Anatomy

OUTLINE

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The etiology and pathogenesis of otitis media are multifactorial, and the structure and function of the eustachian tube are key components. In this chapter, we describe the anatomy of the eustachian tube in relation to the organs to which it connects. We also explain why the infant eustachian tube is developmentally immature and contributes to the prevalence of otitis media in this highly susceptible age group.

Recently, one of us (C.D.B.) published *Eustachian Tube: Structure, Function, Role in Otitis Media*, which is an up-to-date, extensive review of the state of our knowledge of the anatomy of the eustachian tube that also includes other aspects of the tube related to otitis media.¹

This chapter summarizes the structure of the eustachian tube, but for a more comprehensive understanding of the anatomy, the reader is referred to this current text.

THE EUSTACHIAN TUBE IS AN ORGAN WITHIN A SYSTEM

In reality, the eustachian tube is not just a tube but an organ consisting of a lumen with its mucosa, cartilage, surrounding soft tissue, paratubal muscles (ie, tensor veli palatini, tensor tympani, levator veli palatini, and salpingopharyngeus), and bony support (sphenoid sulcus and medial pterygoid plate). The term *middle-ear cleft* is often used to describe the eustachian tube, middle ear, and mastoid gas cells (Table 1). The larynx, another organ in the airway, has many similarities to the eustachian tube in that both have comparable (1) anatomy, including a lumen that is covered by mucosa, cartilage support, and a muscular opening mechanism; (2) physiologic...
functions (eg, ventilation, protection, and clearance); and (3) pathophysiologic processes (ie, the lumen can be too open or too closed or the opening or closing mechanism may fail). The eustachian tube is part of a system of contiguous organs. The nasal cavity, palate, and pharynx are at its proximal end, and the middle ear and mastoid gas cells are at its distal end (Figure 1).

### Table 1. COMPOSITION OF THE EUSTACHIAN TUBE AS AN ORGAN

<table>
<thead>
<tr>
<th>Lumen mucosa</th>
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<tbody>
<tr>
<td>Osseous portion</td>
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<tr>
<td>Lateral membranous wall</td>
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<tr>
<td>Extraluminal soft tissue</td>
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<tr>
<td>Cartilage</td>
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<tr>
<td>Ostmann fat pad</td>
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<tr>
<td>Muscles</td>
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<tr>
<td>Tensor veli palatini (and tensor tympani)</td>
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<td>Levator veli palatini</td>
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<td>Salpingopharyngeus</td>
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<td>Blood supply</td>
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<td>Lymphatics</td>
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<tr>
<td>Osseous support</td>
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<tr>
<td>Sphenoid sulcus</td>
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<tr>
<td>Medial pterygoid plate</td>
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The eustachian tube can be divided into three portions: cartilaginous, junctional, and osseous. The cartilaginous portion is proximal and opens into the nasopharynx. The osseous portion is distal and opens into the anterior middle ear. The junctional portion is the part of the tube where the cartilaginous and osseous portions connect; it was previously thought to be the narrowest part of the tubal lumen, the isthmus. A three-dimensional study of nine human temporal bone specimens by Sudo and colleagues demonstrated the isthmus portion of the lumen to be near the distal end of the cartilaginous portion and not at the junction of the cartilaginous and osseous portions (see later). Respiratory mucosa lines the entire tubal lumen. Figure 2 shows the anatomy of the tube and its muscles. A description of the growth and development of the tube is important for understanding why infants and young children have more middle-ear infections than older children and adults do.

**Figure 1.** The eustachian tube is part of a system; the pharynx, palate, and nasal cavities are at its proximal end, and the middle ear and mastoid gas cells are at its distal end.
Figure 2. A, Complete dissection of the eustachian tube and middle ear. The relationship of the eustachian tube, paratubal muscles, and cranial base and the positioning of the juncture between the osseous portion of the eustachian tube and the middle ear (ie, the aural orifice of the tube) are evident. B, Appearance of the nasopharyngeal orifice of the eustachian tube. Note the large torus tubarius and its inferior continuation at the salpingopharyngeus muscle.
Developmental Anatomy

The structure of the adult eustachian tube is the culmination of 18 years of development and growth. Thus, we can best appreciate the eustachian tube’s structure and function in the context of these processes. Further, identification of abnormalities and their consequences depends on a knowledge of normal anatomy.

Prenatal Growth

The eustachian tube lumen develops from the persistence of the first pharyngeal pouch in the embryo. The entodermal lining of the first pharyngeal pouch extends laterally, making contact with the ectoderm of the bottom of the first gill furrow on either side of the gill plate. The distal pouch becomes elongated and expanded to form the tubotympanic recess, the primordium of the middle-ear cavity. The proximal portion then becomes narrowed to form the eustachian tube. The lumen at this stage has a smooth margin with unciliated low columnar epithelium. The structures associated with this lumen develop from the surrounding mesenchyme in a predictable sequence.

Swarts and colleagues studied tubal development in 20 human fetuses between 7 and 38 weeks after conception. Their results confirmed and extended those of Wolff and Tos. Before 10 weeks after conception, only the epithelial lining of the lumen has differentiated. Between 10 and 12 weeks after conception, the levator veli palatini and tensor veli palatini muscles develop. The first evidence of the third muscle, the tensor tympani muscle, is apparent approximately 2 weeks later. At about the same time (14 weeks after conception), the initial differentiation of the cartilage begins. Also at this time, the lumen begins to show folding of the epithelium into the rugae, characteristic of the eustachian tube after birth.

