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Eighth nerve contributions to cat auditory brainstem responses (ABR)

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There is a temporal correspondence between some of the early components of the auditory brainstem potentials in cat and the negative peak of the triphasic nerve action potential recorded from selected points along the VIII cranial nerve. The intracranial portion of the VIII nerve in cat has a conduction velocity of 10 meters/s. The initial peak of the ABR, P_{1a}, is coincident with the negative portion of the triphasic VIII nerve action potential within the cochlea as recorded from the round window. The next peak (P_{1b}) of the ABR occurs 400 μs later and is coincident with the negative portion of the triphasic VIII nerve action potential recorded from just within the lateral border of the cochlear nucleus. These results are similar to studies of the human ABR that show waves I and II are correlated with activity of the VIII nerve. It is likely that waves P_{1a} and P_{1b} in cat are homologous to waves I and II in human. In cat, these first two peaks of the ABR can be distinguished in vertex to neck recordings but not in vertex to ipsilateral mastoid derivations.

Auditory brainstem responses; Cat; Human; Generators

Introduction

The correspondence between the components of auditory brainstem potentials recorded from the scalp of animals and those recorded from man are uncertain. In man there are up to seven principal vertex positive components beginning at approximately 1.8 ms after a moderately intense stimulus which are labelled sequentially by Roman numerals (I–VII). In most animals the number of vertex positive components can be up to six beginning at about 1.0 ms and labelled by their polarity (P for positive) and their approximate latency in ms or their sequence of occurrence (e.g., $P_{1,2,3}$, etc.). The component in man which is of largest amplitude is wave V (Jewett and Williston, 1971) whereas in experimental animals it is wave P_4 (cat, Achor and Starr, 1980; monkey, Allen and Starr, 1978), P_3 (guinea pig, Wada and Starr, 1983) or P_2 (rat, Plantz et al., 1974). There are significant differences in the size of the brainstem between animals and human. For instance, the intracranial portion of the VIII nerve in man is 5 to 10 mm in length (Møller et al., 1988) whereas in the cat it is only 0.5 mm (results from the present study). Moreover the distance between the cochlear nucleus and the inferior colliculus is about 2 cm longer in humans than in cats (Moore, 1987). These size disparities must contribute to the absolute latency differences of the ABR components and, perhaps, the number and relative amplitudes of the components making up the human and animal ABRs (Møller et al., 1981, 1988; Fullerton et al., 1987).

Several studies have demonstrated that wave I of the human ABR is coincident in time with activity of the VIII nerve in the cochlea (Sohmer and Feinmesser, 1967) whereas wave II is coincident in time with activity of the intracranial portion of the VIII nerve lying between the cochlea and the brainstem (Møller et al., 1981; Spire et al., 1982) or of the VIII nerve within the brainstem (Hashimoto, 1981). Møller and his associates (1986) reported that the intracranial portion of the nerve in monkey is too short relative to its con-

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duction velocity to allow the identification of both distal and proximal VIII nerve activities in the far field. These authors concluded that the decrease in the number of ABR components in animals relative to human is due to the inability to detect activity originating in the proximal intracranial portion of the VIII nerve.

This conclusion may not be entirely accurate since the detection of these early components can be influenced by the recording montage. When the ABR in the cat is recorded between the vertex and a noncephalic reference, the first vertex positive component is followed, in approximately 400 μs, by a second component (Achor and Starr, 1980; Fullerton et al., 1987). If the ABR is derived instead from a differential montage, vertex to the ear being stimulated, the second subcomponent is attenuated or even absent (Achor and Starr, 1980; Fullerton et al., 1987). Three-channel Lissajous' trajectory analysis of the ABR in cat (Martin et al., 1987) reveals three planar segments to be present during the time of these two earliest components (P_{1a} and P_{1b}) compatible with activity originating in several distinct generators. Legatt et al.(1986a) using a vertex to noncephalic derivation in monkey also distinguished that the first vertex positive wave of the monkey is closely followed in 350 μs by a second wave, labelled P_2.

The present experiments were undertaken to further define the relationship between the initial portions of the scalp derived ABR components in the cat and activity recorded from several positions along the VIII nerve: in the cochlea, in the auditory canal close to the porous acousticus, proximally within the cranial cavity at the junction between the nerve and the cochlear nucleus, and even more proximally from the nerve within the cochlear nucleus. The effect of VIII nerve section on the scalp derived ABR components was examined as well. The results contribute to clarifying the generator sites for the early ABR components in cat suggesting homologies between components of the cat and human ABRs.

