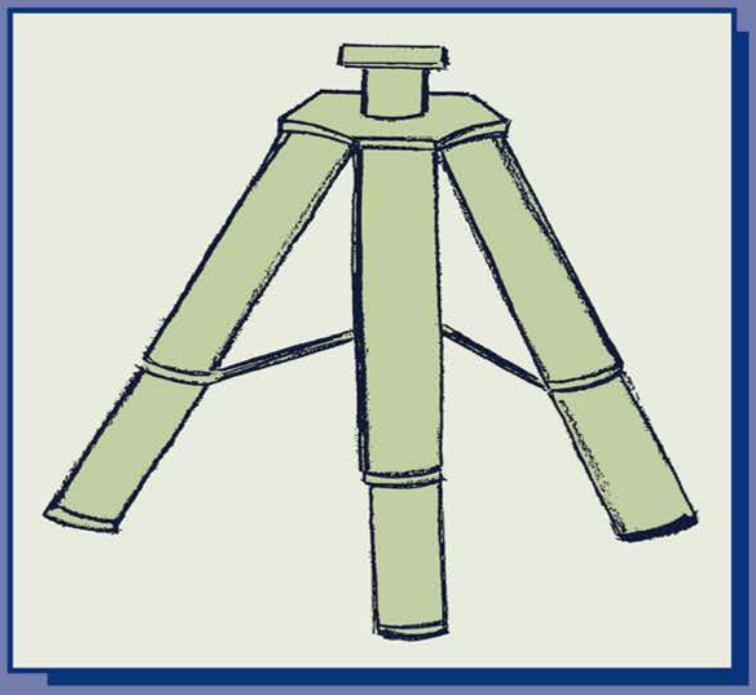
An Introduction to Auditory Processing Disorders in Children



Edited by

Teralandur K. Parthasarathy

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Psychology Press
Taylor & Francis Group
NEW YORK AND HOVE

First published 2006 by Lawrence Erlbaum Associates, Inc., Publishers
10 Industrial Avenue
Mahwah,
New Jersey 07430

Psychology Press is an imprint of the Taylor & Francis Group, an informa business

This edition published 2013 by Psychology Press

Psychology Press
Taylor & Francis Group
711 Third Avenue
New York, NY 10017

Psychology Press
Taylor & Francis Group
27 Church Road,
Hove,
East Sussex, BN3 2FA

Library of Congress Cataloging-in-Publication Data

An introduction to auditory processing disorders in children / edited by Teralandur K. Parthasarathy.
p. ; cm.
Includes bibliographical references and index.
ISBN 0-8058-5392-8 (cloth : alk. paper)—ISBN 0-8058-5393-6 (pbk. : alk. paper)
1. Word deafness in children. I. Parthasarathy, Teralandur K.
[DNLM: 1. Auditory Perceptual Disorders—diagnosis—Child. 2. Auditory Perceptual Disorder—physiopathology—Child. 3. Auditory Perceptual Disorders—therapy—Child. 4. Attention Deficit Disorder with Hyperactivity—Child. 5. Needs Assessment—Child. WV 272 I62 2006]
RF291.5.C45I58 2006
618.92′855—dc22 2005018304

To my mother and my brother Dr. T. K. Raghunath
for their unrelenting help and support

and

To my wife Dr. Gita Malur, my daugther Shilpa, and my son Shrikanth
for their love, understanding, and support

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Contents

Preface

Auditory processing disorder (often called central auditory processing disorder) is not a new entity. Over the past 10 years, numerous advances have been made in the area of assessment and management of auditory processing disorders (APD). Advances in brain imaging technology and electrophysiogic techniques have helped us significantly in our understanding of the brain mechanisms that process auditory information.

In recent years, APD has become an important clinical entity within the field of communication disorders. More and more children are being diagnosed with APD or, in some cases, are described with behavioral manifestations that are compatible with the symptoms of APD.

The purpose of this book is to assist audiologists, speech-language pathologists, and other related professionals in gaining knowledge and understanding in the three major areas related to APD: basic science, assessment, and management.

Many specialists have a keen interest in various aspects of APD. However, there is a tendency for these groups to communicate primarily within their own defined disciplines. Thus, the significant contributions of each group are often not shared with the others. This potentially reduces both our understanding and effectiveness in dealing with APD. It is the purpose of this book to bring together the knowledge base of different professional groups that all too often do not work together and to improve the prospects for a better quality of life for children with a diagnosis of APD. The strength of this book lies in the skill and breadth of knowledge of its contributing authors. They are not only highly regarded clinicians and researchers, but they are also dedicated professionals who have tried to explain, clarify, and demystify APD.

This book will not be the final word on APD. It is, however, a highly positive step in the direction of collating the most recent evidence from all the relevant fields.

With time, assessment and management of APD have taken on a multidisciplinary approach. Taken together, the chapters in this book are an extraordinary compendium of the status of APD in children today. It is the hope of all contributing authors that this information will serve students in audiology and speech-language pathology, practicing audiologists, speech-language pathologists, and members of related professions, notably psychologists, special educators, and physicians.

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Neuroanatomy and Neurophysiology of the Central Auditory Pathways

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HEARING SENSITIVITY

Normal hearing is essential to the acquisition of oral language and effective verbal communication. Any impairment of the auditory system, either congenitally or adventitiously acquired, that affects the transmission and/or perception of sound is likely to have a profound effect on one's ability to hear and comprehend spoken language. The human ear is sensitive to acoustic events within the frequency range of 20 to 18,000 Hertz (Hz). However, the human ear is not equally sensitive to all frequencies within this range. The ear is most sensitive to sound frequencies between 500 and 4,000 Hz—the frequency range most important for speech reception. In terms of intensity, the human auditory system is sensitive to a range of 0 to 140 dB Sound Pressure Level (SPL). Normal conversational speech falls within the range of approximately 50 to 77 dB SPL. Repeated and prolonged exposure to intensity levels above 85 dB SPL can cause permanent structural damage to the inner ear. Sound pressure levels above 140 dB SPL can cause pain sensation and instantaneous structural damage to the hearing mechanism.

OVERVIEW OF AUDITION

The process of audition (hearing) begins when sound waves enter the external auditory meatus and impinge on the tympanic membrane. The subsequent movement of the tympanic membrane serves to convert sound energy into mechanical energy. The motion (vibration) of the tympanic membrane causes the middle ear bones—malleus, incus, and stapes—to be set into motion. This mechanical energy is transmitted to the cochlear fluids of the inner ear via stapes movement in and out of the oval window of the inner ear, thus converting mechanical energy into hydraulic energy. The resulting movement of the fluids causes patterned membrane movement in the cochlea.

