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GENERATION OF AUDITORY BRAIN STEM RESPONSES (ABRs). II. EFFECTS OF SURGICAL SECTION OF THE TRAPEZOID BODY ON THE ABR IN GUINEA PIGS AND CAT [1]

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The ABR is thought to derive from activity in the brain stem auditory pathway. However, there is uncertainty as to the precise relationship between individual components and particular auditory pathway structures except for P1 (wave I in human) which is the far-field reflection of VIIIth nerve activity. Several alternatives have been suggested: (1) the ABR components reflect the sequential activation of auditory nuclear groups (Jewett 1970) and more particularly the activity of special groups of neurons having a precise latency of discharge following auditory stimulation (Buchwald and Huang 1975); (2) the ABR components reflect activity in fiber tracts connecting the nuclear groups (Starr and Squires 1982). With both of these suggestions it is unclear whether just one structure (be it a nuclear group or fiber tract) contributes primarily to each component (Jewett and Williston 1971) or whether each structure contributes to several components (Achor and Starr 1980).

In a companion paper (Wada and Starr 1983a) we demonstrated that injection of a local anesthetic into the trapezoid body of guinea pigs and cat had profound effects on the latency and amplitude of ABR components beginning with N2. We could not distinguish whether the trapezoid body itself generates these components or whether it serves merely to connect the various brain stem nuclear groups generating the ABR. In the present experiments, in which the trapezoid body was surgically sectioned in the midline, the relationship between the trapezoid body and the ABR components is clarified in more detail.

Methods

The experimental procedures followed were the same as those described in the initial paper (Wada and Starr 1983a) with the following exceptions.

Subjects

The effects of surgical section of the trapezoid body were observed in 16 guinea pigs and 5 cats.

Recordings

The ABR was recorded as in the initial paper. In addition, in 7 guinea pigs and 2 cats direct recordings from the trapezoid body were made with bipolar metal electrodes (100 μm tip diameter with a horizontal tip separation of 0.5 mm). In two of the guinea pigs the electrodes were inserted into the trapezoid body. In the other animals the recordings were made from the ventral surface of the pons with the electrodes arranged in parallel with the direction of the fibers.

In all referential recordings positivity was displayed upward. An amplification factor of 100,000 was used for the ABR and 10,000 for the direct recordings.

Experimental lesions and histology

The trapezoid body was exposed from a ventral approach as described in the initial paper. Surgical section of the trapezoid body was made with No.

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11 scalpel near the midline. If there were little or no changes in the ABR, additional incisions were made at different levels on the opposite side of the brain stem. Table I details the number of cuts employed in each animal. In two animals, the surgical section was accompanied by brain stem edema and/or vasospasm of the basilar artery. The ABR in these animals decreased in amplitude and/or had a delay in latency of all components. These results were not included in the analysis.

The ABR was recorded in 3 epochs: prior to exposing the brain stem; after the brain stem was exposed, but prior to sectioning of the trapezoid body; and after the section. There were no significant changes in the ABR between the two epochs prior to sectioning the trapezoid body. After sectioning, recordings of the ABR were continued for 3–18 h. During the recording period rectal temperature was maintained at 36–38°C by means of a circulating water pad. In the initial 4 animals studied, body temperature was not monitored and their latency measures were not included in the analysis.

The animals were perfused through the heart with normal saline followed by a 10% buffered formaline solution. The entire brain stem was removed, blocked and stored in 10% buffered formaline for at least 1 week prior to processing. Reconstruction of the extent of the lesion was made from 60 μm frozen sections stained with cresyl violet. Every section around the trapezoid body was examined to define both the rostrocaudal and dorsal extents of the lesion.

Results

Guinea pig

Fig. 1 contains the ABR evoked by monaural stimulation both prior to (solid line) and after (dotted line) surgical section of the trapezoid body in one of the guinea pigs (GPI) in whom almost the entire structure (92%) was sectioned. The lesion, also shown in Fig. 1, was close to the midline extending from the lower pons to the upper medulla and penetrating the brain stem to a depth

