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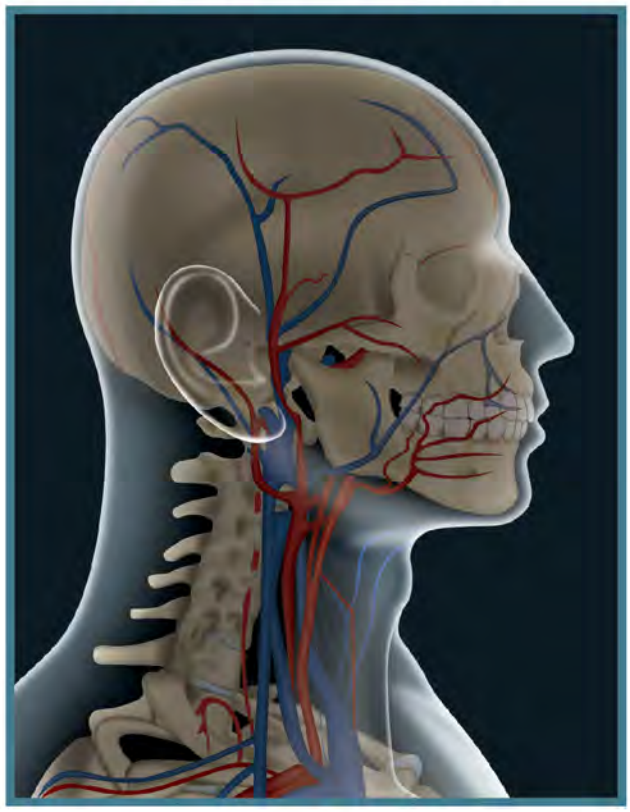
Sataloff's Comprehensive Textbook of
Otolaryngology
Head & Neck Surgery

Pediatric Otolaryngology

Series Editor
Robert T Sataloff



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CHAPTER 3

Impact of Evolution on Eustachian Tube Function and Otitis Media

Charles D Bluestone, J Douglas Swarts, Jeffrey T Laitman

Otitis media (OM) is a major health-care problem since it is the most frequent diagnosis made by health-care professionals who provide care for infants and children, and is not uncommonly encountered in adults. There is general agreement that the etiology and pathogenesis of middle-ear (ME) disease is multifactorial, but the consequences of human evolutionary history[1] for the presence and prevalence of OM have not been addressed until relatively recently. We have hypothesized that otitis media is most likely a disease that occurs only in humans and is unlikely to be present in other species in the wild.[2] Since hearing loss is associated with ME disease, if animals in the wild developed it they most likely would have been selected out by predation. Normal hearing is essential for survival in the wild.

We also posit that OM in humans is probably a consequence of human evolutionary history, i.e. a side effect of adaptations for bipedalism, the development of a large brain, and speech, producing changes in craniofacial skeleton that underlie the pathogenesis of this unique human disease. In this chapter, we describe these evolutionary events and how their consequences may impact management. Similarly, this evolutionary pathway may also contribute to the occurrence of rhinosinusitis in humans, which is also posited to be present only in humans, and is described in Chapter 27.

BIPEDALISM, BIG BRAIN, AND BORN TOO SOON

Habitual bipedalism is one of the distinguishing features of hominids when compared with our predecessors, including our nonhuman primate ancestors (Fig. 3.1). Adaptations for bipedality are evident in one of our earliest ancestors, *Ardipithecus ramidus*, who lived > 4 million years ago.[3] There is no consensus on the evolutionary advantages of either walking and running on two legs or knuckle walking as in the great apes over quadrupedal locomotor patterns. But among the hypothesized advantages of bipedality are improved thermoregulation due



Fig. 3.1: Cartoon depicting the locomotive posture of the chimpanzee (*Pan troglodytes*) with that of the human (*Homo sapiens*). With permission from Bluestone and Swarts.[2]

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to decreased exposure of the body to sun, ability to carry (including food and infants), the ability to see over the savannah for food and predators, and freeing of the hands, allowing tool- and weapon-making.

