

Hearing loss can muddy the waters of otologic disease detection

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A few decades ago, there was a strong movement to develop non-invasive physiological measures correlated to the presence of hearing impairment. For simplicity, let us define hearing impairment as difficulty in processing acoustic information. The impetus was identification of hearing loss in infants and children in whom behavioral measures were difficult to interpret. In the ensuing years, reliable physiological measures correlated to hearing loss of cochlear origin have been developed. We now have physiological measures to screen for hearing loss in infants and children. Currently, we are focused on the clinical refinement of these measures to detect and quantify the degree of hearing impairment more quickly, more easily and with greater accuracy.

While a major focus was the detection of hearing impairment in infants and young children, there was parallel development of physiological measures to aid in the diagnosis of hearing problems in adults. However, the focus was not to detect the presence of hearing loss because reliable behavioral measures were available. Rather, the focus was on determining either the presence or the underlying cause of the hearing problem including any neurological problem affecting the auditory central nervous system. Thus, the question is not whether there is hearing impairment, but rather, is the impairment in the cochlea, auditory nerve, or in the higher nervous system?

Peripheral hearing impairment (i.e., cochlear insult) is a common manifestation of otologic diseases. However, often in adults, the clinical goal is not simply to establish the presence of this peripheral hearing impairment but to detect objectively the presence of a specific underlying otologic disease. In the search for physiological correlates of a specific otologic disease, we often find that the simple presence of hearing loss confounds the correlated physiological measures and dilutes their diagnostic value. Two obvious solutions to this problem are: (1) determine ways to compensate for the confounding effect of the hearing impairment on the physiological measure, or (2) develop physiological measures that are essentially unaffected by the hearing loss. This paper provides examples of these confounds and solutions when using auditory brainstem responses (ABRs) measures.

ABRs have been used extensively over the years to assess hearing impairment. For many clinicians, there was high expectation for what ABRs could tell us about auditory function. From this author's viewpoint, due to the lack of understanding of what ABRs represent, the over-interpretation of these measures, and the inappropriate use has led to disappointment as a reliable and accurate measure for assessing auditory function. However, its proper use, interpretation, and implementation can provide valuable information about the hearing impairment.

INTRODUCTION

Hearing impairment in infants and children: Pre-cochlear and cochlear origins

For infants and children, the main focus of physiological measures has been for screening for the presence of hearing loss and determining the degree and the audiometric configuration of any cochlear hearing loss. A cochlear loss is the primary diagnosis after ruling out conductive problems and the risks for auditory neuropathy and other neurological problems. In addition to ABRs in response to clicks and tones, other physiological measures such as otoacoustic emissions (OAEs) and auditory steady-state responses (ASSRs) can be used to make a rough estimate of the cochlear hearing loss using a variety of stimuli. Typically, for screening purposes the response parameter of the physiological measure is simply the presence of a response at varying levels of stimulation for a given stimulus. Usually, the response in terms of the latency and amplitudes of peak components is not of diagnostic interest, but rather, the response threshold for the given stimulus. Further specific and more accurate information regarding the degree and configuration of the hearing loss require more sophisticated testing paradigms and stimuli. Nonetheless, the major parametric measure involved is the assessment of response threshold; thus, the physiological correlates to the hearing impairment are rather straightforward in this respect. Of course, there are difficulties in determining response thresholds accurately and quickly.

Hearing impairment in adults: Cochlear and retro-cochlear diseases

As mentioned earlier, the use of physiological measures such as ABRs in adults is not to ascertain the presence of hearing impairment. Audiometric behavioral measures can easily clinically define the presence, the degree, and the audiometric configuration of the hearing loss. ABR testing in adults is typically aimed at determining the cause, origin and location of the impairment in the auditory system. Thus, response parameters such as peak latencies and amplitudes are measured and analyzed. To this end, there have been numerous studies over the years correlating these measures to varying cochlear and retro-cochlear pathologies. In particular, for those concerned with otology, objective physiological measures correlated to the presence of acoustic tumors (i.e., vestibular schwannomas) and Meniere's disease/ cochlear hydrops in adults are desirable to assist with the diagnoses.

