Poor Speech Following the Pharyngeal Flap Operation; Etiology and Treatment

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Although the pharyngeal flap operation is generally successful in the correction of palato-pharyngeal incompetence, a significant number of patients continue to have hypernasal speech following the procedure. The purpose of this paper is to; 1) discuss the physiology of the poorly functioning pharyngeal flap; 2) present electromyographic and histologic evidence that the pharyngeal flap is an adynamic fibrotic bridge; 3) describe our approach to the secondary surgical management of poor speech associated with an inadequately functioning pharyngeal flap.

Normal velopharyngeal closure is dependent upon the action of the palatine levator muscles. Contraction of these muscles produces upward and posterior excursion of the soft palate and medial constriction of the lateral pharyngeal walls. This action results in a sphincteric closure of the nasopharynx and contact of the mid soft palate with the pharyngeal wall at or above the level of the tubercle of the atlas. It has been observed by measurement of cinefluorographic films (*1*) that minimal posterior wall movement occurs during normal speech. This suggests that the pharyngeal constrictor muscles play little if any role in normal speech production (*2*).

Owsley and Blackfield (*3*), Skoog (*4*), and Webber, Chase and Jobe (*5*), have all emphasized the importance of maintaining the upward-posterior vector of motion of the soft palate when attaching a pharyngeal flap. The low pharyngeal flap attached at the posterior margin of the soft palate produces traction in an inferior or straight posterior direction, and may actually restrict normal palatal elevation. A significant number of patients with residual velopharyngeal incompetence after a pharyngeal flap operation have been found in our experience to have just this type of restrictive flap (*6*). Clinical and cinefluorographic examination of these patients suggest that the central pharyngeal flap acts as a static obturator. It appears that closure of the lateral pharyngeal apertures at the low level of such a flap occurs as an acquired action of the pharyngeal constrictor muscles.

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The pharyngeal flap was postulated to be a dynamic structure by Broadbent and Swinyard who based their conclusions on electromyographic findings in patients who had undergone either superior or inferior based pharyngeal flaps (*7*). Using monopolar EMG needles inserted through the palate into the area of the flap, they observed normal motor unit potentials during swallowing.

Recently, Fara and Vele reported their EMG findings in a large series of patients with either inferior or superior based pharyngeal flaps (*8*). They reported that inferior based pharyngeal flaps tended to preserve their nerve supply and therefore their EMG activity, whereas superior based flaps tended to become denervated. The latter finding occurred despite their efforts to preserve blood and nerve supply to the pharyngeal constrictor muscle by basing their superior flaps several millimeters below the upper border of the muscle mass. Significantly, speech analysis of their series of patients revealed better speech improvement in the superior based pharyngeal flap group.

Because of our clinical impression of the adynamic nature of the pharyngeal flap, an experimental study was performed to evaluate the nerve supply and function of the pharyngeal constrictor muscles. The initial investigation was performed in experimental animals.

Methods

Adult mongrel dogs were anesthetized lightly. Base line EMG recordings of the pharyngeal muscle activity during swallowing were obtained. Bipolar 13K0823 Disa electrodes going to Disa differential amplifiers were used. Some recordings were photographed directly from the oscilloscope while others were recorded on tape with Ampex SP 300 tape for later photographing with a Cambridge photographing oscilloscope.

A lateral sagittal incision was then made on one side of the posterior pharyngeal wall through the pharyngeal muscle down to the plane of the prevertebral fascia. EMG recordings were then taken along the medial edge of the incision. There were no muscle potentials when swallowing was stimulated. Recordings along the lateral edge of the incision indicated normal muscle function.

The flap was then incised across its base and along the opposite lateral margin and dissected free from the prevertebral fascia. EMG recordings at this time indicated complete absence of any muscle potentials at any point along the length of the flap. It is important to note that these flaps were based high on the posterior pharyngeal wall and included the entire posterior width of the pharyngeal constrictor muscles. The results were consistent in three experimental animals.

