GENERATION OF AUDITORY BRAIN STEM RESPONSES (ABRs). III. EFFECTS OF LESIONS OF THE SUPERIOR OLIVE, LATERAL LEMNISCUS AND INFERIOR COLICULUS ON THE ABR IN GUINEA PIG

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While there is consensus that the ABR recorded at the scalp represents the far-field activity of brain stem structures comprising the auditory pathway (Jewett 1970; Buchwald 1982), there is uncertainty as to the precise localization of the generators for some of the components. While waves I (P1, N1), and perhaps II (P2, N2), reflect activity in the VIIIth nerve both within the cochlea and proximally near the brain stem (Møller et al. 1981), wave III (P3, N3) has been localized only roughly to the region of the pons (Starr and Hamilton 1976; Stockard and Rossiter 1977), perhaps contralateral to the stimulated ear (Buchwald and Huang 1975), and IV and V (P4, N4) to the region of the midbrain (Starr and Hamilton 1976; Hashimoto et al., 1981; Møller and Jannetta 1982) perhaps bilaterally (Buchwald and Huang 1975). Furthermore, it is not known whether each wave is generated primarily by one brain stem structure or whether activity in several different sites combine to account for the appearance of that wave (Jewett 1970; Buchwald and Huang 1975; Achor and Starr 1980a).

In the first two articles of this series on the role of the trapezoid body in the generation of the ABR (Wada and Starr 1983a, b) we demonstrated that components P1, N1, P2 and P4 are generated in the brain stem ipsilateral to the ear stimulated. In this final article of the series we made fairly discrete lesions in the superior olivary complex, lateral lemniscus and inferior colliculus to further define the locus of the generator site(s) for the ABR components.

Methods

Subjects

Twenty-six adult guinea pigs weighing between 0.7 and 1.1 kg were subjected to lesions of the pons and midbrain.

Stimulus generation, ABR recording and data analyses were the same as in the first two studies (Wada and Starr 1983a, b).

Surgery

The animals were deeply anesthetized with sodium pentobarbital. Following a tracheotomy and fixation of a small screw at a point 3 mm posterior to the bregma to serve as the 'active' electrode for recording the ABR, 3 different surgical approaches were employed to make the brain stem lesions. A ventral approach was similar to that described in the first two papers and was used to lesion the superior olivary complex. A dorsal approach consisting of removal of the occipital bone and aspirating the cerebellum was used for lesions of the midbrain and lateral lemniscus. A lateral approach through the bulla and temporal bone was used to expose the lateral aspect of the brain stem for unilateral lesions of the superior olivary complex or lateral lemniscus. This last
approach damaged the cochlea so that the ABR only to contralateral input could be tested.

**Experimental lesions and histology**

Electrolytic lesions were made in 19 animals by placing a 100 μm stainless steel wire, insulated except for the bare cut end, into the appropriate structure guided by the potentials recorded from that electrode to clicks. Discrete lesions (1–4.5 mm) were then made by passing 1 mA of cathodal current for varying times (5–50 sec). In 6 animals, the brain stem was sectioned transversely with a blunt spatula just caudal to the inferior colliculus without damaging the basilar artery. A unilateral section was made in 4 and bilateral sections in the other 2 animals. In one other animal the inferior colliculus was removed bilaterally by aspiration. Following the lesions, recordings of the ABR were continued for at least 2 h.

At the end of the experiment, the animals were perfused through the heart with normal saline followed by a 10% buffered formalin solution. The entire brain was removed, blocked and stored in 10% buffered formalin for at least 1 week prior to sectioning. Serial reconstruction of the extent of the lesion was made from 60 μm frozen sections stained with cresyl violet. Lesions made by transection were confirmed both by visual inspection and by examining serial sections from the lesion sites.

**Results**

The effects of lesions of the region of the superior olivary complex, the lateral lemniscus (unilateral and/or bilateral) and inferior colliculus on the amplitude and latency of the ABR are summarized in Tables I and II.

