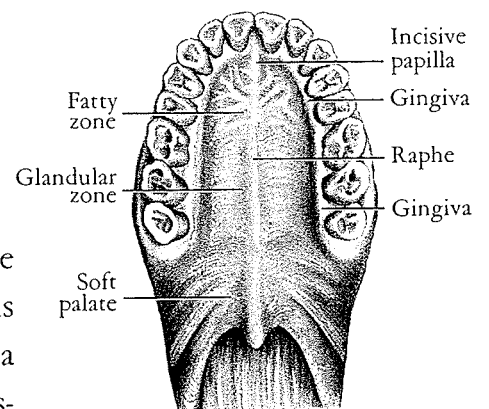
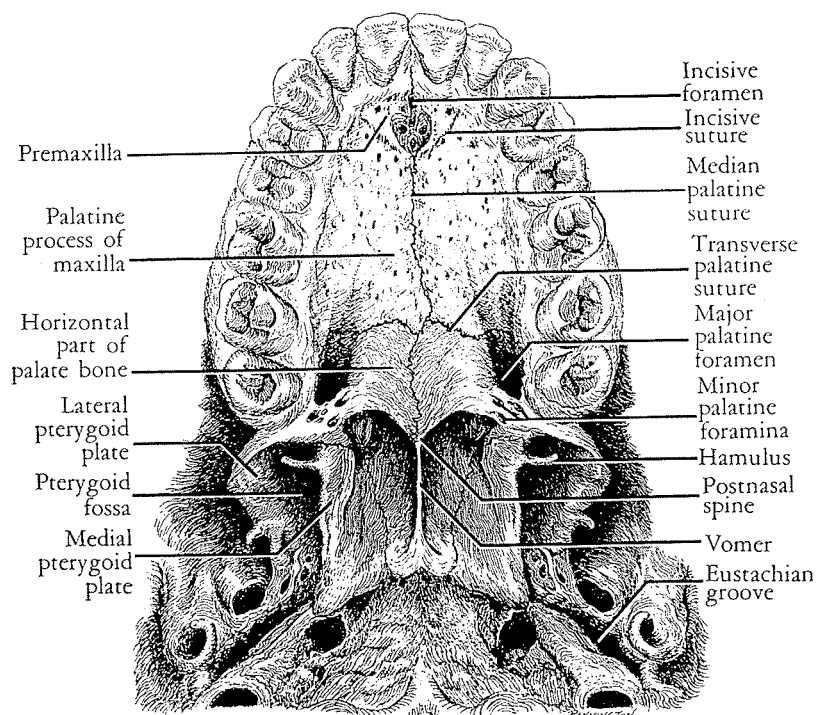


## 2. Anatomy of the Palate

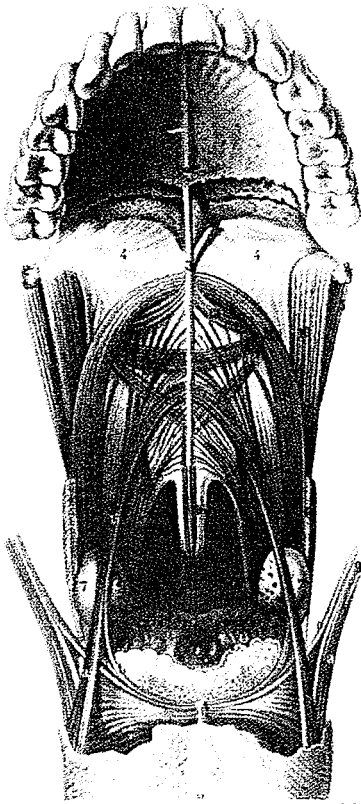
### BONY SKELETON

THE bony skeletal scaffold for the palate consists of the bones adjacent to the base of the skull, including the sphenoid and temporal, the premaxilla, the maxilla proper, and the palatine bone. These bones offer the origins and insertions of the muscles and provide the exits of the nerves and blood vessels serving the palate.



These bones, of course, are covered with a periosteum before receiving the palatal muscles and aponeurosis. The entire area is covered finally with a mucous membrane. In the hard palate area the mucosa is grayish pink and tightly adherent to the perios-

teum. The mucous membrane of the oral surface of the soft palate is highly vascular and red in color, covered by a non-keratinized, stratified squamous epithelium. A layer of elastic fibers separates the lamina propria from the submucosa, which contains many mucous glands. At the free border of the soft palate, the oral mucosa changes to pseudostratified, ciliated, columnar epithelium of nasal mucosa.

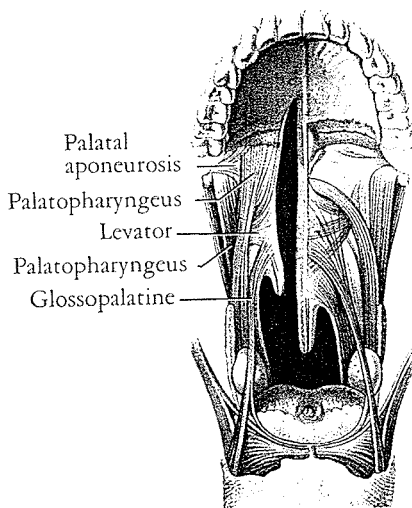


von Luschka

## PALATAL MUSCULATURE

Gustavo Sanvenero-Rosselli of Milan, a historical scholar who accumulated an extensive plastic surgery library, reminded cleft specialists at the 1973 Copenhagen International Cleft Palate Congress that Leonardo da Vinci understood the function of the soft palate in using vowels in speech and was cognizant of the varying levels attained by the palate during speech.

The first true anatomical descriptions of normal anatomy of the palate and pharynx were published by H. von Luschka in German in 1868.



Palatal  
aponeurosis  
Palatopharyngeus  
Levator  
Palatopharyngeus  
Glossopalatine

Veau

### Veau

Victor Veau of l'Hôpital des Enfants Assistés, Paris, published his classic book, *Division Palatine*, in 1931. Here he described the anatomy and pathology of cleft palate, gleaned from his vast number of cases and his hundreds of surgical procedures. In an explicit diagram of the palatal anatomy, he compared the normal aponeurosis and musculature with that in a cleft palate, showing some of the displacement and misdirection of fibers. One of his important contributions in palate surgery was the metallic suture used in gathering the fibers of the displaced muscles of the cleft edges parallel to each other.



James Whillis

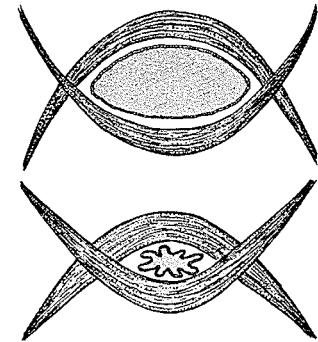
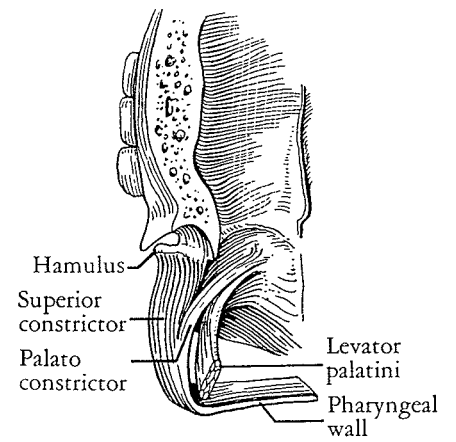
### Whillis

In 1930 anatomist James Whillis of Guy's Hospital, London, showed that some fibers of the superior constrictor were inserted into the palatal aponeurosis and constituted a lamella he referred to as the "palato-pharyngeal sphincter." It is possible in the

normal palate that Whillis' lamella may narrow the lateral pharyngeal recess, thus helping obtain ultimate palatal occlusion of the nasopharynx during contracture of the levator.

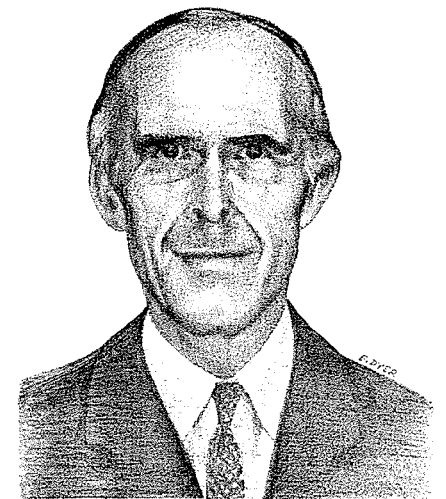
*Browne*

In the December 1935 *British Medical Journal* the eccentric, innovative Denis Browne of the Hospital for Sick Children, Great Ormond Street, London, postulated an orthopedic operation for cleft palate, justifying his design by his evaluation of the muscular activity during nasopharyngeal closure. He diagrammed two overlapping muscle slings involving the levator palatini and the superior constrictor and noted the efficiency of this sphincteric action, which avoids the necessity for the large amount of contraction required by a simple, single muscle ring sphincter.

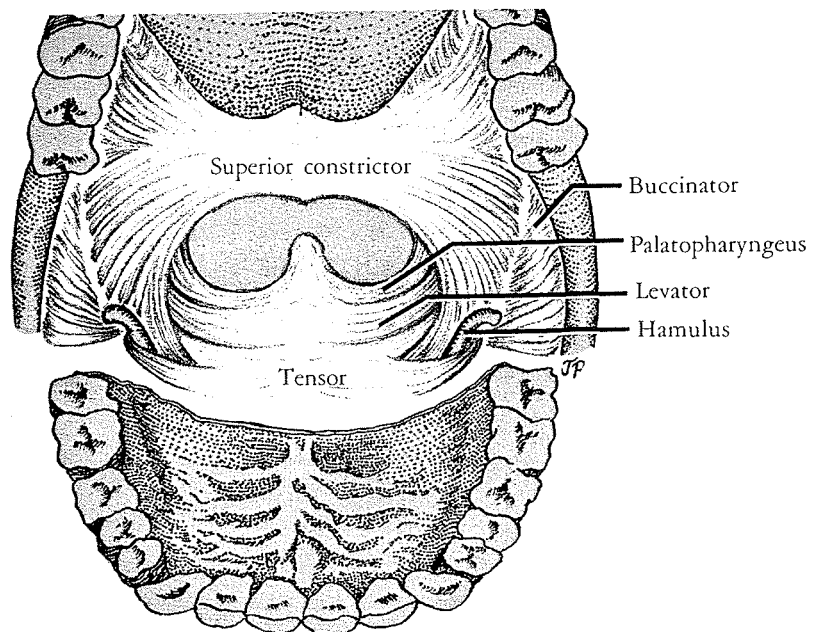
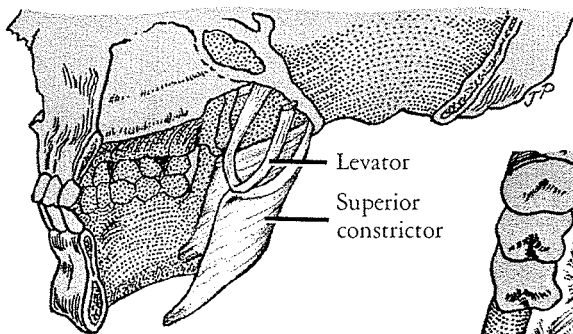


*Oldfield*

In 1941 Michael Oldfield of the General Infirmary at Leeds, England, noted that the muscular elements of the soft palate, apart from the uvular muscle, consist essentially of four slings. These are actually bilateral muscles which effect the sling-like function through their common insertion into the tissues of the soft palate. Superiorly, these muscles are the levator and tensor palatini; inferiorly, they are the palatoglossus and palatopharyngeus.



*Michael Oldfield*

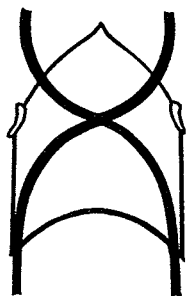


*Braithwaite*

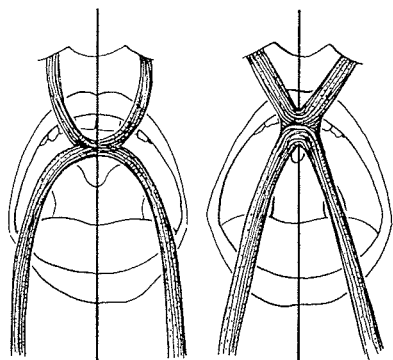
Fenton Braithwaite of Newcastle upon Tyne received his master's in mathematics at Cambridge prior to his study of medicine and training with McIndoe at East Grinstead. He collected such exquisite antiques that it has been said his furniture was either just coming back from or just going off on loan to the Victoria and Albert Museum. Braithwaite was the first to realize the importance of correcting the malposition of the levator muscle in cleft palate surgery.

At the 1964 Cleft Palate Congress in Hamburg, Braithwaite noted:

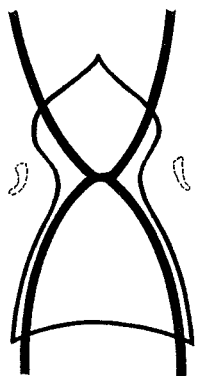
Of the four muscular slings [of Oldfield] . . . , the levator descending on each side and passing downwards and forwards and the palatopharyngeus passing upwards and forwards, are more intimately concerned with speech.



He presented a diagram of the anterior view of the normal upper levator and lower palatopharyngeus muscle slings (heavy line) and the superior constrictor (fine line) from its attachments at the base of the skull to the hamulus and pterygomandibular raphe. He explained:



The levator sling elevates the soft palate in a backward and upward direction whilst the palatopharyngeal slings on contraction will approximate the posterior faucial pillars and narrow the pharyngeal aperture. When these two slings act together, each loop will afford counter purchase for the other and the interposed palati steadied by simultaneous contraction will convert each group into a "V," and the whole mechanism partakes of the features of an "X," as suggested by Podvinec (1952).



When this mechanism is examined from an anterior view through the open mouth, it may be seen that an isthmus is produced at the level of the soft palate by pulling in the lateral walls of the pharynx. It is obvious, therefore, that if the slings are not intact or indeed if the soft palate is scarred, this will not be effected.

As early as 1949 Braithwaite advocated constructing the levator muscle sling.

