

Ventricular Dysphonia: A Case of False Vocal Fold Mucosal Traveling Wave

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(Editorial Comment: The observation of a symmetrical traveling wave on the false vocal cord has not been previously reported.)

Ventricular dysphonia, also known as dysphonia plicae ventricularis, is a disorder of speech in which the ventricular folds (false vocal folds, FVFs) participate pathologically in phonation.^{1,2} Although the first anatomic illustrations of the false folds were done in 1775 by Santorini,³ their role in phonation was not completely understood. In 1860, 6 years after the introduction of the laryngeal mirror by Garcia, J.N. Czermak was the first to recognize that involvement of the false folds in phonation is a pathological phenomenon.¹

The ventricular folds are composed of soft and elastic connective tissue containing fat cells with scattered muscle fibers from the thyroarytenoideus (TA) muscle.² They are lined by pseudostratified ciliated columnar epithelium and tubuloacinar glands.^{4,5} Under normal circumstances, these folds adduct with the arytenoids and assist in laryngeal closure. However, they do not participate in normal voice production.¹ In some pathological states, the false folds adduct before the true vocal folds (TVFs) and hence hamper normal voice production. This pattern is caused by either underactivity of the TVFs or overactivity of the FVFs.² However, in some other reports, it has been stated that during laryngeal examination of a patient with ventricular phonation, the FVFs adduct either immediately before or after the adduction of TVFs.⁶

The vocal quality in ventricular dysphonia has been described as a rough, weak, breathy,

low-pitched rattling with frequent voice breaks. In some cases, the patient may produce diplophonia in which both the TVFs and the FVFs are simultaneously involved in phonation. Some patients with ventricular dysphonia complain of voice deterioration over time. Others state that they constantly try to clear their throat. The history is usually negative for any symptoms of dyspnea or stridor. In laryngeal examination, the FVFs are seen to adduct during phonation. In some cases, hypertrophy of the ventricular bands is also observed.^{4,6}

Jackson and Jackson² estimated the incidence of dysphonia caused by ventricular phonation to be approximately 4%. Other investigators reported it to be approximately 1% of the general population.^{7,8} Therefore, ventricular phonation is therefore a relatively common phenomenon but easily overlooked by otolaryngologists. According to Saunders,⁴ this is because the etiology of ventricular phonation is unclear and it is not always present during laryngeal examination.

Despite the discovery of ventricular dysphonia a century and a half ago, this disorder remains poorly understood. Many investigators have proposed classification systems, possible etiologies, and therapies for this disorder. Jackson and Jackson² separated ventricular dysphonia into two types: vicarious, which usually results from an intrinsic laryngeal disease; and usurpative, which is secondary to vocal abuse. Compensation by the ventricular folds is a desirable effect in the vicarious type, whereas it is problematic in the usurpative type.⁶

Arnold and Pinto¹ described six different forms of this disorder: habitual, emotional, paralytic, cerebral, cerebellar, and vicarious. The habitual form may be caused by vocal abuse. The emotional type may result from an emotional crisis, sometimes as a response to

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psychotherapy. In the paralytic form, the ventricular folds replace the function of the paralyzed true folds. Sometimes the vocal deficit may result from cerebral disease. Similar deficits are also observed in cerebellar lesions. Lastly, in the vicarious type ventricular phonation substitutes for the defective vocal folds.

Appropriate management and therapy is based on correctly identifying and treating the underlying cause of the ventricular dysphonia. Some of the therapies used in the past include voice therapy, voice rest, psychotherapy, medications, and surgery.⁶ Kosokovic et al⁸ recommend therapy based on a histological staging system of the hypertrophied ventricular bands. The first stage consists of reversible inflammatory changes. The hypertrophy of the ventricular fold is soft and elastic, and involves the anterior one third of the fold. Voice therapy is relatively successful for this stage. In stage II, the false folds are involved throughout their length. This stage may also be reversible and voice therapy may be successful, but Kosokovic et al⁸ recommend microsurgical excision of the hypertrophied folds for better and quicker results. The histological changes in the third stage are irreversible, because fibrosis of the bands renders them inelastic. The ventricular bands in this stage do not respond to conservative voice therapy, so surgical excision is recommended for stage III. Success also has been reported with CO₂ laser surgery for microsurgical excision of Kosokovic's stage III ventricular fold hypertrophy.⁵

