Laryngology Seminar
Ventricular Dysphonia

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Introduction
1. Sataloff RT, ventricular dysphonia (Vd), synonymous with dysphonia plica ventricularis, defined as "phonation using false vocal fold vibration rather than true vocal fold vibration, most commonly associated with severe muscular tension and occasionally may be an appropriate compensation for profound true vocal fold dysfunction."
2. The estimated incidence of hoarseness following ventricular phonation is 4%.

Symptoms
1. A low-pitched voice is typical because the false folds contain more mass.
2. A hoarse, harsh, rattling, rumbling, cracking voice has been mentioned.
3. The false vocal folds are not as sophisticated in their vibration as the true vocal bands, causing an unpleasant-sounding voice.
4. There can be a restricted vocal range characterized by reduced loudness, little pitch variability, and monotonous voice.
5. Voice breaks and diplophonia (a double sounding voice pattern resulting from simultaneous action of true and false vocal cords) can also be associated with Vd.
6. In addition, voice fatigue and aphonia have been mentioned.
7. Furthermore, dysphagia, odynophagia, stridor, and dyspnea have not been reported.
8. Somatic symptoms may also appear, such as effortful phonation and fatigability, unproductive throat clearing, and pain or lump in the throat.
Etiology
1. Depending on the cause, some authors have referred to two groups, as follows:
   1) Vd as a consequence of excessive muscular tension in the laryngeal area
   2) Vd as a substitute voice resulting from severe true vocal fold disease
2. Dworkin and Meleca (1997), Stemple et al. (1995), and Andrews (1995) have reported two similar kinds of Vd, as follows:
   1) Vd as a substitute for glottal incompetence
   2) Vd as a functional result of psychoemotional interference and physical and emotional tension
3. Arnold and Pinto (1960), and Pinho et al. (1999) differentiated the following types of Vd caused by several somatic and psychological circumstances:
   1) habitual, resulting from hyperkinetic dysphonia with continuous vocal abuse;
   2) stress-induced (through hyperkinesis of the ventricular folds);
   3) compensatory as a reaction to laryngeal paralysis;
   4) a cerebral type signaling dysarthria (with a low, rough, and squeezed sound);
   5) a cerebellar or midbrain (eg, parkinsonism) type;
   6) vicarious as a desirable adjustment to defective true vocal folds.
4. Previous vocal cord surgery was most commonly related with Vd, followed by surgical intubation, unilateral true vocal cord paralysis, and irritant exposure.
5. Other precipitating medical conditions are vocal abuse, spastic dysphonia, respiratory disease, and neck or chest surgery.

Pathophysiology
1. After intubation, the inflated cuff was placed just under the true vocal folds, resulting in a compression injury to the more medial thyroarytenoid nerve fibers to the vocalis muscle, sparing the more distal nerve fibers to the false vocal cords.
2. This may have caused true vocal cord weakness and compensatory false vocal fold hyperadduction during phonation.
3. Kendall and Leonard (1997) thought that, next to the involvement of the thyroarytenoid muscle, the lateral cricoarytenoid muscle is also accountable for the sphincteric contraction of the false vocal cords.

4. Normally, the ventricular folds adduct with the arytenoid cartilages and assist in laryngeal closure. However, they do not participate in normal voice production.

5. The constantly increased ventricular function and repetitive closure may lead to new functional and anatomical changes in the interior of the larynx (such as ventricular hypertrophy) and, possibly, to a new system of innervation.

**Diagnosis**

1. Besides noticing the symptoms, perceptual evaluation of the sound of the voice, and a complete history, laryngeal examination is indispensable in diagnosing Vd.

2. In the case of hypertrophied false vocal folds, satisfactory visualization of the true vocal folds is often impossible.

3. Videostroboscopy is important to reveal deficient true vocal fold oscillation and can record actual ventricular fold adduction and vibration.

4. Fiberoptic nasopharyngoscopy can also be applied for diagnosing ventricular phonation, showing the ventricular fold action during connected speech.

5. Aerodynamic analysis showed abnormally high subglottic pressure and high translaryngeal airflow (indicating a glottal air leak).

6. The maximum phonation time as measured was short.

7. Acoustic analysis revealed severe acoustic perturbation.

8. Quantification of the restricted vocal range can be useful.

**Therapy**

A therapeutic management scheme regarding Vd.
**Pharmacological Therapy**

**Anesthetic injection.**

1. In an effort to suppress the ventricular movement, lidocaine (an anesthetic agent) was injected.
2. Laryngoscopically, normal ventricular folds occur without hyperactivity.
3. In cases of ventricular phonation caused by true vocal fold disease, this experimental approach should not be considered.

**Botulinum toxin.**

2. After treatment all patients showed a relaxed supraglottis in voicing.
3. All patients persisted in true vocal fold vibration without recurrence of Vd.
4. Supraglottic botulinum toxin injection may be an accessory to voice therapy,
   1) can improve primary vocal fold disease identification,
   2) may allow restoration of normal voicing.

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**False Vocal Fold Surgery**

1. Excision of the false vocal folds can be considered in cases with irreversible ventricular hypertrophy and normal true vocal fold function.
2. Using a CO2 laser for excision of redundant mucosa and connective tissue of the false vocal folds, (Feinstein et al. 1987) having the advantages of minimal edema, scar formation, and destruction of tissue.
3. It is important that the patient has normally functioning true vocal cords.
4. Kosokovic et al. suggested that the therapy of Vd is based on the degree of false vocal fold hypertrophy.

1) The first stage involves inflammatory alterations with soft and elastic hypertrophy at the anterior one-third of the fold. This condition is reversible with voice therapy.
2) The second stage implicates hypertrophy of the entire length of the ventricular fold. This is still reversible with voice therapy but microsurgical excision of the hypertrophied folds results in a more rapid improvement.
3) The third stage involves irreversible histological changes. There is fibrosis of the ventricular folds making them inelastic. Extirpating these folds is recommended.

References