Movement of the membranes in the cochlea stimulate the cilia of the hair cells, which in turn causes depolarization of the hair cells. Depolarization activates chemical channels, triggering the release of neurotransmitters, across the synapse between the hair cells and auditory nerve fibers. The neurotransmitter depolarizes the terminals of the auditory nerve fiber and a nerve action potential is generated. The action potential discharges are transmitted by the fibers of the auditory nerve to the cochlear nuclei in the brain stem, which project these nerve impulses to multiple synaptic points in the brain stem as well as the thalamus. The combined signals from both ears are analyzed in the brain stem by their intensity and frequency to localize sound. Auditory impulses finally travel to the primary auditory cortex located on the superior surface of the temporal lobe in the gyri of Heschl, which are involved with sound perception. The auditory impulses further travel to Wernicke's (associational language) area, where the auditory signals are analyzed and interpreted into language-specific meaningful messages and the comprehension of spoken language occurs.

This chapter provides a functional description of the neural circuitry and physiology of hearing from the inner ear to the primary auditory cortex. Each intersecting level of the brain stem is listed and functionally discussed in terms of its significance to the transmission of auditory information.

INNER EAR MECHANISM

The inner ear contains the sensory organs of balance and hearing. The sensory organ for balance is called the vestibular system and includes the utricle, saccule, and three semicircular canals. The sensory organ for hearing is called the acoustic or auditory system. The auditory portion of the inner ear is a snail-shaped structure called the cochlear. Both the vestibular and auditory systems are encased in the same bony capsule, contain the same fluid systems, and share the same cranial nerve—Cranial nerve VIII (Fig. 1.1).

There are two labyrinths in the inner ear: the osseous (bony) labyrinth and the membranous labyrinth. The osseous labyrinth is a bony capsule covering a series of irregular cavities in the petrous portion of the temporal bone. Inside the osseous labyrinth is the membranous labyrinth, which includes the cochlear duct (scala media) and the vestibular apparatus of saccule, utricle, and three semicircular canals. The inner ear fluid perilymph protects the membranous labyrinth from the bony labyrinth. Endolymph is a closed fluid system within the membranous labyrinth of the cochlear and vestibular portions of the inner ear.

COCHLEAR ANATOMY

The cochlea resembles a snail-shaped structure winding two and a half times around a bony, central core called the modiolus. A cross-section of the cochlea is depicted in Fig. 1.2.

With a length of approximately 35 mm, the cochlea is comprised of three fluid-filled chambers or canals: scala vestibuli, scala media, and scala tympani. Scala vestibuli is the uppermost chamber and follows the inner contour of the cochlea. Scala tympani lies at the bottom and follows the outer contour of the cochlea. Scala vestibuli communicates with scala tympani through a small aperture at the apex of the cochlea called the helicotrema.

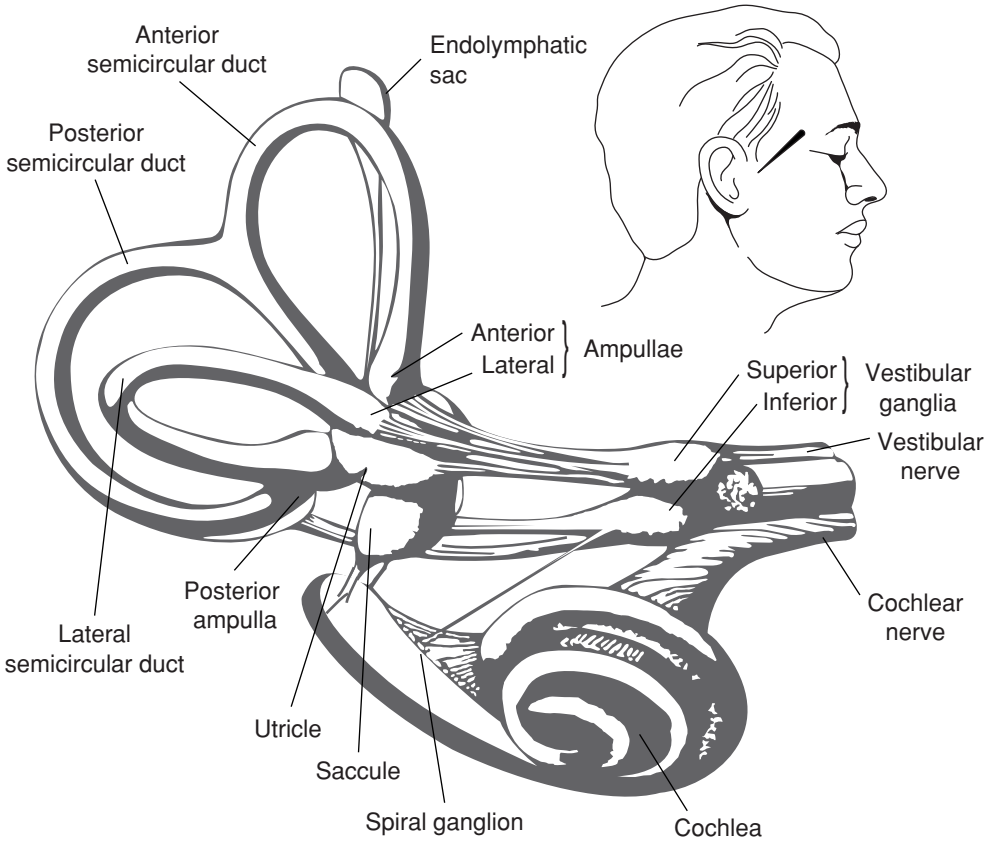

FIG. 1.1. Inner ear labyrinth composed of the semicircular ducts, vestibule, and cochlea along with vestibular and cochlear nerves. From *Neuroscience for the Study of Communicative Disorders,* 2nd. ed. by Subhash C. Bhatnagar, 2001. Copyright 2001 by Lippincott, Williams and Wilkins.

Reisner's membrane, or vestibular ligament, separates scala vestibuli from scala media. The basilar membrane separates scala media from scala tympani. The end organ of hearing—the organ of Corti—is supported by the basilar membrane. Both membranes extend from the osseous (bony) spiral lamina, a bony shell that extends from the modiolus, and attach to the spiral ligament located on the wall of the bony labyrinth.

Scala vestibuli and scala tympani are filled with perilymph, which is secreted by the periosteal lining of the scalae. Scala media is filled with endolymph, which is secreted by stria vascularis, a highly vascularized band of cells on the internal surface of the spiral ligament within scala media. Both of these fluids are marked by different concentrations of sodium and potassium ions. The higher sodium (Na+) relative to the lower potassium (K+) concentration of perilymph makes it similar to cerebrospinal fluid (CSF) or extracellular fluid. The ionic composition of endolymph, which has a higher concentration of K+ relative to Na+, is similar to the intracellular fluid (Haines, 2002).

