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Anatomic Relationships Between the Human Levator and Tensor Veli Palatini and the Eustachian Tube

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To define the interrelationships of the human levator and tensor veli palatini muscles and the Eustachian tube, fetal heads were serially sectioned and anatomic reconstruction done. Cephalometric points on fetal and adult skulls were compared to evaluate the effect of growth and development on these interrelationships. Based on the results of this study, we propose a mechanism for Eustachian tube function in the normal and in the cleft palate patient. This mechanism offers some explanations for many previously unexplained and paradoxical clinical observations.

Introduction

The patient with a cleft palate frequently has middle ear and hearing problems as well as speech problems. Although an abnormal insertion of the levator veli palatini muscle (Dickson, 1975; Hoopes et al., 1970; Kriens, 1975) may be implicated as the cause of hypernasal speech, the basis for the conductive hearing loss remains poorly defined (Bluestone et al., 1972; Dickson et al., 1974). To say that a "primary (Eustacian) tubal dysfunction" causes the universal presence of serous otitis media in cleft palate children (Bluestone et al., 1972) still fails to identify the etiology of that dysfunction.

Current concepts of cleft palate genesis suggest that neural crest cells fail to migrate sufficiently to support epithelial fusion (Johnson et al., 1975; Stark et al., 1958). Rather than postulate an independent component of the cleft palate phenotype involving the pharyngeal pouch/cleft system from which the Eustachian tube is derived, we postulate that the tubal dysfunction is secondary to the altered structure-function relationships of the abnormal cleft musculature (Edgerton et al., 1974). The physiology of the normal Eustachian tube remains controversial (Bosma, 1975; Cleland, 1869; Donaldson, 1972; Negus, 1943; Proctor, 1973; Rich, 1920; Robinson, 1923; Simpkins, 1943) because the anatomy of the epipharynx and base of the skull is inaccessible and difficult to study (Broomhead, 1957; Dellon et al., 1970; Dickson et al., 1972). Depending upon the author one reads, the tensor veli palatini muscle either opens (Cleland, 1869; Dickson, 1975; Dickson et al., 1972; Grant, 1958; Gray, 1949; Proctor, 1973; Rich, 1920; Robinson, 1923; Rood, 1972; Ross, 1971) or closes the tube (Simpkins, 1943) while the levator veli palatini muscle either opens (Dickson et al., 1974; Edgerton et al., 1974; Grant, 1958; Proctor, 1973; Simpkins, 1943) or closes the tube (Robinson, 1923). Unfortunately, non-primate mammals, in whom the epipharynx relates differently to the cranial base than it does in primates (Cave, 1960; Cave, 1967) have been experimental models (Rich, 1920).

Since a basic tenet of physiology lies in the interdependence of structure and function, we re-examined the anatomic relationships between the levator and tensor veli palatini muscles and the Eustachian tube in order to provide a foundation upon which to build a coherent thesis integrating disordered speech and hearing problems. These relationships were examined by serial reconstructions from

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fetal heads and by a developmental comparison between fetal and adult skulls.

Methods

Blocks for serial section were cut from human fetal heads aged approximately 38 weeks (A) and 32 weeks (B). They were cut in the following planes: (1) midsagittally, (2) sagittally through the parotid gland, (3) horizontally just above the inferior wall of the orbit, (4) horizontally at the base of the tongue, (5) coronally through the bony portion of the auditory tube (see Figure 1).

The formalin-preserved blocks were decalcified, embedded in paraffin, and sectioned at 20 micra. Every 10th section was mounted and stained with Mallory's trichrome connective tissue stain.

The microscopic slides were projected via a Prado Universal Projection System, using a ×10 magnification as relevant structures were traced slide by slide. The tracings were then rotated along a horizontal axis and redrawn in an orthogonal projection (without perspective) as described by McCann (1977). This technique was used in place of two-point perspective because the illusion of depth was considered unnecessary in structures of small magnitude (less than 15 mm. long and 2 mm. deep). Serial reconstruction was done according to the methods outlined by Gaunt (1971), Payne (1973), and McCann (1977).