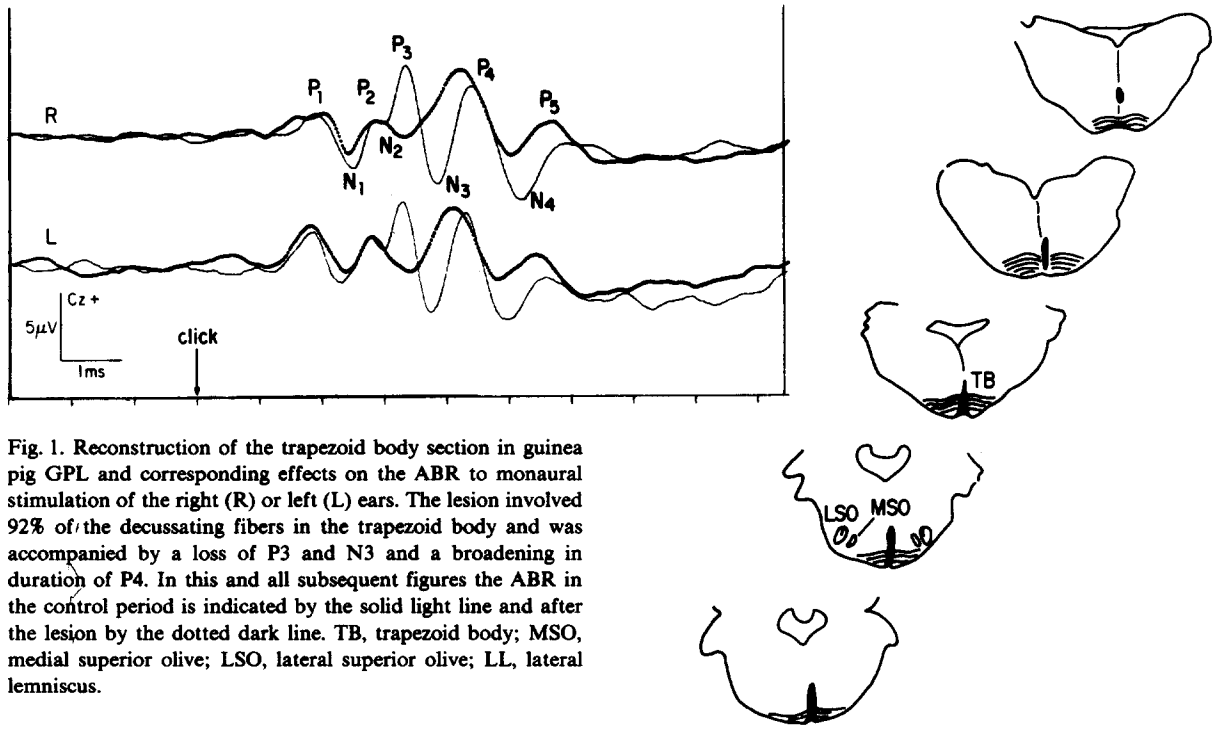


Fig. 1. Reconstruction of the trapezoid body section in guinea pig GPL and corresponding effects on the ABR to monaural stimulation of the right (R) or left (L) ears. The lesion involved 92% of the decussating fibers in the trapezoid body and was accompanied by a loss of P3 and N3 and a broadening in duration of P4. In this and all subsequent figures the ABR in the control period is indicated by the solid light line and after the lesion by the dotted dark line. TB, trapezoid body; MSO, medial superior olive; LSO, lateral superior olive; LL, lateral lemniscus.

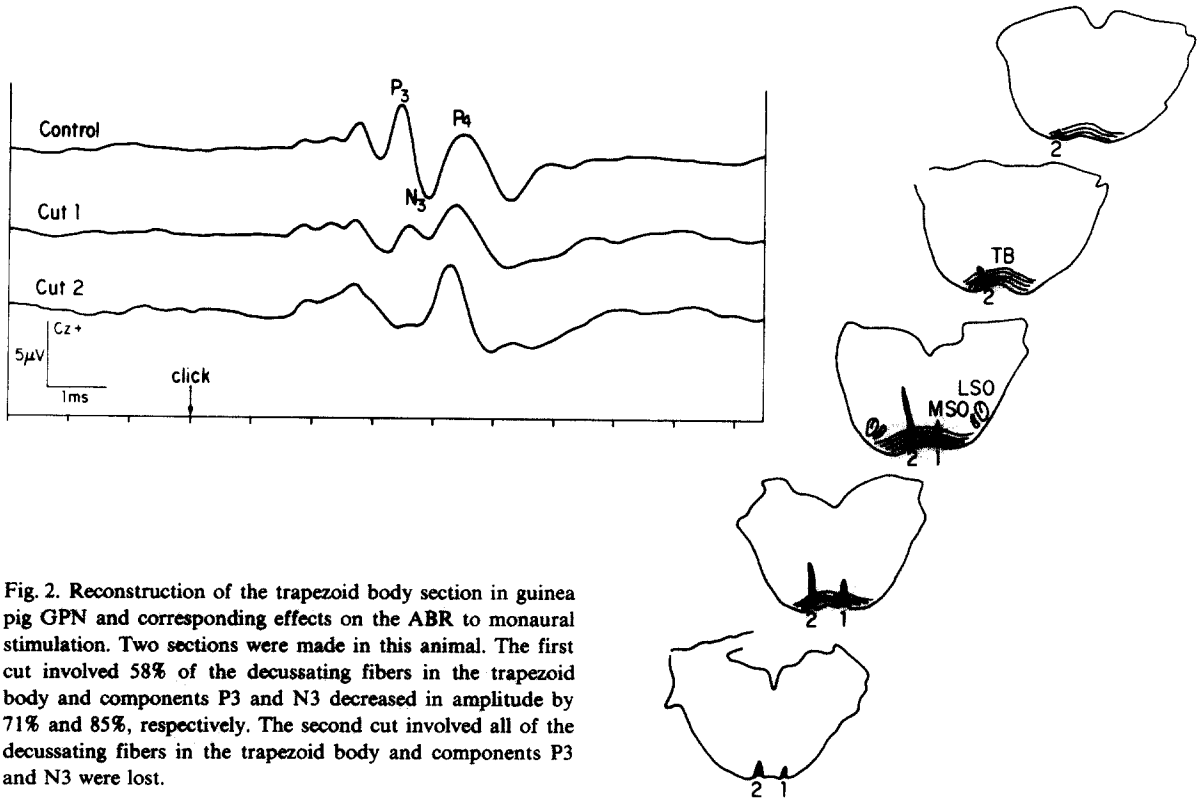


Fig. 2. Reconstruction of the trapezoid body section in guinea pig GPN and corresponding effects on the ABR to monaural stimulation. Two sections were made in this animal. The first cut involved 58% of the decussating fibers in the trapezoid body and components P3 and N3 decreased in amplitude by 71% and 85%, respectively. The second cut involved all of the decussating fibers in the trapezoid body and components P3 and N3 were lost.

of 2 mm. Dramatic changes in the ABR occur from the stimulation of either ear beginning with component N2 which is delayed, P3 and N3 are lost, P4 broadens in duration and shifts to an earlier latency, N4 is attenuated and shifts to an earlier latency, and P5 increases in amplitude and occurs earlier.

