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Development of Auditory Function in Newborn Infants Revealed by Auditory Brainstem Potentials

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ABSTRACT. Auditory brainstem potentials were recorded from scalp electrodes in 42 infants ranging in gestational age from 25 to 44 weeks. The latencies of the various potential components decreased with maturation. Wave V, evoked by 65-dB sensation level clicks, changed in latency from 9.9 msec at 26 weeks of gestation to 6.9 msec at 40 weeks of gestation. Central conduction times in the auditory pathway also decreased with maturation from 7.2 msec at 26 weeks to 5.2 msec at 40 weeks. The effects of brainstem and cochlear disorders on auditory brainstem potentials were noted in several abnormal infants. The application of all of these techniques could permit an objective definition of both normal and abnormal sensory processes in newborn infants. *Pediatrics* 60:831-839, 1977, AUDITORY DEVELOPMENT, BRAINSTEM POTENTIALS, NEWBORN INFANTS.

The clinical assessment of neurological and sensory functions of the newborn infant requires considerable experience and the results are usually described in qualitative terms. Recently, scalp-derived cortical brain potentials evoked by auditory,¹⁻⁴ visual,⁵ and somatosensory stimuli⁶ have been used to provide quantitative measures of changes in sensory function in the newborn. These techniques primarily sample cortical activity and the evoked potentials' amplitude and latency can vary with the infant's level of arousal. Procedures have now been developed for measuring sensory function in subcortical portions of one of the sensory systems, the auditory brainstem pathway, where arousal asserts relatively little effect.⁷

Auditory brainstem potentials are the far-field reflection of electrical events generated within the auditory pathway in its course through the brain. These potentials can be recorded from

scalp electrodes in humans, using computer averaging techniques, and consist of seven deflections of submicrovolt amplitudes in the initial 10 ms following a click signal.⁸ The components, designated by Roman numerals, are thought to derive from the sequential activation of the nuclei and pathways comprising the auditory system. Studies in both animals^{9,10} and humans¹¹ suggest that wave I represents activity of the VIII nerve, wave II represents activity of the cochlear nuclei, wave III represents activity of the superior olive, and waves IV and V represent activity of the inferior colliculus. The origins of waves VI and VII have not yet been established. The latencies of the brainstem potentials change in an orderly manner with signal intensity and are not influenced by level of arousal. The technique has been used to assess hearing in newborn infants,¹² to measure maturation of newborn infants,¹³ and to evaluate brainstem function in neurological disorders in adults.¹⁴⁻¹⁷

The present study utilized measures of auditory brainstem potentials in newborn and preterm infants to define the maturation of both peripheral (i.e., the cochlea) and central portions of the auditory pathway. In the course of study, we have encountered instances in which abnormalities of these potentials could be related to the infants' clinical condition.

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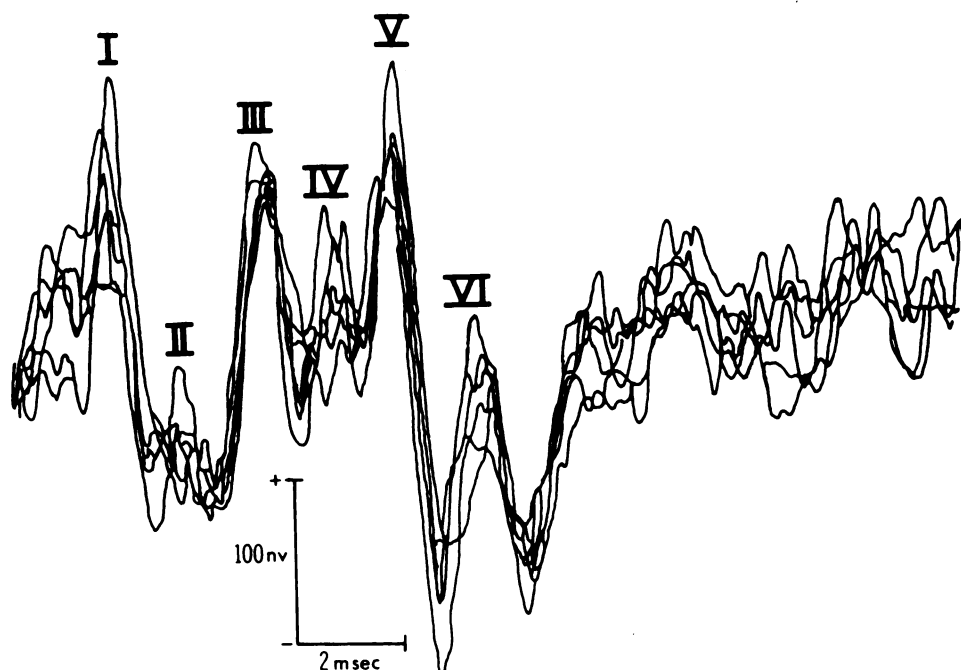


