ARYTENOID ADDUCTION IN VOCAL FOLD PARALYSIS

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Medialization laryngoplasty with Silastic (MLS) has become the mainstay of treatment for patients with unilateral vocal fold paralysis. However, a significant number of these patients undergoing MLS fail to achieve near normal voice quality, because of the fact that medialization alone often fails to adduct the arytenoid. Without adduction of the arytenoid, the vocal fold fails to achieve the physiologic phonating position. In this subset of patients, the addition of arytenoid adduction to MLS results in a significantly stronger voice and less vocal fatigue. This discussion based on over 125 patients undergoing arytenoid adduction (AA), emphasizes the surgical technique of performing AA in conjunction with MLS.

The introduction of laryngeal framework surgery by Isshiki et al in the 1970s has dramatically altered the management of the patient with vocal fold paralysis.[1] Silastic medialization laryngoplasty (SML) has been shown to improve voice fundamental frequency, increase voice intensity, and lengthen maximal phonation time.[2-8] Because SML has potential reversibility and adjustability, it has replaced teflon injection as the primary modality of therapy in many surgeons' hands.[2-4,6,7] In addition, SML seems to preserve the relationship between the vocal fold cover and the underlying body, thus preserving the mucosal wave.[5] More recently, the addition of arytenoid adduction (AA) has been shown to further improve voice outcomes in patients with vocal fold paralysis.[6,9,10] First described by Isshiki et al in 1978, AA has emerged as a crucial adjunct to SML.[9] AA allows the surgeon to place the arytenoid cartilage into a physiological phonating position.

This article will describe our evolving approach to the use of AA in conjunction with SML to rehabilitate patients with unilateral vocal fold paralysis.

INDICATIONS/CONTRAINDICATIONS

The traditional indication for AA has been a persistent posterior glottal gap as seen in patients with high vagal injuries. Initially, AA was used in patients who failed SML or in patients with large posterior glottal gaps. As our experience with AA increased, we became convinced that it added a significant functional and qualitative difference to voice outcome. We now routinely expose the muscular process of the arytenoid cartilage as a component of our laryngeal framework surgery to assess the benefit of AA in conjunction with SML.

Relative contraindications to AA include advanced chronic obstructive pulmonary disease and paresis of the contralateral vocal fold. The posterior portion of the glottis is the ventilating portion and as such a decrease in this region may significantly increase laryngeal resistance and produce dyspnea in selected patients.

PREOPERATIVE ASSESSMENT

All patients undergo a complete head and neck examination including flexible laryngoscopy and are seen in conjunction with a speech pathologist. This evaluation includes videostroboscopy and stroboscopy. Objective testing includes a battery of perceptual, acoustic, and aerodynamic studies including mean flow rate, maximum phonation time, and a voice desirability score.

The evaluation of idiopathic vocal fold paralysis includes imaging the entire course of the vagus nerve with either a computed tomographic or magnetic resonance imaging scan.

SURGICAL TECHNIQUE

The AA in conjunction with SML is usually performed under local anesthesia with intravenous sedation as previously described.[2,3,10] A flexible fiberoptic laryngoscope is positioned to provide an optimal view of the glottis on a monitor.

An incision is outlined over the midportion of the thyroid cartilage and subplatysmal flaps are elevated to the hyoid bone and the cricoid cartilage. The soft tissue is dissected in the midline, elevating the ipsilateral sternohyoid muscle off the underlying thyrohyoid muscle. Approximately 1 cm of the sternohyoid is divided from the hyoid bone and resutured at the completion of the procedure (Fig 1). An incision through the perichondrium of the anterior thyroid cartilage allows the elevation of the perichondrium and strap muscles back to the posterior border of the thyroid cartilage (Fig 2). The lower border of the thyroid cartilage is exposed to allow identification of the muscular tubercle with the cricothyroid muscle attachment.

