

# The Auditory System

*Anatomy, Physiology,  
and Clinical Correlates*

**SECOND EDITION**

Editor-in-Chief for Audiology  
**Brad A. Stach, PhD**



# The Auditory System

*Anatomy, Physiology,  
and Clinical Correlates*

**SECOND EDITION**

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## PREFACE

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The first edition of our book, *The Auditory System: Anatomy, Physiology, and Clinical Correlates* was written to provide a comprehensive text on the anatomy and physiology of the peripheral as well as the central auditory systems—an approach that is maintained in the current edition. The approach to this book is slightly different than what is generally planned for books on the structure and function of the auditory system. This book is written primarily for graduate students with a clinical slant in hope of drawing more future and current practitioners into the important process of reading and learning more about the anatomy and physiology of the auditory system. After conducting surveys as well as extensive discussions with students and clinicians, we have learned several concepts and approaches that may make a book on anatomy and physiology more appealing to graduate students and clinicians. These concepts and approaches follow and provide the impetus for this book.

1. As noted by the title of this book, we will highlight clinical correlates to the basic science principles that are being presented. Whenever possible, a case study, a brief review, or a clinical comment will be connected to the basic science principle being discussed. This added clinical information will be highlighted in the text as *clinical or pathologic correlates*. The purpose of this feature is to help establish the link between science and practice in a brief but relevant way. We believe this will make this text more interesting and useful for the clinically oriented student and professional.
2. This book makes generous use of secondary references because many review chapters and articles are often easier to follow and are more relevant to the graduate student and the clinician. Our interaction with clinicians has taught us that basic science articles are not usually read—even when recommended. Instead, review articles are sought for a better grasp on the subject. Hence, we have tried to provide some key basic science readings (original or key articles) for each of the topics covered, and whenever possible, we have also included review articles or chapters as supplementary references. Finally, we have tried to select basic science articles that have a clinical slant or clinical implications for inclusion in this text.
3. In the past decade or two, considerable interest and research has been focused on neuroscience and the central auditory system. However, prior to this time, far more attention was devoted to the peripheral system. In this text, we have tried to balance the coverage of the peripheral and central systems so that the reader can be exposed to both portions of the auditory system in sufficient depth to be able to appreciate the nature of the processing that occurs at the two levels. The text will present information regarding the similarities in the auditory processes conducted at the two levels, as well as the unique types of processing that occur not only between the two levels, but also among some of the structures within each level of the auditory system.
4. Although it has been difficult to do, we have tried to use the human model as much as possible in our discussion of the anatomy and physiology of the auditory system. So much work has been completed

on animals and so little on humans that it often was a challenge to limit the discussion to the human model—but a serious effort has been made to do this in this book.

5. This book is aimed at the graduate student—especially those enrolled in doctor of audiology (Au.D.) programs who need to understand the anatomy and physiology of the human hearing mechanism as it is relevant to clinical audiology. A number of Au.D. programs have split their hearing science course into two courses: one on anatomy and physiology and one on psychoacoustics. We feel this is an appropriate approach and one for which this book is well suited. This book was also written to provide the practicing clinician with a relevant reference source that can be easy to use.
6. The fields of anatomy and physiology have exploded, with large quantities of new research information appearing in the literature on a regular basis. Most of this is important indeed, but it is not always relevant to the clinician. We therefore have tried to sort out what is most salient to the audiologist (perhaps our most difficult task) and keep the book at a reasonable length.

## IMPORTANCE TO THE CLINICIAN

An understanding of the biological aspects of the auditory system is essential to the knowledgeable clinician. At first glance many clinicians perhaps wonder how knowledge of anatomy and physiology of the auditory system will help them in their everyday activities. However, if one closely reflects upon what a clinician in hearing and hearing disorders does on any given day, the role of biology becomes obvious. A number of clinical activities come to mind that are dependent on the provider being familiar with the structure and function of the auditory system.

Communication with other clinicians in the same as well as different disciplines can be enhanced greatly by understanding anatomy and physiology. Long-lasting opinions are formed in the clinical arena by brief discussions of difficult patients, diagnoses, and treatments. One who is well grounded in anatomy and physiology can better understand and contribute to these types of discussions.

Clinicians are responsible, to varying degrees, for test selection and interpretation. These are the evaluation tools that are critical to the proper diagnosis of the patient. Tests are a measure of function and function is intimately related to structure. Knowing how to administer a test but not understanding the underlying functions that it is assessing is doing only half of the job. For example, an absent acoustic reflex is of little value if one does not know the anatomy of the acoustic reflex circuit. Clinicians well oriented toward the anatomy and physiology of the hearing mechanism are in a much better position to optimally utilize tests and interpret test results than those who are not.

Radiological information is becoming more available and more sophisticated. Diagnostic clinicians can be helped by radiological information on a patient. However, radiology is a specialty that is based on anatomy. Without anatomical knowledge, radiological information cannot be utilized efficiently. Correlating test results with radiological findings is the backbone of diagnostic audiology, and the establishments of such correlations (or lack of) cannot be accomplished without a solid anatomical grounding.

Clinicians work with people who have a variety of auditory disorders. In order to understand a given disorder, the locus and function of the structure(s)

affected must be known. At times the anatomy and physiology related to a given disorder may provide insight as to the nature of the problem and what is to be expected. For example, we know that kernicterus primarily compromises the cochlear nucleus in the brainstem even though it often manifests as a high-frequency sensorineural hearing loss, which could be interpreted as a peripheral problem. Utilizing only a peripheral audiological evaluation would miss the key aspects of this disorder.

Finally, patient counseling is dependent on knowledge of the anatomy and physiology of the auditory system. Better explanations of the patient's problems come from clinicians who understand the basics of structure and function. Anatomical models and illustrations can help enhance understanding for the patient. With common use of the Internet, patients are more knowledgeable and can ask demanding questions about the underlying anatomy and physiology of their disorders. Many of them know and appreciate what has been known in science for many years: that anatomy along with its physiology is the common denominator for understanding how we hear or do not hear. It therefore is critical for hearing health care professionals to have a solid grounding in anatomy and physiology in order to be able to effectively counsel patients with auditory disorders and to be able to answer their patients' questions with knowledge and confidence.

## NEW FEATURES

In our second edition of this book the reader will notice a number changes and updates. Though not all of these can be covered in detail here, there are several changes that are worth mentioning. One of the major additions is Chapter 16, which presents an overview of normal development of the auditory system, plasticity of the central auditory system, and aging effects on the peripheral and central auditory systems. This new chapter reviews basic studies that focus on auditory system changes over the lifespan that have a clinical impact. Chapter 3 includes several new illustrations of the anatomy and physiology of the middle ear. Among the new illustrations is a set of photos showing a variety of pathological conditions of the tympanic membrane. In Chapter 4 on cochlear anatomy the reader will notice several new illustrations and a discussion of synaptic ribbons highlighting novel findings in regard to deleterious interactions between the inner hair cells and the auditory nerve when exposed to noise without the usual accompanying hearing loss. In Chapter 6 some new views on the role of neuropharmacology of cochlear function are discussed. Chapter 12 includes both text and illustrative changes which focus on a discussion of cryoloop cooling as a new procedural approach to better elucidate the effects of pathology on the auditory cortex. In addition, new information on tonotopic organization of the auditory cortex and the variable locus of the angular gyrus is offered in this chapter. Finally, in Chapter 14 updated information on the vascular network of the brainstem, specifically the vertebrobasilar system, is provided.

## CLOSING COMMENTS

We trust that this book has helped make explicit the link between the structures and functions of the auditory system and the specific clinical correlates that are tied to the underlying basic science principles and concepts that have been presented. It was with much deliberation and careful consideration of this

intent that we decided on the title for our text: *The Auditory System: Anatomy, Physiology, and Clinical Correlates (2nd Edition)*. Finally, we hope the reader of this book will become a better, more informed provider of care to individuals with hearing loss and hearing deficits, having gained these insights.