Energetic Otto B. Kriens, professor of plastic and maxillofacial surgery, Bremen General Hospital, Germany, started his training in cleft lip and palate in 1963 under Karl Schuchardt in Hamburg. This was like commando training for combat because his teacher was a taskmaster whose students had to be strong to survive. I once heard Sanvenero-Rosselli refer affectionately to his friend Schuchardt as "der Führer." The pressure pushed Kriens into the study of palatal anatomy, and this is what he wrote to me in 1973:



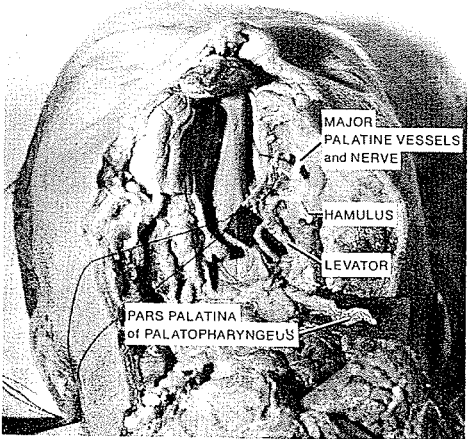
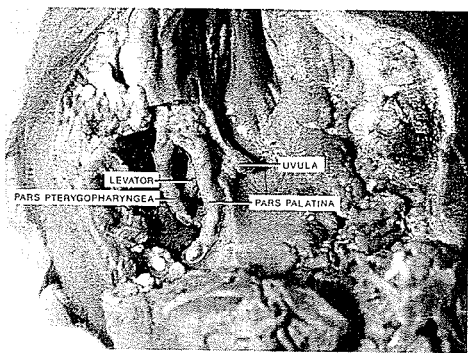
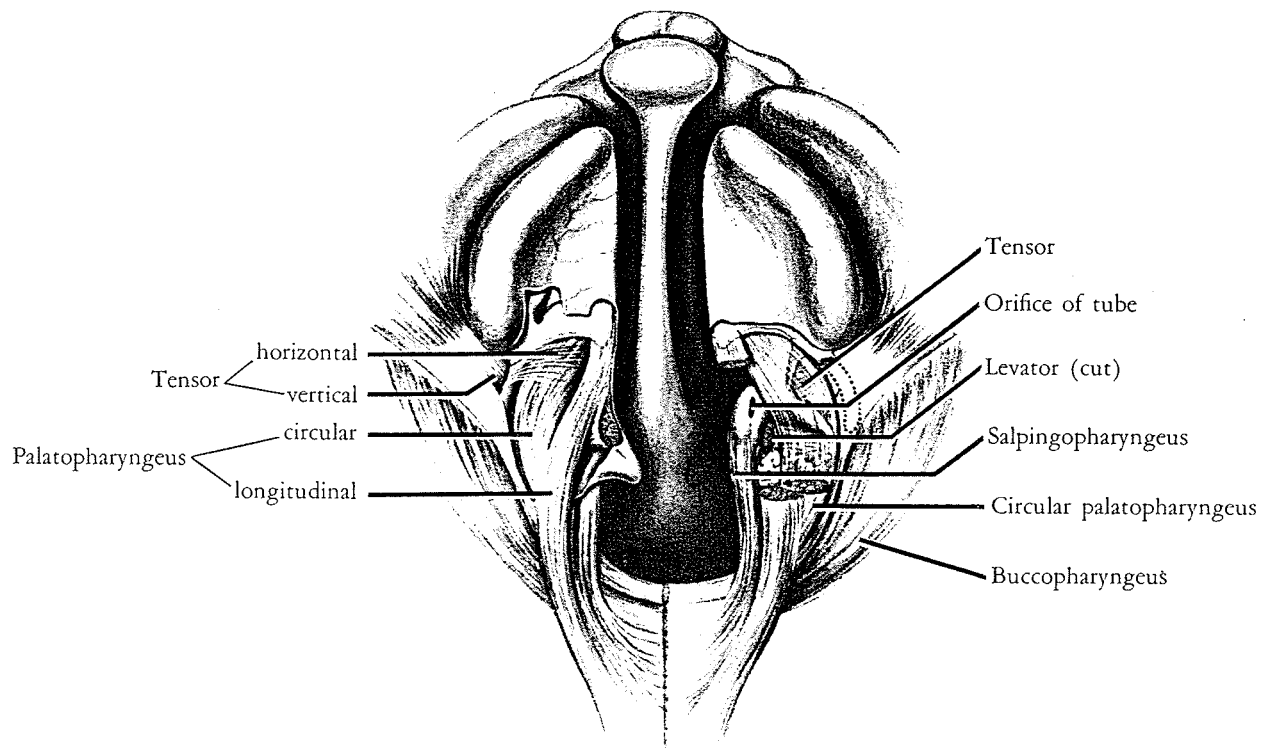
*Otto Kriens*

When I started to study the anatomy of the palate, astonishingly there was hardly any literature on the cleft velum! Although the first reports about veloplasties date back prior to 1820, the first detailed anatomic descriptions of the normal anatomy of the palate and pharynx were given by Luschka in 1864. Thus the beginning of cleft palate surgery had to be without knowledge about the pathology to be corrected. No wonder the operations were morphological ones and in all methods the velar halves were used as entities, as architectural units, which had to be moved medially and/or dorsally so to achieve integrity.

Veau had postulated a closure in layers and he drew sketches of what he had seen during interventions. Braithwaite followed Veau's principle even further and dissected "muscles," apparently not exactly knowing which were where.

The dissections I did on normal palates and pharynges trained my three dimensional perception but did not reveal the essential pathology. Thus Professor Schuchardt was not wrong in rejecting an article speculating what seemed to be wrong in the velum. His dissent was not too polite, which probably prompted Professor V. Karfik to spontaneously invite me to Prague for an anatomical study (1966), which revealed characteristic findings (O. Kriens, *Anatomische Untersuchungen am gespaltenen weichen Gaumen. Chir. Plast. Reconstr.*, 1967). The necessary changes of the velar closure were only too evident after the pathology had been exposed to sight!

Here is his 1967 diagram of the dissected soft palate from the oral aspect:



Also shown are two of his actual dissections with labels: (1) Total bilateral cleft; fresh specimen. On the right can be seen the pars palatina/palatopharyngeus with its insertion on the posterior edge of the palatine bone, as well as the pars pterygopharyngea with its insertion on the hamulus, continuing to the aponeurosis. Between these, the belly of the levator is visible. (2) Total bilateral cleft; material fixed for some weeks in 10% formalin. On the left side of the specimen the levator belly with all its insertions is dissected and the pars palatina of pharyngeus, freed from its hard palate insertion, has been turned laterally.

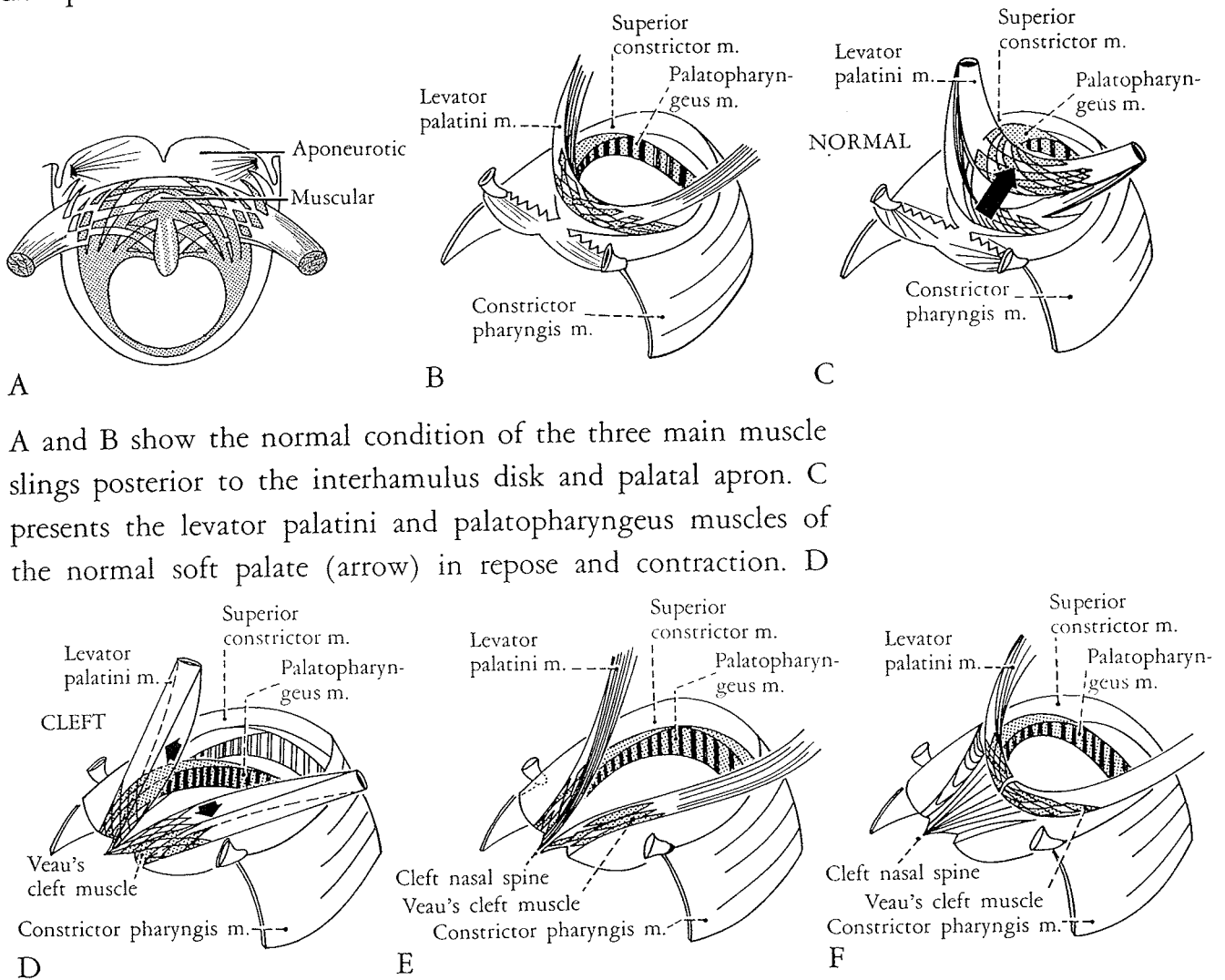
In *Plastic and Reconstructive Surgery* in 1969, Kriens summarized the pathology of the cleft palate anomaly as the basis for planning reconstruction.

1. The forward and transverse displacement of muscular insertions (but the origins are normal).
2. The synergistically interwoven anterior portions of the levator veli palati and of the longitudinal portion of the palato-pharyngeal muscle (Veau's cleft-muscle).
3. The palato-salpingeal fascia forming a functional boundary between the constrictor pharyngeal and palato-pharyngeal muscles (levator space of cleft palate).

4. The plane of loose connective tissue between the palato-pharyngeal sphincter and the palato-salpingeal muscle (plane of blunt dissection in the velum proper).
5. The altered insertion of the hypoplastic horizontal (palatine) tendon of the tensor veli palati (no aponeurosis in the cleft velum).

*the Dicksons disagree*

His explicit drawings, presented in the *Cleft Palate Journal* in January 1970, show the normal palate, the basic pathology of cleft palate and the ideal goal of corrective surgery:



presents the levator palatini and palatopharyngeus muscles of cleft soft palate in repose and contraction. E portrays Veau's cleft muscle. F shows the major portion of Veau's cleft muscle being joined to form the levator sling.

Always interested in controversy, I asked David Dickson to review Kriens' outstanding 1967 article. These are his 1976 comments:

His statement that the tensor attaches to the Eustachian tube cartilage is debatable, if one views the literature, and probably wrong. The lack of aponeurosis in the cleft velum is a consistent finding of other investigators, but one in which we do not concur, based on our own histologic sections. His statement that the uvulus muscle is not involved in velopharyngeal closure is without support and is very likely wrong.

## DIVIDENDS OF MODERN RESEARCH

Closure of palate clefts has been in progress for over two centuries, and with reasonably good functional results in a majority of cases during the last half century. Yet only in the last few years has the sophisticated knowledge of palatal anatomy and physiology been clarified to the extent that the plastic principle "return normal to normal position and retain it there" has finally been appreciated and applied to cleft palate surgery.

Velopharyngeal function, as its name implies, is a combination of movement patterns of the velum and of the pharynx. The principal components of the velum are a single intrinsic muscle, the insertions of several extrinsic muscles, a large amount of glandular material in the anterior inferior portion of the velum and an anterior aponeurosis. The single intrinsic muscle is the azygos uvulae. Muscles typically referred to as extrinsic muscles of the velum include the tensor veli palatini, levator veli palatini, palatopharyngeus, palatoglossus, and fibers of the superior constrictor. Pharyngeal muscles usually described as having a functional role in velopharyngeal movement include the superior constrictor and the salpingopharyngeus. Research over the past 40 years presents us with a view of the anatomy and physiology of this system which is frequently at variance with descriptions found in modern textbooks and old wives' tales.

### *Dickson*

As a guest speaker at the Ohio Valley Society of Plastic and Reconstructive Surgery in 1970, I was impressed by a presentation on "New Studies on Velopharyngeal Musculature" by David Ross Dickson, professor of anatomy and speech at the University of Pittsburgh. Dickson, dedicated, sensitive, tireless, is a teacher who chose speech pathology because of a long-standing interest



in human communication. He has an inborn quirk that compels him to attack ideas simply accepted, without reason or because of tradition. He believes in the scientific method, not just as an exercise in academia, but also in personal conduct, social rules and political viewpoints, searching to know the “real question” and considering unconsidered options. Yet he can be found in all weather collecting English brass rubbings, made by a technique developed in eleventh-century Netherlands, atop famous British sarcophagi, such as those of Sir Thomas Bullen, father of Anne Boleyn, Lady Margaret Chayne, and especially the Black Knight of Canterbury.

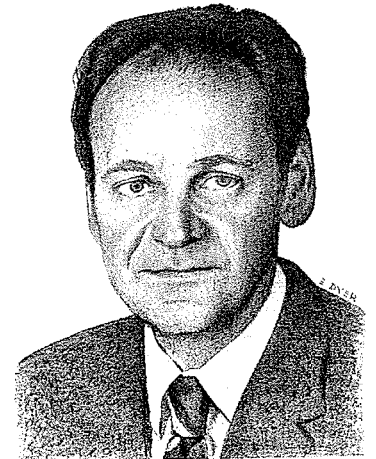
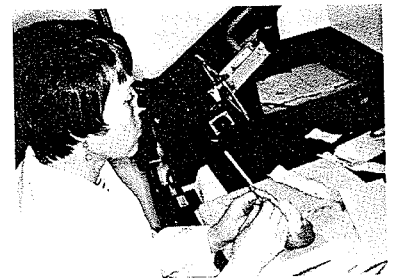
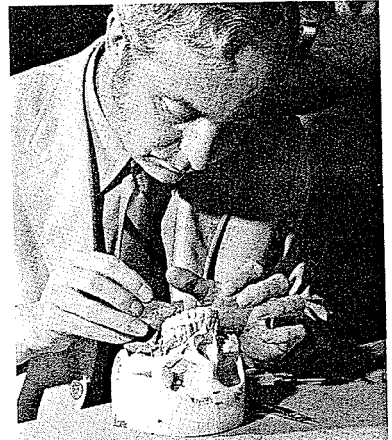
In 1972 Dickson was invited to Miami to lecture and at that time helped place the palatal muscles in plasticine on the base of a skull. Now, as professor of pediatrics at the University of Miami School of Medicine and director of speech and hearing at the Mailman Center for Child Development, he will describe the current knowledge on the palatal musculature with its variations from the previous standards in *Gray's Anatomy* and even in *Cleft Lip and Palate*, published in 1971. The accompanying drawings were made from dissections, aided by the microscope, of embryonic heads by Wilma Maue-Dickson, previously of the University of Pittsburgh and now associate professor of anatomy at the University of Miami School of Medicine.

Comparisons of muscles of all the usual types of clefts in 18 mature stillborn children with those of four normal stillborn children have been obtained from Miroslav Fára's and Jiří Dvořák's dissections at the Charles University, Prague.

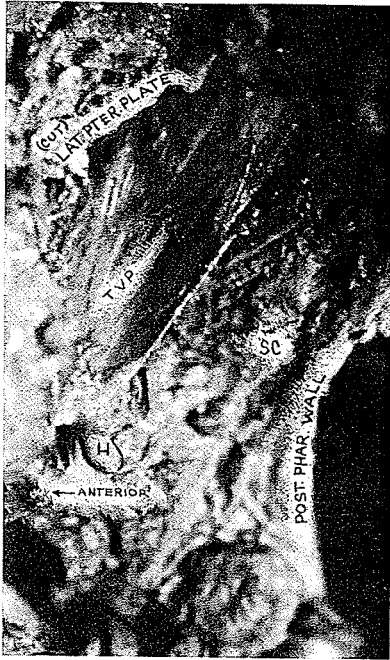
Dickson will describe muscles in the normal palate; Fára and Dvořák, muscles in cleft palate.

*Dickson:*

The *tensor veli palatini muscle* arises from the angular spine of the sphenoid bone, the scaphoid fossa, and the pterygoid fossa. Medial to these attachments, fibers of this muscle arise from the lateral membranous wall of the Eustachian tube. These fibers converge on a medial tendon which passes around the hamulus of the medial pterygoid plate. At this point the tendon is covered with a synovial sheet. The tendon then bends into a horizontal plane and enters the velum as the anterior aponeurosis, which is composed of a flat tendinous sheet in the anterior third of the velum, superior to the



Miroslav Fára



Adult lateral head dissection shows the tensor veli palatini muscle.

glands and muscles which are found in that portion of the velum. It has been thought that since the notch of the hamulus through which the tendon passes is slightly inferior to the velum, muscular contraction of the tensor palatini muscle would place tension on the velar aponeurosis. There is no evidence whatever that such tension exists as a function of contraction of this muscle. Nor is there any convincing evidence that tension placed on the aponeurosis would have any particular functional significance. It has been well documented, however, that this muscle, upon contraction, opens the Eustachian tube, and that no other muscle serves this function. In 1862, Henle disclosed that the tensor palatini muscle exerts a force predominantly effective on the auditory tube with only a minor part of its activity directed to the normal palate. In 1923, Rich confirmed this in dogs and in 1964 Ruding claimed that the tensor palatini muscle had only an auditory function in the cleft palate. It is interesting to note that the superior part of the tensor palatini muscle forms a tendon which passes through the cranial base and forms the inferior tendon of the tensor tympani muscle, the other end of which attaches to the malleus. Thus these two muscles, the tensor tympani and the tensor palatini, form a two-bellied muscle. There has been speculation that these two muscles may have a conjoint function in aeration of the middle ear; the tensor veli palatini by opening the Eustachian tube, and the tensor tympani by increasing middle ear pressure by drawing the tympanic membrane medially, which would, in turn, possibly lower the threshold for Eustachian tube opening. This speculation has arisen from the evidence that three factors are necessary for Eustachian tube function. The first is contraction of tensor palatini muscle, the second is production of surfactant to release surface tension within the Eustachian tube, and third is air pressure differential between the middle ear and pharynx.

