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COLLEGE OF GRADUATE STUDIES AND RESEARCH

ESTIMATED PREVALENCE OF COCHLEAR DEAD REGIONS IN AN AUDIOLOGICAL CLINIC POPULATION AND THE ASSOCIATED HEARING AID SUCCESS

By

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AUDIOLOGY DOCTORAL THESIS APPROVAL PAGE

This is to certify that the Audiology Doctoral Thesis prepared by Sara Brittingham, entitled Estimated Prevalence of Cochlear Dead Regions in an Audiological Clinic Population and the Associated Hearing Aid Success, has been approved by this committee as satisfactory completion of the Audiology Doctoral Thesis requirement for the degree Doctor of Audiology (Au.D.) in the department of Audiology, Speech-Language Pathology, and Deaf Studies.

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ABSTRACT

ESTIMATED PREVALENCE OF COCHLEAR DEAD REGIONS IN AN AUDIOLOGICAL CLINIC POPULATION AND THE ASSOCIATED HEARING AID SUCCESS

Sara Brittingham

It is unknown how many patients in a typical caseload have audiometric findings associated with cochlear dead regions (Moore, 2004). The purpose of this study was to (1) examine a university clinic population and estimate the number of patients with suspected cochlear dead regions and (2) examine the hearing aid success for patients with this profile compared to patients without this profile.

Results indicate a profile suggestive of cochlear dead regions in 11.4% of the patients. Mean word recognition scores (WRS) for patients with suspected cochlear dead regions were significantly poorer for patients with the dead-zone profile compared to patients without this profile, but with similar pure tone average. There was no significant difference between the number of post-fitting hearing aid visits to the clinic between groups and there was no significant difference in the number of returned hearing aids between these two groups.

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CHAPTER 1

INTRODUCTION

Moore (2004) defined cochlear dead regions as places in the auditory system where there are no functioning inner hair cells and/or auditory neurons. These regions are identified by the characteristic frequencies that correspond to the location of damaged inner hair cells and/or the neurons immediately adjacent to the dead region (Moore, 2001). The measurement of psychophysical tuning curves (PTCs) and the Threshold Equalizing Noise (TEN) test are two methods used to identify cochlear dead regions. When a cochlear dead region is present, living regions in close proximity to the dead region may respond and result in a pure-tone audiogram that gives the false impression that hearing exists in regions that are not active, particularly if the dead regions are located in the apex of the cochlea (low-frequency) (Moore, 2002a).

Halpin (2002) discussed hypothetical tuning curve audiograms that reflect damaged regions within the cochlea that may be mistakenly interpreted as regions with residual hearing. He suggested pure tone audiograms showing similar configurations should be interpreted with caution and that the hypothetical tuning curve audiograms may be useful as a guide in distinguishing healthy from damaged regions within the cochlea. Information regarding the patient's speech recognition ability might be used as a secondary indicator when examining the status of specific auditory regions. If dead regions are suspected based on the audiometric pattern and speech recognition test

results, hearing aid strategies can be focused on frequency regions of the cochlea capable of contributing to speech intelligibility, rather than on areas that are most likely dead. Without this consideration, hearing aid gain may be focused on regions incapable of assisting with hearing and potentially interfering with hearing due to the spread of masking.

The presence and extent of these dead regions will impact the benefits and overall success of a hearing aid (Moore, 2001). Various hearing aid fitting strategies have been suggested for patients with cochlear dead regions including use of compression hearing aids, use of the NAL-NL1 fitting rationale, and use of transposition hearing aids (Moore, 2001); however, research in the area of hearing aid fitting outcomes for patients with cochlear dead regions is limited. In addition, it is unknown how many patients on a typical caseload have audiometric findings associated with cochlear dead regions. This study examined a large patient population and estimated the number of patients with an audiometric profile suggestive of cochlear dead regions. Further, the hearing aid success was examined for patients with this profile and compared with the hearing aid success for patients with similar pure tone average, but without the characteristic pattern associated with dead regions.

CHAPTER 2

REVIEW OF LITERATURE

Anatomy and Physiology

Outer and middle ear. The peripheral auditory system consists of three parts: the outer ear, middle ear, and inner ear. The outer ear is made up of the pinna and the external auditory canal. The primary function of the outer ear is to collect and direct sound waves toward the tympanic membrane (Rappaport & Provençal, 2002). The outer ear also serves a secondary function by enhancing auditory stimuli in the range of 2000 to 7000 Hz, as a result of the resonance characteristics of the pinna and external auditory canal (Shaw, 1974). Additionally, spectral changes to sound created by physical characteristics of the outer ear provide useful information related to sound localization (Hebrank & Wright, 1974).

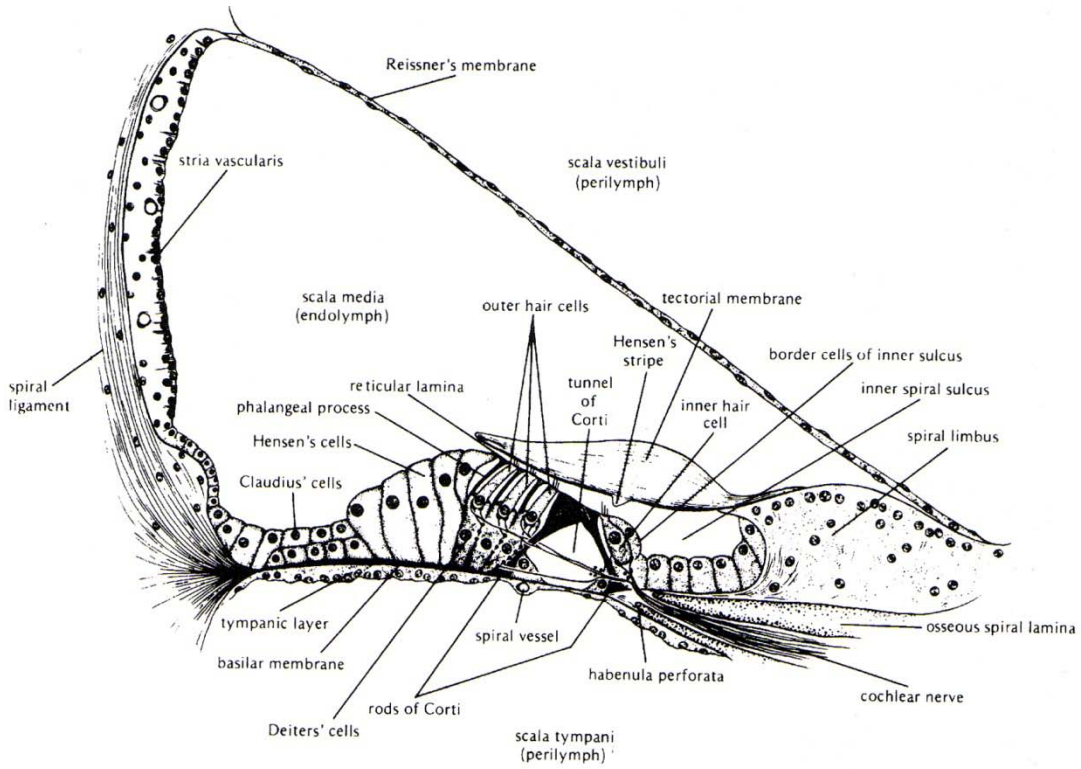
The middle ear is comprised of the tympanic membrane, the tympanum, and the middle ear bones, the malleus, incus, and stapes, known as the ossicles. The ossicles are attached to the tympanic membrane at the lateral end and vibrate in response to tympanic membrane vibrations (Tonndorf & Khanna, 1970). On the medial end, the ossicular chain, specifically the stapes bone, articulates with the oval window membrane of the inner ear. Vibration of the ossicular chain results in displacement of the stapes toward the inner ear, thus creating motion of fluids within the inner ear (Bekesy, 1960). The impedance of the fluids of the cochlea is much greater than the impedance of air. Due to this impedance mismatch, the middle ear system must act as a transformer to allow

efficient transmission of sound energy to the cochlea (Bekesy, 1960; Moller, 1965). The middle ear is able to efficiently transmit sound energy as a result of three mechanisms. These include the difference in surface area between the tympanic membrane and the stapes footplate, the curved buckling mechanism of the tympanic membrane, and the lever action of the ossicles (Moller, 1965).

Inner ear anatomy. The inner ear can be classified into two separate portions, an osseous or bony portion and a membranous portion. The bony outer wall of the cochlea is located within the petrous portion of the temporal bone (Rappaport & Provençal, 2002). A membranous channel, known as the membranous labyrinth, spirals within this bony labyrinth (Davis, 1962). The inner ear contains both vestibular and cochlear sense organs. The vestibular portion of the inner ear is responsible for the sense of balance and will not be discussed in this review.

The cochlear portion of the inner ear is a fluid-filled, spiral-shaped organ. The osseous spiral lamina of the cochlea makes approximately 2 turns around the central core of bone called the modiolus. It is divided into three sections: the scala vestibuli, scala media, and scale tympani, as shown in Figure 1 (Yost, 2000). Reissner's membrane separates the scala vestibuli from the scala media, while the basilar membrane separates the scala media from the scala tympani. The scala vestibuli and scala tympani contain a fluid known as perilymph, which is rich in sodium, and the scala media contains a fluid known as endolymph, which is rich in potassium (Rappaport & Provençal, 2002). The basilar membrane runs the entire length of the scala media. It is narrow and stiff at the basal end of the cochlea and is wider and more flaccid toward the apical end. The Organ

Figure 1. Cross-section of the cochlea.