The problem with physiological measures correlated with hearing impairment

Figure 1 is a brief schematic of factors leading to hearing impairment and abnormal physiological responses. It can easily be seen that there are a number of factors that can lead to hearing impairment which in turn yields abnormal physiological responses. Detection of hearing impairment using physiological measures is generally easily accomplished using simple measures of response presence and response thresholds. Determining if the hearing impairment is cochlear or retro-cochlear or even some combination is a bit more difficult but can often be achieved using a combination of and/or more sophisticated test measures. Similarly, determining the degree and the configuration of cochlear hearing loss associated with the impairment are also more difficult but also can be frequently achieved using more sophisticated test measures or combination of tests to get

fairly decent estimates. Thus, detecting hearing impairment and tracking back in Figure 1 from the abnormal physiological responses to determining whether the origin is cochlear or retro-cochlear, is achievable as demonstrated by a number of studies. The real problem is to go back further in Figure 1 to the source, location and/or cause of the hearing impairment. Can the abnormal physiological responses provide information about the underlying disease responsible for the impairment? The following is a discussion of various ABR measures and some of the typical abnormalities used in the diagnosis of auditory processing (see Don and Kwong, 2002).

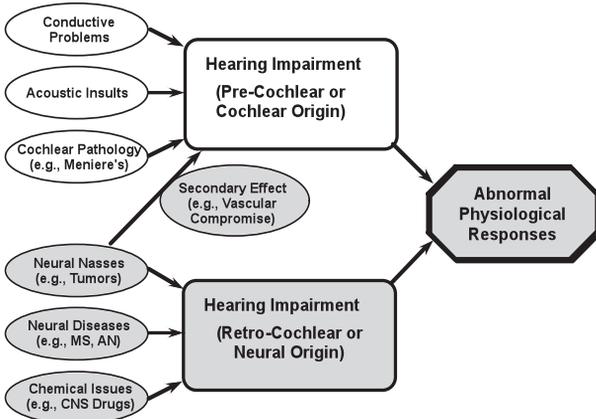


Fig. 1: Schematic of factors leading to hearing impairment and abnormal physiological responses.

Figure 2 shows in a normal hearing young adult a typical ABR to click stimuli presented at about 60 dB normal hearing level (nHL). The basic measures are the latency and amplitudes of the peak components, particularly waves V, III, and I. In addition, interpeak latency delays (e.g., between wave I to wave V) and peak amplitude ratios (e.g., wave V amplitude to wave I amplitude) are also studied. Often these measures are compared between ears in cases where unilateral disease is suspected. Table I summarizes the most common ABR measures.

Single Ear ABR Measures	ABR Interaural Difference Measures
Wave V Latency	Wave V Latency
Wave III Latency	
Wave I Latency	Wave I Latency
Latency Delay Between Waves I and V	Latency Delay Between Waves I and V
Latency Delay Between Waves I and III	
Amplitude Wave V	Amplitude Wave V
Amplitude Wave I	Amplitude Wave I
Amplitude Ratio Between Waves I and V	Amplitude Ratio Between Waves I and V

Table 1: Most Common ABR Measures

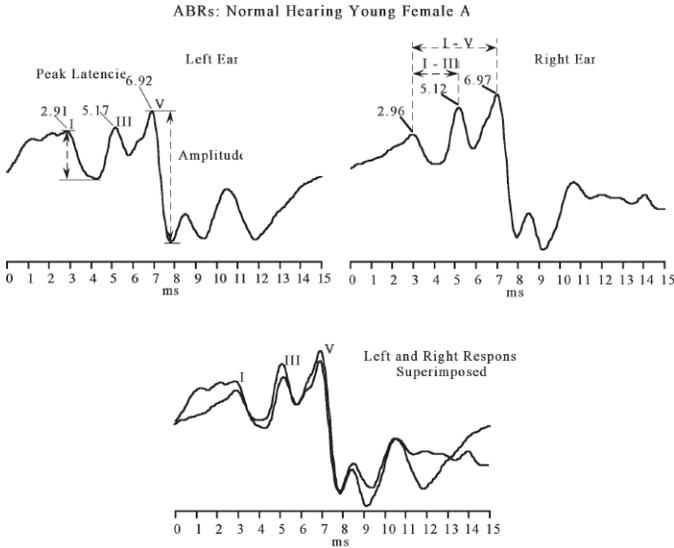


Fig. 2: ABRs from a normal-hearing young adult female showing the various components and typical parametric measures.