As described in previous studies, a 6 × 13-mm window is outlined on the cartilage 5 to 7 mm back from the anterior commissure while preserving a 3-mm strut of thyroid cartilage inferior to the window.[2,3] The cartilage

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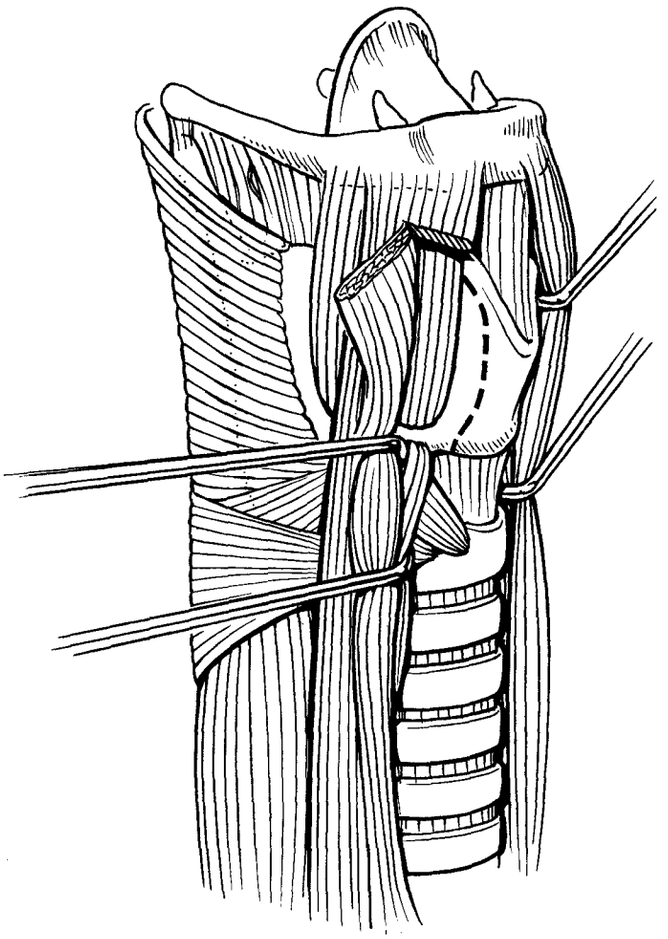

FIGURE 1. One to two centimeters of the sternohyoid muscle is divided away from its attachment on the hyoid bone to facilitate exposure of the posterior border of the thyroid cartilage.

window is removed with a small cutting burr and the inner perichondrium is incised at the superior, inferior, and posterior margins of the window. Elevation in the plane between the inner perichondrium and the thyroarytenoid fascia is then performed for approximately 1 cm inferior and superior to the window and posteriorly to the posterior border of the thyroid cartilage (Fig 3A). With the thyroarytenoid muscle and fascia elevated from the inner thyroid cartilage, the depth gauge may used to medialize the vocal fold and assess the voice.

To access the arytenoid cartilage, a skin hook is placed on the posterior border of the thyroid ala to rotate the larynx and facilitate posterior exposure. The previously elevated thyroid perichondrium is divided at the posterior border continuing the dissection deep to the medial side of the cartilage to elevate the pyriform sinus away from the thyroid cartilage (Fig 3B). At this point a 5-mm Kerrison rongeur is used to remove a window of cartilage from the posterior thyroid ala to visualize the paraglottic space lateral to the muscular process of the arytenoid (Fig 4). The cartilage is removed until the muscular process of the arytenoid can be palpated. The pyriform sinus is distended by having the patient blow against pursed lips. The anterior extension of the pyriform is then dissected away to expose the posterior cricoarytenoid muscle (Fig 5). The muscular process of the arytenoid can be palpated and grasped to move it along its plane of abduction and adduction while observing the monitor.

A double armed 4-0 prolene suture is then passed through the lateral edge of the muscular process in a figure eight fashion to secure the arytenoid (Fig 6). The needles are then passed through the previously dissected paraglottic space into the window. One end of the suture is passed through the cartilage at the anterior border of the window. If this area is calcified then a small hole is drilled with a 1-mm wire pass burr. The second needle is passed from the window below the lower cartilage strut then through the cricothyroid membrane soft tissue in the midline. The goal of the AA suture is to mimic the pull of the thyroarytenoid muscle. This concept of creating a vector of force that rotates the arytenoid medial-inferior is critical to understanding how the AA suture mimics the thyroarytenoid muscle and puts the arytenoid into a physiological phonating position. It is vital that the vector of pull not be altered by the implant or soft tissue in the paraglottic space (Fig 7). The larynx is then allowed to fall back into its anatomical position, and the arytenoid is slowly adducted and abducted by gentle traction on the AA stitch to show arytenoid movement on the monitor.