Methods

Experiments were performed on four cats, one of which was studied twice over an interval of two weeks. The animals were anesthetized with sodium phenobarbital (40 mg/kg) and an intravenous line placed for administering additional anesthetic when needed. Body temperature was monitored by a rectal probe and maintained by a heating pad. In three instances the round window was exposed and a fine wire electrode placed adjacent to the membrane for recording cochlear potentials. In all animals the VIII nerve was exposed by making a craniotomy over the posterior fossa, aspirating the lateral portion of the cerebellum, and exposing the cochlear nucleus and the temporal bone. The VIII cranial nerve is not visible from this approach as the edge of the cochlear nucleus is close to the porous acousticus and covers the nerve. Dissection of this area post mortem revealed the intracranial portion of the VIII cranial nerve (i.e., from the porous acousticus to the lateral edge of the cochlear nucleus) to be approximately 0.5 mm in length with almost all of the nerve actually lying ventral to the cochlear nucleus and under the lip of the pororus acousticus. The VIII nerve then merges with the cochlear nucleus to become part of the brainstem.

Recordings of VIII nerve activity from within the auditory canal was made in five studies with a 31 gauge tungsten wire bent at a right angle 2 mm from the tip and insulated except for its tip. Using an operating microscope for verification, the bent portion of the electrode was introduced into the porous acousticus to lie along the intracanalicular portion of the nerve within the temporal bone. In one animal, the conduction velocity of the intracranial portion of the VIII nerve was defined bilaterally by recording the nerve's activity at three other positions in addition to the intracanalicular site: 1) at the lateral border of the cochlear nucleus where the nerve is free within the intracranial cavity, 2) 0.5 to 0.7 mm medially where the nerve is within the nucleus, and 3) another 2 mm medially, further within the nucleus. These activities were recorded from a glass pipette with a tip diameter of 30 μm that was advanced through the nucleus at an angle of 30 degrees till the triphasic action potential of the VIII nerve was of large amplitude.

In two of the cats the ABR was recorded from a screw electrode placed in the midline 2 cm behind the coronal suture, from another screw electrode 4 cm rostral in the frontal sinus, and

from a needle electrode placed under the skin near the bulla ipsilateral to the ear being stimulated, all referenced against a noncephalic needle electrode placed in the neck. In the other two cats only the vertex-frontal sinus derived ABR was recorded. At the end of two of the studies, the VIII nerve was sectioned and the effects on the scalp derived ABR monitored.

Electrical activity was amplified and filtered between 30 and 3000 Hz. The potentials were digitized at a 20 μs resolution to 1000 click trials presented at a rate of 9.4/s. The resulting averages were plotted and the components' latencies measured on the computer.

The acoustic stimuli were clicks generated by applying 100 μs pulses to Beyer type earphone in the first three cats and Sony type MDR-E225 in one cat. The maximum intensity employed was 80 dB nHL. The effect of stimulus intensity, phase (condensation or rarefaction), rate of presentation, and the presence of accompanying masking noise were examined.

At the end of the experiment the animals were administered a lethal dose of anesthetic, perfused with formalin, and the brain and eighth nerve examined to confirm by visual inspection that the VIII nerve had been transected.

Results

ABR

The ABR of the cat recorded between the vertex or the frontal sinus referenced to the neck consists of up to six major vertex positive and negative components, labelled by their polarity at the vertex and their sequence of occurrence (Fig. 1a). The first component, P_1, can be subdivided into two subcomponents labelled P_{1a} and P_{1b}. These first two components and the next component, P_2, attenuate at low signal intensities. (Fig 1b). P_3 and P_4 are the largest components and persist at low signal intensities. P_5 is broad and of low amplitude. P_6 is of variable occurrence. The amplitude of the recordings from the vertex were approximately twice that derived from the frontal sinus, when both were referenced to the neck (Fig. 1a). Differential recordings between vertex and frontal sinus showed the components to be attenuated in amplitude and to differ slightly in latency (Table I) compared to the neck reference recordings. The ABR recorded between the mastoid and the neck consisted of two major negative components; the initial one peaking shortly after the initial vertex positive deflection (P_{1a}) and the second mastoid negativity peaking at the time of P_2. Differential recordings between vertex and mastoid accentuated the amplitude of components P_{1a} and