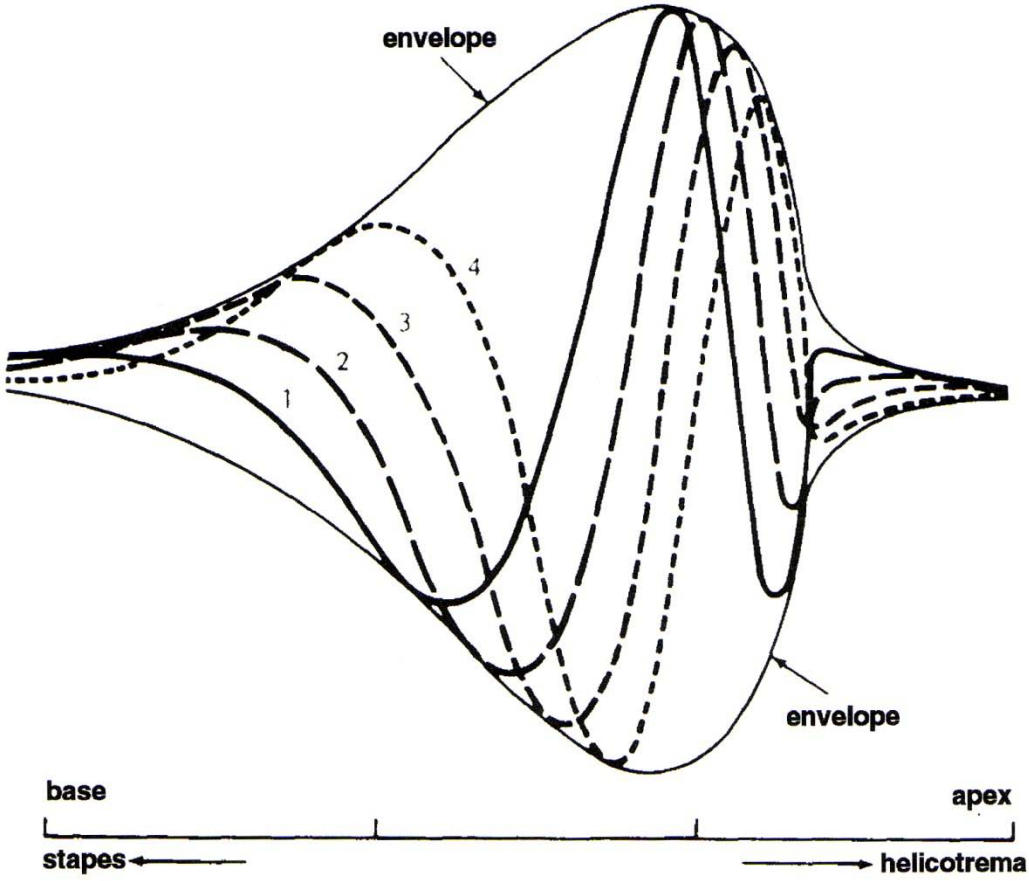
Note. From *Fundamentals of Hearing* (p. 84), by W.A. Yost, 2000, San Diego: Academic Press.

of Corti is located on the basilar membrane. The Organ of Corti is divided along its entire length into inner and outer sections by pillars or rods of Corti, which form a support structure in the shape of a triangle (Lim, 1986). The inner ear contains approximately 3500 IHCs and 12,000 OHCs. The inner hair cells (IHCs) are found in a single row medial to the Tunnel of Corti. The outer hair cells (OHCs) are found in three rows lateral to the IHCs. Small, microscopic projections (hairs) called stereocilia can be found at the apical end of each OHC (Davis, 1962). The stereocilia extend upward through the cuticular plate, a thickened region on top of each hair cell. The reticular lamina is formed by the cuticular plates of the hair cells and isolates the underlying structures of the Organ of Corti from the scala media (Davis, 1962).

Inner Ear Physiology

The traveling wave theory. As previously discussed, the vibrating motion of the ossicular chain results in the stapes moving in and out of the oval window, thus displacing the fluid adjacent to the window. The result of this oscillation within the inner ear was first described by Bekesy and is known as the traveling wave theory. This theory provides an explanation for the main way the cochlea codes the frequency of the sound waves reaching the cochlea (place coding). Because the basilar membrane widens and becomes more flaccid from base to apex, variations in the width and stiffness along the basilar membrane cause different frequency sounds to reach their points of maximum displacement at different points along the membrane (Bekesy, 1960). If the vibrations of the basilar membrane motion are examined across time, the traveling wave pattern can be illustrated via the traveling wave envelope, illustrated in Figure 2 (Yost, 2000). The

Figure 2. Traveling wave envelope.



Note. From *Fundamentals of Hearing* (p. 97), by W.A. Yost, 2000, San Diego: Academic Press.

traveling wave envelope is characterized by a gradual increase in amplitude along the basilar membrane, a peak amplitude at a certain point, and a rapid decrease in amplitude immediately following the peak (Bekesy, 1953). The separation of a complex signal into multiple points of basilar membrane displacement is evidence that the basilar membrane is performing an early spectral analysis of the signal, i.e. a separation of sound in the frequency domain. High frequencies cause displacement of the basilar membrane in the basal region, while low frequencies cause the greatest basilar membrane displacement in the apical region (Gelfand, 2001).

The basilar membrane also plays a role in the coding of the intensity and temporal characteristics of the stimulus (Bekesy, 1953). Higher intensity stimuli create greater amplitude basilar membrane displacement and a wider traveling wave envelope. The temporal pattern of the stimulus is also conveyed through the timing of the vibration of the basilar membrane (Bekesy, 1947). Specifically, the basilar membrane displaces, or moves up and down, synchronously with the vibration of the stimulus. Information related to the frequency, intensity, and temporal characteristics of the stimulus beginning at the basilar membrane is continued at the level of the hair cells.

The basilar membrane is attached along the medial and lateral borders by the osseous spiral lamina and the spiral ligament, respectively. The traveling wave causes displacement and creates longitudinal and radial force along the basilar membrane, resulting from the attachment of the spiral lamina and the spiral ligament. The radial force of the traveling wave causes the stereocilia of the OHCs to bend away from modiolus of the cochlea during excitation (Dallos, Billone, Durrant, Wang, & Raynor, 1972). The stereocilia of the hair cells are firmly embedded in the reticular lamina.

When the basilar membrane moves, the tectorial membrane and the reticular lamina move in opposite directions, creating a shearing force, which causes the stereocilia to bend (Zwislocki, Chamberlain, & Slepecky, 1988). The bending of the stereocilia in an excitatory direction activates the hair cells, if the force is sufficient enough in magnitude to cause electrochemical changes within the hair cells. With sufficient enough stimulation, the hair cell will release a neurotransmitter from its base, potentially causing an excitation of the auditory neurons directly below the hair cell (Dallos et al., 1972). In order for this process to work, the tallest stereocilia of the outer hair cells must provide a strong connection between the hair cell and the tectorial membrane. The stereocilia must be strong, resilient, and resistant to breakage in order to function for an extended period of time (Yost, 2000).

The inner and outer hair cells. The inner hair cells (IHCs) and outer hair cells (OHCs) differ in both form and function. The IHCs are flask-shaped, while the OHCs are tube-shaped. The IHCs and OHCs contain various cell organelles, including mitochondria, necessary for metabolic activity. In addition, the OHCs contain contractile proteins called actins that are structurally similar to muscle cells (Gelfand, 2001). These contractile proteins allow the OHCs to expand and contract, similar to muscle cells. The expansion and contraction, which affects the length of the OHC, is known as motility (Brownell, Bader, Betrand, & de Ribaupierre, 1985). There are two types of OHC motility: fast motility and slow motility. Fast motility of the OHCs occurs approximately 0.15-0.2 msec following hair cell stimulation. Slow motility occurs several seconds following hair cell stimulation and is initiated through a biochemical process, described in the next paragraph (Brownell et al., 1985). The motility of the OHCs is also affected

by the action of efferent fibers that originate from the brainstem and terminate below the OHCs. Because the signal originates at a location removed from the ear (e.g. the brainstem), this OHC motility process is considered slow (Yost, 2000).

The concept of an active cochlear mechanism was developed in the 1970's and early 1980's. This concept is based on the idea that normal cochlear function is dependent on an active, mechanical feedback process created at the level of the OHCs (Dallos, 1992). The ability of the OHCs to change shape alters the micromechanical process within the cochlea; specifically, the expansion and contraction of the OHCs affects the amplitude of the traveling wave within the Organ of Corti (Dallos, 1992). The electromotile response during acoustic stimulation is caused by a receptor potential produced by the OHCs. Changes in the length of the OHCs resulting from alternating depolarization and hyperpolarization deliver a mechanical force (increasing the amplitude) to the basilar and tectorial membranes, while changes in the length of the OHCs resulting from direct current potentials may alter the effective stiffness of the basilar membrane (Dallos, 1988).

The IHCs are the transducers of sound energy, as evidenced by the large number of afferent auditory nerve fibers innervated at the level of the IHCs (Yost, 2000). It is estimated that approximately 95% of afferent auditory neurons innervate the IHCs, while only 5% of the afferent auditory neurons innervate the OHCs. The opposite is true of the efferent fibers. Approximately 95% of the efferent fibers synapse with the OHCs, while only 5% of the efferent fibers synapse with the IHCs (Gelfand, 2001). Damage to either the IHCs or OHCs may severely compromise the transduction of auditory stimuli. (Kiang, Liberman, Sewell, & Guinan, 1986).