The depth gauge is used to medialize the vocal fold and assess the voice (Fig 8). Once the size of the implant is measured the silastic is carved to the appropriate size. The implant is inserted into the window taking care to keep the

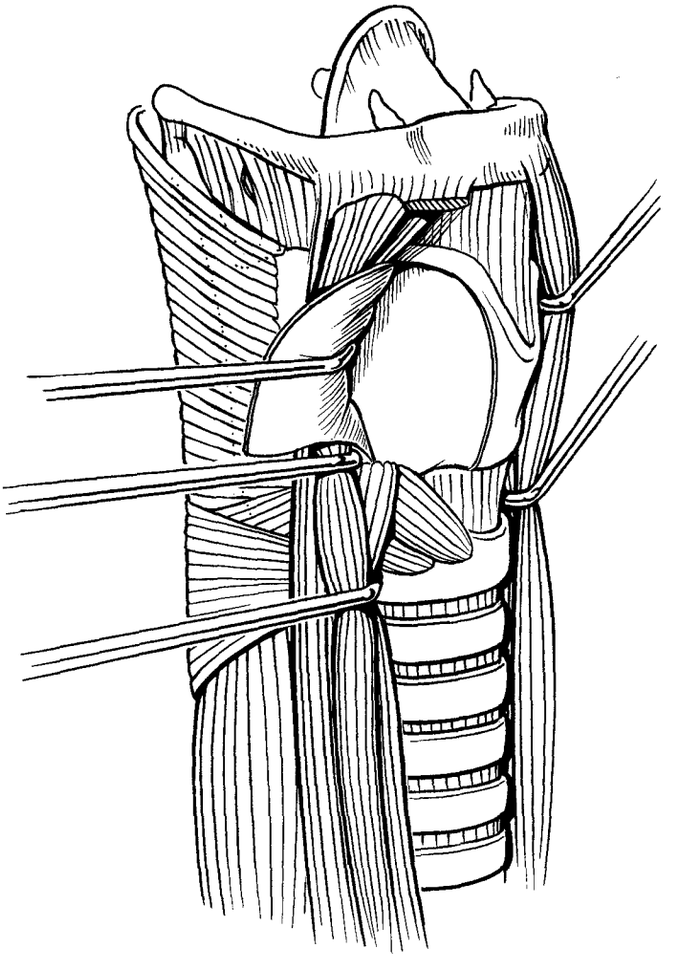

FIGURE 2. Subperichondrial dissection is used to elevate the overlying strap muscles and the perichondrium until the posterior border of the thyroid cartilage is seen from a few millimeters above the cricothyroid joint up to the superior cornu.