## ACKNOWLEDGMENTS

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The second edition of *The Auditory System: Anatomy, Physiology, and Clinical Correlates* presents a new set of challenges for which I received much help from many people. I am grateful to many people who have helped me in a variety of ways with this book. For many years Jane Baran, my coauthor, has been a close friend and colleague. Her many contributions to the completion of this work have been immeasurable, and I thank her for these and her continued support. A special thanks to The Royal Arch Research Assistance (RARA) people who have helped support much of the work that contributed to this book. I also want to thank Tony Sahley for his insightful review and revisions of Chapter 6. I would also like to acknowledge the help from my students in the NeuroAudiology Lab, Speech, Language and Hearing Department at the University of Arizona—at the risk of omission they include: Nicole Denny, Barrett St. George, Alyssa Everett, Laura Sommerfield, Bryan Wong, and Aaron Whitely. Appreciation is also extended to Plural Publishing—especially Angie, Val, and Nicole for all their help and encouragement.

Finally I wish to thank my gracious and lovely wife, Sheila, and my two wonderful sons, Erik and Justin, for their constant support and interest in everything I do.

—FM

I also am grateful to the many fine individuals who have contributed in many important ways to this book. First and foremost I want to thank my colleague and coauthor, Frank Musiek. I have had the privilege and honor of working with him for many years, and this book is but one of the fruits of this collaboration. Without his diligence, persistence, and insights, this book would not have become a reality. He is one of those rare individuals who is not only a good colleague but also a valued and trusted friend.

I also would like to recognize the individuals that Frank Musiek has identified in his acknowledgments. I will not mention all of these individuals again here, but I would like to add my sincere thanks to all of these individuals, as each of them has made an important contribution to the completion of this edition of our book, *The Auditory System: Anatomy, Physiology, and Clinical Correlates*.

Finally, I especially want to thank my husband, David Hoffman, and my daughter, Sarah Jane, who continually offered their encouragement, assistance, and support throughout this endeavor.

—JAB





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**Jane A. Baran, PhD, CCC-A/SLP**, is Professor Emerita in the Department of Communication Disorders at the University of Massachusetts Amherst. Her primary research and clinical interests are in the areas of (central) auditory processing disorders, auditory evoked potentials, and neuroanatomy and neurophysiology of the auditory system. She is coauthor of three books, *Neuroaudiology: Case Studies*, *The Auditory System: Anatomy, Physiology and Clinical Correlates*, and *Disorders of the Auditory System*. In addition, she has authored or co-authored a number of journal articles and book chapters in the areas of normal and disordered auditory processing, language processing, auditory evoked potentials, and auditory neuroanatomy and neurophysiology. She has presented numerous research papers and educational workshops at regional, national, and international meetings. Dr. Baran is a Fellow of the American Speech-Language-Hearing Association (ASHA), the 2001 Recipient of the Clinical Educators Award from the American Academy of Audiology, a 2005 Recipient of a Recognition Award for Special Contributions to Higher Education from ASHA, and the 2013 Recipient of the Journal of the American Academy of Audiology Editor’s Award. She also is one of a select group of scholars to receive the Carnegie National Scholar Fellowship Award from the Carnegie Foundation for the Advancement of Teaching and Learning.





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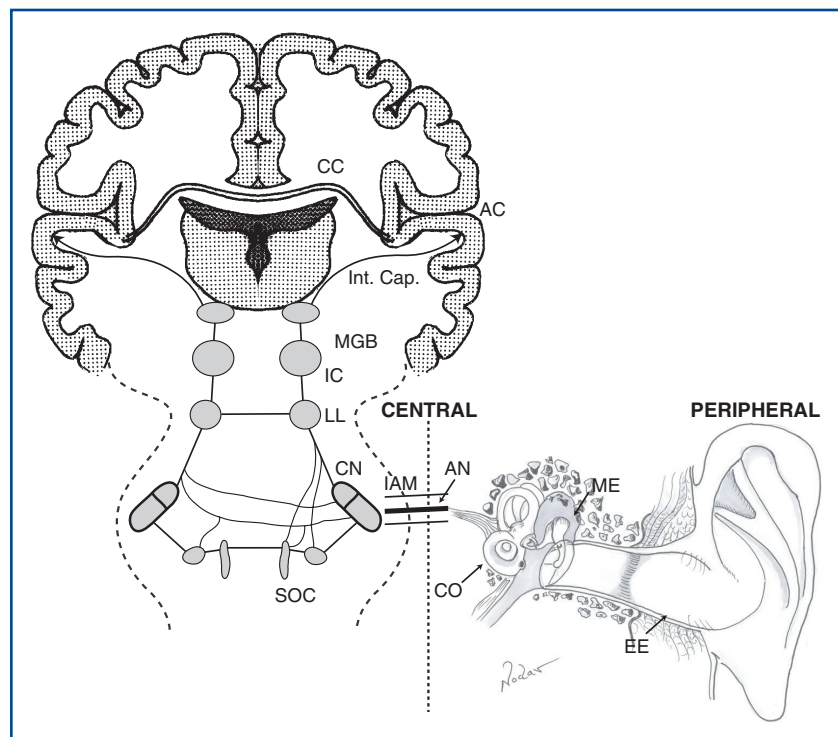
# 1

## Overview of the Anatomy and Physiology of the Auditory System



### INTRODUCTION TO THE AUDITORY SYSTEM

One of the key features of this book is a balanced review of the two major portions of the auditory system; i.e., the peripheral and central auditory systems (Figure 1-1). The peripheral system includes the outer ear, the middle ear, the cochlea, and the auditory nerve (AN). The central auditory system includes the cochlear nucleus (CN), the superior olivary complex (SOC), the lateral lemniscus (LL) (both nuclei



**Figure 1-1.** Highly schematized drawing of the peripheral and central auditory systems. KEY: EE = external ear and canal, ME = middle ear, CO = cochlea, AN = auditory nerve, IAM = internal auditory meatus, CN = cochlear nucleus, SOC = superior olivary complex, LL = lateral lemniscus, IC = inferior colliculus, MGB = medial geniculate body, Int. cap. = internal capsule, AC = auditory cortex, CC = corpus callosum.

and pathways), the inferior colliculus (IC), the medial geniculate body (MGB), the auditory subcortex (subcortical white matter and basal ganglia region), the cortex, and the interhemispheric pathways (including the corpus callosum).

The peripheral auditory system is located for the most part in the temporal bone, which is part of the cranium, and the central auditory system is located in the brain. Specifically, the CN, SOC, and LL are situated in the pons, the IC is in the midbrain, and the MGB is in the caudal thalamus. The auditory subcortex and cortex involve structures such as the internal capsule, the insula, Heschl's gyrus, the planum temporale, and other parts of the superior temporal gyrus. Auditory responsive areas also include segments of the frontal lobe, the parietal lobe, the angular gyrus, the supramarginal gyrus, and the corpus callosum. This entire system is often referred to as the auditory afferent system, meaning it courses from the ear up to and including the brain. There is also an efferent system, which is almost the reverse of the afferent system in that it runs a similar, but not an identical, route but from the cortex down to the cochlea.

In general, the auditory system performs two kinds of processing of acoustic stimuli—sequential and parallel. Sequential processing involves the transferring of information from one area or level of the auditory system to the next. This type of processing lends itself to a hierarchical organization as one ascends the auditory afferent system. Parallel processing, on the other hand, involves overlapping functions that occur at about the same time along different channels. Both of these major types of processing are needed for optimal



## CLINICAL CORRELATE

In diagnostic audiology, considerable effort is expended in differentiating conductive hearing loss from sensorineural hearing loss. Conductive hearing loss involves dysfunction or compromise of the conductive apparatus (i.e., the outer and/or middle ear). Most conductive hearing losses can be medically or surgically corrected, whereas the majority of sensorineural losses are not typically amenable to medical intervention, which means that they tend to be permanent. The hearing deficits associated with conductive and sensorineural losses are quite different in their nature; therefore, it is important for the clinician to differentiate these two types of losses so that the best management approaches can be implemented.