On the other hand, there are disadvantages to walking on two legs, such as disorders of the back and lower limb joints, both which are relatively common, but develop after reproductive age. But an even more significant disadvantage arising from bipedality is the constriction of the female pelvic outlet. The narrowing of the outlet is thought to arise as a consequence of the need for osseous support of the abdominal contents and changes that increase biomechanical efficiency in locomotion. For early hominids, these anatomical changes did not impair the delivery of newborns since their brains and bodies were small relative to their mother's size. However, during the subsequent 2 million years the hominid brain doubled in size, such a large increase that humans are born 12 months too early because of the constraints imposed by our big brain on passing through the narrowed pelvic outlet. This sequence of events is well known to anthropologists, as Martin[4] concluded, based on brain development, humans should have a 21 months' gestation period: 9 months in the uterus and 12 months outside the womb. This is illustrated in Figure 3.2 which depicts the relative sizes of the female bony pelvic birth canal to the size of the brain in the chimpanzee and human.[5] Delivery of human newborns through the birth canal is so tight that almost all pregnancies require birth attendants, a situation unique to humans. Anthropologists describe humans as being "secondarily altricial"; some higher mammals, including primates, are mature ("precocious") at birth, whereas others, such as dogs and cats, are "atricial", i.e. relatively helpless at birth. Humans should be precocious at birth but are not.

We previously described, in detail, the comparative anatomic and birthing differences between humans and our primate relatives, and the consequences of being "born too soon" for the ears, nose, and throat.[6] Among these consequences is that the Eustachian tube (ET) is too short and floppy during the first year of life. This structural and functional immaturity, in the context of an immature immune system, helps to explain the high incidence of acute OM in the first year of life, especially now that child day-care attendance exposes these highly susceptible babies to respiratory pathogens. A report from Norway finds recurrent ME infection more prevalent during the first 18 months of life in premature infants when compared with normal term babies. This difference was attributed to gestational age differences, not weight at birth[6]; thus, premature infants are "born way too soon". However, being born too soon does not explain why OM remains common throughout childhood and in some individuals into adulthood.

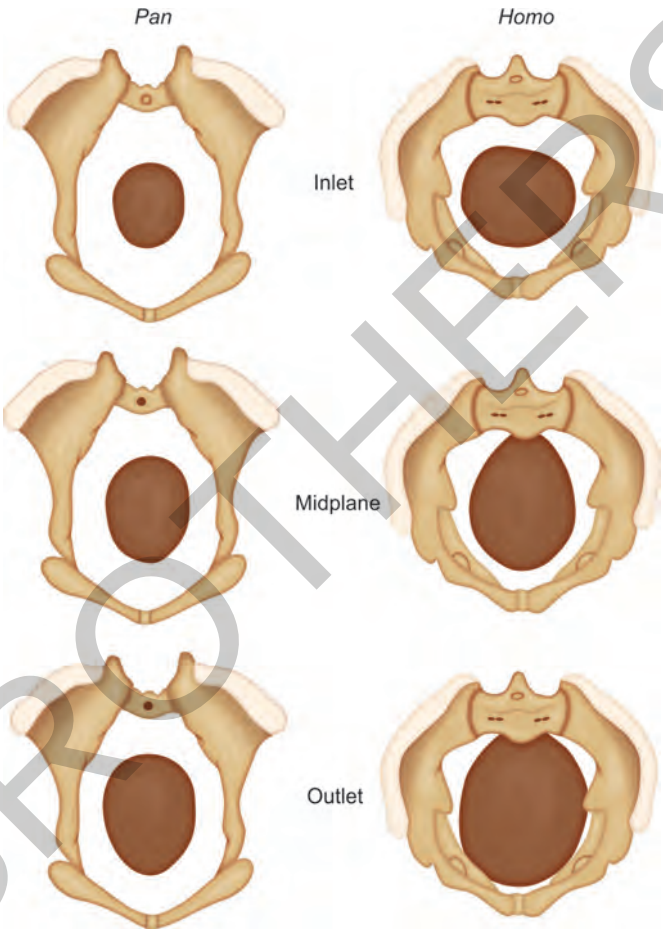

Fig. 3.2: Line drawing comparing the pelvic outlet with the brain size (shaded) of *Pan* (chimpanzee) to that of *Homo* (human) at birth. The brain of the chimpanzee easily fits in the relatively large pelvic outlet, but the human brain even at 9 months' gestation has an extremely tight fit. With permission from Bluestone and Swarts.[2]

LOSS OF HUMAN PROGNATHISM

Another important difference of modern humans, when compared with our hominid ancestors and extant non-human primates, is facial flattening, or the loss of facial prognathism.[7] This is depicted in Figures 3.3A and B, a human-chimpanzee skull comparison that shows the reduction and repositioning of the maxillofacial complex in the human. Facial flattening, along with descent of the hyoid, contributed to shortening of the palate.[8] We have described these evolutionary adaptations in detail elsewhere.[9] So why have we lost our facial prognathism?

