded by hand. Hocevar-Boltezar *et al.* [16] recorded sEMG from 18 pairs of differential electrodes on the face and anterior neck in 11 women with disorders associated with vocal hyperfunction (nodules, muscle tension dysphonia) with respect to five women with healthy normal voice. Although this study found significant differences between the mean sEMG of many electrode positions in the two groups, sEMG signals were not normalized. In order to reduce the variability due to neck surface electrode contact and participant neck mass, sEMG signals should be normalized to a reference contraction before they are compared among conditions and/or participants [17], a difficult task when assessing speech musculature. Both of these studies were limited to root-mean-squared (rms) analysis, without an attempt to assess the patterns of sEMG between electrode positions.

Given the high number of degrees-of-freedom involved in speech motor control and the problematic nature of appropriate amplitude normalization of sEMG data, intermuscular coherence may provide a reliable objective measure of vocal hyperfunction based on the activity of the extrinsic laryngeal muscles. Although a few studies have employed physiological coherence in the study of speech and voice [18]–[20], the measure has not been widely explored.

Although the rhythmic nature of muscle discharge has been appreciated for some time (see [21] for review), one aim of recent investigations has been to determine whether patterns of physiological drives to muscle are diagnostically relevant. Use of the coherence function has been used extensively to assess the oscillatory coupling between the central nervous system and EMG by computing coherence between EMG and magnetoencephalographic (MEG) signals (e.g., [22], [23]) and between EMG and electroencephalographic (EEG) signals (e.g., [24], [25]). Further, coherence between multiple EMG signals can be used to measure the common presynaptic drive to motor neurons [26].

The coherence function, written as  $|R_{xy}(\lambda)|^2$ , is a frequency domain measure of the linear dependency or strength of coupling between two processes—here, two time-series  $x(t)$  and  $y(t)$  as a function of the frequency  $\lambda$ . The coherence function is mathematically bounded from 0 to 1, with 0 representing independence, and 1 indicating a perfect linear relationship [27]. This function is defined by (1), as in [27]–[29], where  $d_y^T$  denotes the finite Fourier transform of the  $\ell$ th segment of length  $T$  ( $\ell = 1, \dots, L$ ) of  $y(t)$  in which the dependency on  $\ell$  has been removed by allowing  $T$  to approach infinity, and  $\text{corr}\{a, b\}$  indicates the correlation coefficient between  $a$  and  $b$

$$|R_{xy}(\lambda)|^2 = \lim_{T \rightarrow \infty} |\text{corr}\{d_x^T(\lambda), d_y^T(\lambda)\}|^2. \quad (1)$$

One commonly studied frequency band is the beta band (15–35 Hz) which is thought to originate chiefly from the primary motor cortex [21]. Beta band coherence is thought to represent transmission from the primary motor cortex to spinal motoneurons, with cortical–muscle interactions following a rough somatotopic map in the primary motor cortex [23]. Significant beta coherence has been found in trunk muscles (paraspinal and abdominal) as well as limb muscles (which have been studied more extensively), although their modulation by the CNS is weaker and may be bilateral [30]. While efferent pathways may be the primary source of beta band coherence, several studies argue for a role of sensory feedback using evidence from cooling, anesthesia, and short-term ischaemic sensory deafferentation [24], [31], [32] and from a deafferented individual [33].

Intermuscular coherence in the beta range is thought to arise from common presynaptic motoneuron drive [34], [35]. Brown *et al.* validated the idea that beta band intermuscular coherence is qualitatively similar to beta band corticomuscular coherence, testing individuals with cortical myoclonus [26]. However, unlike corticomuscular coherence, intermuscular coherence methods will reflect all oscillatory presynaptic drives to spinal motoneurons, not just those of cortical origin.

This study marks the first one of its kind in ascertaining normal bilateral EMG–EMG coherence in neck strap muscle during speech production, as well as comparing that activity between healthy normal speakers and individuals with a vocal hyperfunction. This parameter may be useful as a marker of vocal hyperfunction for use as a clinical tool.

