Auditory brain stem responses in infants with posthaemorrhagic ventricular dilatation

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SUMMARY

Nineteen infants with posthaemorrhagic ventricular dilatation had auditory brain stem responses measured during the period of maximal ventricular dilatation. These showed various patterns ranging from normal, through various abnormalities, to complete absence of responses. When serial auditory brain stem responses were studied in parallel with the evolution of posthaemorrhagic ventricular dilatation it was seen that the abnormalities of auditory brain stem response usually resolved irrespective of the persistence or progression of ventricular dilatation. No correlation was found between cerebrospinal fluid pressure and prolonged interpeak intervals on the auditory brain stem response. In three patients with posthaemorrhagic ventricular dilatation improvement in the auditory brain stem response occurred when cerebrospinal fluid was withdrawn. Intermittent withdrawal of cerebrospinal fluid (by ventricular tap or lumbar puncture) in two of these infants was followed by improvement in the auditory brain stem response after a period of 24 hours (but not sooner). In one infant born at full term improvement in the auditory brain stem response was noted one week after shunting.

Disturbances in auditory brainstem responses have been reported in disorders associated with ventricular dilatation in both infants and children, and the use of the auditory brain stem response to assess the possible impact of ventricular dilatation on the brainstem has been described.

The aim of the present study was to evaluate the auditory brain stem response abnormalities in infants with posthaemorrhagic ventricular dilatation and to determine whether such abnormalities could be altered by drainage of cerebrospinal fluid.

Patients and methods

Of the 855 infants admitted to the Hammersmith Hospital neonatal intensive care unit between October 1981 and April 1984, 145 had periventricular haemorrhage diagnosed on ultrasound scanning during the first week of life; 18 of these (12%) developed posthaemorrhagic ventricular dilatation. Two further infants who were transferred to the unit with established posthaemorrhagic ventricular dilatation at the ages of 2 and 8 weeks, respectively, were included in the study. Of these 20 infants, 19 were fit for testing. The remaining infant was critically ill and considered unsuitable for assessment of auditory brain stem response; he subsequently died.

The diagnosis of posthaemorrhagic ventricular dilatation was made when two or more measurements of ventricular width on ultrasonography exceeded by at least 4 mm the 97th centile of the measurements appropriate for the gestational age determined by Levene.

Of the 19 infants with posthaemorrhagic ventricular dilatation, 16 were preterm (gestational ages 26–34 weeks) and three had been born at full term (38–39 weeks). Gestational ages were calculated from gestational assessment and the dates of the mother's last menstrual period, which agreed within one week in all cases.

The recordings of auditory brain stem response were usually made while the infant was lying prone with one ear on the mattress. An earphone was held gently over the other ear taking care not to occlude or collapse the external auditory canal, which may result in appearances simulating a conductive lesion. Rarefaction clicks, 100 µs square waves, at a rate of 10/s were made into one ear through a TDH-39 earphone. Each response was calculated from a total of 1024 to 2048 stimuli. Two machines were used; the first a Medelec MS6 model using a bandpass filter of 250–1600 Hz, the second a Medelec Sensor machine using a filter of 300–3000 Hz. Three standard electrodes were used, the active electrode was placed over the vertex (M1), and the reference...
eleven preterm infants and one born at full term were studied. In the preterm infants periventricular haemorrhage or intraventricular haemorrhage of variable severity were diagnosed during the first week of life. During the same time auditory brain stem responses were recorded and were abnormal in all 11 infants. The haemorrhages and sequential auditory brain stem responses were measured and showed improvement at a time when the ventricular dilatation was still present on the scans, or even increasing in size. In five of the 11 preterm infants the auditory brain stem responses returned to normal although there was still pronounced dilatation of the ventricles. Fig 1 gives an example of these five infants. In three the auditory brain stem responses remained abnormal at the ages of 10, 14, and 20 weeks, respectively. Though the ventricles decreased in size during this period, they still remained moderately dilated.

**Results**

**Effects of the Evolution of Posthaemorrhagic Ventricular Dilatation on the Auditory Brain Stem Response**

Eleven preterm infants and one born at full term were studied. In the preterm infants periventricular haemorrhage or intraventricular haemorrhage of...
Auditory brain stem responses in infants with posthaemorrhagic ventricular dilatation

In one infant the auditory brain stem response remained abnormal and the child was referred to hearing specialists. Two infants died at the ages of 2 and 4 weeks, respectively.

A single infant born at full term with posthaemorrhagic ventricular dilatation was also studied and showed an initially normal auditory brain stem response. Ventricular dilatation increased progressively over the next 10 weeks. A repeat recording early in this period remained normal. Subsequent auditory brain stem responses measured at the age of 3 months showed deterioration.