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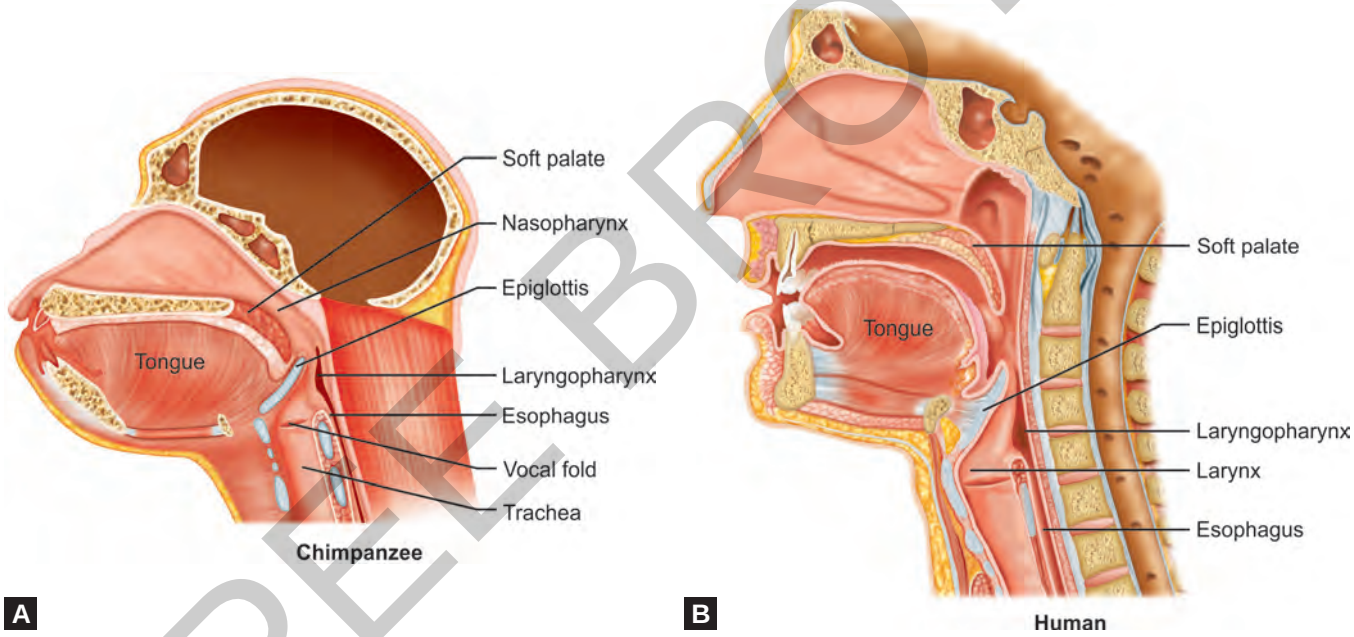
Figs. 3.3A and B: Comparison of the facial prognathism in skulls of the chimpanzee (A) and the facial flattening of the human (B).



Figs. 3.4A and B: Line drawing in the midsagittal plane comparing the chimpanzee and human showing the shorter palate, an oropharyngeal tongue, a narrower pharynx and a descended larynx (loss of "epiglottic—soft palate lock-up") in the human when compared with the chimpanzee.

Adaptation for Speech

Another unique adaptation of *Homo sapiens* is that we are the only species that developed speech. During our evolution, in a short 40,000 years, our larynx descended, elongating the supralaryngeal vocal tract that enhanced speech[8] (Figs. 3.4A and B). This adaptation narrowed the pharyngeal airway and shortened the palate, which probably aids in the production of vowels and consonants, but also has consequences for the palatal muscles (described later).

