Accent method of voice therapy for treatment of severe muscle tension dysphonia

Khalid H. Malki, MD, PhD, Nasser H. Nasser, MD, PhD, Sabah M. Hassan, MD, PhD, Mohammed Farahat, MD, MSc.

Nonorganic dysphonia may present a challenging diagnosis, and management. Here, we present a severe form of nonorganic dysphonia, which we termed as arytenoidal dysphonia. It was a severe form of muscle tension dysphonia, which was described earlier in literature although with different nomenclature. The outcome of the accent method of voice therapy was also presented. We concluded that accent method of voice therapy is proven to be an effective treatment modality of arytenoidal dystonia.


From the Communication and Swallowing Disorder Unit (Malki, Nasser, Hassan), Oto-Rhino-Laryngology Department, King Abdulaziz University Hospital, Riyadh, Kingdom of Saudi Arabia and the Oto-Rhino-Laryngology Department (Farahat), Ain Shams University, Cairo, Egypt.

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Address correspondence and reprint request to: Dr. Khalid H. Malki, Consultant and Assistant Professor of Phoniatrics, Communication and Swallowing Disorders Unit, Oto-Rhino-Laryngology Department, King Abdulaziz University Hospital, PO Box 7805, Riyadh, Kingdom of Saudi Arabia. Tel. +966 (1) 4786100 Ext. 5203. Fax. +966 (1) 4775682. E-mail: kalmalki@ksu.edu.sa

Case Reports

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N onorganic dysphonia refers to impairment of voice production in the absence of mucosal or neurogenic disease of the larynx. The diagnosis is confounded by inconsistent nomenclature, and lack of uniform diagnostic classification systems. The list of labels includes: functional dysphonia, muscle misuse dysphonia, and muscle tension dysphonia. Laryngeal postures observed in nonorganic dysphonia have different classification systems. Such postures were described by Van Lawrence, Koufman and Blalock, Morrison and Rammage, and Rosen and Murry. The aim of this article was to present a severe form of nonorganic dysphonia (muscle tension dysphonia), in which there was a severe anterior-posterior supraglottic compression during phonation. The patient was using the redundant arytenoidal mucosa for phonation. We termed this severe form of muscle tension dysphonia as arytenoidal dysphonia, as compared to ventricular dysphonia. The outcome of the accent method of voice therapy is presented.

Case Report. A 14-year-old Saudi female presented to our Communication and Swallowing Disorders Unit, Ear-Nose and Throat Department at King Abdulaziz University Hospital, Riyadh, Kingdom of Saudi Arabia, with a markedly low-pitched voice, severe dysphonia, and a history of abnormal voice since early childhood. There was a history of unintentional high-pitched voice, which could be produced occasionally by the patient especially during singing. There was no history of dyspnea, dysphagia, diabetes mellitus, surgical intervention, laryngeal trauma, or psychiatric disturbances. Voice handicap index (VHI), as rated by the patient, was 43 on presentation. This indicates a moderate vocal handicap. The auditory perceptual assessment (APA) revealed, that the overall grade of dysphonia was 3 (severe) with irregular quality. Telescopic laryngostroboscopic examination using digital video laryngoscope (Model RLS 9100B, Kay Elemetrics Corp., Lincoln Park, NJ, USA) revealed that the true vocal folds appeared only during respiration (true vocal fold abduction). They were pearly white in color, with mild mucosal congestion (Figure 1). The true vocal folds showed small bilateral vocal fold nodules. During phonation, the true vocal folds could not be seen. She was using her redundant arytenoidal mucosa to produce the voice by contacting it against the posterior surface of the epiglottis, with appearance of mucosal waves on
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Figure 1 - Laryngeal video stroboscopic image at presentation. During breathing, early bilateral vocal fold nodules and bilateral shallow sulcus vocalis type 2 are noticed.

Figure 2 - Laryngeal video stroboscopic image at presentation. During phonation, the patient is using her redundant arytenoid mucosa to produce the voice by contacting it against the posterior surface of the epiglottis, with appearance of mucosal waves.