With partial lesions near the midline of the trapezoid body the changes were less drastic but equivalent from stimulation of either ear. In guinea pig GPN, for instance, two cuts were made. The first cut (Fig. 2) was localized to the right of the midline, extending from the lower pons to upper medulla, and entering into the brain stem 1.5 mm to affect 58% of the decussating fibers in the trapezoid body. To right-sided stimuli components N2 and P3 were delayed, P3, N3 and N4 decreased in amplitude by 71%, 85% and 36%, respectively, P4 increased slightly in amplitude (34%) and decreased slightly in latency (0.15 msec). A second cut was then performed to the left of the midline that involved all of the decussating fibers in the trapezoid body. After the second cut P3 and N3 disappeared and P4 shifted to an even shorter latency.

The effects of a small lesion of the trapezoid body near the midline can be seen in guinea pig GPL (Fig. 3). The initial cut involved 8% of the decussating fibers in the rostral portion. To right-sided stimulation, only components P3 and N3 were affected, decreasing by 26% and 18%, respectively. There were no significant latency changes. A second cut was then made 2 mm to the left of the midline involving 48% of the trapezoid body fibers. Component P3 and N3 further decreased in amplitude by approximately 50%. If the surgical section did not involve any of the decussating fibers in the trapezoid body, there were no changes in the ABR.

Tables I and II detail the effects of surgery on the trapezoid body on the amplitude (Table I) and latency (Table II) of the ABR to monaural stimu-

TABLE I

Effects of surgical section of the trapezoid body on ABR in guinea pig.

Guinea pig	Cut	% of TB cut	Ear	Threshold (dB SPL)	% change in amplitude as an (f) of control P3 or wave III												
					I	P1	N1	II	P2	N2	III	P3	N3	IV	P4	N4	P5
GPA	3	100	R	54				−17		−20	−100	−100	−100	+21	+40	−15	**
GPF	3	100	R	44			+11		−19	+20″	−100	−100	−100	−45	−19	−52	**
GPN	2	100	R	54				+11		+20	−100	−100	−100	+28	+72	−18	−10
			L	54			−10	+39	+42	+37	−100	−100	−100		+37	−36	+11
GPI	1	92	R	54			−14	+11		+18	−100	−100	−100	−25	+23	−63	+31
			L	54			−12	+20		+33″	−100	−100	−100	−31	+12	−62	+34
GPM	1	92	R	54				+13	+20		−100	−100	−100	−17		−27	**
			L	54				+21	+19	+15	−100	−100	−100		+33	−37	**
GI	1	92	R	64				−15	−13	−12	−90	−86	−61″	−11	+18	−36	**
			L	64					−11	+13	−100	−100	−100		+40	−33	**
GB	2	90	R	54							−94	−96	−46″	−51	−34	−43	+12
			L	44				−22	−41		−90	−101″	−67	−34	−13	−50	+21
GB	1	86	R	54				−36	−36	−18	−78	−93	−25″	−18	−27		−100
			L	44						+10	−84	−88	−69	−19	−15	−20	−100
GH	1	74	R	54				−12	−21		−88	−75	−80″				−23
			L	54				−15	−29		−75	−69	−81″	−25	+13	−64	
GPD	1	69	R	64							−87	−71	−69″	+42	+65		**
			L	44							−81	−70	−48″		+14		+14
GPK	1	60	R	64				+11		+13	−76	−49	−41	+14	+27	−10	**
			L	54				−13	+10	−36	−72	−76	−67		−10		**
GPN	1	58	R	54					−10	+17	−82	−71	−85″		+34	−36	−100
			L	54							−63	−72	−57	−13	−10	−16	
GL	1	56	R	54							−86	−75	−68				−28
			L	54					−22	+16	−78	−63	−89	−33	−38	−26	**
GPB	2	50	R	54				+23		+25	−90	−62	−37″	−28	+25	−56	**
GPH	1	48	R	64							−79	−46	−65	+49	+75		**
			L	44					−21	+31″	−67	−65	−33				−22
GPL	2	48	R	54					−16		−61	−53	−50″	−15		−25	**
			L	54					−10		−78	−64	−60	+16	+26		**
GPC	1	43	R	54							−87	−62	−42″	+16	+19		
			L	44							−85	−58	−33″	+14	+26	−15	**
GPG	1	38	R	44							−72	−46	−66	−10	+41	−57	**
			L	44							−64	−60	−64		+13	−21	**
GPL	1	8	R	54					−11		−27	−26	−18			−12	**
			L	54				−14	−13	−13	−16	−10	−16	−14		−22	**

TB = trapezoid body. −100 = component lost. ** component not identified prior to section. ″ polarity change. + = increase in amplitude as a % of control P3 (or III) amplitude. − = decrease in amplitude as a % of control P3 (or III) amplitude.