Unfortunately, a shortcoming of these typical ABR measures is their lack of specificity for any disease. The reason is that the typical measures of peak latencies (absolute or relative) and their amplitudes are abnormal in many diseases whether they are cochlear or retro-cochlear in origin. In short, otologic diseases often result in an impaired auditory system that produces abnormal physiological response measures. Because a cochlear hearing loss can result in abnormal physiological measures shown in Table I, it makes it difficult to determine the underlying disease and “muddies” the water of disease detection. The patient will feel little justification of undergoing physiological test measures if the measures simply point to a hearing problem that can be easily shown with standard audiological behavioral tests.

APPROCHES TO “CLEARING THE MUDDY WATERS OF DISEASE DETECTION”

Typically, disease specification using physiological responses such as the ABR is accomplished by the process of elimination and making certain reasonable assumptions. For example, if there is no history nor symptoms that suggest neural disease above the brainstem, but symptoms are consistent with a possible eighth nerve tumor, it is then assumed that an abnormal physiological measure (e.g., IT5 or I-V delay), is diagnostic for an acoustic tumor. Regrettably, this is often times the best we can do. But even being able to conclude that the test results are consistent with the presence of a tumor is of value in justifying more definitive and expensive tests such as magnetic resonance imaging (MRI).

In the following are examples of ways to reduce the confound of hearing loss in assess-

ing a patient with a small acoustic tumor and a patient with Meniere's disease/cochlear hydrops.

Small acoustic tumors and ABR wave V latency measures

Unfortunately, cochlear hearing loss often accompanies the tumor. The cochlear loss is thought to be due to vascular compromise by the tumor (Eggermont *et al.*, 1980). However, cases of unilateral hearing loss without a tumor are frequently seen in the clinic. Thus, the physiological test must distinguish these cases. Here we find that cochlear hearing loss does compromise the standard ABR latency measures used for the diagnosis.

In the early attempts to use ABRs for detecting acoustic tumors, Selters and Brackmann (1977) found that in using responses to high level click stimuli, the interaural time difference of waves V (IT5) between the suspected ear and the non-tumor ear yielded better detection than any of the standard ABR measures that had been applied. This IT5 measure is demonstrated in Figure 3A. The presumption was that the latency increase of wave V on the tumor side was due to tumor pressure on the 8th nerve causing the delay. Similarly, as shown in Figure 3B, it was presumed that the pressure of the tumor would also cause a delay between waves I and V as well as I and III. However, many studies showed that these measures failed to detect small (≤ 1 cm) tumors in a high percentage (30-50%) of cases (see reviews in Don *et al.* 1997; 2005). As discussed by Don and Kwong (2002) and Don *et al.*, (2005), the failure is due in part to a failure of the small tumor to affect the high-frequency fibers.

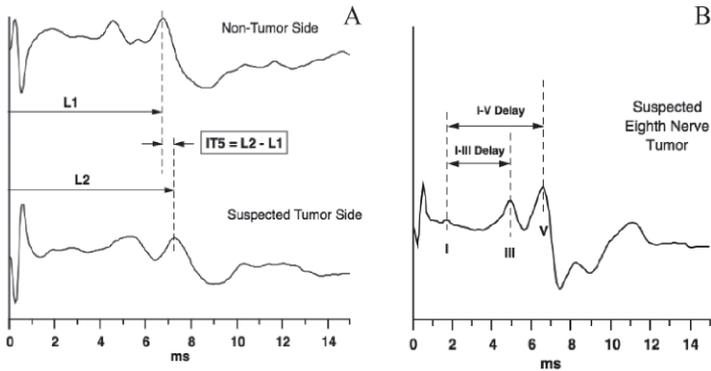


Fig. 3: A. The interaural wave V difference (IT5) is the difference in latency of wave V between the non-tumor ear and the ear suspected of a tumor. B. The I-V or I-III delay is the latency delay between waves I and V or waves I and III and is measured in the suspected ear.