*Fára and Dvořák:*

The tensor [in the newborn cleft] was somewhat thinner than in a normal newborn child . . . [with] a few bundles attached to the hamulus and the tendon itself. . . . The front part of its bundles extended along the rudimentary palatine aponeurosis, toward the posterior nasal spine, and was partially attached to the spine or (laterally) to the posterior edge of the palatine bone. Some of the tensor fibers radiated into the aponeurosis. The main part of the tendon, however, arched backward to the cleft edge of the velum . . . [and thus became either] partly dispersed, and then a triangular area passed into the front bundles of the levator muscle, or [in two-thirds of the autopsies] . . . not disperse [d] at all, but passed into the anterior bundles of the levator muscle as . . . a thick and free single muscular-tendinous bundle.

## APONEUROSIS IN CLEFT PALATE

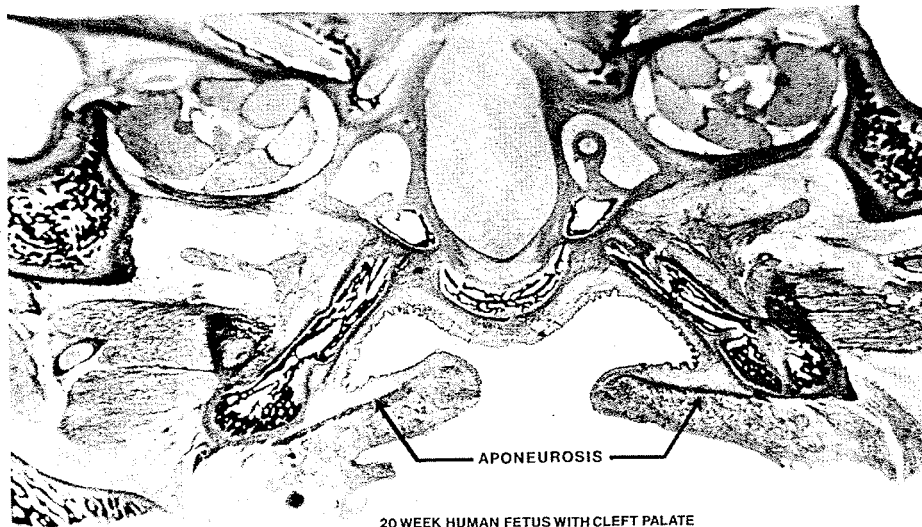
This is one area where Veau, Fára and Dickson have some disagreement. As indicated by Veau in *Division Palatine* (1931), the classic schema of the normal palate described by Fára consists of an anterior third (aponeurosis), a middle third (muscular) and a posterior third (mucous membrane). Veau stated:

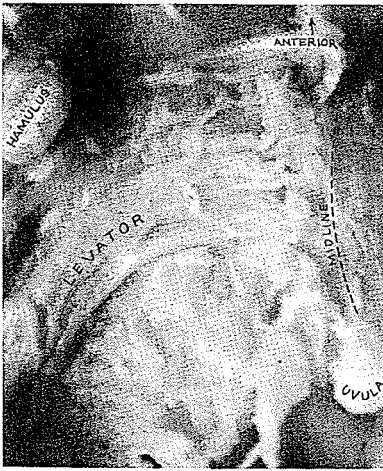
In cleft palate, the anterior third (aponeurosis) is lacking and there remains only a solid fibrous fasciculus for the aponeurosis; this fasciculus is attached to the bone. *There is no real palatine aponeurosis in the palatine division* [in cleft palate].

In 1970 Miroslav Fára and Jiří Dvořák discussed the deficiency of the palatine aponeurosis in the cleft palate. They wrote of the tensor veli palatini muscle:

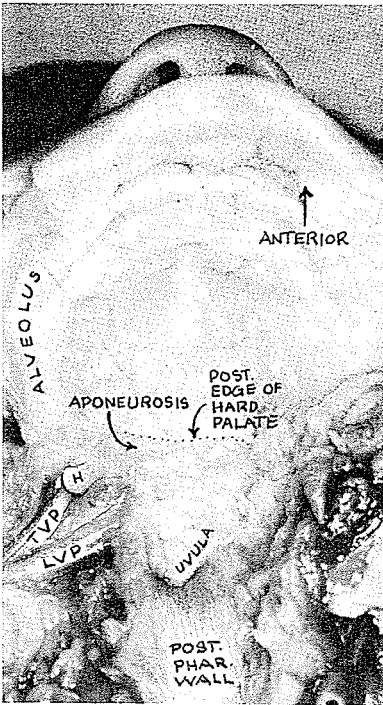
It has no proper chance to function fully; thus, it does not develop as it should. The absence of a fixed point in the midline (which is necessary for the insertion of the fan-shaped tendon) causes not only an incomplete and atypical growth of the tendon itself, but a marked hypoplasia of the palatine aponeurosis as well. Indeed, the very existence of this aponeurosis is due to the extension and penetration of the tensor tendon into it. Thus, the very aponeurosis in its lateral area is now very short; as it approaches the cleft margin, it practically disappears.

David and Wilma Dickson of the University of Miami take a different stand and present microscopic sections of a fetus with cleft palate which clearly demonstrates the palatal aponeurosis.





Inferior fetal palatal dissection shows the levator veli palatini anterior muscle entering the palate.



Inferior fetal palatal dissection shows the tensor and levator muscles entering the palate.

*The levator palatine is also responsible for lateral pharyngeal wall motion in speech.*

*Dickson:*

The *levator palatini muscle* is a cylindrical muscle which has its origin from the petrosal portion of the temporal bone anteromedial to the entrance of the carotid canal. From that point the muscle courses inferior to the Eustachian tube and occasionally gives off a few fibers to the cartilaginous wall of the tube. Just before entering the velum, the muscle passes lateral to the torus tubarius, which is the enlarged inferior end of the Eustachian tube cartilage. From that point the muscle passes into the soft palate with fibers spreading over the posterior three-fourths of the velum. These fibers cross toward the midline where they join with fibers from the opposite levator palatini muscle. Within the velum the levator palatini muscle is the most superior muscle with the exception of the azygos uvulus.

The levator palatini muscle displaces the velum in a superior and posterior direction. The left and right levator muscles form a sling widely separated superiorly and interlacing in the velum inferiorly. From studies of this muscle it is probable that the course of the muscle from velum to cranial base is in a direct line with motion of the velum toward velopharyngeal closure for speech.

*Fára and Dvořák:*

[In the newborn cleft this muscle was] considerably hypoplastic bilaterally; sometimes, [it] did not exceed half the muscle thickness in normal newborn children. The thinner the muscle belly, the thicker the layer of loose connective tissue in its bed [Veau, 1931] . . . The posterior bundles ran posterolaterally toward the . . . palatopharyngeus, penetrat[ing] the posterior palatine arch [near] the base of the uvula. . . The medial bundles radiated like a fan into the margin of the cleft. The anterior bundles were either . . . attached by a triangular tendinous area coming laterally from the posterior nasal spine to the posterior edge of the palatine plate ( . . . [and also] into the tensor tendon), or . . . directly linked up with the compact part of the tensor tendon. . . In the less serious forms of clefts . . . some anterior bundles of the levator advanced for some millimeters along the cleft margin of the palatine plate as a part of Veau's "cleft muscle."

*Dickson:*

The *palatoglossus muscle* is slender and arises from transverse bundles of the tongue. It passes through the palatoglossal arch and into the inferior middle portion of the velum. It would appear capable of lowering the velum or raising and retracting the tongue. However, electromyographic evidence suggests that the palatoglossus muscle is active in tongue function but not in velar lowering. It may also act to narrow the opening between the mouth and pharynx during swallowing.

*Fára and Dvořák:*

[This muscle in the newborn cleft] passed [forward] in the cleft margin to the posterior edge of the palatine plate . . . [and] extended, in many cases, beyond the posterior edge . . . [to become] inserted more frontally (3 to 5 mm) in the oral periosteum of the hard palate.

*Dickson:*

The *palatopharyngeus muscle* arises from the lateral and posterior pharyngeal walls and inserts into the velum. Its superior fibers arise from a complex intermingling with the superior constrictor muscle. These fibers arise from a level just below the most superior fibers of superior constrictor and pass horizontally into the posterior three-fourths of the soft palate, inferior to the fibers of the levator palatini muscle. Lower fibers of the palatopharyngeus muscle arise from the inferior part of the lateral wall of the pharynx, medial to the middle and inferior constrictor muscle. Some fibers may arise from the thyroid cartilage. These lower fibers pass through the palatopharyngeal arch to insert into the velum.

While there is little electromyographic evidence regarding the function of this muscle, it is felt that it decreases the distance between the palatopharyngeal arches during swallowing and also acts to lower the velum.

*Fára and Dvořák:*

[This muscle in the newborn cleft was] relatively well developed. . . . Even though the smaller part of its fibers ended in the cleft margin, most of its bundles passed forward along this margin and inserted on the posterior edge of the palatine plate. . . . Some fibers advanced along the cleft margin, together with the bundles from the levator as a part of the "cleft muscle." . . . The circular fibers of the posterior pharyngeal wall were difficult to distinguish from the bundles of the superior constrictor. . . . (50 percent of our cases) had condensation and even some thickening of the circular fibers . . . cross [ing] in the Passavant pad, which bulged visibly in the autopsy material. This was not seen in any case of sectioned normal newborns.

The powerful insertion of the *pars pterygoidea* extended from the hamulus across the medial plate of the pterygoid, as far as the lateral portion of the aponeurosis.

*Dickson:*

The *azygos uvulus muscle* is a well-developed unpaired muscle traversing the velum in an anterior-posterior direction superior to all other muscles of the velum. Its origin is apparently from the anterior velar aponeurosis with occasional tendinous fibers passing forward to the posterior nasal spine. It

passes posteriorly through the superior midline of the velum to insert into the uvula.

The function of this muscle has never been studied. However, this muscle is thickest as it passes superior to the main mass of the levator palatini muscle, approximately two-thirds of the way back in the velum from the hard palate. This is the portion of the velum which is most displaced in velopharyngeal closure. During velopharyngeal closure, particularly in the male, the superior surface of the velum become convex from anterior to posterior, creating a considerable hump in the superior portion of the velum above the level of contact of the velum with the posterior pharyngeal wall. This superior hump in the velum has been called the "levator eminence" but is more likely to be created by contraction of the uvulus muscle which could thicken the velum in this area. Therefore, at least until some further evidence develops, it is probably more reasonable to call this area the uvular eminence. This could create some confusion but no more so than is presently available in velopharyngeal terminology.

uvulus muscle  
is the "patella"  
of the levator  
of knee

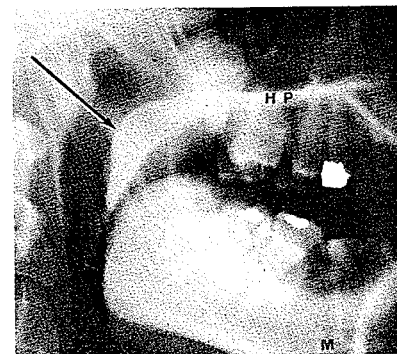
It is of interest that as early as 1880 R. Falscon commented on the bulk of the musculus uvulae. In 1969 R. Pigott confirmed with nasendoscopy the presence and importance of the musculus uvulae in speech:

The enormous bulk of the musculus uvulae could be seen. The majority of subjects had a large ridge down the soft palate, occupying the central  $\frac{1}{3}$ , rising to a height almost equal to its width. In other subjects, it was less prominent, but it was never absent. . . . During speech movements. . . the levator sling could be seen to tighten into a bar throwing the convexity of the musculus uvulae bulge up and back to fit into the concavity of the posterior superior pharyngeal wall. . . . The possible role of the musculus uvulae in giving a suitable convexity to the upper surfaces of the levator at the point of contact . . . is of particular importance in the light of Broomhead's finding that this muscle is not supplied by the pharyngeal plexus, but by the lesser palatine nerve.

As part of the fruit from David and Wilma Dickson's research on the morphology of the musculus uvulae, Nabil A. Azzam and David P. Kuehn of the University of Iowa reported their findings, confirming the Dicksons' observations in the 1977 *Cleft Palate Journal*. They noted:

The *morphology* of the *musculus uvulae* was studied utilizing detailed gross anatomical dissections and *histological sectioning* of the soft palate in seven adult human cadavers. The results indicated that the musculus uvulae is paired. . . . Each bundle takes origin lateral to the midline from the

tendinous palatal aponeurosis posterior to the hard palate and just anterior to the insertion of the levator veli palatini muscle. The two bundles converge in an area overlying the sling of the levator muscle and course along the dorsum of the soft palate, terminating as two separate bundles which subdivide and insert between the mucous glands of the uvula proper into the connective tissue and basement membrane of the mucosa. Because of its location and size, it appears that contraction of the musculus uvulae would add bulk to the dorsal surface of the elevated soft palate [arrow], thus aiding in *occlusion* of the *velopharyngeal portal* during speech and deglutition.

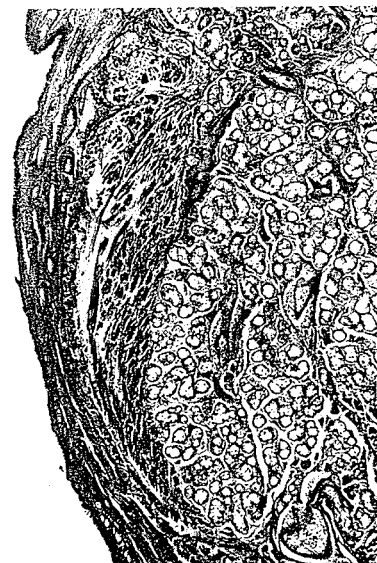
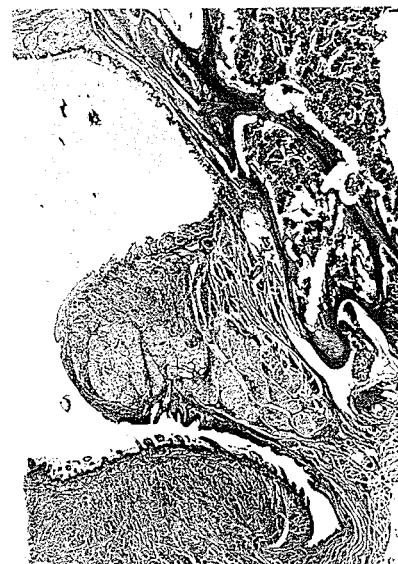


*Fára and Dvořák:*

[This muscle in the newborn cleft] passed in the cleft margin and its bundles intermingled with those of the palatopharyngeus and the levator.

A more detailed description of the uvular muscle in cleft palate was given by Elizabeth Ann Latham at the Third International Congress on Cleft Palate in Toronto, based on serial histological sections of two postmortem infants and a Plexiglas reconstruction.

The *Musculus Uvulae* was divided by the cleft into its bilateral component muscle bundles which were seen on the medial border of each palatal shelf. The *M. Hemiuvulae* originated anteriorly in relation to the border of the hard palate formed by the palatine bone. Here it blended with other velar muscles. It stayed in the medial border as it coursed posteriorly beneath the mucous membrane and turned more onto the inferior border prior to entering and dispersing in the hemiuvula. A coronal section through the cleft soft palate at the level of the pterygoid hamulus showed the hemiuvular muscle on the medial border of the palatal shelf between the mucous membrane and deeper glandular tissue. This was clearly seen at a higher magnification which showed the muscle fibers sectioned rather obliquely.



