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AUDITORY BRAIN STEM POTENTIALS IN MONKEY (*M. MULATTA*) AND MAN *

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In humans, the potentials originating from the brain stem portion of the auditory pathway can be detected with scalp electrodes using far-field averaging techniques and consist of a series of 7 waves during the initial 10 msec following click stimulation (Jewett and Williston 1971). Auditory brain stem potentials have been measured in cats and rats to define the neural generators of the various potential components (Plantz et al. 1974), but the results are still inconclusive (Jewett 1970; Buchwald and Huang 1975).

The study in this report was undertaken to define the scalp distribution of auditory brain stem potentials in the rhesus monkey. Topographical analysis was utilized to define changes in both latency and amplitude of the various components across the scalp to provide clues as to the location and participation of brain stem structures giving rise to the surface potentials. The rhesus monkey was selected because of its close relation to man on the phylogenetic scale and the hope that the results might approximate those derived from humans.

Detailed depth recording and the effects of lesions on the surface recorded potentials in monkeys could serve to help define the generators of these potentials in humans.

Method

Adult female rhesus monkeys weighing 5.7–7.3 kg were anesthetized with pento-

barbital 30 mg/kg and studied in a sound-attenuating chamber. Supplemental doses of pentobarbital were administered if the animals moved. Body temperature was monitored and maintained at 38°C.

The scalp distribution of the far-field auditory brain stem potentials to monaural clicks was tested in three monkeys randomly selected from our colony using the electrode configuration shown in Fig. 1A. Electrodes were placed both in the midline and parasagittally using the following criteria. The vertex was the point midway between the inion and 1 cm above the nasion. Two additional electrodes were placed along the midsagittal line one-quarter of the distance between the inion and the point above the nasion. Parasagittal placements were located midway between the vertex and the tragus of the ear. Two additional electrodes were placed equidistant anterior and posterior to this lead along a line connecting the midparasagittal points with the temporal margin of the palpebral fissure. The location of the anterior points was 1.5 cm from the superior orbital ridge. In addition, electrodes were placed on each ear lobe. An electrode on the sternum served as the 'indifferent' lead. Positivity at the scalp produced an upward deflection in our recording procedure. The electrodes were 6 mm diameter metal disc electrodes attached to the skin by collodion. The interelectrode resistance was less than 5 k Ω . Electrical activity was amplified by a factor of 100,000, filtered between 100 c/sec and 3 kc/sec (3 dB down point) and led to a summing computer. The computer was triggered to sample at

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25 kc/sec for 10.24 msec from onset of a click signal. The click signals were produced by a 0.1 msec positive pulse applied to a TDH-39 earphone positioned 1 cm from the external auditory meatus. The intensity of the clicks was adjusted to be 60 dB SL referenced to six normal hearing adults tested while wearing the TDH-39 earphones.

Electrocardiographic potentials recorded with our large interelectrode distances were not included in the averaging process by restricting the presentation of click signals to the period between qRs components of the electrocardiogram. This was accomplished by using a delaying circuit triggered by the qRs complex which in turn activated a signal generator to produce the positive clicks. The delay was adjusted to prevent any blocking of the amplifier by the large electrocardiographic potential.

Potentials from each of the two scalp sites were simultaneously averaged. Activity from the vertex electrode was always recorded in one channel while the activity from one of the other 12 sites was recorded in the second channel. This technique provides a constant site, i.e. the vertex against which amplitude changes from other scalp locations could be compared.

Auditory brain stem potentials evoked by duplicate sets of 1024 clicks presented at 10/sec rate were summed and the results plotted for measurement of latency and amplitude of the various components. Latency measures were taken at both the peak of each positive deflection as well as the following trough.

The effects of click intensity and rate on the latency and amplitude of auditory brain stem potentials were tested in three monkeys used in the mapping study and in two other adult rhesus monkeys. The electrode configuration employed was the vertex referenced to the earlobe ipsilateral to the side receiving the click signal. Clicks were presented at 10 dB intensity intervals and at a rate of 10/sec. Stimulus rates of 10, 30, 50 and 100/sec at 3 different levels (30, 50,

70 dB HL) were also tested. Two sets of 1024 click trials were summed and the latencies and amplitudes of the various peaks and troughs determined.