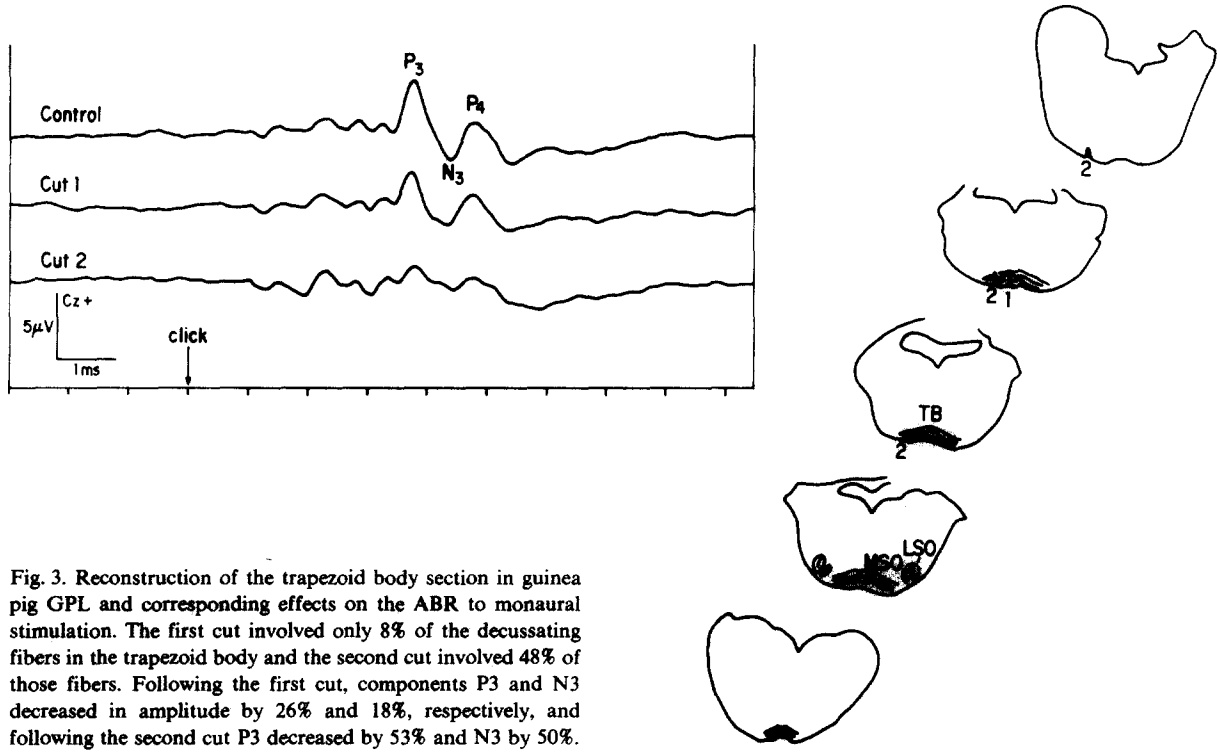


Fig. 3. Reconstruction of the trapezoid body section in guinea pig GPL and corresponding effects on the ABR to monaural stimulation. The first cut involved only 8% of the decussating fibers in the trapezoid body and the second cut involved 48% of those fibers. Following the first cut, components P3 and N3 decreased in amplitude by 26% and 18%, respectively, and following the second cut P3 decreased by 53% and N3 by 50%.

lation in guinea pig. Both tables have been arranged so that the largest cuts are at the top. There were no effects on P1 or N1; P2 decreased slightly in amplitude in 14 instances, increased slightly in 4 instances, and was unaffected in 17 instances. The amplitude of N2 was also affected inconsistently whereas its latency was usually prolonged (16 instances). P3 and N3 were consistently and markedly attenuated with N3 occasionally becoming positive in polarity. P4 usually increased slightly in amplitude (20 instances) and occurred at a shortened latency; N4 was slightly attenuated and shortened in latency; P5, which was identified in only 50% of the animals in the control period, was inconsistently affected by the lesion.

When the extent of attenuation of P3 is graphed as a function of the magnitude of trapezoid section, the relationship is clearly linear with a slope close to unity (Fig. 4A). That is, the amplitude of P3 remaining after the section reflects the proportion of intact trapezoid fibers remaining intact. The relationship of N3 to the extent of trapezoid body section could not be assessed as directly, since this component's polarity changed in a number of animals making the measure of amplitude invalid. Instead, we chose to measure the amplitude of wave III (peak of P3 to the trough of N3) which would be unaffected by N3 polarity changes and related wave III amplitude to the extent of trapezoid section (Fig. 4B). Since P3 amplitude is linearly related to the extent of trapezoid body section, any deviation from this linearity, using measures of wave III, must be attributable to changes in N3. It is evident in Fig. 4B that wave III has a poorer linear relationship to the extent of trapezoid body section than does P3 reflecting the relative lack of correlation between N3 amplitude and trapezoid fiber loss.

Binaural interaction in the ABR was examined in 11 guinea pigs before and after section of trapezoid body. Binaural interaction represents the non-linear interaction of simultaneous monaural stimulation on the ABR compared to the sum of the separate monaurally evoked potentials. The effects are seen only in components P4 and N4. In the example shown in Fig. 5A in which the

