Peer Commentary on "Clinical Usefulness of Auditory Evoked Potentials: A Critical Evaluation" by T.W. Picton

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Reading Picton's paper has much in common with eating delicately spiced food: One cannot stay indifferent and one is provoked to comment in a laudatory way, albeit that after some delay doubts about the indulgence in and substance of the meal occasionally appear. Thus, on first reading of this paper I had the impression that the author was completely justified in his opinions and reservations, that the weak and strong points of the clinical use of evoked potentials had once and for all been stated, and that one only had to do some more research on the indicated weak points or at least be careful in applying them. The main impression was also that Picton's assessment of the clinical use of evoked potentials was well balanced and based on his extensive knowledge reflected in an extensive list of references. On second reading and by limiting myself to a more restricted topic than that of the entire paper, I started to see that some, may be unconscious, bias had entered the presentation. It is the aim of this commentary to provide some additional information, mention a few overlooked references that are crucial for some of the conclusions, and make some remarks on the topic of frequency specificity of threshold determination using evoked potentials so as to provide a more balanced approach.

Frequency specific evoked potential audiometry has a rather long history: from the use of tone bursts to evoke the long latency cortical evoked potentials (reviewed in Davis, [1976]), the first publication of objective audiograms obtained with tone-burst electrocochleography [Eggermont, 1974]); to their subsequent evaluation in both children (once they could be tested subjectively [Spoor & Eggermont, 1976]) and adults by comparing electrocohleography with audiograms made the same day (Eggermont, 1976); to the measurement of accurate click ABR derived response audiograms (Don et al., 1979) or tonepips-in-notched-noise ABR (Picton et al., 1979). Basically what these measurements show is that whenever the subject cooperates (especially in the case of long latency potentials) and is relaxed (especially in ABR and MLR), the objectively measured audiogram, based on thresholds at audiometric frequencies of 500 Hz to 8 kHz, corresponds remarkably well with the subjective audiogram. The slopes of the regression lines for the comparison of ECochG and subjective audiometry range from 0.72 at 500 Hz to 0.95 for 2 kHz, and the standard deviations for the difference histograms are less than 10 dB at all frequencies (Eggermont, 1976).

The question should therefore not be "whether the evoked potentials can provide an audiogram" as Picton states, but under what conditions reliable objective audiometry can be expected. These conditions depend as much on the subject's state as on the stimulus used to make the recordings, and this is addressed to some extent in Picton's article. I fully agree with Picton's conclusion that one has to use frequency and/or place specific stimuli to arrive at an estimate of an audiogram and that some form of masking to limit the spread of activity has to be used. It is plainly impossible to infer audiograms from clicks (Eggermont, 1982) unless one has a precise concept of the type of hearing loss in advance of the ABR test!

There is a suggestion in Picton's paper that longer latency evoked potentials, such as MLR, 40 Hz, and long latency cortical potentials, can produce more frequency specific results because they allow the use of tone bursts with longer rise times. Longer rise times mean less frequency splatter in the stimulus and therefore a more frequency specific stimulus. Although it is correctly noted that only the first few cycles during the rise time of the burst are evoking the potentials, the notion of greater frequency specificity of the entire stimulus remains a suggestive argument for greater frequency specificity of the response. For clinical practice, one can only resolve this issue by comparing statistics on the relation between objective and subjective thresholds in electrocochleography, ABR, MLR, and long latency potentials (see my discussion in Eggermont, 1983). This comparison does not substantiate the claim of greater frequency specificity: All methods if applied at the state of the art are equally good, that is, the results show the same standard deviation of about 10 dB. An interesting study by Van Heusden and Smoorenburg (1981) investigated some aspects of frequency selectivity on the AP thresholds in guinea pigs: Band-pass filtering the test tones, which had zero rise time, with filters having slopes of up to 132 dB/octave did not result in sharper AP tuning curves, although the filtered test tones required a higher level, less than 5 dB, to evoke the same AP amplitude. Thus I am not compelled by the rise-time argument to favor, for example, MLR-based audiograms, above those obtained by the ABR as suggested in Table I.

Picton makes the suggestion is made that it is impossible to use derived response ABR's to measure low frequency thresholds, and if one accidently gets a response for example at 500 Hz, it is because of the also present MLR. The argument for this suggestion is that the low frequency derived response only shows up when the repetition rate is close to 40/s (as in Don et al. and in Picton et al.'s data, which had rates of 34/s and 40/s, respectively) and does not show up when the presentation rate is lower, for example at 27/s, as in Laukli et al.'s

data in which the steady state (40 Hz) MLR response is quite small. It must be remarked that one of the first papers to show the recording of low frequency derived ABR responses down to at least 20 dB SL in normal ears (Eggermont & Don, 1980) used a click rate of only 13/s. So it appears that these are responses at near threshold values that cannot be explained as resulting from MLR's, and consequently they will be wave V components of the ABR. It seems that the experimental set up with appropriate high-pass filter characteristics and the appropriate signal-to-noise ratio is at least as important in obtaining reliable derived ABR responses as is the click rate. Incidentally, at low click levels the derived responses at both the low frequency end (500 Hz) and the high frequency end (8 kHz) decrease faster than those at the middle frequencies, and ultimately only the responses in the 2-4 kHz region remain.