Although much of the early diagnostic work in audiology was directed toward differentiating conductive versus sensorineural hearing losses, the focus was centered primarily on differentially diagnosing conductive versus "sensory" hearing losses (i.e., hearing losses originating within the inner ear or cochlea). More recently, however, because of advances in both basic science and diagnostic methods, it has become important for the audiologist to identify cochlear versus retrocochlear (all structures beyond the cochlea) dysfunction. This need to differentially diagnose cochlear versus retrocochlear lesions was initially driven by the fact that many lesions within the auditory system

involved lesions of the AN or serious lesions of the brain. Hence, the accurate identification of the site of lesion had important implications for the medical management for many patients with retrocochlear lesions. Even more recently, it has become important for audiologic management purposes that cochlear sites of lesion be differentiated from retrocochlear sites even if the lesions are benign. This is because a different set of hearing problems arise from compromise of the retrocochlear system compared with those seen with involvement of the cochlea. Most recently, audiologists have begun to diagnose and manage central auditory disorders or central auditory processing disorders (CAPDs). These disorders are caused by dysfunction or compromise of the auditory system in the brain. Central auditory disorders are retrocochlear disorders that exclude disorders arising from the AN.

It therefore has become increasingly important for audiologists to know and to be able to define the different areas of the auditory system as various regions are responsible for various auditory functions. Each area within the auditory system contributes its own special aspect of acoustic signal processing. However, it is also important to realize that the auditory system is well orchestrated and works as a whole. The different parts of the auditory system are in fact interdependent.



functioning of the auditory system. Sequential processing takes place in the entire auditory system, but parallel processing takes place, for the most part, in the neural system. Examples of parallel processing are noted throughout the auditory neural system. The AN has type I and type II fibers that proceed in parallel to the brainstem. Likewise, the left and right sides of the brainstem pathways proceed in parallel to the cortex. The cortex is highly sequential but also has separate groups of fibers coursing in parallel between the primary and secondary auditory cortex as well as to other areas of the brain (Rouiller, 1997).

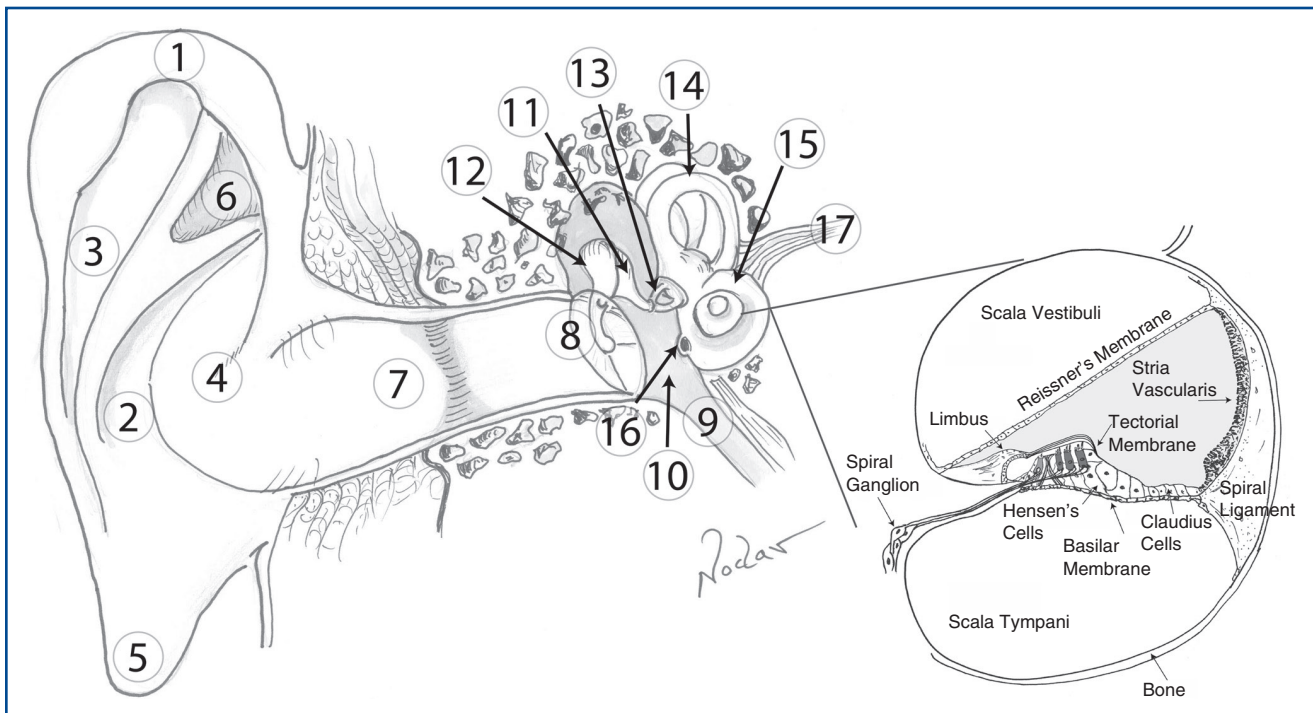
Another principle that applies to the entire auditory system, although it is manifested primarily at the cochlear, AN, and brain levels, is the influence of inhibition and excitation. More evidence is emerging to indicate that a balance between excitation and inhibition processes in the auditory system (as well as other systems) is critical for normal function. It is speculated that maladies such as tinnitus and hypersensitivity to sound may have as their basis an imbalance between inhibition and excitation in the auditory system.

## THE PERIPHERAL AUDITORY SYSTEM

### External and Middle Ear

#### *Anatomy and Physiology*

Sounds entering the auditory system initially travel through the outer ear to the middle ear and then are passed on to the inner ear and the neural pathways of the peripheral and central auditory nervous systems (Figure 1–2).



**Figure 1-2.** Main structures of the peripheral auditory system and cross section of the cochlea. KEY: 1 = helix, 2 = anti-helix, 3 = scaphoid fossa, 4 = concha, 5 = lobe, 6 = triangular fossa, 7 = external auditory meatus, 8 = tympanic membrane, 9 = Eustachian tube, 10 = middle ear space (tympaanum), 11 = incus, 12 = malleus, 13 = stapes, 14 = superior semicircular canal, 15 = bony cochlea shell (otic capsule), 16 = round window, 17 = eighth nerve.

As part of this process of sound transmission within the auditory system, the sounds detected by the ear are converted from acoustic energy at the outer ear to vibratory (or mechanical) energy in the middle and inner ears to a neural representation of the acoustic signal at the cochlea and beyond. Anatomically, the outer ear consists of the pinna and the external auditory meatus (EAM), also known as the ear canal. The pinna protrudes from the side of the head and has a foundation of ligaments and cartilage that is covered by skin. This outer ear structure has a number of ridges and depressions and serves as collector of sound energy. Because of the unique configuration of ridges and depressions or grooves in this structure, it tends to respond maximally to some sounds within the high-frequency range (around 5000 Hz). The result of this preferential response is that acoustic signals in this frequency range are differentially enhanced or amplified before they are directed to the ear canal. The differential sound-collection properties of the pinna are believed to assist in sound localization (Yost, 2000).

The EAM accepts the sounds arriving at the ear and directs these acoustic signals to the tympanic membrane (TM). Because of its configuration, which is much like a pipe or tube that is closed at one end and open at the other, the EAM generates an ear canal resonance that is determined, at least in part, by the dimensions of the ear canal. In adults, this resonance (the enhancement of an acoustic signal) typically occurs at a frequency in the vicinity of 3000 to 4000 Hz. The presence of this ear canal resonance is important for the natural perception of sound. The loss or compromise of the normal ear canal response can result in the perception of speech and other acoustic signals as being “unnatural” or “tinny.” Such perceptions are often noted by patients who have their ears occluded by earmolds or hearing aids.

The middle ear is an air-filled space within the temporal bone, sometimes referred to as the tympanum. The TM (or eardrum) forms the lateral wall of this structure, while the medial wall is formed by the dense portion of the temporal bone that houses the inner ear. Two openings within the part of the temporal bone that forms the medial wall of the middle ear allow for communication between the middle ear and inner ear (i.e., the oval and round windows). The other borders of the middle ear space are formed by portions of the temporal bone. Important structures within the middle ear include the three smallest bones in the human body (i.e., malleus, incus, and stapes), which collectively form the ossicular chain, and the Eustachian tube, which provides fresh air to the middle ear space and equalizes middle ear pressure when necessary. The Eustachian tube (often referred to as the auditory tube) changes size, shape, and angle of orientation within the head with age. Other structures within the middle ear include the tendons from two middle ear muscles (i.e., tensor tympani and stapedius) and a branch of the facial nerve, which traverses the middle ear space. The two muscle tendons help support the ossicular chain within the middle ear space and contract in response to loud stimuli, which results in a stiffening of the ossicular chain referred to as the acoustic reflex. Additional support for the ossicular chain is provided by ligaments housed within the middle ear cavity.