Results

(1) Distribution

Auditory brain stem potentials could be recorded from all sites over the scalp and neck. In contrast the averaged activity detected from the precordial electrode referenced to the tail was flat after 1024 click trials allowing the sternum to be used as the 'indifferent site' in the mapping study.

Wave I was negative over the entire scalp with a peak latency that depended upon scalp location (Fig. 1B). The earliest latencies occurred on the side ipsilateral to the stimulation and were delayed up to 0.2 msec in scalp locations at the vertex or contralaterally to the stimulated ear. Wave II was positive and had a distinct morphology as a function of recording site. The wave consists of two components; the leading peak (IIa) was accentuated at sites ipsilateral to stimulation and the trailing peak (IIb) was accentuated at contralateral scalp locations. The two peaks would often fuse into a single broad wave II at the vertex. The latency of the two components of wave II varied up to 0.36 msec as a function of recording site (Table I). Wave IIa occurred earliest ipsilateral to stimulus site whereas wave IIb occurred earliest contralateral to stimulus site.

Wave III was positive and consisted of two distinct components. The initial component (IIIa) was of smaller amplitude than the second component (IIIb). The two peaks were often fused into a single broad component at the vertex. The initial component (IIIa) was always more prominent on the side ipsilateral to the stimulus and occurred at a latency approximately 0.1 msec earlier than at the vertex. This early component was identifiable at the contralateral mastoid region on only 2

Fig. 1. A: disc electrodes were applied at the locations indicated. Not shown are symmetrically placed electrodes at locations corresponding to 8, 10 and 12. Electrode 13 is at the inion and 3 at the vertex. The wave form at the bottom of the figure is a representative average recorded between the vertex and the sternum. Click intensity was 60 dB HL (human) applied to the right ear. B: representative wave forms from one animal from all recording sites are placed at the appropriate site on the dorsal view of the monkey scalp.

of 6 mapping runs. Wave IIIb appeared earliest on the side contralateral to the stimulus and could be delayed by as much as 0.2 msec in records derived from the vertex.

Wave IV was easily identified at the vertex in all animals. The morphology of the wave varied at the various locations on the scalp from a quite prominent and distinct peak to fusion with wave IIIb or V. Identification was thus difficult at some recording sites which may account for the variability in latency between monkeys (Table I). Wave IV had the shortest latency at the earlobes and the region of the mastoids occurring 0.2 msec before its appearance at the vertex.

Wave V was the most difficult component to identify because of its small amplitude and fusion with wave IV. The latency of wave V was earliest in the posterior region of the head bilaterally.

To recapitulate, for each monkey the peak latency of the various components varied over the scalp from 0.2 msec for wave I up to 1.15 msec for wave V. Moreover, waves II and III consist of two separate components and the recording site is the significant factor determining which component of each of the waves was accentuated. The variability of the latency measure defined from a single electrode site, i.e. the vertex at different times throughout the mapping study, varied from 0.08 msec for waves I–IV to 0.50 msec for wave V (Table I). The variability at this one site is less than one-half that seen across the scalp, indicating that only a portion of the latency shift across the scalp can be attributed to response variability over time.

The amplitude of the brain stem potentials also varied as a function of recording site. Fig. 2 is the mean of the data obtained from three monkeys using 6 distinct maps to monaural clicks with the vertex lead as a reference (value 100).

Wave I was largest at scalp locations ipsilateral and anterior to the ear receiving the acoustic signal.

Wave IIa was also of maximum amplitude anteriorly and ipsilateral to the stimulus. The

TABLE I

Maximum range of latencies (msec) of auditory brain stem potentials in the monkey at different recording sites across the scalp (top half of table) and from a single recording site, the vertex (bottom half of table).