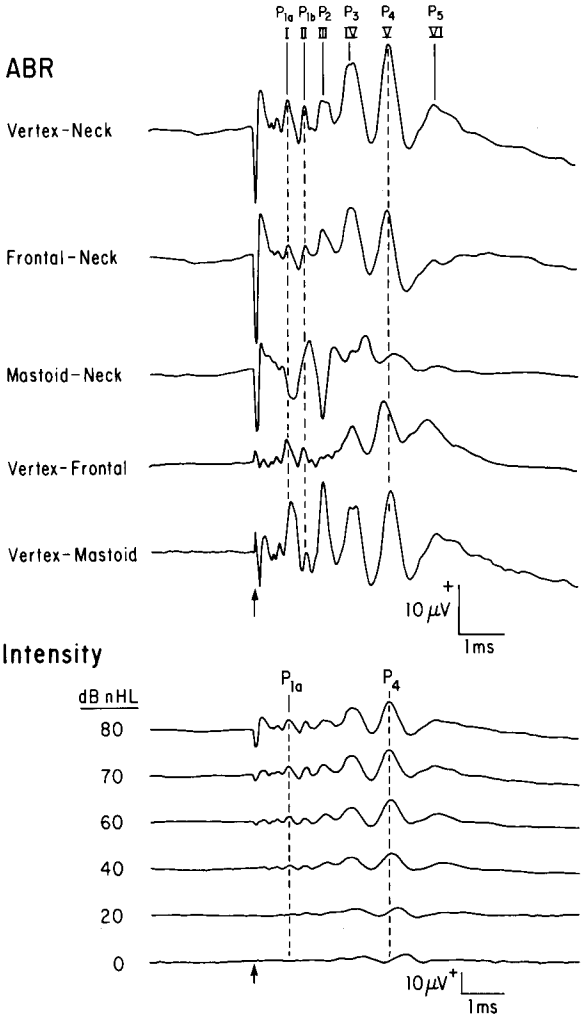

Fig. 1. Auditory brainstem responses (ABR) recorded from several electrodes (vertex, frontal sinus, mastoid ipsilateral to stimulation) referenced to the neck or to one another. Note that the definition of the first two components, P_{1a} and P_{1b}, are affected by the recording montage. In particular, the second component, P_{1b} is obscure relative to the other components in the vertex-mastoid derivation. The lower panel shows the effect of signal intensity on the detectability of the ABR components.

TABLE I

ABR Latencies			
Components	Cz-Neck	Frontal-Neck	Cz-Frontal
		Differences from Cz-Neck (ms)	
P_{1a}	0.90	−0.03	+0.04
P_{1b}	1.30	+0.06	−0.08
P_2	1.68	0.00	+0.08
P_3	2.38	−0.02	+0.06
P_4	3.14	+0.01	+0.06
Ps	4.06	−0.06	0.00

P_2 but markedly attenuated the second subcomponent, P_{1b}.

Cochlear microphonic and neural activities

Round window, intracanalicular VIII nerve activity, and scalp derived ABR recordings contain both microphonic and neural components. The two types of potentials could be distinguished by manipulating stimulus parameters. Microphonic components 1) reversed polarity with click phase, 2) did not change in latency with changes in click intensity, 3) persisted in the presence of masking noise, and 4) did not shift in latency at fast stimulus rates. The neural components, in contrast, 1) did not change in polarity with stimulus phase, 2) did shift in latency with changes in stimulus intensity, 3) did attenuate with masking, and 4) did shift in latency and attenuate at fast stimulus rates. The separation of the potentials into cochlear microphonic and neural components using the first of these methods is shown in Fig. 2a for the round window. The potentials at the round window consisted of low amplitude waves intermixed with at least one clear larger negativity, labelled N_1. By changing the click polarity from condensation to rarefaction and then adding the two responses, the low amplitude components were abolished, identifying them as cochlear microphonics, leaving two clear negative components, N_1 and N_2, the neural components of the VIII nerve (Fig. 2a). This additive procedure slightly prolongs the latency and broadens the N_1 and N_2