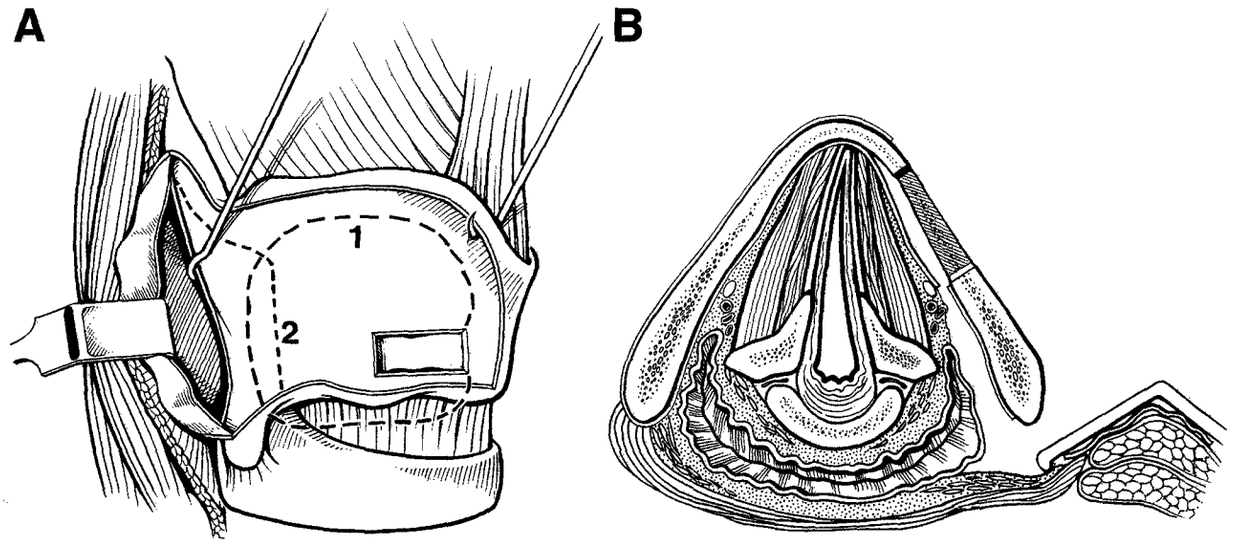

FIGURE 3. (A) The perichondrium is divided along the posterior border of the thyroid cartilage preventing elevation of the inner surface perichondrium. 1. The extent of the elevation of the thyroarytenoid muscle fascia medial to the inner perichondrium through the window. 2. The extent of the elevation of the pyriform sinus and the posterior thyroarytenoid muscle from the posterior view. (B) The final elevation as seen from an axial view showing the yet to be elevated pyriform sinus overlying the muscular process of the arytenoid cartilage.

AA stitches medial to the implant to prevent the sutures from catching on the edge of the implant and changing the vector of pull on the arytenoid (Fig 9). The AA sutures are then lightly pulled to show any further improvement in voice as well as closure of the posterior commissure. The prolene sutures are then tied down to secure the AA. Very little tension is needed to adduct the arytenoid with this technique.

During the course of the AA procedure, great care is taken to prevent injury to the pyriform sinus mucosa. After completion of the procedure, the perichondrium is approximated in the midline. The sternohyoid muscle is reattached to the hyoid bone and the strap muscles are loosely closed. A ⅛ inch closed suction drain is placed and the wound is closed in layers. All patients receive perioperative antibiotics as well as 24 hours of decadron steroids.

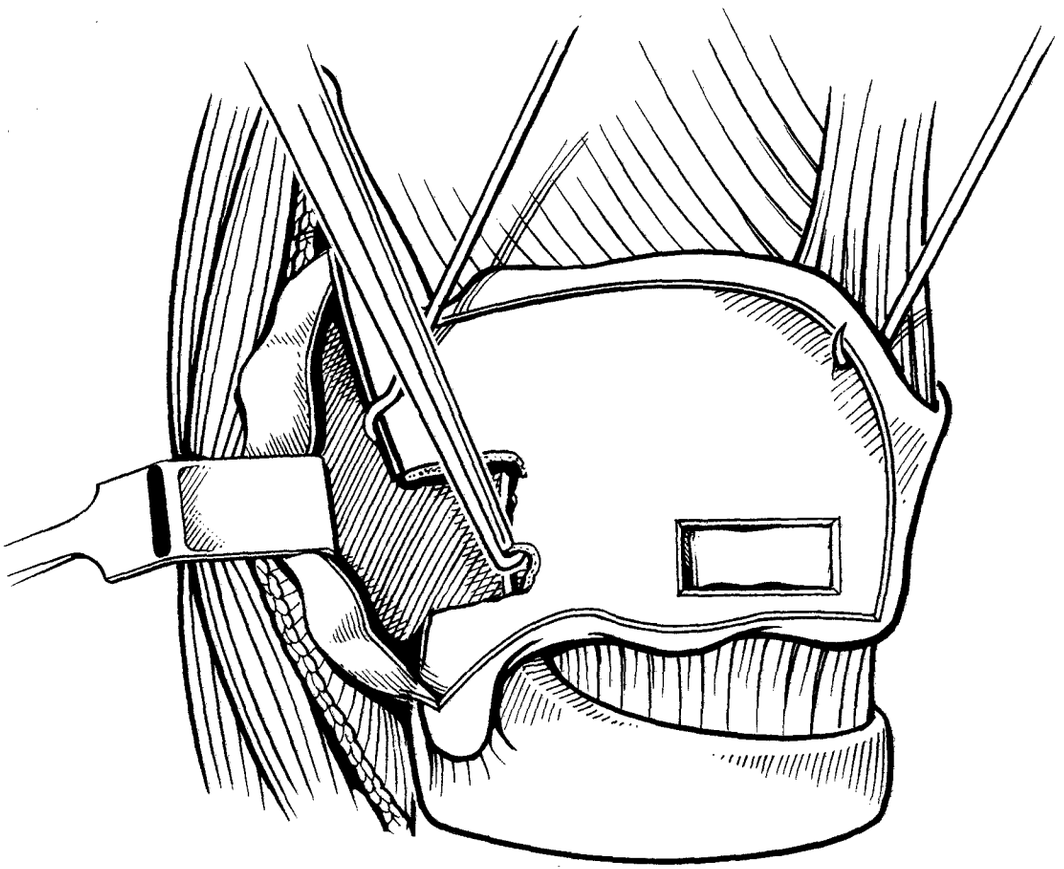

FIGURE 4. A window of cartilage is removed from the posterior border of the thyroid cartilage with a 5-mm Kerrison rongeur. The size of the window ranges from 10 to 15 mm in height and extends forward approximately 10 mm or until the anterior aspect of the pyriform sinus can be identified.