## II. METHODS

### A. Participants

Participants were 18 adult females diagnosed with vocal fold nodules prior to any therapeutic intervention (mean age = 26.1 years, SD = 10.7 years) and 18 female volunteers with healthy normal voice (mean age = 24.2 years, SD = 3.1 years). The diagnosis of vocal fold nodules in disordered individuals was based on visual examination using digital videoendoscopy with stroboscopy by a team comprised of a laryngologist and one or more certified speech-language pathologists. None of these participants had a history of any other voice disorder (e.g., vocal fold paralysis, laryngeal cancer). The individuals with healthy normal voice were volunteers with no complaints related to their voice who had no abnormal pathology of the larynx as observed during standard digital videoendoscopy with stroboscopy. Informed consent was obtained from all participants in compliance with the institutional review board of the Massachusetts General Hospital.

### B. Tasks

Simultaneous neck sEMG and acoustic signals from a lavalier microphone (Sennheiser MKE2-P-K, Wedemark, Germany) were filtered and recorded digitally with Delsys (Boston, MA) hardware (Bagnoli Desktop System) and software (EMGworks 3.3) with a sampling frequency of 20 kHz. The EMG recordings in this study were taken in view of current European standards [36]. Participants’ necks were prepared for electrode placement by cleaning the neck surface with an alcohol pad and

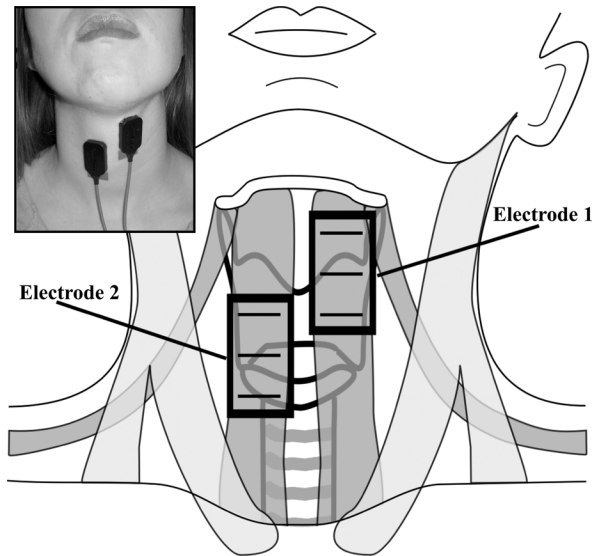


Fig. 1. Schematic of the anterior neck with the locations of double differential sEMG locations.

“peeling” with tape to reduce electrode-skin impedance, noise, dc voltages, and motion artifacts. The neck sEMG was recorded using two two-channel Bagnoli systems (Delsys Inc.) with two double differential electrodes placed parallel to the underlying muscle fibers. Double differential electrodes were utilized to increase spatial selectivity and to minimize electrical cross-talk between the two electrodes. Electrode 1 was placed superficial to fibers of the thyrohyoid, omohyoid, and sternohyoid muscles. Electrode 2 was placed on the contralateral side superficial to the cricothyroid and sternohyoid muscles; however, based on examination of the sEMG during pitch glides, it is unlikely that any activation from the (deeper) cricothyroid was detected. The Delsys 3.1 double differential surface electrodes consist of three 10-mm silver bars with inter-electrode distances of 10 mm. Electrode 1 was centered about 1 cm lateral to the neck midline, as far superior as was possible without impeding the 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. A schematic indicating the locations of these electrodes is shown in Fig. 1. A ground electrode was placed on the superior aspect of the participant’s left shoulder. The EMG recordings were preamplified and filtered using Delsys Bagnoli systems set to a gain of 1000 and a band-pass filter with roll-off frequencies of 20 and 450 Hz.