Activation of the hair cells is an electrochemical process, resulting in electrical signals called receptor potentials (Davis, 1983). These potentials include the cochlear microphonic and the summating potential.

The cochlear microphonic, or "cochlear amplifier", is an alternating current potential that occurs only in the presence of acoustic stimulation (Yost, 2000). It was first recorded by Wever and Bray (1930) and was originally misidentified as a neural potential. Later studies found the cochlear microphonic was a cochlear potential that is present over a wide range of sound levels and is able to reflect both sound intensity and frequency. The origin of the cochlear microphonic is thought to be the stereocilia of the OHCs (Tasaki, Davis, & Eldredge, 1954). The summating potential is a direct current potential that, similar to the cochlear microphonic, only occurs only in the presence of acoustic stimulation. The presence of a stimulus creates a baseline shift in cochlear potentials, in either a positive or negative direction. There is evidence to suggest that the summating potential is actually composed of several different potentials that interact in a complex manner (Dallos, Popper, & Fay, 1996). It is theorized the increase in potassium (K+) and influx and output of various other chemicals are the cause of the summating potential (Fay & Popper, 1992).

Neurons produce electrical charges called action potentials. Action potentials are an all-or-none phenomenon (Rappaport & Provençal, 2002), meaning the nerve will "fire" a chemical charge down the length of the cell only after the change in voltage across the cell membrane has reached the threshold of the cell. The threshold is the smallest voltage change that will elicit an action potential from the neuron. Auditory neurons also have a dynamic range. The dynamic range is the range of intensities in

which the neural firing rate increases with each increase in intensity. The neuron will eventually reach saturation, whereby the firing rate will not increase even with an increase in stimulus intensity. The variation in threshold and dynamic range among neurons allows the auditory system to respond to a wide range of stimuli (Liberman, 1988).

The Auditory Neural Response

Auditory neurons transmit the place and time coded information from the cochlea to the central auditory nervous system. Tuning curves describe the threshold of response of auditory nerves to stimuli of various frequencies. The characteristic frequency (CF) of each nerve fiber is shown as the point of each tuning curve and it is the frequency where the auditory neuron fires at its lowest threshold. Auditory neurons will typically respond to frequencies below their CF but rarely respond at frequencies much higher than their CF (Liberman, 1982). This function of the auditory nerve preserves the frequency selectivity found at the level of the basilar membrane and traveling wave (Yost, 2000).

Damage to the hair cells within the cochlea usually results in elevated thresholds. Damage to the OHCs impairs the active mechanism of the cochlea. This results in a reduction in basilar membrane vibration for low sound levels. IHC damage results in a decreased capacity of the hair cell to transmit basilar membrane movements into electrochemical potentials. This means that the amount of basilar membrane vibration needed to reach thresholds in increased (Moore, Huss, Vickers, Glasberg, & Alcantara, 2000).

Cochlear Dead Regions

Moore (2004) defined cochlear dead regions as places within the cochlea containing no functioning IHCs and/or auditory neurons. Basilar membrane vibration will not result in electrochemical signal transduction if IHCs and/or auditory nerve fibers are absent or non-functioning. In certain cases, IHCs and/or neurons may be present, but functioning poorly. These areas would not be considered dead by definition; however, a stronger than normal signal would be required for threshold detection. High intensity stimuli may lead to off-place listening and a region of the cochlea may be considered functionally dead if a place in the cochlea is damaged such that tonal stimuli are detected via off-place listening. Off-place listening is defined as the detection of a specific tone by IHCs and neurons with characteristic frequencies different from the tone of interest. Off-place listening may occur in normal hearing people, particularly when one sound must be detected in the presence of another. It occurs with greater frequency in ears with damaged auditory systems, specifically with cochlear dead regions (Moore, 2001).

It is estimated that detection thresholds for pure tones are unaffected until the loss of IHCs and/or neurons exceeds 80-90% in a specific frequency region. However, the discrimination of sounds, including speech stimuli, may be affected by an IHC and/or neuronal loss exceeding 50% (Moore, 2004). This may result in the need for a less strict definition of cochlear dead regions. Cochlear dead regions may be more loosely defined as regions in the cochlea where IHCs and/or neurons are functioning poorly enough that a stimulus presented to that region is detected by off-place listening (Moore, 2004).

Dead regions may be identified by the range of characteristic frequencies of the IHCs or neurons typically associated with the region (Moore et al., 2000). There are

several problems associated with use of this definition, as identified by Moore (2004). Damage to outer hair cells (OHCs) may lead to shifts in characteristic frequency of the basilar membrane relative to "normal values" (Liberman & Dodds, 1984); therefore, the tuning of the basilar membrane, IHCs, and auditory neurons may be abnormal in an ear with a dead region, even over regions that are not dead (Moore, 2004). Dead regions can also be identified by the characteristic frequencies of the IHCs and neurons immediately adjacent to the dead region (Moore & Alcantara, 2001). This method of identification remains useful even if the characteristic frequencies of the IHCs and neurons are shifted, as a result of auditory system damage (Moore, 2004).

Identifying Cochlear Dead Regions

Psychophysical tuning curves. The measurement of psychophysical tuning curves (PTCs) is one method of identifying cochlear dead regions. Dead regions are identified through use of simultaneous masking to measure PTCs (Moore, 2002a). The signal for the measurement of PTCs is fixed in both frequency and in level. The stimulus level is typically just above absolute threshold, typically at 10 dB sensation level, while the masker may be either sinusoid or narrow band. A narrow band is typically used in an effort to reduce the influence of beats between the masker and the stimulus signal. The level of the masker required to sufficiently mask the signal is determined at each of the several masker frequencies. The tip of the PTC represents the frequency at which the masker level is the lowest (Moore, 2004). In ears without cochlear dead regions, the tip of the PTC always lies close to the signal frequency (Moore, 1978). When there is no cochlear dead region present, both in individuals with normal hearing and hearing loss, the tip of the PTC lies close to the signal frequency. In contrast, those with cochlear dead

regions typically have PTCs with tips shifted well away from the signal frequency. This occurs when the signal frequency falls within a dead region (Moore & Alcantara, 2001). It is assumed that the signal frequency is detected via off-place listening. In ears with a cochlear dead region, the most effective masking level occurs at the point of maximum basilar membrane displacement and the signal is detected (Moore, 2004). The tip of the PTC is assumed to represent the boundary of the dead regions (Moore & Alcantara, 2001). It should be noted that the frequency at the tip of the PTC may not always provide an accurate estimate of the boundary, particularly if the shift is relatively small, usually less than a few hundred hertz (Hz) (Moore, 2002a).

Limitations of PTCs. PTCs are considered to be the "gold standard" for identifying cochlear dead regions (Summers, Molis, Musch, Walden, Surr, & Cord, 2003). There are several factors, however, that may complicate the interpretation of PTCs. One factor is known as combination tone detection. In some cases, a combination of the signal tone and the masker may be more easily detected than the signal itself. In normal hearing individuals, two types of combination tones may be detected; the first is known as the cubic difference tone, or $2f_1$-f_2. This combination tone appears to reflect nonlinearity within the cochlea, which is dependent on the active mechanism. In normal hearing individuals, this combination tone may affect the masked thresholds if the signal lies just above the masker frequency. However, the audibility of this combination tone is greatly reduced by cochlear damage. The cubic difference tone appears to have little effect on the masked thresholds of hearing-impaired individuals (Moore, 2004).

A second combination tone is known as the simple difference tone, or simply f_1-f_2. This combination tone is usually only detectable when the primary tones are at high sound levels. The simple difference tone may result from distortion produced within the middle ear (Helmholtz, 1954). This tone may affect hearing-impaired individuals when primary tone frequencies fall in an area of substantial hearing loss, while the combination tone frequency is contained in an area of near-normal hearing (Alcantara & Moore, 2002). Detection of the simple difference tone may result in a PTC with a sharp tip at the signal frequency, even when the signal frequency is contained with a cochlear dead region. PTCs will most often be affected when individuals have relatively good low-frequency hearing combined with poor high-frequency hearing (Moore, 2004).

Beat detection is another factor that can influence the shape of PTCs. When the signal and masker are close in frequency, the interaction between the signal and masker creates an audible amplitude fluctuation that occurs at a rate corresponding to the difference in frequency between the two stimuli. This interaction results in the creation of beats. Beats are most readily detectable when both the masker and the signal are sinusoids. Beats are less easily detected when the masker signal is narrowband noise. This occurs because of the random amplitude fluctuation of the noise that makes the detection of beats more difficult. Beats provide detection cues requiring a greater masker level for PTCs. Beat detection can result in a minimum threshold in the PTC at the signal frequency even if the signal frequency is with a dead region (Moore, 2004).

Recommended procedures when using PTCs. To reduce the influence of errant tone detection and beat detection, several procedures are recommended when measuring PTCs. If the individual has better hearing for low frequencies and poorer hearing at high

frequencies, low pass noise should be used in addition to the standard masking signal to reduce the influence of the simple difference tone. The masking signal should have a large bandwidth to minimize the effects of beat detection. It should be noted, however, that use of a very large bandwidth may result in a loss of frequency resolution which makes determining the exact tip frequency more difficult (Moore, 2004). A noise bandwidth of 320 Hz is recommended for signal frequencies of 2000 Hz and above, as it should not result in a significant loss of resolution (Glasberg & Moore, 1990). However, for signal frequencies below 2000 Hz, a bandwidth of 16-20% of the signal frequency is recommended (Glasberg & Moore, 1986). If a high-frequency dead region is suspected, the signal frequency should be chosen as high as possible. However, the signal level at absolute thresholds should not exceed uncomfortable loudness levels. A practical guideline for absolute thresholds is approximately 90 dB SPL (Summers et al., 2003).