Von Doersten et al⁶ proposed a therapy based on the vicarious model in which the ventricular dysphonia results from an intrinsic laryngeal disease. If the underlying cause is identified, it can be corrected in some cases. The investigators reported successful results that included thyroplasty for fold paralysis and medical therapy for reflux laryngitis. Botulinum toxin injection was used in patients with ventricular dysphonia as a result of recurrent laryngeal nerve section for spasmodic dysphonia. Von Doersten et al further recommend a conservative course of speech therapy in cases where an underlying cause cannot be corrected or identified.

This report describes a patient with ventricular dysphonia apparently occurring as a compensation for glottal insufficiency. Laryngovideostroboscopy (LVS) showed ventricular

fold adduction and vibration during phonation. The unique feature of this case was the presence of mucosal traveling waves on the FVFs during certain segments of phonation, a phenomenon not previously reported. This patient later underwent bilateral thyroplasty type I for the treatment of his glottal insufficiency and ventricular dysphonia.

CASE REPORT

A 47-year-old man required ventilation by endotracheal tube for about 3 weeks after a successful orthotopic liver transplant surgery and afterwards complained of aphonia which persisted for 2 months. He began voice therapy, but his voice remained rough and breathy with little variability in loudness. He complained of a chronic cough, which was controlled with codeine. His medical history was also significant for gastroesophageal reflux for which he took omeprazole, an H⁺/K⁺ ATPase inhibitor.

On stroboscopic examination, prominent ventricular fold adduction and vibration were observed during phonation (Fig 1). The ventricular adduction made it difficult to assess true fold mucosal wave characteristics. During some segments of phonation, the FVFs displayed a bilaterally symmetrical mucosal wave motion. At other times, the folds vibrated asymmetrically. Glottal closure was absent during most portions of phonation. Bilateral bowing of the middle segments of the TVFs was observed when the FVFs were sufficiently abducted. However, the arytenoid processes moved normally during inspiration and phonation. No mass lesions were apparent on the vocal folds.

The patient's voice was rough and mildly strained during continuous speech, which was reflected in abnormal acoustic values. His pitch was lower than normal for an adult male. Aerodynamic analysis showed abnormally high subglottic pressure and high translaryngeal airflow (Table 1).

He underwent a bilateral medialization thyroplasty type I under local anesthesia. A week after the operation, his TVFs appeared medialized, although he still had some ventricular phonation. A month later, the patient was phonating with the true folds. His voice remained slightly hoarse, but he was satisfied with his speech.

Three months later, the patient underwent repeat LVS, acoustic, and aerodynamic analyses. LVS showed TVF phonatory adduction with the presence of a normal mucosal wave (Fig 2). The patient was still able to phonate with the ventricular folds at a lower than normal pitch level on command. The subglottic pressure, although still abnormally high, reduced from 19.2 to 11.8 cm H₂O. The airflow increased slightly from 0.404 to 0.468 L/s. Postoperative laryngeal airway resistance was lower (Table 1).