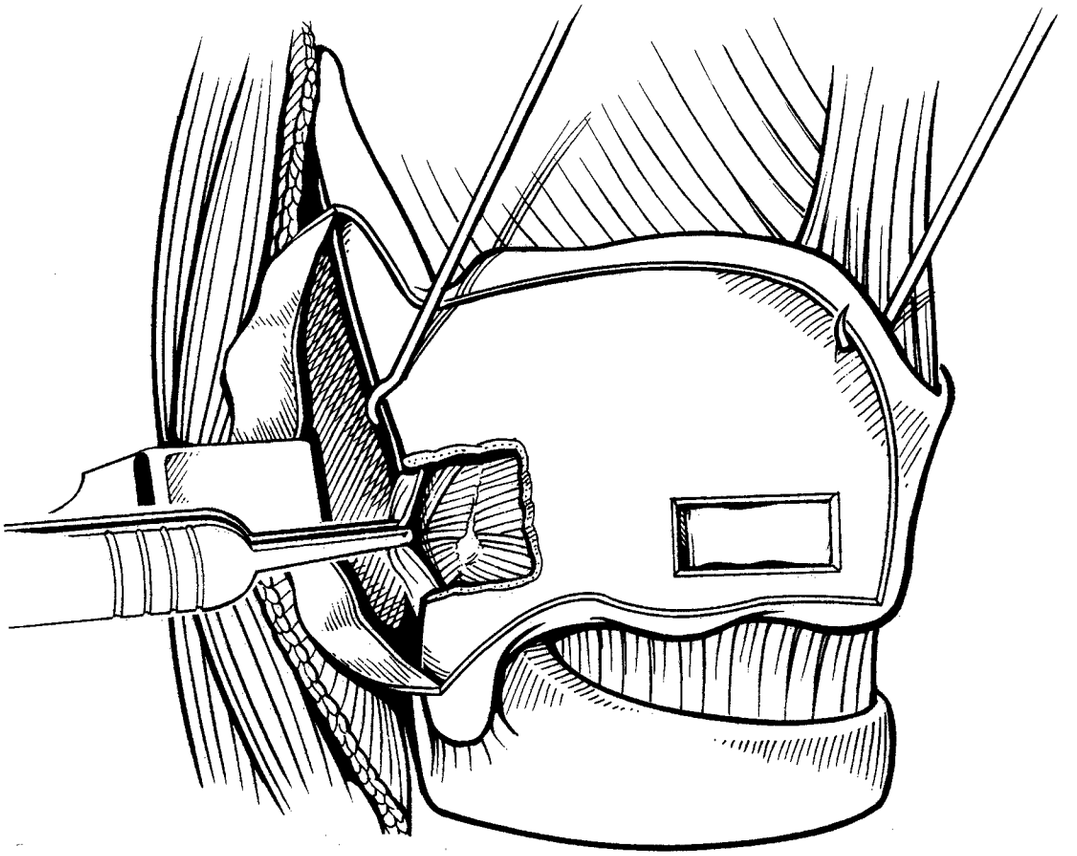

FIGURE 5. The anterior extension of the pyriform sinus is dissected away from the muscular process and the posterior cricoarytenoid muscle. The muscular process is identified with the white zone of perichondrium seen on its lateral extension between the attachment of the thyroarytenoid anteriorly and the posterior cricoarytenoid and interarytenoid muscles posteriorly.