PTCs are a useful method for detecting cochlear dead regions and determining their boundaries. Because PTCs are time consuming to calculate and the choice of appropriate signal frequencies and masker is difficult, PTCs are rarely used in clinical practice (Moore, 2002a; Moore, 2002b).

Threshold equalizing noise test. The threshold equalizing noise (TEN) test is a procedure developed by Moore et al. (2000) for detecting the presence of one or more cochlear dead regions. The TEN test is a much more time-efficient method of identifying cochlear dead regions than the PTC. For this reason, the TEN test may be more suitable for identifying dead regions during a clinical evaluation (Summers et al., 2003). The TEN test is relatively quick and easy and requires tone detection in the presence of spectrally shaped masking noise across a frequency range of 250-10000 Hz. The

intensity level of the noise is described in terms of Equivalent Rectangular Bandwidth (ERB), or center frequency, of the auditory filter. The ERB function is used to derive filter shapes and bandwidths (Glasberg & Moore, 1990). The average ERB for normal hearing listeners occurs at moderate sound levels, specifically one ERB (132 Hz-wide) band centered at 1000 Hz. The signal level of the masked thresholds is essentially the equivalent to the noise level/ERB.

For listeners with normal hearing, the masked threshold for tone detection is equal across all test frequencies (Summers et al., 2003; Moore, 2004). Listeners with hearing impairment but with functioning IHCs and neurons may have slightly elevated thresholds for the TEN Test, when compared to normal hearing listeners. Thresholds for tonal signal of listeners with normal hearing or hearing-impairments with no dead regions are typically within 5 dB of the level/ERB of the TEN (Glasberg & Moore, 1990). For listeners with dead regions, the TEN test shows increased masking thresholds of at least 10 dB above the level/ERB for frequencies associated with dead regions (Moore et al., 2000). When cochlear dead regions are present, a stimulus is detected by IHCs and the associated neurons with CFs different from that of the stimulus frequency; in other words, the stimulus is detected by off-place listening. It is for this reason that the TEN threshold is expected to be higher (Summers et al., 2003).

Advantages of the TEN test. The TEN test has several advantages over the measurement of PTCs. The TEN test is quick to administer. It can be administered in approximately twice the time it takes to conduct an audiogram, if a single noise level is used. A new version of the TEN has recently been introduced where the levels are estimated in dB HL, rather than SPL. The new version, known as the TEN (HL) test,

takes approximately the same amount of time as an audiogram because the absolute thresholds do not need to be recalculated. The procedure for the administration of the TEN (HL) test is similar to that of an audiogram, which is familiar to audiologists (Moore, 2004). The noise level/ERB_N and the test tones correspond to the levels indicated on the audiometer. ERB_N refers to the equivalent rectangular bandwidth of the auditory filter determined for young, normal-hearing listeners at moderate sound levels (Munro, Felthouse, Moore, & Kapadia, 2005). This makes the test simpler to administer and reduces the chance of error (Moore, Glasberg, & Stone, 2004). The results of the TEN test are not affected by the detection of beats between the signal stimulus and the masking stimulus or by the detection of a combination of tones (Moore, 2004). The new version of the TEN test also has a restricted noise bandwidth, which allows the noise level/ERB_N to be increased without distortion. This also reduces excessive loudness and avoids the possibility of further damage to hearing (Moore et al., 2004).

Limitations of the TEN test. The TEN test has several limitations. The TEN test criteria were developed using a small sample size of adults with moderate to severe cochlear hearing loss (Moore et al., 2000). Therefore, the criteria may not be appropriate for use with other populations, such as those with profound hearing loss or younger individuals (Moore, Killen, & Munro, 2003). The TEN test may only provide a rough estimate of the edge frequency of the dead region due to the spacing of the test frequencies at one-half octave intervals (Summers et al., 2003). The TEN test is also dependent on the presence of some residual frequency selectivity in the cochlea. For individuals with severe to profound loss at certain frequencies, limitations of the audiometer may not permit the TEN signal to be made intense enough to produce 10 dB

of masking. In cases such as this, the results of the TEN test must be classified as inconclusive (Moore et al., 2003). The most recent version of this test, the TEN (HL) test, allows higher noise levels to be used due to reduced noise bandwidths and minimal amplitude fluctuations. The results of the TEN test may vary depending on which earphone is used to administer it. The TEN test fails to take into account the non-flat frequency response of the earphone at the eardrum which may result in various outcomes depending on the type earphone that is used. The most recent version of this test, the TEN (HL) test, does allow for this difference, but only includes test frequencies from 500 to 4000 Hz, thus limiting the detection of dead zones at 250 Hz and 6000-8000 Hz (Summers et al., 2003).

Use of the TEN test in a clinical setting. Moore (2004) suggested that the TEN test should only be conducted when there are one or more reasons to suspect the presence of a cochlear dead region. Information gathered from the TEN test may provide useful information for the hearing aid fitting process. Audiogram shape and severity of loss may be indicators for the presence of a dead region. Dead regions are commonly associated with the following audiometric configurations: a hearing loss more than 90 dB at high frequencies and 75-80 dB at low frequencies, a hearing loss of 40-50 dB at low frequencies with near normal hearing at mid- and high frequencies, a hearing loss greater than 50 dB at low frequencies with less hearing loss at higher frequencies, or a rapidly increasing hearing loss (more than 50 dB/octave) with increasing frequency. Subjective reports of patients that pure tones used in audiometry sound more like noise than tones may also be an indicator that the pure tone used may fall within a dead region. A final

indicator of a possible cochlear dead region is a previous lack of benefit from hearing aids (Moore, 2001).

The Effect of Cochlear Dead Regions on the Audiogram

The audiogram is a graphical summary of air and bone conduction thresholds. When a cochlear dead region is present, the audiogram may give the false impression of residual hearing at frequencies contained within the dead region. The audiogram may indicate only a moderate hearing loss at a particular frequency or frequency region, when the loss is actually complete; this is especially likely if there are healthy cells along the borders of the dead regions because the hair cells and neurons adjacent to the dead zone respond to the stimulus (Moore, 2004). A steeply sloping audiogram may be used as preliminary evidence for a high-frequency dead region and further testing should be implemented; however, dead regions may also be present when the audiogram has a shallow slope or a moderate slope suggesting that the slope of the audiogram is not a reliable indicator of the presence or absence of cochlear dead regions (Moore, 2002a; Moore, 2004).

Damage to the hair cells of the cochlea may result in elevated absolute thresholds in two ways. Dysfunction of the OHCs impairs the active mechanism of the cochlea, causing reduced basilar membrane vibration for low sound intensity level inputs, while dysfunction of the IHCs results in reduced transduction efficiency. This means that the amount of basilar membrane vibration needed to reach threshold is increased. Dysfunction of the IHCs may be caused by several factors, including sporadic, or even complete, death of the IHCs, metabolic disturbance, structural problems, or combination of these factors (Moore, 2001).

Psychophysical data obtained from animal and human patients suggest that the maximum gain provided by the active mechanism is approximately 50 dB for low-frequency inputs and approximately 65 dB for high-frequency inputs. This suggests that a hearing loss greater than 50 dB at low frequencies and greater than 65 dB at high frequencies is not the result of OHC damage alone; the IHCs must also be involved (Moore, 2001). Moore (2001) also suggested that absolute thresholds between 0 and 65 dB for high frequencies, or 50 dB for low frequencies, are assumed to be the result of only OHC dysfunction or by a combination of OHC and IHC dysfunction. Absolute thresholds greater than 65 dB for high frequencies, or greater than 50 dB for low frequencies, are assumed to be the results of a combination of OHC and IHC dysfunction, provided there is no retrocochlear involvement. Absolute thresholds greater than 90 dB, or between 75 and 80 dB at low frequencies, are likely associated with dead regions in the cochlea (Moore, 2001).

This is also evidenced by Yates (1990) who estimated that as the threshold value for IHCs exceeds approximately 30 dB, it could be assumed that a dead region is present in the IHCs. It is also assumed that the maximum hearing loss contribution of the OHCs is approximately 65 dB for high frequencies; therefore, hearing losses greater than 95 dB can be likely associated with the presence of cochlear dead regions for both IHCs and OHCs (Yates, 1990; Yates, 1995). The results of Moore et al. (2000) were consistent with these findings and found evidence to support that hearing losses greater than 85 dB are almost always associated with cochlear dead regions.

The presence or absence of cochlear dead regions is even more unpredictable in individuals with severe to profound hearing loss of congenital origin. Congenital hearing

loss may be caused by factors such malformation of the structures within the cochlea. These malformations may be associated with abnormal vibration patterns within the cochlea. As a result, the shape of the audiogram is an unreliable indicator of the presence of cochlear dead regions for those with congenital hearing loss (Moore, 2002a).

Later research by Summers (2004) suggested methods of identifying cochlear dead regions, including PTCs and the TEN test, did not improve clinical decisions regarding amplification for individuals with hearing loss, when compared to information gathered directly from the audiogram. Amplification was not recommended at frequencies where the hearing loss exceeded 90 dB for the same listeners and for similar frequency regions, as indicated by specific testing for cochlear dead regions. The results of this study suggest that use of absolute thresholds can provide audiologists with adequate information for decisions regarding amplification and that more time consuming methods, such as the TEN Test, are not necessary.