Cooking

Another possible explanation for our facial flattening is the cooking of food. As described by Wrangham,[10] the earliest evidence of cooking by hominids dates back about 1.8 million years to *Homo erectus*. He hypothesizes that cooking provided the necessary caloric density to meet the energy requirements of our rapidly growing brain. Cooking reduces the need for large masticatory forces releasing the selective pressures on the teeth and musculature, thus allowing a decrease in dental and craniofacial size, i.e.

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a smaller maxilla and mandible, in comparison to other hominids. We consider this to be an attractive hypothesis to explain both the increase in human neonatal body and brain size and the loss of facial prognathism.[11]

EUSTACHIAN TUBE FUNCTION RELATED TO ALTERED MORPHOLOGY OF THE PALATE

We have proposed that the loss of prognathism altered palatal morphology including the muscles of the palate involved in ET function, the tensor veli palatini, and levator veli palatini muscles. These muscles became less effective physiologically than in the nonhuman primate (*Macaca mulatta*).[2] When we compared the tubal function of monkeys and humans, monkey dilatory tubal function was consistently superior.[12] Humans have relatively poor active tubal function. The consequences of poorer tubal function becomes evident during activities that impose nonphysiologic stresses on the ET, such as flying in airplanes and scuba diving, activities in which equalizing ME negative pressure becomes problematic. In contrast, even the application of sudden large negative ME pressures in the monkey were easily equilibrated with a single swallow.

The relatively poor function of the human ET compared with that of the monkey is most likely due to differences in the anatomy of the paratubal muscles. Figure 3.5 is a cross section of the ET from a human temporal bone specimen showing a relatively slim tensor veli palatini muscle attached to the lateral lamina of the tubal cartilage and the relatively large belly of the levator veli palatini muscle as a rounded mass that abuts the inferior portion of the tubal lumen. Despite their genomic similarity and their use as a model for studies of ET physiology and pathophysiology and the pathogenesis of OM, rhesus monkey paratubal muscles are not identical to those of humans.[13] Comparison of the anatomy of these two muscles between the monkey and the human reveals two major differences: first, the monkey tensor veli palatini muscle is bulkier and attaches along the entire length of the cartilaginous ET, whereas in the human this muscle is less robust and only attaches to the midportion of the ET; and second, the belly of the levator palatini muscle is not as prominent and does not impinge on the inferior portion of the tubal lumen in the monkey in contrast to the human condition (Figs. 3.6A and B).

