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ELECTRO-PHYSIOLOGICAL INVESTIGATION OF AUDITORY FUNCTION.

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Introduction

One of the ways in which we construct a description of our environment is through the sense of hearing. The acquisition of this sensory experience involves the perception of the acoustic stimulation as a sound, and such a perception requires the translation of the acoustic energy into a series of spatially and temporally continuous neural actions, permitting the description of the stimulus. Neural activity is first generated in the cochlea and is subsequently propagated centrally along the complex auditory pathways. In its ascension to higher stations, the complexity of neural activity increases until finally the whole network of central representation of the acoustic stimulus facilitates the perception of sound. The auditory electro-physiologist is able to detect, follow and measure neural activity by recording the concomitant electrical activity of both single neurons and neuronal populations. Such a technique allows the electro-physiological investigation of the peripheral and central structures of the auditory system. Auditory electric responses from these structures have been reviewed by Davis (1) and classified in terms of their latency between 0 and 600 ms. This paper outlines some of the ways in which auditory electric responses are used to investigate auditory function.

Estimation of hearing thresholds

A major advantage of using auditory electric responses to estimate hearing thresholds is that the patient is not required to make a conscious response to the stimulus. The psycho-physical thresholds can be estimated from the electric response thresholds obtained from patients who are unable or unwilling to co-operate in the normal audiometric test procedures. Auditory brainstem electric responses (ABER) have been used to estimate the hearing thresholds of infants up to the age of one year old, by tracing the Jewett V wave (J5) down to its threshold value (2,3,4). While infants can be tested during normal sleep, children up to the age of five years old usually require sedation (as do older retarded or uncooperative children) in order to reduce contamination of the responses by myogenic activity. In normally hearing adults J5 is identifiable down to 10 dB above the psycho-physical threshold and is considered one of the most powerful threshold estimators (2). J5 can also be used to estimate the hearing thresholds of any adult unwilling or unable to cooperate and, in combination with the slow vertex response (SVR) thresholds, can often give the only reliable estimates of thresholds for patients with non-organic hearing loss (NOHL). Since the SVR is probably generated in the auditory cortex and association areas, its threshold values might be considered to most accurately represent the true psycho-physical threshold. The SVR is, however, intrinsically more variable and more affected by attention states and levels of drugs than is the J5 response. Thresholds from both the SVR and J5 can be estimated at different frequencies.

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Location of the Lesion site

1. Response component identification.

One of the important considerations in audiometry is to try to differentiate between conductive, cochlear and neural contributions to the hearing problem. The use of auditory electric responses can give valuable information on location of the lesion site for any patient with a hearing problem. The first requirement, however, is to identify the response and compare it to a standard response from the normal population. Response component identification for an abnormal response can be one of the greatest difficulties. For persons with relatively little experience in the art of identification of auditory electric responses, it is often an easy procedure to identify the components, say, of the brainstem electric response. For an abnormal response, however, component identification must remain questionable. The procedure of identifying components with respect to normal latency values and normal waveform shape is unacceptable since they do not necessarily represent the abnormal response. Probably the safest method for identification of the components of the brainstem electric response is to trace J5 down to threshold. In most cases the sole remaining peak at just above threshold can then be confidently identified as J5. The other components of a supra-threshold brainstem electric response can then be identified using J5 as a reference point. However, even using this technique, abnormal waveforms with either an extra component or an absent component can evade conclusive component identification.

2. The effect of various pathologies.

Having arrived at an acceptable stage of component identification, pathological deviations from the normal can be measured in terms of peak-to-peak amplitude and peak latency values of the components. The most reliable parameter is peak latency, but peak-to-peak amplitude values are also useful. The amplitude and latency measures of the response can then be compared directly to corresponding values for responses from a normally hearing population. Patients with conductive audiometric deficits give abnormally delayed brainstem responses. However, at a stimulation intensity level compensating for the amount of their conductive loss (equivalent value above threshold level), brainstem response latencies are normal. For conductive and cochlear hearing losses which show threshold increase at certain frequencies, the brainstem responses will be affected by the audiogram profile. In cases of high frequency loss latencies are delayed, since they represent neural activity initiated from a less basal part of the cochlea than in the normal case. It is also reasonable to expect the earlier components of the brainstem response (J1 to J3) to decrease in amplitude relative to the later components (J4 to J5) since the former are more dependent on the higher frequencies of stimulation. In cases of severe cochlear deficit components J1 to J3 and often J5 may not be detectable until high intensities of stimulation are used (120 dB PESPL), displaying an abnormally marked change of amplitude and latency values over a relatively small intensity increment. This steep intensity input/output function is common in patients with recruitment and is indicative of a cochlear problem (3). In cases where there is cochlear and peripheral neural involvement in the hearing problem, it is difficult to conclusively demonstrate the neural component. The problem of the peripheral neural auditory system will have an effect on the brainstem responses which may not be detectable in the presence of a cochlear pathology.

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If the hearing problem has a neural aetiology the effect on the brainstem response will depend on the location of the neural lesion. Lesions of the auditory nerve can give varying types of abnormal responses, for example, if the high frequency fibres are affected causing a delay in neural propagation, this can result in the absence of J2 due to waveform component cancellation. Lesions affecting the peripheral end of the acoustic nerve may completely abolish the response altogether (as will severe cochlear pathologies). If in this situation normal cochlear microphonics were obtained, the hearing problem could be localized to a peripheral neural origin. Lesions affecting the central end of the acoustic nerve may give a waveform with component J1 and no other identifiable components, as in cases of cerebellar pontine angle tumours.

In cases where components J1 and J5 can be confidently identified, neural involvement in the hearing problem can be demonstrated by the presence of an abnormally long delay between J1 and J5 (5). The delay is often referred to as the central conduction time and can be tested against J1-5 latency delays in a normally hearing population. The lesion causing the delay can be further localized by measuring the latency delays between the major components of the response (J1, J3 and J5). Abnormal latency delays between J3 and J5 are indicative of a neural problem in the brainstem, while abnormal latency delays between J1 and J2 are indicative of a problem around the central end of the acoustic nerve. A more definitive localization of lesion site in terms of the generating source of each of the waveform components (J1 to J7) should be approached with caution. Experiments (6, 7, 8) have demonstrated the primary sources of the brainstem responses in animals, but there have been a number of different interpretations of these findings when considered in the light of studies in man with specific neural lesions (5, 9, 10). It is likely that, apart from the first neural component (J1) which is generated by the neurons of the auditory nerve, successive components (J2 to J7) are the result of complex summation of contributions from several generating sources. The brainstem electric response, as represented at any one electrode, is the product of neural activity generated in a number of spatially separated structures and at a latency dependent on the neural propagation pathway and the initiating event at the cochlea. The resulting waveform is, therefore, very complex and it is probably a gross over-simplification to attribute any one component (J2 to J7) to any one generating source.

Finally, the effect of delay or attenuation of waveform components, produced by auditory pathologies, can be simulated by a model using the derived components of the brainstem responses (11). The effect of the audiogram profile on the total response can be predicted and, in the absence of recruitment, can be used to assess the possibility of neural involvement in the hearing problem. The recent advances in the types of electro-physiological investigation of the auditory system add a powerful dimension to audiometric testing procedures and allow the audiologist to make a more accurate diagnosis of the many auditory problems presented by patients.

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