**(A) Lesions adjacent to the lateral aspect of the superior olivary nucleus**

In 4 animals the lesion was dorsolateral to the superior olivary nucleus contacting the lateral aspect of the lateral superior olivary nucleus. The lesions, in common, involved a portion of the lateral superior olivary nucleus, the dorsal and intermediate acoustic stria, as well as those fibers of the ventral acoustic stria entering the ipsilateral superior olivary complex. Fig. 1 shows the reconstruction of the lesion and the corresponding effects on the ABR in guinea pig GX. The lesion involved the lateral aspect of the lateral superior olive and dorsal part of the ventral acoustic stria, and extended dorsally into the pons. Changes in the ABR were primarily to stimulation of the ear ipsilateral to the lesion (labeled ‘R’ in Fig. 1); component P4 decreased in amplitude by 39% and was delayed in latency by 0.2 msec; component N4 decreased in amplitude by 35% and was also delayed in latency (0.25 msec). In contrast, component N3 increased in amplitude by 25%. The other components, P1 through P3, were unchanged. The ABR evoked by stimulation of the ear contralateral to the lesion (labeled ‘L’ in Fig. 1) showed no significant amplitude and latency changes except for components N1 and P5 which decreased slightly in amplitude (by 21% and 29% respectively) while P4 increased slightly by 13%. In the other 3 animals with a lesion in this area the major effect on the ABR was a decrease in amplitude (29–70%) and a prolongation of the latency of components P4 and N4 to ipsilateral stimulation similar to the example in Fig. 1. In addition, in these 3 guinea pigs, components P3 and N3 also decreased slightly (13–35%) in amplitude. In summary, lesions of the lateral aspect of lateral superior olive that did not involve the trapezoid body affected the ABR evoked by stimulation of the ear ipsilateral to the lesion with a decrease in amplitude and prolongation of latency particularly of components P4 and N4.

**(B) Lesions of the superior olivary complex**

In 3 animals the lesions were restricted to the superior olivary complex without significant damage to other auditory structures. Fig. 2 shows the reconstruction of the lesion and the corresponding effects on the ABR in guinea pig PF. The lesion involved almost the entire medial superior olivary nucleus and approximately the dorsal 2/3 of the lateral superior olivary nucleus without impinging on the trapezoid body. The ABR to stimulation of the ear contralateral to the lesion (labeled ‘L’ in Fig. 2) had a decrease in amplitude for components N1 (22%), P3 (68%), N3 (57%)
Fig. 1. Reconstruction of a lesion of the lateral region of the pons in guinea pig GX and corresponding effects on the ABR to monaural stimulation. In this and all subsequent figures the ABR in the control period is in the solid line and following the lesion is in the interrupted line. The lesion involved the lateral aspect of the lateral superior olive, the periolivary region including the dorsal and intermediate acoustic striae and the dorsal aspect of the ventral acoustic stria. Note that components P4 and N4 are both delayed and attenuated to ipsilateral stimulation (R) whereas contralateral stimulation (L) is without effect. The following abbreviations are used in this and subsequent figures. TB = trapezoid body; LSO = lateral superior olive; MSO = medial superior olive; LL = lateral lemniscus; IC = inferior colliculus; IV vent = fourth ventricle.

and N4 (48%), and amplitude increments for components P1 (14%) and N2 (22%). (The changes in P1 and N1 appear to be due to a baseline shift since the composite wave I was unchanged.) In contrast, the ABR to ipsilateral stimulation (labeled ‘R’ in Fig. 2) showed components P4 and N4 to decrease in amplitude by 11% and 64% respectively. In the other 2 animals, the lesions also involved the entire medial superior olivary nucleus and the dorsomedial side of the lateral superior olivary nucleus. The changes in the ABR to contralateral stimulation were comparable with a marked attenuation of component P3 and N4 to contralateral stimulation without consistent effect on P4. N3 to contralateral stimulation was attenuated in 2 of the 3 animals in this group.