FIG. 1. Auditory brainstem potentials from normal term infant at 40 weeks' gestation, measured six separate times over two-hour period. Six distinct components designated I through VI can be identified. Click rate was 10/sec; click intensity, 65 dB SL. Positivity at vertex electrode in this and all subsequent illustrations is represented by upward deflection.

METHODS

The infants were patients in the neonatal intensive care unit, the term nursery, or the intermediate nursery of the University of California Irvine Medical Center. Informed consent for the evoked potential study was obtained from the parent. Assessment of gestational age was based on (1) the date of the first day of the mother's last normal menstrual cycle and (2) a detailed physical examination performed shortly after birth using a composite scoring system.¹⁸ The results from 42 infants ranging in original conceptional age from 25 to 44 weeks provide the data for this report.

The infants were tested in their bassinets or incubators. The equipment was on a small cart that was wheeled close to the patient. Three tin disc electrodes, 5 mm in diameter, were attached at the vertex (Cz) and mastoids, using a standard conducting medium (Grass electrode paste) and adhesive tape to hold the electrodes in place. The electrical potentials were recorded between the vertex electrode and mastoid electrode ipsilateral to the ear receiving the click signal. The other mastoid electrode served as the patient ground. The responses were amplified by a factor of 10^3 , filtered between 100 Hz and 3 kHz (3 dB down points) and averaged by a small digital computer. A 20-msec time base with a resolving capacity of

20 to 80 μ sec per point for 1,024 points was used. A total of 2,048 stimulus trials was averaged and a duplicate average was made at each signal intensity to assess reproducibility of results. The averaged auditory brainstem potentials were plotted by an X-Y plotter for measurements of the latency and amplitude of the various components.

The acoustic signals were clicks generated by passing alternating-polarity, 0.5-msec square waves through an attenuator and power amplifier before actuating a TDH 39 earphone housed in an MX-41 coupler. The earphone was held in place over the ear taking care that the pinna was not compressed. Only one ear was tested in each infant. The intensity of the signals was calibrated in decibels of sensation level (dB SL) referred to the mean hearing threshold of six normal hearing adults measured in the nursery environment.

Auditory brainstem potentials were measured with a click rate of 10/sec in response to signal intensities of 65, 45, and 25 dB SL. In some of the infants born before 28 weeks of gestation, additional tests were carried out at 75 dB SL. The potentials were measured within four days of birth in all infants. The evoked potentials in 24 of the 42 infants were reexamined at a later date.

Figure 1 shows averaged auditory brainstem potentials derived from a normal term infant (40 weeks' gestation) measured six separate times over a two-hour period. The components were

remarkably consistent in both form and amplitude except for waves II and IV. The latencies of the most reproducible and easily defined components (waves I, III, the IV-V complex, or V) were defined at their peak. Waves II, VI, and VII were too variable in appearance to allow systematic study. In those instances where a peak was flattened, lines were extended from the rising and falling slopes of the component in question and the peak defined by the intersection. The recording accuracy on repeated measures of the same waves was ± 0.1 msec. Waves IV and V were often fused into a single component designated as the "IV-V complex." Data on the latency of the complex have been included with wave V, based on studies in five sets of twins in whom one member of each set had distinct waves IV and V while the other member had a IV-V complex. The difference in latency between the measurements in each member of the twin set was within the error of the measuring technique.

A listing of the infants studied arranged according to gestational age; latency of the components I, III, IV-V at three signal intensities; birth weight; and clinical diagnosis is available on request from the National Auxiliary Publications Service.

Slow cortical evoked potentials to clicks were also studied in the four infants who were born before the 28th week of gestation. The same recording electrodes were used. The amplifier band pass was changed to 1 to 100 Hz. The click intensity was 65 dB SL and the click rate was 1/sec. A total of 256 clicks comprised the averaged response recorded on a 512-msec time base. The infants were quiet and appeared asleep at the time of testing. The EEG was not recorded simultaneously, preventing staging of the sleep cycle.