Accompanying these changes, glandular tissue appears in the pharyngeal wall, medial to the cartilage and between it and the more lateral lumen. By 20 weeks after conception, the initial center of chondrification has increased in size and a perichondrium is clearly differentiated in the anteromedial portions of the tube. By parturition, these processes yield a eustachian tube structure similar to an adult’s. The cartilage is clearly delimited by a perichondrium throughout its length and shows the classic J-shaped form. The muscles are well circumscribed and, relative to the cartilage, are positioned similarly to those of the adult. Glandular tissue has proliferated and now occupies the regions between the cartilage and the nasopharynx, between the cartilage and the lumen, and between the lumen and the tensor veli palatini muscle.

As ontogeny proceeds, morphometric changes occur among the eustachian tube structures and with respect to the rest of the head. The most pronounced change is the increase in tubal length from 1 mm at 10 weeks to 13 mm at birth. Most of this increase occurs in the cartilaginous portion of the tube. Because the fetal cranial base is relatively flat, the tube deviates from the horizontal plane only about 10°, a condition that persists into early childhood. During postnatal development, the cranial base angle and the vertical dimensions of the skull increase. The hard palate drops away from the skull base. As this occurs, the angle between the cartilaginous tube and the skull base increases.

Postnatal Growth

After birth, the eustachian tube in the infant and young child is immature in structure and function compared with that in the older child and adult. There are at least 11 major anatomic differences, which are summarized in Table 2. These anatomic differences are as follows:

Length of the Tube. Sadler-Kimes and colleagues used a three-dimensional computer graphic technique to analyze the size, shape, and positional association of the eustachian tube cartilage, lumen, and paratubal muscles. They compared temporal bone histopathologic specimens from children younger than 7 years with those of children 7 years and older. In infants,
the eustachian tube is about half as long as in the adult; it averages about 18 mm. The tube lengthens rapidly during early childhood and by 7 years of age is approximately as long as an adult’s. Ishijima and colleagues found the length of the lumen of the infant tube to be 21 mm compared with the average length of 37 mm in an adult. The cartilaginous tube represents somewhat less than two-thirds of this distance. In contrast, the osseous portion is relatively longer and wider in diameter than it is in the adult. A later study confirmed and expanded on these developmental differences in tubal length. The shorter tube in the infant and young child (the tube is too short compared with adult tubal length) is most likely related to an impaired protective function; for example, nasopharyngeal secretions can reflux or insufflate into the middle ear (see Chapter 3).

The cartilage, lumen, and levator veli palatini muscle increase in cross-sectional area and volume after age 7 years. In older children, the tube’s cartilage and lumen are also more elongated, and the distance from the levator veli palatini muscle to the other structures of the tube is significantly larger. The distances from the tensor veli palatini muscle to the lumen and to the levator palatini muscle are also larger after age 6 years. Holborow demonstrated that in infants, the medial cartilaginous lamina is relatively shorter. Cartilage mass also increases from birth to puberty.

## Angle of the Tube

In the infant, the direction of the tube varies from horizontal to an angle of about 10° to the horizontal, and the tube is not angulated at the junctional portion but merely narrows. In the adult, the tube is approximately 45° related to the horizontal plane. This difference in angles between infants and adults has been thought to impair clearance of the eustachian tube–middle ear in the infant, but it is most likely the muscle vector of the tensor veli palatini that is adversely affected in this age group (Figure 3).

### Angle Between the Tensor Muscle and the Tube

The angle that the tensor veli palatini muscle makes with the lumen is almost identical in children and adults. However, the angle between the tensor veli palatini muscle and the cartilage is different. In the child, the angle between the muscle and the cartilage is larger in the nasopharyngeal portion of the tube and decreases posteriorly toward the middle-ear end of the tube. In the adult, this angular relationship is stable throughout the length of the tube. This angular difference between children and adults may be related to the known inefficient tubal function (the tube will not open)

<table>
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<tr>
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<td>Angle/length of TVP to cartilage</td>
<td>Variable vs stable angle, shorter attachment</td>
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<tr>
<td>Lumen</td>
<td>Smaller area/volume</td>
<td>Kitajiri et al22 Suzuki et al23 Ishijima et al11</td>
</tr>
<tr>
<td>Cartilage volume</td>
<td>Less</td>
<td>Takasaki et al20</td>
</tr>
<tr>
<td>Cartilage cell density</td>
<td>Greater</td>
<td>Yamaguchi et al18</td>
</tr>
<tr>
<td>Elastin at hinge portion of cartilage</td>
<td>Less</td>
<td>Matsune et al17</td>
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<tr>
<td>Ostmann’s fat pad</td>
<td>Relatively wider</td>
<td>Aoki et al24 Orita et al25 Orita et al26</td>
</tr>
<tr>
<td>Mucosal folds</td>
<td>Greater</td>
<td>Sudo and Sando28</td>
</tr>
<tr>
<td>Lumen glands</td>
<td>Variable type</td>
<td>Orita et al25</td>
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<td>Connective tissue lateral to tube</td>
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<td>Orita et al26</td>
</tr>
<tr>
<td>Middle-ear volume</td>
<td>Smaller</td>
<td>Ikui et al29</td>
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Adapted from Bluestone C.1

TVP = tensor veli palatini muscle.
in children compared with that in adults and the increased incidence of otitis media in children.\textsuperscript{16,17}