Effects of posthaemorrhagic ventricular dilatation on the auditory brain stem response at time of maximum ventricular dilatation

Auditory brain stem responses were measured in 19 infants during the phase of maximum dilatation of the ventricles. Five records were normal and 14 were abnormal. The abnormal tracings showed various patterns. Two infants had ‘severe’ impairment: absent responses in one, and appreciably reduced wave amplitude in the other (figs 2a and b, respectively). Two infants had ‘peripheral’ abnormalities including absent wave amplitude or wave I more than two standard deviations from the mean (fig 2c). Five infants had ‘central’ abnormalities. Three showed prolonged I–V interpeak intervals (more than two standard deviations from the mean), one with an absent wave V, and one with an abnormal V:I amplitude ratio (figs 2d and e). Two infants had short I–V interpeak intervals but normal wave configuration. Three infants had abnormal and non-reproducible traces on repeated testing. These were classified separately.

Effects of cerebrospinal fluid pressure on the auditory brain stem response

Nine infants had serial cerebrospinal fluid pressures and auditory brain stem responses recorded, six of whom had the auditory brain stem responses recorded on the same day and within a few hours of recording the cerebrospinal fluid pressures. In these six infants therefore (five preterm and one born at full term) a total of 21 direct cerebrospinal fluid pressure measurements were correlated with the auditory brain stem response measurements that were recorded simultaneously. There was no correlation between the cerebrospinal fluid pressure and the presence of auditory brain stem response abnormalities as assessed by the I–V interpeak interval (fig 3).

Effects of cerebrospinal fluid drainage on the auditory brain stem response

In seven infants the auditory brain stem responses were studied before and after drainage of cerebrospinal fluid. In all seven the auditory brain stem responses were abnormal before the drainage was carried out. Repeat measurement of auditory brain stem responses after two to six hours showed little change, but auditory brain stem responses measured 24 hours after drainage showed an increase in amplitude in two of the four cases where this was tested (fig 4).

Four infants had shunts inserted. Auditory brain stem responses measured before the shunts were inserted were normal in one, unequivocally abnormal in another, and showed a pattern of uncertain importance in the remaining two, with normal wave
Lary, De Vries, Kaiser, Dubowitz, and Dubowitz

morphology and hearing thresholds, but interpeak intervals shorter than were appropriate for their ages. The shunting procedure had no effect on the auditory brain stem response in one patient in whom the auditory brain stem response was normal before shunting or in two patients who had abnormalities in the auditory brain stem response of uncertain importance. In the one infant born at full term, however, the abnormal auditory brain stem response recorded before the shunt reverted to normal within a week of the shunt being inserted (fig 5).

Discussion

Previously reported abnormalities in auditory brain stem responses in children with hydrocephalus were found in much older subjects or in children of unspecified age. In some instances the cause of the hydrocephalus was not documented. Previous investigators have showed that in the presence of ventricular dilatation there were increased I-V interpeak intervals that they attributed to increased intracranial pressure. We know of no previous publications on the association between the changes in auditory brain stem response and the development of posthaemorrhagic ventricular dilatation, the

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**Fig 3** Simultaneous cerebrospinal fluid pressures and interpeak intervals I-V recorded on 12 occasions from five preterm infants (black circles), and on nine occasions from one infant born at full term (white circles). The upper limit of normal cerebrospinal fluid pressure is shown by the horizontal line.

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**Fig 4** Effect of ventricular tap on the auditory brain stem response of a baby girl born at 29 weeks' gestation with right sided periventricular and intraventricular haemorrhages on ultrasonography. Before drainage the auditory brain stem responses (both left and right) showed abnormal waveforms and latencies. One hour after cerebrospinal fluid tap there was little change in the auditory brain stem response (a unilateral increase in the amplitude of wave I on the left). The responses remained abnormal. When auditory brain stem responses were measured again 24 hours later, there was considerable improvement, particularly on the left, where both latencies and I-V interval had returned to normal limits. The right side remained abnormal (prolonged I-V interval and abnormal waveforms). All stimuli at 80 dB and 10 clicks/s.
Auditory brain stem responses in infants with posthaemorrhagic ventricular dilatation

Effects of intermittent drainage of cerebrospinal fluid, or the rise in intracranial pressure. In the present study, we found no correlation, either positive or negative, between the I-V interpeak interval and the intracranial pressure in preterm infants, and this is probably due to better adaptation of the immature brain to increased intracranial pressure.