RESULTS

The latencies of the most prominent components (I, III, IV-V) of the brainstem potential from each infant to 65-dB SL clicks are plotted in Figure 2 as a function of gestational age. The latencies of all of the components decrease as gestational age at birth increases, with the maximal shift occurring before 34 weeks.

For instance, a two-week difference in gestational age between 32 and 34 weeks was associated with a 0.8-msec decrease in the latency of the IV-V component. A comparable two-week difference between the 38th and 40th weeks of gestation was accompanied by only a 0.1-msec latency change.

The first infant tested in the most immature

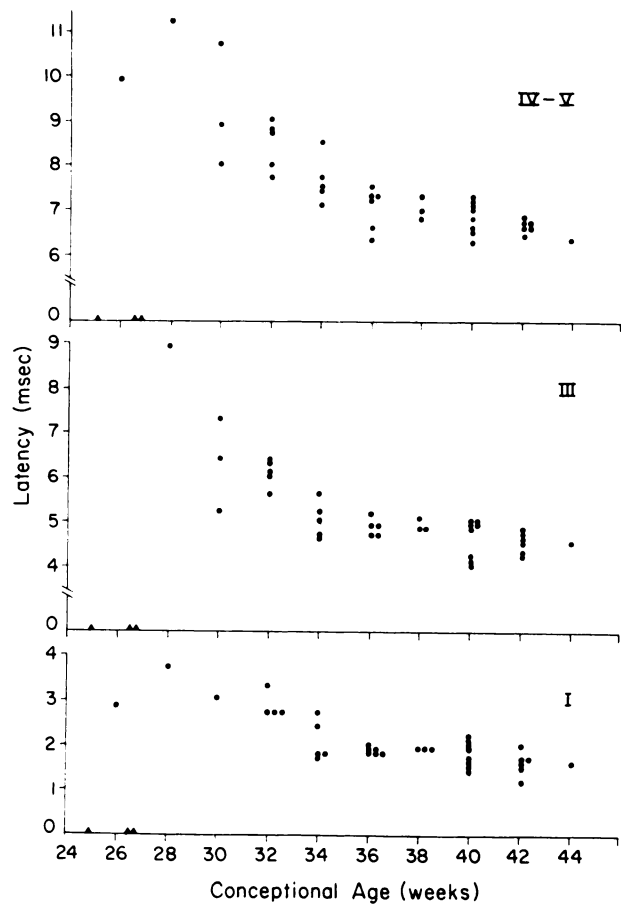


FIG. 2. Auditory brainstem potentials from 42 newborn infants tested within four days of birth to clicks at 65 dB. Latencies of most prominent components, waves I, III, and IV-V complex, are plotted as a function of conceptional age. Triangles refer to tests in which no potential components could be identified.

group (less than 28 weeks of gestation) did not have auditory brainstem potentials to the 65-dB SL click but they subsequently appeared when the infant was retested several weeks after birth. Absence of auditory brainstem potentials at this early age was confirmed in two of the next three infants in this group. The possibility that these results indicated the presence of a critical period for the appearance of auditory function in human infants around the 28th week of gestation was rejected by additional studies. First, an increase in the intensity of the click signal by 10 dB SL to 75 dB SL resulted in the appearance of brainstem potentials in the two infants (Fig. 3). The IV-V complex occurred at latencies of 10.5 and 15 msec, respectively. Second, an examination of slow cortical evoked potentials to the same 65-dB click signals that were ineffective in eliciting brainstem potentials revealed a negative-going deflection between 100 and 200 msec in these infants (Fig. 3). Thus, auditory brainstem poten-

