Chapter Twelve

Dead Regions in the Cochlea: Implications for the Choice of High-Frequency Amplification

Brian C. J. Moore

Introduction

Cochlear hearing loss is sometimes associated with complete destruction of the inner hair cells (IHCs) within the cochlea (Schucknecht 1964, 1974; Engström 1983). For reviews, see Schucknecht (1974) and Borg, Canlon and Engström (1995). Sometimes the IHCs may still be present, but may be sufficiently abnormal that they no longer function. The IHCs are the transducers of the cochlea, responsible for converting the vibration patterns on the basilar membrane into action potentials in the auditory nerve. When the IHCs are non-functioning over a certain region of the cochlea, no transduction will occur in that region. For this reason, I refer to such a region as a *dead region.*

I have proposed (Moore 2001) that a dead region should be defined in terms of the characteristic frequencies (CFs) of the IHCs and/or neurones *immediately adjacent* to the dead region. For example, if there are surviving IHCs and neurons with CFs up to 2000 Hz, but not above, this would be described as high-frequency dead region starting at 2000 Hz. This definition is appropriate even if the CFs of the IHCs and neurones are shifted from "normal" values, as can happen in cases of cochlear hearing loss (Sellick, Patuzzi and Johnstone 1982; Ruggero, Rich, Recio, Narayan and Robles 1997). The definition also allows for an easy interpretation of the psychoacoustic results that will be presented later in this paper.

The diagnosis of dead regions is clinically important for two reasons. Firstly, if there are one or more extensive dead regions, the benefit of a hearing aid is likely to be limited, and aided speech intelligibility is likely to be poor (Halpin, Thornton and Hasso 1994; Baer, Moore and Kluk in press; Vickers, Moore and Baer 2001). Secondly, results presented below suggest that there is little or no benefit of amplifying frequencies *well inside* a dead region. Therefore, the diagnosis of dead regions has important implications for the fitting of hearing aids.

It should be emphasised that the experimental work on dead regions conducted in my laboratory has used adult subjects with acquired hearing loss. Such hearing loss is often, but not exclusively, associated with dysfunction of the IHCs and outer hair cells (OHCs) (Schuknecht 1974; Wright, Davis, Bredberg, Ulehlova and Spencer 1987). Causes of congenital hearing loss are likely to be much more varied, and may involve abnormalities in many structures and systems other than the OHCs and IHCs, for example the stria vascularis and the tectorial membrane (Friedman 1997).

Dead Regions and the Audiogram

Basilar-membrane vibration in a dead region is not detected via the neurones directly innervating that region. Say, for example, that the IHCs at the basal end of the cochlea are non-functioning. Neurons innervating the basal end, which would normally have high CFs, will not respond. However, if a high-frequency sinusoid is presented, it may be detected if it produces sufficient basilar-membrane vibration at a more apical region; this corresponds to downward spread of excitation. In other words, a high-frequency sound may be detected via neurons that are tuned to lower frequencies. Similarly, when there is a low-frequency dead region, a low-frequency tone may be detected via the upward spread of excitation to a more basal region of the cochlea.

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It has been recognized for many years that, when a dead region is present, the audiogram will give a misleading impression of the amount of hearing loss, for a tone whose frequency falls in the dead region (Gravendeel and Plomp 1960; Halpin et al. 1994). Effectively, the "true" hearing loss in a dead region is infinite, but the audiogram may sometimes indicate only a moderate hearing loss. This is especially true when there is a low-frequency dead region. To explain why this is so, I will make use of the concept of the excitation pattern. Normally, this is taken to refer to the amount of excitation evoked by a sound as a function of "place" on the basilar membrane or the associated CF (Zwicker 1970; Moore and Glasberg 1983; Moore 1997). However, within a dead region, basilar-membrane vibration does not lead to neural activity, whereas the word "excitation" could be taken to imply such activity. Here, I use the term excitation pattern to refer to the amount of basilar-membrane vibration, plotted as a function of the associated CF, regardless of whether or not that vibration leads to neural activity.

The solid curve in figure 1 shows schematically the excitation pattern that might be evoked by a low-frequency (250 Hz) tone in an ear with a low-frequency dead region, with normal hearing at medium and high frequencies. The dead region is indicated by the shaded area. The tone becomes audible when it produces sufficient excitation in the region where there are functioning IHCs. In this example, the 250-Hz tone needs to be presented at about 60 dB to produce detectable excitation just outside the edge of the dead region (CFs just above 1.3 kHz).

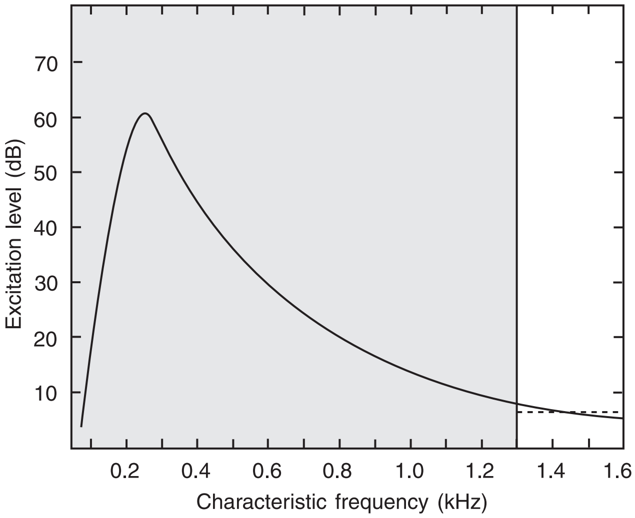

Figure 1. The solid curve shows the excitation pattern that might be evoked by a low-frequency (250 Hz) tone in an ear with a low-frequency dead region, with normal hearing at medium and high frequencies. The dead region is indicated by the shaded area. The tone level was chosen so that it was at absolute threshold; the excitation in the frequency region just above 1.3 kHz lies a little above the "threshold" excitation level indicated by the horizontal dashed line.

It is not possible to determine from the audiogram alone whether or not a patient has a low-frequency dead region. For example, Halpin et al. (1994) described two patients with very similar audiograms, both having a low-frequency hearing loss with nearly normal mid-frequency hearing. Post-mortem examination showed that one had no survival of the organ of Corti in the apical region, while the other had an organ of Corti which was present and of normal appearance. Because of the difficulty in using the audiogram to diagnose a dead region, many researchers have advocated the use of masking sounds as a way of diagnosing the presence of dead regions. This approach will be discussed in more detail later on.

Consider now the effect of a high-frequency dead region on the audiogram. It is often the case that downward spread of excitation in the cochlea is very restricted (i.e. the low-frequency side of the excitation pattern is steep), even in ears where the hair cells are damaged. However, there can be considerable individual variability, and some hearing-impaired ears show marked downward spread of excitation as well as upward spread of excitation (Glasberg and Moore 1986). Because the excitation pattern usually has a steep low-frequency side, a dead region at high frequencies is usually associated with a severe or profound hearing loss at high frequencies, and the audiogram is often steeply sloping. This is illustrated in figure 2, which shows excitation patterns calculated for a hypothetical ear with a 40-dB hearing loss at low frequencies, and a dead region extending from 1 kHz upwards (indicated by the shaded area). The patterns were calculated using the model described by Moore and Glasberg (1997) which takes into account the broadening of the excitation patterns that occurs with increasing level and increasing OHC damage (Moore 1998). Each of the curves represents the excitation pattern for a tone with frequency falling in the dead region; the frequencies used are 1.1, 1.2, 1.3, 1.4 and 1.5 kHz. It is assumed that this tone is detected because of the downward spread of excita-

