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AUDITORY PHONETICS FOR THE SPEECH CLINICIAN
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Introduction

Phonetics is a branch of linguistics (the study of language) that is concerned with human speech sounds. The *phone* part of the word *phonetics* originates in the Greek ϕωνή [foˈni] which means “voice.” Voice is an attribute that may be simultaneously associated with different entities: the speaker, the sound itself, the listener. Because of this, the study of phonetics is distinguished into three main categories: articulatory (planning and execution of movements for speech production), acoustic (the physical properties of speech sounds) and auditory (the conversion of speech sounds into linguistic information), as also represented in the thematic contents of this volume. Expertise in phonetics is a vital asset in the speech clinician’s repertoire of skills.

The present chapter focuses on the latter of the three categories, auditory phonetics, in lay terms referring to “hearing,” as derived from the Latin term audire “to hear.” Thus, what follows is a succinct discussion of the fundamental themes and notions involved in the systematic study of normal and impaired hearing within the scientific field of audiology. Audiology came about in the mid-1900s as a result of the fields of speech pathology (*pathos* Greek “suffering,” *logy* Greek “speaking of”) and otology (*oto* Greek “ear”) coming together to represent, respectively, nonmedical and medical perspectives in our understanding of human hearing, the identification of hearing impairment, the assessment of hearing capacity/loss and subsequent rehabilitation procedures (Newby, 1979).

As with most scientific fields, audiology is wide-spectral in that it may be tackled from different perspectives while also informing and being informed by experts from diverse fields of investigation, three of which are medical: pediatrics (*pedia* Greek “children”), gerontology (*gero* Greek “elderly”) and neurology (*neuron* Greek “nerve”); and three are nonmedical: physics (*physi* Greek “nature”), psychology (*psycho* Greek “soul”) and education (*educare*, Latin “bring up, rear”). Pediatrics and gerontology are closely related to audiology because they are concerned with patients at two extreme age-groups that mostly evidence hearing impairment/loss. Similarly, neurology is germane to audiology because of the involvement of human nerves in transmitting the speech signal/sound to the brain for processing during communicative acts.
Among the nonmedical fields just mentioned, the physics of the speech signal refer, first, to **acoustics** (waves, the physical propagation of sound) and, second, to **electronics** (technological applications dealing with the emission, flow and control of electrons in matter) as used in diagnostic and rehabilitation procedures of hearing impairment (e.g. audiometers, hearing aids). Consultation with a psychology expert may also be required when hearing impairment adversely affects the psychological wellbeing of the person suffering it. Finally, hearing impairment/loss in children may have detrimental effects to the development of language and communicative skills, and the way these affect several aspects of children’s educational development.

The clinical emphasis of the present volume necessitates the inclusion of information on what is involved in hearing, both normal and in the presence of impairment/disorder—a dichotomy that will be also reflected in the thematic undercurrent of this chapter. Within the study of speech science, normal speaker/listener interaction is represented by a diagram referred to as *The Speech Chain* (e.g. Denes & Pinson, 1993), pictorially depicting interrelationships between a Speaker’s mouth, brain and nerves, the speech *Sound* waves, and a Listener’s outer ear, brain and nerves. Illustrations of the speech chain diagram are freely available online. Thus, the “fundamentals of hearing” involve: (i) the **ear** (“peripheral auditory anatomy and physiology”); (ii) the **sound** itself (“auditory stimulus”); (iii) the listener’s **sensitivity** to the sound (“auditory sensation”) in relation to pitch, loudness, sound localization, i.e. the ability to identify the location/origin of the sound, and sound masking, i.e. the ambient sound, specifically engineered to match human speech and used to minimize the extent of conversational distractions, (iv) how the **speech sound is processed** and the intermediary role of nerves transmitting signals to the brain (“auditory perception and the central nervous system”) (Yost, 2013).

Figure 3.1 is an illustration of the speech chain notion that is specifically adapted here to have a focus on hearing for the requirements of this chapter.