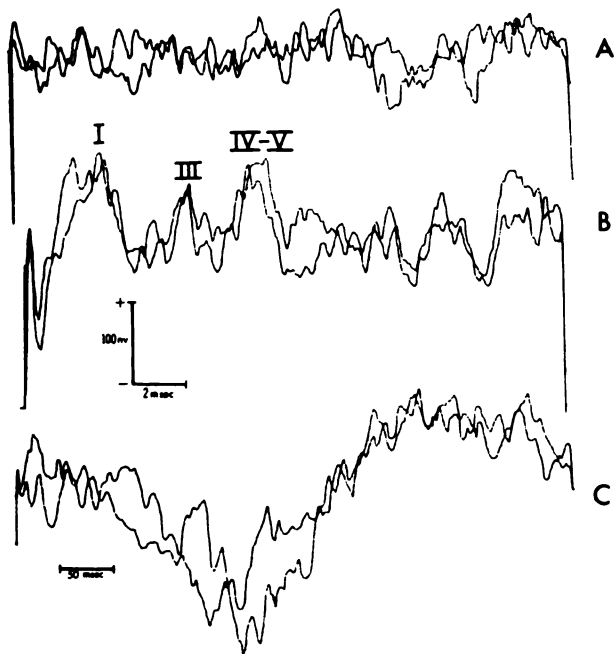


FIG. 3. Effects of stimulus intensity of auditory brainstem potentials prior to 28 weeks' conceptual age. (A) No clear potential components are evoked by 65-dB SL clicks. (B) Potentials evoked by 75-dB SL clicks have definite components. (C) Slow cortical evoked potential to the same 65-dB SL clicks msec in A, showing clear negativity at 150 msec.

tials can be recorded from preterm infants even as young as 25 weeks of gestation, if the signal is sufficiently intense. However, at this early age the click intensity necessary to elicit cortical potentials is lower than that needed for evoking brainstem potentials.

Retesting preterm infants after birth revealed that maturation of auditory function proceeded at about the same rate in both extrauterine and intrauterine environments. Figure 4 contains the mean latency of auditory brainstem potentials to 65-dB SL clicks of the initial test in all of the infants as well as the later tests in some of the infants. The age of retest is designated as the conceptual age for this comparison. For instance, an infant born at 30 weeks' gestation and tested on the fourth week of life is included in the 34- to 35-week conceptual age group and can be compared with the results of the initial test at birth obtained from our preterm population born at the 34th to 35th week of gestation.

The relative contribution of peripheral (cochlea and middle ear) and central auditory pathway processes to the maturational changes can be derived from measures of auditory brainstem potentials. Wave I is assumed to represent activity of the VIII nerve while the other components (II to VII) represent activity arising from auditory pathway structures within the brain-

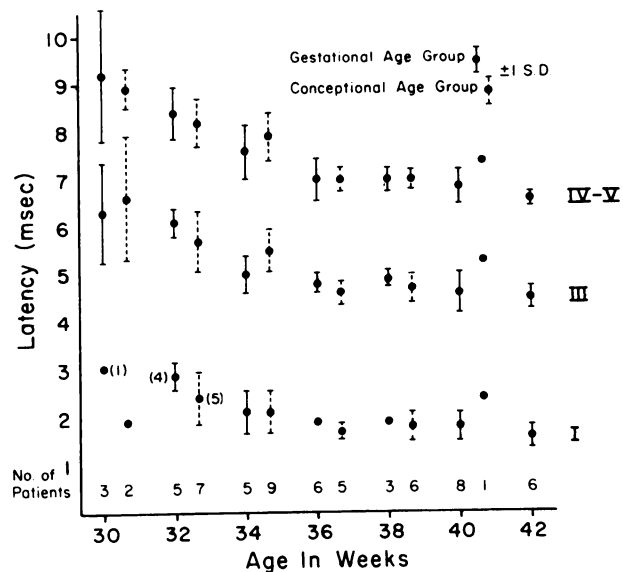


FIG. 4. Comparison of mean latencies at 65 dB SL of major waves between gestational age group tested at birth and conceptual age group tested several weeks after birth at comparable ages. One SD of the mean is indicated by bars; numbers in parentheses indicate number of infants with clearly defined components of total group tested, indicated at lower part of graph.

stem.^{9,11,19} The long latency of wave I in the most preterm infants and the subsequent shortening of latency with increases in gestational age are evidence of changes in peripheral processing during this period. Since cochlear microphonic potentials were not recorded, we are unable to establish the proportion of the latency change of wave I that can be attributed to modifications of middle ear and cochlear receptors during development and the proportion that represents changes in the transduction of receptor potentials into VIII nerve activity.

The time difference between the various components (i.e., waves III minus wave I, wave IV-V minus wave III, or wave IV-V minus wave I) can be used as a measure of maturation of the central auditory pathway. Figure 5 contains a plot of the time difference between waves IV-V and I as a function of gestational age. It is apparent that central conduction time as measured by the difference between the IV-V complex and wave I decreases with gestational age. The scatter in the measures of central conduction times between adjacent waves (waves III and I or waves IV-V and III) is too large to allow an estimate as to the site of maximal change along the central pathway.