\textbf{Cartilage Cell Density.} Yamaguchi and colleagues studied the cartilage cell density in the eustachian tube of human temporal bone histopathologic specimens ranging from 26 weeks’ gestation to 85 years (24 were younger than 3 years).\textsuperscript{18} The cartilage cell density was statistically greater in specimens from children younger than 7 years compared with specimens from older children and adults. This variation may be related to (1) the observations that infants and young children have increased compliance of the eustachian tube (the tube is \textit{too floppy}) contributing to their inability to effectively open or dilate the tubal lumen when the tensor veli palatini muscle contracts (ie, functional obstruction) and (2) increased distensibility of the tube, which can promote insufflation of nasopharyngeal secretions into the middle ear during crying, closed-nose swallowing (the Toynebe phenomenon), and blowing the nose.\textsuperscript{19,20}

\textbf{Elastin in the Cartilage.} Matsune and colleagues assessed the amount of elastin in the hinge portion of the cartilage of the eustachian tube (ie, the intermediate portion between the lateral and medial laminae) in temporal bone histopathologic specimens from infants and adults.\textsuperscript{21} They found that elastin was statistically less dense in the infant specimens and postulated that this may be related to the hypothesis of functional obstruction in children (ie, failure of the tubal opening mechanism or the tube will not open). The relatively less dense elastin in the hinge portion in the infant could also result in inadequate passive tubal closure and a lumen more distensible to nasopharyngeal positive pressures. Both possibilities would impair the eustachian tube’s protective function.

\textbf{Tubal Lumen Area.} Kitajiri and colleagues studied the postnatal development of the eustachian tube lumen and found that the area of the lumen increased almost fivefold from the newborn to age 20 years; the midcartilaginous portion increased most dramatically.\textsuperscript{22} The

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure3.png}
\caption{The difference in the angle of the eustachian tube in the infant and adult. This most likely adversely affects the function of the infant tensor veli palatini muscle.}
\end{figure}
cross-sectional length of the lumen significantly increased during development, especially in the pharyngeal area of the tube. There appeared to be little growth in the width of the lumen from the newborn to the adult. A larger luminal area of the eustachian tube in the adult would promote more effective pressure regulation of the tube compared with that in the infant. Suzuki and colleagues confirmed these early findings, as did a more recent study by Ishijima and colleagues.

**Ostmann's Fat Pad.** Aoki and colleagues measured the amount of Ostmann's fat pad from temporal bone specimens ranging from neonates to adults. They found an increase in volume with advancing age until adulthood, primarily in height, but little growth in width. Because the fat pad is positioned in the inferolateral aspect of the eustachian tube, it may prevent excessive dilation of the tubal lumen. On the other hand, the relatively greater mass of the fat pad in the infant could contribute to less effective opening of the lumen of the tube. Later studies by Orita and colleagues extended these findings.

**Mucosal Folds.** Sando and colleagues found the inferior portion of the lumen of the eustachian tube to contain numerous mucosal folds, which increase the surface area. In contrast, the superior portion of the tubal lumen has relatively no folds, and these folds progressively decrease until 20 years of age. The significance of this developmental change is currently uncertain but may be related to the growth of the tubal luminal area. Other differences were as follows: (1) the middle ear was smaller in the infant; (2) the connective tissue lateral to the eustachian tube was different in the child as opposed to the adult; and (3) the glands in the mucosa of the lumen were variable in the infant.

**Significance of Developmental Differences**

The observed differences in the anatomy of the eustachian tube as an organ in the infant, young child, and adult provide convincing evidence to explain some of the major functional differences also identified in these age groups. This helps our understanding of the increased incidence of middle-ear disease in the pediatric population. Pressure regulation function is less efficient in the young, most probably because of the tubal lumen's ineffective active opening (ie, the tube will not open) by contraction of the tensor veli palatini muscle. This is probably due to the difference in the muscle vector, the highly compliant tubal cartilage (ie, the tube is too floppy), or both. Inefficient ventilatory function of the tube (the tube is too closed) results in middle-ear underpressures—especially during periods of upper respiratory infections—which, if prolonged, can progress into middle-ear effusion.

Because infants and young children have eustachian tubes that are too short and too floppy compared with those of older children and adults, nasopharyngeal secretions can reflux or be insufflated more readily into the middle ear and result in middle-ear infection (see Chapter 3).

**Adult Anatomy**

The length of the adult eustachian tube has been reported to be as short as 30 mm and as long as 40 mm, but the usual range of length reported in the literature is 31 to 38 mm. In a more recent study, Sudo and colleagues found the average length of the cartilaginous, junctional, and osseous portions from temporal bone specimens to be 24, 3, and 6 mm, respectively (ie, total length of 33 mm) and forming a 42° angle with a parasagittal plane through the medial pterygoid plate.

In the adult, the eustachian tube begins in the nasopharynx and passes posteriorly and laterally through the petrous temporal bone. The tube does not take a straight course from the nasopharynx to the middle ear; rather, it follows a slowly curving inverted S course. Spilberg found that in adults, the tube makes two curves from the tympanic cavity, arching downward and forward across the space between the anterior canal wall and the bony external auditory meatus.
in the condyle of the mandible. Before the pharyngeal orifice, it makes another slight curve downward and forward. Additional observations support Spilberg’s observations, although variability is great.

The nasopharyngeal end of the eustachian tube lies about 20 mm above the plane of the hard palate. The cartilage protrudes into the nasopharynx and is known as the torus tubarius (see Figure 2B). A thick layer of epithelium continuous with the soft tissue lining of the nasopharynx covers it.

**Lumen of Tube**
The osseous and cartilaginous portions of the eustachian tube lumen resemble two truncated cones attached at a junctional area. Their broadest ends represent the nasopharyngeal and tympanic orifices. The nasal orifice is 8.5 mm in height. This dimension decreases steadily to a minimum of 3.5 mm after the eustachian tube enters the petrous portion of the temporal bone. A 20° angle exists between the roof of the lumen and its floor. The sum of this angle with that formed by the cranial base and roof of the lumen accounts for the approximately 45° ascent the eustachian tube makes in its course from the nasopharynx to the middle ear (see Figure 3).