I cannot refrain from presenting an interesting, historical observation. In the 70's it was very common that ABR and ECochG publications dealing with the estimation of hearing loss were based on either tone bursts or clicks in high-pass noise, and in such studies audiograms routinely were presented for at least three frequencies, always including 1000 Hz (references in Eggermont, 1983). The trend in the 80's seems to shy away from this trend (with the notable exception of Gorga and colleagues) and to emphasize more and more the precise relationship between click thresholds and the subjective audiogram. This relationship will obviously be colored by the shape of the audiogram and the type of hearing loss, and is therefore not unambiguous. The time restraint that most audiologists work under seems to take its toll; time consuming ABR or MLR studies with tone bursts or clicks or tone bursts in high-pass masking noise are replaced by the simple wide band click screening. The consequence is of course that this practice may lead to sub-optimal assessment of hearing in the difficult-to-test cases.

A final remark on the potential use of the otoacoustic emissions (OAEs) in estimating the degree of hearing loss. Until recently it had been demonstrated only that spontaneous and click stimulated emissions are absent in ears with hearing loss exceeding about 40 dB (e.g., Probst et al., 1987). Recent use of distortion product emissions, in which an emission is produced at the cubic difference tone frequency 2f1 -f2 by stimulating with two tones simultaneously (Brown & Kemp, 1984), suggests that this distortion product emission threshold correlates very well with the audiometric threshold at the cubic difference tone frequency as long as this threshold is below about 50 dB HL (Kimberley & Nelson, 1989). This suggests a potentially very useful screening function for distortion product OAEs. J.J.E.

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Picton's article raises several issues that are important in the clinical application of auditory evoked potentials (AEPs). In contributing to his discussion, we have focused our comments on three of the applications reviewed: (1) infant hearing screening, (2) determination of threshold sensitivity, and (3) diagnosis of retrocochlear disease. Because Picton's paper concentrates on the clinical usefulness of the auditory evoked potential, we have approached these areas from the clinical viewpoint, basing our comments on our experience with over 15,000 clinical cases during the past ten years.

Infant Hearing Screening

Picton's assessment of the ABR as a screening tool for identification of hearing loss in infants appears to conclude that the AEP is not well suited for screening all infants. One reason is because of its high cost. While it is true that many protocols for hearing screening with the ABR that are currently in clinical use are expensive, much of the high cost is manageable. For example, two of the major expense areas, the skilled operator and the number of false positives, have been controlled in an automated ABR hearing screener called the Algo-1 Infant Hearing Screener (Thornton, Herrmann,& Berrick, 1985; Kileny, 1987; Jacobsen, Jacobsen,& Spahr,1990). An expensive, skilled operator is not needed because of automation which insures an adequate test and returns a pass or refer outcome. If the conditions of the test are not adequate, the test will not be completed;

it does not accept an invalid test. The accuracy of the screening is as good as ABR hearing screening under optimal conditions using a skilled operator, and the false positive rate is less than 1% (Sprague-Herrmann, 1987).

A device such as this automated screener makes low cost testing of infants possible at a time that also promotes efficiency, that is, when the babies are still in the hospital. Costs of the screening are less than the costs of determining which babies are high risk for hearing loss and tracking them effectively. In addition, babies are not lost to testing, and all the babies with congenital hearing impairments are identified, not just those who fall into a risk category. While otoacoustic emissions may well become a better screening tool than the ABR, as discussed by Picton, low cost, accurate hearing screening of all infants with ABR is feasible now.

Besides high cost, Picton cites several other factors that argue against using the ABR to screen in the NICU. One is the presence of conductive hearing loss. This problem is difficult to assess because the incidence of conductive loss is not well documented and may vary with the NICU. In our NICU, the incidence of conductive loss is less than 1%. Therefore, it does not make the screening ineffectual. Because few clinics routinely test air and bone conduction within days of a screening failure, the incidence of conductive hearing loss is often assumed when follow-up testing several months later indicates normal hearing. In one center where automated screening is done for all newborns regardless of risk factors (12,000 per year), there is only an 8% total failure rate. Those infants failing with conductive hearing loss tend to have recurrent otitus media and mild hearing loss throughout their early childhood (Marlowe, personal communication). Their early identification is beneficial.

Although not directly stated, many of Picton's citations regarding false positive ABR screening results (i.e., an infant who fails a screening and has normal hearing at follow-up testing) refer to infants who are classified as failures because of ABR amplitude and latency criteria. In other words, the infants are failing because of mild neurologic anomalies, not because of an absent response. Because many of these mild anomalies resolve in the first few months, this mixture of neurologic criteria and hearing in one screening evaluation continues to add noise to the discussion of the effectiveness of the ABR in hearing screening. We have found that focusing the purpose of the screening on identification of hearing loss identifies children with peripheral ear disease, children who can benefit from known medical and rehabilitative therapies. If neurological screening of these infants is important, its purpose and results should be separate from those of hearing screening.

Along the same lines, Picton also brings to the readers' attention that there have been cases reported of normal hearing and no ABR. While these reports frequently are referred to in the literature, we have never seen this in over 4000 cases of evaluating hearing with the AEP in patients with a large variety of medical problems. We have seen a few cases in which the ABR was very small with an unusual morphology and was difficult to record. For instance, one case had a grossly abnormal ABR with only wave I and a small, broad positive wave at 15ms. This abnormal response tracked to 0 dB HL. These rare cases with unusual and difficult-to -record waveforms do not pose a problem for screening purposes because a screening failure means that further testing will be done. Then, during a complete hearing threshold evaluation, the characteristics of the patients AEP can be evaluate critically and the necessary precautions for adequate signal averaging and optimal recording parameters taken to ensure that an accurate test is done.