**PHARYNGEAL MUSCLES**

The constrictor pharyngeus muscles consist of superior, medial and inferior portions. The superior portion of the constrictor complex may be involved in velopharyngeal function. The superior constrictor muscle fibers have their origin at the hamulus and the adjacent pterygomandibular raphe. Fibers pass around and through the lateral pharyngeal wall and "join corresponding fibers of the opposite side in a tendinous strip, the pharyngeal raphe, which runs in the midline from the pharyngeal tubercle of

the occipital bone throughout the entire length of the pharynx.” To complete the circle of this “U,” Whillis found that some of the upper fibers inserted into the palatal aponeurosis formed an actual palatal-pharyngeal sphincter. In 1948 Whillis at Guy’s Hospital patiently pointed out to me in detail in cadaver specimens the muscles involved in the “palato-pharyngeal sphincter.” He emphasized the importance of the superior pharyngeal constrictor’s lateral attachments into the soft palate, which he had found in 1936 under Wardill’s none-too-gentle prodding.

*Dickson:*

While the *superior constrictor muscle* undoubtedly has an important function in narrowing the pharynx during swallowing, its function in velopharyngeal closure is debatable. While Passavant’s ridge has never been studied electromyographically, it is quite probable that this inbulging of the lateral and posterior pharyngeal walls seen in many patients with cleft palate and other forms of velopharyngeal insufficiency is a function of the most superior fibers of superior constrictor which enter the velum. Some writers have suggested that the superior constrictor is also responsible for the lateral wall motion seen in normal velopharyngeal closure. However, the fact that Passavant’s ridge occurs below the normal site of velopharyngeal closure, the fact that the lateral pharyngeal walls move medially and posteriorly rather than medially and anteriorly in normal velopharyngeal closure and the fact that the superior constrictor lies at the level of the hamulus, rather than higher in the nasopharynx, suggests that it probably is not involved in velopharyngeal function during speech. Available electromyographic evidence supports this contention. The continuing debate regarding the role of this muscle in lateral wall movement during speech has been reinforced by findings of considerable variability in patterns of motion of the velum and pharynx from subject to subject in radiographic research.

The *salpingopharyngeus muscle* consists of a few fibers arising from the inferior portion of the palatopharyngeus muscle which passes through the lateral pharyngeal wall superiorly to attach to the torus tubarius. These muscle fibers are few in number and frequently absent in normal specimens. Commonly, the salpingopharyngeal fold through which these fibers pass consists entirely of gland and loose connective tissue.

*Fára and Dvořák:*

Fára noted no difference in the pharyngeal muscles in the newborn cleft and the normal.



*Dickson:*

In summary, from a functional standpoint it is apparent that the levator veli palatini muscle is the principal and quite possibly the only muscle to function for elevation of the velum in speech. In addition, the medial and posterior motion of the lateral pharyngeal wall during velopharyngeal closure for speech must be accounted for. Since the salpingopharyngeus muscle is frequently absent in the normal, and since the superior constrictor muscle is probably too low to account for motions seen in the nasopharynx, and because available electromyographic evidence is not supportive of the contention that this muscle acts during velopharyngeal closure for speech, some other mechanism must be found. A likely answer is to be found by noting the slinglike arrangement of the entrance of the levator muscles through the pharynx and into the velum. As was noted above, the levator passes lateral to the torus tubarius prior to entering the velum. Thus, on contraction it would be quite likely that the levator muscles would not only lift the velum but would displace the lateral pharyngeal walls and tori tubarius in a medial and posterior direction. It is, therefore, a most reasonable hypothesis that both the pharyngeal and velar components of normal velopharyngeal closure for speech are a function of the levator palatini muscle.

With regard to the cleft condition, here are a few of Fára's deductions:

The differences between the normal and the cleft arrangement of the muscles of velopharyngeal closure are considerable, but . . . occur because the muscles extending toward the central line of the soft palate cannot attach themselves to the *punctum fixum* in the midline of the velum; so they insert at some substitute points. These points, however, prevent the muscles from becoming fully functional; therefore, their development is retarded. With the preservation of normal origins, the atypical insertions and the hypoplasia of the muscles are the main pathological features in the cleft velum. . . .

The levators in clefts illustrate clearly the effect of a morphological disorder on function, not only from the point of view of quantity but also of quality. Indeed . . . the effect of the activity of these muscles in a cleft palate is almost opposite to that in a normal one. While the muscles of both sides normally join in the raphe to form a sling lifting the palate upward, in cleft palates each muscle pulls its own half of the soft palate in an entirely different direction (i.e. supero-laterally), causing a further widening of the cleft.



*Robert Shprintzen*

Robert Shprintzen of Montefiore Hospital, the Bronx, New York, of Spanish descent with a Russian rinse, made an interesting study, with McCall, Skolnick and Lencione.

The frontal and lateral cinefluorographic views of five normal subjects performing speech, blowing, and whistling tasks were synchronized in order to observe where the greatest degree of medial movement in the lateral aspects of the pharyngeal walls was occurring in relation to structures observed in lateral view. The results . . . indicate that for all five subjects, maximal medial excursion in the lateral walls of the pharynx occurred at the level of the full length of the velum and hard palate, well below the levator eminence. It is hypothesized that the observed interaction may be due to the select contraction of those fibers of the superior constrictor muscle which enter the velum via the lateral walls and those fibers attached to the pterygoid plates, as well as levator muscle activity.

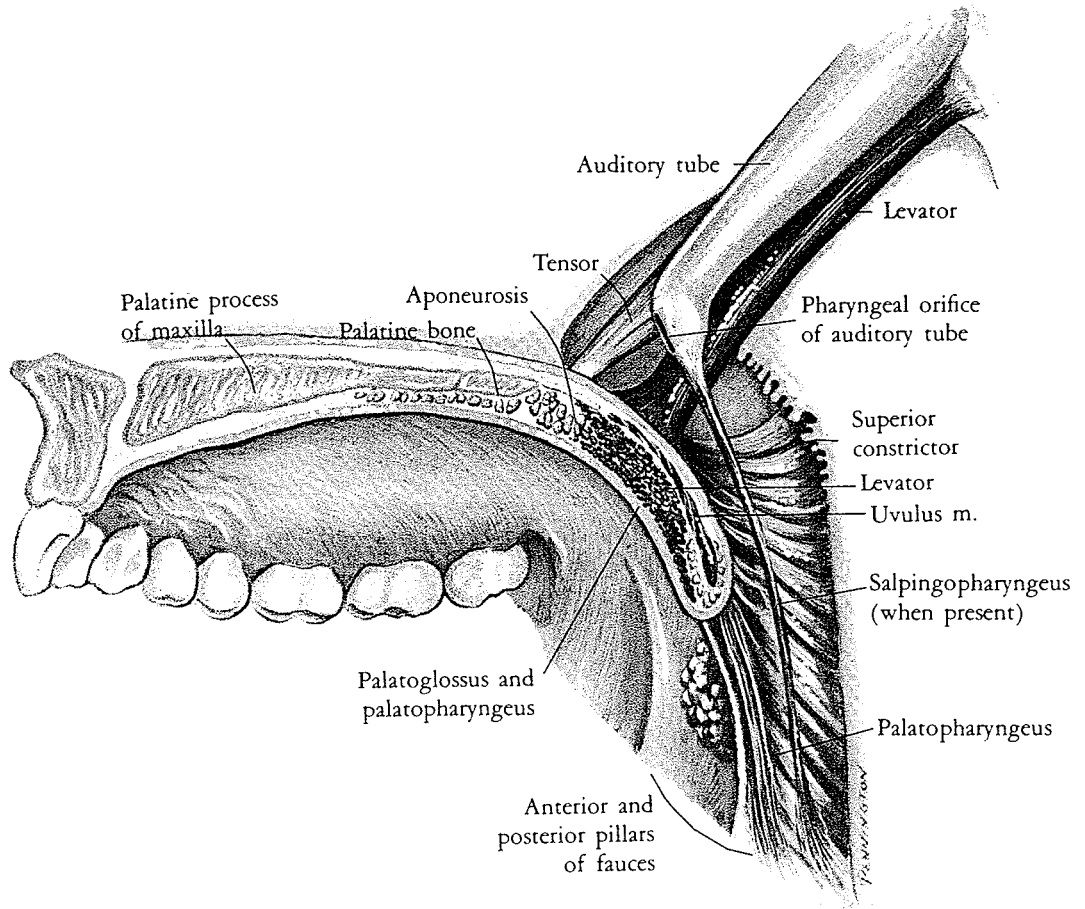
This led to their suggestion of the possibility that

the kinesiological observations of this study would seem to indicate that both the levator and the superior constrictor are necessary to closure.

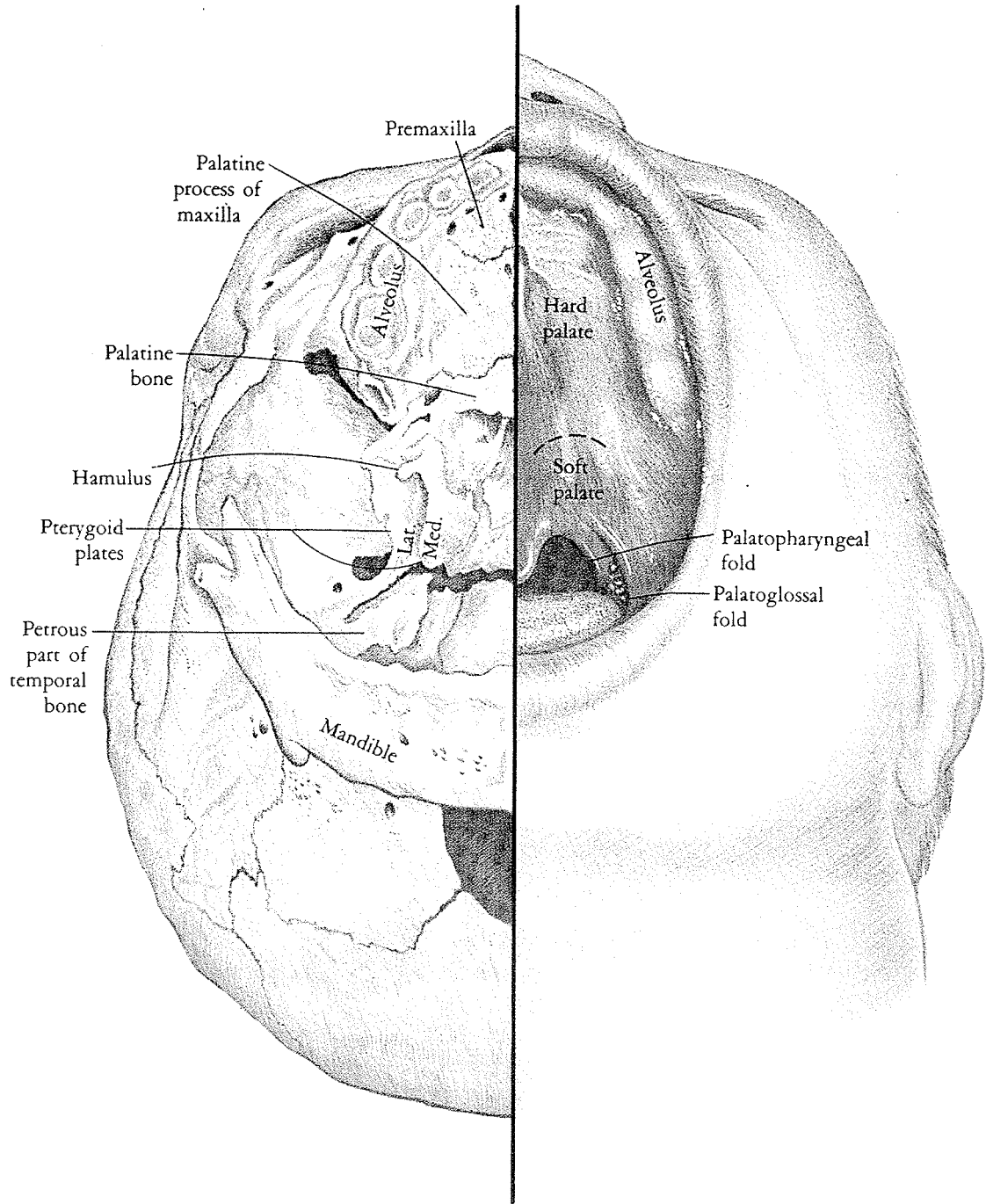
## N A S O P H A R Y N X

The pharynx is related to the sphenoid bone and to the basilar part of the occipital bone above, and descends into the esophagus. Anteriorly, it opens into the nasal and oral cavities and the larynx; posteriorly, from before backward, it is related to the prevertebral layer of fascia, prevertebral muscles and upper six cervical vertebrae. Laterally, such structures as the styloid process and its associated musculature, the medial pterygoid muscle, the carotid sheath and its contents and the thyroid gland are present.

The nasopharynx is actually the posterior portion of the nasal cavity communicating with the oropharynx through the pharyngeal isthmus or hiatus and bounded by the palatopharyngeal arches, the soft palate and the posterior wall of the pharynx. Embedded in the mucous membrane of the posterior nasopharynx is the mass of lymphoid tissue known as adenoids. The pharyngeal isthmus is encircled by a lymphatic ring, the nasopharyngeal tonsil (adenoids) above, the palatine tonsils laterally and the lingual tonsils below.

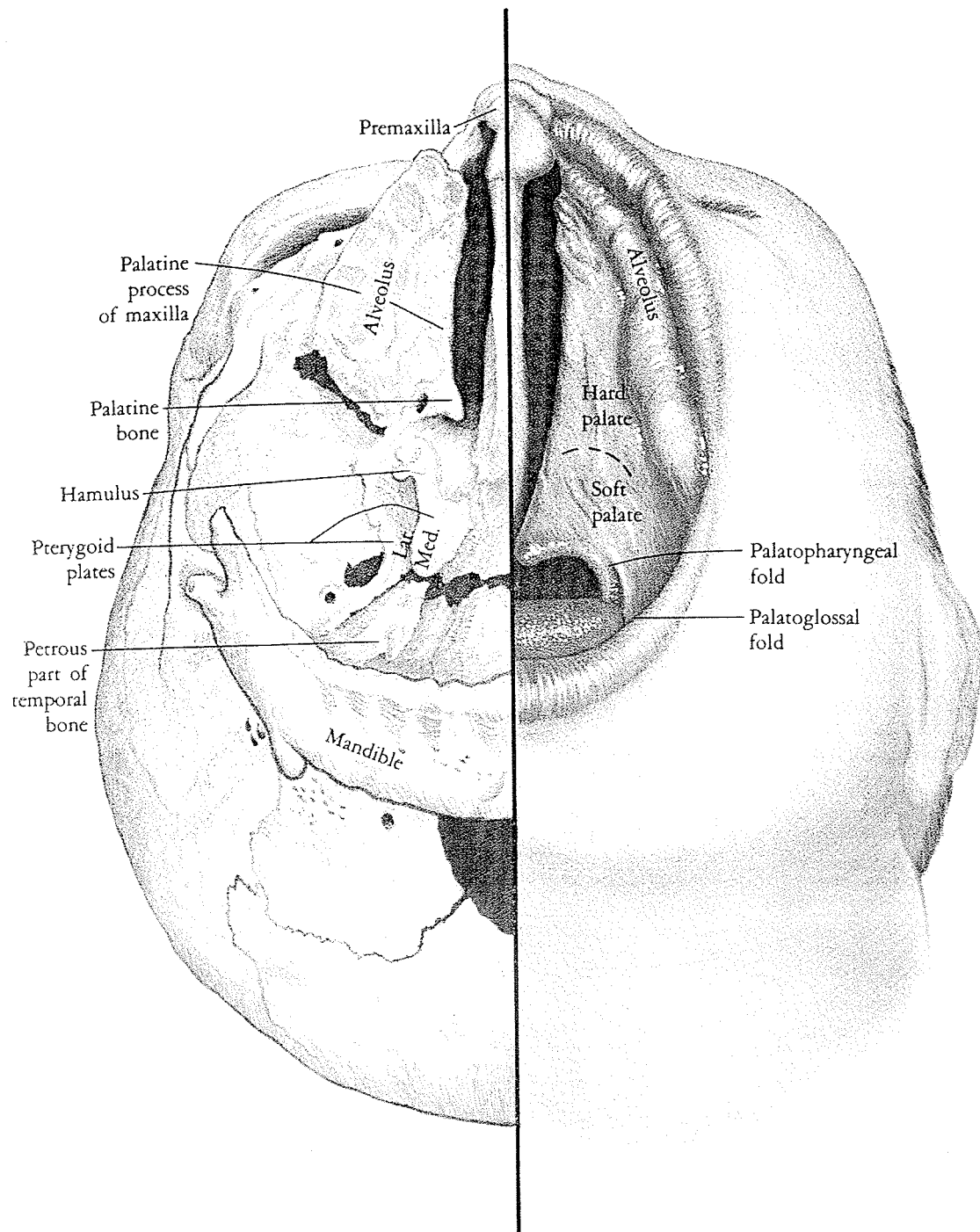


Dissected sagittal view—normal adult palate and pharynx showing auditory tube, pharynx and musculature of the velum (palate).