The narrowest segment of the lumen of the tube is the isthmus. The isthmus is not at the junctional portion (ie, where the cartilaginous and osseous portions meet), as formerly thought; it is within the cartilaginous part. Three-dimensional measurements of the tube in temporal bone specimens by Sudo and colleagues revealed the isthmus to be 21 mm from the pharyngeal orifice and 3 mm from the pharyngeal margin of the junctional portion, that is, the most anterior margin of bone surrounding the tubal lumen. The reduced caliber of the lumen at the isthmus is a critical component of the physiologic protective function of the eustachian tube, that is, the flask effect in preventing nasopharyngeal secretions from entering the middle ear (see Chapter 3). However, any increase in the lumen at the isthmus impairs the protection of the middle ear because of reflux, aspiration, or insufflation of nasopharyngeal secretions into the middle ear. Sadé and Luntz found no difference in the calibers of the isthmus in comparing children with otitis media and those without. This finding lends support to the hypothesis that functional obstruction of the eustachian tube (ie, failure of the opening mechanism), as opposed to anatomic (mechanical) obstruction of the tube, is a cause of otitis media.

**Mucous Membrane of Tubal Lumen**
The tubal lumen is lined with pseudostratified, columnar epithelium of the ciliated type, which sweeps material from the middle ear to the nasopharynx (Figure 4). The mucosa is continuous with the lining of the tympanic cavity at its distal end and with the nasopharynx at its proximal end. Goblet cells are associated with these ciliated epithelial cells and compose about 20% of the cell population. Tos and Bak-Pedersen studied temporal bones from premature and newborn infants, children, and adults that...
were free of signs of otitis media and made counts of 30,000 to 60,000 goblet cells in different portions of the eustachian tube (eg, pharyngeal to tympanic ends and the lateral wall, medial wall, floor, and roof of the mucosa of the lumen). They found low densities in all parts of the tube in premature infants, gradually increasing densities in the pharyngeal portions through childhood, and high densities in the adult. Similar densities were reported between the lateral and medial walls and in the floor of the tube, but lower densities were found in the roof.

This is consistent with the findings reported by Sando and colleagues, who described more mucosal folds in the inferior half than in the superior half of all portions of the tube (ie, pharyngeal, cartilaginous, isthmus, and osseous), a finding that statistically increased the mucosal margin in the floor compared with the roof. Copious ciliated cells, glands, and goblet cells were within the mucosa of the floor. From these findings, they postulated that the superior portion of the lumen in the cartilaginous portion of the tube is probably involved in ventilation of the tube (ie, pressure regulation) and the lower portion is related to the clearance function of the tube. These mucosal folds progressively decrease in the first 20 years of life.

Matsune and colleagues identified mucosa-associated lymphoid tissue within the mucous membrane of the cartilaginous portion of the eustachian tube in human temporal bone specimens from adults with no evidence of middle-ear disease. These lymphoid follicles develop by extravasation of lymphocytes from the postcapillary high endothelial venule into mucosal inflammatory sites. They are more abundant in the osseous portion of the eustachian tube and middle ear when middle-ear infection has been present.

**Cartilaginous Portion of the Tube**

The cartilaginous portion of the tube is angled in most cases 30 to 40° to the transverse plane and 45° to the sagittal plane. The tube is closely attached to the base of the skull and is fitted to a sulcus tubae (ie, sphenoid sulcus) between the greater wing of the sphenoid bone and the petrous portion of the temporal bone (Figure 5; see also Figure 2). The cartilaginous part of the tube is firmly attached at its posterior end to the osseous orifice by fibrous bands and usually extends some distance (3 mm) into the osseous portion of the tube. At its inferomedial end, it is attached to a tubercle on the posterior edge of the medial pterygoid lamina.

**Cartilage Structure and Composition.**

The cartilage of the eustachian tube is intimately related to its functioning. Thus, function depends on cartilage structure, composition, and attachment to the cranial base and paratubal muscles. The cartilage of the tube is shaped like an inverted J in cross section (see Figure 5). The cartilage has been described as being composed of a short lateral lamina and an elongated medial lamina. Some investigators describe two laminae, the medial and lateral, but the cartilage is actually a dome-shaped structure with arms of different lengths. The lateral arm has a constant height. The medial arm, however, starts as a
short structure and increases rapidly in height just posterior and lateral to where the cartilage attaches to the medial pterygoid plate.

Elastic fibers are in the cartilaginous portion of the tube. Guild described a radial organization of elastic fibers around the dome of the cartilage, which suggests that motion of the lateral arm relative to the medial arm is possible. Matsune and colleagues found a rich, mesh-like distribution of elastin along the luminal side of the intermediate portion (also called the hinge portion) between the medial and lateral laminae of the tubal cartilage. They later speculated that this acted to return the lateral lamina to its original position at rest after contraction of the tensor veli palatini muscle and dilation of the upper half of the tube. Also, as stated before, the elastin in the cartilage is less dense in the infant than in the adult.

Cartilage mass increases from birth to puberty, and cartilage cell density is greater in infants. These differences may be related to the tube’s relatively ineffective opening by the tensor veli palatini muscle in infants and young children compared with that in older children and adults. In the young, the cartilage probably does not provide adequate support during attempts at opening and may buckle (see Chapter 3).