The middle ear’s main function is to increase the energy that is imparted to the cochlea. This is accomplished by three mechanisms: an area differential between the TM and the footplate of the stapes, the lever action afforded by the configuration of the ossicular chain, and the buckling of the curved tympanic membrane as it responds to sound. Since the TM is considerably larger in area than the stapes’ footplate, energy from vibrations arriving at the eardrum is concentrated on the smaller stapes with an increase in applied force. Addi-

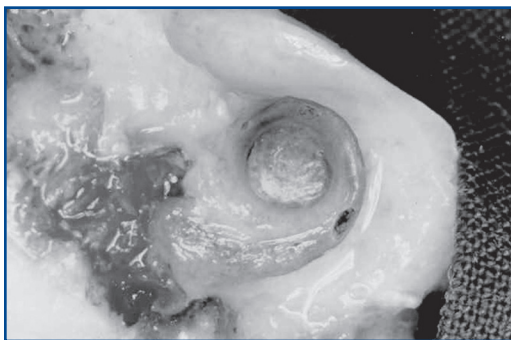
tional increases in force are realized because the ossicular chain works as a lever in conducting sounds to the cochlea, and the curvature of the TM results in greater movement of the TM with less displacement of the manubrium of the malleus. The overall increase in energy that results from these mechanisms is needed because an impedance mismatch exists between the outer ear (low impedance) and the cochlea (high impedance). This mismatch is created primarily by the transfer of energy from an air to a fluid medium (Yost, 2000).

## The Cochlea

### Anatomy

**Temporal Bone.** The cochlea as well as the middle and external ear, vestibular apparatus, and seventh and eighth cranial nerves are all housed in the temporal bone. The **temporal bone**, which is part of the skull base, is a hard bone that has many cavities, channels, and canals that subservise the organs of hearing and balance; hence, the term *labyrinth* is often applied to several of the structures housed within the temporal bone. The temporal bone has four main segments: the squamous (superior to the ear canal), the mastoid (posterior to the pinna), the petrous (deep in the cranium housing the inner ear), and the tympanic (ear canal). The petrous segment is where the middle ear apparatus, cochlea, vestibular structures, and internal auditory meatus (IAM) are located. The triangle-shaped petrous segment divides the cranial skull base into the posterior and middle fossa. The IAM houses the facial, auditory, and vestibular nerves and opens into the lateral aspect of the brainstem (see Anson & Donaldson, 1967).

**The Bony Cochlea.** The bony cochlea is embedded in the petrous portion of the temporal bone and is a totally enclosed structure resembling the shell of a snail (Figure 1–3). It has two openings covered by membranes. These openings include the oval window and round window, which interface with the middle ear cavity. The bony cochlea has about 2½ turns, with the basal turn (closest to the middle ear) being larger than the apical turn. A perforated bony central core called the **modiolus** runs through the cochlea. The **osseous spiral lamina** is a shelf-like structure that winds around the modiolus from base to apex (Figure 1–4). This lamina looks like a fir tree, with the cochlear base correlating

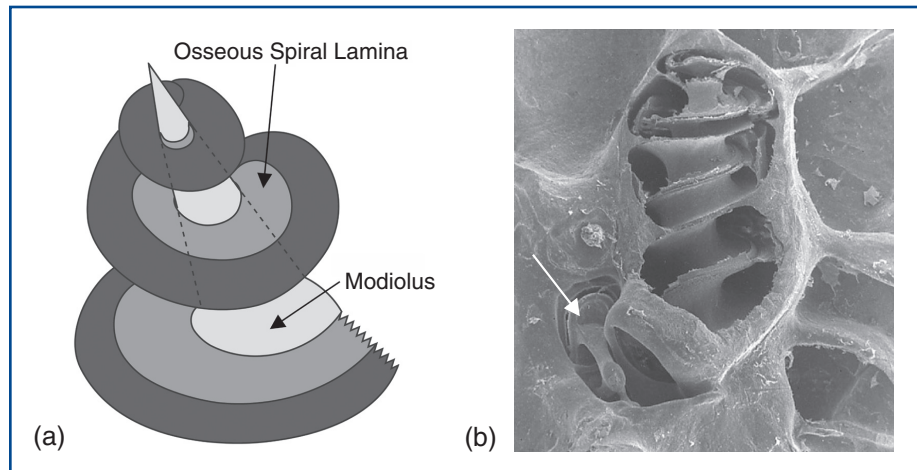


**Figure 1-3.** Photograph of the bony cochlea exposed in the temporal bone. The top layers of bone have been drilled away so the coils of the cochlea can be observed. (Courtesy of Marilyn Pinheiro.)



### CLINICAL CORRELATE

Temporal bone fractures are a major clinical entity for both otologists and audiologists. There are two types of temporal bone fractures: longitudinal and transverse. Longitudinal fractures occur along the long axis of the temporal bone, and transverse fractures run perpendicular to the long axis. Longitudinal fractures are often encountered in patients with head injuries and may be accompanied by hearing and balance deficits. Transverse fractures occur less frequently but can result in major hearing loss and vestibular symptoms (Huang & Lambert, 1997). More recent categorizations of temporal bone fractures are aimed at defining if the otic capsule is or is not involved in fractures of the temporal bone (see Brodie & Thompson, 1997).



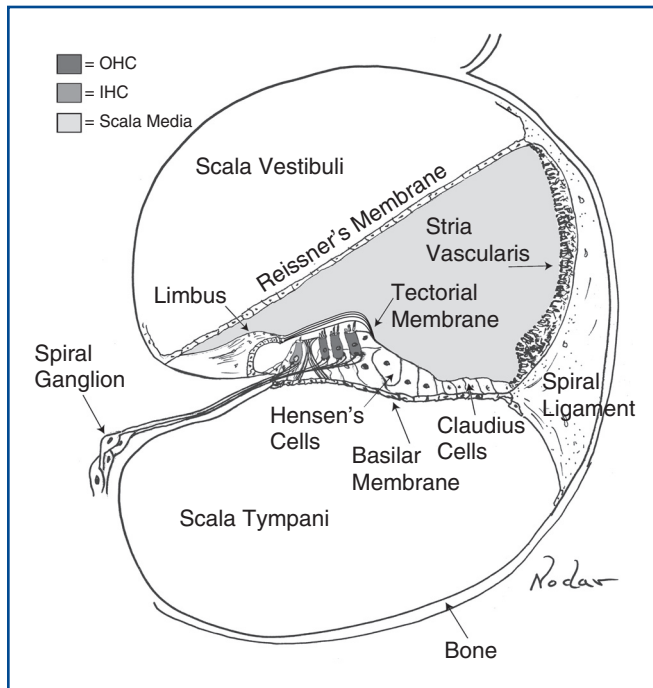
**Figure 1-4.** (a) The osseous spiral lamina. The bony shelf spirals around the modiolus (within the dotted lines) from the base to the apex of the structure. (b) An exposed (chinchilla) osseous spiral lamina from base (bottom) to apex (top) with the stapes footplate in the oval window (arrow). (Courtesy of Richard Mount, Hospital for Sick Children, Toronto.)

with the wider branches and the apex of the cochlea correlating with the narrower branches at the top. Small perforations in the lateral-most aspect of these shelf-type structures are referred to as the habenula perforata. These perforations allow the AN fibers (from the hair cells) to pass through the openings and form the modiolus and (eventually) the AN trunk. The spiral lamina shelf is wider at the base than at the apex and helps form the beginnings of the anatomical divisions of the cochlea by serving as an anchor for the medial aspect of the basilar membrane (Gulya, 1997).