The organ of Corti, which is located in the scala media on the basilar membrane, contains the sensory cells of hearing—the hair cells. The hair cells are equipped with specialized stereocilia on their tips. There are two types of hair cells: inner and outer (Fig. 1.2). In humans, these sensory cells run in parallel rows from the base of the

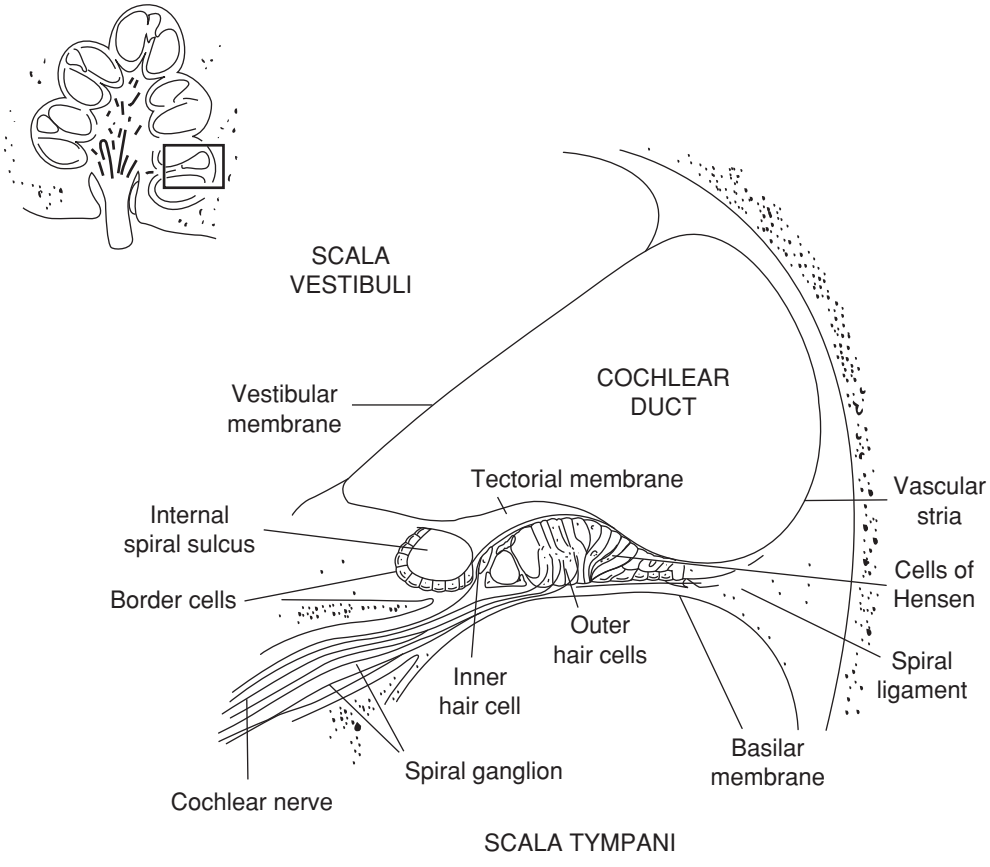

FIG. 1.2. A diagrammatic illustration of the cochlear duct on a radial section of the cochlea. From *Neuroscience for the Study of Communicative Disorders,* 2nd. ed. by Subhash C. Bhatnagar, 2001. Copyright 2001 by Lippincott, Williams and Wilkins.

cochlea to the apex of the cochlea. Inner hair cells (IHC) run in a single row, outer hair cells (OHC) in rows of three or four. The arch of Corti separates the row of IHC from the rows of OHC. There are approximately 12,000 OHCs and 3,000 IHCs. Fifty to 70 stereocilia project from each IHC, forming a "V" pattern. Forty to 150 stereocilia project from each OHC, forming a "W" pattern. The cilia of the OHCs fit into indentations on the underside of the overlying gelatinous tectorial membrane. The cilia of the IHC do not directly insert into the tectorial membrane.

IHC and OHC differ in terms of their innervations: IHC are predominantly innervated by ascending auditory nerve fibers, whereas the OHC are predominantly innervated by projections from the descending auditory pathways including the olivocochlear bundle (OCB).

COCHLEAR FUNCTION

The cochlea converts the mechanical energy received from the middle ear ossicles into hydraulic mechanical energy and initiates neural activity. As the stapes moves into the oval window, it displaces perilymph toward scala tympani through the helicotrema.

Due to the incompressibility of fluids of the cochlea because of the surrounding bony labyrinth, the resulting fluid pressure is relieved by the outward movement of the round window in the scala tympani. As the stapes moves out of the oval window, perilymph is displaced toward the scala vestibuli with a corresponding inward movement of the round window. Because the basilar membrane (BM) is structurally flexible, it responds to this pressure by its own displacement. As the basilar membrane is displaced beginning at its base, the deformation moves toward the apex of the cochlea as a traveling wave. As the wave moves toward the apex of the cochlea, its velocity slows but the amplitude increases. Amplitude of basilar membrane displacement reaches a maximum at a specific point along the membrane before gradually attenuating. The point of maximum amplitude of basilar membrane displacement corresponds to the frequency of the stimulus. Thus, different sound frequencies produce different traveling wave patterns, forming peak amplitudes at different regions of the cochlea. Maximum amplitude of the basilar membrane traveling wave occurs near the base of the cochlea for high-frequency sounds. As the frequency of the stimulus decreases, the peak amplitude of the traveling wave moves toward the apex of the cochlea. A signal consisting of many frequencies will cause a traveling wave with multiple peaks along the basilar membrane.

The hair cells located at the point of the maximum basilar membrane movement are the most stimulated, suggesting that cochlear frequency selectivity is initially related to the mechanical properties of the basilar membrane. However, the frequency selectivity also may be related to structural and electrical properties of hair cells. This relationship between the maximum cochlear response and characteristic frequency is the basis of the place theory of hearing, and this tonotopic organization is preserved throughout the auditory system.

The deformation of the basilar membrane results in the mechanical displacement of the cilia of the hair cells by the overlying tectorial membrane. The mechanical displacement of the cilia—or shearing action—leads to the depolarization of the hair cells. The depolarization triggers the release of neurotransmitters from the synaptic vesicles at the base of the hair cells. The neurotransmitters have a depolarizing effect on the terminals of the auditory nerve fibers that results in the generation of action potentials which travel to the brain stem.

ELECTRIC TRANSDUCTION

The cilia of the hair cells are embedded in the endolymph, which has an electrical potential of +80 mV. This endolymphatic potential is supplied by stria vascularis. Stimulation of the cilia of the hair cells allows this potential to flow through the hair cell, initiating neural activity in the cochlea. Genetic or acquired structural pathology of stria vascularis is likely to alter the biochemical process for nerve impulses (Haines, 2002).