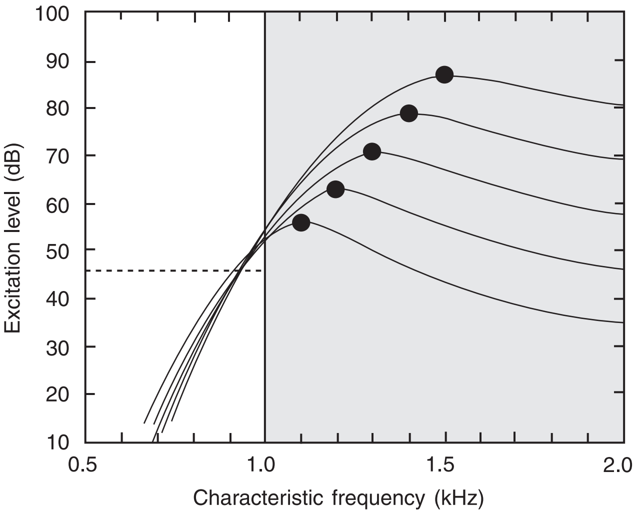

Figure 2. Excitation patterns calculated for a hypothetical ear with a 40-dB hearing loss at low frequencies (threshold excitation level indicated by the horizontal dashed line), and a dead region extending from 1 kHz upwards (indicated by the shaded area). Each of the curves represents the excitation pattern for a tone with a frequency falling in the dead region; the frequencies used are 1.1, 1.2, 1.3, 1.4 and 1.5 kHz. The levels of the tones were chosen so that each was at the absolute threshold. Solid circles indicate the levels at the peaks of the excitation patterns.

tion. To be detectable the excitation must fall above the horizontal dashed line, whose position is determined by the absolute threshold for frequencies below the dead region (the excitation is plotted here in units related to dB SPL; a 40-dB hearing loss corresponds to an absolute threshold of about 46 dB SPL between 0.5 and 1 kHz). The level of each tone has been chosen so that the excitation spreading to the region with CFs just below 1 kHz is just detectable. The solid circles represent the frequency and level at the peak of each excitation pattern. The function traced out by the circles is closely related to the expected absolute threshold for such a hearing loss, specified in dB SPL. Notice that the expected threshold changes by about 32 dB as the frequency changes from 1.1 to 1.5 kHz, which corresponds to a relatively steep slope of 72 dB/octave.

Whenever the audiogram has a very steep slope, the threshold worsening rapidly with increasing frequency, this should be taken as preliminary evidence for a high-frequency dead region, and further testing should be carried out. However, dead regions do sometimes occur when the audiogram is not steeply sloping (Moore, Huss, Vickers, Glasberg and Alcántara 2000), and moderately steep slopes can occur in the absence of a dead region. Thus, the slope of the audiogram cannot be taken as a reliable indicator of the presence or absence of dead regions.

In adults with acquired hearing loss, losses up to about 55 dB may be associated purely with OHC dysfunction (Schuknecht 1974). Losses greater than this tend to be associated with both OHC and IHC dysfunction, and losses greater than 90 dB at high frequencies or 75–80 dB at low frequencies are often associated with dead regions (Moore et al. 2000; Moore 2001). However, in people with congenital loss, the picture is much less clear. It may often be the case that severe or profound hearing loss is caused by some factor other than hair cell dysfunction, for example malformation of one or more structures inside the cochlea (Kiernan and Steel 2000). Such malformations may also be associated with abnormal patterns of vibration on the basilar membrane. Thus, in cases of congenital hearing loss, the shape of the audiogram is an even less reliable indicator of the presence or absence of a dead region than is the case for adults with acquired hearing loss.

Diagnosis of Dead Regions Using Psychophysical Tuning Curves

To measure a psychophysical tuning curve (PTC) (Chistovich 1957; Small 1959), the signal is fixed in frequency and in level, usually at a level just above the absolute threshold, say, 10 dB Sensation Level (SL). The masker can be either a sinusoid or a narrow band of noise; often a band of noise is used to reduce the influence of beats between the signal and masker (Egan and Hake 1950; Moore, Alcántara and Dau 1998). For each of several masker center frequencies, the level of the masker needed just to mask the signal is determined. For normally hearing subjects, the tip of the PTC (i.e. the frequency at which the masker level is lowest) always lies close to the signal frequency (Vogten 1974; Moore 1978). Put another way, the masker is most effective when its frequency is close to that of the signal.

When hearing-impaired listeners are tested, PTCs have sometimes been found whose tips are shifted well away from the signal frequency (Thornton and Abbas 1980; Florentine and Houtsma 1983; Turner, Burns and Nelson 1983; Moore et al. 2000; Moore and Alcántara 2001). This can happen when the signal frequency falls in a dead region. For

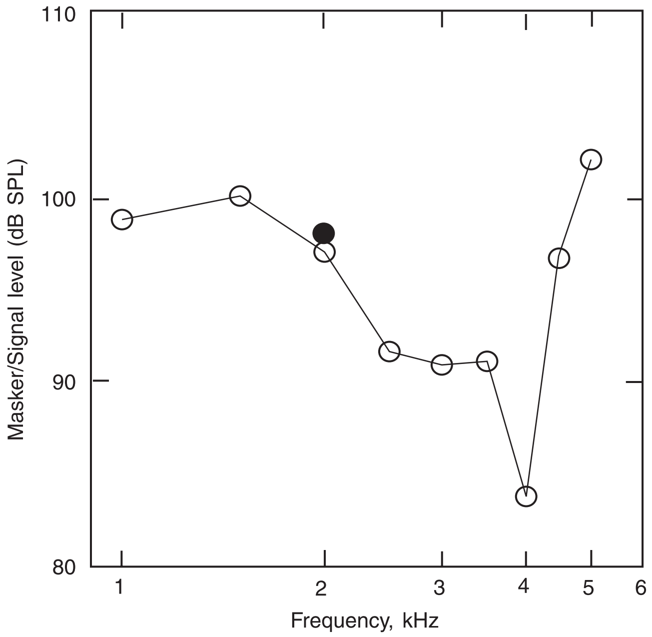

Figure 3. A PTC obtained from a patient with an extensive low-frequency dead region. The patient had a relatively flat hearing loss, but her hearing improved slightly around 4 kHz. The PTC was obtained using a 2-kHz signal (indicated by the filled circle). The tip of the PTC is clearly shifted, to a frequency of 4 kHz.

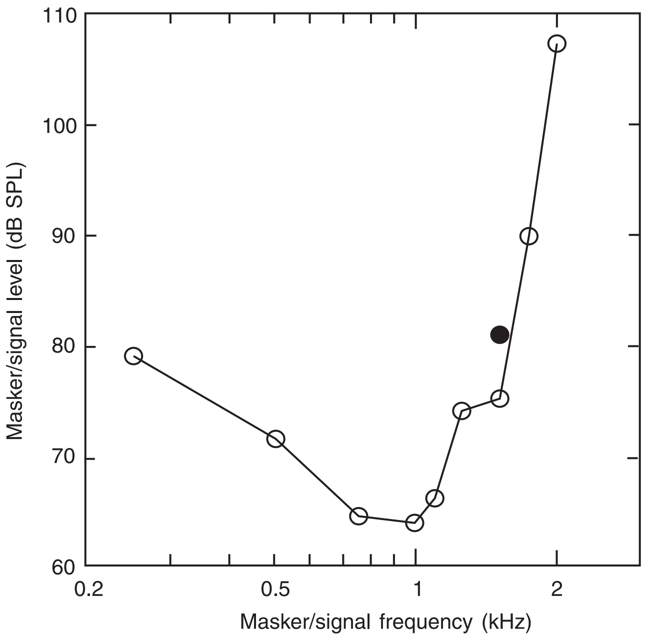

Figure 4. A PTC obtained from a subject with a high-frequency dead region. The PTC was obtained using a 1.5-kHz signal (indicated by the filled circle). The tip of the PTC is shifted towards lower frequencies.

example, when there is a low-frequency dead region, and the signal to be detected has a frequency within the dead region, the tip of the tuning curve lies well above the signal frequency. This is illustrated in figure 3. The subject had a relatively flat hearing loss, but her hearing improved slightly around 4 kHz. The PTC was obtained using a 2-kHz signal (indicated by the filled circle). The tip of the PTC is clearly shifted, to a frequency of 4 kHz. This suggests that the dead region extended up to about 4 kHz; the 2-kHz signal was detected at the 4-kHz place on the basilar membrane, and so the most effective masker frequency was 4 kHz. When a dead region falls at high frequencies, the signal is usually detected via the downward spread of excitation, and the tip of the PTC is shifted towards lower frequencies. An example is given in figure 4.