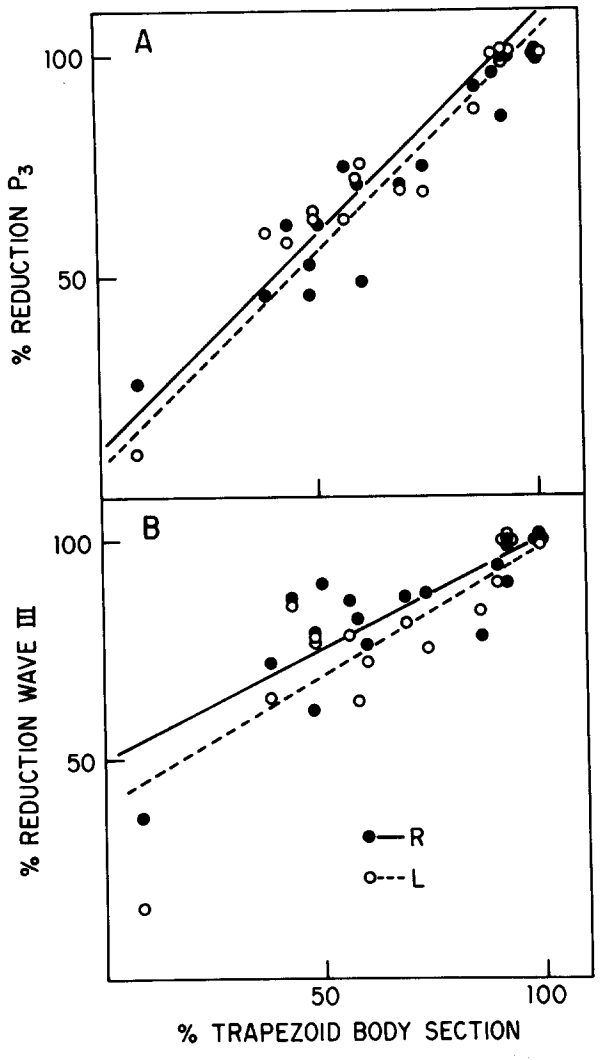


Fig. 4. A: the relationship between the percent of decussating fibers sectioned in the trapezoid body and the percent of decrement of P3 amplitude. The relationship is clearly linear. Left: $r = 0.97$, $r^2 = 0.94$ ($P < 0.001$); right: $r = 0.95$, $r^2 = 0.90$ ($P < 0.001$); left vs. right; $t = 1$ (P = W.S.). B: the relationship between the percent of decussating fibers sectioned in the trapezoid body and the percent of decrement of wave III amplitude. The relationship is also linear but is less than for P3 and the slope is no longer close to unity. Left: $r = 0.87$, $r^2 = 0.77$ ($P < 0.001$); right; $r = 0.85$, $r^2 = 0.72$ ($P < 0.0001$); left vs. right; $t < 1$ (P = W.S.)

trapezoid body has been sectioned by approximately 85%, binaural interaction was reduced approximately 65%. When changes in the extent of binaural interaction are expressed as functions of the extent of trapezoid body section (Fig. 5B), the relationship is linear. That is, the magnitude of binaural interaction persisting after surgery reflects the numbers of crossing fibers remaining intact in the trapezoid body.

We attempted to resolve whether the components of the ABR affected by the surgical section were generated directly by trapezoid body activity by recording from the surface of the trapezoid body before and after such section. The results from one of these studies are shown in Fig. 6. The recording electrodes were placed at various points on the surface of the trapezoid body parallel to the fibers. Prior to section large amplitude potentials were evoked from all recording sites by monaural stimulation. Following partial section of the trapezoid body in its caudal half, the potentials evoked from the fibers contralateral to the stimulated ear in the caudal portion were attenuated whereas the remainder of the trapezoid body potentials were either unchanged or even of larger amplitude.

Cat

The effects of surgical section of the trapezoid body on the ABR evoked by monaural stimulation in cat (Fig. 7) were quite similar to the findings in guinea pig with exceptions that P2 could be significantly attenuated in cat (> 50%) (Tables III and IV) and not in guinea pig, and component P4 was reduced in amplitude but unchanged in latency in cat whereas this component was unchanged in amplitude and shifted in latency in guinea pig. In both species binaural stimulation evoked a larger P4 after surgical section of the trapezoid body than in the control period.

Discussion

The results of this study show that complete surgical section of the trapezoid body in the midline of guinea pig affects many components of the ABR: N2 is delayed, P3 and N3 are lost, P4 shifts to a shorter latency but is unchanged in amplitude, and N4 both shifts to a shorter latency and is slightly attenuated. These findings are comparable to those observed following injection of a local

TABLE II

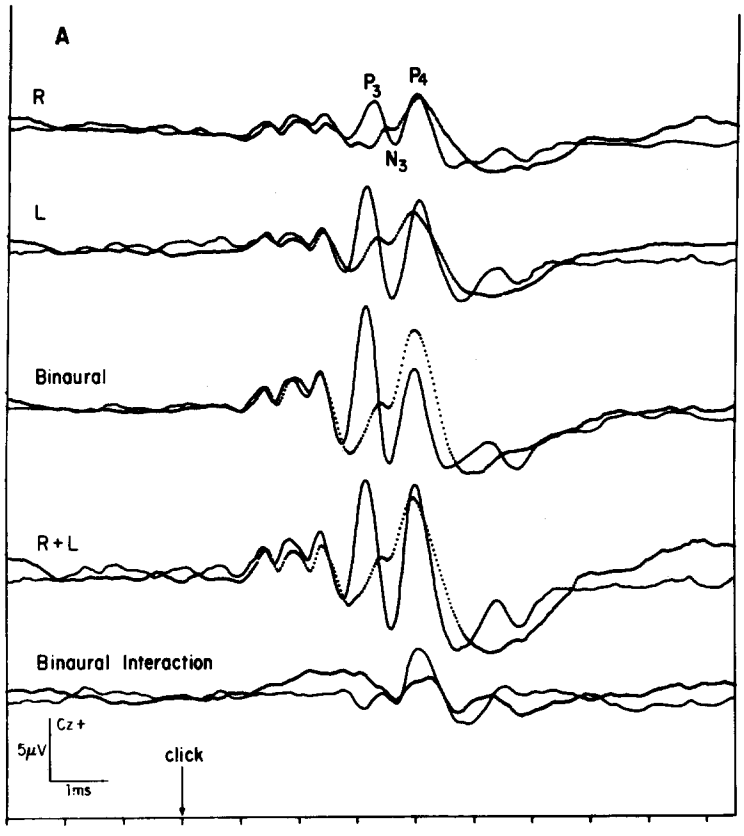
Latency shifts (msec) of ABR components following surgical section of trapezoid body of guinea pig.

