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Auditory brainstem evoked potentials in sudden deafness†

by

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Abstract

Auditory Brainstem Evoked Potentials (ABEP) were recorded from 53 adult subjects suffering from sudden deafness at the time of examination. The onset of sudden deafness was up to three years prior to the study. ABEP were recorded in response to 75 dBHL clicks presented at rates of 10/sec and 40/sec. ABEP peak latencies as well as interpeak latency differences, and also the effect of increasing stimulus rate, were determined for each patient and compared with the clinical symptoms. The psychoacoustic and ABEP detection thresholds were also compared.

A highly significant correlation was observed between ABEP detection threshold and the psychoacoustically determined hearing threshold, and the differences between them were found to be insignificant. The results of this study indicate a central component in sudden deafness. This impairment manifests itself in abnormal effects of increasing stimulus rate on ABEP, and may be related to reduced central synaptic efficacy.

Introduction

Sudden deafness is an important clinical entity which demands immediate attention and treatment (Goodhill and Harris, 1979; Ballantyne, 1979). It may be the result of damage in one or more of the following sites: external ear, middle ear, inner ear, central nervous system or the cerebello—pontine angle. Thus, sudden deafness may be conductive or sensori-neural (Siegel, 1975) with severity ranging from mild, through severe, to total hearing loss. In the majority of cases it is unilateral and accompanied by vertigo, tinnitus and vomiting.

Sudden deafness is a term applied to a wide range of etiological, histopathological, prognostic and medical management entities. In this study, we compared Auditory Brainstem Evoked Potentials (ABEP) of patients with sudden deafness with the accompanying clinical symptoms, in an attempt to localize the site affected and to indicate the possible etiology.

Materials and methods

Over a period of three years, 72 patients were hospitalized for sudden deafness in ENT departments of four hospitals serving a population of 750,000. In this study, 53 of these patients were examined. Of these, 23 had left ear impairment (43.5 per cent) and 30 had right ear hearing loss (56.5 per cent). The time between the appearance of the sudden deafness and hospitalization was between one day and two months. Sudden deafness lasted up to

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one week in 32 of the patients (60 per cent). The patients' age ranged from 21 and 80 years (average 46 years). In addition to hearing loss, three other complaints were reported: tinnitus (in 64 per cent of the patients); vertigo (in 32 per cent); and vomiting (in 24 per cent).

Various treatments were applied to these patients, depending on the policy of the respective department. Medication included vasodilators, steroids, anticoagulants, antibiotics and muscle relaxants.

In 68 per cent of the patients an improvement in hearing was observed within up to three years of the onset of treatment. The improvement was from 5–90 dB, of which 50 per cent were in the 5–30 dB range.

ABEP were recorded from all patients within three years of their hospitilization. Potentials were evoked by 75 dBnHL clicks (maximum energy between 1.5 and 3 kHz) presented to each ear in turn at a rate of 10/sec, and then 40/sec. The potential difference between vertex and the mastoid ipsilateral to the stimulated ear (contralateral mastoid grounding the patient) was amplified (×200,000) at a band pass of 100–3000 Hz. The potentials following each click were sampled (dwell time of 100μsec, 256 addresses per sweep), and 2,048 sweeps were averaged to produce each record. Two records of each stimulus condition were superimposed to assess reproducibility.

An electrophysiological estimate of hearing threshold was carried out for each ear in turn by recording ABEP to successively lower click intensities. 20/sec clicks were presented at intensities decreasing by 10 dB at a time, until no potentials could be detected. Following ABEP recording, pure tone audiometry was conducted on each patient.

ABEP normative measures were described in a previous report (Pratt *et al.*, 1981). In short, interpeak latency differences [IPLD] (V–I, V–III and III–I) were computed for 10/sec and 40/sec click presentations. In addition, the net effects of increasing stimulus rate from 10/sec to 40/sec were computed by subtracting the respective IPLD values at 10/sec from those at 40/sec. Data analysis included comparisons of audiometric thresholds with ABEP detection thresholds and concordance of ABEP findings with clinical symptoms.

The correlation between psychoacoustic and ABEP detection thresholds was calculated in terms of linear regression, and its significance was determined by Student's t-test. The significance of the differences in hearing thresholds determined psychoacoustically and by ABEP was determined using Student's t-test for paired differences. Probabilities below 0.05 were considered significant.

Results

In 90 per cent of the ears examined, ABEP detection threshold and psychoacoustic audiometric threshold for 2–3 kHz were within 15 dB of each other. In the ears affected by sudden deafness, a significant correlation was observed between psychoacoustic and ABEP thresholds [$R=0.729$, $t(41)=6.819$, $p<0.0005$]. The average difference between ABEP detection threshold and the psychoacoustic hearing threshold, in the affected ears, was 3.7 dB. This difference was found to be insignificant in a Student's t-test of paired differences [$t(42)=1.15$].