The distance between each mounted section was computed: (thickness of section) × (no. of sections apart) × (magnification) or 20 micra × 10 × 10 = 2 mm. Each rotated section was retraced 2 mm. from the succeeding one. The edges were connected and appropriate shading was added. The cartilagenous tube, membranous tube, paratubal tissue, lumenal epithelium, base of skull, and levator and tensor veli palatini muscles were each reconstructed separately. The cut edge of the midsagittal plane served as a registration device.

For exact relationships, both overlayed reconstructions and cross-sectional tracings were examined. The slides were further examined microscopically to confirm tissue type and to determine muscle origins.

Growth patterns and developmental changes between the fetus and the adult were determined by measuring standard cephalometric points on the four adult and three fetal skulls. Downward and forward growth was determined by measuring distances between the anterior nasal spine (ANS), posterior nasal spine (PNS), and hormion (H), the point on the midline where the vomer meets the base of the skull (Figure 2). The ANS-PNS-H angle formed by the intersection of the ANS-PNS and PNS-H lines was measured. Lateral growth was measured between the hormion, the basion (the midline of the anterior border of the foramen magnum) (Ba), and the small tubercle postero-medial and nearly adjacent to the external opening of the carotid canal (CC). The Ba-H-CC angle formed by the lines H-Ba and H-CC was measured. The sample consisted of four adult skulls and three 32-36-week-old fetal skulls.

Results

The results are demonstrated in Figure 1. The tensor veli palatini muscle (TVP) originates on the base of the skull from the area of the scaphoid fossa anteriorly and the spine of the sphenoid posteriorly. The anlage of the tensor veli palatini and the tensor tympani are continuous. The TVP is consistently attached to the lateral portion and undersurface of the hook of the tubal cartilage and may attach to the lateral side of the membranous portion of the tube. Posteriorly, the TVP muscle fibers are arranged perpendicularly to the axis of the lumen of the tube, and they become oriented more obliquely as they approach the hook of the hamulus. The total shape in coronal section of the tensor veli palatini muscle indicates a relatively flat muscle. The TVP originates from bone and inserts into bone, the hard palate, via the palatine aponeurosis. Therefore, the TVP must contract isometrically.

The levator veli palatini muscle (LVP) originates from the quadrate area of the petrous portion of the temporal bone, which, in the fetus, is almost directly posterior (not medial) to the origin of the tensor veli palatini. With further growth and development, the origin of the LVP moves laterally, lying at a wider angle to the midline.

In the fetus, the LVP is not medial to the cartilagenous portion of the tube at any point. The rounded LVP muscle belly passes below a narrowed portion of the cartilage near the