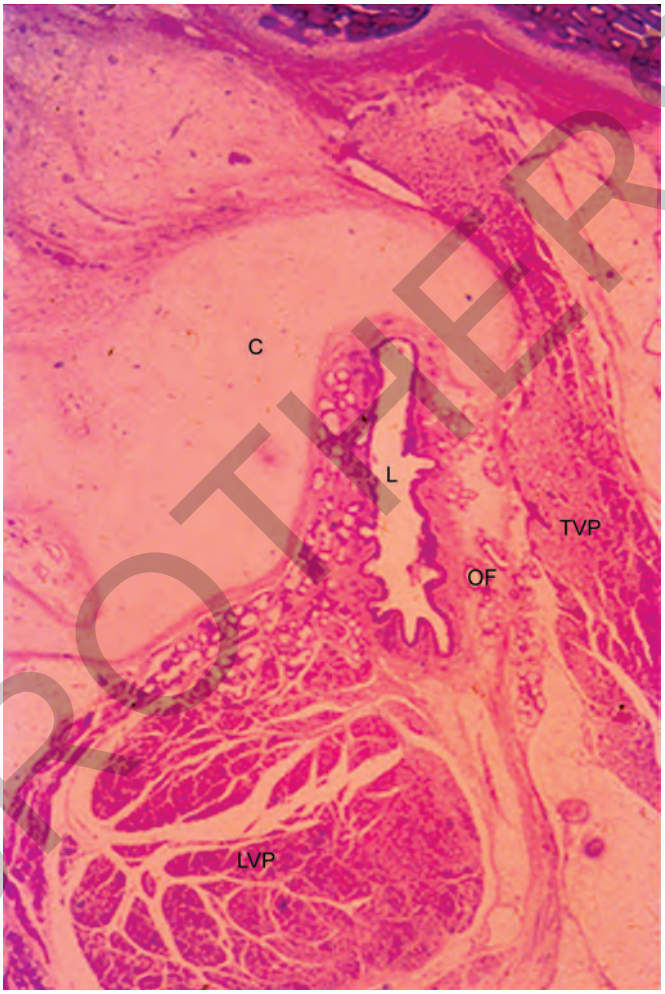

Fig. 3.5: Cross section through the midcartilaginous portion of the left human Eustachian tube, showing the robust rounded belly of the levator veli palatini muscle abutting the inferior portion of the tubal lumen, and the rather thin slip of the attachment of the tensor veli palatini to the lateral lamina of the tube. (C: Tubal cartilage; L: Tubal lumen; LVP: Levator veli palatini muscle; OF: Ostmann's fat pad; TVP: Tensor veli palatini muscle).
Courtesy: Late I Sando, MD (1928-2014).

PATHOGENESIS OF HUMAN OTITIS MEDIA RELATED TO MUSCLES OF THE EUSTACHIAN TUBE

In contrast to the prevalence of OM in humans, especially in infants and young children, we have never observed spontaneous ME disease in the outbred animals (e.g. ferrets and monkeys) used in our laboratory over the past 30 years. Is this remarkable difference in the rate of OM related to the comparatively poor human ET function, a consequence of differences in paratubal muscular anatomy

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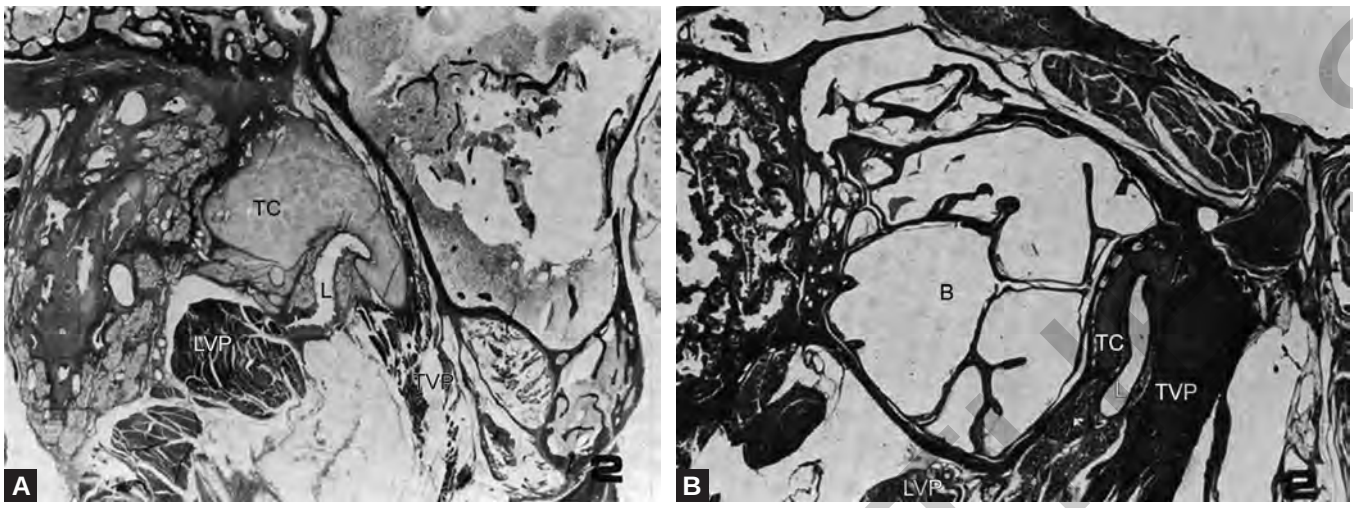