(C) Lesions of both the superior olivary complex and the trapezoid body

In 3 animals, the lesions involved both the superior olivary complex and a significant part of the trapezoid body. Fig. 3 shows the reconstruction of the histology and the corresponding effects on the ABR in guinea pig PE. The lesion began within 2 mm of the dorsum of the brain stem extending ventrally to involve almost the entire medial superior olivary nucleus, approximately 3/4 of the lateral superior olivary nucleus in its medial side, and approximately 60% of the fibers in the trapezoid body just ventral to the superior olivary complex. The ABR to stimulation of the ear ipsilateral to the lesion (labeled ‘R’ in Fig. 3) was affected with components P3, N3, P4 and N4,
Fig. 2. Reconstruction of the superior olivary complex lesion in guinea pig PF and corresponding effects on the ABR to monaural stimulation. The lesion involved the entire medial superior olivary nucleus and the medial part of the lateral superior olivary nucleus without involving the trapezoid body or acoustic striae. The effects on the ABR were an attenuation of components P3 and N3 to contralateral stimulation (L) and N4 to both ipsi- (R) and contralateral (L) stimulation.

being attenuated by 79%, 46%, 51% and 58% respectively. The ABR to contralateral stimulation (labeled 'L' in Fig. 3) showed components P3, N3 and N4 to decrease in amplitude by 88%, 39% and 28% respectively, and to also decrease in latency by 0.18 msec, 0.3 msec and 0.23 msec respectively. Component P4, though its latency was decreased by 0.3 msec, was not attenuated. Thus, the effects of a lesion of both the superior olivary complex and the trapezoid body on the ABR were similar to those of a trapezoid body lesion alone (see Wada and Starr 1983b), with the exception that P4 was not attenuated with section of the trapezoid body alone. P4 becomes modified with the additional involvement of the tegmentum adjacent to the lateral superior olivary nucleus only when the ear stimulated is ipsilateral to the lesion site.

(D) Unilateral lesion of the lateral lemniscus

Two different methods were employed to lesion the lateral lemniscus, i.e., electrolytic coagulation and surgical section. Of the 11 animals with unilateral lemniscus lesions, 5 were by electrical coagulation and 6 by section. Fig. 4 shows the reconstruction of the histology and the corresponding effects on the ABR in one of these animals, guinea pig GQ. In this instance, only the ABR to contralateral stimulation was recorded because the lateral surgical approach to the brain stem employed is accompanied by damage to the ipsilateral cochlea. Almost the entire lateral lemniscus was lesioned with the most conspicuous change in the ABR being the loss of component N3 evoked by contralateral stimulation. The slight changes in components P2, N2 and P3 were not regularly seen in other animals. In all other examples with unilateral lateral lemniscal lesions, component N3 evoked by stimulation of the ear contralateral to the lesion was markedly attenuated. In contrast, P4 was inconsistently affected being
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<th>Approach</th>
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**Table 1:** Effects of the lesions of the pons and the midbrain on ABR in guinea pig.
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Lesion: Lat to SO = lateral lesion to superior olivary complex; SO = superior olivary complex; TB = trapezoid body; LL = lateral lemniscus; uni = unilateral; bi = bilateral; IC = inferior colliculus; MID = midline region of tegmentum.
Approach: D = dorsal; L = lateral; V = ventral.
Method: E = electrolysis; S = section; A = aspiration.
Stimulated ear: I = ipsilateral; C = contralateral; R = right; L = left.
Amplitude change: + = increase in amplitude as a % of control P3 (or III) amplitude; - = decrease in amplitude as a % of control P3 (or III) amplitude; 100% = component lost; ** = component not identified prior to lesion.
Blank spaces represent amplitude shifts of <10%.

slightly decreased in amplitude in 3 cases, and changing less than 10% in the other 5 cases. Components N4 and P5 were attenuated in 3 cases and unaffected in 6 cases.