Selters and Brackmann (1977) noted from their data that hearing loss at 4 kHz could prolong the latency of wave V such that the IT5 measure was abnormal when no tumor was present. This led to their recommendation to correct for the latency delay by 0.1 ms for every 10 dB loss above 50 dB at 4 kHz. This confound with high-fre-

quency hearing loss can be explained in Figure 4. In the left most panel (A) of Figure 4 are shown a series of ABR traces. The top trace is the ABR to a 60 dB nHL click from a normal hearing adult. The succeeding five traces are the derived-band ABRs to that 60 dB nHL click obtained with the high-pass masking procedure (Teas *et al.*, 1962; Don and Eggermont, 1978; and Thornton and Parker, 1978). These derived-band responses represent synchronous activity initiated from successive octave-wide regions across the cochlea with the theoretical center frequency noted beside each of the derived-bands (i.e., 11.3, 5.7, 2.8, 1.4, and 0.7 kHz). If each of the derived band waveforms were added together, the sum would essentially resemble the response to the clicks presented alone (top trace). It can be seen that the latency of the response to the clicks alone (5.92 ms) is determined by the responses from the highest frequency regions in Panel A. In other words, the peak latency of wave V in the ABR to clicks presented alone at a relatively high intensity is determined by responses from the high frequency regions. Activity from the lower frequency regions are phase cancelled in the electric field sum. The next two panels show what happens if activity from the two highest bands are removed. In Panel B, the activity from the highest band (11.3 kHz) is removed and the resulting sum shows an

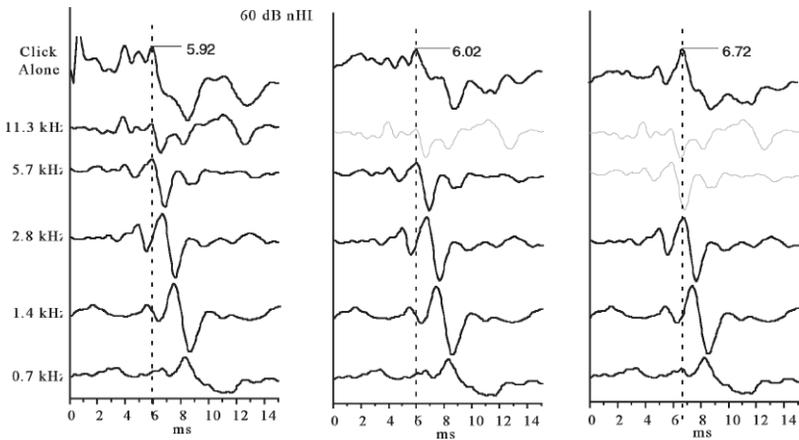


Fig. 4: The dependence of wave V latency of the ABR to clicks presented at 60 dB nHL on activity from the high frequency regions of the cochlea. Panel A: All contributions present and latency is 5.92 ms. Panel B: Removal of contributions from 11.3 kHz octave wide region results in slight latency shift to 6.02 ms. Panel C: Removal of top two bands, 11.3 and 5.7 kHz, resulting in a large latency shift to 6.72 ms.

ABR whose wave V latency has shifted very little. The latency has shifted in this case about 0.1 ms longer to 6.02 ms. This is because the latencies of the 11.3 kHz and the now dominating band of 5.7 kHz are very similar. Finally, in Panel C, we see the wave V latency shift to 6.72 when both of the highest bands are removed. This condition simulates a high frequency loss where the latency to the clicks alone is now dominated by the cochlear frequency region just below the high frequency losses. Thus, an asymmetrical high frequency loss without any tumor involvement could lead to an abnor-

mal IT5 difference. The compensation for the high-frequency loss recommended by Selters and Brackmann (1977) was empirically derived and found useful to improve specificity (correct identification of non-tumors) of the IT5 measure. However, as discussed later, the sensitivity (correct identification of tumor cases) of the IT5 measure to small (≤ 1 cm) intracanalicular tumors was clinically rather inadequate (see reviews in Don *et al.*, 1997; 2005a).