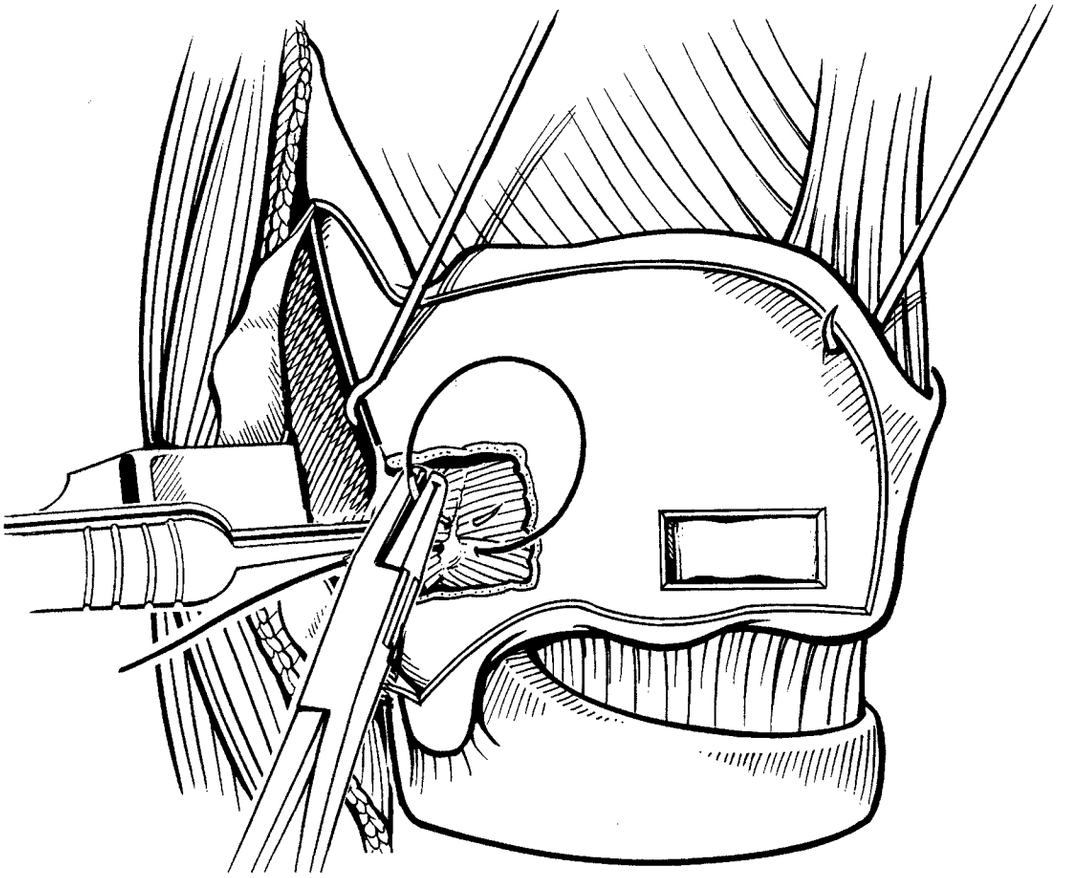

FIGURE 6. With retraction of the pyriform sinus, a 4-0 suture is placed through the lateral surface of the muscular process in a figure eight fashion to ensure a good purchase on the cartilage.

