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Hearing Loss in Japanese Macaques Following Bilateral Auditory Cortex Lesions

HENRY E. HEFFNER AND RICKYE S. HEFFNER

Laboratory of Comparative Hearing, Bureau of Child Research, University of Kansas, Parsons, Kansas 67357

SUMMARY AND CONCLUSION

1. The hearing ability of five Japanese macaques (*Macaca fuscata*) was assessed following two-stage bilateral auditory cortex lesions. The animals were tested using a shock-avoidance procedure with a conditioned-suppression procedure used for comparison in two cases.

2. The animals initially were unable to respond to sound, and the first signs of hearing appeared as late as 13 wk after surgery. Hearing levels improved gradually over time, with maximal recovery reached at 24–35 wk after surgery. Recovery was most pronounced for low frequencies (63–250 Hz) and very high frequencies (32 kHz), which generally returned to normal or near-normal levels. However, the monkeys appeared to have suffered a permanent hearing loss throughout most of their hearing range, especially in the midfrequency range, where they are normally most sensitive.

3. A review of the animal literature reveals little support for the previous view that bilateral auditory cortex lesions have little or no effect on absolute sensitivity in primates and carnivores. Most previous studies did not conduct detailed hearing tests, and those that did often noted a hearing loss.

4. The hearing loss found in monkeys is similar to that noted in human cases following bilateral auditory cortex lesions. The current findings thus provide experimental verification of the clinical phenomenon of cortical deafness.

INTRODUCTION

Over the last 100 years, a number of studies have addressed the question of whether ablation of auditory cortex in animals results in a hearing loss (for reviews, see Refs. 29, 35, 43). Though the results of the various studies have at times been conflicting, the general opinion that has emerged is that auditory cortex ablation has little or no permanent effect on the detection of sound (43). In contrast, numerous clinical reports of stroke cases have indicated that bilateral lesions of the temporal lobe in humans often result in a noticeable hearing loss (e.g., Refs. 1, 9, 17, 18, 31, 36, 41, 42, 45, 56). Yet in spite of these reports, the idea of a cortical hearing loss in humans does not seem to have found universal acceptance, due in part to the lack of supporting evidence in the animal literature (e.g., Refs. 2, 7, 50).

Recently, we have had the opportunity to assess the perceptual hearing abilities of Japanese macaques before and after bilateral ablation of auditory cortex (22). In the course of this study we discovered that the lesions not only affected the animals' auditory perceptual abilities but also resulted in a substantial hearing loss. The results of this study constitute the first clear evidence that bilateral auditory cortex lesions result in a hearing loss in macaques.

METHODS

The seven monkeys used in this experiment were part of another study involving the effect of cortical ablation on the perception of primate vocalizations (22). The postoperative free-field audiograms of five of the monkeys and the audiograms of two normal monkeys were determined with an avoidance procedure similar to that used elsewhere (22). The method of conditioned suppression (27) was briefly used for comparison in two of the operate cases.

Subjects

Seven male Japanese macaques (*Macaca fuscata*) ranging in age from 5 to 7 yr (adolescent) were used in this study. The animals had been born and reared in a free-ranging colony (Arashiyama West Institute). The animals were individually housed in primate cages with free access to food and were trained with a water reward. The ears of each animal were examined during surgery and after death to ensure that they were free of damage or disease.

Surgical and histological procedures

SURGERY. Following preoperative training and testing involving the discrimination of primate vocalizations (22), five of the monkeys received two-stage lesions of the temporal lobes, with additional discrimination testing given between stages. For surgery, a monkey was initially anesthetized with sodium thiamylal (Bio-tal, Bio-ceutic) (18 mg/kg) and given 0.5 mg of atropine sulfate and 100 mg of Lincocin (Upjohn). This was followed by halothane administered via endotracheal cannula as needed to maintain deep anesthesia. The animal's head was shaved and washed, the scalp opened, and the temporal muscle on one side dissected with a cautery. With the edges of the temporal muscle retracted, the portion of the cranium overlying the sylvian fissure was removed, the dura retracted, and the cortical tissue of one hemisphere removed by subpial aspiration. Aseptic procedures were followed throughout surgery.