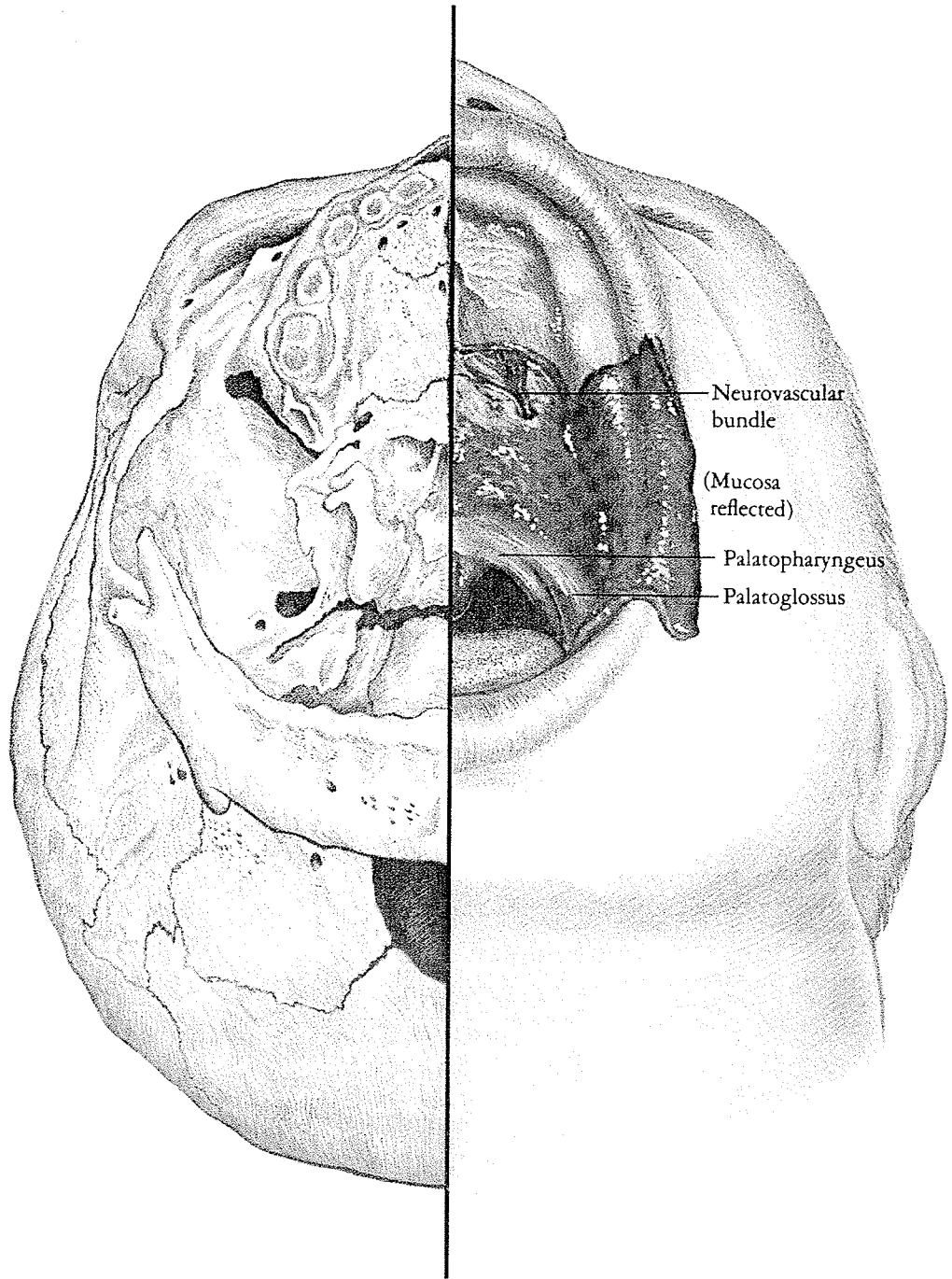


NORMAL

Inferior view--newborn palate and related structures

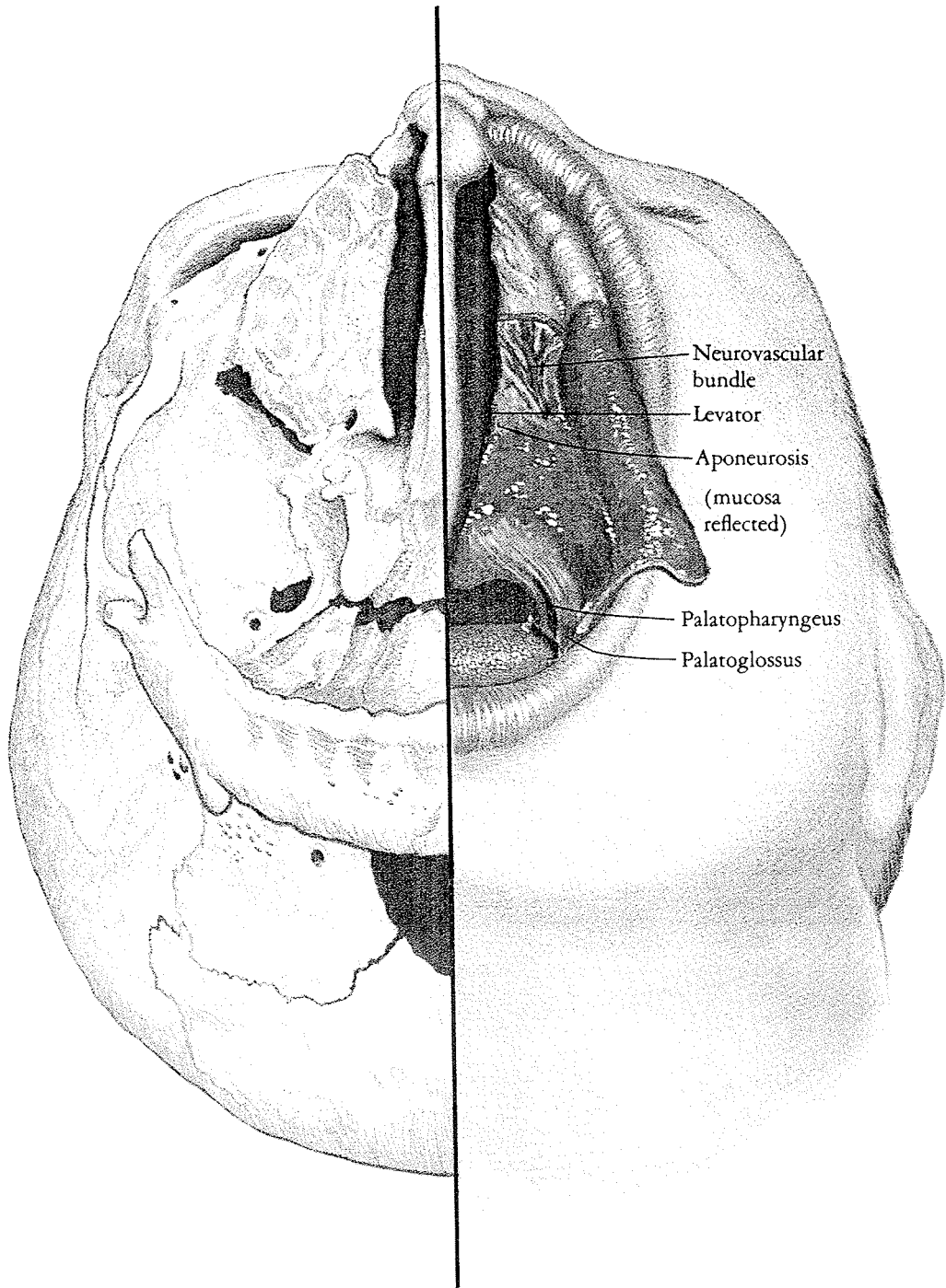


CLEFT

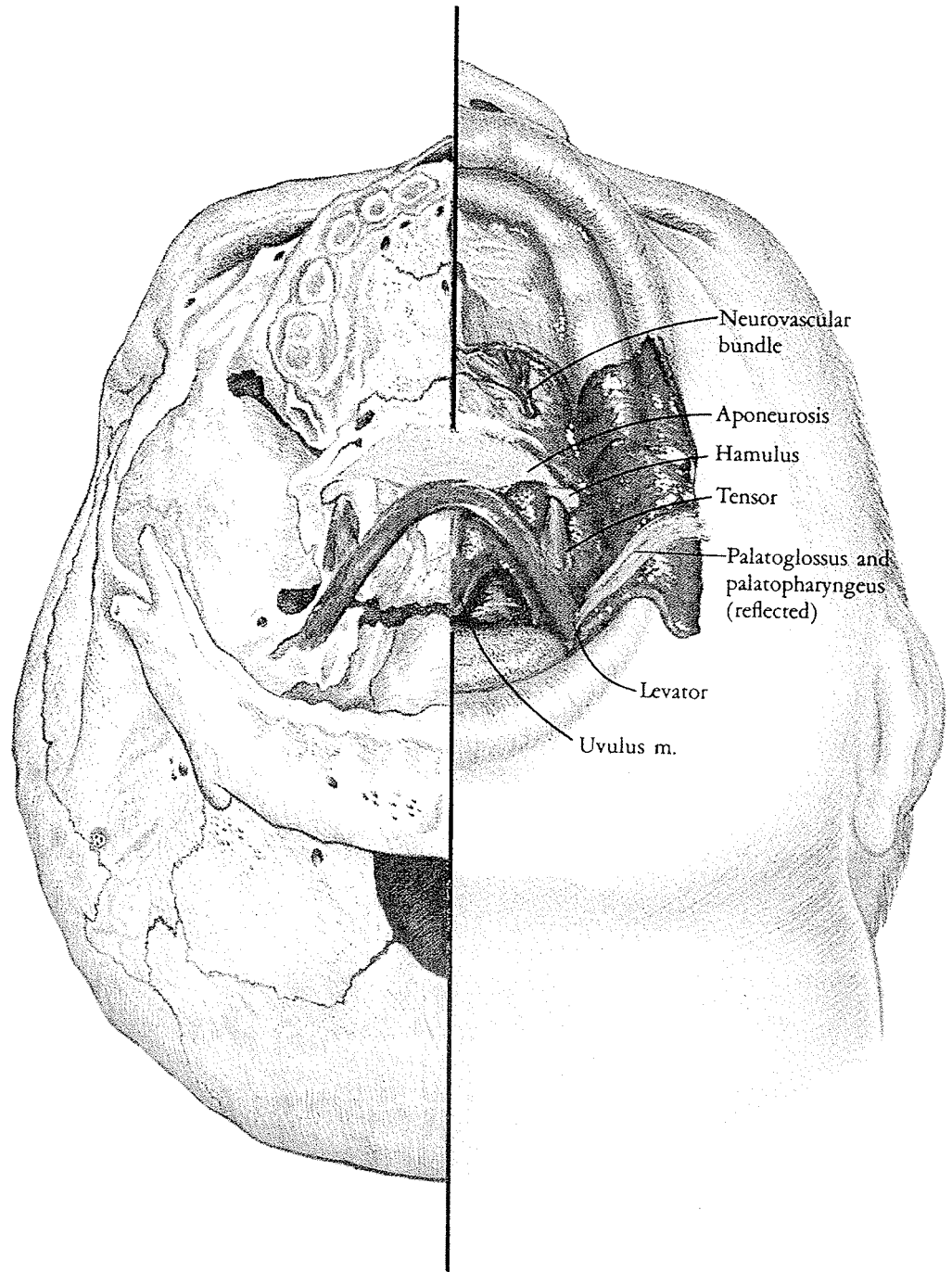


NORMAL

Mucosa reflected and palate dissected.