Monkey	Wave						
	I	IIa	IIb	IIIa	IIIb	IV	V
1	0.20	0.22	0.36	0.25	0.58	0.64	0.68
3	0.16	0.20	0.25	0.15	0.17	0.18	0.28
4	0.20	0.25	0.35	0.50	0.40	0.50	1.15
1	0.08	0.12	0.12	0.16	0.28	0.32	0.36
3	0.08	0.08	0.08	0.08	0.08	0.16	0.16
4	0.08	0.08	0.08	0.08	0.12	0.32	0.50

Fig. 2. Amplitude distribution of each component of the auditory brain stem potentials to 60 dB monaural clicks. The amplitudes of the potentials recorded from the vertex were the controls (i.e., 100%) and all other potential amplitudes were adjusted accordingly. The arrow signifies the stimulus site.

amplitude of both of these components decreased at the posterior and contralateral recording points. Wave IIb was of maximal amplitude over the anterior region. Wave IIIa was difficult to scale and the plot represents only two runs, one from each of two animals. The distribution suggests that the amplitude is largest in the midline and anterior loci. Wave IIIb had maximal amplitude anteriorly and ipsilaterally.

Wave IV was of greatest amplitude in the anterior and frontal sites.

Finally, wave V was maximal in the contralateral anterior and midline sites. In summary, the amplitude distribution of the various potential components is different over the scalp. Waves I and II are largest ipsilateral to the site of stimulation. Wave IIIa is more diffusely distributed whereas the later waves (IIIb–V) have their largest amplitude in the midline and contralateral sites.

(2) Effects of stimulus parameters on brain stem potentials

A vertex to ipsilateral ear recording configuration was utilized to test the effects of changes in signal parameters on the brain stem potentials. This recording array is identical to the recording convention commonly used in human studies in which relative positivity at

Fig. 3. Comparison between auditory brain stem potentials recorded between vertex to sternum (upper trace) and vertex to earlobe (lower trace). Monaural clicks were presented to the same ear.

the vertex electrode produces an upward deflection.

The morphology of the brain stem potentials obtained with the vertex-earlobe configuration is different from our previous descriptions because each of the electrodes is 'active' (Fig. 3). Wave I is positive in this configuration. The second major positive wave

Fig. 4. Representative series of wave forms demonstrating the effect of intensity on the various components of the auditory brain stem potentials.

corresponds to wave IIb and the third positive wave corresponds to IIIb. Waves IIa and IIIa detected with a non-cephalic reference are represented by negative components immediately preceding waves II and III in the vertex to earlobe derivation.

In Fig. 4 the effect of intensity on the latency of the brain stem potentials is seen in a representative monkey. The vertex positive components waves I, IIb, and IV can be readily identified at all intensities.

Latencies of the vertex positive components for the five monkeys as a function of click intensity are seen in Fig. 5. The earlier waves show a much tighter grouping of data points than waves IV and V.

The amplitude of the components varied

Fig. 5. This scattergram represents the latencies of auditory brain stem potentials from five monkeys to different intensities of click stimuli. Recordings were made between the vertex and the ipsilateral ear. Note the tighter distribution of points in the earlier components.

TABLE II

Latency (msec, top half) and amplitudes (μV , bottom half) of auditory brain stem potentials in monkey (n = 5).

