Normal and Cleft Palate Anatomy

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Introduction

There is a great deal of discussion in the literature about what muscles are involved in normal velopharyngeal activity for speech and swallowing. A review of current textbooks reveals a diversity of opinions. The muscles which are commonly described as part of the velopharyngeal mechanism include levator veli palatini, glossopalatinus, palatopharyngeus, constrictor pharyngis superior, salpingopharyngeus, and tensor veli palatini. There is consensus in the literature that levator is primarily, if not totally, responsible for the elevation and retraction of the velum for velopharyngeal closure (Figure 1). There is also consensus that glossopalatinus contributes to or is responsible for lowering the velum. While the palatopharyngeus appears to be anatomically in a position to lower the velum, it is often considered to play a role with levator in velopharyngeal closure. The opinion has been advanced that levator lacks sufficient posterior vector for velar movement observed, thus the additional force of palatopharyngeus is necessary. It is the opinion of many authors that constrictor pharyngis superior is part of the mechanism for moving the velum posteriorly and also is responsible for medial motion of the lateral walls of the pharynx.

In Figure 2 we see an illustration of a superior view of the pharynx. The constrictor muscle fibers course through the lateral walls of the pharynx lateral to the Eustachian tube cartilage (torus tubarius) with its most superior fibers inserting into the velum. The majority of the constrictor fibers insert into the medial pterygoid plate anteriorly. While it has been accepted that the lateral walls of the pharynx do move medially and posteriorly to complete velopharyngeal closure, the muscles responsible for that motion are not agreed upon. Some authors describe either the salpingopharyngeus muscle or the constrictor or both as responsible for lateral pharyngeal wall movement. The salpingopharyngeus muscle arises from the palatopharyngeus and courses vertically through the lateral wall of the pharynx in the salpingopharyngeal fold and inserts into the torus.
FIGURE 1. Diagram of muscle forces apparently acting on the normal velum.

FIGURE 2. Diagram of relationship of constrictor pharyngis superior to the pharyngeal wall.

tubarius (Figure 3). The only way in which these vertical fibers of salpingopharyngeus could accomplish medial movement of the lateral wall would apparently be by increasing their bulk as a result of shortening during contraction. Finally, a number of authors describe anterior tensing of the velum during velopharyngeal closure as a result of contraction of the tensor veli palatini muscle.

The Normal Mechanism. In an analysis of normal velopharyngeal physiology, three components need to be considered: (1) elevation and retraction of the velum, (2) postero-medial movement of the lateral pharyngeal wall, and (3) lowering of the velum. With regard to the first component, levator, palatopharyngeus and constrictor are the only muscles which could contribute. There is no direct evidence that levator lacks the proper anatomical position to accomplish this motion singularly. The electromyographic data of Cooper (4) and Fritzell (6) indicate that contraction of levator is most consistently correlated with velopharyngeal closure; that less consistently correlated contraction of constrictor occurs; but that palatopharyngeus does not contribute to closure. Anatomically, while the superior fibers of palatopharyngeus are horizontal and thus could conceivably pull the velum posteriorly, it would be inconsistent with human muscular function for one muscle to contribute to two physiologically opposing motions—velopharyngeal closure and opening. It would seem most reasonable in light of available data to cast palatopharyngeus with muscles of velopharyngeal opening. Also on anatomical grounds it is difficult to ascribe normal closing motion to constrictor pharyngeus superior. The most fixed attachment of this muscle is anterior at the pterygoid plates. Its pharyngeal insertion is mobile. Thus contraction of this muscle, from a mechanical point of view would be expected to result in antero-medial rather than postero-medial motion of the lateral pharyngeal walls. Antero-medial motion is seen as part of the so-called Passavant’s ridge, a
compensatory, abnormal movement pattern often seen in persons with velopharyngeal inadequacy or cleft palate. This ridge may well be the result of compensatory contraction of the superior fibers of constrictor which insert into the velum. Thus there is reason to question whether constrictor is necessarily involved in any way in normal velopharyngeal closure for speech. It may, however, be involved in the more sphincter-like closure seen in swallowing.