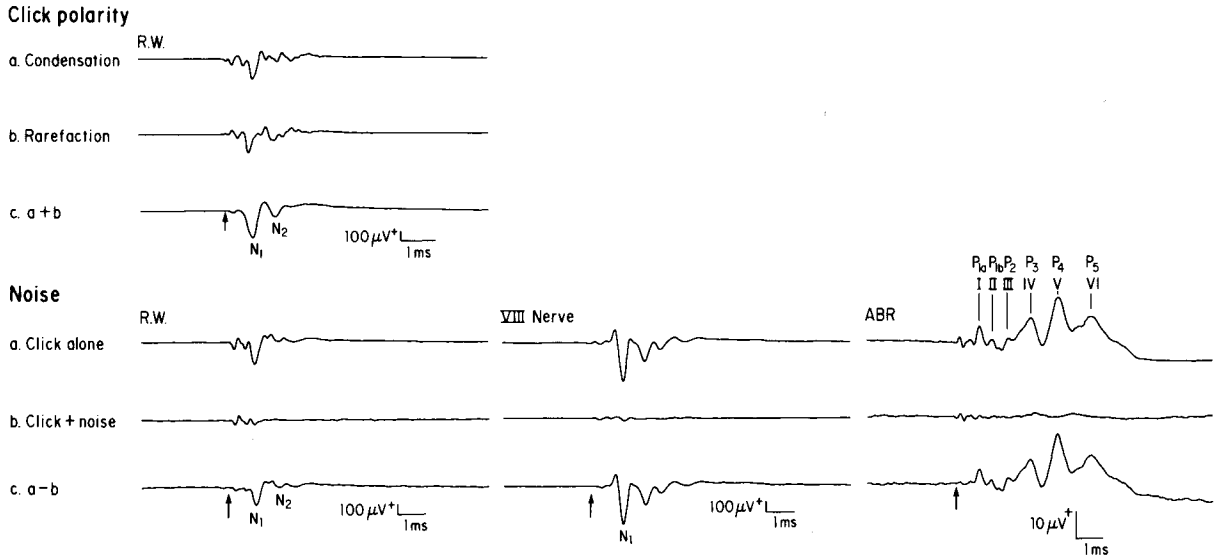

Fig. 2. The effect of click polarity and noise masking on neural and microphonic contributions to click evoked potentials recorded from round window (RW), VIII nerve, and the skull (auditory brainstem responses (ABR)). When click polarity is changed, as for round window recordings, the microphonic potentials are of opposite polarity while the neural components remain of the same polarity but shift slightly in latency. The addition of the potentials evoked to condensation and rarefaction clicks abolishes the microphonics leaving the two neural components, N_1 and N_2 intact. The lower panel of the figure shows that the addition of masking noise to click evoked potentials recorded from round window, VIII nerve, or the skull (ABR) results in the loss of neural components while leaving the microphonic potentials. When the masked potentials are subtracted from the unmasked potentials, the neural components became visible.

component due to the 100 μs latency difference of the neural responses to condensation and rarefaction clicks. The procedure of adding a masking noise to the click could also distinguish between the two types of potentials since the neural responses were abolished without affecting the cochlear microphonics (Fig. 2b). By subtracting the noise masked potentials from the unmasked potential the neural responses, unchanged in latency, were revealed free of cochlear microphonic.

ABR and activity of distal VIII nerve

The activity of the VIII nerve both at the round window and within the auditory canal consisted of two triphasic, positive-negative-positive complexes, N_1 and N_2, with the initial complex being two to three times larger than the second complex. Comparison of the ABR with round window and auditory canal recordings (Fig. 3, Table II) showed the initial vertex positive wave of the ABR (P_{1a}) to be coincident in time with the peak of N_1 from the round window (within 20 μs for the three experiments) and to occur shortly after (between 30 and 80 μs) the peak of the initial positivity recorded from the auditory canal. The second vertex positive deflection of the ABR (P_{1b}) peaked considerably before (240–310 μs) the second neural component (N_2) at the round window and was coincident in time (from 20 to 40 μs) with the peak of the second positivity of the initial nerve action potential recorded from the auditory canal. The negative component of the initial nerve action potential from the auditory canal peaked approximately 175 μs (from 140 to 240 μs in the five studies) after the peak of P_{1a}, at a position fairly intermediate between the peaks of P_{1a} and P_{1b} of the ABR. Thus, at the time of P_{1a} the portion of the VIII nerve within the internal auditory canal shows a positive field and the intracochlear portion of the VIII nerve (sampled at the round window) shows a negative field, findings compatible with depolarization of the VIII nerve within the cochlea at the peak of P_{1a}. The second of the two neural responses recorded from either the round window or the auditory canal was not coincident with the peak of P_{1b}, suggesting that activity of the portion of the VIII nerve extending from the cochlea to the internal auditory canal is not temporally related to the generation of the second component of the ABR in cat.