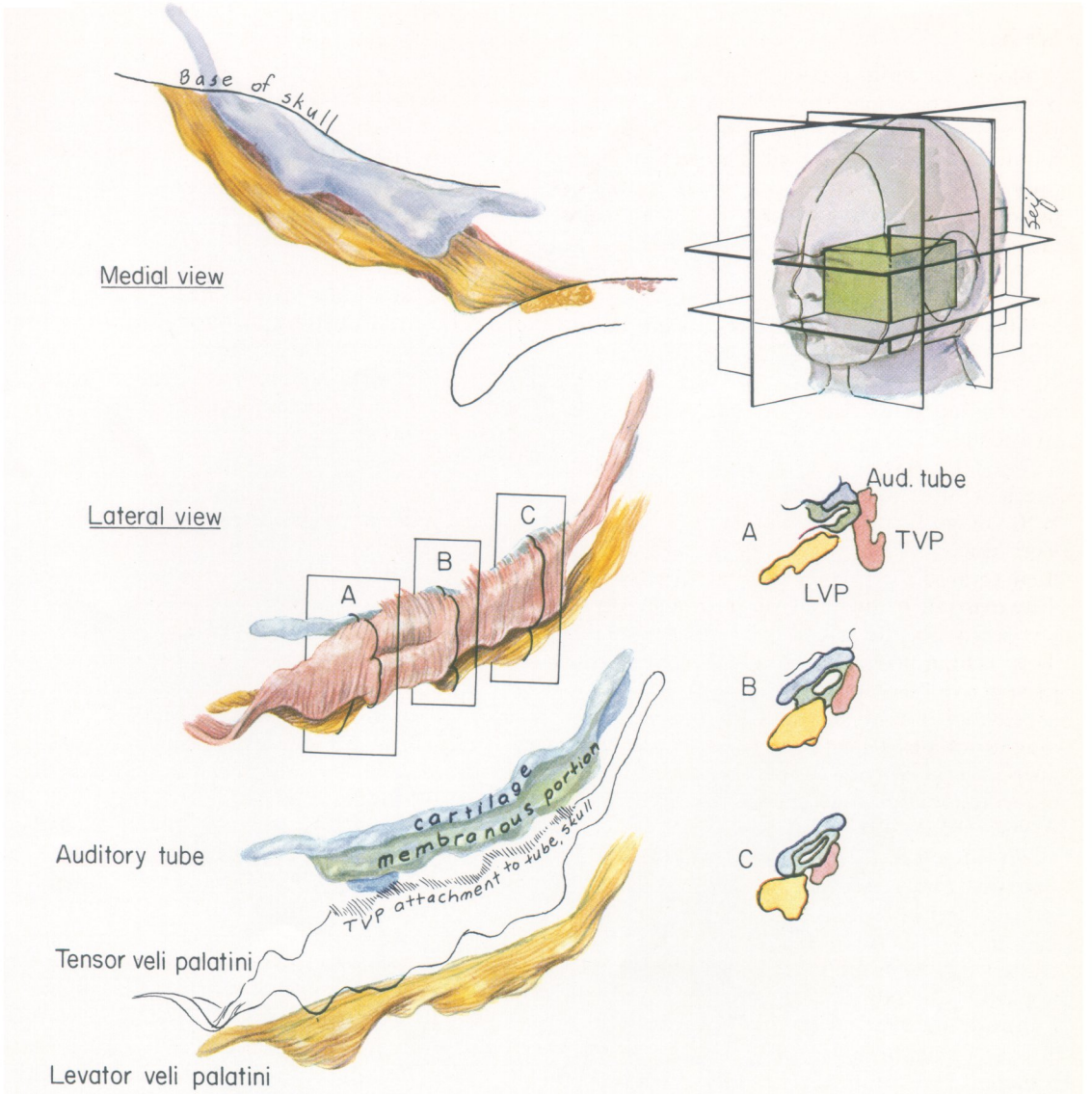

FIGURE 1. Reconstructions of 38-week fetus (A) and 32-week fetus (B), lateral view, left auditory tube and levator and tensor veli palatini muscles; medial view fetus B. Inset: location of block (shaded) serially sectioned. Refer to text for description of planes.

