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Hearing Loss in Dogs After Lesions of the Brachium of the Inferior Colliculus and Medial Geniculate

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ABSTRACT

Seven dogs were tested for their sensitivity to pure tones following lesions of the brachium of the inferior colliculus and medial geniculate body. Bilateral section of the brachium of the inferior colliculus consistently resulted in an average hearing loss of as much as 37 dB in the midrange of the animals' audiograms. Lesions of the medial geniculate appear to produce a similar hearing loss if the ventral division of the medial geniculate is completely destroyed.

Key words: audiogram, behavior, cortex, discrimination, thalamus

A fundamental question in the study of the central auditory system has been the effect of lesions on the detection of sound. Indeed, the knowledge of whether a particular lesion affects an animal's absolute sensitivity is useful for theoretical as well as for practical reasons. Not only is such information necessary for the determination of the neural mechanisms involved in sound detection, but the possibility of a hearing loss must be considered in evaluating the effects of central lesions on more complex auditory discriminations.

Despite the importance of determining the effect of a lesion on absolute sensitivity, few modern studies have examined the audiograms of animals following central auditory system lesions (for a review, see Neff et al., '75). At the present time, the primary source of information on this subject is the study by Kryter and Ades ('43) which investigated the effects of lesions of the lateral lemniscus, inferior colliculus, brachium of the inferior colliculus, and auditory cortex on hearing. In general, the results of their study indicated that lesions above the level of the tectum (i.e., brachium and auditory cortex lesions) have little or no effect on absolute thresholds. Though some of their cases with brachium lesions did show significant hearing losses, they argued that the losses were due to the encroachment of the lesions into adjacent structures such as the inferior and superior colliculi. However, as they did not illustrate the lesions, the extent of such encroachment and the completeness of the smaller brachium lesions must be determined from brief written descriptions.

Recently, we have been studying the effect of brachium and medial geniculate lesions on the hearing ability of dogs. During the course of this study it became apparent that some of these animals had suffered a noticeable hearing loss. The purpose of this paper, then, is to describe both the observed hearing loss and the lesions which consistently produced it.

MATERIALS AND METHODS

Subjects

Seven dogs ranging in size from 4 to 20 kg were used in this study. The animals were housed in rooms with free access to food and were trained using a water reward. The ears of each animal were examined during surgery and following completion of behavioral testing to ensure that they were free of damage or disease.

Surgical and histological techniques

Radio-frequency (RF) lesions were made under aseptic conditions prior to auditory testing. Four animals received bilateral lesions of the brachium of the inferior colliculus and, to varying degrees, part of the medial geniculate. Three additional animals received bilateral lesions of the medial geniculate. The general procedure was to make the lesion on the left side somewhat larger than the one on the right side. This was done to ensure that the left lesion included the entire area covered by the right lesion. Any resulting deficit would then be attributed to the structures bilaterally destroyed, i.e., those included in the smaller right lesion.

For surgery, an animal was initially anesthetized with sodium thiamylal (18 mg/kg), followed by methoxyflurane

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administered via an endotracheal cannula as needed to maintain deep anesthesia. The animal was then placed in a stereotaxic instrument (David Kopf 1504) using nonrupture earbars. The head was shaved and the skin and muscle were incised with a cautery. Bilateral openings of 1–2 cm were made in the skull directly over the lateral midbrain. A 3.5-inch (8.9 cm) 20-gauge spinal needle was placed in the electrode carrier of the stereotaxic instrument and lowered to the location of the intended lesion. This needle was used to penetrate the layers of pia covering the cortex and midbrain in order to provide a smooth, unobstructed path for the electrode which was used to make the RF lesions (Radionic RFG-4). Three penetrations and lesions were made in this manner in the region of the brachium of the inferior colliculus or medial geniculate on each side.

After the lesions were made, the incision was closed in layers, gas anesthesia was withdrawn, an intramuscular injection of acepromazine (0.4 mg/kg) was given, and the animal was placed in a cage to recover. The animals were given an antibiotic (Lincocin, 100 mg per day) for 3 days following surgery. In most cases, training was begun 1 week after surgery and testing was completed 7–10 weeks later. D-2 and D-52, however, were initially tested on sound localization before their audiograms were determined. Absolute threshold testing for D-2 was begun 10 weeks after surgery and was completed 5 weeks later. Testing for D-52 began 7 weeks postoperatively and was completed 3 weeks later.

Following completion of behavioral testing, the dogs were deeply anesthetized with pentobarbital sodium and perfused with isotonic saline followed by 10% formalin. The brains were removed and prepared for frozen sectioning. The midbrain and thalamus were sectioned in the coronal plane at 33 μm and two sets of sections 99 μm apart were stained—one with thionin and one with protargol. These sections were then used to reconstruct the limits of the lesions and any resulting degeneration.

Behavioral apparatus

Testing was conducted in a double-walled sound chamber. To reduce sound reflections the walls of the room were lined with acoustic foam, the floor was carpeted, and burlap was loosely draped along the ceiling. The test cage was constructed of wire mesh (93 × 45 × 80 cm) with a grill bar floor. An opening was provided at one end of the cage, through which the animal placed its head in order to drink from a small cup located in front of a loudspeaker. Water was delivered to the cup via an electrically operated water valve located outside the test chamber. A contact switch, connected at one end to the cup and at the other end to alternate bars on the floor of the cage, served to detect when the animal made contact with the cup. Mild electric shock was provided by a shock generator that was connected between alternate bars on the floor of the cage.

Acoustical apparatus

Sine waves were produced by a generator (Hewlett-Packard 200CD) and gated on and off at zero voltage using two bipolar compadrators (Coulbourn S21-06) and a rise-fall gate (Coulbourn S84-04). The signal was pulsed approximately three times per second (200 msec on, 100 msec off, rise-decay of 50 msec). The signal was then led to an attenuator (Hewlett-Packard 350B), band-pass filter (Krohn-Hite 3202), an impedance matching transformer or, for frequencies above 16 kHz, an amplifier (Crown D75), and finally to either a 12-inch (30.5 cm) loudspeaker (for frequencies from 62 Hz to 2 kHz) or a piezoelectric tweeter (for frequencies above 2 kHz). The speakers were located at ear level 1 m in front of the animals. This sound system proved capable of delivering undistorted tones from 62 Hz to 64 kHz at an intensity of at least 75 dB in the vicinity of the water cup.