Specifically, the initiation of neural activity in the cochlea is a result of the ionic properties of the hair cells and the transmission of charged particles through the hair cell membranes. The presence of a −70 mV intracellular environment and +80 mV endolymphatic potential in scala media results in a 150 mV gradient difference across the apical ends of the cilia. This difference serves to regulate the electrical response of hair cells to mechanical deformation.

Basilar membrane movement deforms the stereocilia against the tectorial membrane, and this results in a graded depolarization of the hair cells. Deformation of the

stereocilia of hair cells increases potassium (K+) permeability and opens K+ sensitive pores in the tips of cilia. The inward K+ current enters the hair cells through the cilia and depolarizes the hair cells. This depolarization also opens the voltage-sensitive calcium (Ca_2+) channels at the base of these hair cells (Castro et al., 2003). This triggers an inward movement of calcium into the hair cells, which in turn initiates the release of glutamate (a fast excitatory neurotransmitter) from the synaptic vessels. The cochlear nerve terminals pick up the neurotransmitter and generate action potentials that travel to the cochlear nuclear complex located at the pontomedullary junction.

COCHLEAR AMPLIFIER

Basilar membrane movement provides passive analysis of sound. The OHC help provide active analysis. In other words, OHC serve to alter cochlear mechanics—that is, basilar membrane movement.

OHC have been found to contain contractile proteins. Stimulation of the cilia of the OHC (shearing action of the cilia) allows the endolymphic potential to pass through and cause the OHCs to contract or oscillate. The stimulated OHCs oscillate at the same frequency of the stimulus. This oscillation produces a receptor potential called the cochlear microphonic. This potential mimics the waveform of the stimulus. Because the cilia of the OHC make direct contact with the tectorial membrane, the contractile property of the OHC causes BM movement to be more finely tuned. The contractile properties of the OHC also serve to increase the sensitivity of the ear as a cochlear amplifier. OHC oscillation increases the motion in the cochlea, allowing the tectorial membrane to stimulate (or shear) the cilia of the IHC—the true sensory cells of hearing. OHC contraction (and thus amplification) is dependent on the intensity of the stimulus. OHC participation is greater at low intensities and less at high intensities.

NEURAL CODING OF AUDITORY INFORMATION

The transmission of auditory information from the cochlea to the central auditory nervous system is coded so that all its elements—timing, intensity, frequency, and others—are fully retained. The exact mechanism and format for coding acoustic information are not completely understood. However, it is likely that this information is coded in a variety of ways to ensure a degree of redundancy. For example, cochlear representation of frequency is based on the stimulation of hair cells at a specific region along the basilar membrane. Although cochlea neural units can be stimulated by a wide variety of sound frequencies, they respond maximally only to a specific frequency with a low threshold for that frequency. Thus, the place of stimulation along the basilar membrane may be the way frequency is initially coded in the auditory system. Sound intensity also could be coded by the number of related neural units stimulated along the basilar membrane or by the rate of neural discharge. Furthermore, the location of nerve fibers in the auditory nerve bundle may dictate their role in transmitting information about the auditory signal. For example, the fibers traveling centrally within the ascending tract may mediate specific tonotopic frequency attributes, whereas the fibers that are tertiary and located in the outer edge of the auditory pathway may be responsible for mediating additional coded properties of sound, such as timing, intensity, and information related to binaural or monaural interactions (Kingsley, 1999).

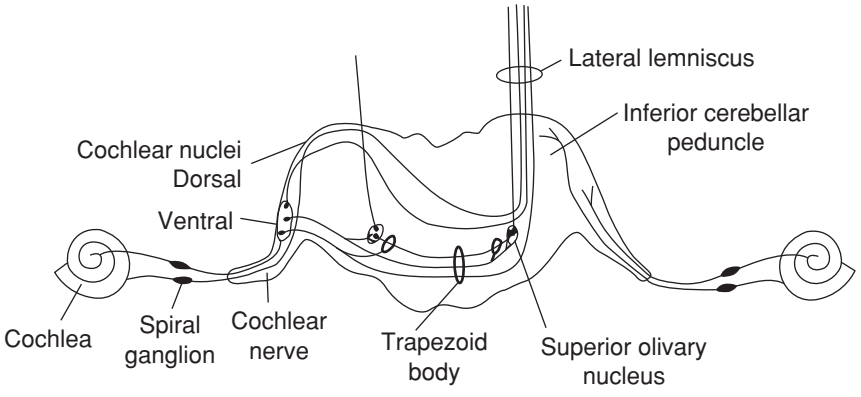

FIG. 1.3. A diagrammatic illustration of the retrocochlear neural mechanism. From *Neuroscience for the Study of Communicative Disorders*, 2nd. ed. by Subhash C. Bhatnagar, 2001. Copyright 2001 by Lippincott, Williams and Wilkins.

RETROCOCHLEAR AUDITORY MECHANISM

The retrocochlear portion of the auditory system (Fig. 1.3) consists of the distal/peripheral (unmyelinated) and proximal (myelinated) processes of the bipolar spiral ganglion cells. Also referred to as the auditory or cochlear nerve, the unmyelinated peripheral process of the spiral ganglion cells enter the basilar membrane to connect with the hair cells. They pick up the neurotransmitter from the synaptic transmission of the hair cells and generate nerve impulses, which are then transmitted through the proximal fibers of the spiral ganglion cells to the cochlear nuclei in the brain stem. There are about 30,000 spiral ganglion cells, made up of two types of neurons: Types I and II. Amounting to about 90% of the total spiral ganglion cells, Type I cells respond to a narrow range of frequency by being connected to only a few selective hair cells in the cochlea. On the other hand, the axonal processes, originating from Type II cells in the spiral ganglion, are known to synapse with 10 or more hair cells—suggesting their sensitivity to a wider range of frequencies with less precision.

The central processes of these spiral ganglion cells form the acoustic branch of cranial nerve VIII and pass along with facial cranial nerve through the internal acoustic meatus, a canal in the petrous portion of the temporal bone, before synapsing on the cochlear nuclei at the ponto-medullary junction. This explains why a pathology in the acoustic meatus may result in an impairment of both audition and facial functions.

CENTRAL AUDITORY PATHWAYS

The auditory cortical projections, unlike the projections of other sensory systems like somatosensation, are perhaps the most complex because of their multiple synaptic relays at many levels between the cochlear nuclei (second-order neurons) and the thalamus (third-order neurons). The transmission of auditory information to the cortex is regulated by a simple rule that relates to the preservation of the tonotopic code from the cochlear hair cells to the primary auditory cortex. Other aspects of the audition that also are preserved involve the contralaterality of projections and the incorporation of monaural and binaural information. Contralaterality refers to the projections that originate in one ear and travel to the auditory cortex on the opposite side. Some of

this information also is transmitted to the auditory cortex on the same side (ipsilateral). Interaural time difference in processing binaural (related to both ears) auditory information is used to identify the direction and source of sound (Bhatnagar, 2001; Kingsley, 1999; Haines, 2002).