A comparison of the clinical and ABEP findings using 10/sec click rate, revealed that 36 of the 49 ears with sudden deafness (75 per cent) were normal with respect of ABEP and 13 (25 per cent) had abnormal ABEP findings. Four ears (7.5 per cent) revealed ABEP records that were considered difficult to interpret. ABEP abnormalities included: (1) prolonged peak latencies (in 11/13, i.e. 85 per cent); (2) Prolonged IPLD (V–I) (in 7/13 ears, i.e. 54 per cent); (3) randomly missing components (in 12/13, i.e. 92 per cent). These 13 patients had an average age of 51 years (range of 21–80), six were male and seven female, nine (70 per cent) of them complained of tinnitus and only a few complained of vertigo and/or vomiting.

In seven of the 13 patients with abnormal ABEP to 10/sec clicks, IPLD (V–I) was prolonged relative to the upper normative value (4.30 msec). Abnormal IPLDs ranged from 4.35 to 4.80 msec. Four of these patients complained of tinnitus (57 per cent), and three

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suffered from vomiting and vertigo (43 per cent). IPLD (V–III) was abnormally prolonged (longer than 2.20 msec) in six of the patients, and ranged from 2.30 to 3.15 msec. Four of these patients (67 per cent), suffered from a combination of tinnitus, vomiting and vertigo, and of these, two had a prolonged IPLD (V–I) as well. When the findings of the affected ear of each subject were compared with the respective findings of the unaffected ear, IPLD (V–I) was found beyond the upper normative limit (interaural difference of 0.35 msec) in 10 of the patients.

When ABEP were recorded in response to an increased stimulus rate of 40/sec, abnormality increased to 27 of the 53 patients (51 per cent). Two records were difficult to interpret. Of the 27 patients with abnormal ABEP to 40/sec clicks, seven complained of a combination of tinnitus, vertigo and vomiting (26 per cent), and the rest complained of combinations of only two of these symptoms in approximately similar proportions.

In seven of the 27 patients (26 per cent) with ABEP abnormality to 40/sec stimulation, the net effect of increasing stimulus rate on IPLD (V–I) was beyond the normative upper limit (0.45 msec). This net effect was calculated by subtracting the IPLD at 10/sec from the respective value obtained with 40/sec click rate. When the net effect of increasing stimulus rate was calculated for IPLD (V–III), abnormality was observed in 14 ears (52 per cent), i.e. the net effect was beyond 0.35 msec). The interaural difference in IPLD (V–I) with a click rate of 40/sec was abnormal (<0.15 msec) in 18 of the patients (67 per cent). Of these 18 patients, only seven showed abnormal interaural difference at 10/sec stimulus rate.

Discussion

In this study, when the psychoacoustic hearing threshold was compared with the ABEP detection threshold, over 90 per cent of the patients with sudden deafness showed concordance within 15 dB. The average difference between the two estimates of hearing was about 3 dB and insignificant. The significant correlation between psychoacoustic and ABEP thresholds can be used as a convenient and reliable indicator of hearing level, to be performed on patients with sudden deafness, who may have a psychogenic component in their hearing loss.

The most striking finding of this study was the high incidence of abnormality in the effect of increasing stimulus rate on ABEP. Half of the patients with sudden deafness had abnormal ABEP findings in response to increasing stimulus rate. Over a half of the patients with this abnormality had no indication of abnormality when only 10/sec clicks were used. The most common indicator of rate-related abnormality was asymmetry in the effect of increasing stimulus rate of IPLDs of the affected and non-affected ear. None of the patients with abnormal effects of increasing stimulus rate regained normal hearing. The effect of increasing stimulus rate has been reported in a variety of clinical entities (Robinson and Rudge, 1977; Daly *et al.*, 1977; Yagi and Kaga, 1979; Pratt *et al.*, 1981; Pratt *et al.*, 1986; Ben-David *et al.*, 1986). The common denominator of all these entities had been suggested to be decreased efficacy of synapses along the auditory pathway (Pratt *et al.*, 1981). The findings of this study may indicate a central component in sudden deafness.

ABEP abnormality was most commonly found in patients with a triad of complaints which included tinnitus, vertigo and vomiting. This group was characterized by a high sensitivity to increased stimulus rate. Over half of these patients had abnormal effects of increasing stinulus rate. Only a quarter of them had abnormality when 10/sec stimulation was used. The coincidence of the three complaints with ABEP abnormality may indicate a more severe pathology in these patients. Accordingly, in these severe cases, the central impairment is expressed not only in auditory symptoms (tinnitus and hearing loss), but in vestibular symptoms (vertigo and vomiting) as well.

The clinical benefits of various treatments of sudden deafness, such as vasodilators, anticoagulants, hyperbaric treatments, etc., have usually been attributed to their effects on the inner ear and its metabolic well being. The same agents could influence more central con-

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stituents of the auditory pathway. The results of this study, in conjunction with other studies that have demonstrated the sensitivity of ABEP to increased stimulus rate in central impairments, support a central effect of these therapeutic agents in addition to the inner ear.

In conclusion, the results of this study indicate a possible central component of sudden deafness. This brainstem impairment is associated with the triad of tinnitus, vertigo and vomiting symptoms, and may be related to decreased efficacy of synapses.

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