The sound system was calibrated and the sound pressure levels (expressed throughout as decibels re 20 $\mu N/m^2$) were measured with either a 1-inch (2.54 cm) microphone (Bruel and Kjaer 4131), sound level meter (B & K 2203), and octave filter (B & K 1613), or a 0.25 inch (0.64 cm) microphone (B & K 4135), preamplifier (B & K 2618), microphone amplifier (B & K 2608), and filter (B & K 1613 or Krohn-Hite 3202). Sound pressure measurements were taken by placing the microphone in the region normally occupied by an animal's ears when drinking from the cup and pointing it directly toward the loudspeaker. Care was taken to ensure that the sound field was relatively homogeneous in the area occupied by the animal.

Psychophysical procedure

The method of conditioned suppression used here is a modification of that described elsewhere (Heffner et al., '71). Briefly, a thirsty animal was trained to make steady contact with the water bowl with its mouth, an action which fixed its head in front of the loudspeaker. This was accomplished by providing a steady trickle of water (2–4 cc per minute) as long as the animal maintained contact with the bowl. Once an animal had learned to drink steadily, auditory training was begun. This consisted of presenting an obviously suprathreshold tone for 10 seconds and following it, at its offset, with a mild electric shock delivered to the feet. After a few tone-shock pairings, the animal ceased drinking at the onset of a suprathreshold tone. This cessation of contact with the bowl, then, was used as an indication that the animal detected the tone.

The test procedure consisted of presenting 10-second trials with a 2-second intertrial interval (i.e., one trial every 12 seconds). Each trial was either a "safe" trial during which no tone was presented or a "warning" trial which consisted of a 10-second train of tone pulses. Warning trials occurred randomly from one to ten trials after the previous warning trial and were followed by shock.

For the purpose of quantifying an animal's response, the duration of bowl contact was measured in 0.1-second increments at the beginning of a safe or warning trial until 10 seconds later at the end of the trial. A score of zero thus indicated perfect suppression while a score of 100 indicated no suppression. The scores were averaged separately for the silent or safe (S) trials and the tone or warning (W) trials for each frequency at each intensity. A measure of discrimination was then expressed in the form of a ratio, (S−W)/S, for each stimulus intensity, where S is the average score of the safe trials and W is the average score of the warning trials. In trained animals, this measure varies from near zero (failure to detect the tone) to one (perfect detection). To reduce the effects of occasional pauses in drinking, the results of a trial were automatically discarded if the animal was not in contact with the bowl at any time during the 1 second immediately preceding the trial (though the trial was presented as usual). Because this criterion was applied equally to safe and warning trials, it did not bias the results.

Auditory thresholds were determined for each frequency at octave intervals throughout the animal's hearing range

by reducing the intensity of the tone in 5-dB steps until the animal could no longer distinguish tone trials from silent trials. Threshold was defined as the lowest intensity that could be detected above a chance level (Mann-Whitney U), which was usually a suppression ratio of 0.30. Testing was considered complete when thresholds obtained in two different sessions were within 3 dB of each other.

RESULTS

Brachium of the inferior colliculus

Anatomical results. The lesions and associated degeneration for the four dogs with lesions of the brachium of the inferior colliculus and parts of the medial geniculate are shown in Figures 1 and 2. For comparison, the lesions have been mapped onto standardized coronal sections approximately 1 mm apart. Though the lesions varied noticeably in their medial extent and their encroachment into the medial geniculate body, the brachium of the inferior colliculus was completely severed in all four animals. As shown in Figure 1, the brachium of D-77 was severed with the lesion extending medially on both sides to the central gray and interrupting the underlying "central acoustic tract" of Papez (Galambos et al., '61; Henkel, '83; Morest, '65; Papez, '29, '36). In addition, the caudal tip of the medial geniculate and the deep layers of the superior colliculus were damaged bilaterally. The lesion in D-79, in addition to severing the brachium, also included the region of the central acoustic tract though it did not reach the central gray. Though the caudal extent of the medial geniculate was damaged on the left side, the medial geniculate was spared on the right side.

The lesions in D-71 and D-80 (Fig. 2), though penetrating less medially on the right side than in the previous two cases, also completely severed the brachium. The caudal tip of the medial geniculate in D-71 was damaged on both sides while the medial geniculate in D-80 was extensively damaged bilaterally. In addition, D-80 sustained some damage to the lateral geniculate and mild degeneration in the posterior pulvinar.

In summary, the brachium of the inferior colliculus was completely severed on both sides in all four cases. The bilateral extent of the lesions ranged from deep lesions which sectioned the tracts medial to the brachium (D-77 and D-79) to shallow lesions which spared the medial tracts (D-71 and D-80). In addition, two of the cases involved little (D-71) or no (D-79) damage to the medial geniculate on the right side. As will be seen, in spite of the variation in medial extent and involvement of the medial geniculate, all four of the animals showed the same deficit in sensitivity.

Behavioral results. After surgery, all the animals demonstrated the ability to respond to loud sounds and soon became reliable observers in the test situation. However, once threshold testing began, it became obvious that their hearing was not normal.

The postoperative audiograms of the four animals are illustrated in Figure 3 along with the average audiogram of four normal dogs which had been tested in a previous study (Heffner, '83). As the figure shows, the effect of the lesions was to "flatten" the audiograms. That is, the animals showed a frequency-dependent hearing loss with the largest losses occurring in the midrange of the audiogram where normal dogs are most sensitive (4–16 kHz). At the extreme ends of the audiogram (63 Hz and 45 kHz), the animals showed virtually normal hearing. However, from 250 Hz to 16 kHz, none of the operated dogs were able to perform within the normal range of hearing.