Determination of Hearing Threshold Sensitivity

Although rigid protocols are appropriate for specific uses of the AEP, such as hearing screening, they do not optimize results for complete evaluations of hearing threshold sensitivity. Many investigators and clinicians report parcelling the AEP into its components of ABR, MLR, Late, and so on, and using rigid recording protocols that are rarely varied, regardless of the patients response characteristics or the stimulus used. This cookbook approach is often ineffective in measuring hearing with the AEP. We have found that a flexible and interactive approach to an evoked response evaluation first can assess the characteristics of a patient's response and then can optimize the conditions of the test to track that response to threshold, resulting in an accurate assessment of hearing threshold sensitivity. For example, although we routinely use 15 ms and 20 ms analysis windows to measure evoked responses to tonebursts, we change those parameters when necessary for better resolution of the patient's response. Sweep counts of 16,000 commonly are used to average 20 nV responses near threshold adequately. In difficult cases, analysis windows of 10 ms and sweep counts of 1,000 to 2,000 are grossly inadequate to measure the threshold of hearing. Because assessment of results on line is necessary to optimize further testing, we have found the best results when the professional who interprets the findings also conducts the test. Data collection by a technician using rigid protocols with later interpretation by a third party not present at the evaluation diminishes the effectiveness of AEP testing.

Consistent with this interactive approach, our choice of which evoked potential is best suited for assessing a specific patient's hearing sensitivity will depend more upon the age and physiologic noise level than the frequency of the test stimulus. We have found that the ABR, more specifically the negativity following wave V, is reliable at all ages, and it is the best potential for assessing hearing threshold sensitivity in children. Our experience with later potentials of the middle latency response (Sprague & Thornton, 1982) is similar to that of Kraus, Smith, Reed, Stein, and Cartree (1985) in that a repeatable, reliable response cannot be recorded during sleep until about age 11. We have also found that the MLR was absent in sleeping children at low stimulus rates of 2 and 4/s using as many as 8,000 sweeps. This absence of the MLR explains why the 40 Hz potential is not reliably recorded in infants (Galambos, Kileny, Stapells, & Thornton, 1983).

In contrast. the MLR is an excellent tool for assessing hearing threshold sensitivity in quietly resting, awake adults. The signal-to-noise ratio of the Na-Pa-Nb complex is much better than the wave V and, like wave V, it can be recorded to within 5 dB of perceptual threshold. In most cases, it is faster to record fewer sweeps at 13/s for an adult MLR than many more sweeps at a faster rate for reliable wave V recording, especially for low to mid-frequency tonebursts. If the patient goes to sleep and the signal-to-noise ratio improves, we may then change to wave V as the most efficient potential for measuring threshold sensitivity for high frequency stimuli. Both potentials can be recorded to within 5 dB of a patient's hearing threshold sensitivity. However, maximum amplitudes are found at different frequencies.

Picton's discussion of measuring hearing sensitivity specific to different frequencies on the audiogram suggests that the use of the notched noise or derived responses are much more useful than tonebursts. In contrast, we have found tonebursts to be an excellent means for measuring hearing threshold sensitivity for different frequency regions of the audiogram. They are simple to generate, responses are not degraded by the presence of masking, and there are no assumptions made about the critical masking bands of a diseased ear. Such simplification contributes to the speed of the evaluation and often results in obtaining more information on a child during the time constraints of a test session. We most frequently obtain thresholds to tonebursts centered at three frequencies for each ear and one threshold by bone conduction during one two-hour evaluation. With adequate averaging, responses are generally recordable to within 5 dB of threshold for tonebursts from 500 to 8000 Hz.

As pointed out by Picton, toneburst spectra limit the precision of measurement for steeply sloping high frequency hearing losses, that is, the maximum slope that can be accurately defined is largely dependent upon the sideband energy. The first sidebands of linear gating functions, used by many clinical instruments, are only 27 dB down from the main lobe and, consequently, cannot define a high frequency hearing loss slope greater than 25 dB per octave. However, even with these stimuli, a slope to the hearing loss is identified and, based on the results obtained and the spectra of the tonebursts, a range of hearing in the high frequencies can often be estimated. This amount of information is most often sufficient for the adequate fitting of a hearing aid. When time permits in the AEP evaluation, different stimuli can be used, such as filtered clicks or if warranted, more complicated procedures, such as notched noise and derived masking techniques, can be used to further define the slope of the hearing loss. Since the majority of the patients we evaluate for threshold sensitivity have sloping high frequency hearing losses, more complicated procedures are only done when necessary, and the time saved by simpler procedures can be used to gain information at more frequencies. When tonebursts are gated with windows that produce less sideband energy, such as the Exact Blackman gating function, the slope restrictions are less. There is no distortion process unique to these stimuli or this evoked potential testing.

The rising slopes of low frequency or reverse frequency hearing losses are well defined by tonebursts. The place specific versus frequency specific question in such losses is not a limitation of the ABR, but it is the same as that presented in behavioral pure tone testing. The audiometric question of whether place information is important is not specifically related to the auditory evoked potential but rather to whether that information is important for proper rehabilitation. If so, masking techniques could be used in both AEP and behavioral audiometry.

Diagnosis of Retrocochlear Disease

With respect to the use of the ABR to identify retrocochlear disease, we have found it to be an excellent diagnostic tool, and we have not found the limitations described by Picton. Although it does not differentiate well between different retrocochlear diseases, such as multiple sclerosis and acoustic neuromas, it effectively screens for those patients who need imaging. We have found its sensitivity to be similar to the 95% figure given in the literature, and it indicates retrocochlear disease in patients without tumors in fewer than 7% of all cases referred for clinical evaluation.