The recording procedure consisted of a brief vocal assessment of the participant including both read and spontaneous running speech. The read passage was the first paragraph of the Rainbow Passage [37]. Spontaneous speech was elicited in response to a prompt from the experimenter to describe their voice issues (in the nodule group), their travel to the facility for the experiment, their job, or a recent trip or holiday experience (e.g., “Can you tell me about your voice issues?”). Recordings were monitored in real-time for signal integrity, ensuring that no recordings included movement artifact or microphonic signals from voice production.

### C. Analysis

Audio signals were examined offline by using visual inspection and by listening to the audio signal to determine periods of speech production. Speech time for analysis was chosen manually from approximately 1 s before and after continuous speech, and avoiding nonspeech activity such as laughing or coughing. Read passages were of mean length 31 s ( $R = 26 - 54$  s), while spontaneous speech samples used for analysis were of more variable length (MEAN = 29,  $R = 8 - 93$  s).

The EMG signals were full-wave rectified and any dc offset was removed from each read and spontaneous speech sample of each participant. Coherence and phase estimates were calculated over a sliding 16 384 point ( $\sim 820$  ms) Hamming window with a 16 384 point FFT, using 50% overlap, mimicking the methods used in Halliday *et al.* [27], using custom software written in MATLAB (Mathworks Inc., Natick, MA). For each speech sample, a 5% significance level for coherence was determined based on sample length (e.g., [27]). These values were catalogued to better reference average coherence values. A two-factor ANOVA analysis of the 5% significance level was performed by group and speech task (read and spontaneous), and showed no effect of group ( $p = 0.93$ ), and a significant effect of speech task ( $p = 0.001$ ), which was not surprising given the varied lengths of the spontaneous speech samples. For read speech samples, the 5% significance levels averaged 0.040 with  $SD = 0.005$ ; spontaneous speech samples had 5% significance levels averaging 0.058 with  $SD = 0.029$ .

The mean of the rms values of sEMG collected from both electrodes was computed in 1 s windows (no overlap) using custom MATLAB (Mathworks Inc., Natick, MA) software for the entire length of the two speech tasks (read and spontaneous), as well as during a period (10–20 s) of silent resting in which no obvious sEMG activity was apparent. Examples of raw and rms signals recorded from representative control and nodule participants are shown in Fig. 2. For each electrode, the ratio between the mean rms during the two speech tasks and the mean rms during the rest period was calculated as an estimate of the signal-to-noise ratio (SNR). For each participant, the absolute difference between the SNR for each electrode was determined. Pearson’s correlations between these absolute differences in SNR and the coherence values during read and spontaneous speech to assess whether noise was a factor in differences in coherence. Further, absolute differences in SNR in the two groups were compared using Student’s *t*-tests.

Tests on the null hypothesis that the beta coherence (15–35 Hz) would be of the same level in the nodule and control groups were performed on average coherence values over the frequency range as well as  $\text{Tanh}^{-1}$  transformed values to account for the possibility of unstable variance (e.g., [29]). Statistical testing was performed by ANOVA and Student’s *t*-tests using Minitab Statistical Software (Minitab Inc., State College, PA).

## III. RESULTS

The mean coherence spectra for each group (speech task data pooled) are shown in Fig. 3. Mean coherence was relatively high over beta and low gamma (30–60 Hz) frequencies for all groups