The degree of individual variation shown by the four operated animals does not seem to be related to differences in the lesions and, indeed, their variation was no larger than that found among normal dogs. In particular, the animals with lesions extending into the central acoustic tract (D-77 and D-79) do not noticeably differ from the other two animals in which this tract was spared. Furthermore, the involvement of the medial geniculate does not appear to be a factor because the hearing of D-79, in which the right medial geniculate was spared, does not differ appreciably from that of D-80, whose lesion included much of the medial geniculate. The existence of a hearing loss, then, appears to be due to the fact that the brachium of the inferior colliculus was transected in each case.

Considering the operated animals as a group, then, the average thresholds differed from the normal average by as little as 4 dB at 63 Hz to as much as 37 dB at 8 kHz. When averaged across octave frequencies, the animals showed an overall hearing loss of 22 dB. Thus, it appears that severing the brachium of the inferior colliculus bilaterally results in a moderate, but consistent, hearing loss.

Medial geniculate

The brachium of the inferior colliculus constitutes the primary pathway for auditory fibers from the inferior colliculus to the medial geniculate. Since lesions of the brachium result in a hearing loss, it would be expected that lesions of the medial geniculate would produce a similar hearing loss. The question arises, however, as to whether the entire medial geniculate must be ablated or whether a deficit can be produced by a restricted lesion.

The following three cases illustrate the effect of medial geniculate lesions on absolute thresholds. Because each of the animals received a different lesion with a different behavioral consequence, each case is discussed separately.

D-2. The lesion in D-2 (Fig. 4) was bilaterally symmetrical and restricted almost entirely to the medial geniculate body. It included the entire ventral division, nearly all of the dorsal division and magnocellular region, most of the caudal tip, and approximately half of the posterior group. The audiogram of D-2 (Fig. 5) reveals the presence of a hearing loss similar in magnitude to that shown by the brachium cases (cf., Figs. 3, 5). Because this animal was not tested at the extreme high and low frequencies, it is not known whether its hearing loss was smaller at those frequencies, as was the case with brachium lesions. However, from the available results, it appears that D-2 experienced a hearing loss which was similar in magnitude to that displayed by the animals with bilateral brachium lesions.

D-76. The case of D-76 illustrates the result of a large lesion of the medial geniculate which nevertheless spared some part of every subdivision except the caudal tip. The smallest lesion in D-76 involved less than a quarter of the ventral division, only half of the dorsal division (on the left side), all of the caudal tip, less than a quarter of the magnocellular region, less than a quarter of the posterior group (on the left side), and half or more of the lateral geniculate (Fig. 4). Despite the substantial size of the lesion and the fact that every subdivision of the medial geniculate was damaged bilaterally, behavioral testing revealed that this animal had normal thresholds at 250 Hz, 2 kHz, and 16 kHz (Fig. 5). Because the animal showed no sign of abnor-