Signal intensity had significant effects on the latency of auditory brainstem potentials. A decrease in signal intensity was accompanied by a lengthening of latency of brainstem potentials for

all infants that varied with gestational age. The latency change of the IV-V complex in preterm infants for a 20-dB decrease of signal intensity (65 to 45 dB) varied from 0.2 to 2.0 msec, whereas the same intensity change in adults results in a latency shift of less than 0.9 msec. The effect of signal intensity on the latency of auditory brainstem potentials in preterm infants is most likely due to immaturity of peripheral auditory mechanisms, since central conduction times in the auditory pathway (wave IV-V minus wave I) were unaffected by the same 20-dB change in signal intensity.

The amplitude of the brainstem potentials varies considerably in both adults and newborn infants. However, measurements of the IV-V complex suggest that there is an increase in amplitude during maturation.

DISORDERS OF AUDITORY BRAINSTEM POTENTIALS IN NEWBORN INFANTS

There were several newborn infants whose auditory brainstem potentials deviated significantly from the normal patterns. These infants were neurologically or audiotologically impaired and were not included in the core population.

Their auditory brainstem potentials were either delayed in latency or altered in amplitude. The components' designation as wave I, II, etc., could be uncertain from inspection of the averaged potentials to just one click intensity. Several strategies were used to help delineate the components. First, waves I, II, and III are attenuated as click rates increase above 25/sec, whereas the amplitude of wave V is little affected.¹² Second, all of the components shift in latency as click intensity is reduced, whereas the time difference between each of the components remains unchanged. Thus, there is an orderly relationship between the components independent of click intensity.⁶ Finally, the amplitude of certain of the components (waves I and III) can be profoundly modified by moving the mastoid electrode to the ear contralateral to the click stimulation, whereas the other components are little affected.¹⁴ Nevertheless, the identity of some of the components of an abnormal auditory brainstem potential may still be imprecise even after all of these techniques have been utilized. An example of one such case that has come to postmortem examination was associated with multiple diffuse lesions of the brainstem (case 6 in reference 11).

Results from representative infants in the present study suggest some clinical applications of this procedure in the newborn nursery.

Patient 1. This infant was born at term and was

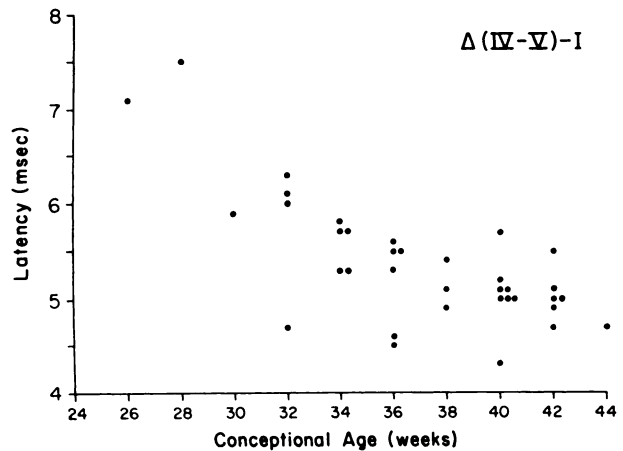


FIG. 5. Time difference in milliseconds between various auditory brainstem potential components for each infant evoked by 65-dB SL click signal.

normal except for a petechial rash localized to the face. She was admitted at 6 weeks with failure to thrive, abnormal head lag, seizures, and lack of clear behavioral response to sounds. Audiometric testing with a 3,000-Hz tone at 100 dB evoked "a marked startle and ocular reflex." In contrast, at 2 months and 4 months of age, auditory brainstem potentials were absent to click signals presented to either ear at 75 dB SL. At 5 months a definitive diagnosis of congenital cytomegalovirus disease was established. The child is now 8 months of age and both the parents and the pediatrician feel the child is deaf. Her psychomotor development is otherwise normal.

Comment. The recording of auditory brainstem potentials provides an objective means for defining auditory function in difficult-to-test newborn and developing infants and particularly infants suspected of hearing loss.

Patient 2. This term infant was noted to bleed from the left external ear canal at birth. Measurement of auditory brainstem potentials revealed a marked disparity between latencies of the IV-V complex evoked by stimulation of the two ears (Fig. 6). The potentials were of normal latency for the infant's age for the right ear but delayed 1.0 msec in the left ear. The potentials were absent in response to 45-dB SL clicks in the affected ear. No other hearing tests were carried out and follow-up has been unsuccessful.

Comment. Auditory brainstem potentials provide sufficiently precise measures to distinguish differences in auditory sensitivity between the ears due to trauma of the cochlea or middle ear at birth.