![Figure 3.1](image-url)

*Figure 3.1*  Hearing in the speech chain.
Source: S. Qiouyi Lu.
Because terminological clarity is not de facto, disambiguation of terms is also addressed where necessary. Both hearing “impairment” and “disorder” are in use in medical and non-medical academic articles and books to refer to a range of hearing problems, including hearing loss (Bagai et al., 2006; Crandell & Smaldino, 2000; Hudson & DeRuiter, 2019; Katz, 2014; Kennedy et al., 2005; Libb et al., 1985; Miceli et al., 2008). “Hearing impairment” will be the generic term preferred here. Definitions of “impairment” and “disability” allude to divergent but associated concepts: impairment is the loss of a body part or a significant deviation of a body function or structure, whereas disability is a person’s inability to be functional in personal, social or professional settings as a result of the presence of impairment (e.g. Dobbie & Van Hemel, 2005).

The remaining of this chapter consists of the following sections: the hearing mechanism, anatomy and physiology of the auditory system, routes of hearing, the measurement of hearing, causes and types of hearing impairment, and conclusion.

The hearing mechanism

Oral language in a speaker’s voice (speech signal or sound stimulus) takes the form of sound waves that enter the ear; the speech signal is then transmitted through the ear, and onward via the nerves to the brain where the meaning associated with the original linguistic signal is processed. In other words, the ear behaves like a biotransducer that changes the form of energy from sound wave, to mechanical vibration, to electrical impulse.

Anatomy and physiology of the auditory system

The human ear may be described in terms of its peripheral (peripheral auditory system) and internal (internal auditory system) constituents. The peripheral component is an exterior section, or a prefatory stage, leading to internal or more central organs and functions of the hearing mechanism.

The peripheral auditory system (PAS)

This section will describe the anatomy (or physical apparatus) of the ear. Figure 3.2 is an abstract representation of the right ear’s PAS (assuming it is facing the reader); see also Clark et al. (2007). Anatomical illustrations are freely available online.

There are three parts in the PAS: the external ear, the middle ear and the inner ear. The external ear consists of the auricle or pinna (the only visible part of the human ear) and the external auditory canal (or meatus). The auricle functions like a funnel capturing the sound signal in the environment (cf. similarly, the hand cups behind the ear in noisy contexts); then, the sound wave passes through the auditory canal and briefly stumbles on the eardrum penetrating it to enter the middle ear. The eardrum, tympanic membrane (Greek “thin tissue shaped like a drum”) has a semitransparent, quasi-circular shape coning inwards. It is the physical boundary of the middle ear between the auditory canal and the first of the three auditory ossicles (tiny bones) that form a chain (ossicular chain) in the middle ear. In order of appearance and decreasing size, these bones are: the malleus (Latin “hammer”), the incus (Latin “anvil”) and the stapes (Latin “stirrups”)—the last one being the smallest in the human body (3 mm long).

The ossicular chain has two functions: (i) it is a mechanical lever system that increases the force of the transmitted signal and ii) it behaves like an acoustic reflex mechanism that contracts to regulate the sound level when the noise reaching the middle ear is extremely loud.
It is the *stapedius* muscle that actually stabilizes the stapes, attenuating intense sounds in the area. The degree of movement of the ossicular chain resulting from the stapedius’ contraction is called *amplitude*. The time between the onset of sound and the contraction is called *latency*. The entire middle ear section is called the *tympanum* "drum," because it transforms the sound pressure vibrations coming from the auditory canal into actual mechanical movements of the ossicular chain, as the ossicles vibrate consecutively, much like a chain reaction.

The stapes are attached to the *oval window*, a breach in the skull’s bone structure which actually has a smaller surface than the eardrum. Given that pressure is great on a smaller surface than on a larger one, the resulting acoustic energy on the oval window is 35 times greater than on the eardrum. Pressure differences between the external and the middle ear are balanced by the *Eustachian tube* (*pharyngotympanic tube*) that connects the middle ear with the nasal and oral cavities releasing the pressure (cf. the function of swallowing to equalize pressure in the ear in airplane flights). The purpose of the middle ear, thus, is to transmit, regulate and amplify the incoming signal, as the signal is further thrust toward the inner ear.