Fig. 5 shows the effects on the ABR following both unilateral and bilateral section of the lateral lemniscus in guinea pig PK. The physiological completeness of the section was assessed by monitoring long latency evoked potentials (time base of 128 msec, bandpass of 1-3000 Hz) to slow rates of click stimulation (1/sec). The sections were made caudal to the inferior colliculus. The effects of unilateral section of each ear were fairly symmetrical. Component N3 was attenuated by 76% and shifted to a positive polarity. Component N4 decreased in amplitude by 20% to right stimulation and by 50% to left stimulation. In contrast, component P4 decreased in amplitude by 78% to right stimulation and by 50% to left stimulation. The latencies of P5, N3 and N4 slightly increased.

The results in guinea pig TQ were similar to those in guinea pig PK, with component N3 being markedly attenuated. The effects of bilateral section of the lateral lemniscus were similar to those of unilateral section. Component N3 was attenuated by 51% to right stimulation and by 69% to left stimulation, whereas the effects of unilateral section were attenuated in 3 cases and changing less than 10% in the other 5 cases. Components N4 and P5 were attenuated in 3 cases and unaffected in 6 cases.

When a dorsal surgical approach was used to allow a section of the lateral lemniscus without damaging the cochlea, and there were little or no effects on the ABR to ipsilateral stimulation, whereas the attenuation of N3 to contralateral stimulation still occurred. In summary, the effects of a unilateral lateral lemniscus lesion (or disappearance of contralateral stimulation) on the ABR were similar to those of bilateral section, with a consistent slight attenuation of N3 to contralateral stimulation.
TABLE II
Latency shifts (msec) of ABR components following the lesion.

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+, increased in latency (msec) following the lesion; -, decreased in latency (msec) following the lesion. For the other explanations of abbreviations see Table I footnote.
In summary, the effects of bilateral lateral lemniscus lesions were characterized by a marked attenuation of component N3 and N4 to both right and left stimulation. In contrast, P4 was only slightly affected.

(F) Lesions of the inferior colliculus
In two guinea pigs the inferior colliculi were destroyed. In guinea pig TP, the lesions were electrolytic without any significant effect on the ABR. In guinea pig TR both inferior colliculi were aspirated under visual control. Fig. 6 shows the reconstruction of the lesion in guinea pig TR and absence of effects on any of the components (P1-P5) of the ABR to stimulation of the right ear.

(G) Lesions of non-auditory brain stem
In 3 animals the lesions intended for the auditory pathway missed their mark and ended close to the midline of the tegmentum. However, in all 3 instances the ABR was still affected. Fig. 7 shows the reconstruction of the histology and corresponding effects on ABR in guinea pig GV. The lesion was restricted to the central tegmentum extending from the upper pons to lower midbrain. There was an amplitude increment of P4 and N4 (from 20 to 43%) to both right and left stimulation. Similar effects were observed in the other two animals. In our experience the amplitude of the ABR component does not vary more than 10% in the absence of any intervention.
Binaural interaction

Binaural interaction in the ABR represents the non-linear processing of simultaneous monaural stimulation and takes the form of a reduction in amplitude of components P4 and N4 to binaural stimulation compared to the sum of these components to separate monaural stimulation (Dobie and Berlin 1979). In the earlier papers (Wada and Starr 1983a, b) we demonstrated that binaural interaction depends upon the integrity of the trapezoid body and in addition showed that its amplitude is linearly related to the number of crossing fibers remaining intact. In the present study we investigated the effects of rostral brain stem lesions on binaural interaction. Lesions of the superior olivary complex were associated with an attenuation of binaural interaction independent of their effects on the monaurally evoked ABRs. For instance in guinea pig GX, in which the lesion was accompanied by an attenuation similar to that of guinea pig PF in which P3, N3 and N4 but not P4 were attenuated. In contrast, unilateral lesions of the lateral lemniscus were without significant influence on binaural interaction whereas bilateral lateral lemniscal lesions were associated with a loss of binaural interaction in the N4 component but not in the P4 component. Thus, the intactness of both superior olivary regions is essential for the demonstration of binaural interaction in P4 and N4 of the ABR. With bilateral section of the lateral lemniscus only the binaural interaction occurring at the time of N4 is lost, whereas the interaction associated with P4 is preserved.
Discussion