Figure 3 - Laryngeal video stroboscopic image after 6 sessions of voice therapy. During phonation, the patient is now using her true vocal folds to produce the voice.

the redundant arytenoidal mucosa (Figure 2). Acoustic analysis was performed using Multi-dimensional voice program (MDVP Model 4305) (Kay Elemetrics Corp., Lincoln Park, NJ, USA) installed into computerized speech lab (CSL model 4300, Kay Elemetrics Corp., Lincoln Park, NJ, USA). The sampling rate was set to 50,000 Hz. In a quiet room, she was asked to sustain the vowel /α/ for 4 seconds at a comfortable pitch and loudness after she was instructed to clear her throat. A dynamic microphone (Shure prologue 14 H) was positioned at a mouth-to-microphone distance of 20 cm. A 3-second mid-vowel segment was selected and analyzed. The voice signal was considered adequate if it was free from overloads (red signals on the screen), and audible variations in pitch and loudness. The following acoustic parameters were then obtained: average fundamental frequency (Fo), absolute jitter (Jita), jitter percent (jitt), relative average perturbation (RAP), pitch perturbation quotient (PPQ), fundamental frequency variation (vFo), shimmer in dB (ShdB), shimmer percent (Shim), amplitude perturbation quotient (APQ), smoothed amplitude perturbation quotient (sAPQ), noise-to-harmonic ratio (NHR), and voice turbulence index (VTI). The results of the acoustic analysis are represented in Table 1. She was managed by the accent method of voice therapy after elicitation of the high-pitched voice that the patient was occasionally producing. Voice therapy was performed by one of the authors. We believe that this high-pitched voice sets the true vocal folds into vibration, and eliminates the vibrations of the arytenoidal mucosa. The newly acquired voice was stabilized by the accent method of voice therapy. The sessions were given twice weekly, and each lasted for 30 minutes. She was instructed to use her newly acquired voice as much as possible. The first post-therapy assessment was carried out after 6 sessions of voice therapy. All the assessment tools mentioned earlier were repeated. Voice handicap index was rated by the patient as 20, which indicates a minimal

Table 1 - Acoustic (MDVP) variables.

<table>
<thead>
<tr>
<th>MDVP parameters</th>
<th>Pre-therapy evaluation</th>
<th>1st post-therapy evaluation</th>
<th>2nd post-therapy evaluation</th>
<th>3rd post-therapy evaluation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fo, Hz</td>
<td>101.01</td>
<td>329.31</td>
<td>297.09</td>
<td>272.27</td>
</tr>
<tr>
<td>Jita, µs</td>
<td>592.21</td>
<td>107.13</td>
<td>52.12</td>
<td>89.96</td>
</tr>
<tr>
<td>Jitt, %</td>
<td>5.96</td>
<td>3.52</td>
<td>1.55</td>
<td>2.45</td>
</tr>
<tr>
<td>RAP, %</td>
<td>3.38</td>
<td>2.13</td>
<td>0.95</td>
<td>1.48</td>
</tr>
<tr>
<td>PPQ, %</td>
<td>3.87</td>
<td>2.09</td>
<td>0.90</td>
<td>1.44</td>
</tr>
<tr>
<td>sPPQ, %</td>
<td>4.83</td>
<td>2.08</td>
<td>0.94</td>
<td>1.46</td>
</tr>
<tr>
<td>vFo, %</td>
<td>6.40</td>
<td>3.32</td>
<td>1.67</td>
<td>2.84</td>
</tr>
<tr>
<td>ShdB</td>
<td>1.06</td>
<td>0.45</td>
<td>0.38</td>
<td>0.43</td>
</tr>
<tr>
<td>Shim, %</td>
<td>12.16</td>
<td>5.19</td>
<td>4.33</td>
<td>4.91</td>
</tr>
<tr>
<td>APQ, %</td>
<td>8.43</td>
<td>3.86</td>
<td>3.19</td>
<td>3.36</td>
</tr>
<tr>
<td>sAPQ, %</td>
<td>10.50</td>
<td>5.17</td>
<td>4.35</td>
<td>4.94</td>
</tr>
<tr>
<td>NHR</td>
<td>0.32</td>
<td>0.12</td>
<td>0.14</td>
<td>0.19</td>
</tr>
<tr>
<td>VTI</td>
<td>0.24</td>
<td>0.06</td>
<td>0.06</td>
<td>0.07</td>
</tr>
</tbody>
</table>