The inner section of the ear, also referred to as the *labyrinth*, is very complex in structure, and it includes *fluid*, the *cochlea*, a snail-like organ that coordinates hearing, and the *vestibular apparatus* that is responsible for balance. Balance is achieved because of the position of the organs of the vestibular apparatus that stays the same irrespective of the position of the head in space. The *semicircular canals* form part of the vestibular apparatus. The cochlea and semicircular canals are connected in a shared area, the *vestibule*. One side of the cochlea, the *base*, connects to the oval window leading to the stapes and the other side, the *apex*, is the innermost section of the cochlea. The cochlea itself consists of three tubes rolled up like the shell of a snail—each tube divided by the next by a membrane: the *vestibular* and the *basilar*. Inside the basilar membrane, the *organ of Corti* has tiny inner and outer hair cells that link to the auditory nerve. Fluid in the cochlea is of two types: *perilymph* and *endolymph* that stimulate the
sensorineural hair cells of the organ of Corti. The function of the organ of Corti is pivotal for our perception of the speech signal.

The inner ear has a similar function to that of the middle ear transmitting, altering and evaluating incoming sounds. First, the inner ear converts the vibrations coming from the middle ear into neural signals, as follows: middle ear vibrations are transported by the cochlear fluid into the basilar membrane, which sets in motion the hair on the organ of Corti, and the hairs convert vibrations into electrical impulses that are sent, in turn, to the brain via neurotransmitters. It is, thus, apparent that proper functioning of the hair on the organ of Corti is critical for normal hearing. Another function of the inner ear is that of sound analysis. This is done by the vibration of the hair on the organ of Corti that amplifies sounds. The basilar membrane sorts out the incoming sounds based on their frequency: sounds with higher frequency (max. 32,000 Hz for humans) activate the cochlea at the base, while sounds with lower frequency (min. ca. 20 Hz for humans) activate the cochlea at the apex; intermediate frequencies are activated accordingly around the cochlea curl. Due to higher frequencies processed there, the cochlea base is more vulnerable to damage from intense vibration, especially around the 3,000–6,000 Hz range. In fact, the point of most hearing loss is localized around the 4,000 Hz dip. Figure 3.3 shows the tonotopy, i.e. the spatial arrangement (Greek <topos> “place”) of tones (sounds of different frequency) in the cochlea; see also Lahav and Skoe (2014).

Finally, the basilar membrane has a bidirectional function in that neural vibrations coming from the brain can also affect its resting status due to cellular or mechanical causes, thus, causing it to move; this vibration forms a brain-prompted sound, known as otoacoustic emission (OAE) (e.g. Kemp, 2002). OAEs can be measured with sensitive microphones and are routinely used as a simple, noninvasive hearing test; complete lack of OAEs is evidence of hearing impairment of the inner ear.

Figure 3.3 Cochlea and its tonotopy across the frequency spectrum.

Source: S. Qiouyi Lu.
The internal auditory system (IAS)

The IAS is responsible for transmitting sound information to the brain for processing (high executive function). The IAS consists of the auditory nerve and the central auditory system (CAS). The auditory nerve utilizes electrical impulses to transfer the speech signal information that was coded in the cochlea to the brain. In particular, the inner hairs act as transducers converting vibration into neural discharge. The bending of the hairs on the inner hair cells causes a neuroelectrical response, while the size of the outer hairs changes leading to mechanical alteration of the cochlea. This coupling activates a response from the nerves which is transferred through the auditory nerve to different parts of the brain for further analysis. Different nerve cells (and their fibers) are linked to different parts of the organ of Corti, each specializing in a specific sound frequency. Under normal hearing, the nerve fibers go past the auditory nerve, enter the brainstem (posterior part of the brain) at the cochlea nucleus, gradually proceeding to the superior olivary complex (SOC) and the inferior culliculi in order to eventually reach the cerebral cortex (or auditory cortex). The areas of auditory reception are mainly in the temporal lobes on both sides of the cortex, but other parts of the cortex are also involved in the representation of hearing. Each ear’s representation in the cortex is bilateral; as a result, disruption of the auditory track on one side of the brain does not impede hearing in either ear. The interested reader is directed to Musiek and Baran (2007), Rouse (2019) and Seikel et al. (2020) for more detailed anatomical descriptions of IAS, and Delgutte (1997) for a discussion on auditory neural processing of speech sounds.