Stimulated by these initial findings, Smith and Dedo then performed an extensive anatomical study of the nerve supply to the superior pharyngeal constrictor muscle in the dog (*9*). After eight dissections, which included direct nerve stimulation, they concluded that the nerve supply to the

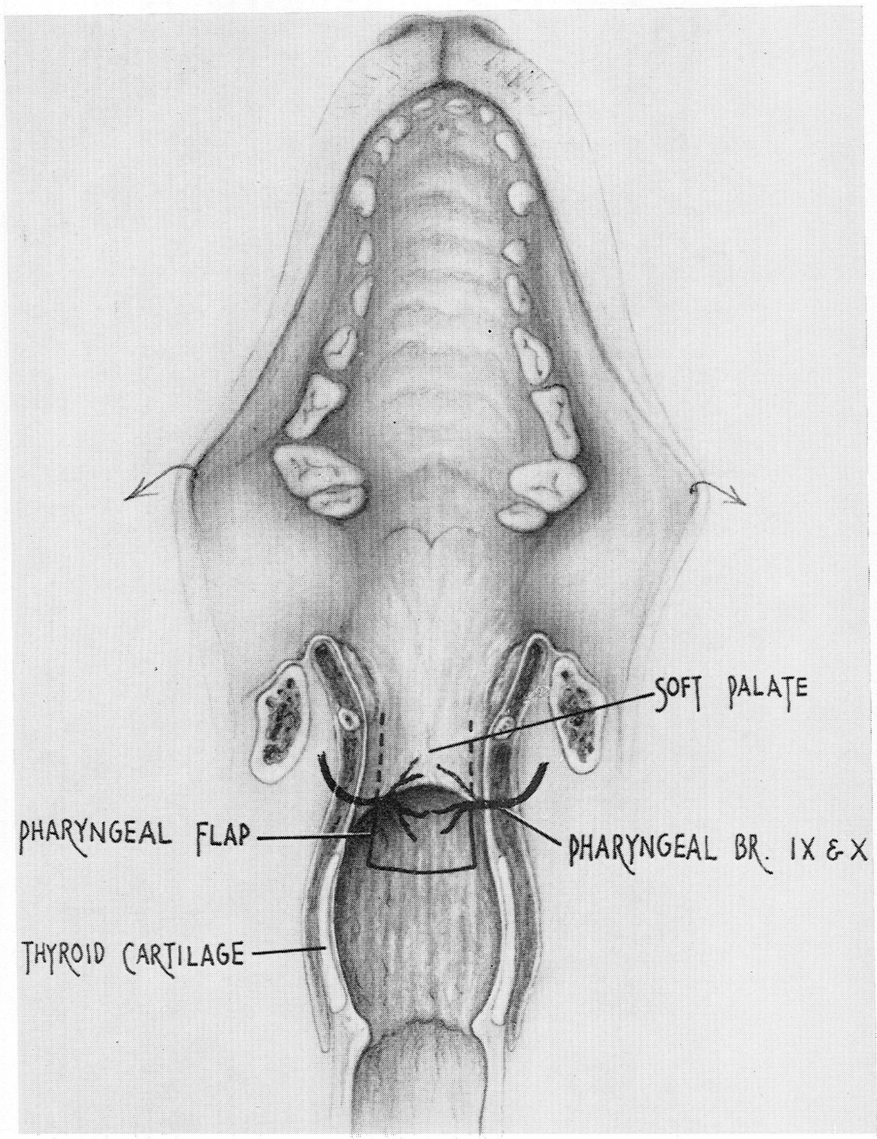

FIGURE 1. Drawing of an anatomical dissection in the dog showing the level that the nerve to the pharyngeal constrictor muscles enters at the lateral margin.

muscle enters at the mid-point of the lateral margin and therefore would be divided during the creation of any pharyngeal flap either superior or inferior based (Figure 1).