**The Membranous Cochlea.** The osseous cochlea is lined with elastic, membranous structures that follow the shape of the bony cochlea. The membranous cochlea has three ducts: the scala vestibuli (superior), the scala media, also referred to as the cochlear duct (middle), and the scala tympani (inferior). The three divisions of the cochlea (ducts) are created by two important membranes. The **basilar membrane** (BM) separates the scala tympani from the inferior portion of the scala media, and **Reissner's membrane** divides the superior aspect of the scala media from the scala vestibuli. At the apex of the cochlea, the scala vestibuli and scala tympani communicate, and this point of communication is termed the **helicotrema**.

The membranous cochlea is a fluid-filled system. The scala tympani and scala vestibuli are filled with perilymph, and the scala media contains endolymph and cortilymph (see Yost, 2000, for review). Perilymph has essentially the same chemical composition as cerebral spinal fluid (CSF). It is low in potassium and high in sodium and has a 0 mV electrical charge. Endolymph is high in potassium and low in sodium and has a +80 mV electrical charge. **Cortilymph** (0 mV electrical charge) is similar in chemical composition to perilymph. Endolymph is most likely produced by the stria vascularis, which is a highly vascular structure located on the lateral wall of the cochlear duct next to the spiral ligament (Figure 1-5).

Playing an important role in the hydrodynamics of the cochlear fluid system are the vestibular (endolymphatic) and cochlear aqueducts. Within the



**Figure 1-5.** Cross section of the cochlea (the scala media, inner hair cells, and outer hair cells are shaded in light to dark shades, respectively).



## CLINICAL CORRELATE

The fluid dynamics in the membranous cochlear labyrinth are critical for appropriate function of the cochlea. If the fluid system doesn't function adequately, inner ear disorders can result, such as Ménière's disease (endolymphatic hydrops) and related disorders such as perilymph fistulas (see Schwaber, 1997, and Thai-van, Bounaix, & Fraysse, 2001). Ménière's disease symptoms include episodic vertigo, hearing loss, and tinnitus. The exact etiology of Ménière's disease is not known, but its basis is believed to be an overproduction of endolymph possibly related to dysfunction of the stria vascularis. There could also be excess endolymph in the scala media because the fluid is under-absorbed related to dysfunction of the endolymphatic sac and/or duct. In the pathological state, Ménière's disease is characterized by an expanded cochlear duct and sometimes even ruptures of Reissner's membrane, usually in the apical end of the cochlea. This is thought to be related to the low-frequency sensorineural hearing loss typically seen in Ménière's disease. A perilymphatic fistula is a tear in the oval or round window membrane through which perilymph leaks. This leakage causes a condition similar to Ménière's disease because there is unequal

pressure between the scala media and the other scalae. The administration of diuretics often alleviates the symptoms of Ménière's disease. These drugs increase osmotic action in the cochlea and may rid the cochlea of excess endolymph—at least temporarily (see Chapter 4 for more on Ménière's disease and cochlear fistulas).

The hearing loss, tinnitus, and vertigo associated with Ménière's disease fluctuate with the episodic nature of the disease. Early on, symptoms completely clear after a so-called Ménière's attack. This is best demonstrated by changes in the pure-tone audiogram, which after an episode returns from a low-frequency sensorineural loss back to normal hearing sensitivity, at least in the early stages of this disease. Long-term Ménière's disease often results in permanent hearing loss, although fluctuations in hearing often continue to occur. A type of auditory evoked potential procedure, electrocochleography (Ecog), is often used in the diagnosis of Ménière's disease (see Chapters 5 and 6). Ecog recordings often show a large summing potential (SP) relative to the AN's action potential (AP); this is known as an abnormal SP/AP ratio (Schwaber, 1997).

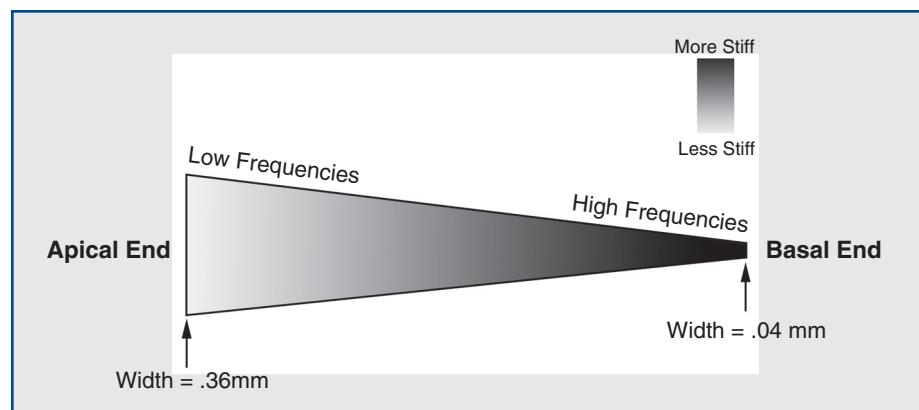


**vestibular aqueduct** are the **endolymphatic sac** and the **endolymphatic duct**. The vestibular aqueduct connects the posterior aspect of the vestibule to the posterior side of the petrous bone. This channel is thought to be a type of overflow valve for excess endolymph. The **cochlear aqueduct** courses from the basal turn of the cochlea to the posterior aspect of the temporal bone. It permits the transfer of CSF to the *scala tympani* and *vestibuli* (Gulya, 1997).

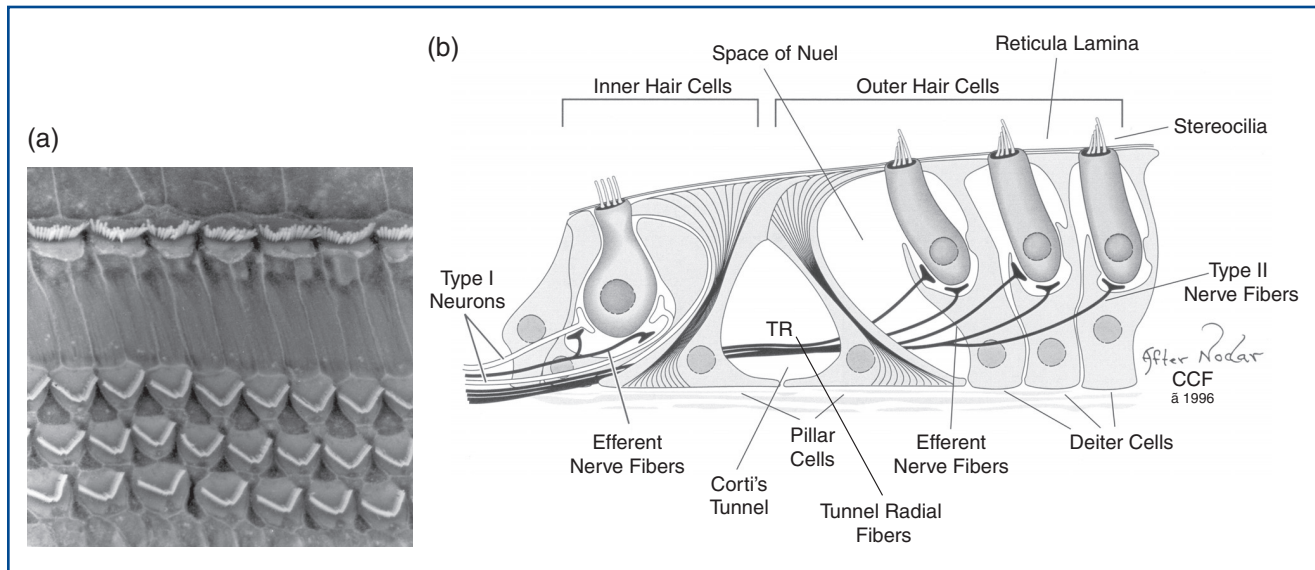
The BM runs the length of the cochlea (25 to 35 mm) and is wider at the apex than at the base. It is composed of fibers that are stiffer at the base than at the apex (Figures 1-5 and 1-6). This allows better high-frequency tuning at the base and better low-frequency tuning at the apex. Reissner's membrane is a thin, elastic membrane that is responsible for keeping separate the perilymph of the *scala vestibuli* from the endolymph in the *scala media* (see Figure 1-5). This membrane and the BM define the upper and lower boundaries of the *scala media* (see Gelfand, 1998).