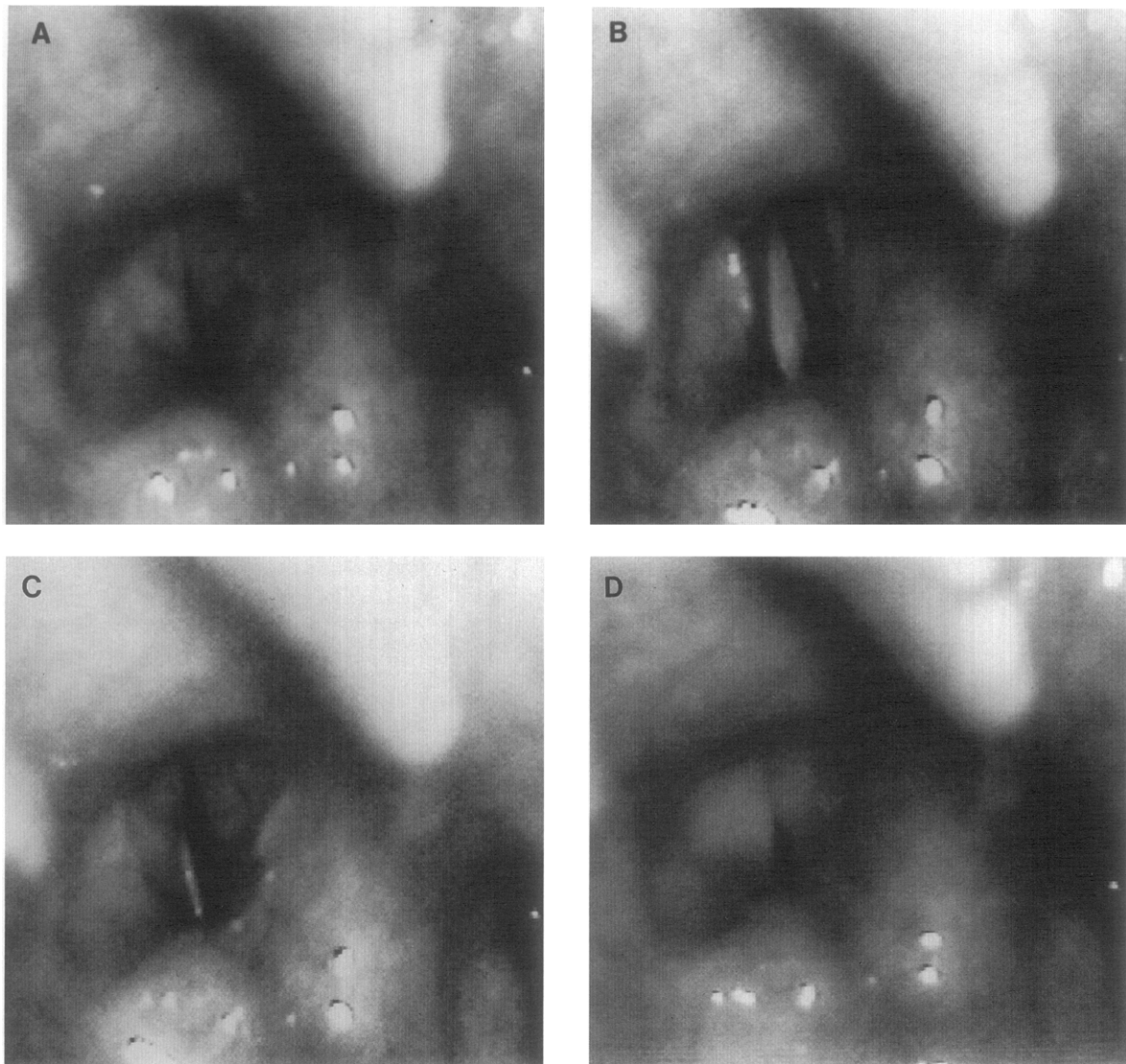


Fig 1. Video frames during a full glottal cycle illustrating opening and closing of the false vocal folds. (A) Beginning of the opening phase. (B) False folds fully open. One can see the true folds that are partly open. (C) False folds halfway closed. (D) False folds almost completely closed.

DISCUSSION

In this case, preoperative stroboscopy showed not only ventricular fold adduction and vibration during phonation, but also a

prominent mucosal wave bilaterally on the false folds, traveling from the most medial to the most lateral aspect of the folds. As noted, a traveling mucosal wave on the false folds has not been described previously.

TABLE 1. Aerodynamic Evaluation

Measure	Preoperative	Postoperative	Normal Range
SGP (cm H ₂ O)	19.2	11.8	5.5-8.0
Flow (LPS)	0.404	0.468	0.11-0.20
Resistance (cm H ₂ O/LPS)	47.5	25.2	40-50

NOTE. Normal range includes 95% confidence interval. Abbreviations: LPS, liters per second; SGP, subglottic pressure.