Our study, cited by Picton (Joseph, West, Thornton, & Nadol, 1987), did not question the efficiency of using the ABR for retrocochlear diagnosis, but rather it was aimed at improving the decision criteria for an already good test. The sample of 17 patients with surgically confirmed tumors was purposely selected to examine characteristics of ABR's that were close to the cut off criteria used in our laboratory. The nine false negative ABR patients represent fewer than 5% of our entire population of patients with tumors. The analysis of these patients, combined with the principal component analysis on the ABR data from over 2,000 patients with cochlear hearing loss (Thornton & Takagi, 1985), resulted in a normalized combination of the latencies of I, III, and V that was more sensitive to tumor identification than our previous clinical criteria. Comparison of the receiver operating characteristics of this new criteria, called PC30, with other commonly used clinical criteria indicated that the PC30 gave the best performance, followed by the interear wave V latency difference, and then the I-V interear-interwave latency difference. In other words,the wave V latency difference between ears is more sensitive and specific than the commonly used I-V interwave and interear-interwave criteria. When waves I and III cannot be identified for both ears, PC30 cannot be calculated. The wave V latency difference then is a very acceptable criteria and better than I-V criteria.

Regarding the other limitations cited by Picton, we have rarely found the ABR to be absent in the presence of adequate hearing sensitivity without also confirming retrocochlear disease, so we would interpret such an absence of the ABR as diagnostic of retrocochlear disease. Also we have not found abnormalities in the ABR to be restricted to large tumors. While some patients with normal ABRs did have small tumors by MRI scanning, others had ABR abnormalities several years before tumors were large enough to be

identified by imaging techniques, including MRI. The location of the tumor may be more important than its size.

Finally, the hypothesis that the delay of the I-V interwave interval is caused by loss of high frequency fibres is highly speculative. We have a patient with a surgically confirmed tumor who presented with a marked low frequency hearing loss below 4000 Hz. She had a large wave I at a normal latency and a wave V latency greater than 7 ms. This large I-V interval remained constant for broadband clicks and for high frequency tonebursts centered at 6000 Hz. Clearly, those results cannot be explained by the selective loss of high frequency fibres.
B.S.H. & A.R.T.

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Picton is one of the few in the AEP field whose experience justifies such a general review article. He has offered some useful insight, and I believe he will succeed in his stated goal "to engender some discussion." I would like to comment first on three specific areas of AEP use, and then elaborate a few general points.

Infant Hearing Assessment

I agree with many of Picton's statements in this area. One of the fundamental issues that is not yet settled is the definition of what it is exactly that we wish to detect, that is, to define the target disease. Assuming that it is hearing loss of some kind, the question is: What kind? What is the etiology, severity, frequency contour, laterality and developmental time course, and what is its impact on speech and language development? Until there are better answers to these questions it is difficult not only to design efficient programs, but also to evaluate the tools to be used. For example, it is possible to get a wide range of answers about the audiometric accuracy of the click ABR, even from a single dataset, depending on the definition of the target disease and the test abnormality criteria.

Our data regarding follow-up validation at 3-6 years of click ABR testing at about three months corrected age generally support the findings of Picton and his colleagues. We, too, have observed children who have normal click ABRs in infancy but have hearing loss at follow-up, with at least one near normal pure tone threshold in the 1 to 4 kHz range. Strictly speaking, we cannot distinguish lack of frequency specificity of the click from lack of development of the impairment at the time of the ABR as the actual cause of the apparent false negative ABR outcome. Of course, it seems inappropriate to look for frequency specific hearing loss with a wide band stimulus, and this relates to the question of the definition of the target disease.

Our early assessment program has always included notch masked tonepip testing at 500 Hz and 4 kHz, as well as the click ABR. We have not noticed any major difficulty recording what we are sure are ABRs to 500 Hz tonepips. Also, except for its value in hearing aid fitting, we have not found testing at 4 kHz useful for increasing the accuracy of detecting high frequency hearing loss at follow-up. Recently we have moved to using 8 kHz. Perhaps an even higher frequency would be desirable as an early warning for progressive high frequency sensorineural loss. We are contemplating a change to the derived band masking approach, if we can work out the problem of a practical trade-off between the number of high-pass masking conditions on the one hand, and the number of intensities and number of stimuli per average on the other.

The issue of the effect of retrocochlear dysfunction on the audiometric accuracy of the infant ABR is certainly difficult and raises the general problem of the AEP as an epiphenomenon of hearing. Which is the better indicator of hearing sensitivity, wave I or wave V? Perhaps the question is simplistic, and neither is always best. As Picton notes, if there was hydrocephalus, then, in the absence of a clear wave V, the audiometry might reasonably be based on wave I. This may not be appropriate for other etiologies of wave V dysgenesis, and an absent wave V in the presence of a clear wave I always causes audiometric uncertainty. Our viewpoint is that the hearing sensitivity is simply not known, but the index of suspicion is raised. Our approach would be to proceed with more rostral AEP audiometry, for example, with the MLR, in the belief that the MLR is less vulnerable to synchronization disorders.

In Picton's suggested infant protocol, many features of which I fully support, ECochG is used on every infant with an abnormal click ABR. I do not see such a major role for this invasive procedure. In our experience, mastoid registration of ABR Wave I is somewhat

better than Picton suggests, so in effect we are doing adequate cochleography at the mastoid in infants (but not in adults). In the sleeping normal infant we almost always see a very clear wave I at the mastoid for a click of 30 dB nHL or less, and the same is true for 4 kHz masked tonepips at 40 dB nHL. Thus, if the click ABR threshold is elevated but the waveform is normal, we usually expect to be able to get a reasonable audiometric description without ECochG. How often is it necessary to have a better signal-to-noise ratio? In my view, ECochG is more defensible if there is no way to obtain satisfactory measurement conditions for ABR testing without sedation or anesthesia, or if the issue of residual hearing is crucial and the ABR indicates a hearing loss that is severe or worse. Furthermore, if the click ABR waveform itself is abnormal, such as with absent wave V, why use another measure caudal to the site of putative lesion?