Guinea pig	Cut	Ear	P1	N1	P2	N2	P3	N3	P4	N4	P5
GPF	3	R			+0.08	+0.4	*	*	−0.23	−0.35	**
GPN	2	R				+0.3	*	*	−0.13	−0.23	
		L		−0.2	−0.23	+0.18	*	*	−0.35	−0.1	
GPI	1	R		−0.08		+0.3	*	*	−0.18	−0.13	−0.13
		L		+0.13		+0.43	*	*	−0.18	−0.13	−0.13
GPM	1	R			+0.15	+0.3	*	*	−0.13	+0.75	**
		L			+0.08	+0.38	*	*	−0.43	+0.45	**
GI	1	R			+0.2		+0.13	−0.28	−0.23	−0.15	**
		L			+0.1	+0.1	*	*	−0.13	−0.15	**
GB	2	R			−0.13		+0.13	−0.15	−0.23	−0.15	−0.6
		L		−0.13	−0.18			−0.28	−0.23	−0.18	−0.43
GB	1	R				+0.38	+0.35		+0.08		*
		L				+0.15	+0.18	−0.08			*
GH	1	R			+0.18	+0.13	+0.15	−0.08	−0.13		−0.15
		L			+0.2	+0.18					−0.23
GPK	1	R			+0.1	+0.23	+0.38	+0.18	+0.2	+0.15	**
		L		+0.13	+0.15	+0.28	+0.18	+0.13			**
GPN	1	R				+0.13	+0.15		−0.15		*
		L				−0.08	+0.08		−0.1	−0.13	−0.18
GPH	1	R				−0.13	−0.13	−0.45	−0.43	−0.73	**
		L			−0.15				−0.43		
GPL	2	R						+0.1			**
		L			+0.1	+0.1		+0.15	−0.13	+0.1	**
GPG	1	R			−0.13			−0.23	−0.48	−0.43	**
		L						−0.18	−0.23	−0.2	**
GPL	1	R									**
		L						−0.08			**

* No component.
** Component not present in control period.
\+ = increase in latency.
− = decrease in latency.

anesthetic (procaine HCl) into the trapezoid body described in a companion paper (Wada and Starr 1983a). However, with incomplete section of the trapezoid body, the relationship between the extent of the section and the extent of the ABR changes could be further defined. For instance, the amplitude of P3 is linearly related to the number of intact crossing fibers remaining in the trapezoid body with the function having a slope close to unity. In contrast, the amplitude of N3 is not as linearly related to the extent of trapezoid body section.

The role of the trapezoid body in generating the affected ABR components was assessed by recording the evoked electrical activity from the surface of this structure before and after surgery. This approach was inconclusive in the experiments using procaine (Wada and Starr 1983a) since the evoked electrical activity was attenuated throughout the trapezoid body following the injection. In contrast, following midline section of the trapezoid body, activity was attenuated only in those portions of the structure which lay contralateral to the ear stimulated whereas high amplitude activity persisted ipsilateral to the section. Thus, if the trapezoid body were generating the components of the ABR affected by the section (e.g., N2, P3, N3, N4) it would have to be from those portions of the trapezoid body contralateral to the ear stimulated. Conversely, the portions of the trapezoid body



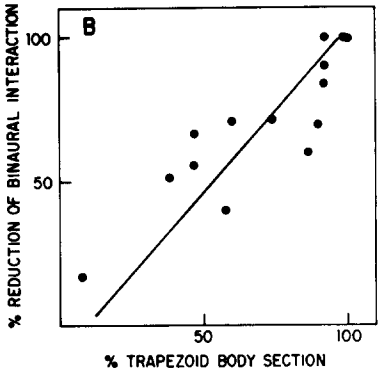


Fig. 5. A: the effects of surgical section of the trapezoid body on binaural interaction in a guinea pig. The ABR in the control period is plotted as a solid line and following surgery by the dotted line. R = right stimulation; L = left stimulation; Binaural = binaural stimulation; R + L = the sum of the two monaural stimulations; Binaural Interaction, R + L − Binaural. Note the diminution of the binaural interaction following trapezoid body section. B: the relationship between the percent of decussating fibers sectioned in the trapezoid body and the percent decrement of amplitude of binaural interaction. The relationship is linear. $r = 0.87$, $r^2 = 0.76$ ($P < 0.001$).

TABLE III

Effects of surgical section of the trapezoid body on ABR in cat.

Cat	Cut	% of TB cut	Ear	Threshold (dB SPL)	% change in amplitude as an (f) of control P3								
					P1	N1	P2	N2	P3	N3	P4	N4	P5
A	3	88	R	34			−35	+46	−100	−100	−18	−92	
			L	74				+14	−100	−100	−11	−87	−17
B	1	33	R	54					−58	−35	+48		
			L	64			−17	+21	−42	−15	+30		
C *	1	82	R	44		+14	−63	+20	−85	−121	−60	−84	
			L	34			−53	+19	−99	−104	−41	−66	+23
E	1	68	R	54			+30	−39	−81	−19	−67	−18	
			L	54			+27	+14	−79	−31	−19	−20	
F	1	92	R	44			−26	+13	−100	−100	−20	−53	−100
			L	44			−13	+34	−100	−100		−43	−100

See footnotes Table I.

* In this animal N3 was larger than P3 in the control period.

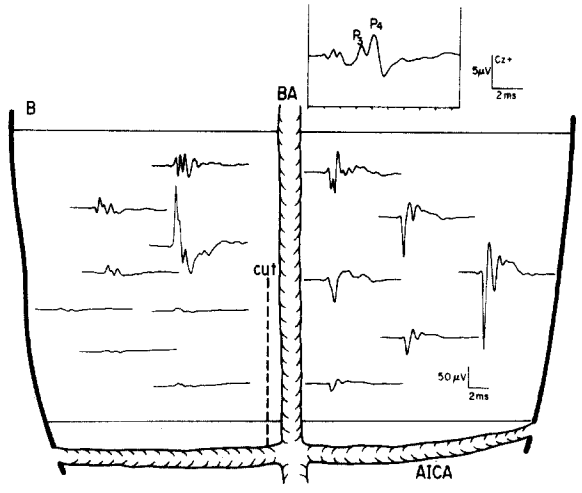


Fig. 6. The ABR (top insert in both A and B) and the distribution of auditory evoked potentials recorded from the ventral surface of the pons before (A) and after (B) section of the trapezoid body. The click occurs at the beginning of each trace. The caudal half of the decussating fibers was cut in midline. Following the section, the evoked potentials from the caudal and contralateral half of the trapezoid body were clearly attenuated compared to the potentials prior to surgery. However, the potentials from the ipsilateral half were either the same or even larger. BA, basilar artery; AICA, anterior inferior cerebellar artery. The thin transverse lines represent the approximate rostral and caudal limits of the trapezoid body.

with unchanged evoked electrical activity following the section could not be the generator of these altered far-field ABR components.