**Lateral Membranous Wall.** The lateral membranous wall is closely associated with the eustachian tube lumen. This structure is not clearly delineated but is invoked in many descriptions of eustachian tube function. It is most clearly defined in the middle part of the cartilaginous portion of the tube (see Figure 2A). Its medial boundary is the submucosa of the lumen. Laterally, a robust connective tissue layer is the insertion of the tensor veli palatini muscle. This fibrous lateral membrane is anchored superiorly to the inferior curvature of the lateral arm of the eustachian tube cartilage. The region between these two boundaries is occupied by glandular tissue anteriorly and adipose tissue posteriorly. Most likely, forces from the tensor veli palatini muscle during contraction are passed to the lateral arm of the cartilage and not to the lateral submucosa of the lumen.

**Ostmann’s Fat Pad.** Ostmann’s fat pad is the fat tissue in the inferolateral portion of the eustachian tube and most likely aids in closing the tube. As described before, the fat pad increases in volume after birth, primarily in height (associated with an increase in tubal height) but not in width; thus, it makes a greater mass in the child.

**Junctional Portion of the Tube**

Sudo and colleagues named the segment where the cartilaginous and osseous portions connect the junctional portion. They determined the length of this segment to be 3 mm. However, there is a gradual transition from the cartilaginous portion to the osseous portion; the lumen in the junctional portion increases from the proximal end (ie, pharyngeal margin) to the distal end (ie, the tympanic margin).

**Osseous Portion of the Tube**

The osseous portion of the eustachian tube—also called the protympanic, aural, bony, or middle-ear portion—is patent at all times when the middle ear is healthy. In contrast, the cartilaginous portion is closed at rest and opens only during swallowing or when it is forced open, such as during Valsalva’s maneuver. This segment of the tube lies completely within the petrous portion of the temporal bone and is directly continuous with the anterior wall of the superior portion of the middle ear. The juncture of the osseous portion of the tube and the epitympanum is 4 mm above the floor of the tympanic cavity (Figure 6A). This relationship is misrepresented in the more popular descriptions and depictions of the eustachian tube–middle ear juncture (too low in the mesotympanum) and is important in the functional clearance of middle-ear fluids.

The course of the osseous portion of the tube is linear anteromedially. It follows the petrous apex and deviates little from the horizontal plane. The lumen is roughly triangular and
measures 2 to 3 mm vertically and 3 to 4 mm horizontally. The medial wall of the bony portion of the eustachian tube consists of two parts: the posterolateral (labyrinthine) and the anteromedial (carotid), whose size, shape, and relationships depend on the position of the internal carotid artery. The average thickness of the anteromedial portion is 1.5 to 3 mm; it is absent in 2% of individuals, exposing the carotid artery. The aural orifice, an oval structure, lies above the floor of the middle-ear space and measures about $5 \times 2$ mm. The medial wall of the osseous portion of the tube lies close to the carotid canal and the labyrinth. Savic and Djeric found that in about two-thirds of the specimens they examined, the carotid canal noticeably impinged on the osseous portion of the eustachian tube.

The mucosal lining of this portion of the eustachian tube is similar to that of the middle ear and includes both mucus-producing and ciliated cells.

Figure 6. A. Anatomy of the osseous (ie, protympanic, middle-ear end, aural) portion of the eustachian tube as viewed from the external auditory canal. Note that the orifice of the eustachian tube is relatively high in the middle ear. B. Sagittally rotated coronal view of the middle ear and ossicles and the relationship to the inner ear.
Muscles of the Tube
Four muscles are associated with the eustachian tube: the tensor veli palatini, the tensor tympani, the levator veli palatini, and the salpingopharyngeus. Each has been directly or indirectly implicated in tubal function.\(^{47-49}\)

The eustachian tube is passively closed at rest. It opens during swallowing, yawning, or sneezing, permitting equalization of middle-ear and atmospheric pressures. Most anatomic and physiologic evidence supports active dilation induced either solely by the tensor veli palatini muscle\(^{50-52}\) or with help from the levator veli palatini muscle.\(^{42,53}\) Tubal closure has been attributed to passive reapproximation of the tube’s walls by extrinsic forces that surrounding deformed tissues exert, by the recoil of elastic fibers within the tubal cartilage, or by both mechanisms. More recent experimental and clinical data suggest that at least for certain abnormal populations, the closely applied internal pterygoid muscle may assist tubal closure by an increase in its mass within the pterygoid fossa. This increase applies medial pressure to the tensor veli palatini muscle and consequently to the lateral membranous wall of the eustachian tube (see Chapter 3).\(^{50,54,55}\)

**Tensor Veli Palatini.** The tensor veli palatini muscle is composed of two fairly distinct bundles of muscle fibers, the tensor veli palatini and the \textit{dilator tubae}, divided by a layer of fibroelastic tissue (see Figure 5). The bundles lie mediolateral to the tube. The more lateral bundle (the tensor veli palatini proper) is of an inverted triangular design. Its origin is from the scaphoid fossa and from the greater wing of the sphenoid bone superior to the eustachian tube. The force the muscle exerts on this origin creates the lateral osseous ridge of the sulcus tubarius. The muscle descends anteriorly, laterally, and inferiorly to converge in a tendon that rounds the hamular process of the medial pterygoid lamina about an interposed bursa. This fiber group then inserts into the posterior border of the horizontal process of the palatine bone and into the palatine aponeurosis of the anterior portion of the velum (Figure 7).

