DYSPHONIA DUE TO ISOLATED CRICOThYROID MUSCLE DYSTONIA: A CASE REPORT AND REVIEW OF LITERATURE

Shannon Kraft MD1, Jana Childes CCC-SLP1, Allen Hillel MD2 and Joshua Schindler MD1

1Oregon Health and Sciences University, Department of Otolaryngology, Portland, OR
2University of Washington, Department of Otolaryngology, Seattle, WA

Abstract

Purpose: We report a case of laryngeal dystonia resulting from isolated cricothyroid (CT) muscle dysfunction.

Methods: We discuss the pertinent clinical features, strobscopic, laryngeal function studies, and electromyography (EMG) findings of the case, and review the literature.

Summary: The patient, a 43 year old otherwise healthy female, presented with 8 months of progressive hoarseness. Her symptoms were particularly bothersome while dictating. At her initial exam, her voice was rated Grade 3, Roughness 3, Breathlessness 1, Asthenia 0, Strain 3. (G3R3B1A0S3). EMG has demonstrated that the lateral cricoarytenoid (LA) muscle had an increased latency of 750 msec with increased activity throughout the task.

Conclusions: EMG can be a useful adjunct in the diagnosis of dystonia that persists despite adequate trials of speech therapy. To our knowledge, this is the only report of laryngeal dystonia due to isolated cricothyroid dysfunction.

Background

Spasmodic dysphonia (SD) is characterized by sustained or repetitive involuntary muscle contractions of intrinsic laryngeal muscles during voicing.1 Patients with SD are evaluated and divided into clinical subtypes based upon perceptual voice analysis. Treatment with botulinum toxin is directed toward the predominant muscle responsible for the clinical subtype.2 Adductor spasmodic dysphonia (ADSD) accounts for about 85% of patients and is treated by thyroarytenoid (TA) injection. Patients with abductor spasmodic dysphonia (ADSD) are treated with posterior cricoarytenoid (PCA) injection.4

Although perceived clinically as ADSD or ADSD, electromyography (EMG) suggests SD is a disorder of mixed adductor and abductor dysfunction (MDX SD) with a predominant phenotype.4 EMG has demonstrated that the lateral cricoarytenoid (LCA) and interarytenoid (IA) can also contribute to ADSD.5 In patients where the IA was determined to be overactive, treatment of the IA in addition to the TA resulted in improved symptom control.6 The authors concluded that EMG could be used to generate a “road map” by which to design a plan for targeted therapy for patients with refractory SD.

Case Report

• An otherwise healthy 43 year old female physician with 8 months of progressive dysphonia
• Worse on phone when dictating
• Better with whispering
• Normal videostroboscopy
• Failed trial of voice rest and steroids
• Failed multiple trials of voice therapy
• No pathology at time of microlyngoscopy

Figure 1A. Patient glides from low pitch to high pitch using a sustained “eee.” CT recruitment at higher pitches confirms wire placement in the CT.

Figure 1B. Sustained “eee.” The TA and LCA demonstrate normal latencies of approximately 400 msec. CT latency is 750 msec, nearly twice the normal value.

Table 1. Comparison of acoustic parameters before and after botulinum toxin injection into the CT muscle.

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<th>VHI</th>
<th>MPT</th>
<th>F0 (Hz)</th>
<th>Fmin (Hz)</th>
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<td>212</td>
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Figure 2. Fine wire EMG. Channel 1 = Voice, Channel 3 = TA, Channel 5 = LCA and Channel 7 = CT.

Figure 3. Fine wire EMG. Channel 1 = Voice, Channel 3 = TA, Channel 5 = LCA and Channel 7 = CT.

Conclusions

EMG can be a useful tool in identifying the specific muscle(s) contributing to the vocal pathology when other methods of evaluation and treatment have failed. EMG allowed us to generate a “road map” of aberrant muscle activity and to tailor a treatment plan for what we believe to be the first reported case of isolated cricothyroid dystonia successfully treated with botulinum toxin. CT dystonia should be considered in dysphonia characterized by elevated pitch that does not respond to voice therapy.