Wave	dB HL re. humans									
	10	20	30	40	50	60	70	80		
I	2.48 $\pm$ 0.28	2.17 $\pm$ 0.20	1.97 $\pm$ 0.14	1.80 $\pm$ 0.11	1.63 $\pm$ 0.098	1.51 $\pm$ 0.09	1.38 $\pm$ 0.09	1.28 $\pm$ 0.07		
II	3.49 $\pm$ 0.27	3.12 $\pm$ 0.198	2.94 $\pm$ 0.16	2.77 $\pm$ 0.15	2.59 $\pm$ 0.14	2.48 $\pm$ 0.13	2.37 $\pm$ 0.15	2.29 $\pm$ 0.13		
III	4.24 *	4.15 $\pm$ 0.04	3.87 $\pm$ 0.16	3.77 $\pm$ 0.20	3.57 $\pm$ 0.17	3.47 $\pm$ 0.19	3.32 $\pm$ 0.22	3.21 $\pm$ 0.17		
IV	5.23 $\pm$ 0.33	4.95 $\pm$ 0.28	4.82 $\pm$ 0.27	4.68 $\pm$ 0.23	4.53 $\pm$ 0.25	4.41 $\pm$ 0.26	4.32 $\pm$ 0.296	4.17 $\pm$ 0.22		
V	**	7.02 $\pm$ 0.86	6.63 $\pm$ 0.60	6.63 $\pm$ 0.61	6.00 $\pm$ 0.62	5.93 $\pm$ 0.60	5.82 $\pm$ 0.59	5.73 $\pm$ 0.59		
I	0.11 $\pm$ 0.05	0.22 $\pm$ 0.08	0.46 $\pm$ 0.13	0.77 $\pm$ 0.18	1.04 $\pm$ 0.28	1.31 $\pm$ 0.33	1.61 $\pm$ 0.54	1.76 $\pm$ 0.61		
IIb	0.61 $\pm$ 0.28	1.01 $\pm$ 0.40	1.60 $\pm$ 0.62	2.02 $\pm$ 0.61	2.43 $\pm$ 0.68	2.58 $\pm$ 0.63	2.66 $\pm$ 0.71	2.72 $\pm$ 0.72		
IIIb	***	0.32 $\pm$ 0.38	0.40 $\pm$ 0.32	0.44 $\pm$ 0.29	0.58 $\pm$ 0.36	0.65 $\pm$ 0.35	0.90 $\pm$ 0.50	0.98 $\pm$ 0.50		
IV	0.53 $\pm$ 0.20	0.81 $\pm$ 0.32	1.06 $\pm$ 0.59	1.16 $\pm$ 0.52	1.36 $\pm$ 0.59	1.53 $\pm$ 0.59	1.83 $\pm$ 0.82	2.19 $\pm$ 0.99		
V	***	0.14 $\pm$ 0.04	0.16 $\pm$ 0.07	0.21 $\pm$ 0.09	0.33 $\pm$ 0.13	0.38 $\pm$ 0.13	0.45 $\pm$ 0.22	0.68 $\pm$ 0.24		

* Only one subject.

** Could not be defined.

*** Insufficient data.

TABLE III

Latency (msec) as a function of click rate and intensity.

Wave	10/sec			30/sec			50/sec			100/sec		
	30 dB *	50 dB	70 dB	30 dB	50 dB	70 dB	30 dB	50 dB	70 dB	30 dB	50 dB	70 dB
I	1.97 $\pm$ 0.14	1.63 $\pm$ 0.098	1.38 $\pm$ 0.09	1.99 $\pm$ 0.17	1.68 $\pm$ 0.15	1.41 $\pm$ 0.13	2.04 $\pm$ 0.19	1.68 $\pm$ 0.15	1.46 $\pm$ 0.14	2.00 $\pm$ 0.18	1.76 $\pm$ 0.18	1.50 $\pm$ 0.12
IIb	2.94 $\pm$ 0.16	2.59 $\pm$ 0.14	2.37 $\pm$ 0.15	3.02 $\pm$ 0.23	2.68 $\pm$ 0.23	2.48 $\pm$ 0.22	3.05 $\pm$ 0.26	2.74 $\pm$ 0.24	2.53 $\pm$ 0.20	3.14 $\pm$ 0.28	2.83 $\pm$ 0.24	2.62 $\pm$ 0.26
IIIb	3.87 $\pm$ 0.16	3.57 $\pm$ 0.17	3.32 $\pm$ 0.22	4.03 $\pm$ 0.25	3.65 $\pm$ 0.22	3.46 $\pm$ 0.22	3.97 $\pm$ 0.20	3.68 $\pm$ 0.26	3.52 $\pm$ 0.23	4.08 $\pm$ 0.23	3.80 $\pm$ 0.24	3.55 $\pm$ 0.24
IV	4.82 $\pm$ 0.27	4.53 $\pm$ 0.25	4.32 $\pm$ 0.296	4.97 $\pm$ 0.42	4.69 $\pm$ 0.42	4.48 $\pm$ 0.38	5.04 $\pm$ 0.45	4.80 $\pm$ 0.46	4.63 $\pm$ 0.45	5.15 $\pm$ 0.46	4.98 $\pm$ 0.46	4.81 $\pm$ 0.54
V	6.63 $\pm$ 0.60	6.00 $\pm$ 0.62	5.73 $\pm$ 0.59	7.20 $\pm$ 0.51	6.06 $\pm$ 0.60	6.14 $\pm$ 0.92	7.47 $\pm$ 0.83	6.40 $\pm$ 0.96	5.76 $\pm$ 0.80	8.20 $\pm$ 0.11	6.52 **	6.33 $\pm$ 1.28

* dB re. human subjects.