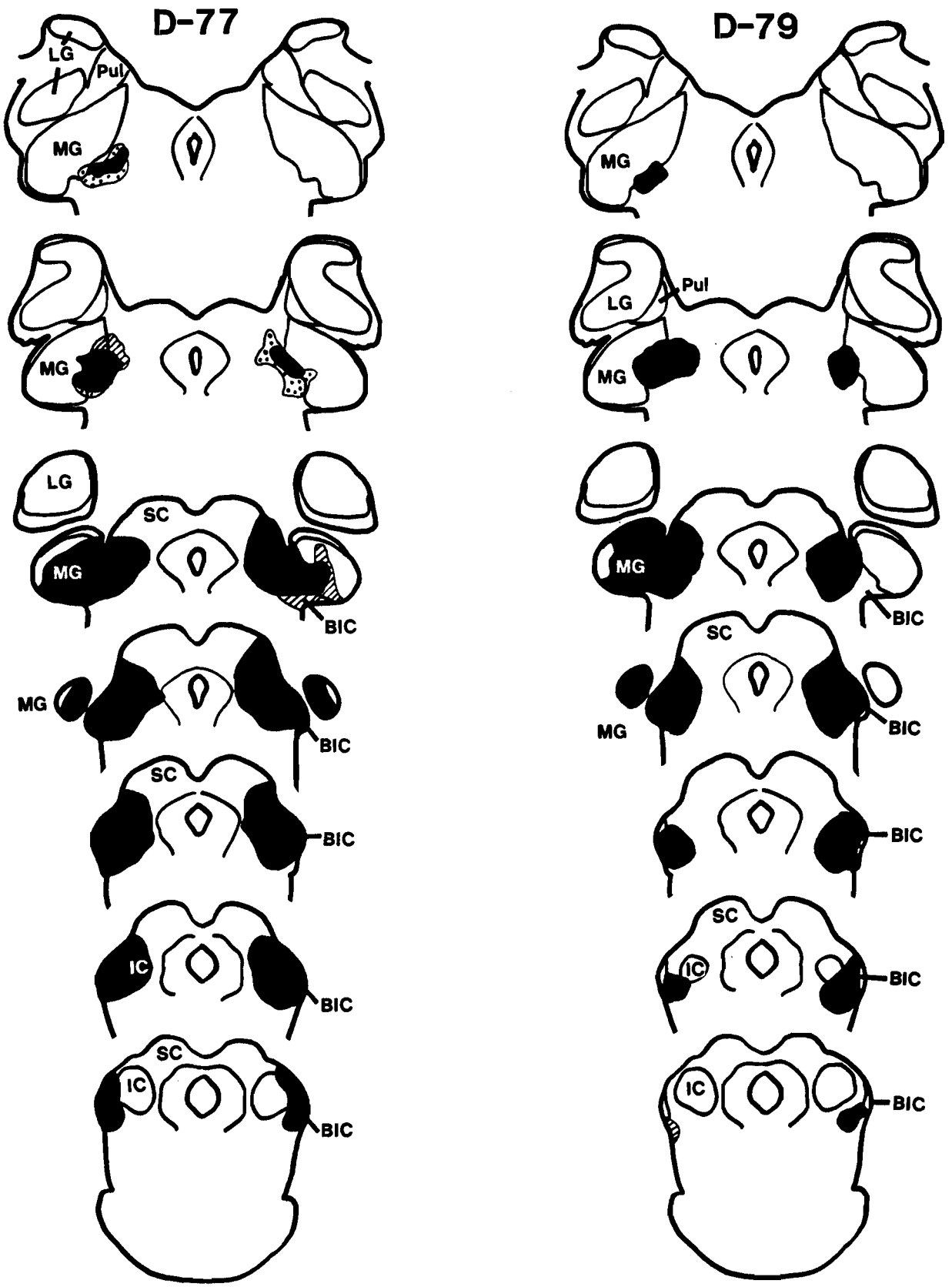

Fig. 1. Reconstruction of the lesions in D-77 and D-79. The lesions in these two cases severed the brachium of the inferior colliculus and the pathways medial to the brachium. Note that the medial geniculate was spared on the right side in D-79. In this and following figures, the black areas denote all cells missing, hatching denotes severe damage (less than 25% of cells remaining), and stippling denotes moderate damage (25–50% of cells remaining). BIC, brachium of the inferior colliculus; IC, inferior colliculus; LG, lateral geniculate; MG, medial geniculate; PO, posterior group; Pul, pulvinar; SC, superior colliculus; VPL, ventroposterolateral nucleus.

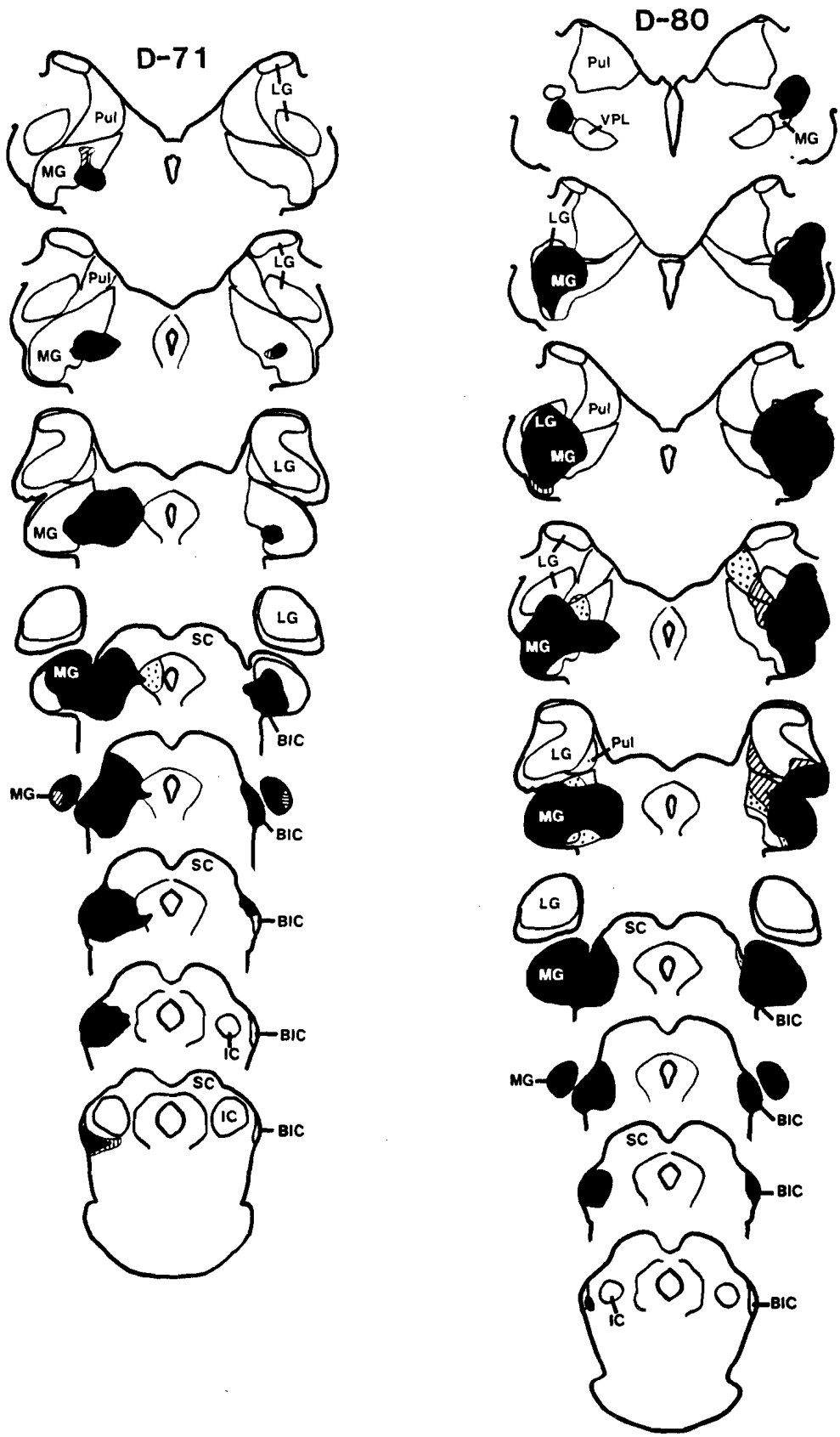

Fig. 2. Reconstruction of the lesions in D-71 and D-80. The lesions in both cases severed the brachium of the inferior colliculus while sparing the fibers medially on the right side.

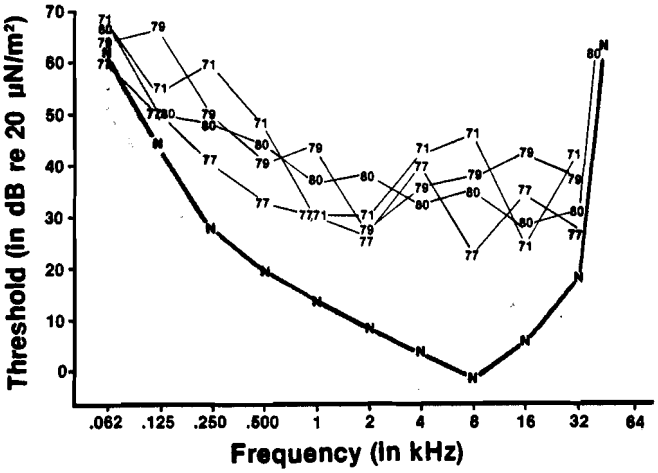

Fig. 3. Audiograms of four dogs with complete section of the brachium of the inferior colliculus (D-71, D-77, D-79, and D-80) compared with the average audiograms of four normal dogs (N). Shaded area indicates the range of variation in the normal dogs. Note that the hearing loss in the operated dogs was greatest in the midfrequencies with their thresholds at very high and low frequencies falling at or near normal levels.