The function of the auditory nerve is often investigated in disorders caused by a tumor (acoustic neuroma) and in rehabilitation procedures using cochlear implants. Similarly, if the capacity of nerve fibers to process different sound frequencies is compromised, our hearing capacity is also compromised. An unexpected loud sound, or long-term exposure to high and intense noise, may permanently impair hair cells that lose their biotransducer capacity and are incapacitated. Hearing impairment due to aging also results from nerve failure to process sound frequencies and transmit the processed information to the auditory cortex.

The acoustic reflex

The acoustic reflex (AR) is often utilized as a clinical tool to assess the healthy status of the hearing mechanism in the middle ear, the cochlea, cochlea nucleus, as well as the low auditory brainstem in the function of SOC. Nevertheless, conductive hearing loss (discussed below) limits ability to observe a patient’s AR. The AR primarily functions to prevent damage to the cochlea from high intensity sounds (see above the function of stapedius). The stapedius contracts more at high intensities and less at low intensities and in silence. When the AR is activated in one ear, both stapedius muscles (one per ear) simultaneously contract, informed by facial nerves on either side of the head. Typical AR thresholds in humans are in the range of 80–90 dB, with frequencies over 250–4,000 Hz having little effect. The amplitude of the AR is proportional to the sound’s intensity level, while its latency is disproportional to the sound’s high intensity and frequency.

Tinnitus

The normal perception (sensation) of sound in the ear with a lack of external acoustic stimulus is called tinnitus and is described as a buzzing, hissing, humming, ringing or whistling sound. Common experiences of tinnitus include the aftermath of having been close to loud speakers playing in concerts, or sensing a pulse beat. Tinnitus can be either objective (rare occurrence...
with an internal acoustic stimulus, e.g. turbulent blood flow near the ear) in that the audiologist can hear it, or subjective (occurring with no acoustic stimulus at all) in that it is only heard by the person experiencing it.

Tinnitus is common in people with hearing loss, but not all people with hearing loss experience it. It may be the result of broken/damaged auditory hair cells, turbulence in a carotid artery or jugular vein, temporomandibular joint (TMJ) issues and problems in the auditory processing pathways of the brain. Tinnitus tones (sound frequencies) are close to the frequencies people have difficulty hearing. The intensity of an external tone matched in loudness to a person’s tinnitus is usually less than 10 dB above that person’s threshold for the tone. Tinnitus does not customarily cause suffering, though trouble concentrating and understanding speech, emotional issues (anxiety, depression, etc.), and sleep disturbance have been reported in extreme cases (e.g. Tyler & Baker, 1983).

**Routes of hearing**

While anatomists are interested in the physiology of the hearing mechanism, audiologists and physicians focus on measuring hearing for the assessment and diagnosis of hearing impairment. In this point of view, the function of the ear can be seen as either conductive or sensorineural. Conduction refers to the process by which sound waves travel through a medium and is distinguished into air conduction and bone conduction. The two fork-like symbols in Figure 3.4 point out the two different conduction paths, but they also represent a tuning-fork, which is an

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**Figure 3.4** Conductive versus sensorineural hearing.

Source: Courtesy S. Qiouyi Lu.
instrument producing tones at different pitch values and traditionally used by audiologists to test hearing sensitivity (i.e. how well the ear hears).

Most of the sounds we hear are airborne; air conduction takes place in the external ear, middle ear and beyond, as sound pressure is channeled via the eardrum (external ear) and ossicles (middle ear) to the inner ear composed of fluid and tissue. This phenomenon whereby a medium carries (“admits”) sound inward is called admittance. As the medium admitting the sound stimulus changes from less to more dense (i.e. from air to fluid and tissue), the process of admitting pressure is impeded, with a pressure loss of up to 35 dB, a phenomenon called impedance. This conduction of pressure inside the ear is known as acoustic immitance (from im[pedance]+[ad]mitance). In spite of this, the entire hearing apparatus functions in a way that tries to counteract the 35 dB impedance.