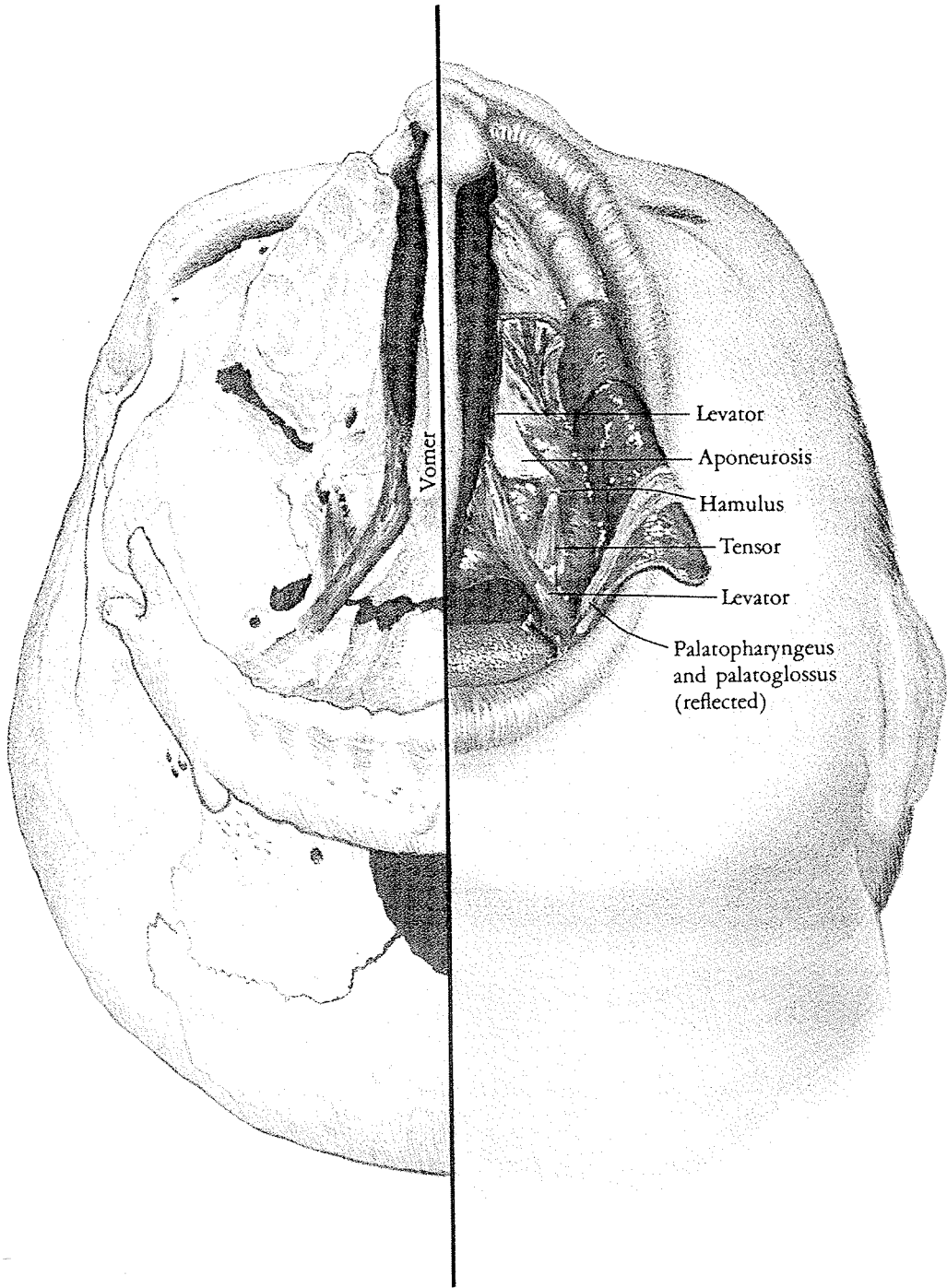


CLEFT

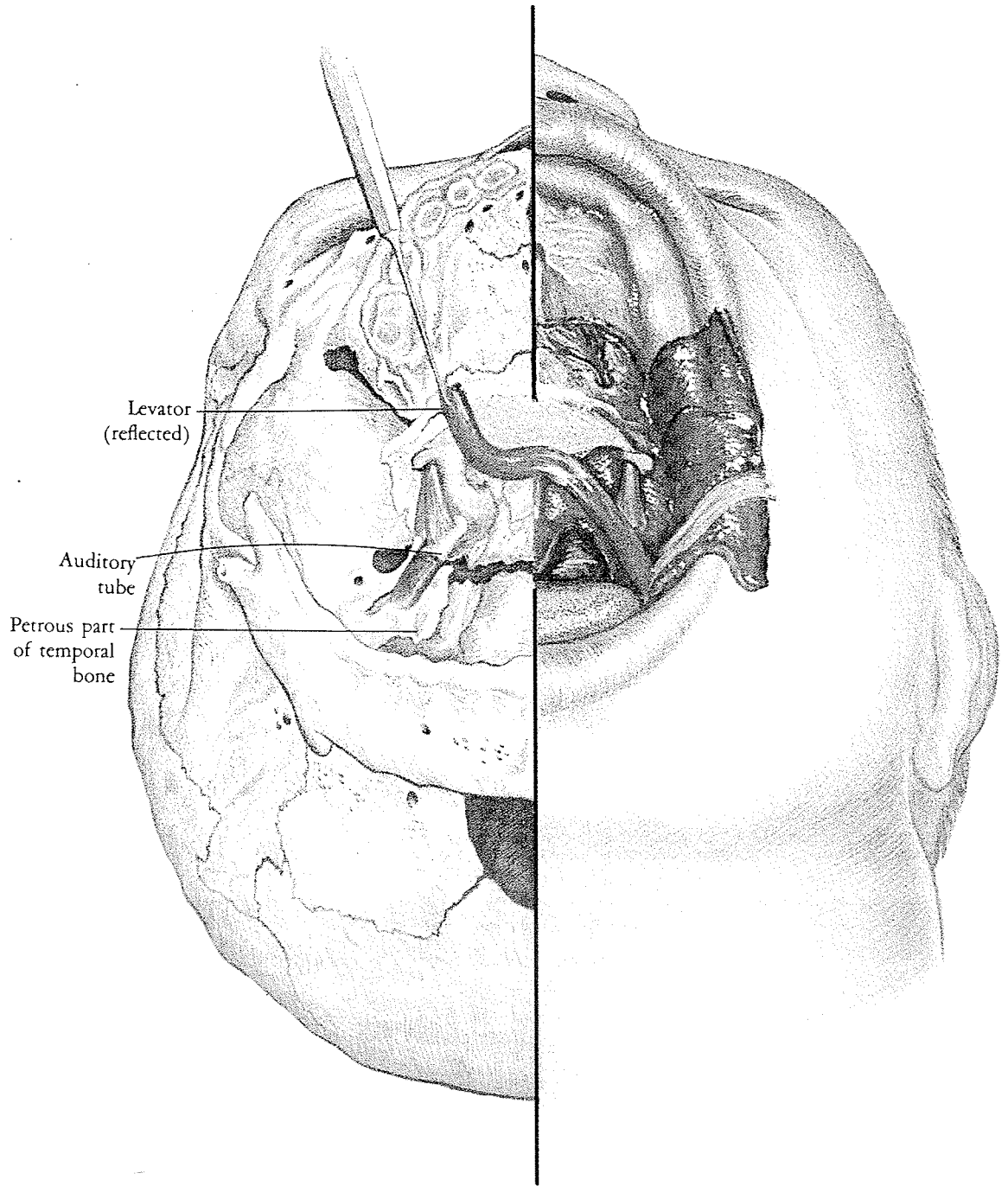


NORMAL



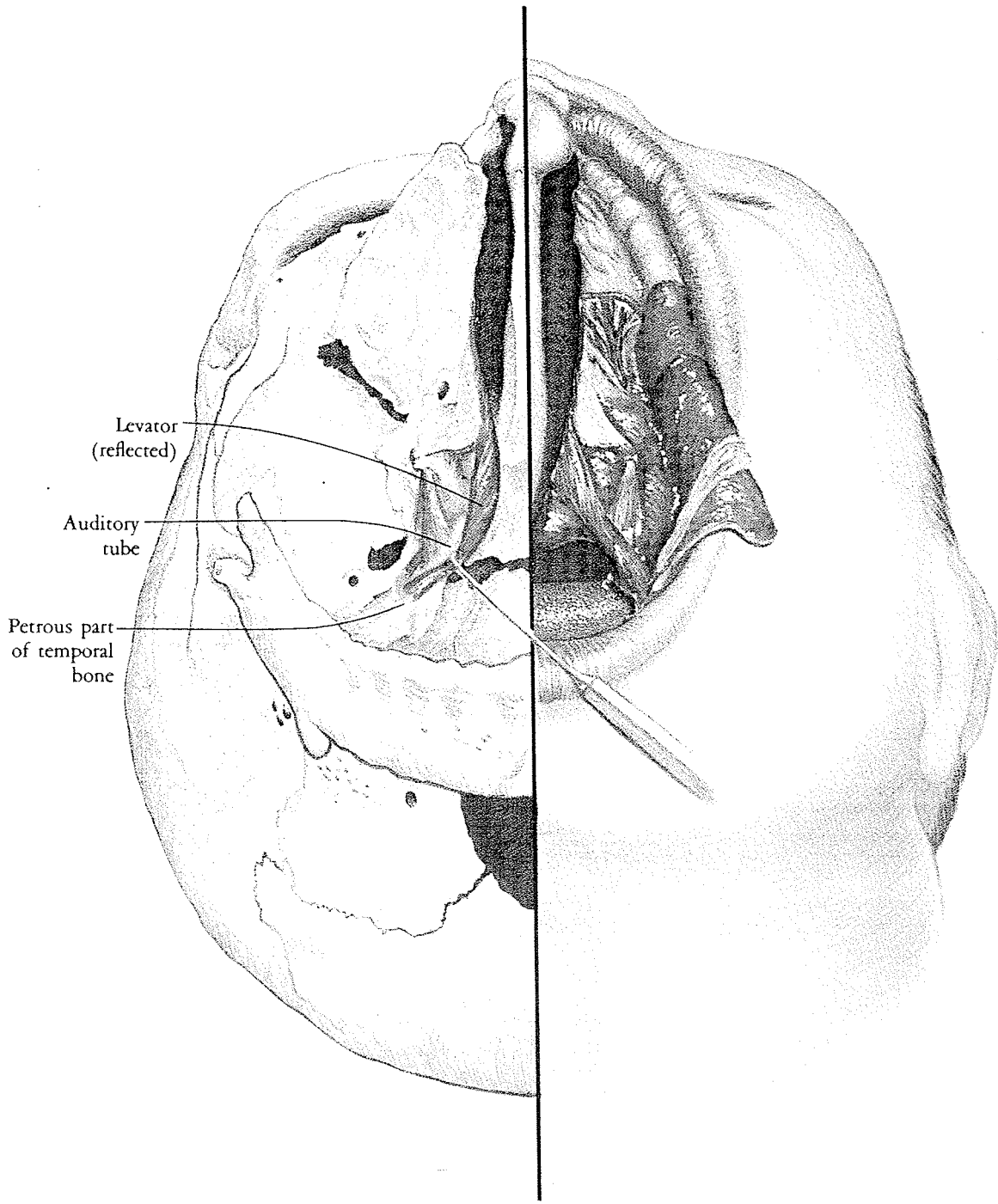


CLEFT

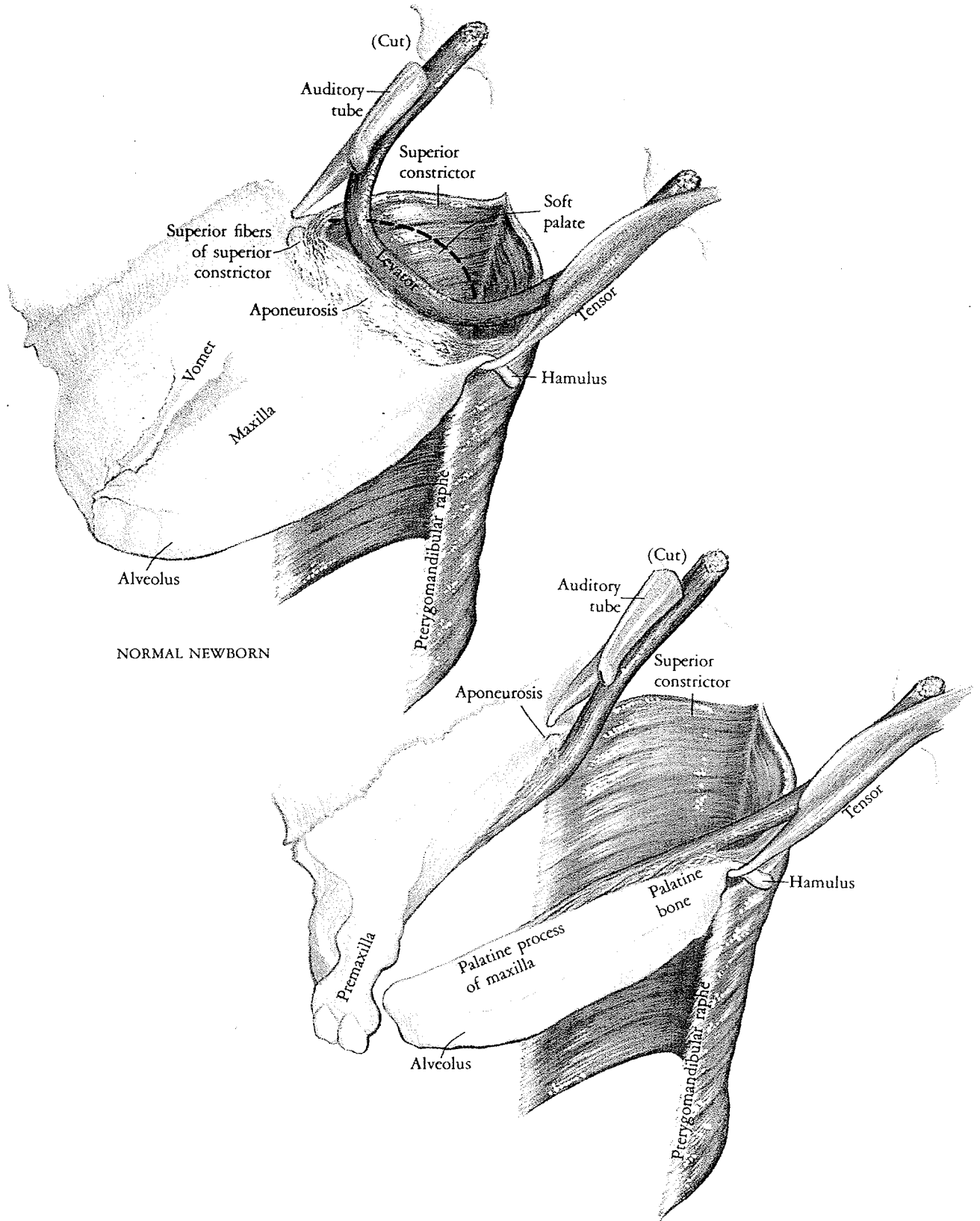


NORMAL

Palate dissected to show auditory tube and related structures.



CLEFT



Superolateral view of the palate and pharynx, showing position of auditory tube and the tensor veli palatini, levator veli palatini and superior constrictor muscles.

The triangular pocket in each lateral wall of the nasopharynx is called the tonsil and is formed by the diverging pillars of the tonsil, the anterior pillar formed by the palatoglossus muscle and the posterior pillar formed by the palatopharyngeus muscle. Cradled between the pillars in each fauces lies the palatine tonsil.

The most significant structure in the lateral wall of the nasopharynx is the auditory, or Eustachian, tube.

## THE EUSTACHIAN TUBE

### *Dickson*

Wilma Maue-Dickson of the University of Miami School of Medicine, a compulsive, analytical perfectionist, played violin in the Exeter Symphony Orchestra in England, climbed Kilimanjaro, and saw her first severe unoperated unilateral cleft lip while in the Peace Corps in Ethiopia. David Dickson was her major professor in head and neck physiology at the University of Pittsburgh. Their mutual love of teaching, research and human communication overcame this obstacle to their relationship and resulted in marriage. Her compulsion not just to observe craniofacial pathologies but to speed their extirpation was probably "fueled" most effectively the day she walked into the storage room for some 15,000 human embryos and fetuses at the University of Pittsburgh's Cleft Palate Center in 1969 and recognized that a gold mine of information for living children with facial anomalies lay in that 15'  $\times$  15' room.

Since then she has carried out numerous microscopic dissections of fetal heads which have placed her among the foremost head and neck anatomists. She has a special affection for the Eustachian tube. This is her 1976 position:

In the sixth century B.C., a man named Alcmocon became interested in how goats "breathe through their ears" and gave us our first description of the structure of the auditory tube, which he felt served this function (Macbeth, 1959). Later, during the sixteenth century, the tube was described in further detail by the Italian anatomist, Bartolomeo Eustachio, for whom the tube was named. He was one of the first to describe accurately its structure, course, and relations. He compared the tube to a quill pen and divided its bony and cartilaginous parts, but did not hazard a guess as to its function.



*Wilma Dickson*



*Bartolomeo Eustachio*

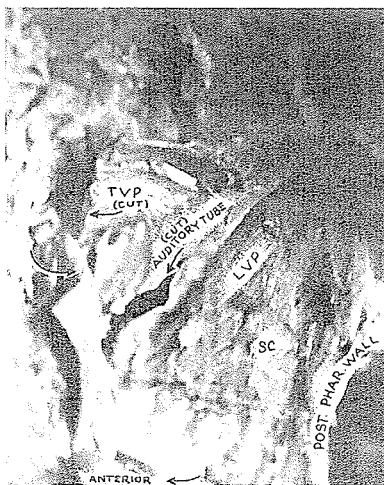
out of respect  
for old Eustachio  
Eustachian Tube  
should be spelled  
with a capital  
E!

The anatomy of the adult auditory (Eustachian) tube has been carefully documented, as has its histology, and large strides have been made in our understanding of its embryologic and fetal development. It courses from the nasopharynx to the middle ear in a posterior lateral superior direction. The anteromedial two-thirds of the tube, called the cartilaginous portion, consists of a superomedial cartilaginous wall and an inferolateral membranous wall. The posterosuperior one-third of the tube passes through the petrous portion of the temporal bone and is referred to as the osseous portion, protympanum, or semicanal. The division between the cartilaginous and osseous portions is marked roughly by a constriction called the isthmus. It has been well documented (Sucheston and Cannon, 1971) that the membranous part of the tube is lined with pseudostratified, ciliated, columnar epithelium, while the medial lamina and roof are lined with cuboidal epithelium. Muco-serous glands and goblet cells are present at the pharyngeal orifice and in the mid-portion of the tube, but are absent at the tympanic orifice. Lymphoid tissue is present at both orifices but is far less abundant in the mid-portion of the tube.

The auditory tube is of particular interest because it provides a dynamic link between the nasopharynx and middle ear. The middle ear has no direct outlet via any other route than the auditory tube. The auditory tube, therefore, provides a mechanism whereby pressure can be equalized across the tympanic membrane. The cartilaginous part of the tube is roughly elliptical in configuration and is normally collapsed, but opens during swallowing, coughing and sneezing. The osseous portion is obligatorily open.

The tube is also of clinical interest because it provides an avenue for the outflow of fluids from the middle ear. Unfortunately, it also provides an access route for the spread of infection from the pharynx to the middle ear and mastoid area.

The specific mechanism for opening the auditory tube involves an interaction of muscular force, pressure differential, ciliary action, and possibly the aid of a surface tension-reducing substance such as surfactant. Muscular activity associated with tubal opening has been a source of debate for years. Normal tubal function has been associated at one time or another with at least six different muscles, including the superior constrictor, the palatopharyngeus, the medial pterygoid, the lateral pterygoid, the levator veli palatini, and tensor veli palatini. Of this group, only the latter muscles have direct attachments or important spatial relationships to the tube. Fortunately, this debate about the function of the muscles associated with the auditory tube has been settled by an elegant study conducted by Arnold Rich [experimental pathologist at Johns Hopkins Hospital] in 1920, which indicated clearly that the tensor veli palatini is the sole muscle responsible for tubal opening at the isthmus. More recent EMG studies have supported this fact.



Adult lateral head dissection shows the tensor reflected, the auditory tube and the levator muscle.