The dilator tubae is the medial bundle of the tensor veli palatini muscle and lies immediately adjacent to the lateral membranous wall of the eustachian tube. It was first described by Valsalva and subsequently confirmed by other anatomic dissections.\(^{48,56,57}\) Its superior origin is in the posterior half of the fibrous lateral membranous wall of the cartilaginous eustachian tube. The fibers descend sharply to enter and blend with the fibers of the lateral bundle of the tensor veli palatini muscle. This inner bundle is primarily responsible for active dilation of the tube. The angular relationship between the tensor veli palatini muscle and the cartilage varies in the infant but is relatively stable in the adult.\(^{15}\)
The dilator tubae of the tensor veli palatini muscle inserts only into the cartilaginous portion of the eustachian tube. It is important for dilation of the tube to equilibrate middle-ear pressure during swallowing. It is apparent that an inefficient tensor veli palatini will not effectively open the tube (ie, the tube will not open) (see Chapter 3).

**Tensor Tympani.** The more posterosuperior muscle fibers of the tensor veli palatini muscle lack an osseous origin and instead extend into the semicanal of the tensor tympani muscle. Here they receive a second muscle slip that originates from the tubal cartilage and sphenoid bone. These muscle fibers converge in a tendon that rounds the cochleariform process and inserts into the manubrium of the malleus. This arrangement gives a bipennate form to the tensor tympani muscle (see Figure 7). The tensor tympani does not appear to be involved in active dilation of the eustachian tube. However, stretch receptors in the tympanic membrane may be related to modulation of the middle-ear pressure through the tensor tympani, thereby affecting the tensor veli palatini opening the eustachian tube (see later).

**Levator Veli Palatini.** The levator veli palatini muscle arises from the inferior aspect of the petrous apex of the temporal bone. The fibers pass inferomedially, paralleling and lying beneath the tubal cartilage and luminal floor (see Figures 2A and 7). In most instances, the interaction between the levator veli palatini muscle and the posterior half of the cartilaginous eustachian tube lumen is precluded by an extension of the medial arm of the cartilage. Near the nasopharyngeal end of the eustachian tube, where the cartilage is at its maximal height, the levator veli palatini lies lateral to its medial arm. The fibers of this muscle insert by fanning out and blending with the dorsal surface of the soft palate. Most investigators deny a tubal origin for this muscle and believe that it is related to the tube only by loose connective tissue. We assessed eustachian tube ventilatory function both before and after bilateral excision of the levator palatini muscle within the palate and found no change in any parameter of the testing. The levator is not the primary dilator of the tube but probably adds to its support and contributes to its function by elevating the medial arm of the cartilage at the nasopharyngeal end of the eustachian tube.

**Salpingopharyngeus.** The salpingopharyngeus muscle arises from the medial and inferior borders of the tubal cartilage through muscular and tendinous fibers (see Figure 2A). The muscle then courses inferoposteriorly to blend with the palatopharyngeal muscle. Rosen examined 10 hemisected human heads and identified the muscle in 9 specimens. In all cases, there were few muscle fibers, and they appeared to lack any ability to perform physiologically.

**Innervation of the Tube**
The pharyngeal orifice of the eustachian tube is innervated by branches from the otic ganglion, the sphenopalatine nerve, and the pharyngeal plexus. The remainder of the tube receives its sensory innervation from the tympanic and the pharyngeal plexuses. The glossopharyngeal nerve probably plays the predominant role in tubal innervation. Sympathetic innervation of the tube depends on the sphenopalatine ganglion, otic ganglion, paired glossopharyngeal nerves, petrosal nerves, and caroticotympanic nerve. Mitchell suggested that the parasympathetic nerve supply is derived from the tympanic branch of the glossopharyngeal nerve. Nathanson and Jackson provided experimental evidence for a secondary parasympathetic innervation by the vidian nerve from the sphenopalatine ganglion. Innervation of the tensor veli palatini and tensor tympani muscles is from the ventromedial part of the ipsilateral trigeminal motor nucleus through the trigeminal nerve (mandibular division). The levator veli palatini muscle receives its innervation from the nucleus ambiguus through the vagus nerve.

**Blood Supply of the Tube**
Five arteries constitute the blood supply to the eustachian tube: the ascending palatine artery,
the pharyngeal branch of the internal maxillary artery, the artery of the pterygoid canal, the ascending pharyngeal artery, and the middle meningeal artery. Venous drainage is by the pterygoid venous plexus.13

**Lymphatics of the Tube**

An extensive lymph network is in the tunica propria of the submucosa of the eustachian tube, and it is more abundant in the cartilaginous portion than in the osseous portion. This network drains into either the retropharyngeal nodes medially or the deep cervical nodes laterally.13 Early investigators described a lymphoid mass within the tube of a 6-month-old infant.14 However, Wolff, in an examination of 250 subjects, and Aschan, in a histologic study of 39 eustachian tubes, failed to find such a structure.5,71 Further, in a study of the developmental anatomy of the tubal system, Rood and Doyle failed to find this lymphoid mass and concluded with Wolff and Aschan that this tubal tonsil was a rare pathologic abnormality.39

**ORGANS AT THE PROXIMAL AND DISTAL ENDS OF THE EUSTACHIAN TUBE**

At its proximal end, the eustachian tube system consists of the nose, pharynx, and palate. At its distal end, it consists of the tube, middle ear, and mastoid gas cells (see Figure 1).

**Nose**

The physiologic functions of the nose are an important part of a healthy eustachian tube system. Functions include humidifying, warming, and filtering gas—initially atmospheric air and then end-expiratory gas in the nasopharynx—supplied to the eustachian tube and then to the middle ear–mastoid. Patent nasal cavities are also important for physiologic functioning of the eustachian tube because nasal obstruction may result in abnormal nasopharyngeal pressures during swallowing (ie, the Toynbee phenomenon). Unfortunately, little information is available concerning nasopharyngeal pressures in the infant (see Chapter 3). However, in infants, with their relatively small nasal cavities, it is possible that nasopharyngeal (and mesopharyngeal and hypopharyngeal) pressures may be abnormal during certain activities (eg, sucking on the nipple or pacifier), even when the nose is open.