Tuning curve audiograms. Due to the conflicting reports regarding the usefulness of the pure-tone audiogram for the identification of cochlear dead regions, Halpin (2002) suggested the use of hypothetical tuning curve audiograms to detect damaged frequency regions within the cochlea. Normal tuning curves demonstrate very sharp tips at the characteristic frequency of the cell being examined. The entire tuning curve is rarely seen in the clinical setting because it would be shown in a pure-tone audiogram only if one functioning region in the cochlea were present and all adjacent cells were dead; however, cases demonstrating both normal thresholds and steep slopes are not uncommon. Dead zones can occur between these two extremes.

Frequency tuning curves have characteristic shapes that are easily recognizable by students of basic psychoacoustics. Because acoustic energy presented to the basilar membrane spreads most effectively from apex to base, the low-frequency slope is a result of damping limitations of the cochlea (Eagan & Hake, 1950). This means that surviving regions of the cochlea are isolated by no more than about 55 dB on the low-frequency side. The cochlea is better damped in the regions associated with high-frequency stimuli, which results in steeper slopes in the high-frequency direction (Kiang & Moxon, 1974). When identifying cochlear dead regions, a steep high-frequency slope, at the frequencies of 2000 and 4000 Hz, is a feature indicating a possible tuning curve tail; however, cochlear dead regions may also be found at 500 and 1000 Hz, where the slope of the possible tuning curve tail will be much less steep. When a cochlear dead region is suspected, the clinician must determine whether the cochlear region along the slope represents the thresholds of attenuated, but functioning, hair cells or thresholds of adjacent hair cells capable of responding to that frequency (Halpin, 2002).

Cochlear dead regions and speech recognition. Small groups of nerve cells may be responsible for responding to pure tone tests; however, word recognition tasks are highly dependent upon the interaction of many functioning hair cells and cochlear neurons. Good word recognition scores imply there are large populations of functioning hair cells and neurons, even in the presence of severe to profound threshold loss (Halpin, 2002). Even with sufficient amplification, individuals with dead regions might utilize little or no information from much of the frequency range of speech for several reasons. When regions within the cochlea are damaged, the frequency components of speech are detected and analyzed by frequency channels best tuned to other frequencies; a mismatch

in frequency and place coding may result in difficulty interpreting the information (Moore, 2001). When the frequency components of speech are being detected and analyzed by channels best tuned for other frequencies, the analysis of the best-tuned frequencies that would have been detected and analyzed by these channels in a normal ear may be impaired. The broadband characteristics of speech may cause certain frequency channels to become overloaded with information. Speech information, specifically regarding formant frequency information, is coded in time patterns of neural impulses, known as phase locking. When a mismatch occurs between the frequencies of speech and the place where they are detected, the temporal coding mechanism may not operate effectively (Moore, 2001).

Cochlear Dead Regions and Hearing Aids

Killion and Fikret-Pasa (1993) identified three types of sensorineural hearing loss. Type I and Type II hearing losses are characterized by loudness growth functions. Type III loss is characterized by intelligibility growth functions. Individuals with Type I hearing loss typically only demonstrate a loss of sensitivity for soft sounds, while those with Type II hearing loss typically show a loss of sensitivity for soft sounds and a partial loss of acuity for loud sounds. Type I and Type II hearing losses typically are the result of damage to the OHCs. Individuals with Type III demonstrate a loss of sensitivity for soft and loud sounds and a reduced dynamic range of hearing and intelligibility. Type III hearing loss most likely is the result of damage to both the IHCs and OHCs. It is because of these differences that each type of hearing loss requires a unique method of hearing aid processing.

The fitting of hearing aids for individuals with cochlear dead regions presents a set of unique challenges. Research in the area of speech perception suggests that individuals with high-frequency dead regions receive little or no benefit from amplification of frequencies within the dead region (Vickers, Moore, & Baer, 2001; Baer, Moore, & Kluk, 2002). For individuals with high-frequency cochlear dead regions, there may be several benefits to reducing high-frequency amplification. A reduction in gain in areas with known cochlear dead regions may actually lead to improved speech intelligibility. A decrease in high-frequency gain may also reduce problems associated with feedback and reduce distortion within the hearing aid. It also allows the audiologist to concentrate efforts toward appropriate amplification in the areas with known residual hearing (Moore, 2001).

For individuals with low-frequency cochlear dead regions, decisions regarding amplification are more difficult. Similar to those with high-frequency dead regions, individuals with low-frequency dead regions may not gather useful information for speech intelligibility from low-frequency amplification. However, there has been some evidence to suggest that individuals with low-frequency dead regions may be able to extract some useful low-frequency information related to speech from areas of the cochlea best tuned to higher frequencies (Halpin, Thornton, & Hasso, 1994). Therefore, it may be advantageous to these individuals to provide some amplification into the cochlear dead region (Moore, 2001).

Vickers et al. (2001) examined the benefits of high-frequency amplification for individuals with and without diagnosed high-frequency cochlear dead regions in quiet. In the patients without cochlear dead regions, speech intelligibility performance improved

progressively with increasing cutoff frequencies. In individuals with cochlear dead regions, speech intelligibility performance initially improved with increasing cutoff frequency. Performance of the individuals with dead regions worsened when the cutoff frequency was approximately 1.5-2 times the estimated edge of the dead region. These results indicate that amplification of high frequencies contained within the dead region negatively impacted performance.

A later study by Baer et al. (2002) examined the benefits of high-frequency amplification for individuals with and without diagnosed high-frequency cochlear dead regions in noise. Results of this study were similar to those found by Vickers et al. (2001). Specifically, individuals without cochlear dead regions benefited from amplification of high frequencies during the speech in noise condition, while those with cochlear dead regions did not. In some cases, however, individuals with dead regions did benefit from the amplification of frequencies 50-100% above the estimated edge frequency of the dead region.

Mackersie, Crocker, and Davis (2004) examined the ability of 14 adults with and without cochlear dead regions to utilize wide-band amplification. Results from this study indicated that in quiet or low-level noise situations, patients with and without cochlear dead regions benefited equally from wide-band amplification. These findings differ from prior research conducted by Vickers et al. (2001) and Baer et al. (2002) where the performance of individuals with cochlear dead regions deteriorated with increased high-frequency amplification. In high noise level situations, however, those with cochlear dead regions did not receive benefit from wide-band amplification at frequencies higher than one octave above the edge of the dead region.

Moore (2002a) suggested that hearing aids with automatic gain control (AGC) or compression be used when fitting individuals with cochlear dead regions. When a cochlear dead region is suspected, the gain recommended by the fitting method should be applied only to areas where there are known functioning hair cells, or in special cases, such as in individuals with low-frequency dead regions, gain may be applied to areas extending 50-100% inside the dead region. Use of the NAL-NL1 model for hearing aid fitting may be beneficial for individuals with cochlear dead regions. The NAL-NL1 model decreases the amount of high-frequency gain as the hearing loss increases. It has been found that individuals with high-frequency hearing loss get progressively less benefit from the amplification of high frequencies as the hearing loss increases (Moore, 2002a).

The use of frequency transposition has also been suggested as method of increasing the audibility of speech information (Velmans & Marcuson, 1983; Parent, Chmiel, & Jerger, 1997; McDermott, Dorkos, Dean, & Ching, 1999). The most commonly used method has been downward frequency transposition for those with high-frequency dead regions. Use of these devices may present several problems including an overload of information to the area with residual hearing. The transposed information is also presented to areas of the cochlea that are not the best tuned for analyzing the information. As a result, there is an extended learning period that must be completed in order to effectively use the transposed information (Rosen, Faulkner, & Wilkinson, 1999). This form of amplification may also present a unique set of problems in the presence of background noise. Specifically, when background noise that was previously inaudible, due to the frequency region, is transposed to a frequency region where it is

easily audible, the benefits gained through use of transposition are negligible (Moore, 2001; Moore, 2002a).

Statement of the Problem

The audiometric configuration that is associated with dead zones has been published in a number of journal articles, both research and clinical protocols exist for the detection of dead zones, and amplification protocols have been published for hearing aid fitting in patients with suspected dead zones; however, the prevalence of suspected dead zones in a typical patient population is unknown. How often should audiologists suspect a patient with a cochlear dead region be seen in their typical practice? A known prevalence would give audiologists more information regarding how often special procedures for diagnosis and treatment need to be used. Therefore, this study was designed to estimate the potential prevalence of cochlear dead regions within a university clinic population using criteria from Moore (2001). This study also investigated the word recognition scores associated with the Moore profile and hearing aid success of patients whose audiograms indicate possible dead regions with the hearing aid success of patients without dead regions. For this study, hearing aid success was defined as the number of return visits to the clinic for hearing aid adjustments and the number of hearing aids returned to the clinic.

CHAPTER 3

METHOD

Participants

The audiological records from 500 patients (214 males and 286 females) of the Towson University (TU) Speech-Language-Hearing Clinic were examined to estimate the potential prevalence of cochlear dead regions in this population. The TU Speech-Language-Hearing Clinic was established in 1965 and offers both speech-language pathology and audiology services. Patients included in this study were adults 18 years and older. Three-hundred-seventy-five patient files were located in the TU Clinic, while 125 files were retrieved from storage. The clinical staff at TU typically keeps active patient files in the clinic for 1 year. If the patient has not been seen in the TU clinic for more than one year, the files are moved to a separate storage facility and are typically kept for 7-8 years and then destroyed. Patient files from 1997-2005 were examined as part of this study.