Patient 3. The patient was a normal full-term infant who was febrile on the second day of life.

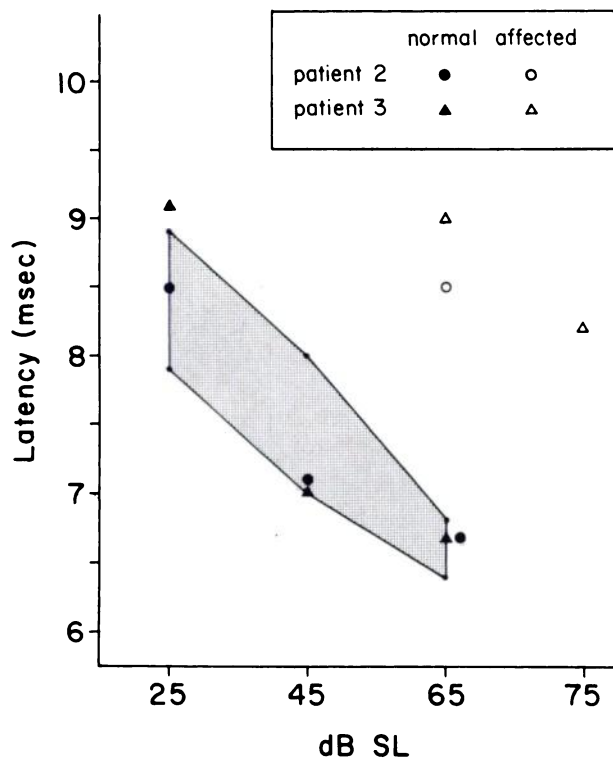


FIG. 6. Latency of wave IV-V complex for two infants with unilateral hearing losses. Shaded region represents range of values for normals in this age group.

The diagnosis of *Escherichia coli* meningitis was established and the infant was treated. The meningitis resolved and auditory brainstem potentials were requested to assess hearing function at 3 weeks of age. The IV-V component was of normal latency when evoked by stimulation of one ear but considerably delayed on stimulation of the opposite ear at both 65 and 75 dB SL (Fig. 6). The latency difference was comparable to a 40-dB hearing loss. Repeated auditory brainstem potential measurement at 11 weeks of age showed marked improvement in the affected ear with the components returning to normal latency. There were no other tests of auditory brainstem performed. It is likely that the child experienced ischemia of one of the cochlea secondary to meningitis with almost complete recovery.

Comment. Auditory brainstem potentials provide early evidence of the presence of a temporary hearing loss (in this patient, a unilateral deficit) that followed meningitis.

Patient 4. The patient had a gestational age of 32 weeks and weighed 1,360 g. She had severe respiratory distress syndrome and required assisted ventilation. There was a sudden deterioration of the clinical condition on the second day of life compatible with an intracranial hemorrhage. Her condition stabilized for a week but

then she gradually developed signs of hydrocephalus. A ventriculostomy was performed but the baby developed sepsis and died on the 22nd day of life.

Auditory brainstem potentials were recorded on the day before death while she required complete respiratory assistance. She had intermittent generalized seizures with hypotonia in the periods between seizures. The pupils were 4 mm in diameter and did not react to light. Auditory brainstem potentials consisted of an abnormally high-amplitude wave I of normal latency and a IV-V complex appearing at a delayed latency of 20 msec. The central conduction time (wave V minus wave I) was considerably prolonged to 17 msec (upper limit of normal in our core population at this age was 6 msec). The brain was covered by a gray exudate, most severe around the brainstem. There was a massive intracerebral hemorrhage, extensive necrosis, and advanced encephalomalacia of both hemispheres and the brainstem. *Citrobacter diversus* was identified on tissue sections and culture.

Comment. Measures of auditory brainstem potentials revealed severe impairment within the CNS manifested by markedly prolonged central conduction times. These potentials provide an objective method for assessing the integrity of the brainstem in neurologically impaired infants.

Patient 5. The infant was born after 38 weeks' gestation. The neurological examination revealed marked hypotonia. All of the components of the auditory brainstem potentials were present, but the central conduction time (wave V minus wave I) was prolonged (7.0 msec). At 19 months of age the brainstem potentials still showed a prolonged central conduction time of 6.6 msec (normal, 4.3 msec) (Fig. 7). The patient was severely retarded and could not sit without support. She could not crawl, had poor coordination of the upper extremities, and made sounds that were not intelligible. Horizontal nystagmus was noted in addition to the severe hypotonia.