Figs. 3.6A and B: Coronal view of the left Eustachian tube and related structures in the temporal bone specimen from a 13-year-old human female (A) compared with a coronal view of the left Eustachian tube in a temporal bone specimen from a rhesus monkey (B). Note that the levator veli palatini muscle in the human has a rounded belly and closely approximates the inferior portion of the tubal lumen. In the monkey, this muscle is more sparse and there is a distinct separation between the muscle and tubal lumen. Also note, the robust belly of the tensor veli palatini muscle in the monkey compared with the human. (TC: Tubal cartilage; L: Tubal lumen; TVP: Tensor veli palatini muscle; LVP: Levator veli palatini; B: Bulla of monkey). With permission from Doyle and Rood.[13]

in *H. sapiens* as compared with the monkey? Experiments with the monkey in our laboratory undergirded our understanding of the pathogenesis of ME disease in the human.[14] We have successfully created OM in this animal model by inactivating the tensor veli palatini muscle either by severing the tendon at the hamulus of the pterygoid bone or by injecting botulinum toxin into its belly. The consequence of these manipulations is an inability to dilate the ET lumen during swallowing, resulting in a ME effusion. Our conclusion was that a healthy tensor veli palatini muscle is critically important in the prevention of ME disease. We postulated that the monkey's larger tensor veli palatini muscle with a longer insertion on the cartilaginous portion of the ET could explain their excellent tubal function. Furthermore, if tubal inflammation did occur in the monkey its efficient ET function would prevent the development of ME disease. By contrast, since humans have comparatively poor tubal function and are susceptible to upper respiratory tract infections, OM is a common disease. Buchman and colleagues[15] reported that some adult volunteers who had nasal challenge with virus developed ME disease. Why viral infection affected some subjects and not others may be explained by the results of a later study in which adult volunteers who had signs of tubal dysfunction prior to a viral challenge developed more severe dysfunction and ME negative pressure. Subjects with good tubal function before the challenge did not develop OM.[16] We concluded from these observations and experiments that the relative inefficiency of the human tensor veli palatini muscle is a viable explanation for the pathogenesis of ME disease in some individuals, especially under nonphysiologic and inflammator conditions.

Obviously, due to their transient nature, these conditions cannot be invoked to explain why ME disease persists in a subgroup of patients. We have reported that older children and adults with chronic OM with effusion had ET dysfunction characterized by constriction of the lumen as opposed to dilation during swallowing during the forced response test.[17] This observation in the context of experimental electrical stimulation of the monkey paratubal muscles during the forced response test[18] suggests that the observed constriction is most likely due to contraction of the levator veli palatini muscle, which collapses the dilated tubal lumen. This hypothesis has yet to be confirmed, but is currently under investigation in our laboratory.

Eustachian tube constriction has been identified in children with cleft palate. The infant with an unrepaired cleft palate is a natural model of chronic OM with effusion, due to a functional, as opposed to an anatomic, obstruction of the ET. Similarly, surgical clefting of the monkey soft

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palate induced OM with effusion that persisted until the cleft had healed.[19] During the interval when the soft palate was clefted, ET function tests revealed constriction of the tubal lumen during swallowing that we now attribute to a dysfunction of the levator veli palatini muscle. We posit that in an effort to prevent OM in these babies, surgical repair of the palate should focus on anchoring the tensor veli palatini muscle at the hamulus and repositioning the levator veli palatini muscle to preclude tubal constriction, as well as correcting their velopharyngeal insufficiency and hypernasal speech.

OTHER FACTORS INVOLVED IN PATHOGENESIS

The above describes the consequences of our evolutionary history for the pathogenesis of OM in humans; however, the extremely high incidence of OM today cannot be explained solely as a consequence of evolutionary adaptations in humans. Other factors, such as heredity and immunity,[20,21] as well as those derived from living in novel environments (those we are not adapted to) such as a decrease in breast-feeding, smoking in the household, use of pacifiers and very importantly, child day-care attendance, are well known to increase the risk of ME disease. [22]

SUMMARY AND CONCLUSION

It is well known that the pathogenesis of OM is multifactorial, but the role of evolutionary adaptation on its development has not been addressed until recently. Our hypothesis is that OM is most likely restricted to humans, in contrast to other species in the wild, because the associated hearing loss would have reduced the fitness of affected individuals. The byproducts of two human adaptations may have resulted in an increased prevalence of OM: the interaction of bipedalism with increased brain size, and the loss of facial prognathism, which may have been the result of adaptation for speech or the advent of cooking almost 2 million years. As a consequence of our adaptation for bipedalism, the female pelvic outlet is constricted, which in the context of a rapidly enlarging brain results in humans being born 12 months too soon. Significantly, immature ET structure and function in conjunction with an immature immune system helps to explain the high incidence of OM in the first year of life. But the persistence of ME disease beyond that stage is not explained by "immaturity". The morphology of the palate changed with the adaptations that produced facial flattening, with concomitant effects on the paratubal muscles that affected ET function. These changes resulted in relatively poor human physiologic tubal function in comparison to a nonhuman primate.