The results of these studies showing that particular components of the ABR are affected with lesions of small areas of the auditory pathway are compatible with the hypothesis of the focal generation of the ABR components. Thus, P3 is attenuated with lesions in the region of the medial superior olivary nucleus contralateral to the ear stimulated; N3 is lost with lesions in the ventral region of the lateral lemniscus contralateral to the ear stimulated; P4 is attenuated with lesions in the lateral portions of the pons involving the tegmentum adjacent to the lateral superior olivary nucleus ipsilateral to the ear stimulated; N4 is attenuated with lesions in any of the 3 aforementioned areas. These findings support the conjectures of Jewett (1970) and, more recently, Buchwald (1982) that the generation of each of the components of the ABR is principally from a limited region of the auditory brain stem pathway. An alternate concept proposed by Achor and Starr (1980a, b), that each component of the ABR arises from simultaneous activity in several different sites in the auditory pathway, is not supported by these new observations. Thus, even though electrical events in the brain stem may be widespread at the time of the peak of many of the components (Achor and Starr 1980a), only a restricted portion of this activity is probably detected at the scalp as a ‘far-field’

Fig. 5. Reconstruction of the brain stem following surgical section of both lateral lemnisci in guinea pig PK and the corresponding effects on the ABR. The effects on the ABR following a unilateral section of the right lateral lemniscus (Cut 1) are in Fig. 5A and following bilateral section (Cut 2) are in Fig. 5B. An anatomical rendering on the right side of the figure shows the level of the cuts from both the dorsal view (bottom drawing) and from a transverse section of the midbrain (top section) taken at the level of transection. Following the first cut, component N3 to contralateral stimulation (L) markedly decreased in amplitude. Following the second cut (Fig. 5B), the effects on the ABR were fairly symmetrical to stimulation of either ear with N3 being markedly attenuated.
Fig. 6. Reconstruction of the inferior colliculi lesion in guinea pig TR and the effects on the ABR to monaural stimulation. Following both unilateral and bilateral aspiration of the inferior colliculus, no significant changes occurred in the ABR.

Fig. 7. Reconstruction of a midline tegmental lesion in guinea pig GV and the effects on the ABR to monaural stimulation. This lesion was located in the midline of the tegmentum extending from the IVth ventricle to the lower midbrain and did not involve any auditory structures. Both P4 and N4 to monaural stimulation increased in amplitude.
event. One must still be cautious in accepting such

a conclusion since the effects of brain stem lesion

on the ABR, if considered in isolation, can be

misleading. For instance, in the second paper of

this series (Wada and Starr 1983b), we demon-

strated that section of the trapezoid body is associ-

ated with a loss of P3 and N3. This result should

not be taken to mean that the trapezoid body is

the generator of these components. Rather, as has

been made evident in the present study, the gener-

ator sites of the affected components are other

brain stem auditory structures that receive input

from the trapezoid body. Thus, it is only by having

knowledge of the connections of the auditory

pathway and by making sequential lesions along

the pathway that the results can be fairly interpre-

ted.

The question as to which neural structures

within a focal brain stem region contribute to a

particular far-field ABR component is not known.

Buchwald (1982) argues cogently that the far-field

components reflect synaptic events rather than

fiber discharges. However, the possibility that nerve

fiber discharges contribute to the ABR receives

support from analyses of evoked potentials from

the somatosensory system in which the first posi-
tive potential recorded between the scalp and a

non-cephalic reference to stimulation of the median

nerve is the far-field reflection of activity in pe-

ripheral nerve fibers in the arm (Wiederholt and

Iraqui-Madoz 1977). In the present experiments

we have no way of distinguishing whether a lesion

in the region of the medial superior olivary nucleus

which is accompanied by an attenuation of P3 has

its effect due to a disruption of nerve fiber activity
reflecting input or output axons to this area or to a

disruption of synaptic events in the cell bodies

themselves.