The auditory pathway contains two types of fibers: centrally located core fibers and peripherally located belt fibers (Kingsley, 1999). The core fibers are organized tonotopically and maintain tonal representation throughout their course. The belt fibers are less organized in terms of their frequency representation and may be sensitive to timing and spatial aspects of sound patterns.

The central auditory pathway (Fig. 1.3) extends from the cochlear nuclear complex to the primary auditory cortex in the temporal lobe. The myelinated, proximal fibers of the auditory nerve enter the brain stem laterally at the pontomedullary junction and synapse upon the cochlear nuclear complex. Hierarchically organized structures in the central auditory pathway include the cochlear nuclei, fibers of the acoustic stria, trapezoid body, superior olivary nucleus, lateral lemniscus, and inferior colliculus. The auditory pathway from the midbrain to the cortex includes the brachium of inferior colliculus, the medial geniculate body, the auditory radiations (geniculocortical fibers), and the primary auditory cortex located in the transverse gyri of Heschl.

COCHLEAR NUCLEAR COMPLEX

Fibers of the auditory nerve enter the brain stem at the pontomedullary junction dorsolateral to the inferior cerebellar peduncle (restiform body). They terminate in the cochlear nuclear complex, which contains dorsal (posterior) and ventral (anterior) groups of nuclei (Fig. 1.4). The dorsal cochlear nucleus is located dorsal–lateral to the restiform body, whereas the ventral cochlear nucleus is located ventral–lateral to the restiform body. The entering fibers of the auditory nerve divide into dorsal and ventral bundles (stria) in order to synapse onto the cochlear nuclei. Each of the afferent fibers makes specialized synaptic contacts with multiple cell types in the cochlear nuclear complex. These synaptic contracts are orderly distributed in rows in order to maintain discrete tonotopic representation. The cochlear nuclear complex is also known to contain different specialized cells like bushy and multiform (Kingsley, 1999). Some of these cells may provide a sustained response to tones mediating important sound attributes like phase and timing, while the other cells may respond to changes in sound pressure level.

An important principle governing the functional representation at the cochlear nuclear complex and through the ascending auditory pathway is the preservation of precise tonotopic organization. The frequency-related projections from the hair cells in the cochlea synapse ipsilaterally on the specialized cells in the cochlear nuclear complex so that a discrete tonal representation is retained. The fibers from the apex of the cochlea, which represent lower frequencies, terminate at the superficial layers of the cochlear nucleus. The fibers from the base of the cochlea, which represent higher frequencies, penetrate deeper in the nucleus and thereby preserve the tonal correspondence. This discrete tonotopic representation is retained throughout the ascending fibers of the central auditory pathway and all its nuclei up to the auditory cortex.

ASCENDING PROJECTIONS FROM COCHLEAR NUCLEAR COMPLEX

The cochlear nuclear complex gives rise to parallel pathways that carry coded auditory information like intensity as well as monaural and binaural aspects of sounds. These projections, called the lateral lemniscus, ascend in both the ipsilateral and/or contralateral auditory pathways. The exact path taken by these ascending fibers is not as clear as it seems to be in the commonly illustrated diagrams of the brain stem auditory pathways. Most auditory fibers are known to cross the midline and project to the contralateral auditory system, although a small amount of fibers do not cross the midline and instead ascend ipsilaterally (Fig. 1.4).

The cochlear projections that cross the midline travel in three bundles: the dorsal acoustic stria, the intermediate stria, and the trapezoid body. There is a functional

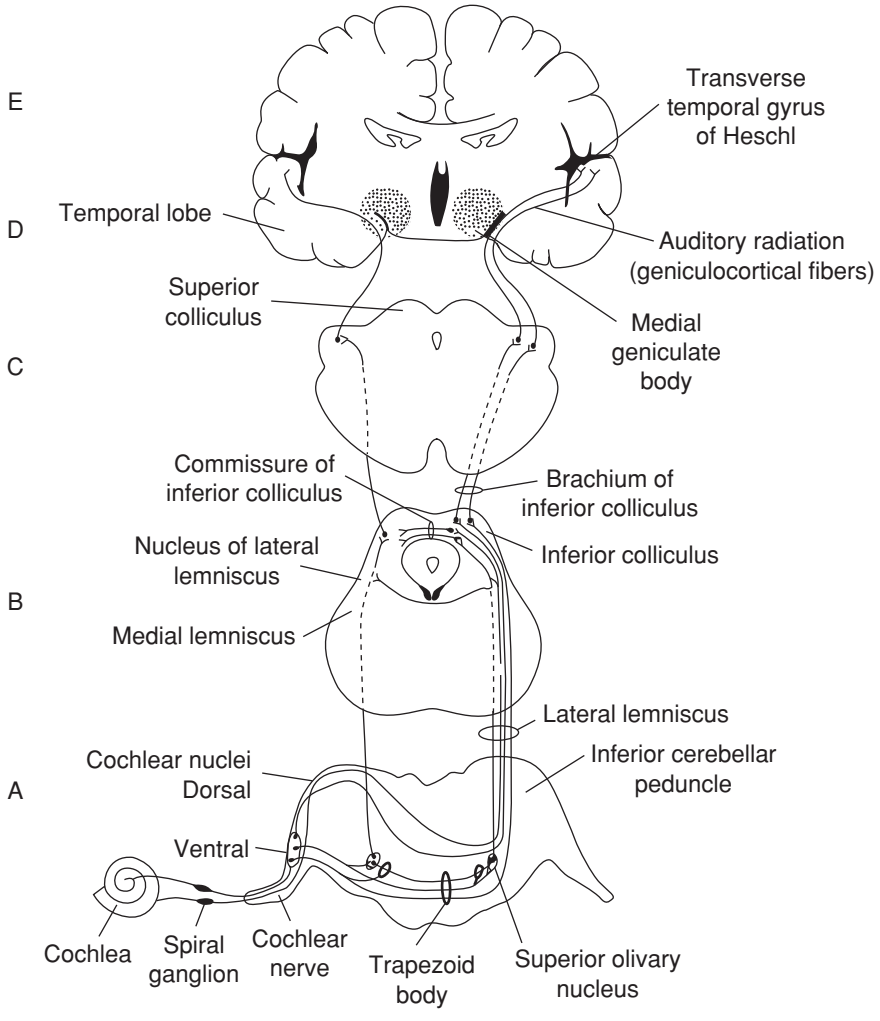

FIG. 1.4. A diagrammatic illustration of the central auditory pathway. From *Neuroscience for the Study of Communicative Disorders*, 2nd. ed. by Subhash C. Bhatnagar, 2001. Copyright 2001 by Lippincott, Williams and Wilkins.

purpose for each of these axonal channels in terms of mediating specific signal attributes; however, our knowledge of this coding as well as the encoded ones remains incomplete. The cells along the crossing fibers form the nucleus and the trapezoid body. The fibers of the dorsal acoustic stria cross the midline and ascend in the contralateral lateral lemniscus without sending projections to any of the superior olivary nuclei (SON). Some collaterals from the fibers of the intermediate acoustic stria may project to the ipsilateral and/or contralateral superior olivary nucleus while the main body of fibers joins the contralateral lateral lemniscus. The fibers of the trapezoid body, the largest stria of the three bundles, cross the midline to terminate in the SON, which is located laterally in the dorsal pons next to the nucleus of the trapezoid body. Some of the auditory fibers that cross the midline may bypass the SON on their way to the contralateral lateral lemniscus.