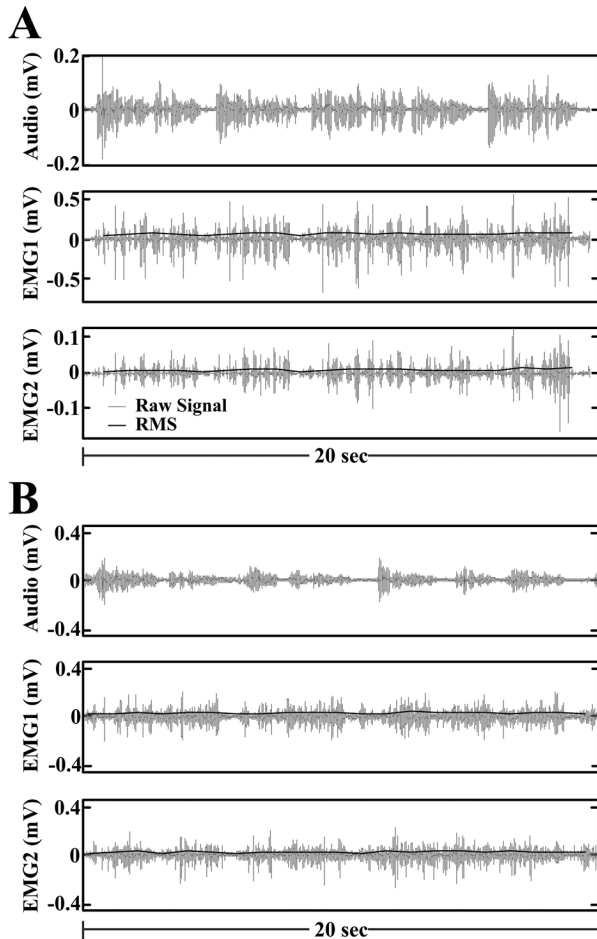


Fig. 2. Examples of raw and rms signals during 20 s of reading. Panel A displays signals recorded from a representative control participant with high mean beta coherence (0.63), whereas Panel B displays signals recorded from a representative participant with nodules with low mean beta coherence (0.03).

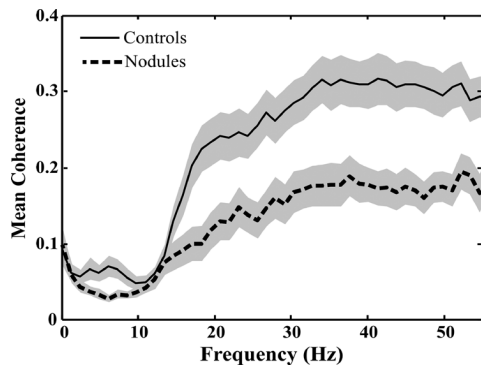


Fig. 3. Mean coherence spectra for the two participant groups. The solid black line refers to the healthy normal controls (“Controls”) and the dashed black line to the individuals with vocal nodules (“Nodules”). Grey shading indicates standard error of each group by frequency.

relative to the 5% significance values for the speech samples compared to previous reports of bilateral intermuscular coherence for speech tasks measured in respiratory muscles and the masseter [20]. No obvious peaks were seen in any frequency range. A two-factor ANOVA analysis of the average beta band coherence (15–35 Hz) by group and speech task (read and spontaneous) showed a statistically significant effect of group ( $p =$

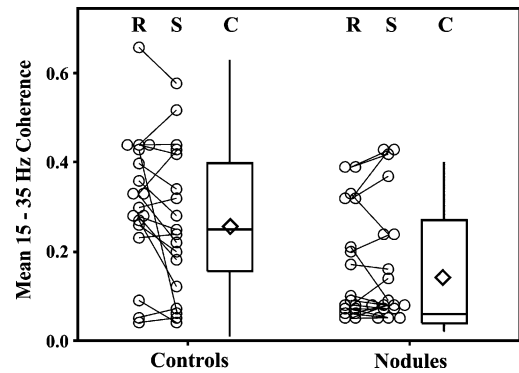


Fig. 4. Individual values and boxplots of the mean beta coherence by group. “Controls” refers to healthy normal controls and “Nodules” to individuals with vocal nodules. Individual values are shown for read speech (labeled “R”) and spontaneous speech (labeled “S”) separately. Boxplots are shown for all combined (labeled “C”) data. Horizontal box lines indicate the lower and upper quartiles of the data, with the center line marking the data median. Diamonds show the location of the data means. Vertical whiskers extend from the boxes to the minimum and maximum values of each dataset. Individual values of coherence values are shown to the left of the boxplots with circles. ANOVA found a statistically significant difference ( $p = 0.001$ ) between the mean beta coherence in the control and nodules groups ( $p_{\text{adj}} < 0.001$ ).