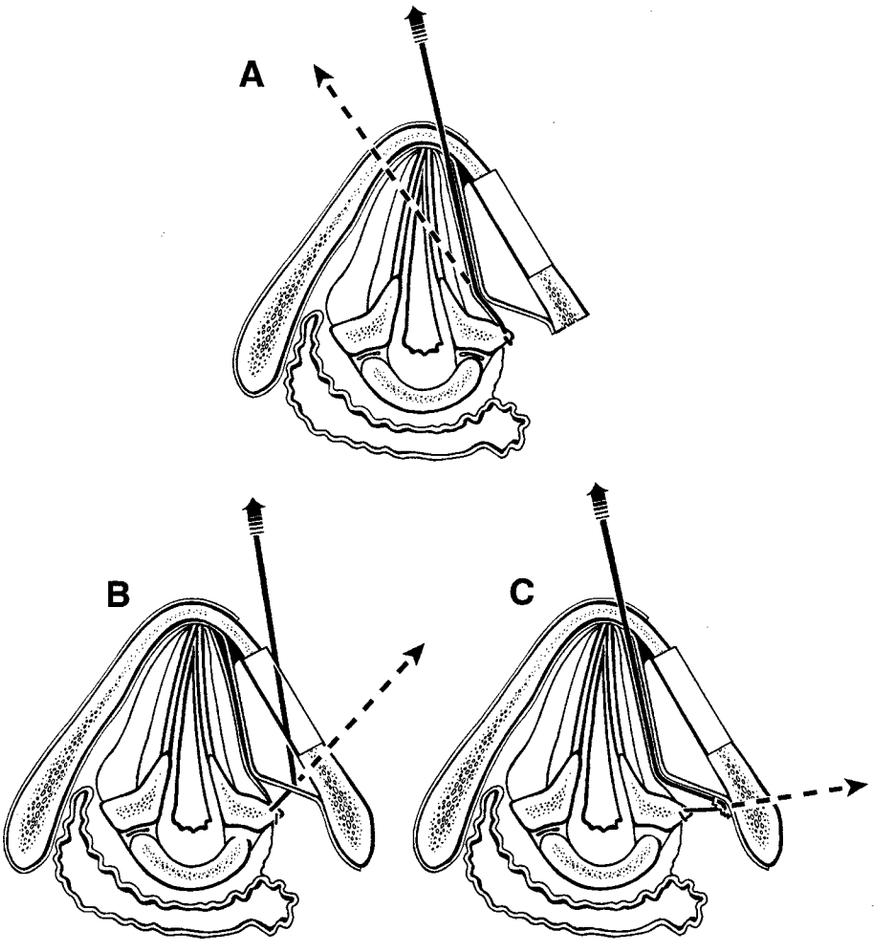

FIGURE 7. (A) The physiological vector of force acquired when the AA suture lies unencumbered medial to the implant. (B) The altered vector of the AA suture when it passes through the implant. (C) The altered vector of the AA suture when it is caught in the lateral paraglottic soft tissue or the partially elevated perichondrium. This vector of force concept is critical to allow the AA stitch to rotate the vocal process medial and inferior into the physiological phonating position.

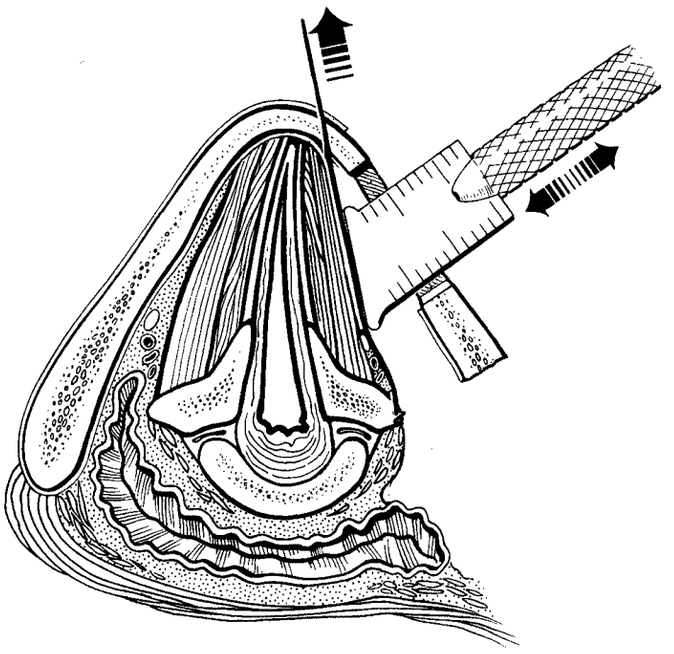

FIGURE 8. The depth gauge simulates the implant while the voice is assessed both subjectively and by the video monitor. The additional effect of the AA is observed by gentle traction on the suture. Both the medialization and the AA stitch can be tested jointly or independently.

DISCUSSION

The purpose of the AA is to place the arytenoid into a physiological phonating position. The arytenoid adduction suture mimics the vector of force of the thyroarytenoid muscle to rotate the arytenoid vocal process medial-inferior, thus closing the posterior commissure. During a review of our first 200 phonosurgery patients undergoing SML with or without AA, we noted a significant qualitative and functional difference in the small group of patients having undergone AA as an adjunct to SML; the AA patients fared better in a number of objective tests.

In a recent study, we have quantified this improvement with both aerodynamic tests as well as perceptual voice analysis. We know from previous reports that improvement in glottal closure secondary to SML alone results in less glottal airflow escape and is reflected by a significant increase in maximum phonation time as well as a reduction in mean flow rate through the glottis.[2,3] When comparing the SML + AA group to the SML alone, there was a statistically significant improvement in maximum phonation time (219% *v* 122%) for the SML + AA group ($P = .06$), and there was a trend towards improvement of mean flow rate (48% decrease *v* 42%) and overall voice score (157% increase *v* 139%).[10] What these scores do not express is the change in voice quality that AA produced in patients in whom it was obvious intraoperatively that SML alone would not result in a good voice.