What is the optimal age for testing? In 1984 we began testing at about 56 weeks post-conception because of the large discrepancies between 40-week and 56-week click ABR abnormality rates, even in well babies tested in an audiometric sound room. These discrepancies increase with decreasing click ABR threshold abnormality criteria, but are quite large even at 40 dBnHL. Resolving conductive disorders are probably the main culprit, and in neonates or young infants, accurate quantification of conductive and sensory loss components is not yet entirely straightforward. Also, it seems reasonable to defer testing to allow every opportunity for the expression of a disorder up to the point in time at which intervention should be initiated.

Our patients return for 56-week testing with compliance in the range 85-90%. This is due to the time invested in garnering community physician support, as well as to persistent recall efforts by letter and telephone to parents and physicians. This is expensive and may not always be feasible or effective. In that case, neonatal testing seems the only answer. The question is: Will a parent who refused to bring the child back for testing at four months comply with a habilitation program based upon neonatal testing? Litigation based on the rights of the child might help, but parent education and community follow-up seem to be important avenues for further efforts. These procedures also address the problem of children with hearing loss who would not be detected by a program based on current risk registers because the factors are inappropriate, because there are no accessible factors (e.g., recessive familial disorders), or because the losses are progressive and can only be discovered by a monitoring process.

Finally, with regard to otoacoustic emissions, I agree that there seems little doubt that they are a useful tool, especially as a screen for the ABR. However, I believe that there is a significant bandwagon effect now in operation. There is equally little doubt that significant practical constraints, accuracy limitations, and caveats on OAE validity and use will emerge. The biggest practical difference between screening with emissions and with the ABR is the electrode application, which may yet yield to further engineering development.

Functional Hearing Loss

I have used this heading because I would prefer to restrict the term objective audiometry to refer to use of completely automated systems that include stimulus control and computer assisted interpretation. Such systems are not yet common, but will emerge over the next few years.

Picton is more temperate than I can be, especially in relation to functional hearing loss. The lack of widespread use of the cortical AEPs with 100 to 200 ms typical latency is one of the real puzzles of North American audiology. This AEP is widely used elsewhere in the world and has been so for many years, yet one can still find new North American audiology textbooks that virtually ignore it, dwelling upon procedures as archaic as galvanic skin response testing or as limited as threshold prediction from the acoustic reflex. It is doubly curious because of the North American origins of this particular AEP technique (in St. Louis).

At our clinic, testing with the late tone burst response N100-P180 is almost mandatory when there is a need to estimate or validate pure tone thresholds in a passively cooperative, awake person other than a young infant. With proper training and test protocol, it is possible to estimate pure tone thresholds by air and bone conduction, masked and unmasked, to within 10 dB in over 90% of cases (Hyde, Alberti, Matsumoto, & Li, 1986.). The frequency specificity of this response is excellent, and sharp notches or high slopes can be resolved. At the present stage of knowledge regarding frequency specificity, it seems to me that the ABR is a markedly inferior tool; it might even be called wholly inappropriate if the goal is accurate estimation of the pure tone audiogram. Abnormalities of tuning curves in cochlear pathology cast doubt even on the validity of the derived band ABR for this purpose.

Picton raises the very interesting problem of the distinction between place specificity and frequency specificity. Which is the more fundamental or more useful measure of auditory function? Is the conventional audiogram in any sense wrong? Perhaps the discrepancy between frequency and place measures might contain important information. I believe that the derived band ABR and ECochG are the tools to explore these questions.

Acoustic Tumors

Picton raises several interesting points. With respect to our environment, at least, the ABR has a stronger role than might be inferred from Picton's discussion. We do about 2,000 ABR tests per year in adult patients with various degrees of suspicion of acoustic tumor. In this general otology and neuro-otology clinical environment, the acoustic tumor is about one hundred times more prevalent than the next most common intracranial neuropathy, so the lack of pathology specificity in the abnormal ABR waveform is not really a major problem.

As Picton suggests, we find no ABR wave I at the mastoid in about one-third of our patients, but this does not cause much of a

problem. Our approach is based on a nonlinear model of the expected wave V latency as a function of age and pure tone cochlear hearing loss, an early version of which was published in Hyde (1985). This model is applied to both males and females (different absolute latency but the same interactive age and hearing loss effects) using a constant SPL click stimulus (115 and 125 dB peak SPL). Essentially, we have refined the Selters and Brackman (1976) method. The protocol includes evaluation of wave III and the use of both condensation and rarefaction stimuli. Our decision criterion is set such that the false positive rate is about five percent. I am aware of two tumors missed over a period of about ten years, although this, of course, does not mean that others were not missed. I currently accept the notion of a false negative rate between two and five percent, but as Picton notes, if patients present earlier and earlier for investigation, the true figure will be higher. I do not see any reason to increase this estimate beyond five percent until peer reviewed large sample evidence becomes available, and I am concerned that Picton's academically reasonable caveat on ABR accuracy might be over-interpreted.

Picton makes an important point that is often taught in introductory epidemiology courses, but seems not to be generally appreciated: Diagnostic tests are usually developed using crystal clear cases with and without the target pathology, and this does not properly reflect the far more blurred differential diagnostic problem encountered in real life, wherein very small tumors and very large cochlear hearing losses intrude. Actual test accuracy or range of application will almost always be less than that claimed in the early stages of test development and application (Sackett et al., 1985). This phenomenon is combined with the problem that it rapidly becomes impossible and unethical to investigate the performance of a good test because it cannot be withheld from the diagnostic protocol. However, the problem of ABR testing being a common part of the referral route, and thereby introducing bias in test accuracy evaluation, does not seem significant in our situation.