0.001), but no effect of speech task ( $p = 0.54$ ); an ANOVA analysis on  $\tan^{-1}$  transformed values produced nearly identical results. Mean beta coherence for nodules participants was 0.14 (SD = 0.13), whereas control participants had a mean average beta coherence of 0.26 (SD = 0.16). Individual values and boxplots of the average beta coherence by group (speech task data pooled) are shown in Fig. 4.

A Student’s  $t$ -test between the absolute differences in SNR in the nodule group relative to the control group did not show a significant difference ( $p = 0.98$ , two-sided,  $df = 19$ ). The average absolute difference in SNR was 9.8 (STD = 7.4) in the control group and 9.9 (STD = 25.7) in the nodules group. Neither the Pearson’s correlation between the absolute differences in SNR and the coherence values during read speech ( $R = 0.15$ ) nor between the absolute differences in SNR and the coherence values during spontaneous speech ( $R = 0.01$ ) were statistically significant ( $p > 0.05$ ).

#### IV. DISCUSSION

Oscillatory coupling in the beta band between the two neck strap muscle recording locations is relatively high for both read and spontaneous speech in healthy normal speakers, as measured by bilateral EMG–EMG coherence compared to bilateral intermuscular coherence measured in respiratory muscles and the masseter during speech tasks [20]. Comparison with coherence measured in nonspeech systems is difficult given the unique properties of the speech motor control system (see [38] for review). The speech tasks used for this study are representative of the typical function of the individuals, but have little in common with the simple press and hold tasks used in previous coherence studies carried out in limbs.

##### A. Bilateral EMG–EMG Beta Band Coherence is Reduced in Individuals With Vocal Nodules Relative to Healthy Controls

In contrast to the relatively high oscillatory coupling between neck strap muscle and motor cortex seen in healthy normal

speakers, individuals with vocal nodules have reduced beta band EMG–EMG coherence for both read and spontaneous speech. This reduced coherence could be a function of a difference between the two populations in electrophysiological noise, differences in measured sEMG cross-talk, or a true difference in neural bilateral coupling. The two groups did not differ significantly in terms of the absolute differences in SNR, nor were the absolute differences in SNR significantly correlated with coherence values during read speech ( $R = 0.15$ ) or spontaneous speech ( $R = 0.01$ ). These findings indicate that the significant difference in coherence between the two groups was not due to a variance in SNR. Using sEMG always carries the risk of cross-talk between electrodes (e.g., [39]), which could increase coherence values. However, all participants were recorded with double differential electrodes to significantly decrease this risk [40], [41]; furthermore, it is unlikely that individuals with healthy normal voice should be any more vulnerable to crosstalk than those with vocal nodules. Reduced coherence in many of the individuals with vocal nodules could also correspond to reduced bilateral neural coupling, which would be consistent with the clinical impression that vocal hyperfunction may be caused by excessive and/or ‘imbalanced’ muscular forces [1]. Based on the present work, the source of reduced bilateral neural coupling requires some speculation, but could implicate degraded sensory feedback, lack of cortical oversight caused by reduced attention, or overexertion.

Loss/weakness of sensory feedback leads to loss or weakness of the beta drive (e.g., [31]–[33]). It is possible that individuals with vocal hyperfunction have degraded sensory feedback. Altered or inappropriate sensory feedback might explain the habit of individuals with vocal hyperfunction to use “too much” or the wrong combination of muscle activity without noticing. In fact, modification of sensory feedback has also been used to treat therapy-resistant vocal hyperfunction. Dworkin *et al.* [42] applied topical lidocaine to three individuals causing near-immediate resolution of their functional dysphonia. To our knowledge, sensory feedback has not yet been studied systematically in individuals with vocal hyperfunction. This work provides further justification for such study.