REFERENCES

1. Nesse RM, Williams GC. Why we get sick: the new science of Darwinian medicine. New York: Vintage Books; 1994.
2. Bluestone CD, Swarts JD. Human evolutionary history: consequences for the pathogenesis of otitis media. Otolaryngol Head Neck Surg. 2010;143(6):739-44.
3. Lovejoy CO. Reexamining human origins in light of Ardipithecus ramidus. Science. 2009;326(5949):74e71-78.
4. Martin RD. Primate origins and evolution: a phylogenetic reconstruction. New Haven, CT: Princeton University Press; 1990.
5. Tague RG, Lovejoy CO. The obstetric pelvis of AL-288-1 (Lucy). J Hum Evol. 1986;15(4):237-55.
6. Bluestone CD. Humans are born too soon: impact on pediatric otolaryngology. Int J Pediatr Otorhinolaryngol. 2005;69(1):1-8.
7. Schumacher KU, Koppe T, Fanghanel J, et al. Morphometric studies on the facial skeleton of humans and pongids based on CT-scans. Kaibogaku Zasshi. 1994;69(5):636-44.
8. Laitman JT, Reidenberg JS. Evolution of the human larynx: nature's great experiment. In: Fried M, Ferlito A (eds), Larynx, 3rd edn. San Diego, CA: Pleural Publishing; 2009. pp. 19-38.
9. Bluestone CD. Impact of evolution on the Eustachian tube. Laryngoscope. 2008;118(3):522-7.
10. Wrangham R. Catching fire: how cooking made us human. New York, NY: Basic Books; 2009.
11. Bluestone CD. Galapagos: Darwin, evolution, and ENT. Laryngoscope. 2009;119(10):1902-5.
12. Swarts JD, Ghadiali SN. Interspecies comparisons of Eustachian tube flow patterns during swallowing in the forced-response test. Paper presented at: 9th International Symposium on Recent Advances in Otitis Media; June 3–7, 2007; St. Pete Beach, FL.
13. Doyle WJ, Rood SR. Comparison of the anatomy of the Eustachian tube in the rhesus monkey (*Macaca mulatta*) and man: implications for physiologic modeling. Ann Otol Rhinol Laryngol. 1980;89(1 Pt 1):49-57.
14. Bluestone CD. Studies in Otitis Media: Children's Hospital Of Pittsburgh-University of Pittsburgh Progress Report-2004. Laryngoscope. 2004;114(11 PART 3).

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15. Buchman CA, Doyle WJ, Skoner D, et al. Otologic manifestations of experimental rhinovirus infection. Laryngoscope. 1994;104(10):1295-9.
16. Doyle WJ, Seroky JT, Angelini BL, et al. Abnormal middle ear pressures during experimental influenza A virus infection—role of Eustachian tube function. Auris Nasus Larynx. 2000;27(4):323-6.
17. Swarts JD, Bluestone CD. Eustachian tube function in older children and adults with persistent otitis media. Int J Pediatr Otorhinolaryngol. 2003;67(8):853-9.
18. Cantekin EI, Doyle WJ, Reichert TJ, et al. Dilation of the eustachian tube by electrical stimulation of the mandibular nerve. Ann Otol Rhinol Laryngol. 1979; 88(1 Pt 1):40-51.
19. Doyle WJ, Cantekin EI, Bluestone CD, et al. Nonhuman primate model of cleft palate and its implications for middle ear pathology. Ann Otol Rhinol Laryngol Suppl. 1980;89(3 Pt 2):41-6.
20. Casselbrant ML, Mandel EM, Fall PA, et al. The heritability of otitis media: a twin and triplet study. JAMA. 1999; 282(22):2125-30.
21. Underwood M, Bakaletz L. Innate immunity and the role of defensins in otitis media. Curr Allergy Asthma Rep. 2011; 11(6):499-507.
22. Casselbrandt ML, Mandel EN. Epidemiology. In: Bluestone CD, Rosenfeld RM, eds. Evidence-based otitis media. Hamilton, Ontario: BC Decker; 2003. pp. 147-62.

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