The role of salpingopharyngeus is also highly questionable. Previous investigators of this muscle (1, 3, 8, 14) have found it to be small, inconsistent, and sometimes absent. Two investigations of this muscle in our laboratories have revealed that in approximately half of the over twenty human heads dissected, this muscle was absent at least on one side. Where muscle was found, the amount of muscle was extremely variable. It was our conclusion that the salpingopharyngeus muscle consists of occasional fibers of palatopharyngeus which bypass the velum and ascend to the torus tubarius and have no functional significance.
If we are to eliminate constrictor pharyngis superior and salpingopharyngeus from consideration in lateral pharyngeal wall motion, what is left? If we examine the lateral pharynx, we find that it is quite likely that the primary site of motion of the lateral wall occurs at the level of the torus, and that as the torus moves, or is moved, postero-medially, the salpingopharyngeal fold which descends from it is carried along. This speculation was strengthened by our examination of frontal cineradiographic studies of this area which we did in cooperation with Ewanowski and Crummy at the University of Wisconsin and with Wilma Dickson at Meric Hospital, Pittsburgh. Skolnick (13) has also completed an excellent series of frontal and basal views and disagrees with this hypothesis. He feels that the site of primary motion may be below the level of the torus and a result of constrictor. Bosma (7), Moss (9) and Ruding (12) speculated that the relative position of the levator veli palatini muscle and the torus tubarius suggested that levator may move the lateral wall. In Figure 4 we see a

FIGURE 4. Medial view of relationship among torus tubarius (1), levator veli palatini (2) and salpingopharyngeus (3) in the adult human pharynx, (4) velum, (5) pharynx.
medial view of the relationship among the torus, levator, and salpingopharyngeus. Were the levator to move medially, the torus and salpingopharyngeus would be carried with it. This view taken with the posterior view of levator shown in Figure 5 indicates that the two sides of levator course inferiorly, medially and anteriorly from the cranial base to their convergence in the velum. Thus, from a mechanical standpoint any tissue on their medial surface, such as the torus, would be displaced superiorly, medially, and posteriorly as the sling of levator contracts. Thus we believe that the most tenable hypothesis is that levator is the sole muscle of velopharyngeal closure, causing both the postero-superior movement of the velum and the postero-medial movement of the lateral pharyngeal walls.
There is ample evidence that the tensor veli palatini muscle has nothing to do with movement of or tension in the velum. Its sole and unique function is to open the Eustachian tube. This was demonstrated by Rich (10). Recent research in our laboratory (11) supports a hypothesis discussed previously in the anatomical literature that tensor may be considered a two-bellied muscle with one end attaching to a long line from the cranial base down the pterygoid plate to the hamulus. An intermediate septum divides this portion from that which extends to the lateral membranous wall of the Eustachian tube at its isthmus. Thus the action of the muscle would be from the lateral pterygoid plate to the Eustachian tube and would dilate the tube. The inferior tendon of the tensor extends around the hamulus into the anterior third of the velum and attaches to the entire length of the posterior rim of the hard palate. Thus it is not surprising to find that recent studies have demonstrated that hamular fracture does not effect Eustachian tube function.