The patient's acoustic and aerodynamic evaluations gave clues about the mechanism of his dysphonia. Subglottic pressure and trans-laryngeal airflow were both higher than normal. The high airflow indicated an air leak at the glottis, typical of glottal insufficiency. An elevated subglottic pressure was required to set the TVFs in vibration; hence, the presence of an elevated subglottic pressure. In all likelihood, the patient's dysphonia resulted from compensatory hyperadduction of FVFs second-

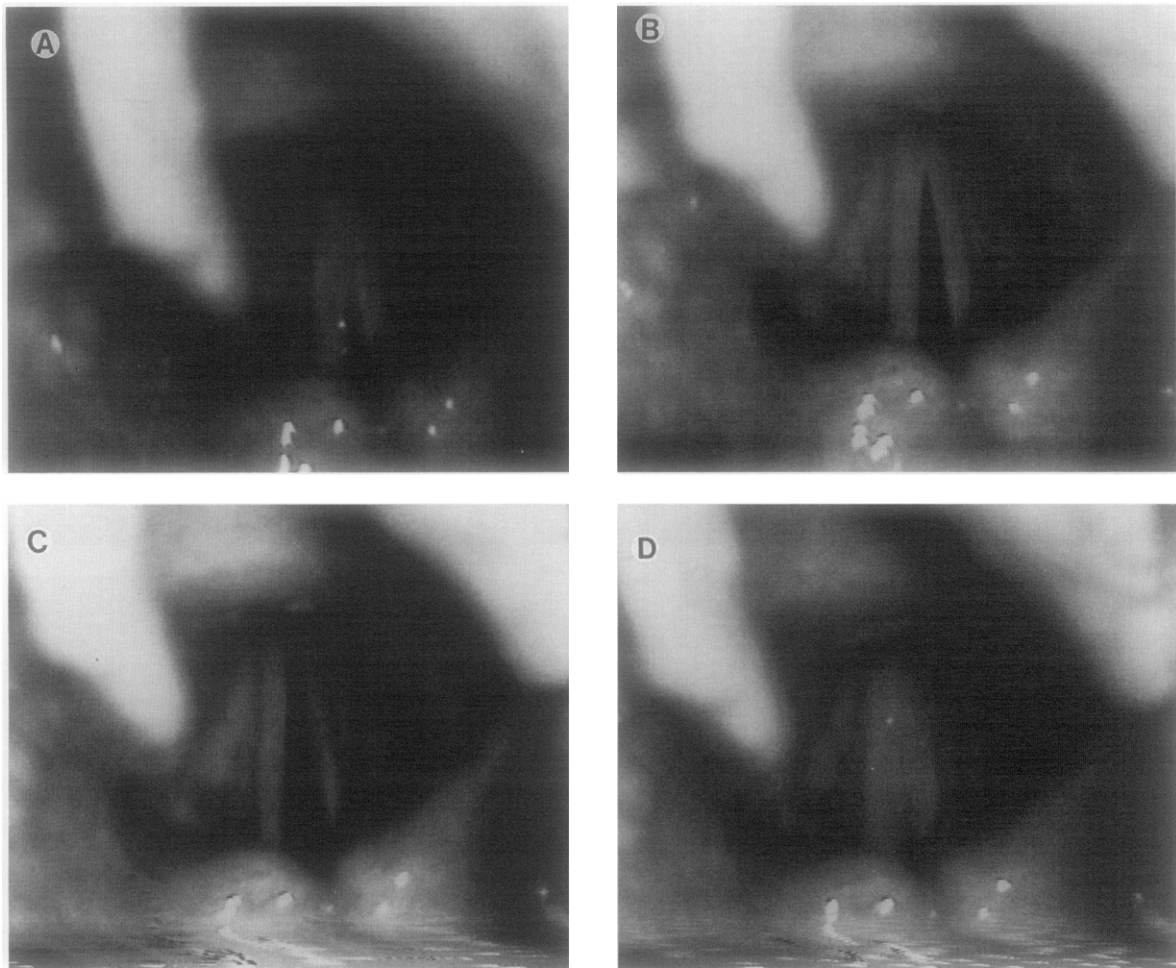


Fig 2. Video frames during a full glottal cycle in the same patient following bilateral thyroplasty type I. (A) True vocal folds beginning to open. (B) True vocal folds fully open. (C) True vocal folds beginning to close with the lower edges approximating. (D) True folds almost fully closed.

ary to an intubation-related glottal insufficiency.