PTCs provide a useful way of detecting dead regions and defining their boundaries. When the tip of the PTC is shifted markedly from the signal frequency, then, to a first approximation, the frequency at the tip indicates the boundary of the dead region. However, the frequency at the tip of the PTC may not give an accurate estimate of the boundary when the shift is small (less than a few hundred Hertz), as beats between the signal and masker can influence the shape of the tip (Moore and Alcántara 2001).

Although PTCs have been very useful in laboratory studies of dead regions, they are time-consuming to measure, and the choice of an appropriate signal frequency and level can be difficult. Thus, they are not really suitable for use as a diagnostic tool in clinical practice. I describe next a simpler method of diagnosing dead regions, based on the use of a broadband noise masker.

The TEN Test

The TEN test is based upon the detection of sinusoids in the presence of a broadband noise, designed to produce almost equal masked thresholds (in dB SPL) over a wide frequency range, for normally hearing listeners. This noise is called threshold equalizing noise (TEN) (Moore et al. 2000). The noise level is specified in terms of the Sound Pressure Level in a 132-Hz wide band centred at 1000 Hz; this is also called the level per ERB, since the equivalent rectangular bandwidth of the normal auditory filter is 132 Hz for a center frequency of 1000 Hz. When the noise level is specified in this way, the "normal" masked threshold for any frequency within the range 250 to 10000 Hz is very close to the nominal noise level. For example, a nominal noise level of 70 dB/ERB should give a "normal" masked threshold of 70 dB SPL (Moore et al. 2000).

For listeners with moderate to severe cochlear hearing loss, but without a dead region at the signal frequency, masked thresholds in broadband noise are usually only 2–3 dB higher than for normally hearing listeners (Glasberg and Moore 1986; Tyler 1986). The same is true for the noise used in the TEN test (Moore et al. 2000). However, when a dead region is present, the masked threshold for a tone falling within the dead region can be markedly higher than normal. Such a tone will be detected using neurones with CFs remote from the signal frequency (hereafter called "off-frequency listening"). The amplitude of basilar-membrane vibration at the remote place will generally be less than the amplitude in the dead region. Therefore, a broadband noise will mask the tone much more effectively than would normally be the case, as the noise only has to mask the reduced response at the remote place.

An illustration of this rationale for a hypothetical patient with a high-frequency dead region is given in figure 5. Consider first the top panel. It is assumed that the dead region starts at about 1.07 kHz (indicated by the shaded area), and that the excitation level required to reach absolute threshold for frequencies below 1.07 kHz is 40 dB (indicated by the horizontal long-dashed line). The solid curve shows the excitation pattern for a tone with a frequency of 1.5 kHz, which falls within the dead region. The level of the tone has been chosen so that the excitation immediately adjacent to the dead region just reaches the threshold value. In other words the 1.5-kHz tone is at the level required to reach absolute threshold. This level is about 67 dB. Consider now the bottom panel. This shows the effect of adding a broadband noise which produces an equal excitation level of 70 dB at all frequencies (the TEN does not quite do this, but a constant excitation level is shown here for simplicity). The 1.5-kHz tone at 67 dB would be completely masked by this noise, as the noise would "swamp" the previously audible excitation at CFs just below 1.07 kHz. To restore audibility of the tone in the noise, its level has to be increased to the point where the tone-evoked excitation at CFs just below 1.07 kHz just exceeds 70 dB. The solid curve shows the excitation pattern for a tone level of 97 dB, which is the level required to achieve this. Thus, the masked threshold of the tone is 30 dB higher than the absolute threshold, and the masked threshold is 27 dB higher than the "normal" masked threshold, which would be around 70 dB.

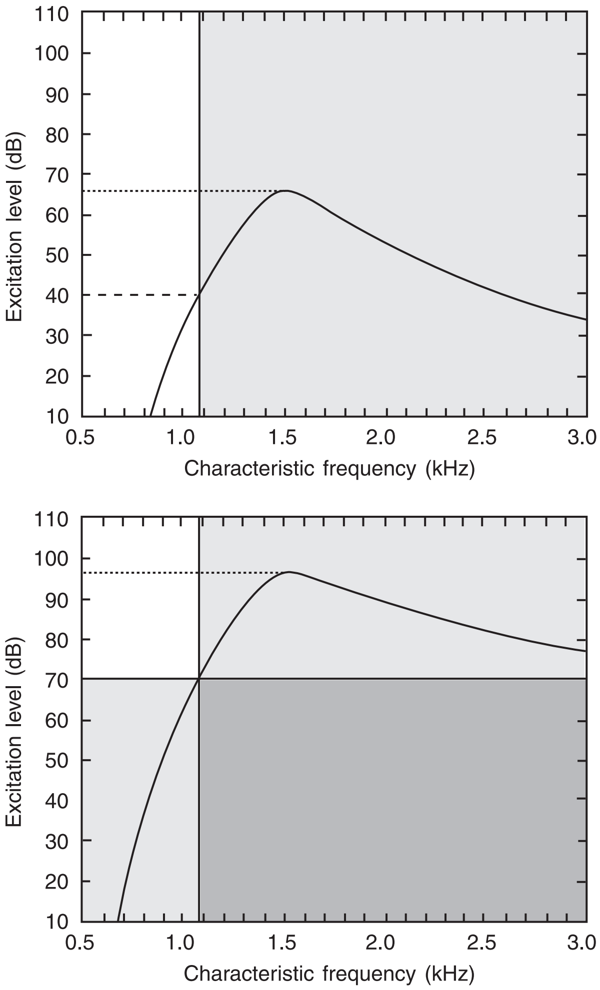

Figure 5. Illustration of the rationale behind the test using TEN for a hypothetical patient with a dead region above 1.07 kHz.

To assess the validity of the TEN test, Moore et al. (2000) measured PTCs using the same hearing-impaired listeners as tested with the TEN. The specific hypothesis tested was that higher-than-normal thresholds in the TEN would be associated with PTCs with shifted tips. Results for a hearing-impaired person who does not appear to have a dead region are shown in figure 6. The lower panel shows results obtained with the TEN, except that filled squares indicate absolute thresholds (obtained using the same