In our view the use of the ABR with severe or profound hearing loss is limited. When hearing is worse than about 80 dB HL at 2 kHz (which usually means off the board at 4 kHz), the upper tolerance limit of our corrected interaural latency difference becomes very large, and we are obliged to say that the test is inconclusive. This occurs in about ten percent of our patients referred for ABR. It is in this group that vestibular function testing becomes valuable.

If MRI were inexpensive and available everywhere, the caseload for ABR testing would be much reduced. However, for us, MRI is a scarce and expensive resource. With publicly funded health care, very few MRI machines, and a strong trend towards cost-benefit analysis, this is unlikely to change soon. It can be argued that any patient with asymmetric or unilateral sensorineural hearing loss, as well as a few others (such as those who report unilateral difficulty with telephone use but have normal audiograms), is at risk for acoustic tumor. Although this means a lot of people, we continue to regard ABR testing as an economical and accurate screening tool. For a more detailed analysis see Hyde et al., (1991).

General Points

I support strongly several of Picton's themes. In all areas of evaluation and therapy it is important to develop decision protocols that link various test procedures in a consistent and structured way. Furthermore, this approach should include the formulation of explicit goals for the diagnostic or therapeutic protocol, such as to achieve particular accuracy and efficiency targets, as well as ongoing quality assurance monitoring to ensure that the protocol is being followed and is achieving its targets. This programmatic approach is superior to the popular informal idea of the test battery, which generally does not seem to inspire either quantification or accountability.

As Picton makes clear, AEPs offer a set of procedures; There is no best test appropriate for all objectives and circumstances. In most areas of interest, a good evaluative protocol ought to include more than one AEP procedure. Unfortunately, there is a tendency to become preoccupied with a single test and to apply it indiscriminately. This is certainly not in the interests of the patient. Unfortunately again, published reports that apparently support almost any position or practice, no matter how inappropriate, can usually be found. Training courses in how to distinguish the "wheat" from the "chaff" are few and far between, but there are good and very readable texts that address study evaluation and pitfalls in analysis (e.g., Sackett, Haynes, & Tugwell, 1985; Norman & Streiner, 1988). Moreover, the choice of evaluative protocol should be based on knowledge of local variables, especially the prevalence of various disorders, as well as upon multi-study meta-analyses of test or protocol performance. There are standard techniques (see, for example, Swets, 1988; Turner, Frazer & Shepard, 1984; Hyde, Davidson, & Alberti, 1991), but they certainly require time, effort, and expertise. Increasingly, it will be necessary for those who wish to influence audiologic practice to have a working knowledge in this area. Particularly with respect to adopting protocols that include several AEP procedures, tester training and maintenance of skills can be a problem. A rule of thumb might be that if a given tester performs any particular AEP procedure less than once a week, then he/she should not be doing it at all. It follows that for some clinical problems in some milieu, compromise test protocols may have to be adopted. Perhaps the most important thing is to be fully aware of the cause and extent of the deficiencies in protocol.

There is another complication. It is often assumed, especially by those with little or no hands on experience with AEP testing, that an ABR is an ABR is an ABR. This is not so. There are good and bad test conditions, good and bad instrumentation, good and bad tactics, and good and bad interpretations. There is a tendency to overlook this problem partly because of the illusion that AEP test results are objective. In reality, subjective judgment intrudes in many facets of AEP testing. There are some testers who achieve almost magical

accuracy in threshold estimation, for example, but there are others for whom no amount of training is sufficient. Thus, it may be desirable for those who require AEP testing services to shop around, and it is also desirable to try and verify whether anticipated test accuracy is actually being achieved. These points apply, of course, to many other non-trivial non-AEP procedures.

The Future

I would like to endorse Picton's prediction that AEPs will offer many more opportunities for functional analysis of the auditory system than are currently practised. The use of slow and late cortical responses (in the terminology of Davis, 1976) has much to offer. I urge interested readers and researchers to set the date limits on their computer searches in this area back to at least 1970, with emphasis on European work, to avoid "reinventing the wheel." It should be noted, however, that the lability of these responses and the complexities of controlling attentional variables have not decreased over the years. For this reason, I believe that the N100-P180 response will prove to be the most useful as a neurophysiologic correlate of perceived change in any feature of the auditory environment.

Finally, despite spatiotemporal dipole analysis, our current methods of stimulation and data analysis are rudimentary. In the next few years, much will be gained from more sophisticated applications of the theory and techniques of random process analysis, especially for cochlear and brainstem potentials. It will become increasingly clear that AEPs provide not one or two tests but an entire investigatory sub-discipline. Along with emissions, perhaps we might call it Clinical Auditory Physiology.
M.L.H.

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Reply to Commentaries: Some Final Words

My provocative review has triggered some excellent replies. These have extended, clarified, and rebutted my original comments. These final words will highlight some of the areas of agreement and disagreement.

Infant Hearing Assessment

The age at which infants at risk for hearing loss should be tested with the ABR remains in some dispute. In Canada, we have tended to test children after the immediate neonatal period. The ASHA Guidelines for Infant Hearing Screening suggest that the testing should be done while the baby is still in the hospital. Turner (1990) recently has critiqued these guidelines. He points out that it might be better to test infants at the age of 3-5 months rather than in the newborn period because the test is more accurate at this time and because the habilitation of the hearing impaired infant does not begin until at least several months of age. Hyde also makes the point that testing at 3-5 months of age will detect infants who have developed a hearing loss after birth or who have a progressive hearing loss. The major argument against testing at several months of age is that parents may not bring their baby back for evaluation. Parents will do so if it does not cost a great deal in terms of time and money; society should ensure that it does not.