Procedures

Demographic (age, gender) and audiological (pure tone thresholds, speech audiometry results) were extracted over a four-week period from patient files. Audiological information included the right and left ear pure-tone thresholds from 250 to 8000 Hz in octave intervals, speech recognition thresholds (SRTs), and word recognition scores (WRS). In cases where a patient was fit with a hearing aid following their initial audiologic evaluation, information regarding the number of return clinic visits from 0-3

and 3-6 months post-fitting, as well as the total number of clinic visits from 0-6 months were recorded. Patients were only included as part of this study if they had been fit with a hearing aid at least six months prior to the beginning of data collection, in order to ensure that follow-up hearing aid data were available. During data collection, only the clinic visits directly related to a patient complaint involving the reprogramming or adjustment to the sound quality of the aids were included. Visits related to other hearing aid problems (i.e. fit of the aid, battery door replacement, etc.) were not included. The patient's decision whether or not to keep their hearing aids by 3 months post-fitting, typically the end of the trial period, was also recorded.

Based on the audiometric findings, the patients were classified into two groups: those with suspected cochlear dead regions and those without suspected cochlear dead regions based on an analysis of the audiogram following criteria suggested by Moore (2001). Specifically, these criteria include a pure tone threshold greater than 90 dB at high frequencies or 75-80 at low frequencies, a hearing loss greater than 40-50 dB at low frequencies with near-normal hearing at higher frequencies, a hearing loss greater than 50 dB at low frequencies with a mild hearing loss at higher frequencies, or a very steeply sloping (greater than 50 dB/octave) hearing loss with increasing frequency. The specific criteria used as possible indicators of cochlear dead regions are listed in Table 1. In addition to pure tone results, the patient's WRS was considered as a possible indicator of a cochlear dead region. Information gathered by Halpin (2002) suggested that individuals with cochlear dead regions have poorer WRS than individuals without cochlear dead regions due to the reduced function of the individual hair cells and cochlear

Table 1

Possible Indicators of Suspected Cochlear Dead Regions based on Moore (2001) Criteria

	Criteria
Low-frequency dead region	
Condition 1	Thresholds greater than 75 dB at 250 or 500 Hz.
Condition 2	Thresholds greater than 40 dB at 250 or 500 Hz with thresholds at 1000-8000 Hz no greater than 35 dB.
Condition 3	Thresholds greater than 50 dB at 250 or 500 Hz with thresholds at 1000-8000 Hz no greater than 45 dB.
High-frequency dead region	
Condition 1	Thresholds greater than 90 dB at 2000-8000 Hz.
Condition 2	Hearing loss with a slope greater than 50 dB/octave at frequencies above 1000 Hz.

neurons. For the purposes of this study, the WRS of patients with suspected cochlear dead regions was compared to the WRS of patients without cochlear dead regions and with the predicted score based on the articulation index (AI).

The articulation index was first introduced by French and Steinburg (1947). It is based on the amount of speech information available to the listener through a number of frequency bands each weighted according to their contribution toward speech understanding, known as band importance (Pavlovic, 1991). The AI is useful for the prediction of speech intelligibility (Humes, Dirks, Bell, Ahlstrom, & Kincaid, 1986), the comparison of predicted and actual speech recognition performance (Kamm, Dirks, & Bell, 1985; Studebaker & Sherbecoe, 1991), and the selection of hearing aids (Fabry & Van Tasell, 1990; Pavlovic, 1988).

For the purposes of this research, the articulation index was calculated by using a method first outlined by Pavlovic (1988). This method for calculating the AI is relatively simple when compared to other methods because the band importance values are assumed to be equal. The available dynamic range (the speech range that is heard by the patients based on his/her audiogram) is calculated at the frequencies of 500, 1000, 2000, and 4000 Hz. The values for all four frequencies are added together and divided by 120. 120 is the total possible value that would be obtained if the entire speech region was audible. The result of this calculation is a number between 0 and 1which represents the AI.

CHAPTER 4

RESULTS

Table 2 provides the basic demographic and audiometric information for 500 patients seen at the Towson University Speech-Language-Hearing Clinic over the past 5 years. Pure-tone averages (PTAs) were calculated using audiometric thresholds at 500, 1000, and 2000 Hz. Pure-tone averages could not be calculated for 3 patients in the right ear and 2 patients in the left ear, due to the presence of no response (NR) on the audiogram at 500, 1000, and/or 2000 Hz.

Patients were categorized based on their PTA using a commonly accepted classification system for severity of hearing loss shown in Table 3 (Goodman, 1965). An examination of hearing sensitivity was conducted for the right and left ears separately. 34% of the right ears had normal hearing sensitivity,13% were categorized within the range of slight hearing loss, 22% were mild, 18% were moderate, 9% were moderately-severe, 3% were severe, and 2% were profound. Using the same criteria for the left ear, 34% of the patients had normal hearing sensitivity, 13% were categorized within the range of slight hearing loss, 21% were mild, 17% were moderate, 8% moderately-severe, 4% were severe, and 2% were profound.

Patients were also categorized based on the slope of their hearing loss using the commonly accepted classification system, as shown in Table 4, to describe audiometric configuration (Carhart, 1945) across all hearing loss categories. Based on these criteria,

Table 2

Subject Demographic and Audiometric Data

	Mean	SD	Range
Age (n=500)	58.5	21.5	18-100
Pure Tone Average			
Right Ear (n=497)	30.4	22.8	0-106.7
Left Ear (n=498)	30.4	23.3	0-108.8
Word Recognition Score (%)			
Right Ear (n=500)	81.3	25.0	0-100
Left Ear (n=500)	81.4	24.4	0-100

Table 3

Classification System Used to Describe Severity of Hearing Loss

Hearing Level (dB)	Classification
-10 to 15	Normal hearing
16 to 25	Slight hearing loss
26 to 40	Mild hearing loss
41 to 55	Moderate hearing loss
56 to 70	Moderately severe hearing loss
71 to 90	Severe hearing loss
91 and above	Profound hearing loss

Note. Scale is a modification of Goodman (1965) by Clark (1981). From J Katz (Ed.), *Handbook of Clinical Audiology* (p. 82), by R. H. Harrell, 2002, Baltimore: Lippincott, Williams, and Wilkins.

Table 4

Classification System Used to Describe Configuration of Hearing Loss (modified from Carhart, 1945)

Term	Description
Flat	Less than 5 dB rise or fall per octave
Gradually falling	5-12 dB decrease per octave
Sharply falling	13 dB or more decrease per octave
Abruptly falling	Flat or gradually falling, then sharply falling
Rising	5 dB or more increase per octave
Trough	20 dB or greater loss at 1000 Hz and/or 2000 Hz than at 500 and 4000 Hz
Miscellaneous	Does not fit any of the above

Note. The primary frequencies considered in describing the audiometric configuration are 500 through 4000 Hz. From *Audiometric Interpretation A Manual of Basic Audiometry* (p. 13), by H. Kaplan, V. S. Gladstone, & L. L. Lloyd, 1993, Boston: Allyn & Bacon.

20% of the right ears were categorized with a flat configuration, 14% were categorized with a gradually falling hearing loss, 26% were categorized with a sharply falling hearing loss, 27% were categorized with an abruptly falling hearing loss, 3% were categorized with a rising hearing loss, 2% were categorized with a trough hearing loss, and 7% of the patients could not be classified in any of the previous categories and were considered miscellaneous for the right ear. Using the same criteria for the left ear, 17% of the patients with at least a mild hearing loss were categorized with a flat configuration, 14% were categorized with a gradually falling hearing loss, 22% were categorized with a sharply falling hearing loss, 33% were categorized with an abruptly falling hearing loss, 3% were categorized with a rising hearing loss, 2% were categorized with a trough hearing loss, and 9% of the patients could not be classified in any of the previous categories and were considered in the miscellaneous category.

The presence of suspected cochlear dead regions was determined for each patient based on Moore's (2001) criteria. Moore identified 3 indicators or conditions for the presence of low-frequency cochlear dead regions and 2 indicators or conditions for the presence of high-frequency dead regions, as shown previously in Table 1. Table 5 shows the number of right and left ears that met the criteria for the presence of suspected cochlear dead regions for each of the five conditions. Due to the severity and/or configuration of the hearing loss, a total of 12 ears, 6 right and 6 left, met more than one criteria for presence of suspected cochlear dead regions. Because several ears were included in multiple categories, the total number of ears from each category is not reflective of the actual number of ears with suspected cochlear dead regions. A total

Table 5

Number of Ears With Suspected Cochlear Dead Regions

Criteria	N=
Low-frequency dead region	
Condition 1	
Right Ear	14
Left Ear	13
Condition 2	
Right Ear	0
Left Ear	0
Condition 3	
Right Ear	2
Left Ear	0
High-frequency dead region	
Condition 1	
Right Ear	13
Left Ear	12
Condition 2	
Right Ear	13
Left Ear	15

of 36 right and 33 left ears were identified with suspected cochlear dead regions. Table 6 shows the mean word recognition scores (WRS) of ears with suspected cochlear dead regions for each of the 5 conditions. The mean word recognition scores (WRS) of ears with and without suspected cochlear dead regions based on pure tone average are compared in Table 7. There is an obvious difference in WRS between these two groups. An analysis of variance (ANOVA) indicated the difference between groups was statistically significant, F (1, 998) = 2283.97, p = .000. A correlation analysis indicated a significant correlation between the pure tone average and the WRS, r = -.739, p = .000, as is expected. Because the pure tone average was significantly correlated with WRS, a second ANOVA examining the effect of dead zone (yes, no) was conducted using PTA as a covariate. An analysis of variance (ANOVA) indicated the difference between groups was still statistically significant, F (1, 992) = 12.682, p = .000. The covariate (pure tone average) was also statistically significant, F (1, 992) = 1091.438, p = .000. The comparison of the mean articulation index (AI) values and mean word recognition scores are also shown in Table 8. A linear regression analysis was calculated predicting the WRS of each ear based on the AI for both dead zone profile ears and non-dead zone profile ears. A significant regression equation was found for the dead zone profile ears, F (1, 67) = 70.022, p = 000, with an R^2 of 0.511, and for the non-dead zone profile ears, F (1, 929) = 586.15, p = .000, with an R^2 of .376. The predicted WRS for dead zone profile ears is equal to 36.611 + 74.448 (AI) and the predicted WRS for non-dead zone profile ears is equal to 63.743 + 37.359 (AI). Scatter plots of WRS as a function of AI for patients with and without the suspected dead zone profile are shown in Figures 3 and 4. These figures indicate a similar association between AI and WRS for both groups.