MDVP - multi-dimensional voice program, Fo - average fundamental frequency, Hz - Hertz, µs - millisecond, Jita - absolute jitter, Jitt - Jitter percent, RAP - relative average perturbation, PPQ - pitch perturbation quotient, sPPQ - smoothed pitch perturbation quotient, vFo - fundamental frequency variation, ShdB - shimmer in dB, Shim - shimmer percent, APQ - amplitude perturbation quotient, sAPQ - smoothed amplitude perturbation quotient, NHR - noise-to-harmonic ratio, VTI - voice turbulence index.
vocal handicap. At this stage, she was using her true vocal folds during phonation, as documented by video laryngo stroboscopy (Figure 3), with the appearance of weak, slightly asymmetrical, and slightly aperiodic stroboscopic mucosal waves. After the tenth session, a second post-therapy assessment was carried out, with re-application of all assessment tools. The post-therapy and acoustic analysis showed that Fo had increased. All other acoustic variables decreased after the tenth session of voice therapy. On her third evaluation one year after presentation, all acoustic variables, except Fo, were slightly worsened (Table 1).

**Discussion.** The pathophysiology of nonorganic dysphonia is uncertain. It was hypothesized that it could be due to incomplete relaxation of the posterior cricoarytenoid muscle, excessive activity of intrinsic and extrinsic laryngeal muscles during phonation, in-coordination of respiratory effort with vocal fold tension, or it could be due to a maladaptive compensatory laryngeal strategy as a result of glottal pathology. There is a growing body of evidence that some laryngeal postures attributed to nonorganic dysphonia, may not be signs of abnormal phonatory function. They can be considered as normal variants in some normal subjects, especially during connected speech. The presented patient was using her redundant arytenoidal mucosa for phonation. The arytenoidal mucosa has a large mass, and reduced elasticity. This can explain the irregular character, and the low pitch of her voice. It is not clear why she used her arytenoidal mucosa to produce phonation. We propose that this was the result of inefficient true vocal fold vibration at a certain stage during her early childhood. Transient true vocal fold paralysis, or vocal misuse or abuse might lead to supraglottic hyperfunction, which might induce the use of arytenoidal mucosa as an alternative vibrator. However, the use of arytenoidal mucosa for phonation might also have developed as a habit during early childhood, and then maintained for reasons of secondary gain, although there was no history of clear emotional, or psychiatric disturbances. The use of arytenoidal mucosa, and not the ventricular folds still needs an explanation.

The associated bilateral vocal fold nodules augment the assumption of vocal trauma as a predisposing factor for the development of the proposed compensatory supraglottic hyperfunction in the pathogenesis of arytenoidal dysphonia. Occasional high-pitched voicing in the presented patient can be attributed to the patient’s need to use her true vocal folds. This was one of the reasons why voice therapy used in her management started by training her to phonate with high-pitched voice, aimed at eliminating the use of the arytenoidal mucosa for phonation. The newly acquired voice with its higher-than-normal pitch was then stabilized by the accent method of voice therapy. This type of voice therapy has a holistic approach for management of voice disorders. It entails 2 main tasks: 1) voice hygiene advice, and 2) correction of faulty vocal technique. It entails an integration of abdomino-diaphragmatic breathing, accentuated rhythmic vowel play, phonation, and later articulation. It allows restoration of balance between expiratory phonatory airflow excitor, and the adjustment of the muscles of the vocal folds vibrator. In spite of limited efficacy studies, the accent method was used for this case as it is postulated that it allows restoration of the physiologic balance, and timing between airflow, sound pressure level, and laryngeal muscle contraction, thus allowing emergence of a comfortable pitch. The Fo had increased from 101 Hz pre-therapy to 329 Hz after the sixth session of voice therapy. This increase is attributed to the use of the normal vibrator (true vocal folds). After the tenth session, Fo had decreased slightly to 297 Hertz, which is still slightly higher than her normal age-and-gender-matched pitch. Pitch perturbation and amplitude perturbation variables, in addition to noise-related variables, improved after voice therapy. Acoustic variables of voice are expected to improve when true vocal folds, and not other abnormal vibrators are used for phonation.

Although she was still using her true vocal folds, it was noticed that all acoustic variables of our patient’s voice, except Fo, were slightly worsened on her third evaluation one year after presentation (Table 1). Since she was not compliant to the use of her newly acquired voice at home, and as she was still abusing her voice, this slight deterioration was expected. Such findings emphasize the importance of compliance to voice therapy techniques in nonorganic dysphonia.

**References**


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