A second question concerning the early identification of the hearing impaired infant is who should be tested. The ASHA guidelines propose that all infants should be screened initially by the high risk register and that the ABR should be used for those children who have some risk factor for hearing loss or who are referred because of parental concern. The major problem with this proposal is the significant number of children with a hearing impairment who do not have any recognized risk factors. In the best of all possible worlds (where everything is free) one would check the hearing of all newborn infants. Herrmann and Thornton describe the Algo-1 instrument and suggest that this can be used to test all newborn infants. Testing with this instrument is less expensive than conventional ABR testing because it assesses threshold more accurately and therefore causes fewer false alarms and because the testing does not require the expensive time of a skilled ABR interpreter. The recent paper by Jacobson and his colleagues provides clear support for the effectiveness of the Algo-1 instrument. A clear cost-benefit analysis of testing all newborn infants is now needed.

Conductive hearing loss in the neonatal period remains an interesting issue. The results of Durieux-Smith and her colleagues (1987) suggest that about 25% of infants in a neonatal intensive care unit have a conductive hearing loss. Many of these conductive hearing losses improve within the first few months of life. By the time of follow-up testing, the incidence of conductive hearing loss has decreased to about 8%. However, infants who have had a conductive hearing loss in the first few months of life are at a significantly greater risk for developing conductive hearing loss at a later age than those who do not have any middle ear problems in the newborn period (Durieux-Smith, Picton, Bernard, MacMurray, & Goodman, submitted). Herrmann and Thornton raise the possibility that some of the infants who appear to have a conductive hearing loss may actually have a transient neurological disturbance. This possibility needs to be investigated using bone conduction studies (Yang, Rupert, Moushegian et al., 1987; Yang & Stuart, 1990).

One of my suggestions was that electrocochleography should be more widely used in the assessment of infants. If there is an absent or distorted ABR, electrocochleography can accurately assess the threshold in the cochlea independent of the central nervous system (Aran, 1978). Hyde correctly points out that electrocochleography need not necessarily be done using trans-tympanic electrodes. The cochlear nerve action potential can be recorded quite well from the mastoid in infants. Ear canal or trans-tympanic recordings therefore may only be necessary if the mastoid recordings are not convincing.

Herrmann and Thornton express some scepticism about patients who have normal hearing thresholds but absent ABRs. Although rare, such patients do exist. Starr, McPherson,Patterson, Don, Luxford, Shannon, Sinninger, Tonakawa, and Waring (in press) have described in detail a patient without ABRs who had relatively good thresholds for pure tones and no clear neurological disorder. This patient performs very poorly on psychophysical tests that require accurate timing. She therefore seems to be missing the synchronized activity of "time keepers" in the central nervous system.

Otoacoustic Emissions

My paper considered the otoacoustic emissions elicited by the transient stimuli. Continuous stimuli can also evoke emissions, although these have not been studied as extensively. Continuous emissions at the same frequency as the stimulus are difficult to differentiate from acoustic echoes. However, the continuous response may also contain a "distortion product" otoacoustic emission that cannot be explained by acoustic echoes (Lonsbury-Martin & Martin,1990). Two elicited frequencies (f1 and f2, f1<f2) are presented. The nonlinear processing of the hair cells distorts the signal and creates some new frequencies, one of which is 2f1-f2. The presence of this distortion product can be used to assess the function of the cochlea in the frequency range between f1 and f2. The threshold for recording the distortion product emission can be related to the threshold for hearing over a range from about 0 dB to 50 dB HL. This technique can therefore provide information about hearing threshold that is both accurate and frequency specific. At the present time, however, hearing losses with thresholds above 50 or 60 dB cannot be assessed.

As Hyde points out, OAEs as yet have not been as extensively evaluated as the ABR. They hold great promise, but their clinical usefulness must be validated in the same way as the ABR. The ABR will always have some advantage in that it assesses auditory function in the auditory nerve and brainstem, whereas the OAE assesses only the hair cells. There may be disorders that can affect the auditory nerve fibres but not the hair cells. However, even these might show up in the OAEs because the hair cells are under the control of the efferent nervous system to the cochlea.

The relationship between the OAEs and the auditory evoked potentials will become a focus for research in the next few years. What is the relationship between the transient OAEs and the transient evoked potentials from the cochlear nerve and the brainstem? Are there any relationships between the distortion products seen in the OAEs and those seen in electrical recordings (Chertoff & Hecox, 1990; Rickman, Chertoff, & Hecox, in press)?

Frequency Specificity

Eggermont points out that frequency specificity depends upon both the stimulus and the response. In general, the longer the stimulus and the more gradual its rise time, the greater its frequency specificity. However, one must also consider the response system. Most evoked potentials respond to a wide range of stimulus frequencies. The actual generators may be different for each frequency, but the recorded waveforms are similar. Thus, an evoked potential may be elicited by whatever frequencies are present in the stimulus. The frequency specificity of the whole system combines the stimulus energy with the response transfer function. For example, the 40 Hz response is larger for low frequency stimuli than for high frequency stimuli. When a brief high frequency tone is presented, the response may be elicited by both the high frequencies (large stimulus energy but small response)and the low frequencies (low stimulus energy from spectral splatter but large response). The frequency specificity of the whole stimulus-response system can only be properly tested in patients with steep hearing losses. Masking can be used to evaluate the more peripheral responses (cochlear nerve, brainstem) because the masking of these responses appears to occur by means of a busy line mechanism. Unfortunately, for later evoked potentials, masking may have other effects. For example, noise of one frequency may attenuate a response to another frequency through inhibitory connections in the central nervous system and not because the noise is occupying a particular frequency region in the cochlea.