Conduction by bone involves vibrations reaching the ear through the movement of the bone section of the skull in which the inner ear is nested. Thus, bone conduction can only happen in the inner ear and beyond. Hearing by bone conduction is admittedly less effective than by air conduction. This is because: (i) the sound stimulus must be very intense to cause vibrations in the skull, and (ii) no matter how intense the sound is, its progress through the outer layers of human body (skin, flesh) to reach the bone, dissipates the vibration eventually reaching the bone. For this reason, bone conduction generally results in sound distortion. Also, hearing by bone conduction is less conscious to us than by air, as when we rest our head on the floor and feel the vibration of footsteps. The reason that our voice sounds different in recordings is because this is how others hear our voice by air conduction. The way we hear our own voice is (i) while we talk: by a combination of air and bone conduction, as the sound is first air-conducted via the cavities of the head and neck and, subsequently, bone-conducted through the skull’s bones and (ii) after we talked, that is, via external air conduction that sends the sound wave of our voice to our ears. This multi-plane process leads to our “distorted” acuity regarding our voice.

Last, sensorineural hearing refers to the part of the auditory track where no conduction takes place; on the other hand, there is transmission of the sound stimulus within and beyond the inner ear via mechanical and electrical impulses to the auditory nerve ultimately reaching the cerebral cortex.

The measurement of hearing

The normal threshold of hearing for humans is between 20 and 30 Hz (hertz, cycles per second) and 0 and 20 dB (decibel, sound level). The normal hearing range is between 20 and 30 Hz and 32,000 Hz and at intensities between 10–90 dB (<30 dB: whisper, leaves rustling, birds chirping; 30–60 dB: conversation; 60–90 dB: car passing, dog barking, baby crying, telephone ringing, loud music; 90–110 dB, discomfort: motorcycle, airplane, train; >110, pain: thunder, explosion; 140 dB: ear damage). The hearing range for speech and language is 500–8,000 Hz. In frequencies above 1,000 Hz, our capacity to hear small changes in frequency decreases, while our capacity to hear high-frequency sounds deteriorates with age. Voiced speech sounds (phones: z, v, j, m, l, o, i) have high frequencies, while voiceless speech sounds have low frequencies (phones: t, s, f, θ). A combination of sounds is called “white noise” when all frequency components have the same average sound level. Hearing sensitivity in normal, impaired hearing or hearing loss is measured by audiometry. Specifically, to test hearing sensitivity, pure tones (simple sinusoidal waves) with different frequencies spanning from low to high, are successively played to a listener’s ear and the listener is expected to indicate when the tone was heard. The average threshold for different frequencies is used to give an estimate of auditory sensitivity or hearing loss. This is referred to as the pure-tone average (PTA) threshold,
where the frequencies used for threshold averaging are listed in abbreviated form, e.g. a PTA threshold obtained at 300 Hz (.3 kHz), 1,000 Hz (1 kHz) and 2,000 Hz (2 kHz) is noted as PTA 512. An audiogram shows the hearing threshold for each frequency established. Based on such PTAs, loss of hearing, indicated by dB, is classified as follows:

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<td>slight</td>
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<td>moderate</td>
<td>moderately severe</td>
<td>severe</td>
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The criteria that determine hearing disability require that the ear functioning better shows one of the two following: (1) a PTA air conduction threshold, for 500, 1,000 and 2,000 Hz, (PTA 512) of 90 dB hearing level or greater, with corresponding bone conduction thresholds, or, (2) a speech discrimination score of ≤40%, at a sufficient level to decide maximum discrimination skill.

**Types and causes of hearing impairment**

Normal hearing refers to the hypothetical average hearing of a young adult (aged 18–22) with no known hearing pathology (infection, disorder). Any considerable deviation from normal ear behavior is considered a disorder, classified under hearing impairment (HI). In lay terms, differentiation in the seriousness of HI has led to two characterizations: hard-of-hearing and deaf, the first denoting variable degrees of loss of hearing sensitivity, and the second denoting complete lack of hearing. HI may be organic or nonorganic. Nonorganic HI has psychogenic (i.e. psychological disorder) origins. On the other hand, impairments resulting from malfunction or damage in the operation of any intercepting link within the hearing apparatus are organic and form four categories: conductive HI (hindrance in the auditory track involving conduction), sensorineural HI (setbacks within the inner ear and auditory nerve), mixed HI (instances of both conductive and sensorineural HI) and finally, central auditory processing disorder (where hearing loss is caused by damage in the neural pathway of sound inside the brain). These impairments are called organic.