**The Organ of Corti.** Resting on the BM is the **organ of Corti**, which is the end organ of hearing. The organ of Corti is composed of sensory cells, supporting cells, and a variety of membranes. It runs the entire length of the cochlear duct. The top of the organ of Corti is defined by the **tectorial membrane**, which on its underside touches the taller cilia of the **outer hair cells (OHCs)**. The cilia of the **inner hair cells (IHCs)** do not touch the tectorial membrane. At the base of the stereocilia is the **reticular lamina**, which forms a tight juncture around the cilia. The reticular lamina prevents the endolymph from entering the area of the hair cells and is formed by the flattened top surfaces of supporting cells (**Deiters' cells**) and the **cuticular plate** at the top of the hair cells.

The sensory cells in the organ of Corti include the IHCs and the OHCs that are arranged on either side of the pillar cells, which define the tunnel of Corti (Figure 1-7). In the human, there is a single row of about 3,500 IHCs that runs the length of the cochlea and multiple rows (three to five) of about 12,000 OHCs that also run the length of the cochlea (Figure 1-7b). The IHCs are flask shaped and robust in their appearance. The OHCs are cylinder shaped and are thinner and longer than the IHCs. The electrical charge in the hair cells is  $-40$  to  $-70$  mV. At the top of the hair cells are stereocilia (also called cilia), and at



**Figure 1-6.** A drawing looking down on the basilar membrane from above showing changes in the width of the membrane from the apical to basal end and corresponding frequency representation along the length of the basilar membrane.

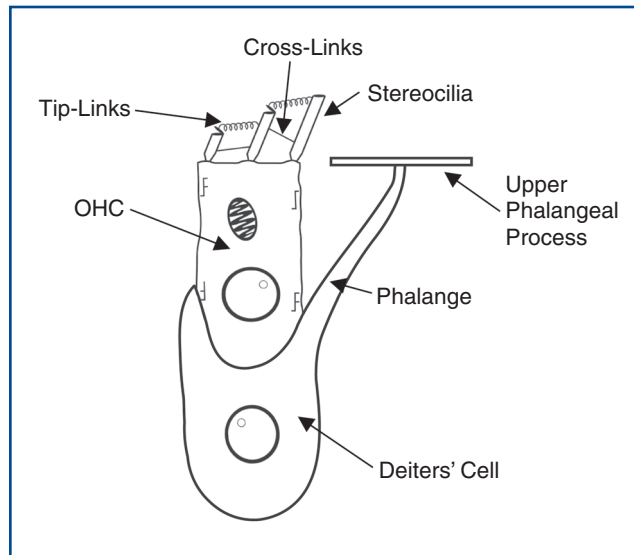


**Figure 1-7.** (a) Photograph looking down on the top of inner (one row) and outer (three rows) hair cells and the reticular lamina (area in between cells) after removal of the tectorial membrane. The region between the row of inner hair cells and first row of outer hair cells is the pillar cell region. Note that the cilia of the outer hair cells form a “W,” while the inner hair cells look like a flattened “U.” (Courtesy of Richard Mount, Hospital for Sick Children, Toronto.) (b) Hair cells and related structures. (Modified from Sahley et al., 1997 and the Cleveland Clinic Foundation, with permission.)

the bottom are AN fiber connections (often called terminal buttons). The cilia on the hair cells are graded in length. The OHCs’ cilia form a “W” shape, while the IHCs’ cilia form a flattened “U” shape (Figure 1–7a). Located on the cilia, probably on the top, are pores that open when the cilia are bent toward the lateral wall of the cochlea duct. **Tip-links** are small filaments at the tops of the cilia that connect one cilium to another cilium and help with the opening and closing of the pores (which allows potassium [K<sup>+</sup>] ions to flow into the cell and start the transduction process). **Cross-links** are similar to tip-links; however, they are located at the sides of the cilia (Figure 1–8). The cross-links help the cilia move in concert when the cilia are stimulated. Hair cells are present along the entire length of the cochlea. The hair cells at the basal end respond best to high frequencies, and those at the apical end respond to low frequencies (see Raphael & Altschuler, 2003).

The organ of Corti is highly dependent on not only hair cells, which are sensory transducers, but also supporting cells. The **phalangeal cells** support the IHCs, and Deiters’ cells support the OHCs. The hair cells rest on the bases of the supporting cells, with the stalks of these supporting cells angularly projecting upward across the cells to form the reticular lamina. Pillar supporting cells are at angles facing each other to form a triangular structure known as the **tunnel of Corti**. More supporting cells (Hensen and Claudian) are seen as one views the more lateral aspect of the organ of Corti as it progresses to the lateral wall (see Geisler, 1998, and Slepecky, 1996).

The lateral wall of the cochlea marks the end of the organ of Corti and it encompasses two key structures. Progressing laterally, one first encounters the stria vascularis and then the spiral ligament, which rests against the osseous cochlear wall.



**Figure 1-8.** An outer hair cell (OHC) and its supporting structure.



## CLINICAL CORRELATES

High-intensity noise exposure typically results in damage, primarily to the OHCs. If sound is of high intensity and/or there is a sufficiently long duration of exposure, then the IHCs and supporting cells can also be damaged. New data implicate synaptic ribbons as especially fragile structures for potential damage, which if compromised can disallow proper auditory nerve function (Kujawa & Liberman, 2009). It appears that temporary threshold shift can occur subsequent to noise exposure without permanent threshold shift resulting, yet permanent damage to the synaptic ribbons (**synaptopathy**) can occur, which in turn affects AN function (Kujawa & Liberman, 2009). The basal portion of the cochlea is most susceptible to damage from high-intensity sound. Most people with noise exposure typically incur the greatest amount of hearing loss at 4 kHz, with the adjacent frequencies of 3 kHz and 6 kHz also often affected. Susceptibility to hearing loss from high-intensity sound is highly variable, with some people being highly vulnerable while others are affected minimally.

Ototoxic drugs can also cause damage to hair cells, with the OHCs being the most susceptible. Audi-

ologically, the high frequencies are affected first, with subsequent progression to the mid and low frequencies. Because of this trend for high-frequency compromise to occur prior to mid- and low-frequency compromise, two specialized audiometric procedures (high-frequency audiometry and otoacoustic emissions) have been used to define early ototoxic effects on hearing. Probably the two best known categories of ototoxic drugs are the aminoglycosides (the mycins; for example, gentamycin) and the salicylates (aspirin). While most ototoxic drugs result in permanent hearing loss, the hearing loss associated with salicylates is often reversible. Ototoxic drugs can also damage the vestibular system and, in at least some cases, the balance system is affected before the auditory system.

Autoimmune disorders, head trauma, viral and bacterial infections, and vascular disorders are other maladies that can damage the hair cells. In most instances, the OHCs at the basal end of the cochlea are more susceptible to damage than the OHCs at the mid and apical ends. Therefore, in most of these disorders, high-frequency hearing loss is more common than low- or mid-frequency hearing loss.

### Cochlear Physiology

How does the cochlea work to help provide the hearing experience? From a general perspective, the cochlea performs some key functions. It changes

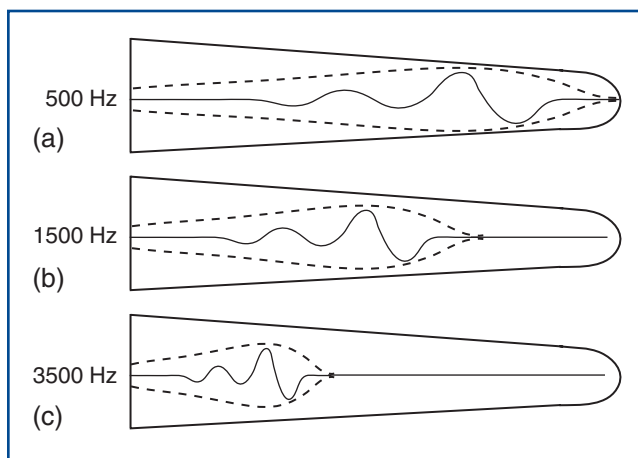


vibratory energy (sound) into electrical impulses so the brain can utilize these signals. The cochlea also provides fundamental coding of the intensity, frequency, and temporal aspects of sound.