SUPERIOR OLIVARY NUCLEUS

The superior olivary nucleus (SON), a collection of nuclei in the pons, is located adjacent to the facial motor nucleus and the facial cranial nerve fibers. This nucleus plays a significant role in localizing sound and is found to be highly developed in animals. Known to be an important auditory relay nucleus, it is the first nucleus to receive inputs from both the ipsilateral and contralateral cochlear nuclei.

The SON contains binaural cells: the lateral superior olive and medial superior olive, which are surrounded by small peri-olivary nuclei. The cell bodies in the SON contain two large dendrites extending in opposite sides from the soma. The medial dendrite receives crossed projections from the contralateral cochlear nuclei, whereas the lateral dendrite receives projections that are from the ipsilateral cochlear nuclei. This anatomical arrangement allows the SON to use interaural difference in sound intensity in addition to the difference in time of sound arrival to calculate the direction and determine the location of sound. Even a slight time difference of as little as 400 msec is considered functionally significant. This ability to localize sound is remarkable, as the path difference between both ears is only approximately 5–6 inches or so.

In addition to receiving binaural afferents, the SON also sends massive inhibitory efferents in olivocochlear fibers to the cochlear hair cells, particularly the OHC. This allows the olivary nucleus to not only regulate the responsiveness of the hair cells to stimuli, but also suppress background noise to improve speech perception in noise.

LATERAL LEMNISCUS

The lateral lemniscus (LL), the primary ascending auditory pathway, extends from the cochlear and superior olivary nuclei to the inferior colliculus of the midbrain (Fig. 1.4). Its fibers climb laterally in the tegmentum (central area) of the pons toward the midbrain. The cell bodies located along the ascending fibers form the nucleus of the LL. Receiving most of its crossed afferents from the ipsilateral superior olivary nucleus and uncrossed afferents from the ipsilateral cochlear nuclear complex, the LL retains bilateral representation with added and stronger representation from the opposite ear. This bilaterality of representation explains why pathology of the central auditory pathway at any point does not always lead to reduced hearing sensitivity in only one ear. The fibers of the LL ascend adjacent to the fibers of the medial lemniscus

that mediate fine discriminative touch in the brain stem toward the inferior colliculus in the midbrain (Fig. 1.4). On their way to the midbrain, the fibers of the lateral lemniscus pass dorsolaterally in the tegmentum of the pons, potentially making numerous connections.

INFERIOR COLLICULUS

The fibers of the LL, containing projections from contralateral and ipsilateral ear, ascend through to the inferior colliculus (IC), the round egg-shaped structure in the lower midbrain (Fig. 1.4). Virtually all of the LL fibers are actually known to synapse upon the central cells in the IC; the remaining fibers bypass the nuclei of the IC. Both IC are connected through the bidirectional commissural fibers of the IC, traversing through the pericentral nuclei and connecting with the ascending fibers bilaterally. This anatomical organization permits crossing and further integration of monaural and binaural auditory input, which has additional implications for the sound localization and for determining its other attributes.

The cellular organization in the IC is known to contain frequency-specific regions. However, with increased neuroanatomical complexity and projectional diversity, the central and pericentral cells of the IC are likely to respond to complex patterns of auditory stimuli indicating a higher level of information processing.

With its projections to the deeper cellular layers of the adjacent tectum and superior colliculus (the midbrain structure that mediates visual reflexes), the IC is likely to be concerned with auditory–visual and auditory–motor reflexes. Part of the auditory information from the IC is projected to the cerebellum and adjacently located reticular formation. The information received about the angular location of the sound source is integrated by the cerebellum with visual and other sensory inputs. This integrated information is projected on different ascending and descending pathways to coordinate reflexive movements of the eye, head, and body toward the sound source. With projections to the midbrain reticular formation, the cells in the IC are also likely to be involved with attentional processes and screening of auditory information.

The primary output of the inferior colliculus is to the medial geniculate body of the thalamus. The projections to the thalamus intersect central/external nuclei of the IC and travel through its brachium (Fig. 1.4).

MEDIAL GENICULATE BODY

The medial geniculate body (MGB) is the thalamic relay center for the transmission of auditory information. Shaped like a small protruding ball, it is located in the lateral–caudal portion of the thalamus between the lateral geniculate body and pulvinar (the posteriormost thalamic nucleus) and receives its input from the IC in the midbrain (Fig. 1.4). There is no known crossing of fibers directly at the level of the MGB, but nonetheless the possibility remains for some information to cross to the other side through the thalamic commissural fibers known as the massa intermedia and/or through the corpus callosum. The anterior portion of the MGB, with afferents from the central cells of the IC, projects the frequency representation to the primary auditory cortex in transverse gyri of Heschl, Brodmann area 41. The middle and posterior MGB regions, with afferents from the central and pericentral cells in the IC, project to the primary and secondary auditory cortex, Brodmann areas 41 and 42.