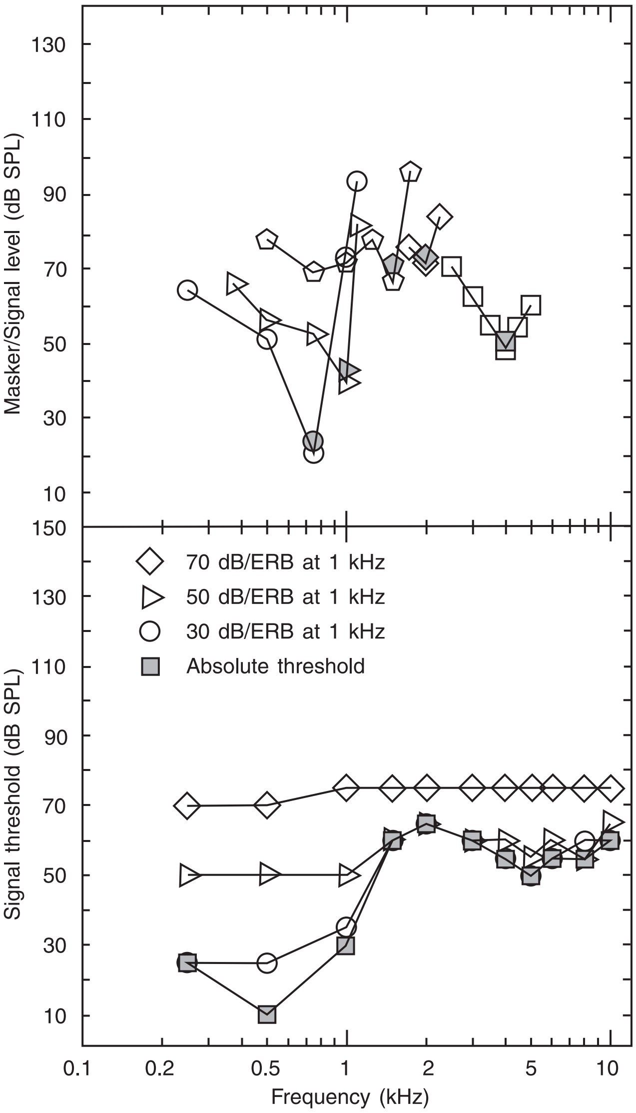

Figure 6. Results for a hearing-impaired person who does not have a dead region. The lower panel shows results obtained with the TEN, except that filled squares indicate absolute thresholds. The upper panel shows PTCs determined for five signal frequencies. In each case, the signal level and frequency are indicated by a filled symbol. The corresponding PTCs are indicated by open symbols of the same shape.

audiometer and earphones as employed for measuring thresholds in the TEN, and specified in dB SPL). This person has near-normal hearing for frequencies up to 1000 Hz, but a moderate loss at higher frequencies. Over the frequency range where the TEN produces masking, the masked thresholds are only slightly higher than normal, being around 75 dB for the TEN level of 70 dB/ERB.

The upper panel of figure 6 shows PTCs determined for five signal frequencies. In each case, the signal level and frequency are indicated by a filled symbol. The corresponding PTC is indicated by an open symbol of the same shape. For each PTC, the tip is close to the signal frequency. The PTCs are consistent with the results using the TEN, indicating that each signal was detected via IHCs/neurones with CFs close to the signal frequency.

Figure 7 shows an example of results for a hearing-impaired person who probably does have a dead region. This person has near-normal hearing for frequencies up to 1000 Hz, but a severe-to-profound loss at higher frequencies. In this figure, the symbols with up-pointing arrows indicate cases where the threshold was too high to be measured; the highest measurable threshold was 120 dB SPL. The specific symbol used with the arrow indicates the *lowest* TEN level for which a threshold could not be measured. For example, for a signal frequency of 3000 Hz, a masked threshold could be measured for the TEN level of 30 dB/ERB, but not for levels of 50 and 70 dB/ERB, so the symbol associated with the arrow at 3000 Hz is a diamond, the symbol for 50 dB/ERB. A filled square with an arrow indicates that the absolute threshold was too high to be measured.

For signal frequencies of 1500 Hz and above, masked thresholds in the 70 dB/ERB noise were 10 dB or more higher than the mean normal value. For signal frequencies from 3000 to 5000 Hz, the masked thresholds in the 30 dB/ERB noise were elevated above the absolute thresholds and were at 120 dB SPL, i.e. 90 dB higher than for normal-hearing subjects! This strongly suggests that tones with frequencies of 1500 Hz and above were being detected via IHCs/neurones with CFs below 1500 Hz.

The PTC for this subject for a signal frequency of 500 Hz has a tip at 500 Hz. However, the PTCs for signal frequencies of 1200 and 1500 Hz are shifted downwards to about 1000–1200 Hz. This suggests that the dead region starts at 1000–1200 Hz, and extends upwards from there, which is consistent with the finding that thresholds in the TEN were near normal for frequencies up to 1000 Hz, but were higher than normal for frequencies of 1500 Hz and above.

In total, Moore et al. (2000) tested 20 ears of 14 subjects with sensorineural hearing loss. Generally, there was a very good correspondence between the results obtained using the TEN and the PTCs; if, for a given signal frequency, the masked threshold in the TEN was 10 dB or more higher than normal *and* the

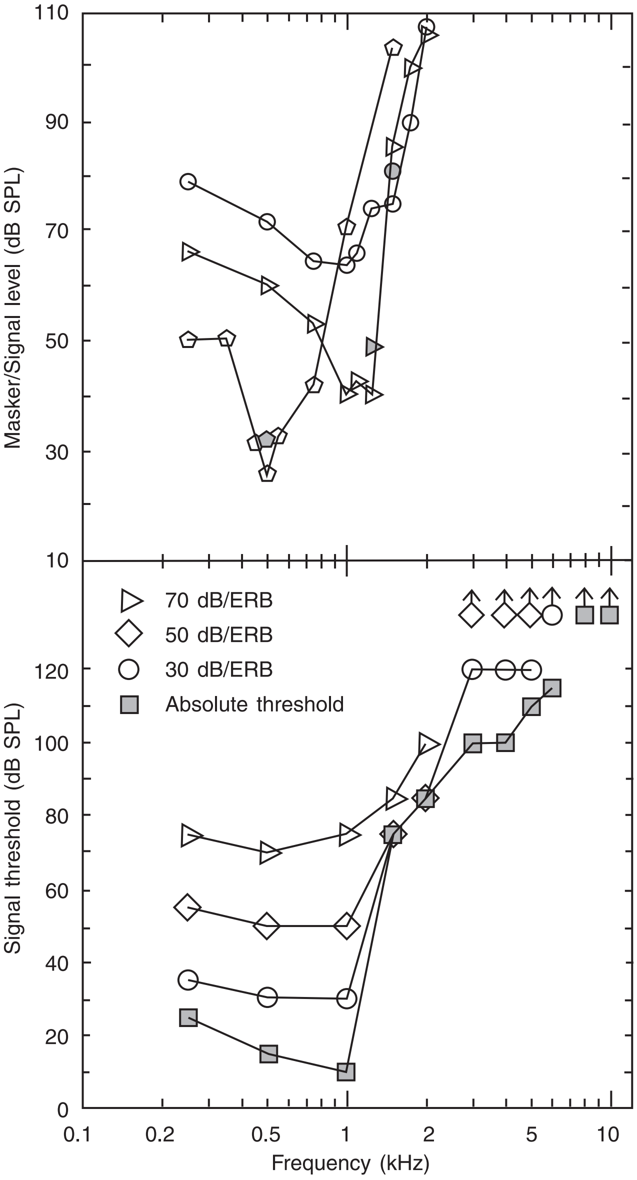

Figure 7. Results for a person with a dead region at high frequencies. Otherwise, as figure 6. Symbols with up-pointing arrows indicate cases where the threshold was too high to be measured; the highest measurable threshold (determined by equipment limitations) was 120 dB SPL. The specific symbol used with the arrow indicates the *lowest* TEN level for which a threshold could not be measured.

TEN produced at least 10-dB of masking (i.e. the masked threshold was 10 dB or more above the absolute threshold), then the tip of the PTC determined using that signal frequency was shifted. If the masked threshold in the TEN was not 10 dB or more higher than normal, the tip of the PTC was not shifted. Hence, the following "rule" was formulated: If the threshold in the TEN is 10 dB or more above the TEN level/ERB, and the TEN produces at least 10 dB of masking, this is indicative of a dead region at the signal frequency. However, some exceptions to this rule, and some limitations of the TEN test should be noted:

(1) For some hearing-impaired subjects, the absolute thresholds at high frequencies may be so high that they cannot be measured. In such cases it is not possible to determine whether the TEN produces masking at these frequencies. In all probability, such extreme losses are associated with dead regions, although it is difficult to demonstrate this directly. However, the thresholds in the TEN for frequencies where the hearing loss is not so extreme can be used to determine whether there is a dead region corresponding to these lower frequencies and, if so, to estimate the frequency at which the dead region starts.