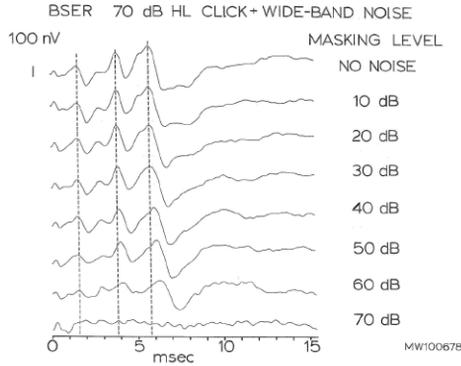


Fig. 5: Effect of broadband noise on the I-V delay in ABRs from a normal-hearing subject.

Even the wave I-V delay is dependent on compromise of the high-frequency fibers. The usual interpretation assumes the delay occurs because the tumor compresses the eighth nerve and thus, an abnormal (e.g., 2 s.d.) I-V delay is an indicator of a neural problem. However, as seen in Figure 5, an abnormal delay can be produced in a normal-hearing individual without any neural deficit. In this figure are plotted a series of ABRs from a normal-hearing subject free of any known neurological problems. The top trace is the ABR to clicks presented at 70 dB nHL. In the succeeding traces, are ABRs to these clicks with ipsilateral broad-band white noise. The noise level is adjusted to a level that masks the ABRs to the 70 dB nHL clicks. This can be seen as the last trace in the series and is labeled as 70 dB masking level. ABRs to the clicks and masking noise at various masking levels in 10 dB steps are noted. In the top trace (no masking noise) the I-V delay is noted. In the succeeding traces where the masking noise level is increasing, reduction in the amplitudes of waves I and V can be seen. In addition, the I-V delay also begins to increase at 40 dB masking level and continues to increase up to the 60 dB masking level where the I-V delay is about 0.5 msec longer than in the response to clicks alone (no noise). This delay increase would be considered abnormal in neurological diagnoses. Thus, an abnormal delay can be produced using varying stimuli alone. Thus, it is questionable that the I-V delay can be considered as strictly a neural measure and free of cochlear influences. Again, this is strong suggestion that delays in the wave V are related to the relative contributions from the high-frequency regions of the cochlea. As mentioned earlier, this latency measure also fails to detect small (≤ 1 cm).

Stacked ABR, small tumors and hearing loss

It was hypothesized that the failure of the standard clinical ABR measures to detect small tumors is due to their reliance on latency changes of wave V of the ABR (Don *et al.*, 1997; Don and Kwong, 2002; Don *et al.*, 2005a). Because the high frequency fibers dominate the standard click-evoked ABR latency measure, small tumors will be missed if they do not affect these high-frequency fibers sufficiently. These studies also hypothesized that a solution to the problem of detecting small tumors is to use a measure that assesses activity from essentially all nerve fibers, not just a subset. This led to the development of the Stacked Auditory Brainstem Response (SABR) measure that is related to the total amount of synchronous neural activity evoked by click stimulation (Don *et al.*, 1997; Don *et al.*, 2005a). Using the derived-band ABR and stacking techniques (Don *et al.*, 1994), the SABR is formed by temporally aligning and summing the synchronous activity of octave-wide bands of activity initiated across the cochlea in response to click stimulation. Because, the SABR is composed of neural activity initiated across the whole cochlea, reduction of activity by a small tumor will reduce the SABR amplitude. These papers (Don *et al.*, 1997; 2005a) demonstrated that the SABR amplitude measure can be a highly sensitive, widely-available, cost-effective, and comfortable tool for screening small acoustic tumors.

However, a reduction of synchronized neural activity can also occur with cochlear hearing loss independent of any tumor. Hearing loss is frequently a consequence of an acoustic tumor likely due to vascular compression of the cochlear blood supply (Eggermont *et al.*, 1980). Thus, any concomitant cochlear hearing loss will improve the sensitivity of the SABR measure because of the added reduction of the SABR amplitude owing to the hearing loss. Given that the major deficit of standard ABRs was the lack of sensitivity to small tumors, the SABR measure has overcome this deficit. However, because of hearing loss, specificity is compromised. That is, false positive results can occur when there is no tumor but significant unilateral hearing loss.