mality at points in either the low-, or the middle-, or the high-frequency region of its hearing range, it was judged to have normal hearing and testing was discontinued. While it is not impossible that the animal might have suffered a loss at intermediate frequencies, it should be noted that testing at these three points would have easily revealed the deficits shown by the animals with the brachium lesions as well as the previous medial geniculate case (D-2). The lack of a hearing loss in D-76, then, appears due to the fact that significant portions of each subdivision, including the ventral division, were spared. Taken together with the results of D-2, the results of this animal indicate that a large lesion of the auditory thalamus does not result in a hearing loss unless certain critical areas are involved. Just which areas these may be is suggested by the next case.

D-52. The lesion in D-52 (Fig. 6) involved all of the ventral division on the left side, but spared the posterior and medial portion of the ventral division on the right side. The dorsal division and the posterior group were nearly entirely destroyed while part of the caudal tip on the right side and one quarter of the magnocellular region on both sides were spared. In addition, the lateral geniculate was damaged on both sides.

The audiogram for D-52 (Fig. 5) shows that the animal had normal hearing in the low- and midfrequency range of its audiogram (i.e., from 250 Hz to 8 kHz). However this animal showed a distinct high-frequency hearing loss at the two higher frequencies, 16 kHz and 32 kHz. At 16 kHz the animal's threshold was outside the range of hearing for normal dogs and within the range for animals with lesions of the brachium of the inferior colliculus (cf., Figs. 3, 5). At 32 kHz, the animal's threshold of 59 dB was 18 dB higher than the threshold of any of the brachium cases at that frequency. Thus, this animal had a high-frequency hearing loss which equaled or exceeded that shown by the animals with brachium lesions.

Given the restricted nature of the deficit, the question arises as to whether this high-frequency hearing loss can be related to the locus of the lesion. To answer this question, it is first necessary to compare D-52's lesion with that of D-2, which showed a hearing loss throughout its hearing range. A comparison of these two lesions (Figs. 4, 6) reveals three subdivisions in which the lesion in D-2 was larger than that of D-52. First, the entire ventral division was destroyed in D-2 while part of it was spared on the right side in D-52. Second, the magnocellular division was almost completely destroyed in D-2 while about a quarter of it was spared bilaterally in D-52. Finally, slightly more of the caudal tip on the right side of D-52 was spared than was spared on either side in D-2.

Of these differences, we have several reasons for suspecting that it may be the extent of the lesion into the ventral division which accounts for the observed deficits. First, while the lesion in D-2 involved more of the magnocellular region and the caudal tip, neither of these areas was completely destroyed in either animal. Only in the ventral division do we have the situation in which an area was completely destroyed in D-2 and partially destroyed in D-52. Second, of these three areas, the ventral division is considered to be the principal auditory division in the medial geniculate as it is the primary target of fibers from the central nucleus of the inferior colliculus and it appears to receive only auditory innervation (e.g., Harrison & Howe, '74; Winer et al., '77).

Finally, it appears that the locus of the lesion in the ventral division of D-52 and the resulting high-frequency deficit is consistent with the tonotopic organization of that division. In particular, the area of the ventral division which was spared on the right side was the medial and ventral portion of the caudal aspect. In cats, high-frequency units (i.e., 16 kHz and above) are generally found more rostrally and dorsally (e.g., Aitkin et al., '81; Imig and Morel, in press). While the area of the ventral division which was spared is known to contain low- and midfrequency units, it appears to contain few high-frequency units. Thus, it is possible that the high-frequency hearing loss in D-52 resulted from a lesion which destroyed all the high-frequency areas of the ventral division while sparing a portion of the low- and midfrequency areas.

DISCUSSION

Hearing loss following lesions of the brachium of the inferior colliculus

The results show that bilateral ablation of the brachium of the inferior colliculus in dogs results in a consistent impairment in the ability to detect sound. This hearing loss is frequency dependent with the largest threshold shift occurring in the midrange of an animal's audiogram while thresholds at the extreme high- and low-frequency ends of the audiogram appear to be normal or near normal. Thus, while the average threshold shift was 22 dB, the average thresholds ranged from 4 dB above normal (at 63 Hz) to 37 dB above normal (at 8 kHz).

Nonsensory factors as potential explanations. Because it has previously been suggested that lesions of the brachium have an effect on the ability to attend to sounds (Jane et al., '65) or a loss in their emotional significance (LeDoux et et al., '84), the question arises as to whether the hearing loss seen here is the result of an attentional or some other nonsensory deficit.

While it is entirely possible that the animals suffered nonsensory impairments as a result of the lesions, such impairments would not seem to account for the present results. First, the hearing loss shown by these animals was frequency dependent with thresholds being elevated at some