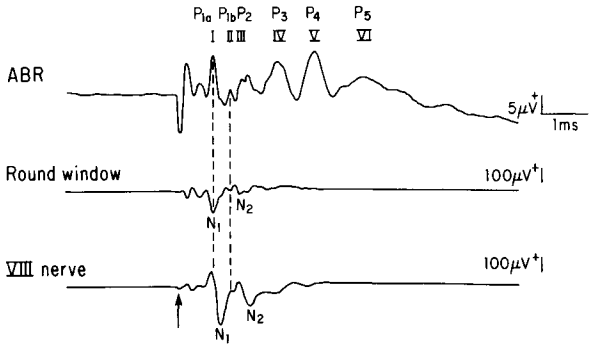

Fig. 3. The relationship between the ABR and initial two negative components of the triphasic action potentials recorded from the cochlea (round window) and from the auditory canal are shown by the dashed lines. P_{1a} peaks coincident with the peak of the N_1 recorded from the round window. P_{1b} peaks at an intermediate position between the N_1 and N_2 components at both the round window and auditory canal recordings. Details are in Table III.

TABLE II

TEMPORAL RELATIONS IN μsec BETWEEN ABR AND VIII NERVE POTENTIALS

	Site of VIII Nerve Recordings				
	Cochlea		Int. Aud. Canal		Cochlear Nuc.
ABR Wave	N_1	N_2	N_1	N_2	N_1
P_{1a}	> 0– 20	< 620–730	< 140–240	< 730–970	< 400–500
P_{1b}	> 460– 510	< 240–310	> 120–240	< 410–500	< 0– 60
P_2	> 820–1 000	> 20–180	> 500–700	> 40–230	> 340–400

N = negative component of triphasic nerve action potential; Cochlea = Round Window Recording; Int. Aud. Canal = 2 mm within Internal auditory canal; Cochlear Nuc. = 0.5–0.7 mm medial to edge of Cochlear nucleus; < means the ABR Wave peaks before VIII nerve negativity; > means the ABR Wave peaks after VIII nerve negativity.

ABR and activity in proximal VIII nerve

Recordings from two sites along the VIII nerve intracranially outside of the brainstem, i.e., from within the internal auditory canal and from its junction with the cochlear nucleus, showed its conduction velocity, in two instances, to be 9.1 and 10 meters/s. The calculation is based on the approximate 200 μs difference in the negative component of the triphasic nerve potential recorded from within the internal auditory canal with that recorded from the nerve at the lateral edge of the cochlear nucleus adjacent to the porous acousticus (Fig. 3), a distance of approximately 2.0 mm. The conduction velocity of the VIII nerve within the brainstem, from the edge of the cochlear nucleus to a position within the cochlear nucleus, 2 mm further medial, was also approximately 10 meters/s. There was a close temporal correspondence (within 60 μs) between the negative peak of the nerve action potential recorded just medial to the junction of the VIII nerve with the cochlear nucleus and the peak of component P_{1b} of the scalp derived ABR (Fig. 4, Table II). Correspondingly the initial positivity of the triphasic action potential recorded from within the cochlear nucleus (position 1, Fig. 4) and the second positivity of the triphasic action potential recorded from the VIII nerve within the auditory canal (position 4, Fig. 4) also occurred close to the peak of P_{1b} recorded from the scalp. Thus, the initial two components of the cat ABR occur coincident in time with activity of different portions of the VIII cranial nerve: the initial peak, P_{1a} occurs when the VIII nerve is active within the cochlea and the second component, P_{1b}, occurs when VIII nerve activity just enters the cochlear nucleus.