HI involves hearing loss (HL), i.e. attenuation of a sound’s strength at variable degrees from slight to profound, as seen before. Further subcategories specify whether one or both ears are affected (unilateral vs. bilateral), whether both ears are equally affected (symmetrical vs. asymmetrical), and the onset of HI compared to language development (prelingual vs. postlingual). Hearing loss may also be described as progressive or sudden, fluctuating or stable, and congenital (present at birth) or acquired (developed later in life).

**Conductive hearing impairment**

Conductive HI (CHI) results from impaired air conduction, though bone conduction is normal. The symptoms include: speaking with a low voice (the patient hears himself well but not environmental noise), hearing speech better in noise contexts because noise has a masking effect
(paracusis willisiana), better tolerance of sounds at discomfort level, loss of sensitivity to all sound frequencies (flat loss), and tinnitus. Likely settings of conductive HI include: blockage of the external meatus by, e.g. cerumen (wax) accumulation, or by tiny objects children play with; in infants born with missing auricle(s) (agenesis) or occluded canals (atresia). Atresia is curable if the inner ear is functioning well. Medical conditions leading to CHI include upper respiratory infections (otitis media) that cause the eardrum to be either distended or retracted, in the second case affecting the patency (opening) of the Eustachian tubes. Another medical condition causing CHI is otosclerosis (the inner ear bone capsule becomes spongy). CHI can in most cases be treated via medication or surgery. CHI is less common than sensorineural HI and rarely leads to deafness; hearing aids and surgical rectification, as necessary, prove beneficial.

Sensorineural hearing impairment

Sensorineural hearing impairment (SHI) refers to hearing loss due to pathology either in the inner ear or the neural track linking the inner ear to the brainstem. Other terms to refer to SHI are perceptive impairment and nerve loss. For SHI to be diagnosed, all conductive hearing is normal, i.e. the sound is conducted by the ear but not processed or perceived. SHI symptoms include: speaking excessively loud (since the patient cannot hear either himself or others) unless the patient has learned to regulate his voice level, better hearing for low frequencies (e.g. for high frequency phones: [f, s, k] in fake, shook, kick, the patient only hears the vowel), difficulty in speech discrimination (even at loudness levels above the hearing threshold), discomfort at loud noise (due to a disproportionate increase of sensation of loudness in the inner ear, when an actual loud sound is heard), and tinnitus that is much higher pitched than in normal or CHI tinnitus (e.g. like a doorbell or phone ringing). SHI is the predominant disorder in infants born with HI, with the exception of congenital atresia mentioned above.

Generally, the causes of SHI may be grouped into congenital and acquired. Congenital causes include heredity (defective genes) and damage to the embryo in utero (due to mother’s diseases, e.g. rubella, in the first three months of pregnancy). Acquired SHI may occur any time in the lifetime as a result of aging, disease, exposure to drugs, or injury, while susceptibility to hearing loss varies across people. Normal loss of hearing, as a result of aging (presbycusis) starts at about 30 years of age, and worsens every following decade. Ménières syndrome (or endolymphatic hydrops) is a disease specific to the inner ear, caused by increased fluid pressure, and it exhibits dizziness (vertigo), hearing loss and tinnitus. The clinician plays an important role in diagnosing Ménières syndrome and referring the patient to an otologist. Diseases causing SHI due to toxicity are influenza, diphtheria, mumps, measles and virus infections.

Exposure to chemicals and drugs may also lead to SHI; in the past, a drug commonly causing hearing loss was quinine, but nowadays dangerous drugs for hearing health are neomycin, kanamycin and dyhydrostreptomysin that are usually reserved for use in life-threatening circumstances only. Other SHI causes, though less common, are abrupt hearing loss, even overnight (as in autoimmune system attacks), and mechanical injury like bone fracture. Trauma, or noise-induced hearing loss (NIHL) is another common cause of SHI. All HI that is not noise-induced is commonly referred to as non-noise-induced hearing loss (NNIHL). NIHL mostly results from sustained exposure to high-frequency/high-intensity sounds, and it is a result of cumulative exposure over several years. Temporary hearing loss (threshold shift) may also occur following short-lived exposure to intense noise though, in this case, normal hearing sensitivity returns. By and large, however, SHI does not on the whole respond well to treatment. When hearing aids are used, they amplify sounds but do little to increase the clarity.
of speech. In such cases, *cochlear implants* (surgically implanted devices) are used to stimulate the hearing nerve which increases the ability to hear sounds and, thus, comprehend speech. Sensorineural abnormality leads to hearing sensitivity loss that is greater by air conduction than by bone conduction. This is because the sound travelling by air conduction is attenuated by the processes taking place in both the middle and the inner ear, while the sound travelling by bone conduction is attenuated only by impairment of the inner ear.