(2) Sometimes, when the absolute threshold is high but still measurable, a dead region may be present (as indicated by a PTC with a shifted tip), but the TEN may not be sufficiently intense to produce 10 dB or more of masking. In some cases it may be impossible to make the TEN sufficiently intense, either because of limitations in the equipment, or because the TEN becomes uncomfortably loud. For most patients with moderate to severe hearing loss, a level of 70 dB/ERB is sufficient, but it may sometimes be necessary to use a TEN level of 80 or even 90 dB/ERB.

(3) When the signal frequency falls *just inside* a dead region, the threshold in the TEN may be less than 10 dB higher than the TEN level per ERB.

(4) Some subjects show higher than normal thresholds at *all* frequencies. This may indicate that they are "inefficient" or "cautious" listeners (Patterson and Moore 1986); they may need to hear the tone very clearly before indicating that they detect it at all. Alternatively, the high thresholds may be indicative of a problem in the central auditory system (Langenbeck 1965) or a dysfunction in the cochlea or auditory nerve linked to factors other than OHC or IHC damage. Such cases need to be treated with caution. A signal threshold that is 10 dB or more higher than normal may not indicate a dead region. A reasonable policy in cases where thresholds using the TEN are in the range 5–10 dB higher than normal,

even at frequencies where the hearing loss is mild or moderate, is to adopt a more stringent criterion for diagnosis of a dead region, for example, requiring thresholds in the TEN to be 15 dB or more higher than normal.

Application of the TEN Test in Cases of Congenital Hearing Loss

Recently, Munro, Killen and Moore (in press) used the TEN test with 31 teenagers, mostly having severe to profound hearing loss. Their average age was 14 years. The hearing loss was classified as congenital in 22 subjects, acquired in 5 subjects and unknown in 4 subjects. Test signals were derived from a CD (available from Starkey: contact details can be found at http://hearing.psychol.cam.ac.uk/), routed through a Grason-Stadler GSI16 audiometer, and presented via Sennheiser HD580 earphones. The TEN was typically presented at a level of 80–90 dB/ERB as this was sufficient to be audible without causing discomfort. Over 60% of the subjects met the criteria described above, apparently indicating a dead region in at least one ear. All five of the subjects with acquired hearing loss met the criteria. More than 50% of the subjects with congenital hearing loss met the criteria.

Although these results seem to indicate a high prevalence of dead regions in teenagers with acquired or congenital hearing loss, there are several problems associated with the administration and interpretation of the test. Firstly, there were many cases where the results were inconclusive, as the TEN level was not sufficient to raise masked threshold 10 dB or more above absolute threshold. Secondly, there were many cases where the criteria were only just met. For example, the masked threshold was often just 10 dB higher than the level per ERB of the TEN. This criterion was chosen partly on the basis of results obtained from subjects with moderate to severe hearing loss who did not have dead regions, as indicated by PTCs without shifted tips (Moore et al. 2000). For these subjects, the masked threshold was usually within 10 dB of the level per ERB of the TEN. However, when dealing with subjects with profound hearing loss, it may be appropriate to use a more stringent criterion. The auditory filters of subjects with profound hearing loss may be very broad (Faulkner, Rosen and Moore 1990), and this may lead to unusually high masked thresholds even for frequencies where there is no dead region.

Part of the difficulty in using the TEN test in cases of profound hearing loss stems from the fact that the noise stimulus was designed to be suitable as a masker for a very wide range of signal frequencies, from about 250 Hz up to 10 kHz (Moore et al. 2000). The use of a single masker for all signal frequencies was intended to simplify the administration and interpretation of the test (Moore 2001). However, the use of a broadband masker has the drawback that the overall level is considerably higher than the level per ERB, and it is the overall level that limits the maximum possible presentation level. To identify dead regions in subjects with severe to profound hearing loss, it may be more appropriate to use a narrowband masker, as is done in the determination of PTCs. However, the limited dynamic range available may make it difficult to obtain meaningful results, even when measuring PTCs.

Effects of Dead Regions on Speech Intelligibility

Several studies have shown that only limited information can be extracted from frequency components of speech falling in a dead region. Hence, it is to be expected that speech intelligibility will be relatively poor (even for amplified speech) when extensive dead regions are present within the frequency range that is most important for speech intelligibility (about 500 to 5000 Hz; see ANSI 1969; ANSI 1997). In what follows, I will focus on two studies of speech intelligibility conducted in my laboratory, in which high-frequency dead regions were identified using both PTCs and the TEN test. Both studies were conducted using adult subjects, mostly with acquired hearing loss. For a more extensive review, see Moore (2001).

In the first study (Vickers et al. 2001), all subjects had high-frequency hearing loss, but some had high-frequency dead regions and some did not; generally, the subjects with dead regions had more severe high-frequency hearing losses than those without dead regions. The speech stimuli were vowel-consonant-vowel (VCV) nonsense syllables, presented in quiet, using one of three vowels (/i/, /a/ and /u/) and 21 different consonants. In a baseline condition, subjects were tested using broadband stimuli (upper frequency limit 7500 Hz) with a nominal input level of 65 dB SPL. Prior to presentation via Sennheiser HD580 earphones, the stimuli were subjected to the frequency-gain characteristic prescribed by the

"Cambridge" formula (Moore and Glasberg 1998). This formula is intended to give speech at 65 dB SPL the same overall loudness as for a normal listener, and to make the average specific loudness (the loudness per ERB or per critical band; see Moore and Glasberg 1997) of the speech the same for all frequencies over the range important for speech intelligibility, i.e. about 500 to 5000 Hz. Of course, this is only possible for a listener without a dead region in that frequency range. For a listener with a dead region, the specific loudness is zero for all critical bands falling within the dead region. In any case, the goal of the frequency-dependent amplification was to restore audibility as far as possible, while avoiding excessive loudness. The appropriate amplification was calculated and applied separately for each ear of each subject. The stimuli for all other conditions were initially subjected to this same frequency-gain characteristic. Then, the speech was lowpass filtered with various cutoff frequencies.

All subjects were given practice to familiarize them with the task. At least two lists of 63 tokens were used for each condition. These were always presented in different test sessions. The order of testing of the different cutoff frequencies was randomized in the first test session. This order was reversed for the second session to balance the effects of practice and fatigue. For subjects without dead regions, performance generally improved progressively with increasing cutoff frequency. An example is shown in the upper panel of figure 8. This indicates that they were able to make use of high-frequency information. For subjects with dead regions, two patterns of performance were observed. For some subjects, performance initially improved with increasing cutoff frequency and then reached an asymptote (figure 8, middle). This indicates that they were not able to make use of high-frequency information. For other subjects, performance initially improved with increasing cutoff frequency, and then worsened with further increases (figure 8, bottom). This indicates that amplification of high frequencies impaired performance.

To give an overall impression of the pattern of results for the subjects having dead regions with edge frequencies below 2000 Hz, the cutoff frequencies used for each ear of each subject were expressed relative to the estimated edge frequency of the dead region for that ear and subject. The data for each ear (percent correct versus relative frequency) were fitted with a cubic-spline function. The cubic-spline functions were then averaged across ears. The result is shown in figure 9 (solid curve). For comparison, a similar analysis was applied to the results for the subjects without dead regions; these results are plotted in figure 9 as a function of absolute (not relative) frequency in kHz (dashed curve). The two frequency scales are roughly comparable, as a relative frequency of 1 corresponds, on average, to a frequency a little below 1 kHz; the geometric mean of the estimated edge frequencies of the dead regions was 942 Hz.