It is also important to note that the anterior velum is amuscular. Figure 6 is a coronal section of a normal fetal head taken just posterior to the hard palate. The velum is seen to be glandular at this point. The tensor tendon which forms the anterior velar aponeurosis is clearly visible. The first muscle fibers encountered as we move back through the velum are transverse and are probably fibers of levator and/or palatopharyngeus. More posteriorly, the first longitudinal fibers—those of the azygos uvulus muscle—are encountered.
THE CLEFT MECHANISM. If we turn back many years to the work of Lushka (15) we see the first description of cleft musculature. He described two essential differences in the structure of the cleft and non-cleft velum. First, the anterior aponeurosis was described as lacking in the cleft. Second, the anterior third of the cleft velum, rather than being amuscular, contained longitudinal muscle fibers which he ascribed to palatopharyngeus and levator. These fibers were described as inserting into the posterior rim of the hard palate and into the medial edges of the bony cleft of the hard palate. Later, in 1931, Veau reported Lushka’s observations and confirmed them by dissection and histologic analysis. Recently Kriens (7) reported a dissection of one cleft palate human fetus, Braithwaite and Maurice (2) described another, and Fara and Dvorak (5) described their dissection of 18 cleft human fetuses. All agreed with Lushka and Veau. They further described the tensor tendon as terminating at the hamulus rather than crossing through the velum. Thus one way of explaining the shortness of the cleft palate is the absence of the aponeurotic layer in the anterior third. In addition, these investigators agreed that while the muscular portion of tensor appeared to be normal, the levator was found to be hypoplastic.

Preliminary investigations of cleft fetuses in our laboratories have agreed with these findings. Figure 7 shows a comparison of a cleft and non-cleft fetus sectioned through the posterior hard palate. While the

![Figure 7: Histologic section in coronal plane of cleft palate (left) and non-cleft palate (right) human fetal heads taken through the posterior hard palate. (1) septum nasi, (2) palate, (3) nasal cavity, (4) oral cavity. Arrows show longitudinal muscle fibers.](image-url)
FIGURE 8. Histologic section in coronal plane of cleft palate human fetal head taken through the velum at the plane of Eustachian tube opening into the pharynx (1). (2) Eustachian tube, (3) tensor veli palatini, (4) nasopharynx, (5) velum, (6) levator veli palatini, (7) oral cavity.

FIGURE 9. Histologic section in coronal plane of non-cleft palate human fetal head taken through the velum at the plane of Eustachian tube opening into the pharynx (1). (2) Eustachian tube, (3) tensor veli palatini, (4) nasopharynx, (5) velum, (6) levator veli palatini, (7) oral cavity.
FIGURE 10. Histologic section in coronal plane of cleft palate human fetal head taken through the velum near the uvula (1). (2) Eustachian tube, (3) tensor veli palatini, (4) nasopharynx, (5) velum, (6) levator veli palatini, (7) oral cavity.

FIGURE 11. Histologic section in coronal plane of non-cleft palate human fetal head taken through the velum near the uvula (1). (2) Eustachian tube, (3) tensor veli palatini, (4) nasopharynx, (5) velum, (6) levator veli palatini, (7) oral cavity.
normal palate is amuscular at this plane, longitudinal muscle fibers are noted in the margins of the cleft. Figures 8 and 9 are sections through the velum at the plane of Eustachian tube opening. It is apparent that the non-cleft velum is far more muscular than the cleft. However no apparent difference is seen in the tensor muscles. Further posteriorly, near the uvula (Figures 10 and 11) we can examine the levator muscle in its position inferior to the Eustachian tube. Again the reduced volume of levator in
the cleft is seen. In some of our specimens (Figures 12, 13) there is a suggestion of a tortuous entrance into the Eustachian tube, narrowing of the tube aperture and enlargement of the medial cartilage as compared to the non-cleft. This is now under investigation and no firm statements can be made at this time. In addition, studies of cleft and non-cleft palate nerve and blood supply are now being undertaken. No information of this kind has been found in the literature on cleft palate.

Conclusions

Further definitive studies of the physiology of normal velopharyngeal function are ongoing and necessary. It is apparent that our information in this area is sufficient to call for coordinated electromyographic-anatomical-radiographic exploration to further test existing hypotheses. This is not true in the area of cleft palate where no definitive studies of specific muscle function or patterns of movement of the velum and pharynx have been completed. Further, delineation of nerve and blood supply is necessary in both cleft and non-cleft.

References