The present studies do not support the concept

that each sequential component of the ABR arises

from higher (more rostral) segments of the audi-
tory pathway. We found that both P3 and P4 arise

from the pons, albeit on different sides, whereas

N3, which occurs intermediate between these two

components, arises from a more rostral region, the

lateral lemniscus. The mechanisms by which P4

occurs later than P3, though both arise from the

pons, could reflect: (1) differences in the timing of

afferent input to the pons from the cochlea, i.e.,
basal versus apical end, for instance, with the

latency difference reflecting differences in the

travel time of acoustic events along with basilar

membrane; (2) differences in the cochlear nucleus

origins of input to the pons with each having
different latencies of activation; or (3) differences

in the synaptic processing at the sites of generation

such that additional interneurons may be inter-

posed in the pons delaying the appearance of P4

relative to P3.

Our findings also bear on how the ABR is used

for the localization and definition of neurological

lesions. The present strategy of measuring the am-

plitude of the composite waves (I, II, III, etc.)

(Thornton and Hawkes 1976) rather than their

components (e.g., P1, N1 for wave I) is incomplete

since, as is evident from the studies presented here,

lesions of the auditory brain stem pathway may

affect only one of the components of a composite

wave. Secondly, if the analogy between human

and animal ABR components is correct, particu-

larly that P4 and N4 in animals are equivalent to

the IV–V complex in humans, then the site of
generation of the IV–V complex must be consid-
ered to arise from the tegmentum of the pons just

lateral to and possibly involving the lateral super-
or olivary nucleus rather than from the inferior
colliculus or lateral lemniscus as has been gener-
ally proposed (Hashimoto et al. 1981; Møller and

Jannetta 1982). Moreover, if the generator sites for

human and animal ABR components are similar,

the lateralized origins of the components from one

or other side of the brain stem should be consid-
ered when making interpretations of abnormal

ABRs in clinical applications.

Fig. 8 summarizes the results of this and the 2

preceding studies (Wada and Starr 1983a, b) on

the effects of discrete lesions of auditory brain

stem structures involving the trapezoid body, su-
perior olivary complex and surroundings, lateral

lemniscus and inferior colliculus on auditory brain

stem responses to monaural stimulation. The

hatched areas represent the degree of attenuation

of each of the components with the extent ex-

pressed as a percentage of each component. The

side of acoustic stimulation relative to the lateral-

ity of the brain stem lesions is indicated in the
Fig. 8. Schematic representation of the effect of various brain stem lesions on the components of the ABR. The site of the lesion is indicated in the brain stem section along with an arrow indicating the ear stimulated. With both midline lesions (trapezoid body) and bilateral auditory pathway lesions (lateral lemniscus, inferior colliculus), the effects of stimulation of either ear on the ABR were comparable. The ABR before the lesion is indicated by the solid line and following by the interrupted line. The differences between the two potentials are highlighted by cross-hatching when the lesion is accompanied by an attenuation and by stippling when the lesion is accompanied by an enhancement. The extent of the attenuation of enhancement is noted below the affected components.

accompanying brain stem figure by the arrow. If the lesions were in the midline (as with the trapezoid body) or were bilateral, the effects from stimulation of either ear on the ABR were comparable. The data are obtained from the tables of amplitude change of the ABR in the 3 papers of the series and include an average of the results from all of the animals with varying extents of the lesion with the exception of the trapezoid body in which only the 4 animals with total transection of these fibers were included.

Note that (1) the amplitude of components prior to P3 are unaffected by pontine and midbrain lesions; (2) P3 is lost with complete midline trapezoid body lesions and is markedly attenuated with partial lesions of the superior olivary nucleus to contralateral but not to ipsilateral input; (3) N3 is lost with midline trapezoid body lesions and is also attenuated by lesions of either the superior olivary nucleus or lateral lemniscus either bilateral or contralateral to the ear stimulated; (4) P4 is attenuated only by lesions in the lateral pons impinging on the lateral superior olivary nucleus and only if evoked by ipsilateral but not by contralateral stimulation (a trapezoid body lesion affects a paradoxical increase in amplitude to P4, dotted area); (5) N4 is affected by lesions of the trapezoid body, the superior olivary nucleus (both ipsilateral and contralateral inputs), the lateral
pontine region similar to that affected P4 (but only to ipsilateral input), and bilateral lateral lemniscal lesions; (6) lesions of the inferior colliculus were without effect.