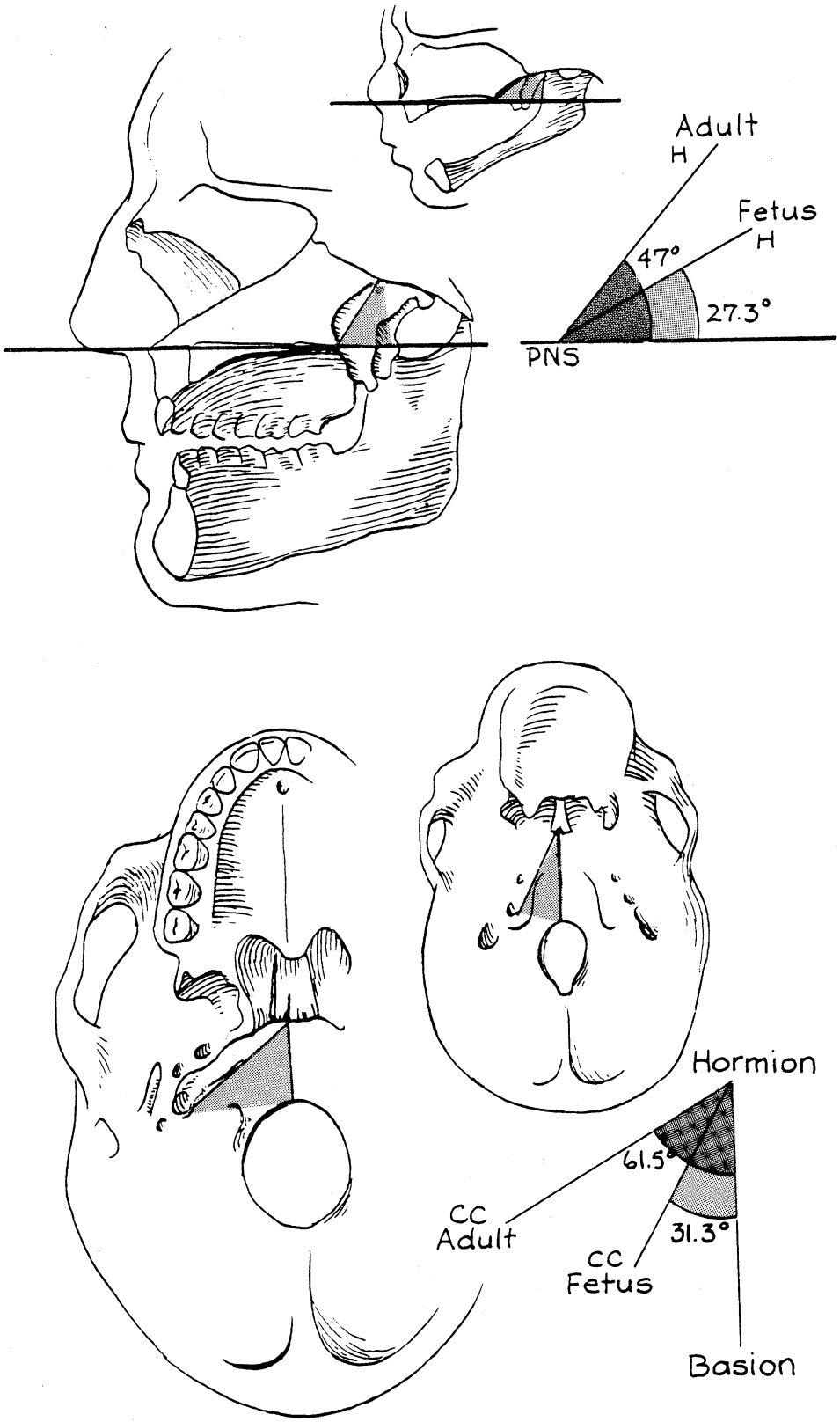

FIGURE 2. Developmental comparison of fetal and adult skulls: the levator veli palatini's insertion (the palate) moves downward and its origin (the quadrate area of the temporal bone) moves laterally with growth.

muscle origin at the base of the skull and then inferior to the lumen of the tube and lateral to the medial sheet-like portion of the tubal cartilage. The LVP muscle fibers are arranged parallel to the axis of the tubal lumen and, at the region of the torus, where the cartilage consists only of remnants of superior hooked portion, the fibers continue medially to enter the soft palate. The LVP does not originate from either the membranous portion of the tube or from the tubal cartilage.

The cartilagenous portion of the auditory tube consists of a short, laterally directed hook-like portion tangent to the cranial base and a large medial sheet of cartilage extending inferomedially from the tubal isthmus to a short distance before the torus. No suspensory ligament was observed between the cartilage and the cranial base. The medial cartilagenous sheet has a notch on its inferior border near the base of the skull through which the LVP passes to achieve its inferior position relative to the tube.

The membranous portion of the auditory tube consists of the epithelium of the tubal lumen, the paratubal tissue, and the dense, collagenous membrane enclosing the paratubal tissue. The membranous margin arises from the lateral side or top of the cartilagenous hook near the base of the skull, surrounds the paratubal tissue and appears to connect to the medial cartilage near the skull base.

The lateral tubal tissue consists of dense collagen fibers, capillaries, and lymphatics. The medial tubal tissue contains glandular tissue, fewer collagen fibers, and relatively more capillaries and lymphatics than the lateral tubal tissue.

Measurements of cephalometric points give the fetal-adult relationships at the base of the skull (Table 1 and 2).

The ANS-PNS-H angle formed between

TABLE 1. Fetal-Adult Characteristics of ANS-PNS-H Region

Specimen	*ANS-PNS*	*PNS-H*	*H-ANS*	*Angle*
		Adult		
A1	46.0 mm	21.2 mm	62.0 mm	58°
A2	47.2	25.8	70.0	41°
A3	50.0	21.9	67.3	40°
A4	57.2	27.8	77.3	56°
				47° mean
		Fetus		
F1	25.5 mm	8.5 mm	32.4 mm	25°
F2	25.8	6.2	34.5	26°
F3	27.4	9.0	32.4	31°
				27.3° mean

Note: all values are actual measurements.