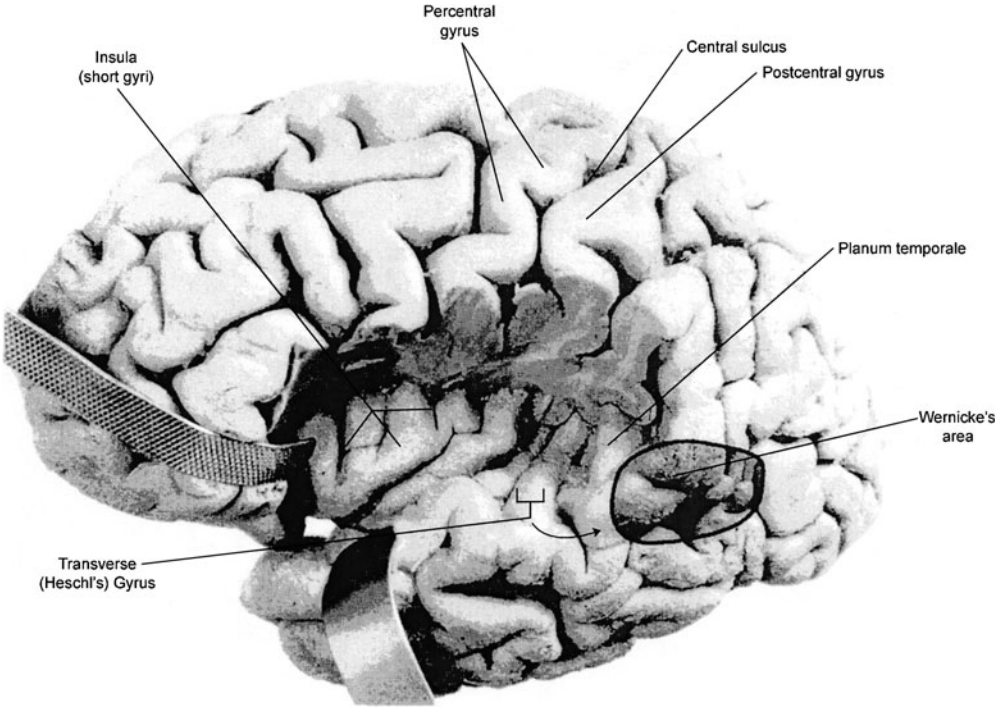

FIG. 1.5. The human brain dissected to expose the primary auditory cortex and the temporal planus in the dominant left hemisphere. From *Neuroscience for the Study of Communicative Disorders*, 2nd. ed. by Subhash C. Bhatnagar, 2001. Copyright 2001 by Lippincott, Williams and Wilkins.

Functionally, this region may be responsible for additional distinctive attributes of the sound.

It is anatomically plausible that with multiple but diverse projections to the adjacent putamen (basal ganglia), amygdaloid nucleus (limbic lobe), and to the tertiary regions of the temporal and parietal cortex, the MGB, with afferents from the brain stem reticular activating system, may participate in regulating attention, activated visceral functions, emotional expression, and integration with pain mechanism.

The projections of the MGB (auditory radiations or geniculo-cortical) pass ventrally (sublenticular) and caudally (retrolenticular) to the lenticular portion of the internal capsule. They terminate in the ipsilateral primary auditory cortex, the gyri of Heschl, which are located in the superior temporal lobe (Fig. 1.5).

AUDITORY CORTEX

The primary auditory cortex (Brodmann area 41) is located on the superior surface of the temporal lobe and is formed by two transversely oriented gyri: anterior gyrus and the portion of the posterior transverse gyrus of Heschl (Fig. 1.5). This area of primary auditory cortex is surrounded by the secondary auditory cortex (Brodmann area 42), which extends onto the lateral surface of the temporal lobe, representing the structural property of the koniocortex with a well-developed inner granular layer similar to the visual cortex and somatic sensory cortex.

The primary auditory cortex receives afferents from the MGB representing crossed and uncrossed fibers from both ears and is known to retain the cochlear tonotopic representation (Fig. 1.4). The geniculo-cortical fibers mediating higher frequencies terminate in the posteromedial region of the gyrus of Heschl and the fibers transmitting lower frequencies synapse in the anterolateral region. The area in between these two regions receives fibers carrying the middle range of frequencies. This tonal representation is based on experiential studies on mammal brains and has not yet been demonstrated on the human brain (Bhatnagar et al., 1989). The secondary auditory cortex along with the primary auditory cortex also represents other essential properties of audition such as timing patterns and spatial attributes, which characterize human speech.

Research carried out on the brains of cats suggests that the primary auditory cortex is not absolutely essential for frequency discrimination; rather, it is of vital importance in recognition and discrimination of sound patterns that are based on the timing and spatial patterns. These timing and spatial attributes of sound patterns play a significant role in the perception of human speech. This has been supported by the observations of unimpaired frequency discriminations and near-normal hearing thresholds in patients with unilateral and bilateral cortical lesions. However, individuals with cortical lesions also are found to exhibit an impaired ability to perceive and discriminate speech. A somewhat similar observation has been made on cats trained to discriminate low–high–low pitch tone sequence from high–low–high pitch patterns. After the cortical ablation, these trained cats could no longer discriminate such temporally based sound patterns.

Located posterior to the auditory cortical region is the planum temporale (temporal planum) area, which is hidden by the overlying operculum of the temporal, parietal, and frontal lobes (Fig. 1.5). In most individuals, the left planum temporal area has been found to be larger in the right brain, a fact that has been related to cerebral dominance by Geschwind and Levitsky (1968).

The primary auditory cortex and surrounding area are the site of auditory sensation and perception. An extensive axonal bundle connects the primary auditory cortex to Wernicke's area (Brodmann area 22), the language cortex, which includes part of the planum temporale and posterior–superior first temporal gyrus. It is concerned with recognizing language stimuli, interpreting their meanings with respect to auditory memories and linguistic experiences, and comprehending spoken language. Wernicke's area, as part of the larger language interpretative cortex, also receives visual and somesthetic information and may contribute as well to language formulation. An injury confined to Brodmann area 22 results in Wernicke's aphasia, a language syndrome characterized by impaired auditory comprehension and fluently produced copious speech marked with a severe word-finding deficit and the verbal output carrying little meaning. Similar linguistic errors are noted in reading and writing. Wernicke's area also extends to the inferior parietal lobule that contains supramarginal (Brodmann area 40) and angular (Brodmann area 39) gyri, the structures that are known to play an important role in reading and writing functions.

DESCENDING AUDITORY PROJECTIONS

Descending fibers, which run parallel to the auditory ascending afferents projections, are known to exist along the entire course of the auditory pathway, with conduction of impulses traveling in the reverse direction (Fig. 1.4). These synaptic connections of the

ascending and descending fibers not only regulate the functioning of all the auditory relay nuclei, but they also serve as feedback circuits to refine the perception of pitch and loudness properties and to sharpen the reception of specific frequencies through the process of lateral inhibition. The recurring connections of the descending fibers begin from the primary auditory cortex and make synaptic relays to the thalamus, brain stem (inferior colliculus), and superior olivary nucleus before terminating in the cochlear hair cells. The descending connections, consisting of cortico-geniculate, cortico-collicular, colliculo-olivary, and olivary-cochlear fibers, serve to modulate responsiveness of hair cells and improve the signal-to-noise ratio. They also are known to enhance cues for sound localization and contribute to the quality of the perceived sound by suppressing competing signals. Furthermore, the descending pathways also help us differentiate between sounds generated externally from ones generated internally. This ability to differentiate sound sources explains why the auditory nuclei are inhibited during self-vocalization. Gamma aminobutyric acid (GABA) and a glycine are the neurotransmitters that are known to work on inhibitory interneurons involved with the descending auditory pathway.