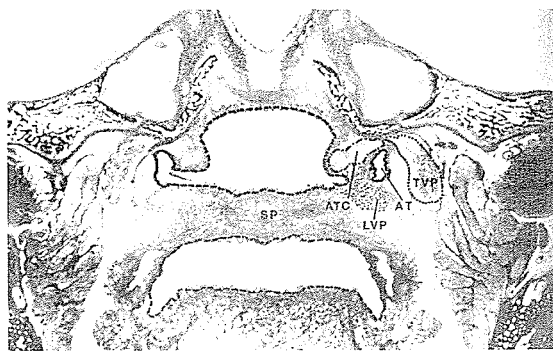
Another debate on tubal function concerned innervation of the tensor veli palatini muscle, which has been variously ascribed to cranial nerves V, VII, X, and XI. However, Rich (1920) demonstrated convincingly that tensor veli palatini is innervated via the mandibular branch of cranial nerve V.



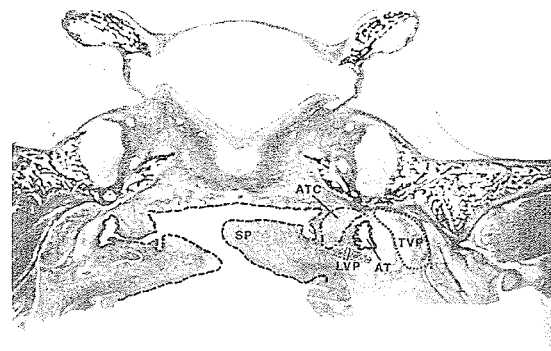
Arnold Rich

Levator veli palatini may assist in opening the pharyngeal orifice of the tube by moving the torus tubarius, the cartilaginous expansion of the tube at its pharyngeal orifice, posteriorly and medially during swallowing. However, this action would not aid in tubal opening at the isthmus.

*Cleft Palate:* The auditory tube is of further clinical interest because of its apparent involvement in cleft palate. It has been demonstrated that babies with cleft palate have an almost 100 per cent incidence of middle ear effusion, frequently accompanied by hearing loss. Numerous studies have been conducted to ascertain why this is true; none have completely solved this puzzle, but it clearly involves abnormal Eustachian tube function. A recent anatomical study conducted by Maue-Dickson (1975) indicated that human fetuses with cleft palate consistently show the following characteristics:



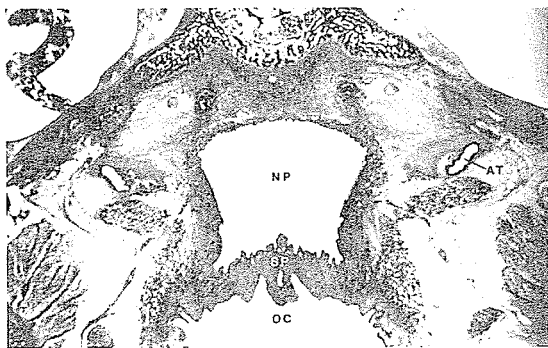
Non-cleft



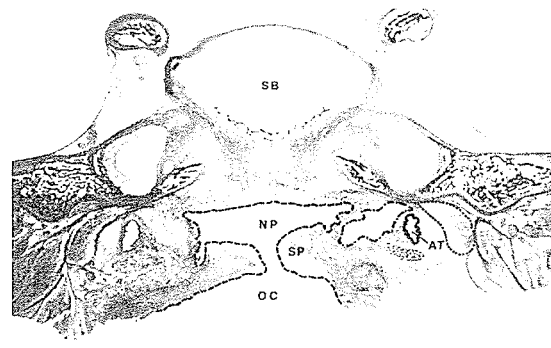
Cleft

- SP Soft palate
- ATC Auditory tube cartilage
- AT Auditory tube
- LVP Levator veli palatini
- TVP Tensor veli palatini

As compared to the non-cleft, there are narrower and smaller auditory tube lumina, which are more widely separated than in normals.



Non-cleft

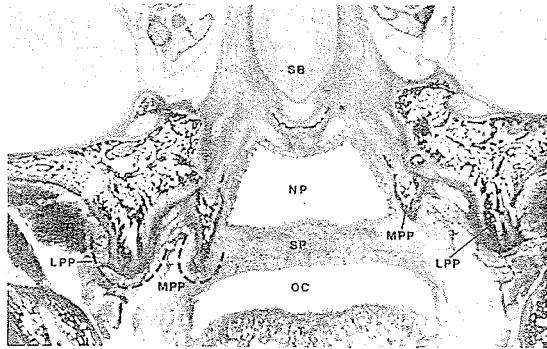


Cleft

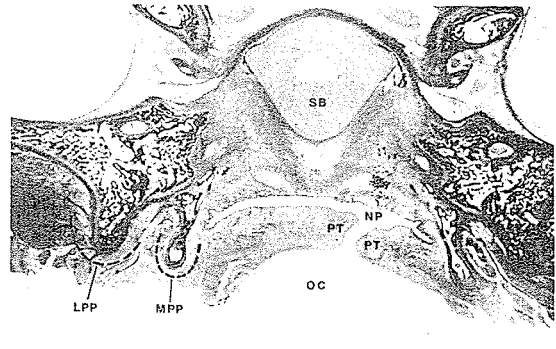
- SB Sphenoid bone
- NP Nasopharynx
- SP Soft palate
- OC Oral cavity
- AT Auditory tube

The greatly enlarged auditory tube cartilages are also more widely separated than in normals.

- LPP Lateral pterygoid plate
- MPP Medial pterygoid plate
- SB Sphenoid bone
- NP Nasopharynx
- SP Soft palate
- OC Oral cavity
- T Tongue
- NP Nasopharynx
- SP Soft palate
- SB Sphenoid bone
- AT Auditory tube
- ATC Auditory tube cartilage



Non-cleft

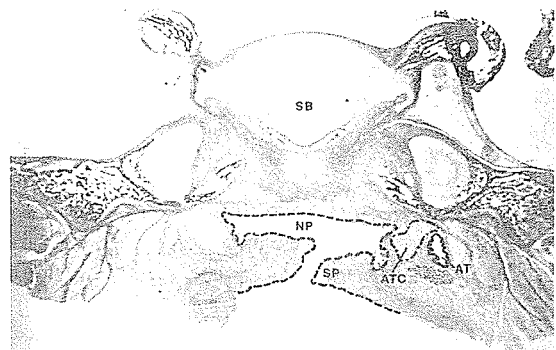


Cleft

There are more widely separated pterygoid plates than in the normals.



Non-cleft



Cleft

There is significantly reduced pharyngeal height, but greatly increased pharyngeal width.

One conclusion from these data is that the space between the lateral pharyngeal wall and the side wall of the cranium is substantially reduced and that *the tube may suffer a mechanical disadvantage as a result*. This problem may be reduced by craniofacial growth, which might relieve stress on the tube. This suggestion is consistent with the observation that children with cleft palate typically have reduced middle ear problems within the first few years of life.

Interestingly, while the interruption of the levator veli palatini muscle and of other muscles which traverse the soft palate is obvious in cleft palate, there is no indication that the extra-palatal anatomy of the tensor veli palatini muscle is abnormal in cleft palate, and even in severe cleft of the palate, the presence of a well-defined palatal aponeurosis (the tendon of tensor) can be demonstrated in the palatal rags in histologic sections.

In summary, while the adult structure of the auditory tube is fairly well understood, more information is needed on the specific morphology of the tube and associated musculature in cleft palate. Based on data collected recently, there is a strong suggestion that cleft palate may in fact co-exist with, or even be caused by, cranial base deformity occurring early in the embryogenesis of the involved structures, and that auditory tube malfunction observed clinically in cleft palate may be related to this deformity.

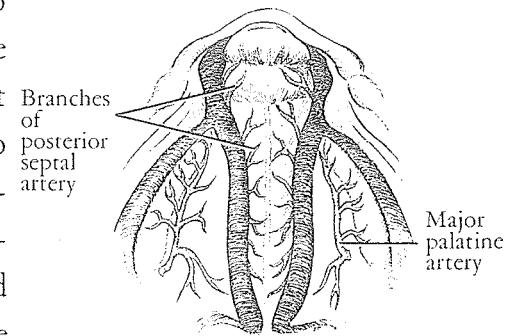
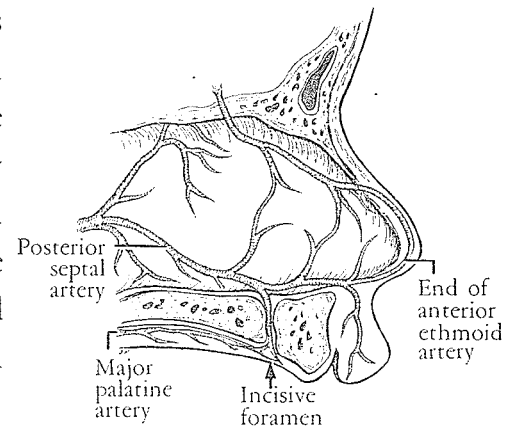


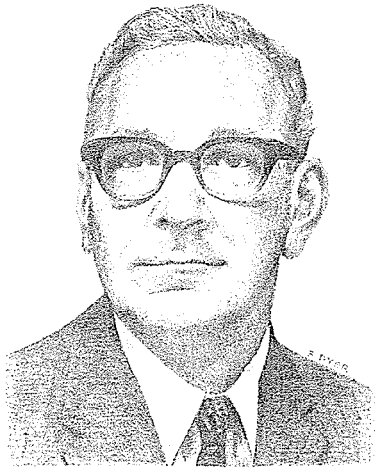
## BLOOD SUPPLY TO THE PALATE

The internal maxillary artery gives off the descending palatine artery, which, in turn, gives off several branches to the tonsils and soft palate. It then passes through the posterior palatine foramen, just above the periosteum, and proceeds forward close to the alveolar margin on each side as the greater (major) palatine artery to the incisive fossa. At that point it sends a terminal branch through the incisive foramen to anastomose with the terminal branch of the sphenopalatine artery. The sphenopalatine artery is also a branch of the internal maxillary artery. One of its branches descends to the incisive canal and anastomoses with the terminal ascending branch of the posterior palatine artery to form the anterior blood supply to the palate. The posterior septal artery arises from the sphenopalatine artery in the roof of the nasal cavity and courses down the groove of the vomer to the incisive foramen. As noted by Brescia, a wide, rich anastomosis is formed between the posterior septal, major palatine and ascending septal branches of the superior labial arteries.

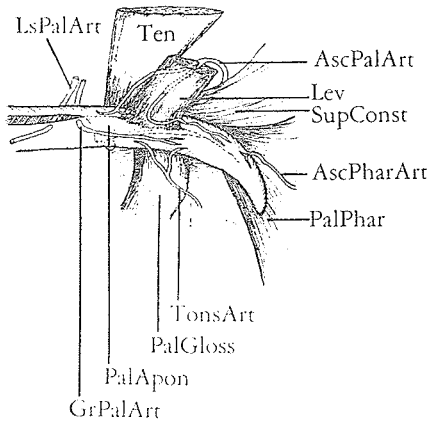
The blood supply to the anterior alveolar process of the maxilla comes from the arterial complex composed of the major palatine, anterior and posterior superior alveolar and branches of the sphenopalatine arteries.

In the complete bilateral cleft lip and palate, as noted by plastic surgeon W. B. Slaughter, along with J. V. Henry and J. C. Berger, the union of the superior labial arteries is lacking; thus they do not contribute to the blood supply of the philtrum. Also absent is the anastomosis of the posterior septal artery with the major palatine artery. Therefore, premaxilla and philtrum must derive their blood supply from the posterior septal artery and to some degree from the lateral and terminal branches of the anterior ethmoid vessels which pass through the columella. Fortunately, in cleft specimens, there is usually one well-developed vessel on either side of the premaxilla in the region where the incisive foramen should have been. Each of these vessels passes anteriorly and inferiorly into the philtrum and forms an arcuate anastomosis across the midline in the inferior part of the philtrum.





Ivor Broomhead



The scholarly plastic surgeon I. W. Broomhead dissected a fetal head for blood supply studies. In 1951, in the *British Journal of Plastic Surgery*, he reported:

The greater palatine artery [GrPalArt] supplies the oral surface of the hard palate and gives off a few fine branches which perforate the horizontal plate of the maxilla to supply the nasal mucosa. It also sends twigs to the gums and palato-glossal arch. The lesser palatine artery [LsPalArt] supplies about the anterior half of the oral surface of the soft palate. A branch of the facial artery, the ascending palatine artery [AscPalArt], is the largest vessel entering the soft palate. It ascends on the lateral side of the superior constrictor muscle to turn downwards and forwards into the soft palate between the tensor [Ten] and levator palati [Lev], giving small branches to these muscles. There are two main terminal branches, the anterior one passing along the anterior border of the levator palati and the posterior one passing through this muscle. On approaching the midline the posterior division turns backwards to run the length of the soft palate to the uvula. Small twigs from the tonsillar [TonsArt] and ascending pharyngeal arteries [AscPharArt] also reach the soft palate. The branches from the tonsillar artery enter along the palato-glossus muscle [PalGloss], and those from the ascending pharyngeal pierce the superior constrictor [SupConst] and pass along the fibres of the palato-pharyngeus muscle [PalPhar].

Broomhead concluded with:

The vascular supply of the soft palate is not endangered by the [standard third-degree cleft palate] operation.

Muriel E. Morley of Newcastle upon Tyne was the speech therapist for many years for Wardill's cleft palate cases. In her little 1962 book, *Cleft Palate and Speech*, she noted:

It is of importance in surgery that the blood supply to the palate enters through the bone and not, as in other parts of the body, through the periosteum. . . . It is therefore possible at operation to strip the periosteum from its bony attachments without interfering with the blood supply.

Stefan Demjen of Bratislava referred to the lack of knowledge of the blood supply to the bony palate:

The hard palate and its mucoperiosteal membrane are supplied by blood from nasopalatine vessels and from the descending palatine arteries. The bony palate is said to have an independent blood supply and is thus protected from necrosis following conventional palate operations.

It is important to know whether the presence of a cleft in the palate changes the vascular arrangement. The data are minimal, but in 1974 David Dickson et al. in their "Status of Research in Cleft Palate" reported:

Blood supply: no specific investigations of the blood supply to the cleft palate have been found except for Frederiks, who reported slight differences between cleft and non-cleft.

Edith Frederiks of Leiden, the Netherlands, did note in 1972:

In the secondary palate the existence or absence of a cleft makes little difference to the vascular pattern.

Yet in the 1973 "State of the Art" report on clinical research in cleft lip and cleft palate, Spriestersbach, Dickson, Fraser, Horowitz, McWilliams, Paradise and Randall proposed the likelihood of a variability in arterial arrangement in cleft and non-cleft humans. This was confirmed when, in 1977, William P. Maher of Marquette University and the Medical College of Wisconsin reported postmortem arteriographic studies of the major branches of the pterygopalatine portion of the maxillary artery in three cleft palate and nine non-cleft near-term human fetuses. The study revealed numerous variations in each facial half in both cleft and non-cleft palates. Maher noted anatomical aspects pertinent to palate surgery:

Lateral branches of the greater palatine arteries provide substantial contributions to all maxillary deciduous and permanent dental structures. Incisions made parallel to the alveolar ridge and lateral to the greater palatine artery are designed for purposes of moving the pedicle flap medially to close the cleft. These incisions completely sever nutritional supply from the palatine network to all the maxillary teeth. However, the developing dental structures are also supplied by dental branches from the superior alveolar artery and gingival-osseous branches from the great facial network. Whether these major contributions are able to provide adequate nutritional sources for normal dental development or whether temporary nutritional loss from the palatal network as the results of sectioning might be responsible for morphological defects in hard dental structures remains to be clarified.

The palatal mucoperiosteum is detached from its bony base in the preparation of a palatal pedicle flap, and as a result the recurrent osseous branches are severed. These branches vary in size, number, location, and

distribution frequency. Bleeding from their cut stumps at the bony surface may be judged . . . insignificant at the time of surgery. However, after the flap is repositioned, should bleeding continue . . . blood may pool beneath the repositioned flap.

Maher also noted:

Terminal branches of the greater palatine artery were found to anastomose variously with: 1) the lateral nasal septal artery, 2) the superior alveolar artery, 3) labial branches of the facial artery via branches of the palatal artery that pass through the maxillary fissure or via the external nares, and 4) with its companion on the contralateral side. One or more combinations of these variations may occur in both facial halves. . . . Furthermore, crossover anastomoses between terminal branches of major vessels can occur.

Maher admits to being a two-kind-of-vessel freak:

I have a canoe that is called *Capillary*, a nice runabout called *Arteriole*, and a larger boat called *Ark of the Aorta*.