** only one subject.

TABLE IV

Central conduction times (msec).

Stimulus intensity (dB)	Monkey	Waves				
		I-II	II-III	III-IV	IV-V	I-IV
80	Mean	1.02	0.91	0.96	1.55	2.89
	Range	0.94-1.12	0.72-1.15	0.64-1.11	1.23-1.81	2.72-3.14
70	Mean	0.99	0.97	0.99	1.53	2.95
	Range	0.92-1.10	0.73-1.18	0.64-1.11	0.92-1.98	2.74-3.14
60	Mean	0.97	1.02	0.93	1.58	2.92
	Range	0.94-1.06	0.75-1.22	0.61-1.21	1.04-2.06	2.77-3.18
50	Mean	0.96	0.99	0.94	1.53	2.89
	Range	0.92-1.05	0.75-1.24	0.56-1.14	1.02-2.0	2.72-3.11
40	Mean	0.97	1.00	0.91	1.91	2.88
	Range	0.93-1.04	0.77-1.26	0.56-1.16	1.41-2.34	2.73-3.03

considerably from session to session independent of changes in latency. Table II shows that waves II and IV had the largest variation about the mean. The etiology of these variations is currently not known. It may be related to the level of anesthesia of the animals.

For all of the waves there is a systematic shift in latency with change in intensity. At the higher intensities there is approximately 0.1 msec shift for each 10 dB decrease in intensity, while at the lower intensities the shift is 0.2 msec. The transition takes place at approximately 40-50 dB HL (re: Human).

Additional waves beyond wave V were occasionally noted. These waves occurred at higher intensities and tended to disappear at or near 60 dB HL. Their latencies to an 80 dB click were 6.2 msec and 7.2 msec.

In Table III the latency of each wave is plotted as a function of click rate at 3 selected intensities. Increasing the stimulus rate from 10/sec to 100/sec produced a latency shift of 0.1 msec in wave IIb and 0.4 msec in wave IV. Wave I did not shift significantly with a change in click rate. These changes in latency were significant beyond the 0.05 level using analysis of variance. Because of variation between the animals, mean latencies do not show significance. However, the amount of shift for each animal from the reference fre-

quency of 10/sec was clearly significant.

There was a significant interaction between stimulus rate and signal intensity utilizing the analysis of variance that could be attributed to wave IIb. This finding is contrary to the finding in humans which showed that the latency shift associated with increasing stimulus rate was independent of intensity (Don et al. 1977).

The difference in peak latency between wave V and wave I has been utilized as a measure of central conduction time in the human with application to definition of disorders of the central auditory pathway in neurologic diagnosis (Starr and Achior 1975; Stockard et al. 1976; Starr 1977). The latency separation between the various components in the monkey is included in Table IV using the vertex to ipsilateral ear configuration. Wave V was not included because its irregular appearance makes the determination of peak latency difficult.

Discussion

Jewett (1970) demonstrated that the far-field auditory brain stem potentials are not due to muscle potentials, cochlear microphonics or electrical artifacts. Several techniques have been utilized to characterize the neural generators of the potential components.

These include (1) latency correlation between the far-field potentials and recordings from auditory structures themselves (Jewett 1970; Jewett and Williston 1971; Lev and Sohmer 1972), (2) topographic analysis of scalp distribution of potentials (Martin and Coats 1973; Picton et al. 1974; Plantz et al. 1974), and (3) lesion studies correlating changes in the far-field potentials with disruption of particular portions of the auditory pathway (Buchwald and Huang 1975). All of these studies suggest that wave I is coincident with VIII nerve activity, wave II originates from the region of the cochlear nucleus and waves III and IV originate in structures between the superior olive and the inferior colliculus.

In the present mapping study a fairly consistent series of waves was obtained from all electrode sites. There were latency shifts as one moved about the scalp as well as marked variation in amplitude.

We found as did Plantz et al. (1974), in the rat, that there are no indifferent points on the head or neck of the monkey. Unlike the cat or human (Terkildsen et al. 1974) the mid or lateral neck proved to be active when referenced against the chest or arm. The sternum proved to be an adequate indifferent.

