The Effects of Vibrotactile Stimulation on Spasmodic Dysphonia, A Cross-over Trial

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Abstract

Objective: To determine the effects of vibrotactile stimulation on the auditory-perceptual characteristics of patients with adductor spasmodic dysphonia (ADSD).

Study Type: Prospective cross-over trial.

Methods: Voice samples from ten patients with confirmed ADSD were collected using research quality recording equipment prior to their routine Botox treatments. The vibrotactile stimuli was provided directly to the anterior thyroid cartilage with the SIRI2 device (125 Hz) and an all-voiced consonant sentence was recorded pre-stimuli, during stimuli, and post-stimuli. These recordings were then rated using a modified CAPE-V assessment by an otolaryngologist and speech language pathologist. The raters were blinded regarding the intervention status of each sample during their analysis via a superimposed hum, mimicking the vibratory stimuli.

Results: Our patient population was majority female (60%),with a mean age of 59.5 years, and average VHI score of 21.5.

Conclusions: The pathophyisiology of ADSD is not well understood, although recent functional imaging studies suggest alterations in the primary motor and somatosensory cortex may be involved. Our results show mechano-vibratory stimulation resulting in a overall changes in level of dysphonia, which although may not be applicable in terms of treatment, may aid in diagnosis and understanding the pathophysiology of ADSD.

Introduction

Spasmodic dysphonia (SD) is a focal dystonia that produces involuntary contractions in laryngeal muscles during speech[1,2]. The most common form of SD, adductor SD, is characterized by a strained, strangled voice quality, frequent breaks in phonation, and highly effortful speech production.

Treatment options for this disorder are limited, and few novel treatments have been proposed since the introduction of botulinum toxin injections in the 1980's. Although effective in improving voice symptoms for those with adductor SD[3], many patients opt out of this treatment due to negative side effects, the invasive nature of vocal fold injections, and the need for re-injection several times a year.

The pathophysiology of SD is not well understood, but structural and functional neuroimaging studies suggest multiple neural regions that differ in patients with SD compared to controls. Functional MRI (fMRI) studies that have included both symptomatic and asymptomatic speech have shown altered activation in primary motor and somatosensory cortex for patients with SD relative to controls, with increased or decreased activation in SD patients reported. In support of sensory system involvement, neural activity in the primary somatosensory cortex is positively correlated with voice breaks in adductor SD.

Abnormal processing of sensory information and deficient integration of sensory and motor information has been proposed as one mechanism for motor abnormalities that occur in SD[6]. Altered sensory feedback at the peripheral laryngeal level may impact cortical activation patterns, and may account for differences in somatosensory activation that occur in patients with SD.

Methods and Materials

Inclusion Criteria: Patients with Adductor Type Spasmodic Dysphonia +/- Muscle Tension Dysphonia (MTD), greater than 5 years of Botox treatment

Study Design: Patients act as their own controls, A Cross over Trial

Intervention: Application of a vibrotactile device (SIRI2 at 125 Hz) to the anterior thyroid cartilage for 1 minute

Data collection just before 3 month Botox(TM) injection schedule

Pre-intervention VHI-10
Practice all-voiced (Rainy Island Ave) sentence 8 times

1. Record all-voiced sentence x2 with superimposed hum, no stimulation
2. Record all-voiced sentence x2 with superimposed hum, w/ stimulation
3. Record all-voiced sentence x2 with superimposed hum, post stimulation

A laryngologist and SLP with over 25 years experience then perform auditory-perceptual analysis using a modified CAPE-V tool to compare the stimuli and post-stimuli groups to the pre-stimuli group, in terms of dysphonia severity level

Table 1. Change in Dysphonia, During and After Stimulation

% Improve During Intervention = 75%	**Mean Improvement = 13%**
%Unchanged During Intervention = 0%	
%Worsened During Intervention = 25%	**Mean Deterioration = 30.5%**
% Improve Post Intervention (vs Pre) = 44.44%	**Mean Improvement = 14.25%**
%Unchanged Post Intervention = 0%	
%Worsened Post Intervention = 55.56%	**Mean Deterioration = 12%**

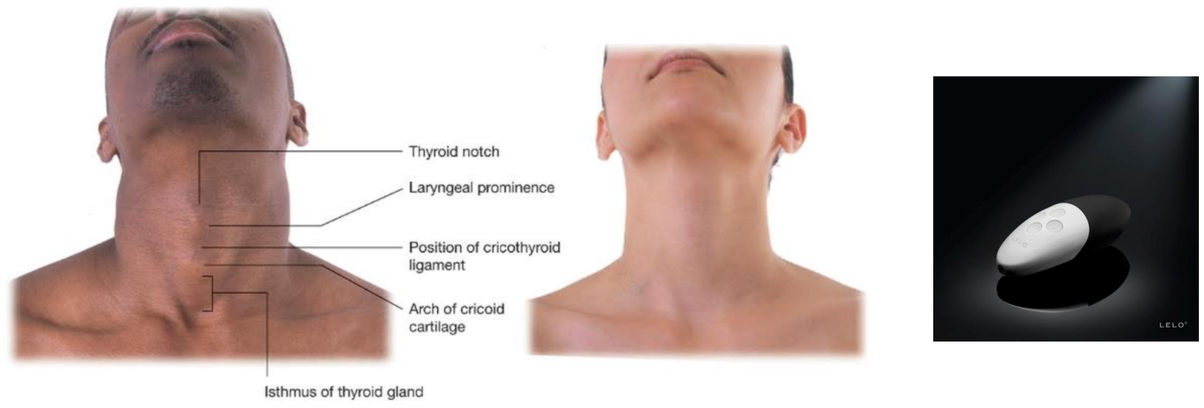

Figure 1. SIRI2 vibrotactile device with constant 125Hz application at highest power was applied to the anterior thyroid cartilage (just lateral to the laryngeal prominence) for 1 minute as the intervention.

Discussion and Conclusions

The majority of patients (75%) improved during the intervention, however, by a small margin, only 13% on average. A sizable portion (25%) however did worsen and by a much larger margin (30.5%) on the modified CAPE-V scale. The post intervention data is more equivocal with 44.44% improving by 14.25% and 55.56% worsening by 12%.

The next step in teasing out the patients who benefit from those who do not would be to see if there are any predisposing factors within each distinct group (patients who got worse, patients who got better).

The amount of MT compensation in each patient may play role in how spasmodic dysphonia patients respond to tactile stimuli. Vocal fry may be another factor that we may be able to use in the future to further categorize how patients with spasmodic dysphonia react to these stimuli. Whether or not this can be used as a therapeutic tool or as a diagnostic one, further studies and analysis is necessary.

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