Clinical EMG studies were performed in the operating room on anesthetized patients undergoing superior based pharyngeal flap operations. Bipolar electrodes were placed in the pharyngeal musculature and base line recordings during swallowing were obtained. The lateral incision was then performed. Following this no further muscle potentials could be found along the medial cut margin. Stimulation of swallowing resulted in pulling of the flap to the contralateral unincised side. Normal motor potentials were demonstrated in the nasopharynx inferior to the incision and also in the soft palate (Figure 2). It was our conclusion that this lateral incision through the musculature to the level of the prevertebral fascia effectively divides the nerve supply to the pharyngeal constrictor in humans as was the case in experimental animals. After both lateral incisions and division of the inferior end of the flap had been completed, the flap was elevated. Following this we were unable to demonstrate any EMG potentials at any location in the flap.

EMG recordings of pharyngeal flaps were also obtained in patients ranging from one to five years post-operatively. Topical anesthesia was used and the electrodes were placed directly into the flap in each case. Normal muscle potentials were recorded from the soft palate and surrounding adjacent pharyngeal musculature but in each instance no muscle potentials were found within the flaps themselves.

Similar observations on the adynamic nature of the standard pharyngeal flap have been reported recently by McCoy and Zihorsky (*10*). Their studies included cadaver dissections which traced the nerve supply of the pharyngeal musculature. The anatomical findings indicate that the incision for all conventional pharyngeal flaps would completely sever the nerve supply to the constrictor muscle included in the flap.

As a result of these consistent laboratory and clinical findings we conclude that a pharyngeal flap based high on the posterior pharyngeal wall and outlined and elevated in the standard fashion will be denervated. The muscle in such a structure becomes atrophic and the bulk of the flap will be replaced with fibrous scar tissue (Figure 3). This flap will have no intrinsic contractility and would not in itself contribute any dynamic

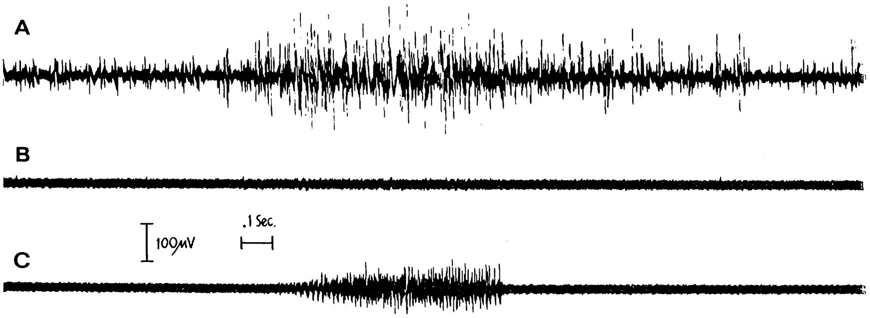

FIGURE 2. Electromyographic recording of a patient taken at the time of operation. Line a., electrode placed in the pharyngeal constrictor lateral to the incision. Line b., electrode placed into the pharyngeal constrictor medial to the incision. Line c., electrode placed in the soft palate. Recording taken as the patient swallows.

FIGURE 3. Photomicrograph of a histologic specimen taken from a low attached pharyngeal flap which was divided. The majority of the field is collagenous connective tissue with a single atrophic muscle fiber in the central field.

action to velopharyngeal closure. Its function will be merely one of traction on the palate towards the pharyngeal wall and obturation by the nature of its location and bulk.

Discussion

Our clinical experience with 230 patients in whom the high anterior attachment of a superior based pharyngeal flap has been performed indicates that effective palate elevation and speech improvement is achieved in a high percentage of patients (*11*). Assuming the static nature of the flap, it would appear that this improved velopharyngeal closure reflects a more effective utilization of the normal speech mechanism, specifically the superior traction and medial constriction of the levator palatine muscles. Neuromuscular pathways controlling these muscles' function are adapted to the normal rapid mobility of the soft palate during speech. Compensatory muscle function utilizing the contraction of the pharyngeal constrictor, sometimes described as Passavant's pad, is a laboriously learned mechanism which is unreliable and easily fatigued (*12*). It is this type of acquired mechanism that we have observed functioning in the low attached pharyngeal flap in many cases. If there is good palate mobility and pharyngeal muscle movement, many low attached pharyngeal flaps will provide non-nasal speech. However, if palate elevation is restricted and pharyngeal