An obvious approach to compensate for the effect of the cochlear loss on the SABR is to correct the SABR amplitude for the hearing loss. The problem is that the normal range of SABR values is more than 2 fold across normal-hearing non tumor subjects. Thus, using an absolute numerical value can be problematic. One approach is to use a measure having a smaller variability and is a relative measure where the individual serves as his/her own control. In our approach, we use interaural SABR measure because the standard deviation of this measure in non-tumor normal-hearing subjects is about 11% whereas the standard deviation for the absolute SABR amplitude is about twice that (Don, unpublished data). To determine how to compensate for hearing loss in testing suspected tumor patients, we examined 17 small tumor cases and measured the interaural difference in the SABR amplitude and the interaural difference in the clinical pure-tone averages (PTAs). The clinical pure-tone average is the average threshold for 500, 1000, 2000, and 3000 Hz. Figure 6 shows the interaural SABR percent difference as a function of the difference in the clinical PTA for each of 17 small tumor patients studied with ER2 insert earphones. Other specifics regarding methodology (e.g., recording and stimulating parameters) can be found elsewhere (Don *et al.*,

2005a). A linear regression line is fitted to the data and results suggest, to a first

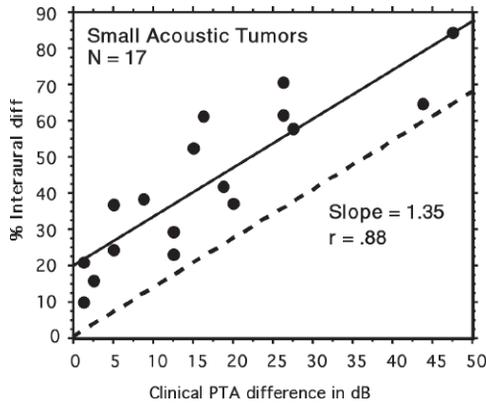


Fig. 6: Interaural percent difference in the SABR amplitude is plotted as a function of the interaural clinical PTA difference in dB for 17 small acoustic tumor cases.

approximation, that for each dB difference in the interaural clinical PTA, there is a 1.35% difference in the SABR amplitude. It can be seen that the intercept of the regression line is at about 20%. This suggests that the small tumor, on average, reduces the SABR amplitude by about 20% above that caused by the hearing loss alone. The dotted regression line has the same slope and indicates the calculated theoretical relationship between the SABR difference and clinical PTA difference independent of a tumor. This is an example of compensating for the effects of hearing loss that can “muddy” the waters of tumor detection.

Meniere’s disease/cochlear hydrops detection

Don *et al.* (2005b) described a method for distinguishing patients diagnosed with an active case of Meniere’s disease (MD) from non-Meniere’s normal-hearing subjects (NMNH). The method involved recording ABRs to moderate level clicks and simultaneous ipsilateral masking pink noise high-pass filtered at 8, 4, 2, 1, and 0.5 kHz. This procedure is identical to the first step described above regarding the Stacked ABR. However, there is no successive subtraction of responses to form derived-bands and no stacking of these bands. Instead, only the high-pass masked responses are analyzed. It was shown that in the control NMNH subjects, the latency of wave V in the ABR increases as the cut-off frequency of the high-pass masking noise is lowered. Normally, the highest unmasked frequency region dominates the latency of wave V. Therefore, as the cochlea is successively masked from 8 kHz and higher down to 0.5 kHz and higher, the peak latency of wave V increases. An example of the normal masking pattern in NMNH subjects is illustrated in Panel A of Figure 7. An increase is expected because with each lowering of the high-pass masking noise cut-off frequency, the response to the click is dominated by the lower unmasked frequency region. Thus, due to factors related to the cochlear traveling wave delay and cochlear response times, the peak latency of wave V of the ABR increases as the area of the unmasked cochlea is successively restricted to lower frequencies. With the full band

noise masking, the ABR is often fully masked and a response is not detected.