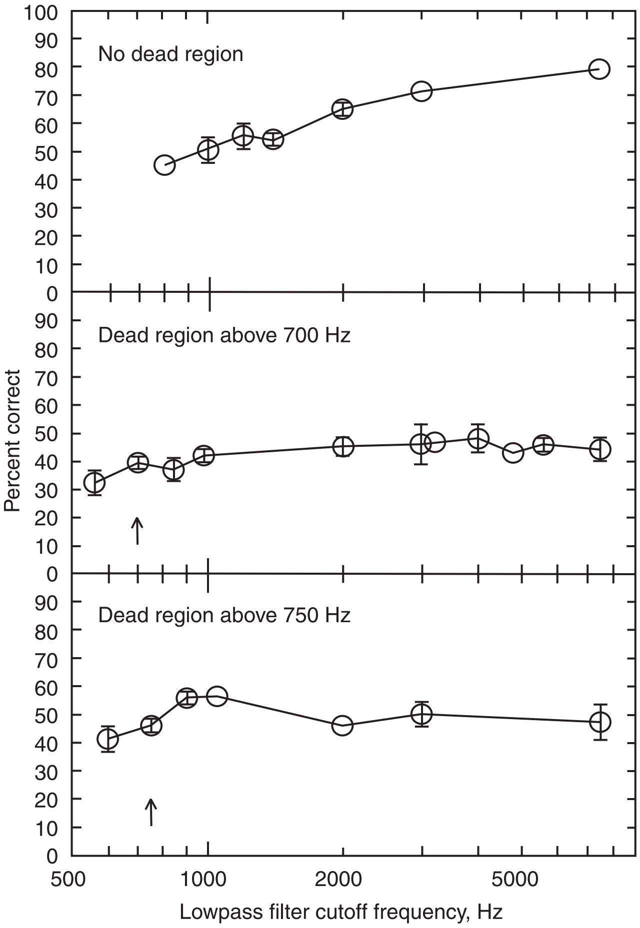

Figure 8. Percent correct scores in identifying VCV syllables for three hearing-impaired subjects, one without (top) and two with (middle, bottom) a dead region. Scores are plotted as a function of the cutoff frequency of a lowpass filter. Prior to lowpass filtering, stimuli were given the frequency-gain characteristic prescribed by the "Cambridge" formula (Moore and Glasberg 1998). Error bars indicate ± one standard deviation across test sessions.

For the subjects with dead regions, the fitted function increases with increasing relative frequency up to about 1.7, and then flattens off. This indicates

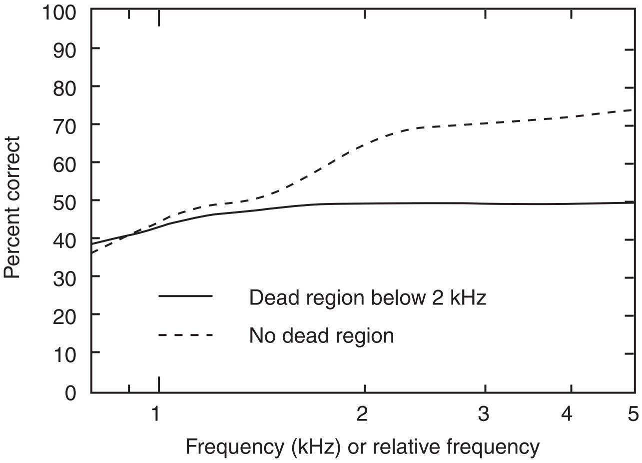

Figure 9. Mean cubic-spline functions fitted to the data (percent correct identification of VCVs in quiet) for subjects without dead regions (dashed curve) and with dead regions starting below 2000 Hz (solid curve). For the group without dead regions, the percent correct is plotted as a function of filter cutoff frequency in kHz. For the group with dead regions, frequency is expressed relative to the estimated edge frequency of the dead region.

that there is typically some benefit to intelligibility of amplifying frequencies up to about 70% above the estimated edge frequency of the dead region. For the subjects without dead regions, the fitted function increases progressively with increasing cutoff frequency, indicating that broadband amplification gives the best performance.

It is noteworthy that, for subjects who showed an "optimum" cutoff frequency, the best performance was achieved when that cutoff frequency was 50–100% above the estimated edge frequency of the dead region. For subjects whose performance reached an asymptote, the asymptote was reached for a cutoff frequency about 70% above the estimated edge frequency of the dead region. To assess the extent to which the subjects with dead regions were able to make use of information from frequencies just above the estimated edge frequency of the dead region, scores were compared for the lowpass filter cutoff frequency closest to the estimated edge frequency, and for the cutoff frequency closest to 1.7 times the estimated edge frequency. This analysis included all twelve ears with dead regions. The mean scores for these two conditions, 50.6 and 55.9%, respectively, differed significantly ($p < 0.001$). Thus, there was a significant benefit from amplifying frequencies up to 70% above the estimated edge frequencies of the dead regions.

In the second study (Baer et al. in press), the design was similar, but stimuli were presented in a speech-shaped background noise. Ten subjects were used, five with high-frequency dead regions extending below 2 kHz and five with high-frequency hearing loss without dead regions. The noise level was chosen individually for each subject to reduce intelligibility by about 15% when the cutoff frequency was about 1.7 times the estimated edge frequency of the dead region (or the edge of the high-frequency loss for subjects without dead regions). The pattern of results was similar to that found for speech in quiet. For all subjects, performance was worst for the lowest cutoff frequencies. For subjects without dead regions, performance generally improved progressively with increasing cutoff frequency. For most subjects with dead regions, performance improved with increasing cutoff frequency until the cutoff frequency was 1.5 to 2 times the edge frequency of the dead region, but hardly changed with further increases. For a few subjects with dead regions, performance improved with increasing cutoff frequency and then worsened somewhat with further increases. Cubic-spline fits to the data were calculated in the same way as before. The results are shown in figure 10. The cubic-spline fits confirm that subjects with dead regions fail to benefit from amplification of components more than about 70% above the edge frequency of the dead region, while subjects without dead regions do benefit from amplification of high frequencies.

The fact that there is some benefit from restoring the audibility of frequency components falling a little

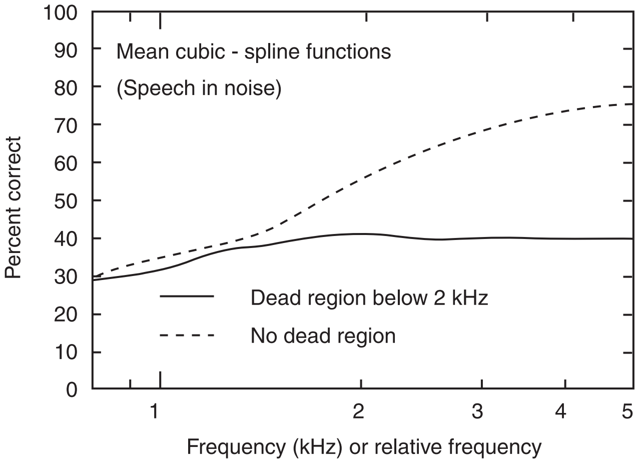

Figure 10. As figure 9, but for VCVs presented in a background of speech-shaped noise.

inside a dead region suggests that useful information can be extracted from speech even when some of that information is transmitted via the "wrong" place in the cochlea (Moore 2001; Shannon, Zeng and Wygonski 1998).

Implications for the Fitting of Hearing Aids

The studies described above suggest that, for adults with high-frequency dead regions, there is little or no benefit to speech discrimination from amplifying frequencies *well inside* a dead region but there may be some benefit in amplifying frequencies up to 50–100% above the estimated edge frequency of the dead region. For patients *without* high-frequency dead regions, amplification of the high frequencies can be (and usually is) beneficial (Skinner and Miller 1983; Vickers et al. 2001; Baer et al. in press). Hence, before deciding what form of amplification should be provided for a patient with high-frequency hearing loss, it is important to determine whether that patient has a high-frequency dead region, and, if so, what its extent is. The TEN test seems appropriate for this purpose for adults with moderate to severe hearing loss. However, more work is needed to determine the applicability and validity of the TEN test for patients with severe to profound hearing loss. For such cases, other methods may be needed.