Table 6

Mean Word Recognition Scores (WRS) of Ears With Suspected Cochlear Dead Regions

Criteria	Mean	SD	Range
	Low-frequency dead regions		
Condition 1			
Right Ear (n=14)	15.6	24.7	0-76
Left Ear (n=13)	4.0	6.4	0-20
Condition 2			
Right Ear (n=0)	-	-	-
Left Ear (n=0)	-	-	-
Condition 3			
Right Ear (n=2)	0	0	0
Left Ear (n=0)	-	-	-
	High-frequency dead regions		
Condition 1			
Right Ear (n=13)	16.3	22.7	0-68
Left Ear (n=12)	12.8	16.8	0-48
Condition 2			
Right Ear (n=13)	84.0	17.6	40-100
Left Ear (n=15)	74.7	18.9	44-100

Table 7

Word Recognition Scores (WRS) of Ears With and Without Suspected Cochlear Dead Regions Based on Pure Tone Average (PTA)

PTA	Mean	SD	Range
0-25 dB			
With (n=13)	92.9	7.6	76-100
Without (n=438)	95.3	7.6	72-100
26-40 dB			
With (n=6)	75.0	8.8	66-92
Without (n=226)	85.5	13.8	32-100
41-55 dB			
With (n=8)	71.8	21.2	44-100
Without (n=168)	69.9	22.7	0-100
56-70 dB			
With (n=7)	38.3	26.1	0-100
Without (n=77)	59.8	26.3	0-92
71-90 dB			
With (n=12)	31.2	27.5	0-76
Without (n=22)	44.9	34.2	0-100
90 dB and above			
With (n=18)	4.0	7.8	0-24
Without (n=0)	-	-	-

Table 8

Mean Articulation Index and Word Recognition Scores (WRS) for Ears With and Without Suspected Cochlear Dead Regions

	Mean Actual WRS Score	AI	SD	Range
0-25 dB				
With (n=13)	92.9	.81	.12	.67-1.0
Without (n=438)	95.3	.87	.16	.46-1.0
26-40 dB				
With (n=6)	75.0	.51	.07	.46-.67
Without (n=226)	85.5	.35	.12	.13-.75
41-55 dB				
With (n=8)	71.8	.19	.17	.04-.58
Without (n=168)	69.9	.10	.08	0-.50
56-70 dB				
With (n=7)	38.3	.10	.10	0-.25
Without (n=77)	59.8	.01	.03	0-.17
71-90 dB				
With (n=12)	31.2	0	0	0
Without (n=22)	44.9	0	.01	0-.04
90 dB and above				
With (n=18)	4.0	0	0	0
Without (n=0)	-		-	-

Figure 3. WRS as a function of AI for patients with suspected dead zone profile.

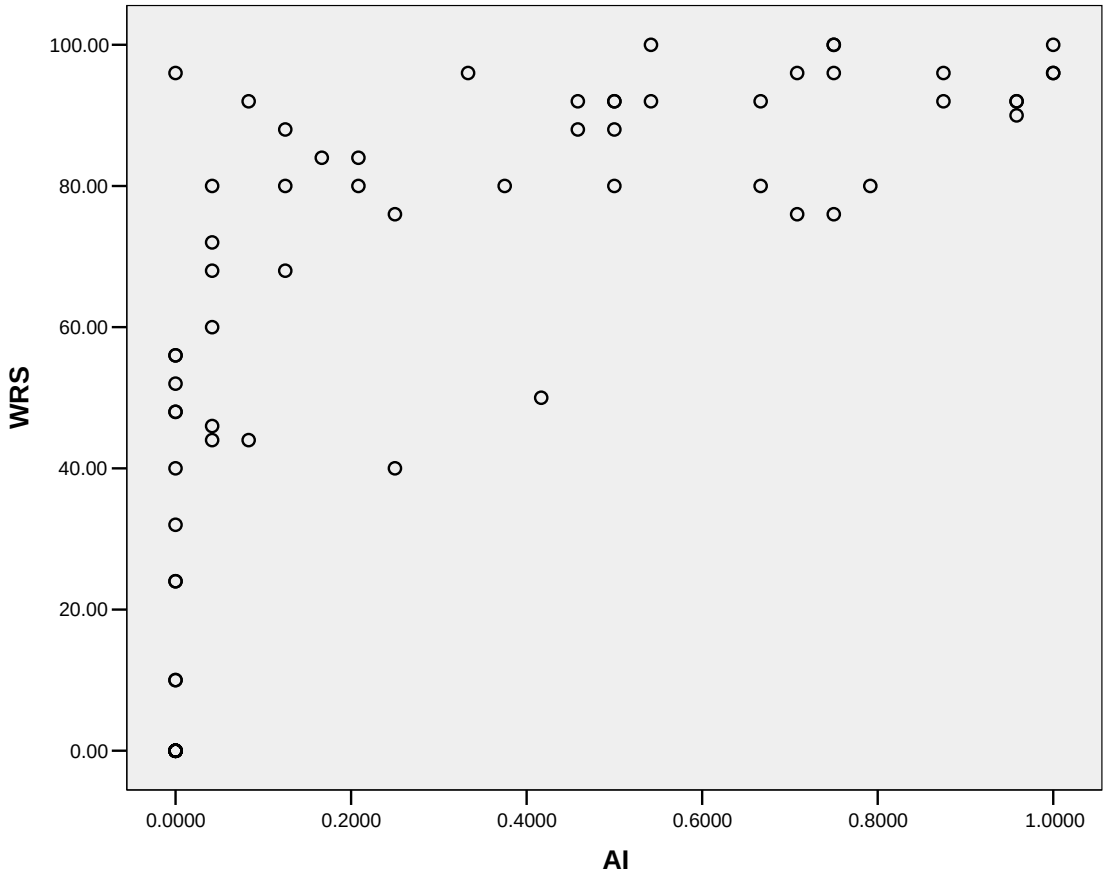
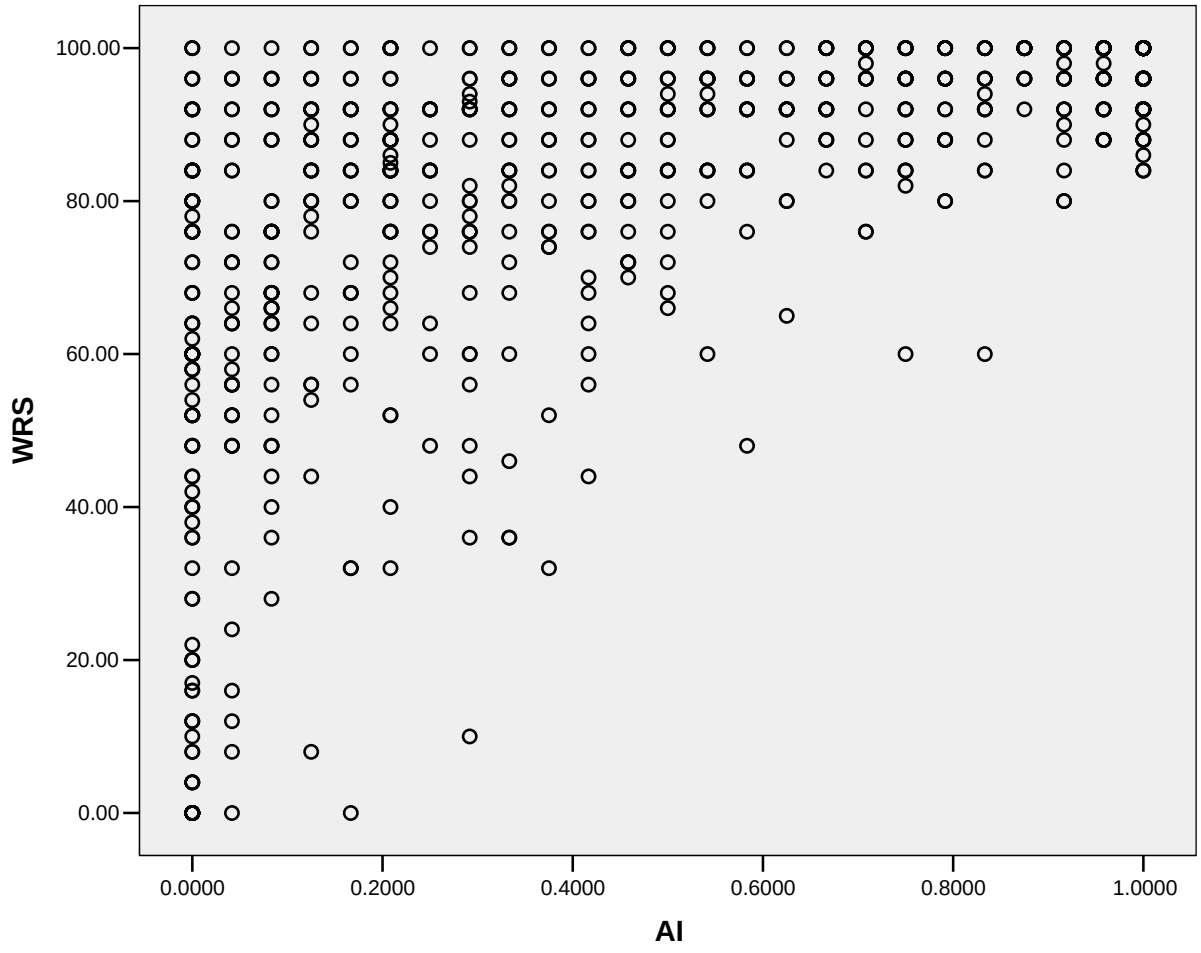


Figure 4. WRS as a function of AI for patients without suspected dead zone profile.