Originating from the SON, the olivo-cochlear bundle (OCB) is composed of two tracts: lateral and medial. The lateral tract is an uncrossed fiber tract that originates from the lateral superior olivary complex (SOC). The fibers composing this tract are unmyelinated and connect with afferent auditory fibers at the base of the IHC. The medial tract is mostly a cross-fiber tract that originates from the medial SOC. Fibers of this tract are myelinated and are fewer in number than those composing the lateral OCB. These fibers connect directly to the base of the OHC. It has been suggested that the OCB functions as a feedback loop that causes some type of regulation or inhibition in the cochlea. The OCB may play a role in suppressing background noise to help people hear in noise. Specially, the OCB may serve to attenuate the transduction of certain sound frequencies by regulating the contractile properties of the OHCs (e.g., cochlear amplifier). By changing the OHC length, it lifts the tectorial membrane and alters the responsiveness of the stereocilia of the selective IHCs by reducing their bend subsequent to a movement of the basilar membrane. It is known that reflexive activity of the olivary-cochlear projections can reduce hearing sensitivity by over 20–25 dB (Kingsley, 1999).

AUDITORY REFLEXES

Auditory reflexes integrate sensorimotor information to coordinate head and eye movements and direct attention toward the sound, control the middle ear ossiculare movements, and also influence vestibular functions. This sound-triggered reflex mechanism involves four anatomical pathways.

The first pathway includes the projections from the inferior colliculus to the deep cellular layers of the superior colliculus and tectum (midbrain region responsible for ocular reflexes), which integrates auditory and visual systems. This information is transmitted through the visceral fibers of the oculomotor cranial nerve for controlling intraocular movements in the startle reflex.

The second pathway involves the integrated auditory efferents that travel through the superior olivary nucleus in the brain stem to the medial longitudinal fasciculus, which projects bilaterally to the motor nucleus of ocular (oculomotor, abducens, and trochlear) cranial nerves. Part of the information is also relayed to the neck muscles. Neural impulses traveling on these two pathways regulate ocular movements and head coordination, and startle response in response to auditory stimuli.

The third pathway includes the auditory projections to the adjacent vestibular nuclei and the brain stem reticular area, which project to cervical and spinal motor neurons through the tectospinal projections. This participates in body equilibrium and controlling head position. It also regulates the whole body movement in response to a loud sound.

The fourth pathway involves regulating the ossicular movements by controlling the muscles of stapedius and tensor tympani. The motor nuclei of the facial and trigeminal cranial nerves are connected with the superior olivary nucleus. Serving as the sensory path of the reflex, the input from the cochlear and superior olivary nuclei activates the facial and trigeminal nerves that contract the stapedius and tensor tympani muscles in the middle ear. This contraction dampens the ossicular movements to protect it from being overdriven, causing damage at the ossicular joints or beyond in the cochlea in response to high intensity stimuli.

CLINICAL CONSIDERATIONS

Disorders of hearing result from unilateral and/or bilateral damage to the peripheral auditory system (outer ear, middle ear, inner ear, auditory nerve fibers) and/or the central auditory nervous system (cochlear nuclear complex to the primary auditory cortex). The specific nature of a hearing impairment is determined by the anatomical location of the lesion rather than its etiology.

The symptoms of any peripheral auditory system lesion, in general, include reduced hearing sensitivity and difficultly hearing and/or understanding speech. These symptoms can be measured using conventional audiometric procedures. Lesions involving the central auditory nervous system (CANS) affect the transmission of information and are associated with signal processing impairments, many times without a significant loss of hearing sensitivity. Pathologies of the CANS predominantly produce symptoms in the ear contralateral to the site of the lesion.

SENSORINEURAL HEARING LOSS

Sensorineural hearing loss (SNHL) is an impairment of hearing that results when there is damage to the neural units within the cochlea and/or the neural structures that lie beyond. SNHL results from neural unit damage—either to the hair cells in the cochlea or to the auditory nerve fibers. In most cases, a SNHL is permanent/irreversible.

COCHLEAR PATHOLOGY

SNHL due to cochlear pathology is an impairment of hearing that results from damage to the hair cells in the cochlea. Symptomatology of a SNHL due to cochlear pathology includes:

Poor word recognition ability. Patients can hear speech but are unable to understand it. They frequently complain that "people mumble when speaking." This poor word recognition ability may still be present even when the intensity of speech increases above normal conversational level.

Greater difficulty hearing in noise. Patients frequently complain that they have much more difficulty hearing in noise. They may state that they have little or no difficulty

hearing in quiet, but if there is any background noise present (e.g., microwave oven, dishwasher, running water, television, etc.) they have extreme difficulty understanding speech.

Tinnitus. Patients frequently complain of tinnitus (sensation of ringing or other sound in the head without external cause), which may take a variety of forms. The most common form is a high-pitched ringing sensation.

Speaking more loudly. Patients have difficulty self-monitoring the loudness of their voice due to the hearing loss. To compensate, they tend to speak at a higher than normal conversational level.

Recruitment. Recruitment is defined as the abnormally rapid growth of loudness. In other words, once the individual's threshold of hearing is reached, any further increase in the intensity of sound will be perceived as being uncomfortably loud. Patients with recruitment complain that they have difficulty understanding speech at the normal conversational level. However, if the speaker raises the intensity of his or her voice above normal conversational level, it appears to the patient that the speaker is shouting. Recruitment is consistent with outer hair cell damage.

AUDITORY NERVE PATHOLOGY

Impairment of hearing also can result from disease, irritation, or pressure on the nerve trunk of the auditory nerve. This lesion typically results in a structural alteration that is visible radiologically. The symptomatology of this SNHL includes hearing but not understanding speech, greater difficulty hearing in noise than in quiet and tinnitus. Typically, a patient with this site of lesion will describe his or her tinnitus in unusual terms—for example, bacon frying, hissing, or buzzing.

AUDITORY NEUROPATHY

Auditory neuropathy is a hearing impairment marked by abnormal functioning at the level of the auditory nerve with no visible structural alteration. Possible sites of auditory neuropathy include IHC, the synaptic juncture between IHC and the auditory nerve, and the auditory nerve or perhaps auditory pathways of the brain stem (lateral lemnicus). Patients may or may not have other neuropathies outside of the auditory system. Auditory neuropathy also is referred to as auditory dysynchrony because of the dyschronous pattern of neural activity. Symptomology of auditory neuropathy includes hearing but not understanding speech and more difficulty hearing in noise than in quiet. This symptomology may exist even without sensitivity loss.

CLINICAL ASPECTS OF THE CENTRAL AUDITORY NERVOUS SYSTEM

The central auditory nervous system includes the lower brain stem (cochlear nuclei, superior olivary nucleus, and the lateral lemniscus), upper brain stem (inferior colliculus and medial geniculate body), and the primary auditory cortex. Clinically, the most identifying feature of CANS dysfunction is a near-normal sensitivity to auditory stimuli but impaired processing of linguistic signals and their metalinguistic properties.