**Mixed hearing impairment**

Mixed hearing loss (MHI) refers to hearing problems demonstrating characteristics from both conductive and sensorineural HI. MHI is not uncommon. A typical example would be an elderly person with presbycusis that also demonstrates conductive hearing loss caused by otitis media. Figure 3.5 is a diagram (see also Martin, 1975, 5) showing all three types of organic hearing impairment discussed above.

**Central auditory processing disorder**

Central auditory processing disorder (CAPD) (or *auditory processing disorder, central loss*) is caused by interferences in the neural pathways from the brainstem to, and including, the temporal lobes of the cerebral cortex. Patients with CAPD exhibit difficulties *comprehending* what is heard (*auditory imperception*) rather than actually hearing the sound stimulus. Loss

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![Diagram of normal and impaired hearing](image-url)  
*Figure 3.5* Normal and impaired hearing.  
Source: Courtesy S. Qiouyi Lu.
of hearing sensitivity is less easily diagnosed in CAPD than in conductive and sensorineural HI. Central loss may also result from lesions in the brain. If CAPD occurs after language has been developed, lesions are not identifiable through routine audiometry, because the ears will respond normally to the demands of these tests, although the patient cannot recognize what he hears. The causes of CAPD may also be brain tumor(s), an abscess, brain damage from trauma, vascular (vein) changes in the brain (e.g. arteriosclerosis), infections (e.g. meningitis, encephalitis), degenerative diseases (e.g. Parkinson’s, multiple sclerosis), erythroblastosis fetalis (a hemolytic disease resulting from blood-group compatibility between mother and fetus), etc. It should be emphasized, further, that the hearing loss discussed in CAPD is not the same as the one discussed for peripheral and internal HI, in the sense that CAPD is a neurological disorder rather than a disorder of the hearing mechanism per se. As a neurological disorder, it has similar symptoms to those of auditory agnosia (ability to hear but not recognize sounds) and auditory verbal agnosia (hearing but not recognizing speech sounds). Auditory agnosia is a type of receptive aphasia (affecting language comprehension), associated with injury of the dominant cerebral hemisphere. Congenital aphasia in children is characterized by auditory imperception, visual imperception, or both; in such cases, children have typical intelligence and hearing sensitivity, but cannot develop language. Although the treatment of CAPD falls within the field of neuropsychology, a speech clinician needs to be able to differentiate between peripheral hearing and central hearing disorders for diagnosis purposes.

Nonorganic hearing impairment

Nonorganic (or functional) hearing impairment (NHI) is instigated by psychological factors, like emotional stress, as a defense against the painful situation. Other terms for NHI are conversion deafness and hysterical deafness. In such cases, the patient is truly convinced that he has suffered hearing loss despite the fact that his hearing apparatus is intact. Similarly to CAPD, hearing loss in functional hearing impairment is not a loss that relates to breakdown of the hearing mechanism, and it requires referral to a psychologist.

Conclusion

A person with normal hearing is always in the midst of a noisy milieu. To a large extent, we are all unaware or less conscious of the constant array of sounds that surround us. Thus, while reading something or having a conversation, we may not pay attention to other noises in the environment like a car going by, the sound of the refrigerator, a TV playing in another room, or the sound of keys being struck when typing. This type of unconscious primitive hearing is taken for granted, but once we are removed from this setting of familiar noise, uneasiness is immediately felt. It is plainly understood, then, how hearing impairment or loss can place patients at a distinct disadvantage. By elaborating on what constitutes normal and impaired hearing, the present chapter has served to highlight the significance of the ability to hear for general quality of living. The chapter is presented to the speech clinician reading this volume as a comprehensive preface to the substantative study of auditory phonetics (part of audiology), a field that verges on speech language pathology, medicine, psychology, physics and linguistics.
Auditory phonetics

References