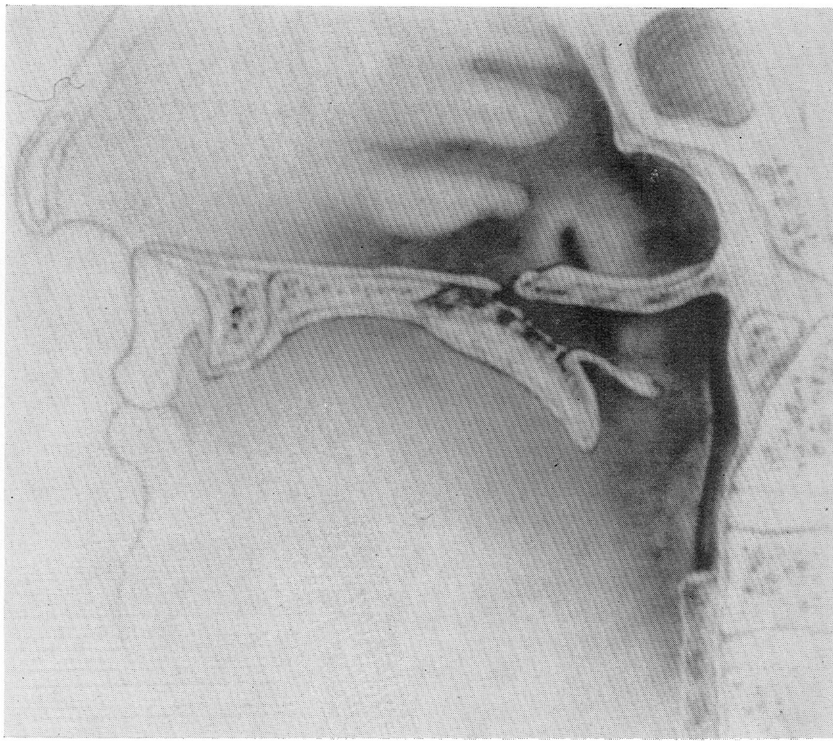

FIGURE 4. A drawing in the midsaggital plane showing the placement of a high anteriorly attached pharyngeal flap. The recipient site is the area of levator muscle insertion. Note the lining flap of reflected palatal mucosa.

muscle activity is ineffective or easily fatigued, then hypernasal speech persists.

It therefore seems reasonable to suggest that poor speech due to a restrictive pharyngeal flap may be improved by division of the flap and reattachment of a new high anteriorly attached flap (Figure 4). The palate is split in the midline after division of the low attached pharyngeal flap. The mucosal defect on the posterior pharyngeal wall is oversewn with a few sutures. It is interesting to note that except for the area of flap division, the posterior pharyngeal wall looks unscarred despite its being the donor site for the previously transferred flap. A new flap is outlined and elevated in the usual manner with its superior base carried to above the level of the tubercle of the atlas. The recipient site is developed on the anterior nasal aspect of the soft palate by reflecting posteriorly based flaps of palate mucosa. After the pharyngeal flap is sutured into the recipient site, the flaps of palate mucosa are sutured beneath the remaining raw surface of the pharyngeal flap thus providing lining and preventing scar attachment to the pharyngeal wall which would result in inferior migration of the base of the flap. This is essentially application of the standard procedure we have used for our primary pharyngeal flap operation for the past ten years. Our experience with this technique as a secondary flap revision in five patients is insufficient to allow for any critical analysis presently. However, subjective evaluation of the patients indicates improved speech in terms of reduced nasal emision and improved general intelligibility.

Summary

The pathophysiology of the poorly functioning pharyngeal flap is described. Electromyographic studies in experimental animals and in patients that suggests that the pharyngeal flap functions as a static obturator are represented. A surgical technique for revision of the inadequately functioning pharyngeal flap which utilizes palate muscle function in a more physiologic manner is described.

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