In the monkey, wave I is clearly of highest amplitude and has the shortest latency on the side ipsilateral to the stimulus which would support the concept of its origin from VIII nerve activity.

Wave II is a complex wave having two distinct components. There is accentuation of each component (IIa or IIb) depending upon the position of the recording electrode. A single wave II is obtained on recording from the ipsilateral earlobe which has a latency that corresponds to the vertex wave IIa. In contrast a single wave II is obtained on recording from the contralateral earlobe which has a peak latency that corresponds to the vertex wave IIb. These data suggest that wave II detected at the vertex is comprised of activity of two generators located on the two sides of the brain stem.

Wave III is also composed of two distinct components with separate latency and amplitude distributions suggesting its origin in bilateral brain stem generators (Plantz et al. 1974).

Wave IV has its greatest amplitude in the midline and occurs simultaneously at lateral recording sites suggesting origins from a midline structure or synchronous activity from bilateral structures.

Wave V occurs earliest contralateral to the stimulus and has its greatest magnitude in the same distribution suggesting its origin from a contralateral structure.

In summary, waves I and V appear to have a single lateralized generator, waves II and III appear to take origin from bilateral structures, and wave IV appears to originate from a midline source.

The latter portion of this study was devoted to a systematic evaluation of the effect of frequency and intensity on the latency of the brain stem potentials recorded in a vertex to ear configuration. The shifts in latency were subtle compared to changes seen in man at comparable stimulus parameters (Starr and Achor 1975; Thornton and Coleman 1975; Don et al. 1977).

There are several explanations that could account for the variations in amplitude between recording sessions. Electrode location has been shown to be critical with regards to amplitude in the mapping study, but the electrodes were applied according to strict criteria in each instance by the same experimenter. Stimulus intensity has also been shown to affect amplitude as well as latency measurements. Since there were no significant changes in latency and the placement of the earphone 1 cm from the external meatus presented no problem it is unlikely that this can explain the observed changes. Level of consciousness has been shown by several investigators (Picton and Hillyard 1974; Starr and Achor 1975) to have no effect on the latency of the early components. However, level of anesthesia would seem a reasonable explanation for the changes

in amplitude. In the course of the experiment it was noted that supplemental injection of i.v. barbiturate was associated with the reduction in amplitude of all waves. Jewett and Romano (1972) noted similar changes in amplitude in both rats and cats relative to depth of anesthesia. Finally, a yet unidentified dynamic process or activity of efferent systems may exert an influence on the auditory system in the brain stem altering amplitude.

Correspondences among auditory brain stem potentials in monkey, man, cat and rat

In all mammals tested there are certain fundamental properties of the far-field brain stem potentials that are relatively constant: (1) a series of four or more waves are seen in the first 10 msec following acoustic stimulation, (2) the vertex serves as the optimal recording site for the later components, (3) predictable shifts in latency occur with changes in stimulus parameters, (4) usually one wave is of highest amplitude and can be followed at all intensities above threshold, (5) the pinna is active and causes alteration in the wave form when it is used as an 'indifferent' recording site, (6) the latency of the various waves is unaffected by sleep or anesthesia, (7) several of the waves probably represent activity from more than one generator, (8) waves beyond wave I originate from within the brain stem, and (9) functional or anatomical disruption of the brain stem pathways produces alterations in the potentials recorded at the scalp.

In Fig. 6 and Table V, latencies and representative wave forms for man, monkey, cat and rat are given. In all species the referential lead was placed at an 'indifferent' site. For purposes of comparison in this study the point at which wave I first goes in a negative direction was taken as the starting point for latency measurements. This choice is based on knowledge of the complex behavior of wave I when recorded in various configurations utilizing cephalic and non-cephalic references. In man, in the vertex to neck con-

Fig. 6. Representative tracings from four different mammals recorded in the same laboratory. The recording configuration was between the vertex and an 'indifferent' non-cephalic reference. Click intensity was 60 dB HL re. human. The tracings of the evoked potentials were adjusted so that wave I (arrow) occurred at the same place. Thus the beginning of the tracings do not represent the stimulus onset. See Table V for analysis of the data.