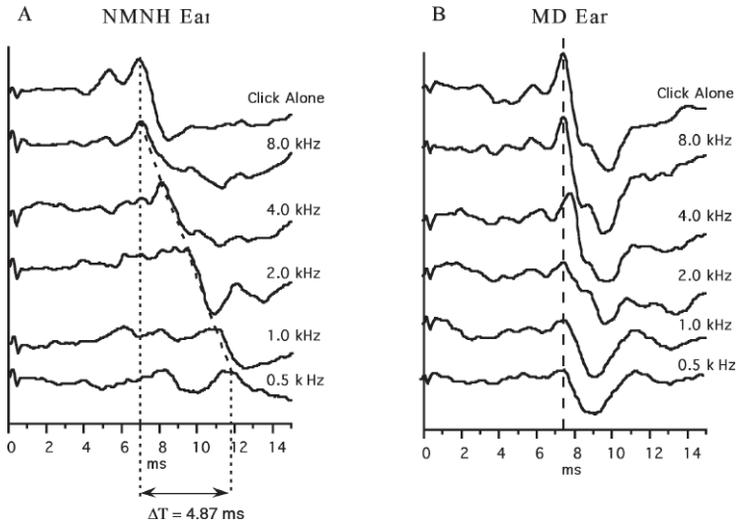


Fig. 7: A. High-pass masked ABRs from a NMNH ear showing increase wave V latency with each successive high-pass masking condition. B. High-pass masked ABRs from a MD ear showing no increase in wave V latency with each successive high-pass masking condition. Modified from Don et al. (2005b).

However, for the MD patients, that masking noise was insufficient such that the latency of wave V in the responses to the clicks and various high-pass masking noise conditions is similar to that of wave V in the response to clicks alone. An example of the undermasked pattern in MD patients is illustrated in Panel B of Figure 7. Don *et al.*, (2005b) hypothesized that the endolymphatic (cochlear) hydrops in patients with Meniere’s disease alter the response characteristics of the basilar membrane such that the high-pass masking noise that normally masks the high-frequency activity that dominates the response in NMNH subjects is less effective in MD patients. [It should be noted that the use of pink noise (-3 dB/octave) emphasizes the reliance of some upward spread of masking.] As a result, in patients with Meniere’s disease, the observed wave V latencies of the responses to the high-pass conditions are similar to that for the response to clicks presented alone as seen in Figure 7B.

In order to quantify this undermasking phenomenon for comparing these two populations, the difference in the latency of the obvious wave V in the response to clicks presented alone and the response to clicks in the presence of 0.5 kHz high-pass masking noise (henceforth referred to as the 0.5 kHz high-pass response) was measured. As shown for the NMNH subject in the left panel in Figure 7A, the delay difference is quite large. However, for the MD patient shown in the right panel (7B), there is virtually no measurable latency change or delay. As demonstrated by Don *et al.* (2005b), the difference in the delays between the MD and NMNH populations investigated in that particular study was such that the sample distributions did not overlap resulting in 100% sensitivity (detection of MD patients) and 100% specificity (correct identi-

cation of NMNH subjects).

In practice a specific measurement may not be necessary. If a wave V peak can be observed to increase in latency as the cut-off frequency of the high-pass masking noise is lowered, a diagnosis of Meniere's disease/cochlear hydrops is not supported. Any latency delay measure should be made on the larger longer latency wave V (Figure 7A). The reason for the focus on the longer latency wave V is that this peak indicates the masking noise is sufficiently effective such that the response is dominated by areas of the cochlea that are lower in frequency than the cut-off frequency of the masking noise. Thus, its latency in the 0.5 kHz high-pass response will be significantly longer than in the response to clicks alone.

How does hearing loss affect the undermasking measure? A major theme emphasized in this paper is that while physiological correlates of hearing impairment can easily be found, finding physiological correlates that are more specific to a given otologic disease is difficult. The reason is that hearing impairment is a usual consequence of most otologic diseases and the presence of hearing impairment affects physiological measures. Because the abnormalities of most physiological measures depend on the hearing impairment and not the cause of the impairment, determining the specific disease from these abnormal measures is virtually impossible. However, proper treatment and rehabilitation may depend on knowing the cause of the impairment and not simply that an impairment exists. The attractive feature of the undermasking test for Meniere's disease/cochlear hydrops is that it appears immune to the effects of hearing loss. Don et al (2005b) demonstrated that patients with hearing loss but without Meniere's disease/cochlear hydrops, show the usual masking pattern seen in NMNH subjects. This is illustrated in Figure 8 where for two patients with equivalent hearing loss (slightly more loss for the non-Meniere's patient in the high frequencies) the audiograms (A), and the high-pass masked responses for the Meniere's disease patient (B) and for the hearing loss only (C) are shown. It can be seen that for the Meniere's disease patient (B), undermasking occurs and the latency of wave V does not change with successively lower high-pass masked conditions. In contrast, the non-Meniere's disease patient with even more hearing loss does show the progressive masking and increase latency of wave V typical of non-Meniere's cases. Even though the hearing impairment may affect other measures such as latency and amplitude of the ABR components, these changes are not part of the diagnostic criteria. The test simply asks whether masking pink noise that is sufficient to mask non-Meniere's normal-hearing subjects can mask the patient in question. If it can, the patient does not meet the criterion diagnosis for Meniere's disease/cochlear hydrops. If it cannot, a diagnosis of Meniere's disease/cochlear hydrops is supported. This is an example of avoiding the confounding effect of hearing loss on a physiological measure by using a measure that is fairly independent of the presence of hearing loss.