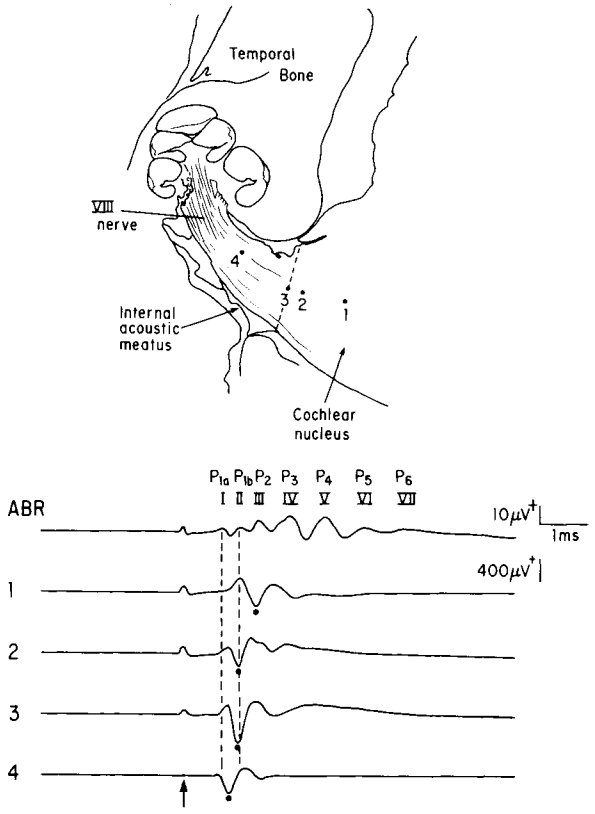

Fig. 4. [The relationship between the initial two components of the ABR, P_{1a} and P_{1b}, and the initial negative component of the triphasic action potential activity recorded at several positions along the intracranial course of the VIII nerve: the internal auditory canal (point 4); 2 mm medial at the edge of the cochlear nucleus (point 3); 0.7 mm further medial just within the cochlear nucleus (point 2); and 2 mm further medial in cochlear nucleus (point 1). The peak of the negative portion of the initial triphasic nerve action potential is indicated by the black dot. Note that the second component, P_{1b}, peaks coincident with negative component of the VIII nerve recorded just within the cochlear nucleus at point 2.

ABR after VIII nerve transection

Sectioning of the VIII nerve at the porous acousticus resulted, in one animal, of almost a complete loss of all ABR components except for the initial vertex positive wave, P_{1a}, which was essentially unchanged (Fig. 5a). In the second animal, low amplitude deflections in the ABR persisted for several ms after P_{1a}. In this latter animal, round window recordings showed an N_1 which was unchanged followed by a series of low amplitude deflections superimposed on a sustained negative shift. It is unlikely that these deflections are microphonics since the technique of processing the evoked potentials using noise masking removed the microphonic in this animal's recordings in the period prior to P_{1a} or N_1. Round window recordings from other animals without VIII nerve section also demonstrated low amplitude deflections superimposed on the N_1 and N_2 responses (Fig. 2). It may be that some of these low amplitude neural events detected from the

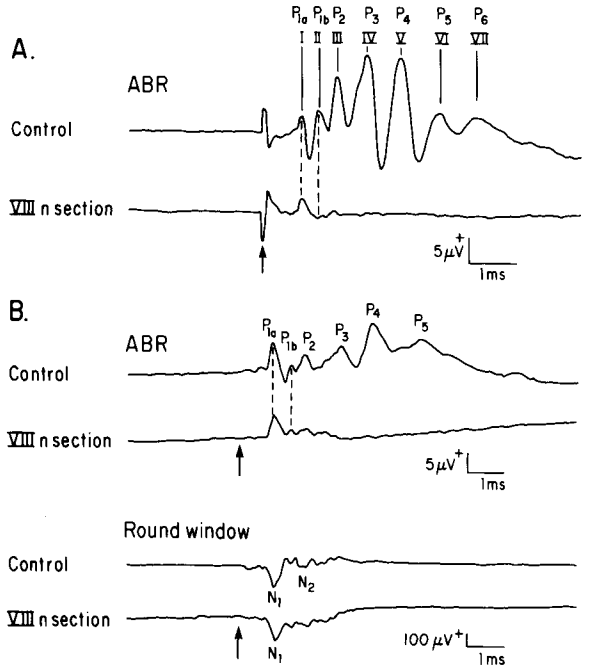

Fig. 5. The effect of sectioning the VIII nerve at the porous acousticus in two cats (A and B) on the ABR and round window recordings. The time scale in the two cats differs. In both cats, the ABR changes dramatically with a loss of all components after the initial P_{1a} peak. The round window potentials which were also recorded in cat B show N_1 to persist following section of the nerve with the addition of a sustained negative shift.

cochlea may normally contribute to the far field ABR.