TABLE V

Auditory brain stem potentials in several mammals. The actual latencies in msec of the various components are compared to their predicted appearance.

		Waves			
		I	II	III	IV
Mean	Observed	1.65	2.93	4.1	5.25
Monkey	Observed	1.40	2.26	3.25	4.15
	Predicted		2.37	3.26	4.14
	Error		0.11	0.01	0.01
Cat	Observed	1.43	2.40	3.15	4.03
	Predicted		2.35	3.16	3.99
	Error		0.05	0.01	0.04
Rat	Observed	1.28	2.10	2.7	3.70
	Predicted		2.14	2.88	3.65
	Error		0.04	0.18	0.05

figuration, wave I is negative going. In the rat wave I is initially positive in locations above the horizontal plane defined by the tip of the nose to the tail. Below this plane wave I is negative. We chose the former tracing for comparison. In the cat wave I is similar in shape to the human. In the monkey wave I has an initial slight positive component before becoming negative (see Fig. 3). The striking similarity between the cat and monkey is apparent. Waves II and III are complex in both animals. In all animals wave IV is prominent and has a simple configuration. The later waves are irregular in appearance and highly variable depending upon intensity and the specific animal tested.

There is 75–100-fold difference in brain stem size and weight between man and rat (Blinkov and Glezer 1968). However, if their differences are adjusted by some simple assumptions several predictions can be made.

Using man as a reference, the latency of waves II, III and IV can be predicted with great accuracy in subhuman species. Since wave I is assumed to be due to VIII nerve activity this serves as a starting point for comparison. The total transmission time between waves I and IV (central conduction time) in man was taken as a standard. The latency difference between each wave beyond wave I (II–I, III–II, IV–III) was divided by the central conduction time to provide a percentage of the total central conduction time. Next the central conduction time was determined by direct measurement in the other three species. Applying the percentage values obtained for the difference between waves in man to the central conduction time of the animal yielded a series of numbers that represented the predicted latency (or separation) of the various wave forms. For example, if the total central conduction time in the cat was 2.6 msec and the percent difference between waves I and II of the total central conduction time in man was 35.5% then the predicted occurrence of wave II in the cat would be obtained by taking 35.5% of 2.6 msec and adding it to the measured value of wave I.

This yielded a value of 2.35 msec which was comparable to the actual measured time of 2.4 msec. The predicted value of the other waves is determined by a similar calculation. The largest error in predicted latency was 0.18 msec. This would suggest that the components of the auditory brain stem potentials in most mammals may originate from similar space generators, though the exact identity of the generators may not be identical.

Careful cross-species studies will undoubtedly increase the predictability and understanding of the components of the evoked potential. The information obtained could be applied to a better understanding of the human auditory system in normal and pathological states.

Summary

Auditory brain stem potentials were mapped over the head of the rhesus monkey following monaural clicks. Latency and amplitude were dependent upon the recording site. No electrically indifferent point could be found on the head or neck. The results are interpreted as showing that waves I and V originate from single generators, whereas waves II, III and IV originate from bilateral structures. Latency varied in a linear fashion with changes in intensity and click rate. In contrast there were variations in amplitude of the potentials independent of stimulus change. A comparison was made of auditory brain stem potentials in man, monkey, cat and rat to demonstrate the similarity of these responses across species.

Résumé

Potentiels auditifs du tronc cérébral chez le singe (macaque) et l'homme

La topographie des potentiels auditifs obtenus par clics monoraux du tronc cérébral a été établie chez le singe rhésus. Il apparaît que la latence et l'amplitude de ces potentiels

dépendent du point d'enregistrement. Aucun point électriquement indifférent n'a pu être trouvé au niveau de la tête ou du cou. Ces résultats sont interprétés comme la preuve que les ondes 1 et 5 proviennent de générateurs isolés alors que les ondes 2, 3 et 4 proviennent de structures bilatérales. La latence varie de façon linéaire avec les modifications d'intensité et de vitesse des clics. Par contre, certaines variations d'amplitude des potentiels sont indépendantes des modifications du stimulus. Les potentiels auditifs du tronc cérébral de l'homme, du singe, du chat et du rat ont été comparés, pour en montrer la similarité d'une espèce à l'autre.

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