Abstract

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

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 were 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 pathophysiology 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 not 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.

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 SD. Changes in mechanosensory transmission may account for differences in somatosensory activation that occur in patients with SD.

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 worsened 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.

References