Even with these results we are still unable to distinguish whether the affected portions of the trapezoid body contralateral to the ear stimulated are generating the ABR or are merely the input to other structures responsible for the far-field ABR. Nevertheless, we can conclude that auditory brain stem structures contralateral to the stimulated ear (e.g., trapezoid body, superior olive, lateral lemniscus tract and/or nucleus, inferior colliculus) are candidate generator(s) for components N2, P3, N3 and N4 whereas components P1, N1, and N2 are generated in structure(s) ipsilateral to the section (VIIIth nerve, cochlear nucleus, trapezoid body, superior olive). The identity of the site(s) of generation of component P4 is particularly complicated. In the guinea pig, midline trapezoid body section did not alter the amplitude of this component to monaural stimulation and thus its generator(s) should also be considered as being ipsilateral to the midline. However, the loss of binaural interaction in component P4 following surgical section indicates that crossing fibers in the trapezoid body are essential for the occurrence of this electrophysiological sign of binaural processing.

TABLE IV

Latency shifts (msec) of ABR components following surgical section of trapezoid body of cat.

Cat	Cut	Ear	P1	N1	P2	N2	P3	N3	P4	N4	P5
A	3	R				+0.6	*	*		−0.1	
		L			+0.13	+0.43	*	*		+0.25	
B	1	R									
		L			−0.23		+0.1				
C	1	R						−0.1	+0.08	+0.13	+0.15
		L				+0.2	+0.08	−0.1	+0.1	+0.18	+0.15
E	1	R						−0.2		+0.2	
		L				−0.08		−0.08			
F	1	R			−0.08	+0.58	*	*			*
		L			−0.13	+0.73	*	*			*

* No component.
+ = increase in latency.
− = decrease in latency.
R = right.
L = left.

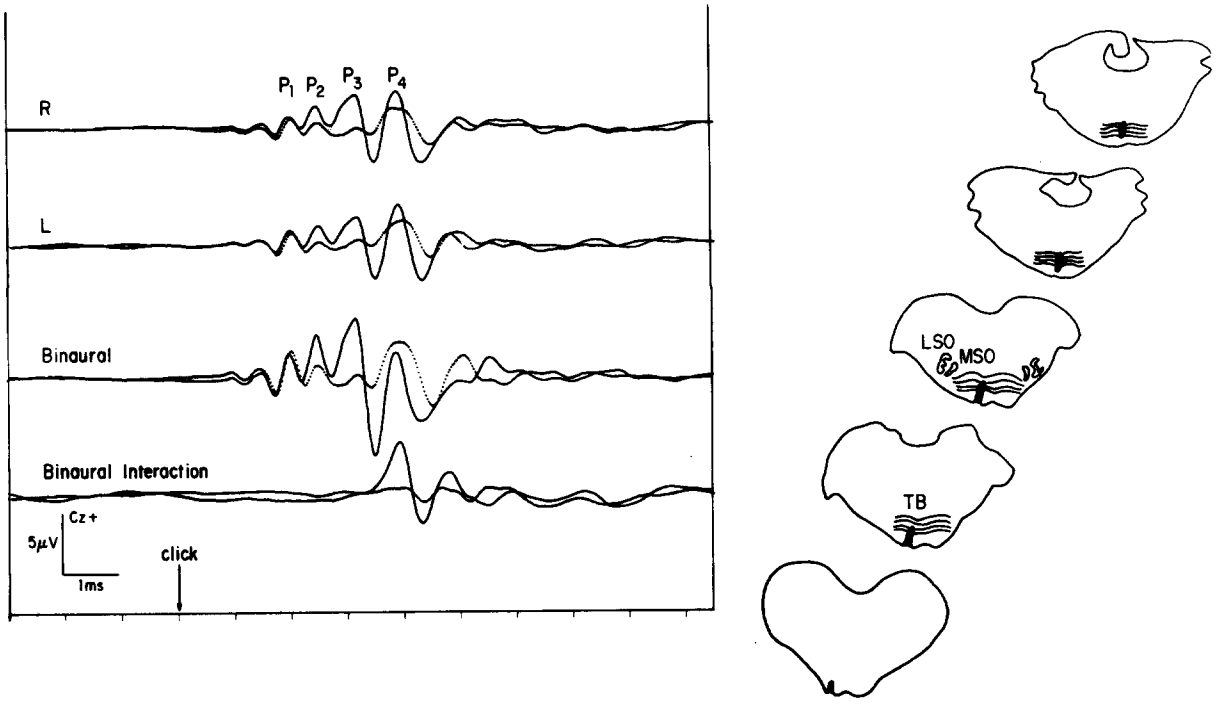


Fig. 7. Reconstruction of the trapezoid body section in cat C and corresponding effects on the ABR to monaural stimulation (upper two traces), binaural stimulation (third traces) and binaural interaction (bottom traces) both before (solid lines) and after (interrupted lines) section of the trapezoid body.