The mechanisms whereby the brains of preterm infants can adapt to increased intracranial pressure are open to speculation. One may be the balance between increased ventricular volume and the resorption of extracerebral cerebrospinal fluid. When the limits of compensation are reached a sustained rise in the intracranial pressure will ensue giving rise to the classic signs of increased intracranial pressure with increasing head circumference, tense anterior fontanelle, and suture diastasis.11

Fig 5  Effect of shunting on the auditory brain stem response in an infant born at full term with posthaemorrhagic ventricular dilatation. (a) Initial auditory brain stem responses show normal latency and wave configuration at 60 and 40 dB. Ultrasonography shows some ventricular dilatation. (b) Effects of progressive ventricular dilatation lasting over three months (pronounced deterioration of wave configuration, and disappearance of responses at 40 dB). Both lateral ventricles show pronounced dilatation on ultrasonography carried out on the same date as the auditory brain stem response. (c) Auditory brain stem response measured one week after shunting showing reversion to normal. The ultrasound scan showed that the ventricles, while still dilated, had diminished in size. All stimuli at 10 clicks/s.
Necropsy studies have also shown that in preterm infants with posthaemorrhagic ventricular dilatation considerable degrees of dilatation can occur before there is any rise in intracranial pressure.\textsuperscript{12, 13} Periventricular oedema, produced by cerebrospinal fluid extravasating through ruptures in the ependyma, may compress periventricular capillaries and cause ischaemic injury.\textsuperscript{14} Hill and Volpe have also stressed that the immature state of the periventricular tissue and the fact that it has been compromised by a variety of hypoxic and ischaemic insults may influence its adaptation to increased intracranial pressure.\textsuperscript{15}

Another factor influencing this adaptation is the comparatively large subarachnoid space within the cranium of the premature infant.\textsuperscript{12, 16} Among preterm infants there is a wide variation in the size of the brain and of the extracerebral space depending on the pressure to which the head has been subjected in the uterus and on variations in fetal maturity and nutrition.\textsuperscript{13} These facts are probably relevant to our findings concerning the association between intracranial pressure and I-V interpeak intervals.

The data should probably not be extrapolated to infants born at full term or to older children. Our data for our single infant born at full term were similar to those previously reported. Nagao \textit{et al} reported associated reductions in the amplitudes of waves IV and V, and prolonged latencies of waves III, IV, and V after experimentally induced intracranial hypertension in cats (the intracranial pressure being raised by expansion of a supratentorial balloon).\textsuperscript{17} Although increased intracranial pressure and abnormalities in auditory brain stem response have been described in animals and in humans,\textsuperscript{17–19} Kraus \textit{et al}\textsuperscript{5} found ABR abnormalities in auditory brain stem response in patients after shunts had been inserted in the absence of increased intracranial pressure.

Much less is known about the specific associations between intracranial pressure and brain stem function. Goodman and Becker studied the vascular abnormalities in the brain stem induced by intracranial hypertension in cats.\textsuperscript{20} They found that as intracranial pressure rose there was a parallel progression of vascular disturbances, first in the microcirculation and finally in the macrocirculation. Such reversible ischaemic changes in the brain stem caused by alterations in blood flow may account for the changes in auditory brain stem response on intermittent withdrawal of cerebrospinal fluid seen in some of our patients. In an experiment on cats, Nagao \textit{et al} found progressive reduction in the amplitude of waves IV and V as the intracranial pressure was gradually increased by balloon infla-

followed by progressive recovery during a three hour period after the balloon had been deflated.\textsuperscript{21} They suggested that these changes were due to direct mechanical pressure. The changes were clearly not irreversible. Recently Little \textit{et al} showed that intraoperative auditory brain stem response recording during surgical treatment of a basilar artery aneurysm resulted in contralateral amplitude reduction of waves III-V only during pontine retraction.\textsuperscript{22} The effect was transient and the patient completely recovered. Another explanation for our data could be the recovery of brain cells from interstitial oedema (or effused cerebrospinal fluid), a recovery reflected in an increase in wave amplitude.

This study has shown that in premature infants abnormalities in auditory brain stem responses may resolve irrespective of the persistence or progression of ventricular dilatation. No correlation was found between cerebrospinal fluid pressure and certain abnormalities of auditory brain stem responses (such as prolonged interpeak interval) but an improvement of the auditory brain stem response—especially of the amplitude—could occur after drainage of cerebrospinal fluid.

A larger number of infants born at full term needs to be studied to observe whether their response to ventricular dilatation and raised intracranial pressure is indeed different from that in preterm infants.

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Auditory brain stem responses in infants with posthaemorrhagic ventricular dilatation


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