Comment. This infant's hypotonia is probably due to a disorder of the CNS. The detection of prolonged central conduction times in the auditory brainstem potentials is evidence of such a brainstem abnormality. The basis for the abnormality has not yet been defined.

DISCUSSION

Far-field recording of auditory brainstem potentials in newborn infants provides information as to the integrity of function of both peripheral and central portions of the auditory pathway. The test requires technical skills for

electrode application and electrophysiological data acquisition, but both are within the area of expertise of the electrodiagnostic technician.

There is evidence that the various components of the brainstem potential represent the sequential activation of neural structures comprising the auditory pathway in its course from the cochlea to the cortex.⁹⁻¹¹ We have demonstrated that measurement of the various components of the auditory brainstem potential can provide the clinician with objective evidence of (1) the status of auditory function in newborn infants in whom the question of altered hearing has been raised and (2) the integrity of brainstem structures in infants with neurological dysfunction.

The results from the present study of auditory brainstem potentials in newborn infants show that reliable components to a 65-dB SL click signal first appear at about the 28th week of gestation. The latencies of the components then decrease as gestation proceeds, with the rate of change being maximal between the 28th and 34th weeks. Auditory maturation is still not complete at birth, as Salamy and McKean²⁰ have shown that brainstem potentials continue to decrease in latency throughout the first year of life and are not at adult values even at that age.

The failure to detect auditory brainstem potentials prior to the 28th week of gestation is not due to an absence of auditory function at this stage of development, as a slight increase in the intensity of the click signal evokes the brainstem potentials. Furthermore, in both the present study and those of Weitzman and Graziani,¹ the initial negative component of the slow cortical evoked potential could also be recorded in preterm infants as early as the 25th week of gestation. The basis for the threshold difference between the brainstem and cortical potentials is unclear but three possibilities will be considered.

First, the relative difference in amplitude between the cortical potentials (1 to 10 μ V) and the far-field brainstem potentials (less than 0.50 μ V) may be a technical factor favoring the detection of the cortical potentials. This possibility is not supported by experimental studies in cats,²¹ in which auditory brainstem potentials abruptly appear postpartum on the 13th day, whereas other authors have reported that cortical evoked potentials to acoustic signals appear earlier, at about the tenth postpartum day.^{22,23}

The discrepancy between the detection of far-field auditory brainstem and cortical potentials may depend on different requirements for neural synchrony of these two events. Auditory brainstem potentials are best elicited by transient

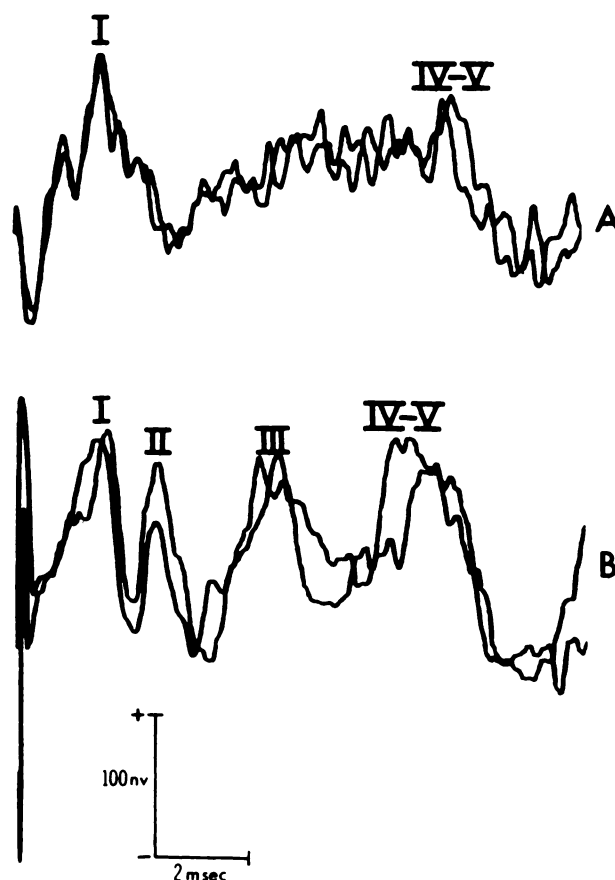


FIG. 7. Patient 5 tested at birth (A) and at 19 months of age (B). Central conduction time (wave IV-V minus wave I) is prolonged on both tests. Value at birth is 7.0 msec (upper limit of normal is 5.5 msec) and 6.6 msec at 19 months of age (upper limit of normal is 4.4 msec).