Both otolaryngologists and anesthesiologists are familiar with the complications of intubation. Unilateral and bilateral vocal fold paralysis have been reported following compression injury to the anterior ramus of the recurrent laryngeal nerve from high intubation.^{9,10} The false folds contain some scattered TA muscle fibers. It is our belief that the motor innervation to the TA fibers in the FVFs also originates from the thyroarytenoid nerve branch of the anterior division of the recurrent laryngeal nerve (Fig 3).

Conceivably, high intubation in this patient placed the inflated cuff of the endotracheal tube just inferior to the TVFs. This resulted in a compression injury to the more medial TA nerve fibers to the vocalis, sparing the more distal motor fibers to the FVFs. This may have

caused weakness of the TA fibers in the true fold area and midfold bowing, with sparing of the FVF nerve supply. The false fold compensatory hyperadduction during phonation, and

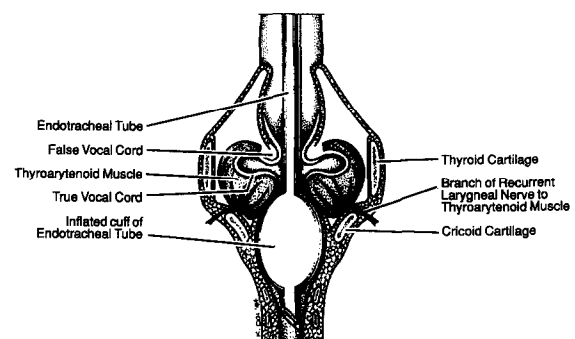


Fig 3. Drawing of a midcoronal section of the glottis with an endotracheal tube in place illustrating the possible mechanism of injury to true fold branches of the thyroarytenoid nerve sparing the branches to the false vocal folds.

hence patient's ventricular dysphonia, could be explained in this way.

The relationship between the traveling wave and the underlying properties of the vocal folds has been emphasized in many previous works. Hirano's body-cover theory^{11,12} divides the true folds into two layers with varying rheological properties. The cover, consisting of squamous epithelium and superficial and intermediate layers of lamina propria, is very pliable and can propagate a wave, but it has no contractile properties. The body, consisting of the deep layer of the lamina propria and the vocalis muscle, contributes to vocal fold stiffness by active contraction.

The finding of a traveling wave in the FVFs is quite rare. According to Hirano's theory, both cover and body components are necessary for a traveling wave to propagate. It is conceivable that the bulk of TA fibers is variable in FVFs. Perhaps it is unusual for this bulk to be substantial enough to contribute adequately to internal stiffness of the fold for a mucosal traveling wave to occur. A second possible reason for this observation of a wave in the FVFs is that the presence of both false and true vocal fold vibration in ventricular dysphonia may make it difficult for the laryngostroboscope to detect the fundamental frequency of the FVFs. As a result, no traveling wave had been found in the past.

A bilateral medialization thyroplasty type I was performed to enhance the possibility of contact between the true folds and allow entrainment. Postoperative improvement of vocal function was reflected in phonatory adduction and vibration of the TVFs, presence of a mucosal traveling wave and decreased subglottic pressure. There was a small leak present at the posterior aspect of the glottis, which was reflected in the slightly increased airflow. The airway resistance, calculated as the ratio of subglottic pressure to airflow, decreased postoperatively. However, it would be inappropriate to compare the preoperative and postoperative laryngeal resistance in this case. Preoperatively, the resistance was derived from

the aerodynamic parameters of ventricular phonation, whereas postoperatively, it was calculated from TVF vibration. This is an example of how resistance may not be the best factor in comparing laryngeal function pretreatment and posttreatment.

The treatment in this patient was designed to decrease the glottal insufficiency resulting from intubation. He was first treated with a conservative course of speech therapy with little improvement. Bilateral type I medialization thyroplasty was eventually performed with excellent subjective and objective results; the patient experienced immediate improvement from surgery in vocal quality, his aerodynamic values normalized, and the TVFs displayed adduction and vibration with bilaterally symmetric mucosal traveling wave.

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