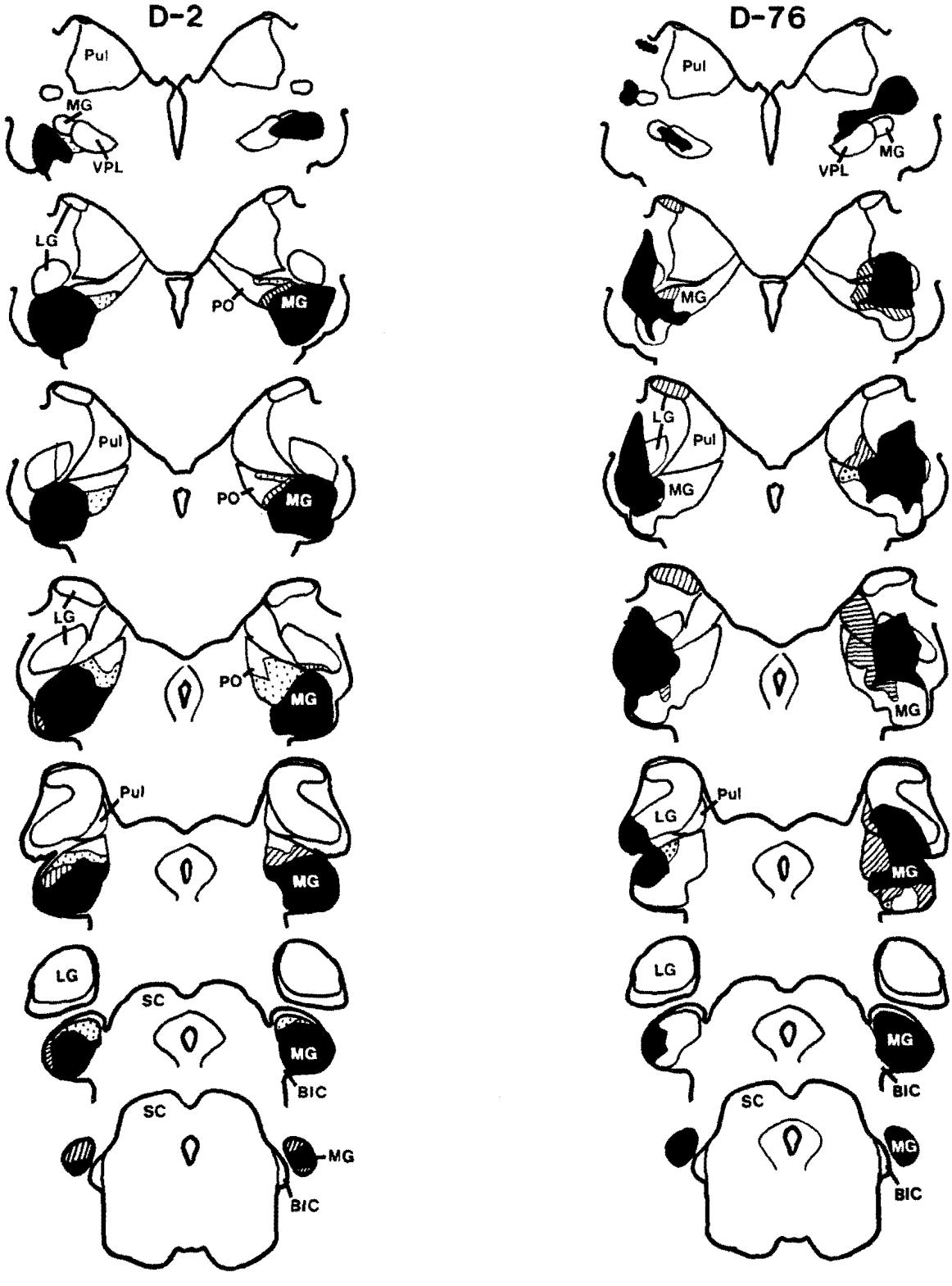

Fig. 4. Reconstruction of the lesions in two dogs with medial geniculate lesions.

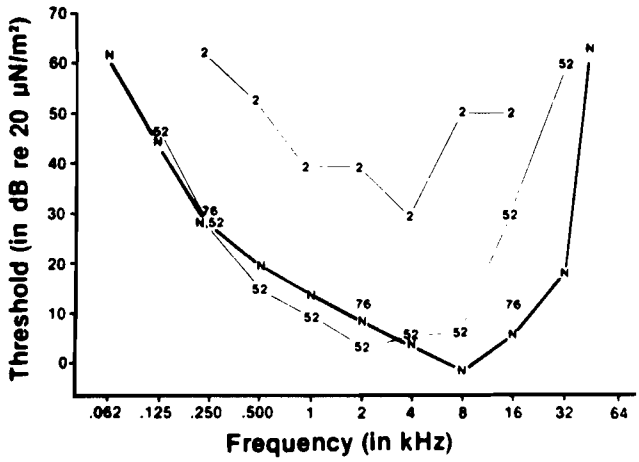

Fig. 5. Audiograms of three dogs with medial geniculate lesions (D-2, D-52, and D-76) compared with the normal dog audiogram. Note the high-frequency hearing loss of D-52.

frequencies, but not at others. If the animals had suffered a simple deficit in attention or emotional responsiveness to sound, it might have been expected to affect all frequencies similarly, with the result that their audiometric curves would have paralleled those of normal dogs. Second, a deficit in attention could potentially lead to increased variability in an animal's threshold either within a session or between sessions. That is, an animal's attention to sounds might fluctuate over time. However, we noticed no unusual variability in the animals' intra- or intersession performance that would suggest an unusual difficulty in attending to the sounds. Thus, the frequency-dependent nature of the hearing loss coupled with the lack of variation in the animals' performances make it difficult to account for the deficit in terms of loss of attention or emotional significance of the sounds.

Previous studies of absolute sensitivity following brachium lesions. In reviewing the literature concerning the effect of brachium lesions on hearing, it soon becomes apparent that few studies have examined the effect of such lesions on absolute sensitivity. Indeed, there are only two reports in which any absolute thresholds were determined and neither of these measured the animals' hearing throughout their entire hearing range. The first report on the effect of brachium lesions is the study by Kryter and Ades ('43) in which absolute thresholds were obtained for cats at 125 Hz, 1 kHz, and 8 kHz. In their study they reported that three cats with lesions confined primarily to the brachium of the inferior colliculus had small threshold shifts that ranged from a few decibels to about 19 dB with the largest losses occurring at 125 Hz. Two additional animals with brachium lesions had average losses of 43–45 dB with the largest threshold shifts occurring at 8 kHz. However, because the lesions in these latter cases also involved either the superior colliculus or the anterior part of the inferior colliculus, they were not considered as illustrating the effect of brachium lesions alone.