**Pharynx**

The nasopharynx is involved in the eustachian tube system, and the mesopharynx and hypopharynx may also be involved if there is abnormal anatomy or pathophysiology in this region. This is because pressures inferior to the palate can affect nasopharyngeal pressures during swallowing when nasal obstruction is present, that is, the Toynbee phenomenon.74 When there is an overt cleft of the palate or any velopharyngeal insufficiency, pressures in the entire pharynx may also affect the eustachian tube (and middle ear–mastoid). In the adult, when nasal obstruction is absent and velopharyngeal closure is adequate, the nasopharynx is the key part of the proximal end of the system. Pressures during swallowing should approximate ambient pressures.

**Nasopharynx**

The torus tubarius is a prominence on the lateral wall of the nasopharynx that protrudes into the nasopharynx (see Figure 2A). Abundant soft tissue overlying the cartilage of the eustachian tube forms this prominence. Anterior to this is the triangular nasopharyngeal orifice of the tube. From the torus tubarius, a raised ridge of mucous membrane descends vertically. This is the salpingopharyngeal fold. The adenoids, or pharyngeal tonsils, lie on the posterior wall and are composed of abundant lymphoid tissue. A variable depression within the mucous membrane, the pharyngeal bursa, is above the tonsils. Behind the torus tubarius is a deep pocket that extends the nasopharynx posteriorly along the medial border of the eustachian tube. This pocket, the fossa of Rosenmüller (see Figure 2B), varies from 8 to 10 mm in height and from 3 to 10 mm in depth.14 Adenoid tissue usually extends into this pocket and gives soft tissue
support to the tube. Niemela and colleagues studied the dimensions of the nasopharynx in 238 school-aged children using radiographs and found that children with a relatively small nasopharynx had a higher incidence of recurrent otitis media than did children with larger dimensions. The findings of this study were similar to one reported earlier by Maw and colleagues.

## Palate

The structure and function of the palate should be normal within the eustachian tube system. Competent velopharyngeal closure during swallowing is important in maintaining the physiologic functions of the eustachian tube. The integrity of the tensor veli palatini muscle within the palate is important for efficient active opening of the tube during swallowing because it is the only active dilator of the eustachian tube.

Fibers from the levator veli palatini muscle pass inferomedially, paralleling the tubal cartilage and lying within the vault of the tubal floor (see Figure 2A). They fan out and blend with the dorsal surface of the soft palate. Loss of function of the levator veli palatini muscle does not impair normal function of the active opening mechanism of the eustachian tube.

## Middle Ear and Mastoid Gas Cells

The eustachian tube’s primary physiologic function is to equilibrate gas pressure in the middle ear with ambient pressure to maintain normal compliance of the middle-ear structures for optimal hearing. Middle-ear abnormalities can affect the eustachian tube system. For example, a nonintact tympanic membrane can enhance liquid flow from the nasopharynx through a patent eustachian tube into the middle ear, that is, reflux (see Chapter 3).

The middle ear and the eustachian tube are connected anatomically and are intimately associated in function. The promontory of the middle ear has a neural gas-regulating reflex arc with the function of the eustachian tube (see later). The mastoid, with its system of interconnected cells, is also an important part of the eustachian tube–middle-ear system. No consensus exists concerning the function of these gas-filled cells. They most likely provide a “surge tank” of gas to maintain a normally pressurized middle ear. The mucosa lining the mastoid gas cells provides transfer of gases between the mastoid gas cells and the middle ear and mucosal blood vessels, or both functions coexist.

## Middle Ear

The middle ear is an irregular, laterally compressed, gas-filled space lying within the petrous portion of the temporal bone between the external auditory canal and the inner ear (Figure 8). This cavity can be considered to be divided into three parts superoinferiorly in relation to the tympanic membrane. The epitympanum, or attic, refers to that space lying above the superior border of the tympanic membrane. The mesotympanum lies opposite the membrane. The hypotympanum lies below the membrane. Within the middle ear are the ossicles: the stapes, incus, and malleus (Figure 9). At birth, the middle-ear cavity and associated structures are the size of an adult's. The vertical and anteroposterior diameters measure about 15 mm. The transverse diameter measures 4 mm at the epitympanum, 2 mm at the mesotympanum, and 6 mm at the hypotympanum. Because of these dimensions, the middle ear has been termed a cleft, or narrow box.

Superiorly, the cavity is bounded by a thin plate of bone, the tegmen tympani. It extends forward to cover the semicanal of the tensor tympani muscle and posteriorly to cover the attic, thus isolating the middle ear from the middle cranial fossa (see Figure 8). Anteriorly, the floor of the middle-ear cavity is raised to become continuous with that of the bony portion of the eustachian tube. Superiorly and beneath the tegmen tympani is the cylindrical semicanal for the tensor tympani muscle (Figure 10). It is separated from the eustachian tube by an upwardly concave thin bony septum, the cochleariform process. This process enters the
middle ear along its superomedial margin to end just above the oval window. There, it flares laterally. This termination of the cochleariform process serves as a pulley about which the tendon of the tensor tympani muscle makes a right-angled turn to proceed laterally to its insertion on the muscular process of the malleus.