A schematic representation of the auditory pathway in the guinea pig is in Fig. 9 and includes the presumed locus of the principal generations for the ABR. Results from previously published studies suggest that P1, N1 and perhaps P2 and N2 originate from the VIIIth nerve (and/or its ganglion) both within the cochlea and proximally as the nerve enters the cochlear nucleus (Hashimoto et al. 1981; Moller et al. 1981; Buchwald 1982). Since the effects of a midline trapezoid body lesion on P3 and N3 of the ABR are essentially mimicked by a combination of lesions of the superior olivary nucleus and lateral lemniscus contralateral to the ear stimulated, we conclude that the trapezoid body does not generate the ABR components but acts to transmit inputs to these other regions which, in turn, generate P3 (contralateral superior olivary nucleus) and N3 (contralateral lateral lemniscus). This conclusion is supported by examples from the lesion experiments of Buchwald and Huang (1975) and Achor and Starr (1980b). In the present series (Wada and Starr 1983a, b) P4 was unaffected by isolated trapezoid body lesions, a finding opposite to the results of Buchwald and Huang (1975) which may reflect that the lesions in the latter studies involved the tegmentum of the brain stem as well as the trapezoid body. Lesions in the lateral pontine region contiguous to and involving the lateral superior olivary nucleus affected P4 to ipsilateral stimulation. It is unclear from the data whether the lesion affects P4 because of damage to (a) inputs to the superior olive (middle, dorsal and ventral acoustic striae), (b) the nucleus itself (lateral superior olive or periolivary nuclei), or (c) outputs from the nucleus. N4 was affected to a moderate degree by lesions of the trapezoid body, superior olivary nucleus and the lateral lemniscus. However, with bilateral lesions of the lateral lemniscus, N4 was maximally attenuated suggesting its generation bilaterally. P5 is too variable in appearance in our experience in both guinea pig and cat to warrant any conclusion as to its site of generation. Finally, the inferior colliculus is not involved in the generation of any of the ABR components. In summary, these results demonstrate the relative complexity of generation of the ABR components and emphasizes both the advantages and disadvantages of the lesion methods in fathoming the relationship between auditory brain stem structures and ABR components.

Summary

Auditory brain stem potentials were recorded between the skull (vertex) and a non-cephalic reference in guinea pig before and after making discrete lesions of the auditory pathway in the pons and midbrain. Lesions of the superior olivary complex were accompanied by attenuation of P3 and N3 to contralateral input. Lesions of the lateral lemniscus were accompanied by attenuation of N3 to contralateral input. Lesions of the lateral portion of the pons adjacent to the lateral superior olivary nucleus were accompanied by attenuation of P4 to ipsilateral input. Lesions of the inferior colliculus were without effect on the ABR. These data are interpreted as supporting the hypothesis that each component of the ABR arises from a focal region of the brain stem auditory pathway.

Résumé

Origine des réponses auditives du tronc cérébral (RATC). III. Effets de lésions de l'olive supérieure, du lemnisque latéral et du colliculus inférieur sur la RATC chez le cobaye

Les potentiels auditifs du tronc cérébral ont été recueillis chez le cobaye, entre le crâne (vertex) et une référence non céphalique, avant et après lésions localisées effectuées le long du trajet auditif, dans le pont et le mésencéphale. Les lésions du complexe olivaire supérieur ont été suivies par une atténuation de P3 et de N3 à des stimulations contralatérales; celles du lemnisque latéral, par une atténuation du N3 à des stimulations contralatérales. La lésion de la région latérale du pont, adjacente au noyau latéral de l'olive supérieure ont été accompagnées d'une atténuation du P4 à des stimulations ipsilatérales. La
lésion du colliculus inférieur n'a pas eu d'effet sur la RATC. Ces données sont en faveur de l'hypothèse selon laquelle chaque composante de la RATC prend son origine dans une région très précise de la voie auditive du tronc cérébral.

References