After removal of cortical tissue, the area removed was packed with Gelfoam (Upjohn) in order to minimize subsequent distortion of the gyri, and Gelfilm (Upjohn) was placed over the opening and under the edges of the cranium to minimize adhesions of the overlying tissue with the pia. The muscle was then sewn together, and a topical antibiotic powder (Neo-Predef, Upjohn) was sprinkled on top of the muscle. The scalp incision was closed with silk suture. The animal was returned to its home cage and was given acepromazine as needed to minimize discomfort.

The order in which each hemisphere was ablated and the elapsed time between stages were as follows: M-37, left lesion followed 35 days later by a right lesion; M-57, left lesion followed 36 days later by a right lesion; M-150, right lesion followed 29 days later by a left lesion; M-246, right lesion followed 109 days later by a left lesion; M-252, left lesion followed 64 days later by a right lesion.

HISTOLOGY. Following completion of behavioral testing, the monkeys were deeply anesthetized with pentobarbital sodium and perfused with isotonic saline followed by 10% formalin. The brains were removed, photographed to aid cortical reconstruction, and prepared for frozen sectioning. The brain was sectioned in the coronal plane at 40 μm and two sets of sections at 200-μm intervals were stained: one with thionin and one with Protargol (Sterling). These sections were then used to reconstruct the limits of the lesions and the resulting thalamic degeneration.

Procedure

BEHAVIORAL APPARATUS. A standard primate chair was modified to accommodate a "double" water spout. This spout consisted of two standard drink tubes mounted parallel and close enough (1 cm apart) so that a monkey could comfortably place its mouth on both spouts. The two spouts were electrically isolated from each other and were attached to a touch switch that detected when an animal placed its mouth on them. (Use of the double spout eliminated the need to tie the animal's foot to a footplate in order to provide a ground for the touch switch.) One of the spouts was attached via plastic tubing to an electrically operated water valve and constant-pressure water reservoir. Mild electric shock was provided by a shock generator connected to the two spouts. A 60-W light was mounted above the chair, and the entire apparatus was located in a double-wall sound chamber (2.7 $\times$ 2.5 $\times$ 2.0 m) the walls and ceiling of which were lined with egg-crate foam to reduce sound reflection. A microcomputer (Apple II) was used for behavioral programming.

ACOUSTICAL APPARATUS. Sine waves were produced by a generator (Hewlett-Packard 209A) and switched by a rise-fall gate (Coulbourn S84-04). The signal was pulsed two times per second (0.250 ms on 0.250 ms off, rise-decay of 50 ms for 63–500 Hz and 20 ms for higher frequencies) with the stimulus timing controlled by external timers (Coulbourn S53-21). The signal was then led to an attenuator (Hewlett-Packard 350B), an impedance matching transformer, or for 32 kHz, an amplifier (Coulbourn S82-24), and finally to either a 12-in. (30.5-cm) loudspeaker (for frequencies from 63–500 Hz), a 4-in. (10.2-cm) midrange loudspeaker (for frequencies from 1 to 4 kHz), or a ribbon tweeter (for frequencies from 8 to 32 kHz). The speakers were located slightly above ear level 1 m in front of the animal and angled down so that they pointed at the center of the animal's head.

The sound system was calibrated, and the sound pressure levels (re 20 $\mu N/m^2$) were measured with either a 1-in. (2.54-cm) microphone (Brüel and Kjaer 4131), sound level meter (B & K 2203), and octave filter (B & K 1613), or a 0.25-in. (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. Care was taken to ensure that the sound field was homogeneous.

Unless otherwise specified, the maximum intensities of the tones were as follows: 74 dB at 63 Hz, 74 dB at 125 Hz, 78 dB at 250 Hz, 73 dB at 500 Hz, 77 dB at 1 kHz, 74 dB at 2 kHz, 72 dB at 4 kHz, 69 dB at 8 kHz, 64 dB at 16 kHz, and 74 dB at 32 kHz.