Discussion

The results of this study show that, in the cat, the initial two vertex positive components (P_{1a} and P_{1b}) occur coincident in time with activity in the distal and central portions of the VIII cranial nerve respectively. The initial component (P_{1a}) occurs coincident with the activity of the VIII nerve within the cochlea, and the second component (P_{1b}) occurs approximately 400 μs later at the time VIII nerve activity has just passed into the cochlear nucleus. The definition of these two initial ABR components is secure when the vertex electrode is recorded against a noncephalic reference whereas in recordings between the vertex and the mastoid ipsilateral to the ear being stimulated, the second component, P_{1b}, is attenuated or even absent as had already been noted (Achor and Starr, 1980; Fullerton et al., 1987; Martin et al., 1987). Thus, studies of the ABR in cat, using vertex to mastoid recordings, would have missed the second of these early ABR components.

The results from the present experiment can be compared to other studies, both in humans and animals, in which electrical events were recorded directly from the cochlea, VIII nerve, and cochlear nucleus while monitoring the scalp derived ABR. Møller in humans (Møller et al., 1981; Møller et al., 1988) showed that wave I of the ABR recorded between the vertex and a noncephalic site occurred up to 1 ms prior to the negative portion of VIII nerve activity recorded intracranially from the porous acousticus. Wave II occurred slightly after the negativity of the VIII nerve action potential recorded from the proximal portion of the VIII cranial nerve adjacent to the cochlear nucleus. These authors concluded that both wave I and II were generated by activity of the VIII cranial nerve with wave I coincident in time with activity in the cochlea and wave II coincident in time with activity close to where the nerve 'exits the brainstem'. Hashimoto and his colleagues (1981) concluded from recordings in man of both brainstem and intracranial VIII nerve potentials that wave II appeared to be generated within the brainstem.

In monkey, Legatt and his coworkers (1986b) showed that activity of the intracochlear portion of the VIII nerve was coincident in time with one of the early peaks of the ABR (component 1_b of their nomenclature) whereas activity of the VIII nerve centrally within the cochlear nucleus was coincident with the peak of the next component (wave 2) that occurred 350 μs after peak 1_b. Moushegian and his colleagues (1962) showed, in cat, that the VIII nerve is active within the cochlear nucleus approximately 300 μs after the peak of the N_1 recorded from the round window (see Fig. 1 of their paper). Møller (1982) in rat, showed the time separation between VIII nerve activity at the round window (N_1) and from within the cochlear nucleus was also 300 μs. Thus, in monkey and cat there is evidence that activity restricted to the VIII cranial nerve within the cochlea and within the cochlear nucleus in the brainstem are separated in time by only 300–400 μs. Moreover, from the present study in cat, the initial two components of the scalp

derived ABR (P_{1a} and P_{1b}) are coincident in time with activity of the VIII nerve in the cochlea and in the cochlear nucleus.