acoustic signals with rapid rise times. These signals evoke synchronous discharges in VIII nerve fibers followed by the sequential activation of synchronous events at each higher level of the auditory system. The detection of these sequential sets of synchronous neural events probably accounts for the distinct components of the auditory brainstem potentials. Acoustic signals that have gradual rise times are less effective in evoking neural synchrony and do not evoke brainstem potentials. In contrast, temporal dispersion of neuronal events has considerably less effect on cortical evoked potentials. Acoustic signals with rather slow onset (up to 50 msec) can still evoke cortical potentials, and the long time period employed in recording the cortical potentials (256 msec) favors temporal integration.²⁴ An experimental study of neural synchrony during development in cats²⁵ reveals that the immature auditory system is impaired. If there were a corresponding alteration of neural synchrony in the immature infant, auditory brainstem potentials would also be altered.

A change in cochlear responsiveness during maturation could account for the differences in detecting cortical and brainstem potentials. In adults with normal hearing, cortical potentials can be evoked by a wide range of signal frequencies within the acoustic spectrum, whereas auditory brainstem potentials are evoked predominantly by signals containing acoustic energy above 2 kHz.²⁴ The *click* signals used in this and most evoked potential studies will not select for either type of potential, as the energy is widely distributed across the acoustic spectrum though peaks occur at the resonant frequency of the particular transducer employed. There is experimental evidence that low-frequency sensitivity is selectively enhanced during cochlear maturation. Electrophysiological experiments in animals^{26,27} reveal that cochlear function is initially restricted to the low acoustic frequencies and that the extension of sensitivity to both the higher and lower frequency ranges appears only later with maturation. It may be that the difficulty in detecting brainstem potentials before the 28th week of gestation in the infants in this study is due in part to such a delayed maturation of high-frequency sensitivity.

In the present experiment, there is evidence that auditory maturation involves both peripheral and central auditory sites. The peripheral changes are manifest by the decrease in latency of wave I (representing VIII nerve activity) to a constant click intensity as gestation progresses. The central changes are evident by the decrease in conduction time between wave I and other brainstem components. The mechanisms accounting for the change in central conduction time could involve changes in nerve conduction velocity associated with myelination and/or changes in synaptic efficiency at the various nuclei of the auditory pathway. The mechanisms accounting for the peripheral change could include impedance changes in the middle ear, the maturation of high-frequency sensitivity of the cochlea, or changes in transduction between hair cells and the dendrites of VIII nerve.

Auditory brainstem potentials provide an objective means for quantifying auditory development in the human infant with implication of assessing the effects of environmental and congenital factors during the critical period after birth. The technique allows the clinician to obtain a precise measure of the function of one of the sensory pathways independent of factors of arousal or attention. It is possible that similar studies of subcortical visual and somatosensory function will be developed in the near future.

A table containing all clinical data with data of the latencies of the various components at 65, 45, and 25 dB SL is available from the National Auxiliary Publication Service, American Society for Info Science, 1440 Connecticut Avenue, N.W., Washington, D.C. 20036.

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THE CULT OF THE C.P.C.

One of the standard and more popular teaching methods in U.S. medical schools is the clinicopathological conference (C.P.C.). . . . An unwritten rule exists that in any C.P.C. the subject must be over-investigated; the whole exercise is likely to be regarded as a failure if a member of the audience can think of an obscure test that has not been carried out. . . .

Every attempt has been made to pile up a sufficiently large data base from which a tentative diagnosis may be suggested and subsequently confirmed by a biopsy procedure, this being the last resort. The costs of these numerous investigations are often staggering, the more so since it is apparent that they seldom lead to a definitive diagnosis, and for the most part are appreciably less helpful in suggesting one than are the history and physical examination.

Fortunately, some physicians are beginning to question the cult of the C.P.C. One would think that the essence of good medical practice would be to diagnose and treat the patient as expeditiously as possible with a minimum of invasive procedures, but such an approach is unusual in the rarefied atmosphere of a teaching hospital. Nether is the fact that the attending physician, in his obsessive desire to acquire a data base, may well be spending the patient's hard-earned savings, given much thought. President Carter is aiming to limit the increase in the medical-care budget to below 10% for the coming year—it has been rising at roughly 25% a year. To do so, he had better start working on the C.P.C. mentality.

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