**Cochlear Mechanics.** Cochlear physiology begins with the mechanical input from the stapes via the oval window, which is accommodated by expansion of the round window membrane. When a compression sound wave is introduced, the stapes is pushed in and the oval window bulges; the opposite happens when the acoustic input is a rarefaction wave.

The vibratory input from the stapes to the cochlea sets up a traveling wave (TW) in the cochlear fluids that moves down the length of the BM. This TW moves faster at the basal end (nearly 100 meters/second) and slower at the apical end (3 meters/second) (Zwislocki, 2002, interpolated from Tonndorf, 1960). This velocity difference is related to the physical characteristics of the BM (short stiff fibers at the basal end equal fast velocity; longer, looser fibers apically equal slow velocity). A compression wave results in an initial downward movement of the BM, while a rarefaction wave results in an initial upward movement of the BM. The BM movement is influenced by where it is attached. The inner part of the BM is attached to the osseous spiral lamina, which serves as a fulcrum for the BM and the organ of Corti. The lateral edge of the osseous spiral lamina is almost directly below the inner pillar, which provides support for BM movement. The outer part of the lateral aspect of the BM is attached to the spiral ligament. The lateral aspect of the BM, as one would expect, is more elastic than the inner aspect; hence, it moves more easily (Raphael & Altschuler, 2003).

**Frequency and Intensity.** Frequency representation at the cochlea is accomplished by the place principle and/or temporally (periodicity) by the rate of neuron firing. Different frequencies produce TWs that reach their maximum deflection at different places along the cochlear partition (Figure 1-9). Simply stated, the point of maximum deflection on the BM is the result of the resonance characteristic of the BM that matches the frequency of the stimulating sound. The BM is tuned from high (basal end) to low (apical end) frequencies. Hence, the point of maximum deflection along the cochlear duct relates the



**Figure 1-9.** The traveling waves and their envelopes (*dotted lines*) for (a) low (500 Hz), (b) mid (1500 Hz), and (c) high (3500 Hz) frequency tones.



## CLINICAL CORRELATE

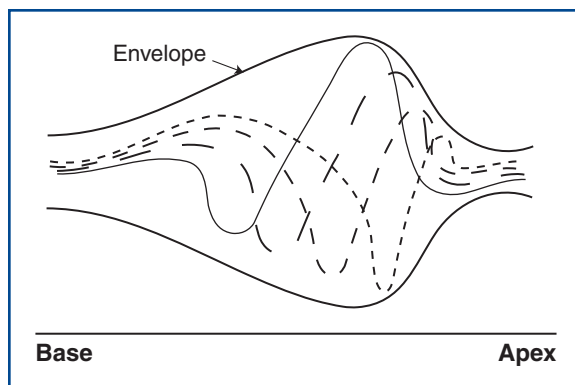
At high intensities there is less frequency selectivity—especially if there is outer hair cell damage. Amplification increases input to the cochlea and thus creates greater broadness of the TW envelope. This broadness creates greater intensity representation at lower sensation levels in individuals with hearing impairment compared with individuals with normal hearing. In addition, frequency discrimination is compromised. For these reasons, it becomes important when fitting hearing aids to determine the lowest level of amplification that results in sufficient audibility. This is because the lower the intensity level, the better the chance for proper intensity and frequency representation. This physiologic concept relates to essentially all audiologic suprathreshold procedures.

frequency of the sound (the higher the frequency, the more basal the location of maximum deflection; the lower the frequency, the more apical the point of maximum deflection). A long-standing problem that surrounded this view was that the frequency tuning of the BM was too broad to explain the psychoacoustic measurement of frequency discrimination (i.e., behaviorally, frequency discrimination was much sharper than could be predicted by the frequency tuning of the BM alone). Hence, the temporal theory of frequency analysis was entertained.

At low and mid frequencies the BM vibrates in a whip-like motion at the rate of the frequency of the stimulating sound (e.g., if the sound has a frequency of 700 Hz, the BM vibrates 700 times per second and stimulates the AN at this rate) (Figures 1–9 and 1–10). This BM vibration happens within the envelope of the TW (often termed the fine movement or structure of the envelope). This may be how frequency is temporally coded at low to mid frequencies either in addition to or perhaps in place of the place principle. At higher frequencies the temporal theory runs into problems, and it becomes more difficult to support this concept (for more discussion of this topic, see Chapter 6). Many agree that both place and temporal theories contribute to frequency coding (see von Békésy, 1970, and Hudspeth, 2000a, 2000b).

Intensity representation in the cochlea is related to the amplitude of the TW envelope. That is, the more intense the stimulus, the greater the magnitude of the maximum deflection of the BM. Because of its physical characteristics, the BM has greater deflections at its apical end than at its basal end (for the same intensity stimulus). As the stimulus intensity increases, the TW envelope becomes not only greater in amplitude but also broader in shape. The broadness of the TW envelope at high intensities decreases the frequency selectivity of the BM. Hence, BM frequency tuning is sharper for low-intensity than for high-intensity stimuli. The greater the amplitude and broadness of the BM deflection, the greater are the number of hair cells that are stimulated. This in turn creates a greater neural response that results in greater loudness perception.

A critical concept in intensity representation in the cochlea is its nonlinearity. Basilar membrane deflection is certainly greater for high-intensity stimuli

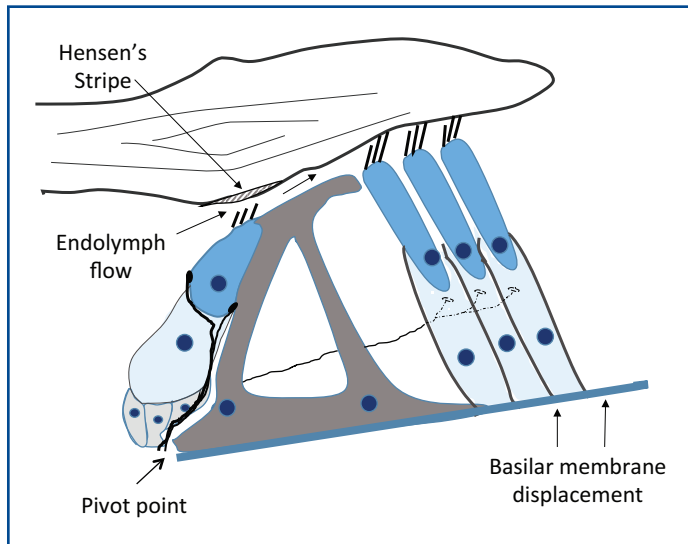


**Figure 1-10.** Close-up sketch of the whip-like fine motion of the basilar membrane and the envelope of its displacement. The peak of maximum displacement is the place on the basilar membrane that corresponds to the frequency of the acoustic stimulus.

than for low-intensity stimuli, but there is progressively less change in the amplitude of the BM deflection as the intensity of a sound increases. Stated another way, there is compression in the cochlea for intensity representation at high levels (Ruggero, 1992).

**Hair Cell Mechanics.** Hair cells change vibratory energy to electrical energy. A key aspect of this transduction process is the mechanics of the hair cells, which begins at the level of the cilia. The OHCs' cilia articulate with the tectorial membrane in an interesting manner. The TW of an acoustic compression wave depresses the BM downward. When this happens, the organ of Corti also moves downward, and this results in a shearing motion of the tectorial membrane that pushes the cilia toward the limbus, which causes the tip-links to close the pores in the cilia. This in turn causes a hyperpolarization of the hair cells (inhibition). When the BM moves upward, the cilia are pushed in the opposite direction (away from the limbus), causing the tip-links to open the pores in the cilia and  $K^+$  to enter the cell and the cells to fire (depolarization) (Figure 1-11).

The IHC cilia do not touch the tectorial membrane; hence, there must be another means of moving these cilia so that these cells can fire. The leading hypothesis explaining how this happens is that fluid flow between the reticular lamina and the tectorial membrane caused by OHC cilia movement results in the IHC cilia rotating in the same direction as the cilia from the OHCs (Geisler, 1998).