TABLE 2. Fetal-Adult Characteristics of Ba-H-CC Region

Specimen	*H-B*	*CC-H*	*CC-Ba*	*Ba-H-c*
		Adult		
A1	26 mm	28 mm	25 mm	54°
A2	36	32	37	65°
A3	22	25	26	66°
A4	25	25	24	63°
				61.5° mean
		Fetus		
F1	20 mm	20 mm	12 mm	34°
F2	20	20	12	34°
F3	25	18	12	28°
				31.3° mean

Note: all values are actual measurements.

the plane of the hard palate and the posterior nasal spine-hormion averaged 27.3° in the fetus and 47.0° in the adult. The Ba-H-CC angle formed by the plane of the hormion and basion and the hormion and a tubercle postero-medial to the external opening of the carotid canal on the quadrate area, averaged 31.3° in the fetus and 61.5° in the adult (Figure 2).

Discussion

The results of this study suggest that the tensor veli palatini (TVP) muscle does not open the Eustachian tube by exerting a force radially outward upon the tubal lumen. The TVP arises from bone, from the cranial base, and from the lateral cartilage hook of the Eustachian tube. This hook, an extremely short lever arm, is also attached to the cranial base, and its movement, if any could occur, would not outwardly displace the tubal lumen. The TVP "insertion" is into bone via the palatal aponeurosis. This inelastic aponeurosis also is immobile, thus rendering the TVP probably incapable of true isotonic (length-changing) contracture. During contracture, the TVP muscle girth must, therefore, increase (isometric or non-length changing contracture) and exert a radially directed inward force against the tubal lumen (Figure 1). Although this would, in the usual sense, suggest that TVP "closes" the tube, we believe this TVP action is a "pumping one" which facilitates tubal content flow under the constant force of gravity.

The results of this study demonstrate that the levator veli palatini muscle (LVP), as it goes from lateral and inferior to the medial plate of the Eustachian tube cartilage medially into the mobile soft palate, is in a unique position to elevate the medial tubal cartilage. The LVP contraction (which does change muscle length because of non-boney velar insertion) elevates the soft palate posteriorly and the tubal cartilage medially. During this LVP muscle contraction, the paratubal tissue would be compressed radially inward from below, while the radially inward "closing" pressure exerted by the "resting" medial cartilage plate would be relieved.

It is concluded that normal Eustachian tube function consists of a constant force, gravity, drawing fluid along the tube from its higher origin, the middle ear, to its lower point of exit, the epipharynx. Forces of cohesion between lumen walls and the weight of the medial cartilage plate combine to prevent free flow of fluid along the tube by keeping the tube "closed" at rest. During swallowing, and to a lesser extent during phonation, a pumping or "milking" action is created by (a) elimination of the pressure exerted by the medial cartilage plate, and (b) application of new pressures to the remaining surfaces of the tubal lumen by the TVP and LVP muscle contractions. No muscle actively opens the tube in the classical sense of pulling open a lumen (Figure 3).

This proposed mechanism of tubal function offers some explanation for the following clinical observations and paradoxes: (a) All cleft palate infants have middle ear fluid (Bluestone et al., 1972a) *because* in the cleft palate the LVP muscle is inserted into the non-mobile boney hard palate. The LVP, therefore, must contract without changing its length. The constant pressure of the medial cartilage plate cannot be released, and the tubal lumen is constantly occluded. (b) After cleft palate repair, improved hearing and tubal function (Bluestone et al., 1972b) may occur *because* the LVP is often separated from its boney insertions. Many cleft palate patients may continue to have hearing problems since it is only recently that the emphasis has been placed not only on releasing the LVP but also on reuniting it across the midline and retrodisplacing it to give it a normal function (Edgerton et al., 1974). One non-cleft patient, in whom only an anterior LVP insertion was corrected by LVP retrodisplacement, had correction of his conductive hearing loss (Edgerton, et al., 1974). (c) The incidence of conductive hearing loss diminishes with increasing age *because*, (Goetzinger et al., 1960; Graham, 1963; Spriestersbach et al., 1962), as our results demonstrate, with growth and development, the LVP origin moves laterally, increasing its effectiveness in releasing the pressure of the medial cartilage plate on the tubal lumen, and the insertion moves downward and forward (increasing its effectiveness as a tubal "opener"). (d) Hamulotomy, or fracturing the pterygoid hamulus during palate repair, has no effect on hearing or tubal function (Noone, 1973), *because* it is the LVP rather than the