The middle ear is bounded medially by the lateral surface of the bone covering the labyrinth of the inner ear. A bulbous, hollowed prominence formed by the outward projection of the basal turn of the cochlea occupies the position between the oval and round windows. This structure, the promontory, is cross-hatched by the various branches of the tympanic plexus of nerves. The Jacobson nerve, a branch of the glossopharyngeal nerve, enters the cavity through its floor, divides, and ramifies about the promontory to contribute to the plexus. The tympanic plexus has connections to the ventral subnucleus of the ipsilateral nucleus of the solitary tract within the brainstem. This has been postulated to provide sensory input from middle-ear chemoreceptors, baroreceptors, or both, and it is thus related to middle-ear ventilation. It has also been postulated that middle-ear baroreceptors may be stimulated by stretch receptors...
in the tympanic membrane that respond to alterations in external ear canal pressure. The lateral wall of the middle ear is formed by the tympanic membrane, the tympanic ring, and a portion of the squamous temporal bone called the septum. The posterior border of the middle ear is demarcated by the anterior wall of the mastoid cavity, the pyramidal prominence, and is connected to the mastoid antrum by the aditus ad antrum (see Figure 1).

**Tympanic Membrane.** The tympanic membrane is the lateral wall of the mesotympanum. Its integrity is essential to a healthy eustachian tube–middle ear; for example, a nonintact tympanic membrane can impair the tube’s protective function, resulting in reflux of nasopharyngeal secretions. Conversely, abnormal function of the eustachian tube can adversely affect the tympanic membrane and cause, for example, atelectasis of the tympanic membrane–middle ear (see Chapter 9).

The tympanic membrane has areas with relatively high compliance (ie, a lack of stiffness). This is related to the pathogenesis of retraction pockets and acquired cholesteatoma. Khanna and Tonndorf used holography to show that the pars flaccida and the posterosuperior quadrant of the pars tensa are the most compliant areas. Thus, extreme underpressures within the middle ear can cause these portions of the tympanic membrane to retract. This is the first stage of the development of a retraction pocket or cholesteatoma. The pars flaccida has high compliance because of its relatively thin medial fibrous layer and lack of an annulus. The posterosuperior portion is the most floppy quadrant of the pars tensa because it has a relatively large surface area. When the middle ear is full of effusion, these two areas become full, eventually bulge laterally, and can rupture. The anterosuperior and anteroinferior quadrants of the tympanic membrane are relatively stiff and can also rupture. The posterior portions are floppy and can either distend or retract with positive or negative middle-ear pressure, respectively.

**Mastoid Gas Cell System**

The mastoid is that portion of the petrous temporal bone that lies posterior to the middle-ear cavity. In the adult, it extends exteriorly and interiorly, forming a process to which the sternocleidomastoid muscle is attached superficially. The mastoid is filled with a system of interconnected cells. The mastoid antrum is posterior, continuous with the epitympanum, and the largest of these gas-filled spaces (see Figure 1). The antrum serves as a patent communication between the middle ear and the mastoid gas cells. In the infant, the mastoid process is small and the degree of pneumatization low. This small gas cell system may be partly related to the susceptibility of infants to high negative middle-ear pressure and, subsequently, otitis media. Between 5 and 10 years of age, pneumatization is, for the most part, complete. Incomplete development of the mastoid gas cell system has also been associated with frequent bouts of otitis media in infants and young children.
Mucous Membrane
The middle-ear and mastoid mucous membrane is continuous with that of the nasopharynx through the eustachian tube. This membrane covers all structures in the middle ear, including the ossicles, vessels, and nerves. Examination of cells of the mucous membrane within the tympanic cavity reveals a gradual change from tall, columnar cells with interspersed goblet cells to cuboidal cells at the posterior portion of the promontory and aditus ad antrum, antrum, and mastoid cells.

Innervation
Innervation of the tympanic cavity and its structures comes from the branches of the tympanic plexus of nerves. As described previously, the Jacobson nerve, a branch of the glossopharyngeal nerve, enters the cavity through its floor, divides, and ramifies about the promontory to contribute to the plexus. Eden and Gannon described the tympanic plexus as having neural connections to the ventral subnucleus of the ipsilateral nucleus of the solitary tract within the brainstem. This has been postulated to provide sensory input from middle-ear chemoreceptors, baroreceptors, or both and is thus related to middle-ear ventilation. In a later report from the same laboratory, a twofold increase in the average myelin thickness and a greater than threefold increase in the ratio of myelinated to unmyelinated fibers of the tympanic nerve (the afferent limb of the neural circuit) were observed from the newborn to the adult cynomolgus monkey.

Experiments in the same animal species have provided physiologic evidence that a neural arc exists between the middle ear and the eustachian tube. Differences in eustachian tube ventilatory function were described when the middle-ear gas composition was altered. This can be explained by the presence of baroreceptors, chemoreceptors, or both in the middle ear (see Chapter 3). Finding a feedback modulation of middle-ear pressure regulation, and the possible immaturity of the mechanism in young animal models, if it is confirmed to be present in the human, may be related to the relatively inefficient tubal ventilatory function in infants and young children. It has also been postulated that middle-ear baroreceptors may be stimulated by stretch receptors in the tympanic membrane that respond to alterations in external ear canal pressure.

Sympathetic innervation to the plexus is provided by the superior and inferior caroticotympanic nerves, and parasympathetic fibers are provided by the smaller superficial petrosal nerve. Oyagi and colleagues described sympathetic innervation of the middle-ear mucosa as arising from the ipsilateral superior cervical ganglion and not the stellate ganglion. They also showed that parasympathetic fibers probably arise from the ipsilateral pterygopalatine ganglion.

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