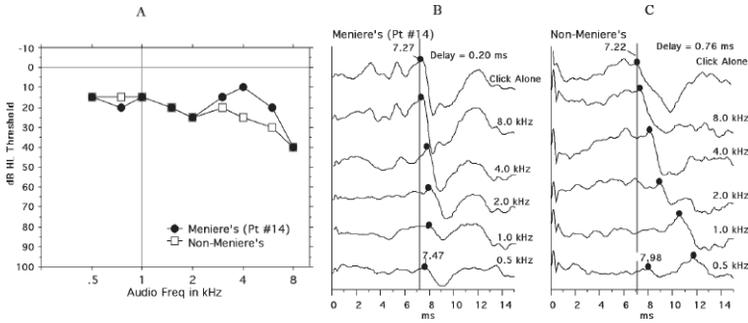


Fig. 8: A. Audiograms of a Meniere’s disease (filled symbols) and a non-Meniere’s disease (open symbols) patient. B. High-passed masked responses from the Meniere’s disease patient showing the undermasking phenomenon. C. High-pass masked response in non-Meniere’s patient (with slightly more hearing loss) showing the normal progressive masking with successive high-pass masking conditions. Modified from Don et al (2005b).

DISCUSSION AND SUMMARY

Measures of evoked neural activity recorded from the surface of the head are attractive for tests in humans because they are non-invasive and do not require behavioral responses. Such measures have had much success and value in identifying the presence and even the degree of hearing impairment in infants and children easily justify the development and improvement of these measures. However, these measures have had disappointingly limited success in the diagnosis of specific otologic diseases in adults. Such diagnosis typically requires a process of elimination and assumptions that other diseases are not involved. A stumbling block for the diagnosis of specific diseases is that hearing loss is a usual consequence of otologic disease and most physiological measures simply reflect the hearing loss and not the underlying disease, thereby mudding the waters of disease detection. To improve their utility for adults beyond what can be obtained from behavioral measures, two approaches were illustrated in this paper. One approach is to develop measures that can be compensated for by the degree of hearing impairment. The example presented here involved determining within the same subject the relationship of the interaural difference in the amount of hearing loss as measured by the clinical PTA to the difference in the interaural amplitude of the Stacked ABR. While not a perfect compensation, it does improve the specificity of the measure and reduces the confounding effect of the hearing loss.

A second and more preferable but more difficult approach is to develop creative physiological measures that are not affected by hearing loss but capitalize on changes due to the underlying disease in the way the auditory system processes acoustic information. The example presented here is the undermasking observed in patients with Meniere’s disease/cochlear hydrops (Don *et al.*, 2005b). It is hypothesized that the cochlear hydrops causes stiffness changes in the basilar membrane that leads to the undermasking phenomenon. The presence and degree of hearing loss appears to be inconsequen-

tial. It is not known yet whether there are other otologic diseases or cochlear problems that could lead to undermasking. At this point, this phenomenon appears to be specific to Meniere's disease/cochlear hydrops.

Non-invasive physiological measures are valuable tools in diagnosing diseases that affect the auditory system. Their value heavily depends on a clear understanding of the physiological bases of the measures, their appropriate application, and their interpretation. While hearing impairment may "muddy the waters" for specific diagnoses, we can begin clearing the muddied waters with thoughtful measures to compensate for the effect of hearing loss or by developing new measures whose diagnostic value is not compromised by the hearing impairment.

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