It is possible that the differences between the results of Kryter and Ades and the present results are due to differences in the placement of the lesions. That is, their smaller brachium cases may have been incomplete and the deficits

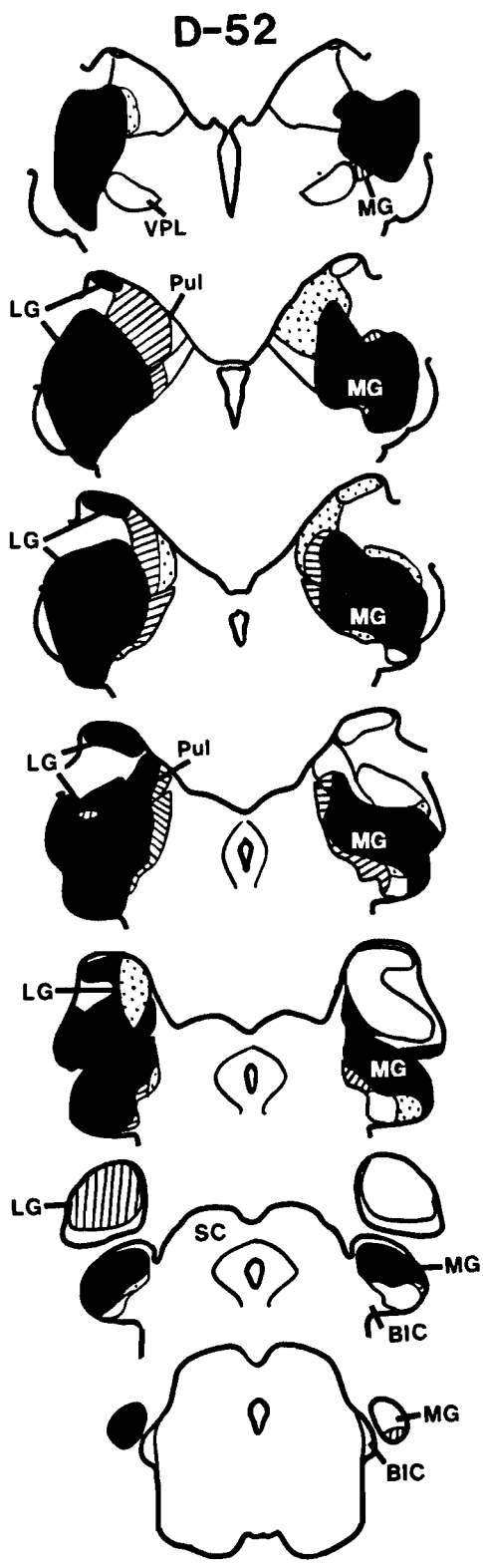

Fig. 6. Reconstruction of the medial geniculate lesion in D-52. This animal had a high-frequency hearing loss.

observed in their larger cases may not have been due to the encroachment of the lesion into adjacent areas. Indeed, our present results indicate that extending a lesion into the superior colliculus does not have an added effect on hearing (cf., D-77 vs. D-71 and D-80, Fig. 3). However, as Kryter and Ades did not publish illustrations of the lesions, it is difficult to account for their conclusion that brachium lesions have little effect on abolute thresholds.

The other study which determined absolute thresholds following brachium lesions is that of Jane and his colleagues (Jane et al., '65) in which the threshold for a 300-Hz tone was determined in a single cat following bilateral brachium lesions. Their results showed that the animal's threshold at 300 Hz was only 5 dB above that of a normal animal. This result, however, does not differ from the present results: because the hearing loss described here is quite small at low frequencies, a large loss at 300 Hz would not have been expected. Indeed, in our test, one brachium animal (D-77) was within 1 dB of the normal hearing range at 250 Hz (cf., Fig. 3). As a result, there appears to be no significant discrepancy between our findings and those of Jane and his colleagues.

Relation of current results to previous studies of brachium lesions

Over the past 20 years, a small number of studies have investigated the effects of brachium lesions on other aspects of audition. These studies have included tests of intensity discrimination, frequency discrimination, sound localization, and auditory attention. At the time these studies were performed, it was generally believed that brachium lesions had little if any effect on absolute thresholds. It is therefore of interest to reexamine these studies to determine whether their results could be interpreted as being due to an absolute threshold shift.

Intensity discrimination. The ability of cats to discriminate differences in the intensity of a 1-kHz tone (65 dB SPL) following bilateral brachium lesions was investigated by Oesterreich and his colleagues, ('71). They found that brachium lesions increased the cats' intensity-difference thresholds from a normal threshold of 2 dB to 12 dB. Because the loudness or sensation level of a tone is an important factor in an intensity discrimination, they discussed the possibility that their results may have been due to a hearing loss. As they noted, a hearing loss even on the order of that demonstrated in the present article would be expected, on the basis of human studies, to raise the thresholds to no more than about 4 dB (i.e., to double the normal thresholds). Thus, it would appear that the increase in intensity-discrimination thresholds which they observed was primarily a direct effect of brachium lesions and not an indirect result of a hearing loss.

Frequency discrimination. The ability of cats to discriminate 800 Hz from 1 kHz following bilateral brachium lesions was assessed by Goldberg and Neff ('61). They found that complete brachium lesions impaired the frequency discrimination while deep lesions which included the tracts medial to the brachium entirely abolished the ability to discriminate frequencies. While it is possible that a hearing loss might have affected their results, it appears that their animals probably had a specific frequency discrimination deficit. First, though the intensity of the sounds was not specified, Goldberg and Neff did demonstrate that the operated animals were still able to detect the two tones. Second, while reducing the intensity of the sounds to near threshold does raise frequency discrimination thresholds slightly (e.g., Shower and Biddulph, '31), the frequency difference of 200 Hz used in their study is well above the normal cat threshold of 8 Hz at 1 kHz (Elliot et al., '60) and should have been easily detected even at a reduced intensity level. Therefore, it does not seem likely that their results can be considered as an indirect result of a hearing loss.

Sound localization. One of the most consistent deficits reported after brachium lesions is an impairment in the ability to localize sound. Bilateral section of the brachium in cats appears to result in elevated sound localization thresholds as well as poorer performance at angles above threshold (Casseday and Neff, '75). In addition, when tested with headphones, cats with brachium lesions appear to have lost the ability to use the binaural time-difference cue to localize sound while retaining the ability to use the binaural intensity-difference cue (Masterton et al., '68). Furthermore, larger lesions of the brachium which involve the pathways medial to the brachium and thus totally deprive the forebrain of auditory input result in a complete inability to localize sound or to use either the time- or intensity-difference cue (Casseday and Neff, '75; Masterton et al., '68; Strominger and Oesterreich, '70).