Other lesion studies that utilized total midline section of the brain stem (Buchwald and Huang 1975; Britt and Rossi 1980) are in partial agreement with the results of the present study using a small midline section restricted to the trapezoid body in that there were no changes in components P1 and P2 whereas P3 was lost. However, in the former studies in cat, P4 was also significantly reduced in amplitude to binaural stimulation whereas, in the present study, in both cat and guinea pig, P4 increased in amplitude to binaural stimuli. This difference may reflect that the extensive surgery required for a hemisection has nonspecific detrimental effects on brain stem function or compromised specifically the function of auditory brain stem structures other than the trapezoid body. We favor the latter possibility since when complications of surgery, such as vasospasm, edema, or hemorrhage, affected the brain stem in our experiments the early components of the ABR (P1 and N1) were also attenuated. In the published studies quoted above the decrement of P4 was not accompanied by changes in P1 or N1. In any case, P4 may be differently generated in the cat and guinea pig since following trapezoid body section this component was attenuated in the cat but not the guinea pig to monaural stimulation.

The conclusions of this study, that many of the components of the ABR may be generated in lateralized portion of the auditory pathway, are in agreement with the analysis of the scalp distribution of the ABR in both animal and humans. Waves I and II in humans (equivalent to P1, N1, and P2, N2 in animal studies) are lateralized to the scalp ipsilateral to the ear being stimulated (Picton et al. 1974) and appear to be generated in both the extra- and intradural portions of the VIIIth nerve ipsilateral to the ear stimulated (Hashimoto et al. 1981; Møller et al. 1981). Wave III (P3, N3), in scalp recordings in humans (Picton et al. 1974; Starr and Squires 1982), is lateralized on the scalp being negative at the pinna ipsilateral to the ear

stimulated and positive at the pinna contralateral to the ear stimulated. In monkeys, wave III can be separated into two subcomponents, P3a and P3b, that have different lateralized scalp distributions (Allen and Starr 1978). The results from the present experiments in guinea pigs indicate that P3 and N3 (wave III) are generated in the brain stem contralateral to the ear stimulated. The different types of scalp distributions of wave III noted for these different species may reflect variations in the far-field representations of such lateralized generator(s). The scalp distribution of wave V in humans (Picton et al. 1974) and P4 in animals (Plantz et al. 1974; Allen and Starr 1978; Dum et al. 1981) does not suggest their generation in lateralized structures since amplitudes are largest over the scalp in the midline. Results from the present study indicate that the equivalent positive component in guinea pig (P4) is generated ipsilaterally to the ear stimulated. The absence of an asymmetry over the scalp may be due to the vertical orientation of the dipole for wave V in human and P4 in animal, whereas the dipoles for waves I, II and III are horizontally arranged and are therefore detected asymmetrically on the scalp.

Alterations of the components of the ABR that were found to accompany surgical interruption of the trapezoid body consist of (1) attenuation of amplitude (P3, N3); (2) broadening of duration (P2, P4); (3) prolongation of latency (N2, N4); and (4) shortening of latency (P4). Only two of these changes, that of prolongation of latency and alteration of amplitude, are routinely used as criteria of abnormality of the ABR in clinical tests (Starr and Achor 1975; Stockard and Rossiter 1977; Chiappa et al. 1979; Rowe 1981). A shortening of latency has only rarely been considered as a criterion of abnormality and restricted to disorders of the cochlea (Coats and Martin 1977) or to Trisomy 21 (Squires et al. 1980). Absolute amplitude changes are utilized by only a few (Thornton and Hawkes 1976; Uziel and Benezech 1978) because of its high variability, whereas the amplitude ratio of component V/I is used more often. Finally, the duration of a component has not been utilized as a criterion of abnormality in clinical tests. We suggest that criteria for abnormality of ABR in clinical tests of disorders of the brain stem should include all of the measures noted above. Moreover, quantification of both the peak and trough of each wave should be made separately, if possible, as it is obvious from the studies reported in this paper that central lesions can affect just one of the components of the composite wave.

The final paper of this series, on the effects of lesions rostral to the trapezoid body (Wada and Starr 1983b), will define further the generator sites of some of the lateralized components of the ABR.

Summary

Auditory brain stem potentials were recorded between the skull and a non-cephalic reference electrode in guinea pig and cat before and after midline section of the trapezoid body from a ventral approach. The ABR after complete section was altered: N2 was delayed, P3 and N3 were lost, P4 and N4 were shortened in latency but only N4 was attenuated in amplitude. With partial section of the trapezoid body the amplitude of P3 was linearly related to the extent of the section. Recordings from the surface of the trapezoid body before and after section revealed a loss of activity contralateral to the ear stimulated and a preservation of activity ipsilateral to the section. Binaural interaction in P4 and N4 was attenuated in a linear manner as a function of the extent of trapezoid body section. This study suggests that auditory brain stem structures contralateral to the midline of the trapezoid body generate components N2, P3, N3, N4, whereas auditory structures ipsilateral to the midline generate components P1, N1, P2 and P4.

Résumé

Genèse des réponses auditives du tronc cérébral (RATC). II. Effets d'une section chirurgicale du corps trapézoïde du cobaye et du chat

Les potentiels auditifs du tronc cérébral ont été enregistrés entre le crâne et une électrode de référence non céphalique chez le cobaye et chez le chat avant et après section du corps trapézoïde le

long de la ligne médiane, ceci par approche ventrale. Après section complète, les potentiels ont été affectés comme suit: N2 a été retardée; P3 et N3 ont disparu; la latence de P4 et de N4 a été raccourcie, mais seule l'amplitude de N4 a diminué. Après section partielle du corps trapézoïde, l'amplitude de P3 s'est révélée linéairement liée à l'étendue de la section. Des enregistrements effectués à la surface du corps trapézoïde avant et après section ont révélé une perte d'activité contralatérale. L'interaction binaurale, pour P4 et N4, a été diminuée de façon linéaire en fonction de l'étendue de la section du corps trapézoïde. Cette étude suggère que la genèse des composantes N2, P3, N3, N4 est due à des structures auditives du tronc cérébral contralatérales par rapport à la ligne médiane du corps trapézoïde, alors que les composantes P1, N1, P2, et P4 le sont par des structures auditives ipsilatérales à la ligne médiane.

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