Because of these findings we now expose the muscular process of the arytenoid in all patients irrespective of the preoperative position of the vocal process to directly assess the benefit of AA in conjunction with SML. This ability to judge the benefit of AA in all patients has significantly

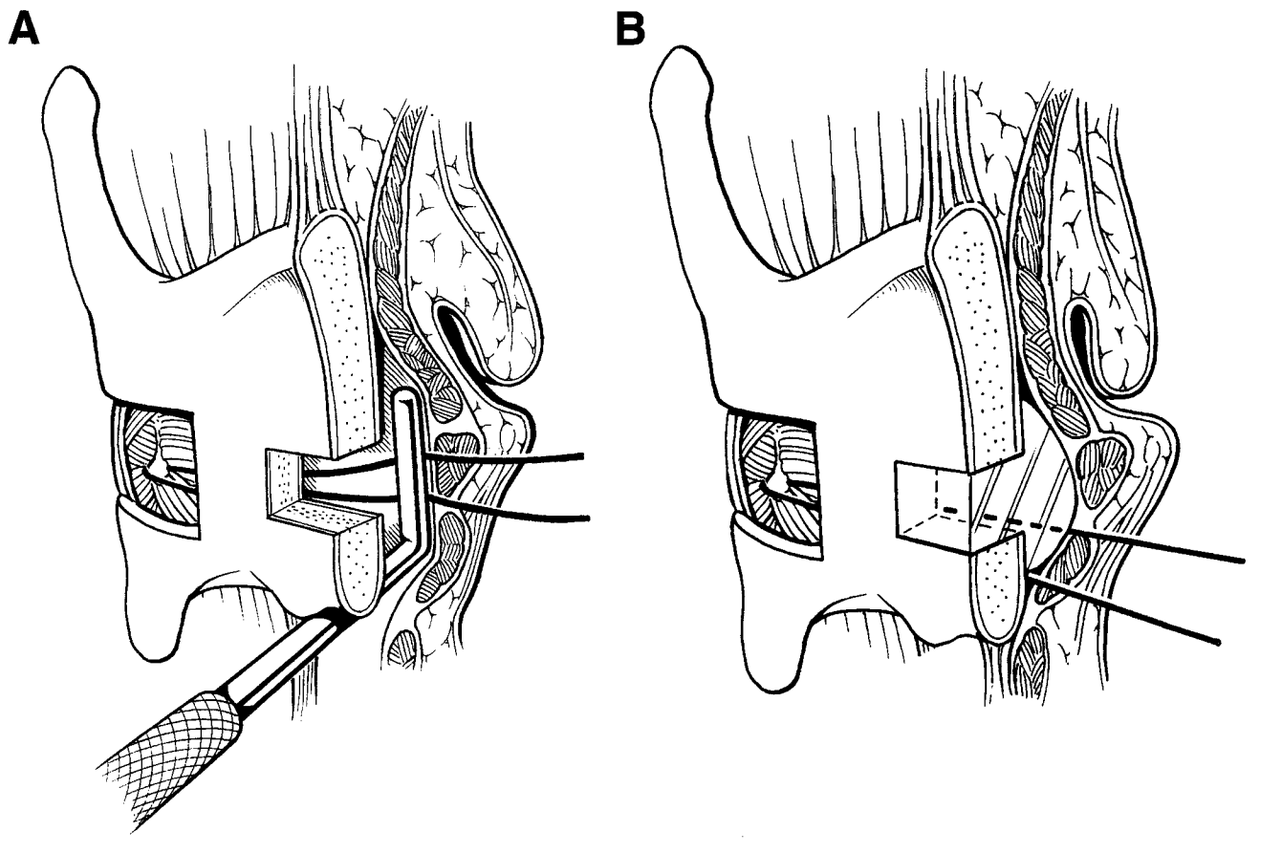

FIGURE 9. (A) The AA suture strands are displaced, as well as the TA muscle, with a small elevator to prevent the binding of the suture while the implant which has previously been carved is placed through the window. (B) The final position of the sutures lies medial to the implant.

improved our ability to obtain the best voice outcomes as reflected in both objective testing as well as perceptual voice scores. Whereas SML may effect the medialization of the membranous vocal fold, it cannot reproduce the vector of force exerted by the thyroarytenoid muscle to adduct the arytenoid cartilage. Only AA can mimic this vector, thus placing the arytenoid into the physiological phonating position. In this study, the indications for the use of arytenoid adduction are probably broader than has previously been reported.

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