**Figure 1-11.** Sketch showing the deflection of the outer hair cells by the tectorial membrane for movement upward of the basilar membrane. Upward movement causes a deflection of the stereocilia in the direction shown and results in depolarization of the hair cells, while downward movement (not shown) results in deflection of the stereocilia in the opposite direction and results in hyperpolarization of the hair cells. Note the pivot point of the basilar membrane near the (inner) pillar cells. Although the inner hair cells' cilia do not touch the tectorial membrane, it is theorized that the endolymph flow (shown by arrows) and its related forces deflect the inner hair cells' cilia.

Both IHCs and OHCs are responsible for the transduction process; however, the OHC's responsibility is primarily one of expansion and contraction. That is, it has been shown that when the OHCs are stimulated, contractile proteins in the cells allow them to expand and contract. This motility works in such a manner that when the BM is moved upward the OHCs contract and when the BM is pushed downward the OHCs expand. Since these cells are firmly connected to the tectorial membrane superiorly and to the Deiters' cells inferiorly (which are connected to the BM), contraction lifts the BM, creating a greater displacement of this structure. When the BM is pushed downward, the cells expand and again cause greater displacement of the BM. This hair cell motility works like an amplifier by increasing BM movement and causing greater stimulation—hence, the term *cochlear amplifier*. The OHC motility that operates at the peak of BM displacement also sharpens the membrane's tuning by reducing the broadness of the membrane's peak. This cochlear amplifier action takes place only for low intensities and is lost for high-intensity stimuli. This is one of the reasons high intensities result in poorer frequency selectivity (see Geisler, 1998).

The expansion and contraction of the OHCs is believed to be the basis for the generation of the small acoustic signals that are referred to as otoacoustic emissions (OAEs). OAEs are not typically present when there is hearing loss or when there is damage to the OHCs. The intensity range over which OAEs are operational is similar to the operating intensity range of the cochlear amplifier. Damage to the IHCs, AN, or central auditory nervous system (CANS) generally do not affect OAEs. Salicylate ototoxicity, which appears to compromise OHC function, arrests OAE activity as well as creates reversible sensorineural hearing loss. By stopping salicylate intake, hearing sensitivity will typically return, as will the patient's OAEs. These research findings, as well as other similar data, have strongly associated OHC motility with the generation of OAEs (Brownell, Bader, Bertrand, & de Ribaupierre, 1985; Geisler, 1998).

### **Cochlear Electrophysiology**

Cochlear mechanics begins the transduction process. The BM, moving in an upward direction (rarefaction sound wave), results in a shearing action at the TM, which pushes the cilia away from the limbus, and the hair cell depolarizes. When the BM moves downward (compression sound wave) the hair cell is in hyperpolarization and doesn't fire. Associated with the hair cell depolarization are three cochlear electrical potentials: the endocochlear or resting potential, the cochlear microphonic (CM), and the summing potential (SP). The action potential (AP) is generated by the AN and follows the SP in time.

The endocochlear potential is observed by passing an electrode through the different fluid compartments of the cochlea and noting how voltages increase to +80 mV when it enters the scala media. This positive voltage creates a large differential with the -40 to -70 mV charge in the hair cells. The endocochlear potential is believed to be generated and maintained by the stria vascularis and is responsible for moving positively charged K<sup>+</sup> ions through the channels of the hair cell stereocilia (Wangemann, 2002a).

The CM is generated mostly by the OHCs, with a minor contribution from the IHCs, and mirrors the receptor currents as they flow through the hair cells; that is, the CM mimics the incoming stimulus. As the intensity of the sound increases, the CM increases in amplitude up to relatively high levels, where it saturates and then decreases in its output. Reversing the polarity of the sound signal reverses the polarity of the CM (Dallos, 1973). Therefore, an alternating



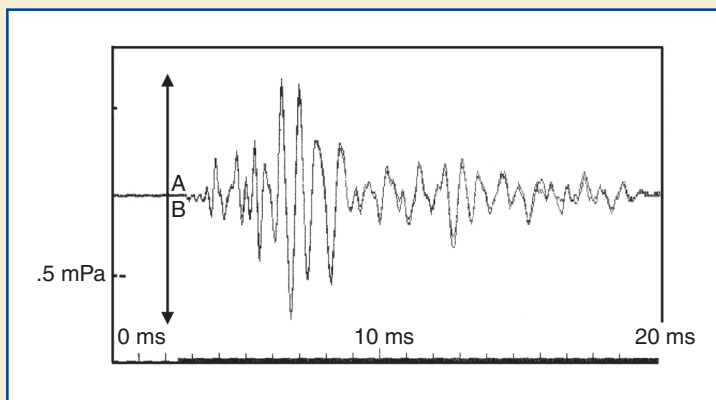
## CLINICAL CORRELATE

Otoacoustic emissions, first discovered by Kemp (1978), have become a highly used clinical tool. Three main types of OAEs have emerged. These include transient (or click) evoked OAEs (TEOAEs, CEOAEs), distortion product OAEs (DPOAEs), and spontaneous OAEs (SOAEs).

Briefly, TEOAEs result from click stimuli presented to the ear at mid to moderately high intensities. A microphone placed in the ear canal picks up the sound oscillations generated by the cochlea (OHCs), which are conducted back through the middle ear apparatus into the ear canal. These sound emissions (oscillations) are subaudible and represent most of the frequencies in the range of hearing tested clinically. The subaudible sounds in the ear canal are represented by oscillations similar to sound waves and represent a wide frequency spectrum. The oscillations, like audible sound waves, contain amplitude and frequency information (Figure 1-12). The high frequencies have a shorter latency and appear first in the recording that is made, and these are then followed by mid and low frequencies. The high frequencies are depicted by tight (narrow) oscillations and the low frequencies by broad oscillations. The amplitudes of these oscillations can be correlated to norms that indicate normal or near normal hearing. TEOAEs are seldom present if hearing loss is greater than 30 decibels hearing level (dB HL). The TEOAEs can be quite frequency specific in that if hearing loss is present at a given frequency, then TEOAEs are absent at (or near) that frequency.

DPOAEs evolve from the presentation of two tones at slightly different frequencies. The frequency ratio of the two tones should hover around 1.2. These two tones result in a distortion production in the cochlea that is manifested by a lower intensity and lower frequency third tone in the cochlea that can also be measured in the ear canal. The frequency of the third tone follows the formula  $2f_1 - f_2$ , with  $f_1$  and  $f_2$  being the frequencies of the stimulating tones. DPOAEs are generated by tone pairs presented at discrete frequencies across the frequency range being assessed. Hence, this method of deriving OAEs is highly frequency specific. DPOAEs are recorded similarly to TEOAEs. They have been recorded in individuals who have 40 to 45 dB HL hearing loss; hence, their intensity range for eliciting responses is greater than for TEOAEs. DPOAEs are frequency specific and can mimic the contour of the audiogram.

SOAEs are emissions that are present without acoustic stimulation. They are essentially only present in people with normal hearing, but not all people who have normal hearing have SOAEs. Most of the available research indicates that 90% or more of people with normal hearing have normal TEOAEs or DPOAEs; this is far more than people with normal hearing who have SOAEs. Hence, most clinical applications involve TEOAEs or DPOAEs. Presently, OAEs are primarily used for screening newborns for hearing loss, but they can also be used for detection of pseudohypacusis and assistance in the differential diagnosis of neural versus sensory involvement (see Robinette & Glattke, 2002).



**Figure 1-12.** An otoacoustic emission from a click stimulus. The arrow at the left indicates the amplitude of the oscillations, and the latency is noted along the bottom. The higher frequencies of the waveform are at the left. The oscillations here are closer together and are of a shorter latency. The low-frequency responses are on the right. Note that the oscillations are wider apart.

polarity signal, when averaged, will cancel the CM (Figure 1-13). The CM is almost instantaneous in its response to the stimulus and it is essentially unaffected by the rate of presentation (Dallos, 1973).

The SP is an extracellular direct current (DC) response that follows the envelope of the stimulus. It is most likely generated by the IHCs but with some