Appropriate diagnosis may be particularly important, but also particularly difficult in children. Psychoacoustic measurements with children tend to be more variable than those with adults, and masked detection thresholds are often higher than in adults (Hall and Grose 1990; Veloso, Hall, and Grose 1990; Hall and Grose 1991; Buss, Hall, Grose and Dev 1999). Therefore, application of tests like the TEN test is more difficult, and the criteria required for positive diagnosis of a dead region may need to be altered. Possibly, electrophysiological methods can be developed for the diagnosis of dead regions in children.

Assuming that a dead region has been diagnosed at high frequencies, there may be several benefits of reducing the gain for frequencies well inside the dead region. Firstly, this may actually lead to improved speech intelligibility, as described above. Secondly, it may reduce problems associated with acoustic feedback. Thirdly, it may reduce distortion in the hearing aid, especially intermodulation distortion. Finally, it allows the dispenser to concentrate efforts on providing appropriate amplification over the frequency range where there is useful residual hearing.

Many modern hearing aids incorporate some form of automatic gain control or compression, and there is increasing evidence that compression is beneficial, both in adults (Laurence, Moore and Glasberg 1983; Moore, Johnson, Clark and Pluvinage 1992; Moore 1993; Hickson 1994; Dillon 1996; Moore 1998) and in children (Stelmachowicz, Kopun, Mace, Lewis and Nittrouer 1995; Jenstad, Seewald, Cornelisse and Shantz 1999). Methods for the initial fitting of multi-channel compression hearing aids have been developed by several research groups. Some of these methods, for example the "Camfit" methods developed in my laboratory (Moore, Glasberg and Stone 1999; Moore 2000; Moore, Alcántara and Marriage 2001), are based on a loudness model (Moore and Glasberg 1997), and have the default assumption that no dead region exists. Before using such methods, a test for the diagnosis of dead regions should be applied (such as the TEN test) whenever the audiogram indicates the possibility of a dead region. When a dead region is diagnosed, the gains recommended by the fitting method should be applied *only* for frequencies where there is not a dead region, and perhaps for frequencies extending 50–100% inside any dead region.

Another method for the fitting of hearing aids, the NAL-NL1 method (Dillon 1999), is partly based on the same loudness model, but is also based on empirical data on the effects of amplification of speech for hearing-impaired listeners with varying degrees of hearing loss (Ching, Dillon and Byrne 1998; Hogan and Turner 1998; Souza and Turner 1999; Turner and Cummings 1999). These data show that, *on average*, people with high-frequency hearing loss get progressively less benefit from amplification of the high frequencies as the hearing loss increases. This is taken into account in the fitting method, by reducing the high-frequency gain progressively below the value needed to restore audibility as the hearing loss increases.

In my view, this approach, based on data averaged across listeners, is inappropriate, since some of the listeners probably had dead regions and some did not. For listeners without dead regions, amplification of the high frequencies so as to restore audibility usually leads to improved intelligibility. For listeners with dead regions at high frequencies, amplification of frequencies well inside the dead region is unlikely to be beneficial. Gains based on averaging data across these two classes of listeners are likely to be inappropriate for both classes.

Conclusions

Dead regions are regions of the cochlea where there are no functioning inner hair cells and/or neurones. High-frequency dead regions appear to be relatively common in adults with acquired hearing loss, when the hearing loss at high frequencies exceeds about 65–70 dB. However, the audiogram does not give a reliable indication of the presence or absence of a dead region. Dead regions can be diagnosed by measurement of PTCs or using the TEN test. However, these tests are difficult to apply in cases of severe to profound loss, and their applicability in children with congenital loss remains to be established. Results for adult listeners indicate that there is no benefit for the intelligibility of speech in quiet or in noise of amplifying frequencies well inside a high-frequency dead region. However, there often is benefit from amplifying frequencies up to about 1.7 times the estimated edge frequency of the dead region. The optimal amplification strategies for children with high-frequency dead regions remain to be established.

Acknowledgements

I thank José Alcántara, Thomas Baer, Brian Glasberg, Martina Huss, Michael Stone, Dianne van Tasell and Deborah Vickers for their collaboration in the work reported in this chapter. I also thank José Alcántara, Thomas Baer, Brian Glasberg, Martina Huss and Thomas Stainsby for helpful comments on an earlier version of this paper. The work was supported mainly by the MRC (UK), with additional support from Starkey (USA), The RNID (UK) and Defeating Deafness (UK).

References

ANSI 1969. *ANSI S3.5. Methods for the calculation of the articulation index.* New York: American National Standards Institute.

ANSI 1997. *ANSI S3.5–1997, Methods for the calculation of the speech intelligibility index.* New York: American National Standards Institute.

Baer, T., Moore, B.C.J., and Kluk, K. in press. Effects of low-pass filtering on intelligibility of speech in noise for listeners with and without dead regions at high frequencies. *International Journal of Audiology*.

Borg, E., Canlon, B., and Engström, B. 1995. Noise-induced hearing loss – Literature review and experiments in rabbits. *Scandinavian Audiology* 24, Suppl. 40: 1–147.

Buss, E., Hall, J.W., Grose, J.H., and Dev, M.B. 1999. Development of adult-like performance in backward, simultaneous, and forward masking. *Journal of Speech, Language and Hearing Research* 42: 844–849.

Ching, T., Dillon, H., and Byrne, D. 1998. Speech recognition of hearing-impaired listeners: Predictions from audibility and the limited role of high-frequency amplification. *Journal of the Acoustical Society of America* 103: 1128–1140.

Chistovich, L.A. 1957. Frequency characteristics of masking effect. *Biophysics* 2: 743–755.

Dillon, H. 1996. Compression? Yes, but for low or high frequencies, for low or high intensities, and with what response times? *Ear and Hearing* 17: 287–307.

Dillon, H. 1999. NAL-NL1: A new prescriptive fitting procedure for non-linear hearing aids. *Hearing Journal* 52: 10–16.

Egan, J.P., and Hake, H.W. 1950. On the masking pattern of a simple auditory stimulus. *Journal of the Acoustical Society of America* 22: 622–630.

Engström, B. 1983. Stereocilia of sensory cells in normal and hearing impaired ears. *Scandinavian Audiology* Suppl. 19: 1–34.

Faulkner, A., Rosen, S., and Moore, B.C.J. 1990. Residual frequency selectivity in the profoundly hearing-impaired listener. *British Journal of Audiology* 24: 381–392.

Florentine, M., and Houtsma, A.J.M. 1983. Tuning curves and pitch matches in a listener with a unilateral, low-frequency hearing loss. *Journal of the Acoustical Society of America* 73: 961–965.

Friedman, I. 1997. Pathology of the cochlea. In J.B. Booth (ed.), *Scott-Brown's otolaryngology, Vol. 3: Otology* (pp. 3/4/1–3/4/61). Oxford: Butterworth-Heinemann.

Glasberg, B.R., and Moore, B.C.J. 1986. Auditory filter shapes in subjects with unilateral and bilateral cochlear impairments. *Journal of the Acoustical Society of America* 79: 1020–1033.

Gravendeel, D.W., and Plomp, R. 1960. Perceptive bass deafness. *Acta Otolaryngologica* 51: 549–560.

Hall, J.W., and Grose, J.H. 1990. The masking-level difference in children. *Journal of the American Academy of Audiology* 1: 81–88.

Hall, J.W., and Grose, J.H. 1991. Notched-noise measures of frequency selectivity in adults and children using fixed-masker-level and fixed-signal-level presentation. *Journal of Speech and Hearing Research* 34: 651–660.

Halpin, C., Thornton, A., and Hasso, M. 1994. Low-frequency sensorineural loss: Clinical evaluation and implications for hearing aid fitting. *Ear and Hearing* 15: 71–81.