Overall, if the AI is low, the WRS can range from 0 to 100%. As the AI increases, low WRS scores are rarely seen.

A total of 234 out of 500 patients were identified with at least a mild hearing loss in one ear; however, they did not meet the criteria for suspected cochlear dead regions. A total of 57 out of 500 patients were identified with a suspected cochlear dead region in at least one ear. 12 of the 57 total patients were identified with suspected cochlear dead regions in both ears.

Of the 234 patients not identified with a suspected cochlear dead region, 198 were fit with a hearing aid at the TU Speech-Language-Hearing Clinic. Of the 57 patients identified with a suspected cochlear dead region in at least one ear, 36 were fit with a hearing aid at the TU Speech-Language-Hearing Clinic. Results for the number of clinic visits are shown in Table 9. On average, patients with a dead zone profile returned for 4.8 visits compared to an average 3.4 visits for patients without this profile. A chi-squared analysis indicated the number of visits was statistically significant between the two groups, χ^2 (1) = 15.447, $p < 0.01$. Of the 198 patients not identified with a suspected cochlear dead region, 180 (91%) chose to keep their hearing aids at 3 months post-hearing aid fitting, while 18 returned the hearing aids. Of the 36 patients identified with a suspected cochlear dead region, 35 (97%) chose to keep their hearing aids at 3 months post-hearing aid fitting, while 1 returned the hearing aids. Thus, the patients with a dead zone profile had a lower return rate than the patients without this profile.

Table 9

Mean Number of Clinic Visits for Patients With and Without Suspected Cochlear Dead Regions

	Mean	SD	Range
Within 0-3 months			
With (n=35)	2.6	1.7	0-6
Without (n=198)	2.0	1.1	0-5
Within 3-6 Months			
With (n=34)	2.4	1.4	0-4
Without (n=180)	2.3	1.2	0-3
Total Visits Within 0-6 Months			
With (n=34)	4.8	2.3	0-8
Without (n=180)	3.4	1.9	0-6

CHAPTER 5

DISCUSSION

This study identified 57 of 500 patients with audiometric profiles suggestive of cochlear dead regions indicating that the prevalence of possible cochlear dead regions, based on the Moore criteria, is 11.4%, although the clinical setting (i.e. university clinic) from which the data were extracted may be a unique population and not necessarily reflective of hospital and private practice clinics. The results from the current study suggest audiologists in a university clinic will see audiometric profiles associated with cochlear dead regions in just over 1 out of 10 patients. This may be more frequent than audiologists realize, although there are no available data at this time to indicate how prevalent audiologists think dead zones are in their patient population and how often they conduct TEN tests or other measures to examine whether patients have cochlear dead regions. This study was conducted to examine only the possibility of dead zones based on a profile system used with a file review. The dead zones were not confirmed in these patients and it is likely that many of these patients did not, in fact, have dead zones. Thus the results of this study indicated only the differences between patients with and without confirmed dead regions.

The mean word recognition scores (WRS), across degree of hearing loss, were significantly poorer for patients with suspected cochlear dead regions compared to WRS of patients without suspected cochlear dead regions, particularly in those patients with pure tone averages in the mild, moderately-severe, and severe hearing loss categories.

Halpin (2002) suggested that WRS may be used as a secondary predictor of cochlear dead zones. The significant difference in WRS supported this assumption. This study identified 26 ears with suspected low-frequency cochlear dead regions and 44 total ears with suspected high-frequency dead regions. These results suggest that high-frequency cochlear dead regions may occur more frequently than low-frequency cochlear dead regions. Research in the area of cochlear dead regions and speech perception has indicated that only limited speech information can be extracted from the frequency components of speech that fall within a cochlear dead region (Shannon, Zeng, & Wygonski, 1998; Rosen et al., 1999). Several studies have been conducted to specifically examine the perception of speech of patients with low-frequency dead regions (Thornton & Abbas, 1980; Van Tasell & Turner, 1984; Halpin et al., 1994). Results from all of these studies indicated that for patients with low-frequency cochlear dead regions, only limited information from the low frequencies of speech is useful; however, there was evidence to suggest that a small amount of low-frequency information may be gathered by the IHCs and neurons best tuned to mid- and high-frequency information (Moore, 2001). Moore (2004) found that individuals with extensive cochlear dead regions, particularly in the low-frequency range, are unable to make effective use of speech information at high frequencies when compared to individuals without cochlear dead regions.

Studies have been completed to assess the speech perception of patients with high-frequency cochlear dead regions (Hogan & Turner, 1998; Turner & Cummings, 1999). The findings from these studies indicate that very little useful speech information is available, particularly in individuals with moderate-to-severe loss in the high

frequencies. This information is particularly useful for the fitting of hearing aids. As discussed previously, it has been indicated that in individuals with high-frequency dead regions, amplification of the high-frequencies of speech is not always optimal and may even be harmful, in some instances (Moore, 2001).

Patients with the cochlear dead zone profile returned more often for follow up visits, on average for 1.4 more follow up visits within the first 6 months following the issuance; however, hearing aid return rates for individuals with suspected cochlear dead regions were not significantly different between individuals without suspected cochlear dead regions, as only 1 out of 36 patients with suspected cochlear dead regions and only18 of 198 patients without suspected cochlear dead regions returned their hearing aids after 3 months. This corresponds to a return rate of 3% for patients with suspected cochlear dead regions and 10% for patients without suspected cochlear dead regions. Both of these values are much lower than the average industry return rate of 18% (Northern & Beyer, 1999), suggesting that the presence of suspected cochlear dead regions, based on the criterion used in this study, had no effect on a patient's decision to keep or return their hearing aid and that the clinic is fitting hearing aids with more success than the average clinic. Because it is unlikely the present sample of patients with suspected dead regions included patients without actual dead regions, it is possible the return rate was not accurately reflected. Future studies should consider the return rate and number of hearing aid visits associated with confirmed cases of dead regions.

The current findings suggest individuals with suspected cochlear dead regions may require more programming changes as they attempt to adjust to amplification. For patients with a profile suggestive of cochlear dead regions, hearing aid fitting rationales

may underestimate the need for amplification in the dead regions (Moore, 2001; Moore, 2002a). For this reason, patients with suspected cochlear dead regions may require more visits post-hearing aid fitting than patients without suspected dead regions. Other factors may also impact the number of return clinic visits post-hearing fitting including the type of hearing aid, the patient's prior listening experience with hearing aids, and clinician preference for the amount of follow-up. It is possible that a prospective study, with the use of confirmatory test of dead zones (e.g. TEN) may provide some clarity as to why patients required more visits to the clinic and yet returned the hearing aids less often.

Implications of Research

The results of this study indicate a prevalence of a profile suggestive of cochlear dead regions significant enough to warrant the inclusion of tests for the detection of cochlear dead regions as part of a routine clinical test battery at this time; however, because it is unclear how accurately the presence of cochlear dead regions can be assessed through the audiogram, the audiologist may want to consider tests such as the TEN (HL) test (Moore et al., 2004), when the patient's audiometric profile is suggestive of a cochlear dead region. The knowledge that a patient has a cochlear dead region may have a significant affect on the selection of amplification and fitting strategies for that patient. Previous studies have suggested the use of automatic gain control or compression (Moore, 2001; Moore, 2002a), the NAL-NL1 fitting formula (Ching, Dillon, & Byrne, 1998; Dillon, 1999), or the use of transposition hearing aids (Parent et al., 1997; McDermott et al., 1999) for patients with cochlear dead regions.

Areas of Future Research

There has been a great deal of research completed in the area of cochlear dead regions to date, however, very little has been focused on the clinical implications of cochlear dead regions. Certainly, further studies need to be completed within other clinic populations in order to accurately estimate the prevalence of cochlear dead regions. A comparison of the use of audiometric test results to PTCs and the TEN test to determine the presence of cochlear dead regions would seem to have clinical value because it remains unclear whether or not audiograms are accurate predictors of cochlear dead regions. Vestergaard (2003) stated that an articulation index (AI) model designed to account for the presence of cochlear dead regions may be useful to more accurately predict the word recognition of patients with cochlear dead regions and in the selection of the best means of amplification for these patients. In the current study, the AI was able to predict the WRS for both the dead zone and non-dead zone profile; therefore, either the AI has a questionable value as a predictor of the presence of dead zones or there were quite a lot of patients without dead zones who were classified as dead zone patients with the current profile. Further research on the prevalence of dead zones in a clinic population not specific to a university, comparison of various diagnostic tests, and comparison of hearing aid fitting rationales may provide helpful information for clinicians selecting hearing aid fitting strategies for patients with cochlear dead regions.

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