While the control tests used in these studies rule out the possibility that the observed localization deficits could have been due to total deafness, both studies suggest that their animals did have a noticeable hearing loss. In the study by Masterton and his colleagues, both of the two brachium cases had difficulty in learning to detect the stimuli delivered to the right ear against background of silence though both eventually proved capable of perfect performance on this task. Similarly, one of the brachium cases in the study by Casseday and Neff required the intensity of the localization stimulus to be increased by 20 dB before it could be localized at large angles. Thus, while the results of these two studies suggest that the cases with brachium lesions may have suffered a hearing loss, they also demonstrated a deficit in localizing sounds which were audible.

Hearing loss following medial geniculate lesions

Because the brachium of the inferior colliculus is the primary pathway between the inferior colliculus and the medial geniculate, it would be expected the bilateral lesions of the medial geniculate would result in a hearing loss similar to that noted following brachium lesions. The results of the present study indicate that medial geniculate lesions do indeed result in such a deficit. Specifically, it appears that bilateral lesions of the medial geniculate which include the entire ventral division result in a hearing loss of a magnitude similar to that found following brachium lesions. In addition it appears that partial lesions of the ventral division of the medial geniculate may result in a hearing loss at some frequencies, but not at others.

The idea that the medial geniculate is tonotopically organized in such a way that partial lesions result in a hearing loss at different frequencies is not new. Some years ago, Ades and his colleagues presented evidence that small, localized lesions of the medial geniculate in cats produced hearing losses at different frequencies depending on the location of the lesion (Ades et al., '39). Unfortunately, they did not illustrate the lesions and the brief verbal descriptions do not lend themselves to a detailed analysis. Though Ades later discounted these results because the audiograms had been completed within 2 weeks after surgery and,

therefore, the hearing losses may have been transient ones (Kryter and Ades, '43), it is of interest to note that their observation that high-frequency losses occurred with dorsal lesions while lower-frequency losses occurred with more ventral lesions coincides with the tonotopic map in the ventral division of the medial geniculate (e.g., Aitkin et al., '81).

Role of auditory cortex in hearing

The primary source of auditory input to neocortex is the medial geniculate body which sends fibers via the auditory radiation to auditory cortex. So close is the relationship between the medial geniculate and auditory cortex that ablation of all primary and secondary auditory cortex in carnivores results in severe degeneration of the medial geniculate body (e.g., Harrison and Howe, '74; Neff et al., '75). Given this relationship, the question arises as to whether bilateral ablation of auditory cortex would result in a hearing loss similar to that found following large medial geniculate lesions. Indeed, the fact that large auditory cortex lesions result in the degeneration and consequent disappearance of virtually all of the neurons in the medial geniculate would seem to require that such lesions produce the same deficit as that produced by direct medial geniculate lesions.

Over the last 100 years, a number of studies have addressed the question of whether auditory cortex ablation results in a hearing loss (for reviews, see James, 1890; Kryter and Ades, '43; Neff etal., '75). Though the results have been conflicting, the general opinion which has emerged is that auditory cortex ablation has little or no permanent effect on the detection of sound (Neff et al., '75). Thus, we are left with the problem of reconciling the observed hearing loss following medial geniculate lesions with the apparent lack of such a loss following auditory cortex ablation.

There are at least two possible explanations for this apparent difference. The first is that the thalamic degeneration produced by auditory cortex lesions is not equivalent to lesions made directly in the medial geniculate. For example, while large auditory cortex lesions result in severe degeneration in the medial geniculate, there are usually a few scattered cells which survive. In addition, medial geniculate lesions may affect adjacent fibers or cells which cortical lesions would leave intact. Furthermore, it has been suggested that severing the fibers from the inferior colliculus, which would occur in the case of a medial geniculate lesion, could result in a deficit because it affected the response properties of the cells in the inferior colliculus rather than because it severed the auditory input to the cortex (Neff et al., '75). Thus, medial geniculate lesions could result in a larger deficit than auditory cortex lesions because they produce more disruption at subcortical levels.

On the other hand, a second possible explanation is that auditory cortex ablation does result in a hearing loss. Indeed, there are several reasons for suspecting that such a deficit does occur. First, it is now known that bilateral auditory cortex ablation in both humans and monkeys results in a permanent hearing loss of about the same magnitude as observed in the animals in the present study (e.g., Jerger et al., '69, '72; Heffner and Heffner, '84). Thus, in higher primates, at least, cortical ablation does result in a hearing loss.

Second, in examining the literature it soon becomes apparent that there has been no complete study concerning the effect of cortical ablation on hearing in either cats or dogs. In particular, many of the older studies did not report the resulting thalamic degeneration which is necessary to determine the completeness of the lesion (cf., Kryter and Ades, '43). Furthermore, most studies have determined thresholds at only two or three frequencies with no study examining an animal's entire hearing range.

Finally, though several studies have reported normal thresholds following cortical ablation in carnivores, an equal number have found a hearing loss. Studies which have reported no hearing loss include Kryter and Ades ('43), unpublished work by both Stroughton and Wegener (cited by Neff et al., '75), and studies by Baru ('71) and Cranford ('79). On the other hand, significant hearing losses in cats or dogs following bilateral cortical ablations have been found by Mettler and his colleagues (Mettler et al., '34), Lipman (cited by Kryter and Ades, '43), Girden ('42), Maruyama and Kanno ('61), and Cranford and Igarashi ('77).

In attempting to reconcile the conflicting reports on the effect of cortical lesions on hearing, we currently favor the idea that a complete bilateral auditory cortex lesion may result in a small, but nonetheless significant, hearing loss in cats or dogs. Those studies which have failed to find such a deficit may have included only incomplete lesions or else may have included tests only at lower frequencies where, based on the present results, the loss would have been small and easily missed. Furthermore, the recent discovery that large bilateral auditory cortex lesions affect auditory thresholds in primates gives rise to the possibility that this deficit may also occur in other species.

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