Beta band coherence also appears to be modulated by the precision required for a task and the amount of attention used. Kristeva–Feige *et al.* saw a reduction in the beta range EEG–EMG coherences during an isometric constant force task when the task required less precision and also when a high precision task was performed while the subject divided his or her attention from the motor task by doing mental arithmetic [43]. Beta coherence also increases with visuo-motor learning, with increases not necessarily related to task performance [44]. Kristeva–Feige *et al.* postulate that beta drive increases with learning or task precision may be due to tighter cortical control. If so, this suggests that individuals with vocal hyperfunction could have reduced cortical control over their vocal anatomy.

Individuals with vocal hyperfunction may be overusing their neck strap muscles for long periods of time, resulting in general fatigue of the vocal system, which could in turn modulate intermuscular coherence. The effect of fatigue on beta coherence is still not completely clear. Tecchio *et al.* measured hand EMG–MEG coherence before and after a fatiguing motor task

in fourteen individuals, finding increased beta coherence post-fatigue [45]. However, more recently, Yang *et al.* [46] measured EMG–EEG coherence during the first and second halves of a fatiguing task in nine individuals, finding that the beta coherence was reduced during the second half of the task.

In their study of sEMG of the masseter, Smith and Denny [20] found that the coherence between right and left muscles showed a large degree of intersubject variability for speech tasks when compared to the more consistent coherence patterns during chewing and jaw clenching. Determining the clinical utility of neck strap muscle coherence in individuals with voice disorders is dependent upon more complete understanding of the possible inter- and intrasubject variability of this speech-related coherence in the healthy normal population, which could vary widely as a function of time and participant. Normative studies over days and weeks are necessary to more fully understand this promising measure.

### B. Speech Type has no Effect on Bilateral EMG–EMG Beta Band Coherence

Whether the speech sample was read or spontaneous did not affect the mean beta coherence. While Fitch [47] did not show significant differences in the average fundamental frequency of spontaneous and read speech in healthy normal participants, speech type may have an effect on speech respiration. Specifically, respiratory variables such as syllables per breath group show larger differences between healthy individuals with normal voice and individuals with vocal nodules when measured using spontaneous speech tasks than with read speech [48]. Our investigation, however, did not show a significant difference between these two speech tasks, demonstrating that the differences among groups seen in read speech were replicated in the recordings of spontaneous speech. This may indicate that the degraded coherence in the participants with vocal nodules is a resilient feature of their speech production, rather than a mere by-product of compensation techniques or attention that may differ with changes in linguistic planning between read and spontaneous speech.

### C. Summary and Indications for Future Work

This is the first work to assess normal bilateral EMG–EMG coherence in neck strap muscle during speech production, as well as to compare that activity between healthy normal speakers and individuals with a vocal nodules. In individuals with healthy normal voice, mean coherence was relatively high over in the beta band (MEAN = 0.26) for both speech types. Individuals with vocal nodules showed significantly lower mean coherence in the beta band (MEAN = 0.14) when compared to controls. There was no significant effect of speech type on average coherence. Results are consistent with previous hypotheses describing vocal hyperfunction as the use of “imbalanced” muscular forces, and suggest that bilateral EMG–EMG beta coherence in neck strap muscle during speech production shows promise as an indicator of vocal hyperfunction for use as a clinical tool.

Patients with hyperfunctionally-related disorders such as vocal nodules are typically offered voice therapy that is designed to reduce vocal hyperfunction. Future studies monitoring

bilateral EMG–EMG coherence in vocal hyperfunction patients across the course of voice therapy are needed to determine whether this measure correlates with rehabilitative outcomes. Investigations are also needed to determine the sensitivity and specificity of this measure when compared to current methods of assessment. Further scientific study is also warranted to elucidate the possible factors affecting bilateral anterior neck EMG–EMG coherence such as sensory feedback, precision, attention, and fatigue.

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