Hickson, L.M.H. 1994. Compression amplification in hearing aids. *American Journal of Audiology* 3: 51–65.

Hogan, C.A., and Turner, C.W. 1998. High-frequency audibility: Benefits for hearing-impaired listeners. *Journal of the Acoustical Society of America* 104: 432–441.

Jenstad, L.M., Seewald, R.C., Cornelisse, L.E., and Shantz, J. 1999. Comparison of linear gain and wide dynamic range compression hearing aid circuits: Aided speech perception measures. *Ear and Hearing* 20: 117–126.

Kiernan, A.E., and Steel, K.P. 2000. Mouse homologues for human deafness. *Advances in Otorhinolaryngology* 56: 233–243.

Langenbeck, B. 1965. *Textbook of practical audiometry*. London: Edward Arnold.

Laurence, R.F., Moore, B.C.J., and Glasberg, B.R. 1983. A comparison of behind-the-ear high-fidelity linear aids and two-channel compression hearing aids in the laboratory and in everyday life. *British Journal of Audiology* 17: 31–48.

Moore, B.C.J. 1978. Psychophysical tuning curves measured in simultaneous and forward masking. *Journal of the Acoustical Society of America* 63: 524–532.

Moore, B.C.J. 1993. Signal processing to compensate for reduced dynamic range. In J. Beilin and G.R. Jensen (eds.), *Recent developments in hearing instrument technology* (pp. 147–165). Copenhagen: Stougaard Jensen.

Moore, B.C.J. 1997. *An introduction to the psychology of hearing*, 4th ed. San Diego: Academic.

Moore, B.C.J. 1998. *Cochlear hearing loss*. London: Whurr.

Moore, B.C.J. 2000. Use of a loudness model for hearing aid fitting. IV. Fitting hearing aids with multi-channel compression so as to restore "normal" loudness for speech at different levels. *British Journal of Audiology* 34: 165–177.

Moore, B.C.J. 2001. Dead regions in the cochlea: Diagnosis, perceptual consequences, and implications for the fitting of hearing aids. *Trends in Amplification* 5: 1–34.

Moore, B.C.J., and Alcántara, J.I. 2001. The use of psychophysical tuning curves to explore dead regions in the cochlea. *Ear and Hearing* 22: 268–278.

Moore, B.C.J., Alcántara, J.I., and Dau, T. 1998. Masking patterns for sinusoidal and narrowband noise maskers. *Journal of the Acoustical Society of America* 104: 1023–1038.

Moore, B.C.J., Alcántara, J.I., and Marriage, J.E. 2001. Comparison of three procedures for initial fitting of compression hearing aids. I. Experienced users, fitted bilaterally. *British Journal of Audiology* 35: 339–363.

Moore, B.C.J., and Glasberg, B.R. 1983. Suggested formulae for calculating auditory-filter bandwidths and excitation patterns. *Journal of the Acoustical Society of America* 74: 750–753.

Moore, B.C.J., and Glasberg, B.R. 1997. A model of loudness perception applied to cochlear hearing loss. *Auditory Neuroscience* 3: 289–311.

Moore, B.C.J., and Glasberg, B.R. 1998. Use of a loudness model for hearing aid fitting. I. Linear hearing aids. *British Journal of Audiology* 32: 317–335.

Moore, B.C.J., Glasberg, B.R., and Stone, M.A. 1999. Use of a loudness model for hearing aid fitting. III. A general method for deriving initial fittings for hearing aids with multi-channel compression. *British Journal of Audiology* 33: 241–258.

Moore, B.C.J., Huss, M., Vickers, D.A., Glasberg, B.R., and Alcántara, J.I. 2000. A test for the diagnosis of dead regions in the cochlea. *British Journal of Audiology* 34: 205–224.

Moore, B.C.J., Johnson, J.S., Clark, T.M., and Pluvinage, V. 1992. Evaluation of a dual-channel full dynamic range compression system for people with sensorineural hearing loss. *Ear and Hearing* 13: 349–370.

Munro, K.J., Killen, T., and Moore, B.C.J. in press. Dead regions in the cochlea of hearing-impaired teenagers. *International Journal of Audiology*.

Patterson, R.D., and Moore, B.C.J. 1986. Auditory filters and excitation patterns as representations of frequency resolution. In B.C.J. Moore (ed.), *Frequency selectivity in hearing* (pp. 123–177). London: Academic.

Ruggero, M.A., Rich, N.C., Recio, A., Narayan, S.S., and Robles, L. 1997. Basilar-membrane responses to tones at the base of the chinchilla cochlea. *Journal of the Acoustical Society of America* 101: 2151–2163.

Schucknecht, H.F. 1964. Further observations on the pathology of presbycusis. *Archives of Otolaryngology* 80: 369–382.

Schuknecht, H.F. 1974. *Pathology of the ear*. Cambridge, MA: Harvard University Press.

Sellick, P.M., Patuzzi, R., and Johnstone, B.M. 1982. Measurement of basilar membrane motion in the guinea pig using the Mössbauer technique. *Journal of the Acoustical Society of America* 72: 131–141.

Shannon, R.V., Zeng, F.-G., and Wygonski, J. 1998. Speech recognition with altered spectral distribution of envelope cues. *Journal of the Acoustical Society of America* 104: 2467–2476.

Skinner, M.W., and Miller, J.D. 1983. Amplification bandwidth and intelligibility of speech in quiet and noise for listeners with sensorineural hearing loss. *Audiology* 22: 253–279.

Small, A.M. 1959. Pure-tone masking. *Journal of the Acoustical Society of America* 31: 1619–1625.

Souza, P.E., and Turner, C.W. 1999. Quantifying the contribution of audibility to recognition of compression-amplified speech. *Ear and Hearing* 20: 12–20.

Stelmachowicz, P.G., Kopun, J., Mace, A., Lewis, D.E., and Nittrouer, S. 1995. The perception of amplified speech by listeners with hearing loss: Acoustic correlates. *Journal of the Acoustical Society of America* 98: 1388–1399.

Thornton, A.R., and Abbas, P.J. 1980. Low-frequency hearing loss: Perception of filtered speech, psychophysical tuning curves, and masking. *Journal of the Acoustical Society of America* 67: 638–643.

Turner, C.W., Burns, E.M., and Nelson, D.A. 1983. Pure tone pitch perception and low-frequency hearing loss. *Journal of the Acoustical Society of America* 73: 966–975.

Turner, C.W., and Cummings, K.J. 1999. Speech audibility for listeners with high-frequency hearing loss. *American Journal of Audiology* 8: 47–56.

Tyler, R.S. 1986. Frequency resolution in hearing-impaired listeners. In B.C.J. Moore (ed.), *Frequency selectivity in hearing* (pp. 309–371). London: Academic Press.

Veloso, K., Hall, J.W., and Grose, J.H. 1990. Frequency selectivity and comodulation masking release in adults and in six-year old children. *Journal of Speech and Hearing Research* 33: 96–102.

Vickers, D.A., Moore, B.C.J., and Baer, T. 2001. Effects of lowpass filtering on the intelligibility of speech in quiet for people with and without dead regions at high frequencies. *Journal of the Acoustical Society of America* 110: 1164–1175.

Vogten, L.L.M. 1974. Pure-tone masking: A new result from a new method. In E. Zwicker and E. Terhardt (eds.), *Facts and models in hearing* (pp. 142–155). Berlin: Springer-Verlag.

Wright, A., Davis, A., Bredberg, G., Ulehlova, L., and Spencer, H. 1987. Hair cell distributions in the normal human cochlea. *Acta Otolaryngologica* Suppl. 444: 1–48.

Zwicker, E. 1970. Masking and psychological excitation as consequences of the ear's frequency analysis. In R. Plomp and G.F. Smoorenburg (eds.), *Frequency analysis and periodicity detection in hearing* (pp. 376–394). Leiden: Sijthoff.

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