Since 1957 he has also been involved in microvascular studies and has developed the technique of injecting blood and lymph vessels with India ink in order to examine the routes and distributions. In fact, he became known as the Wizard of Ink or Inky for short. In 1977 he forwarded this vascular maze to me and wrote on the back of it:

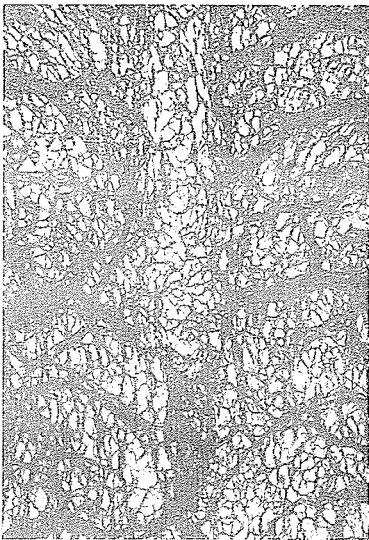
This is a photograph of ink replications of arteries and veins in the vicinity of the palatal raphe that has habitually been described as being relatively avascular. The preparation is that of perinatal man.

#### INVESTIGATION INTO BLOOD SUPPLY TO PALATE BONES

This field has long been the realm of surgery guided largely by the surgeon's supposition. It occurred to me that further study would be of interest, not only from the aspect of safety during surgery, but also for investigating surgery's possible effect on bone growth by reduction in blood supply. Lieutenant Colonel G. Franklin Welsh, U.S.A.F., while in general surgery residency at the Mayo Clinic, visited Miami to investigate a possible residency in plastic surgery. He was challenged to study the blood supply to the bones of the palatal area and to *search for* and *identify* blood



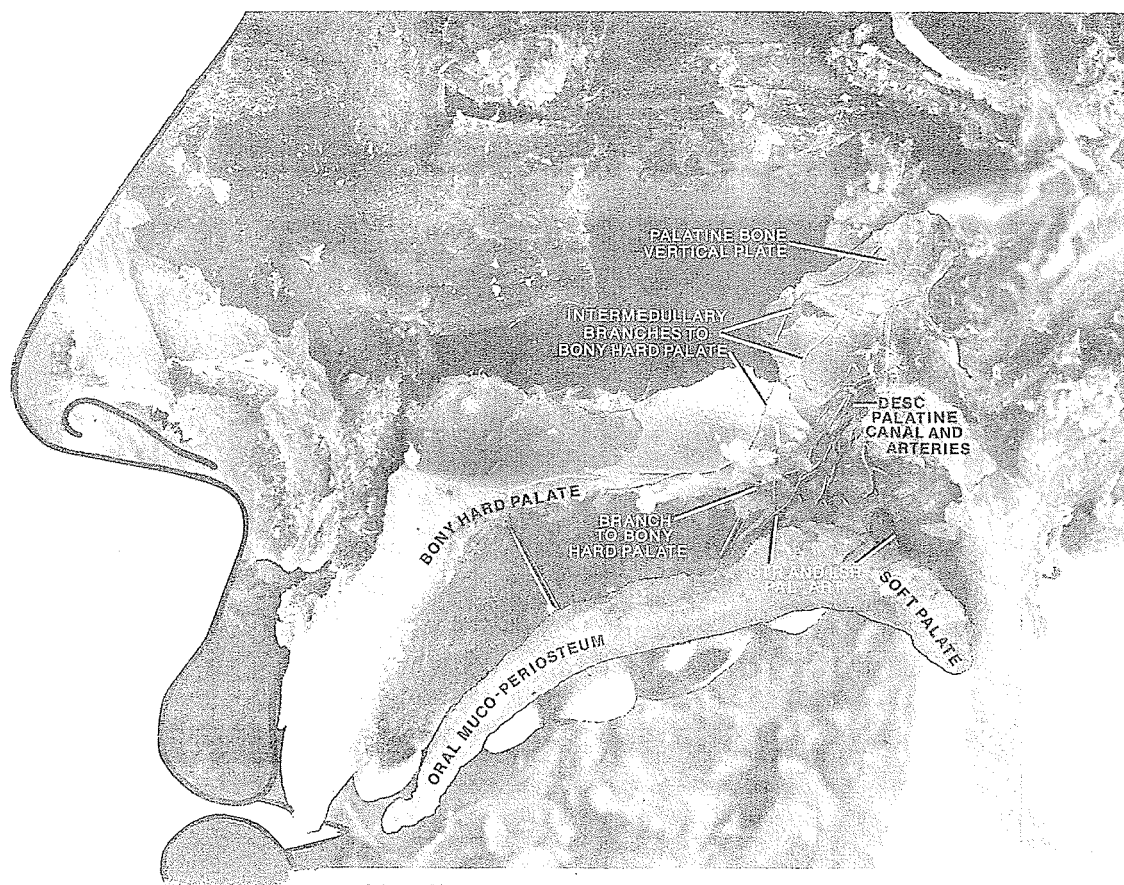
William Maher



Franklin Welsh

vessels that would demonstrate how the oral mucoperiosteum could be reflected with impunity in palate surgery without fear of devascularizing the bony palate. Welsh dissected the blood supply in both halves of a medial-sagittally sectioned head from a cadaver injected with red latex. This was his 1971 report, along with a photograph of the specimen:

Soon after its origin as a terminal branch of the maxillary artery, the descending palatine artery enters a canal in the vertical plate of the palatine bone to give off an intramedullary cascade of arterioles which meander antero-inferiorly into the bony hard palate. Several branches descend through the palatine foramina and divide into an anterior group, the greater palatine arteries, and a posterior group, the lesser palatine arteries, for soft palate. Immediately upon emerging from the greater palatine foramen, these arteries give off multiple small vessels that spread out, closely adherent to the undersurface of the bony palate, yielding several perforators into the bone, rather than lifting away with the mucoperiosteum. Specimen is shown:



Neither the posterior superior alveolar artery nor the anterior superior alveolar extension of the infraorbital artery, all of which penetrate maxillary

bone cortex externally, continues medially beyond the alveolar ridges and tooth sockets into the bony palate.

*Conclusion:* With perforating branches from nasal floor mucosa, multiple intramedullary branches from the descending palatine artery in its canal, and immediate takeoff of bony branches from the emerging greater palatine arteries, the blood supply to the bony palate is well secured even as the mucoperiosteum is lifted, even if the greater palatine artery should be ligated.

Upon receipt of this fine work, I wrote to Welsh to ask if he had also studied the vasculature coming through the incisive foramen. This was his response:

Although the conventional wisdom regarding anastomosis of posterior septal branches of sphenopalatine artery with anterior terminal branches of the greater palatine artery via incisive canal is repeated throughout the texts and atlases, I was unable to confirm this fact. By sprinting back to the gross anatomy laboratory here upon receipt of your letter, I located the same specimen on which I made the earlier observations. Attention to the incisive canals revealed first that the injected latex dwindled rapidly, requiring the final vascular arborizations to be traced via minute unfilled radicles. The course proved to be as follows:

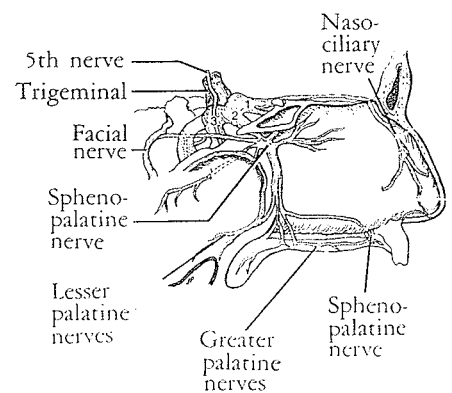
An anterior extension of posterior septal branches of sphenopalatine artery enters the incisive canal, passes inferiorly into oral alveolar ridge cortex, and terminates in the region of the incisor tooth sockets. Although there were no apparent penetrations from incisive canal into oral mucoperiosteum, there were multiple oral mucoperiosteal vessels perforating the oral cortex of hard palate and alveolar ridge. No contributions from the artery of the incisive canal back into bony secondary hard palate were observed.

Although Welsh was unable to obtain clearance from the Air Force for a complete residency, he did accept a Maytag Fellowship and finally, in 1976, returned to Miami to put the finishing touches on his bony palate vascular research. Welsh, a Harvard Medical School graduate and pithy scholar, has a remarkable depth of knowledge and an uncanny way with words. He admits to having *hyperhedonia* when digging through the palate bone for shriveled vessels, defining this state as "*hi*" *par hē dō' ni a, (n)*, abnormal pleasure from doing ho-hum things," taken from *Mrs. Byrne's Dictionary of Unusual, Obscure and Preposterous Words*.

## NERVE SUPPLY

### *Sensory*

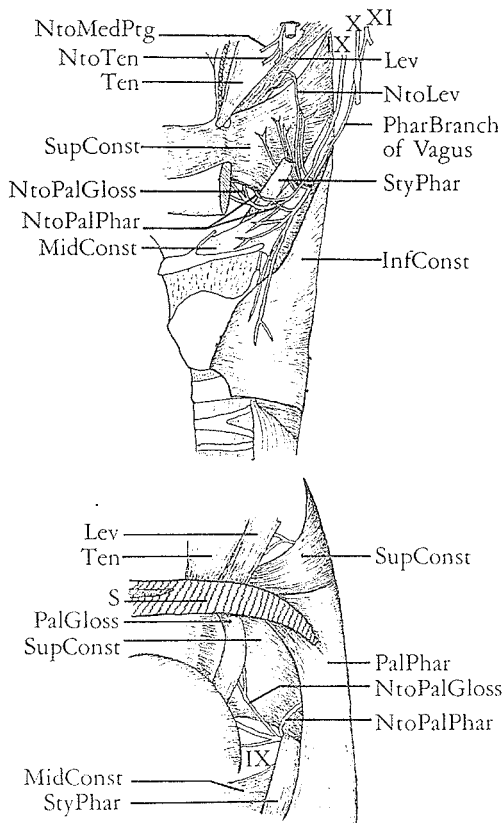
The maxillary nerve, the second division of the trigeminal nerve, passes through the sphenopalatine ganglion, picking up the secretory and sympathetic fibers from the facial nerve. This composite nerve then divides into three and is distributed to the nasal cavities, nasopharynx and palate. One of the branches is the sphenopalatine nerve, which passes through the incisive foramen to the anterior hard palate. Then there is the greater palatine nerve, which comes through the posterior palatine foramen, supplying the remainder of the hard palate. The smaller middle and posterior palatine nerves, branches of the pterygopalatine nerve, emerge through the lesser palatine foramen to supply the soft palate and tonsil with sensory branches.



### *Motor*

It has long been known that the motor nerve supply to the tensor veli palatini muscle is different from the other velopharyngeal muscles. W. A. Turner (1889), L. Rethi (1893), L. Druner (1903) and A. R. Rich (1920) all confirmed that the tensor muscle was innervated by the trigeminal nerve, actually the internal pterygoid nerve, a branch of the mandibular, which in turn is a branch of the trigeminal nerve. It is of interest that Harvey Cushing in 1905 reported movement of the tensor palatini by stimulation of the mandibular division of the trigeminal nerve.

There has been and continues to be much disagreement concerning the motor nerve supply to the other velopharyngeal muscles. Most textbooks seem to be satisfied with the general statement that the vagus and accessory nerves supply motor fibers to the muscles of the pharynx and soft palate, with the exception of the tensor palatini, and that the greater superficial petrosal nerve, arising from the facial nerve, also carries motor fibers to the sphenopalatine ganglion and thence to the palatine nerves and soft palate.

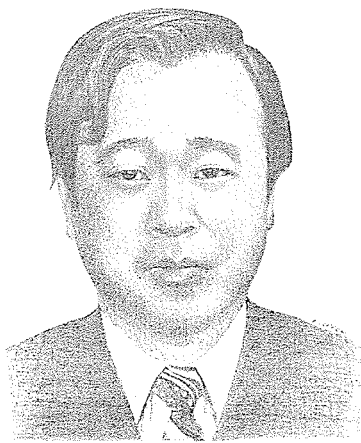


E. Cords (1910) and I. W. Broomhead (1951) described these muscles as innervated by branches of the pharyngeal plexus derived from the glossopharyngeal and vagus nerves.

Ivor Broomhead was house surgeon to David Matthews in 1948 and was inspired by him in 1949 to carry out research on the nerve supply of the soft palate in the Anatomy Department at Cambridge University. He later joined Matthews at the Hospital for Sick Children, Great Ormond Street, London, and worked with him until Matthews retired in 1976. In the *British Journal of Plastic Surgery*, 1951, Broomhead reported important anatomical findings for the palate surgeon. He presented a sketch showing the distribution of the glossopharyngeal nerve (IX) and the pharyngeal branch of the vagus (X) to the constrictor muscles of the pharynx, levator palatini, palatoglossus, and the nerve to the medial pterygoid muscle. He also showed the course of the nerves to the palatoglossus and palatopharyngeus on the medial side of the superior constrictor.

A. R. Rich (1920), however, reported levator veli palatini muscle contractions elicited by stimulation of the vagus and accessory nerves, but not by facial and glossopharyngeal stimulation.

The continuing disagreement in the face of many studies of the motor nerve supply to the velopharyngeal muscles intrigued Juntaro Nishio of Japan. He continued his family's tradition by taking a dental degree, then furthered his studies with a dissertation entitled "The Relationship Between Velopharyngeal Movement and Its Motor Nerves," for which he earned his Ph.D. In 1976, further excellent work was published in the *Cleft Palate Journal* by Nishio, with T. Matsuya, J. Machida, and T. Miyazaki, of the Oral and Maxillofacial Departments of the Matsumoto Dental College and the Osaka University Dental School of Japan. Their experiments, designed to clarify motor nerve supply to the velopharyngeal muscles, were carried out on 20 rhesus monkeys. (J. F. Bosma and S. G. Fletcher in 1961 stated that basic velopharyngeal anatomy was similar in cats, dogs, monkeys and humans, while C. G. Hartman and W. L. Straus, also in 1961, reported that the course of the cranial nerves in the rhesus



Juntaro Nishio



monkey is similar to that in the human being.) Evoked EMG responses of the levator veli palatini, uvula and superior constrictor pharyngeus muscles, which contributed to velopharyngeal closure, were analyzed by the Japanese workers by their stimulating the cranial nerves within the skull. Here is the summary of their results:

1. Muscle action potential (M-waves) from the selected muscles could be recognized on stimulating the facial, glossopharyngeal, and vagus nerves at the petrosal area of the temporal bone but were not noted upon accessory nerve stimulation.

2. At maximal stimulation, the vagus gave a greater increase in muscle amplitude than the other nerves studied. This was followed by the glossopharyngeal with the facial nerve producing the least in amplitude.

3. Also at maximal stimulation, latencies in the response of the levator veli palatini and uvula muscles were reduced to the greatest degree by stimulation of the vagus, to a lesser extent for the glossopharyngeal, and least for the facial nerve.

4. On stimulating the facial nerve below the stylomastoid foramen, M-waves could not be recognized.

From the present study, it was concluded that the levator veli palatini, uvula, and superior constrictor pharyngeus muscles are double innervated by the facial nerve and branches of the pharyngeal plexus derived from the glossopharyngeal and vagus nerves and that the facial nerve plays an important role as one of the motor nerves in the movements responsible for velopharyngeal closure.

They also proposed an interesting theory:

We sometimes encounter cleft palate patients who demonstrate nasal grimace during phonation. This has been considered to be a compensation to velopharyngeal incompetence (Morley). Recently the authors have applied visual training to help cleft palate patients acquire adequate velopharyngeal function. As a result of the training, it was noted that coordinating movements of lip and face, such as nasal grimace or lip-protrusion, during phonation, were useful to activate velopharyngeal movements (Nishio, Yamaoka, Matsuya and Miyazaki). Therefore, the nasal grimace may occur not only to compensate for velopharyngeal incompetence by increasing nasal resistance, but also to fire the facial nerve to complement velopharyngeal movements.



## A WARNING

The dissection of fetal and adult heads by Broomhead at the anatomy school at Cambridge has particular significance to palate surgeons, as he concluded his report with this warning:

During operative repair of a third-degree cleft palate damage may be inflicted on the nerve to the tensor palati, resulting in paralysis of this muscle.

Section of the lesser palatine nerves also takes place, and will result in some anaesthesia of the soft palate and paralysis of the musculus uvulae. Whether the mucous glands suffer any damage following the section of these nerves is not known.

The nerves to the palato-glossus, palato-pharyngeus, and levator palati muscles do not, in any part of their course, enter the operative field.