The relatively long interval of 1.1 ms separating waves I and II in man can be attributed, in part, to the time required for the nerve impulse to traverse the 5–10 mm intracranial portion of the VIII nerve extending from the cochlea to the brainstem. In cat the intracranial portion of the VIII nerve is approximately 10-fold shorter being only 0.5 mm in length. However, this disparity in the dimension of the VIII nerve between human and cat is too great to account for the relatively modest difference (approximately 3-fold) in the interval between the initial two vertex positive ABR peaks in the cat, 400 μs, compared to 1100 μs in man. Even considering the differences in conduction velocities of the VIII nerve in the two species (10 meters/s in cat; 13–25 meters/s in humans (Hashimoto et al., 1981; Møller et al., 1981; Spire et al., 1982)) will not suffice to account for the relatively small difference in the interval between the first two peaks in the two species. We believe that it is the disparity in length of the myelinated portion of the VIII cranial nerve within the temporal bone that is the major factor contributing to the difference in the interval between the first two vertex positive peaks in man and cat. In cat we have measured the length of the VIII nerve within the temporal bone to be 2.5 mm whereas in humans it is more than 10-fold longer at 25 mm (Lang, 1981). Thus, the approximate length of the entire VIII nerve extending from within the cochlea to the cochlear nucleus is 30 mm in man and only 3 mm in cat. Using the measures of conduction velocity of 20 meters/s for humans, the time necessary to travel the length of the VIII nerve is approximately 1.5 ms, a value close to the interval of 1.1 ms found between waves I and II. Using the values obtained in the cat of 3 mm for the length of the VIII nerve and a conduction velocity of 10 meters/s, the time required to travel the VIII nerve from the cochlea to the cochlear nucleus is 0.3 ms, a value likewise close to the observed interval of 0.4 ms between P_{1a} and P_{1b}.

The localization of the generator sites for these first two components to particular segments of the VIII cranial nerve was complemented by the results of sectioning the VIII nerve intracranially. The first vertex positive wave, P_{1a}, was unchanged in amplitude whereas the subsequent components beginning with the second wave, P_{1b}, were markedly attenuated or lost. The initial wave, P_{1a} must be generated by activity in the intracochlear portion of the VIII cranial nerve whereas the second wave is generated by VIII nerve activity within the cranial cavity. Three-channel Lissajous' trajectory analysis of the ABR in cat following intracranial section of the VIII nerve (Gardi et al., 1987) also showed that of the three planar segments that comprised the period of the initial vertex positive complex (P_{1a} and P_{1b}) preoperatively, only the initial planar segment remained though prolonged in duration. Results from another set of experiments (Zaaroor and Starr, 1990b, submitted) in which the neurons of the cochlear nucleus were almost totally destroyed by an excitatory amino acid while sparing VIII nerve fibers are in agreement. In some of these animals the initial two components, P_{1a} and P_{1b}, of the

TABLE III

HUMAN AND CAT ABR

Human	Generator	Cat	Generator
I (1)	VIII n in cochlea	P_{1a} (6)	VIII n in cochlea
II (2)	VIII n close to CN	P_{1b} (6,7)	VIII n in CN
III (3)	CN	P_2 (8)	CN
IV (4,5)	bilateral brainstem	P_3 (8)	contra brainstem
V (4,5)	bilateral brainstem	P_4 (8)	bilateral brainstem
VI (5)	bilateral midbrain	P5 (8)	bilateral midbrain

1) Sohmer and Feinmesser 1967; 2) Møller et al., 1981; 3) Scherg and Cramon 1985; 4) Starr and Hamilton 1976; 5) Stockard and Sharbrough 1977; 6) Achor and Starr 1980; 7) present study; 8) Buchwald and Huang 1975.

ABR were unchanged whereas subsequent waves (P_2 and P_3) were abolished.

We suggest that there are sufficient anatomical and functional similarities between many of the peaks of the ABR in cat and man to allow the Roman numeral labelling scheme initially proposed by Jewett and Willison (1971) for human ABR to be applied to cat ABR (Fig. 1, Table III).

Our experiments can not distinguish which component of the VIII nerve within the cochlea is the generator of wave P_{1a} (I) i.e., the ganglion cells, their dendrites, or their axons. Moreover, these experiments also can not distinguish which portion of the VIII cranial nerve intracranially in cochlear nucleus is the generator(s) for wave P_{1b} (i.e., wave II). There are several sites along a nerve trunk which, when active, generate potentials detectable in the far field (Duepree and Jewett, 1988). For instance, a stationary positive potential can be detected 1) when the nerve impulse traverses impedance boundaries as might occur when the VIII nerve exits the cochlea or enters the cochlear nucleus 2) at sites where nerve activity changes direction as would occur when the VIII nerve turns dorsally after entering the cochlear nucleus; or 3) at the end of a nerve as would occur where the VIII nerve terminates on the neurons of the cochlear nucleus. In our study there was a temporal correspondence between the initial two far field ABR components and the negative peak of the VIII nerve triphasic action potential either within the cochlea or within the brainstem but not with activity of the segment of nerve lying free in the cranial cavity, findings that would be compatible with all of these alternatives.

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