The most frequency specific stimulus is a continuous pure tone. Recently, a novel technique has been used to obtain frequency specific thresholds (Salt & Vora, 1990). An evoked potential is recorded using a brief stimulus. The response is then recorded again in the presence of a continuous pure tone with a particular intensity and frequency. Any significant difference between the two recordings indicates responsiveness to the pure tone. This process is effectively

the opposite of using notched noise because the continuous stimulus elicits the response (a change in the evoked potential to the transient stimulus) rather than prevents a response (to the spectral splatter).

Herrmann and Thornton suggest that brief tones alone may give sufficient information to assess the audiogram. Without any masking, this audiometric analysis will be limited by the frequency specificity of the stimuli. If the hearing loss has a slope of less than 30 dB/octave, the technique will be accurate. Hearing losses with steeper slopes will be underestimated. It is not technically difficult to add notched noise to the tone, to have the noise shift in intensity with the intensity of the tone, and to have the notch shift with the frequency of the tone. Audiometric instruments should provide this capability. Then the ABR information would become more accurate.

Hyde, and Herrmann and Thornton all agree that the middle and late auditory evoked potentials are important audiological tools. The choice of response recorded depends upon the clinical information required. Most people find the middle and late auditory evoked potentials to be reasonably frequency-specific. However, it would be worthwhile to have some clear assessment of their frequency specificity in patients with steep hearing losses.

The problem of the 500 Hz response remains controversial. Is there a clear 500 Hz brainstem response independent of overlapping later waves? If so, what is the threshold for eliciting this stimulus?

Eggermont points out that the derived response technique does show a good 500 Hz response when stimuli are presented at 13/s (once every 77 ms). There still may be a problem with overlapping latency waves at this rate of stimulation. The negative going transition between the Pb wave (60 ms) and the Nc wave (90 ms) will overlap with the response to the next stimulus and accentuate the positive-negative transition of the presumed wave V.

A maximum length sequence analysis (Eysholdt & Schreiner, 1982; Burkard, Shi, & Hecox, 1990) can be used to disentangle overlapping waves in the auditory evoked potential. I have recently used this technique to remove overlapping middle latency responses from the 500 Hz ABR. A preliminary analysis of the results suggests the following conclusions: There is a 500 Hz ABR independent of the middle latency response. However, the threshold for this 500 Hz ABR is about 25 dB higher than for the 2000 Hz ABR or the 500 Hz middle latency response.

Auditory Nerve Lesions

The prolonged I-V interval found in patients with an acoustic neuroma usually is explained in terms of some delay in the transmission of impulses through the region of the auditory nerve affected by the tumor. Eggermont and Don (1986) have provided evidence that the delay may result from disruption or desynchronization of the high frequency fibers so that the wave V is activated only by the low frequency fibers. The case reported by Herrmann and Thornton indicates that delays in transmission of high frequency fibers also may occur.

My original paper described some concern about the accuracy of the ABR in detecting lesions of the auditory nerve. Several people have discussed with me patients who have had an acoustic neuroma despite a normal ABR. I must confess that I misread the study by Joseph and her colleagues. The patients in this study were chosen to test the effectiveness of the ABR technique and not to demonstrate the incidence of false-negative results. It is comforting to get assurance, from those who see many more acoustic neuromas than I do, that the accuracy of the ABR remains about 95%.

However, I still do have some nagging doubts about the effectiveness of ABR in detecting small tumors. It would be worthwhile to know the incidence of abnormal ABRs in a large series of small tumors. There are several questions that could be asked of such a series. Would it be worthwhile to refer patients for MRI at a somewhat lower level of ABR abnormality than we do at present? It is probably worthwhile to err conservatively if the referring physician wants to know about multiple sclerosis, but this may not be so if one is considering the possibility of an acoustic neuroma. The rest of the audiological test battery perhaps could help in the decision to refer for MRI or not. For example, a unilateral attenuation of speech discrimination in the absence of an abnormal ABR may still warrant further investigation with MRI.

Evaluation Processes

Hyde correctly brings up the importance of using proper technique to evaluate our testing procedures. I heartily support this idea. We must learn the accuracy of our tests through signal detection theory. As well as delineating how well a test result indicates a particular pathology, signal detection period can also assist us in detecting the auditory evoked potentials (Valdes-Sosa, Bobes, Perez-Abalo, Perera, Carballo, & Valdes-Sosa, 1987). We must also use our tests in accordance with a full cost-benefit analysis (Prager, Stone, & Rose, 1987). This has not been performed for the use of evoked potentials in screening all newborn infants or for the use of the ABR (versus the MRI) in identifying patients with an acoustic neuroma.

Our data should always be open to critical discussion and sceptical appraisal. On this note, the reader should realize that my provocative comments on the use of auditory evoked potentials in intra-operative monitoring and in neurology have not, as yet, been critically assessed.

T. W. P.

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Call for Papers

A call for papers has been issued for the annual Canadian Acoustical Association Conference, October 9-10, 1991, Edmonton, Alberta, Canada. Abstracts on all aspects of acoustics are welcome, however papers on assistive listening devices, speech intelligibility, electronic augmentative communication devices, and language processing are especially encouraged.

Abstracts of 300 words or less must be received by April 30, 1991 in order to be included in the conference proceedings. Submissions and format enquiries should be directed to:

Dr. M.G. Faulkner, Dept. of Mechanical Engineering, U. of Alberta, Edmonton, Alberta T6G 2G8, Telephone: (403) 492-3446 Fax: (403) 492-2200