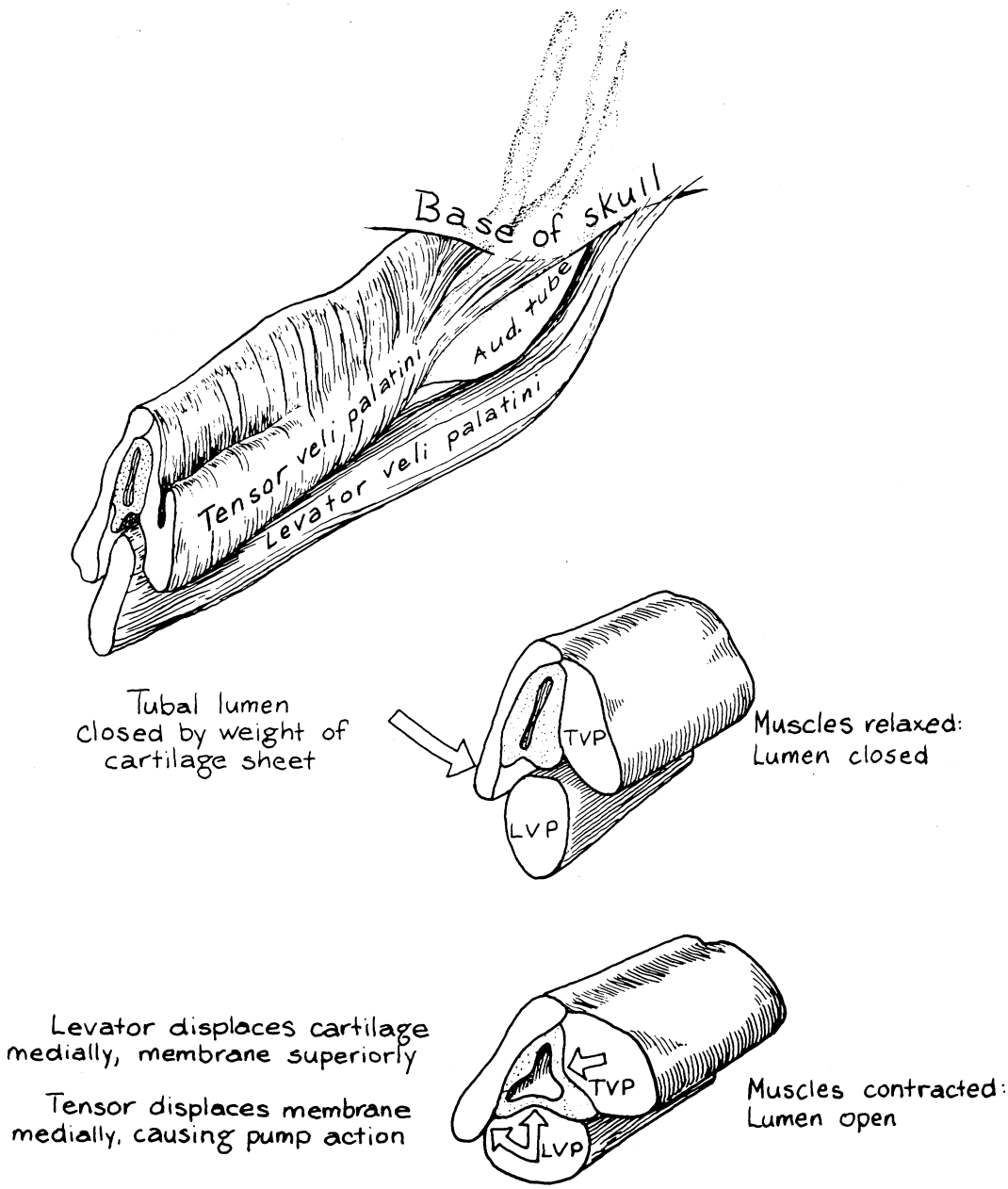

FIGURE 3. Proposed mechanism of auditory tube function. Tube "closed" at rest; during swallowing and phonation a "milking" or pumping action is created by contraction of the LVP and TVP muscles. No muscle opens the tube in the classical sense of pulling open a lumen.

TVP which is primarily responsible for tubal function.

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