Additional stimuli used were primate vocalizations and white noise. The vocalizations consisted of seven so-called "smooth early high" and eight "smooth late high" coos, which have been described elsewhere (3, 19). The coos were presented at either 55 or 70 dB above threshold as determined by reducing the intensity until the animals could no longer detect them. The white noise was presented at 75 dB SPL.

PSYCHOPHYSICAL PROCEDURE. In the avoidance procedure, a thirsty monkey was seated in the primate chair and trained to place its mouth on the water spout, an action which fixed its head in front of the loudspeaker. This was accomplished by providing a steady trickle of water (3–4 ml per min) as long as the animal maintained contact with the spout. Tones were then presented at random intervals and followed at their offset by mild electric shock delivered through the double water spout. Once the animal had learned to associate the tone with shock, it avoided the shock by breaking contact with the spout whenever a tone signaled impending shock. This cessation of contact was used to indicate that the animal detected the tone. The light above the primate chair was momentarily turned on after each warning trial to indicate that the shock had been delivered and that the animal could return to the spout.

The test procedure consisted of presenting 3.5-s trials with a 3.5-s intertrial interval (i.e., one trial every 7 s). Each trial was either a "safe" trial during which no tone was presented or a "warning" trial which consisted of a 3.5-s train of tone pulses. Warning trials generally occurred randomly from one to seven trials after the previous warning trial. Two precautions were taken to prevent an animal from using the time since the last warning trial as a cue that a warning trial might be about to occur. First, the number of warning trials given in each of the seven trial periods was adjusted so that each trial period had the same probability of containing a warning trial (which was 0.25). Second, "catch" trials, in which the seventh trial following a warning trial was a safe trial, were inserted so that the probability of the seventh trial being a warning trial was also 0.25.

The response of an animal on each trial, i.e., whether or not it had made an avoidance response, was determined by whether or not the animal was in contact with the spout during the last 200 ms of the trial. Thus, performance on each trial was dichotomized as either an avoidance response or no response. Basing the response criterion on the last 200 ms of the trial, as opposed to using a longer duration, had two important advantages. First, it allowed the animal sufficient time to react to the tone and break spout contact following tone onset. Second, requiring the animal to break contact for <1 s reduced the response cost to the animal (i.e., it only missed a small amount of water). As a result, a lower level of shock could be used to maintain reliable behavior than would have been necessary had a longer response time been required.

The details of determining spout contact were as follows: The duration of spout contact during the last 200 ms of each trial was measured in 20-ms increments. This generated a number from 0 to 10 where 0 indicated no contact, 10 indicated contact during all 10 of the 20-ms periods, and an intermediate number indicated intermittent contact. While nearly all trials resulted in a 0 or a 10, the ability to record intermediate scores was useful in that it served to indicate potential problems such as the use of marginally effective shock level. For the calculation of chance level, values of 0 to 4 were scored as an avoidance response, whereas values from 5 to 10 were scored as no response.

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 performance 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 ~0 (failure to detect the tone) to 1 (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 spout at any time during the 1 s 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 by reducing the intensity of the tone in 5-dB steps until the animal could no longer distinguish tone trials from silent trials. Once a preliminary threshold had been obtained, final threshold determination was conducted by presenting tones varying in intensity by 5-dB increments extending from 10 dB below to 10 dB above the estimated threshold. The trials at each intensity were presented in blocks of three to five warning trials, and the level of shock was varied to insure optimal performance. Threshold was defined as the lowest intensity that could be detected above the 0.01 level of chance, which was usually a performance ratio of 0.20. Chance level was calculated by comparing the occurrence of responses during the safe and warning trials using the binomial distribution (21). Testing was considered complete when thresholds obtained

in two different sessions were within 4 dB of each other.

For convenience, the animals' thresholds were plotted in terms of hearing loss. This was done by comparing their thresholds with an average audiogram for macaques derived by taking a weighted average of seven macaque audiograms (4, 6, 8, 14, 20, 47, 52). The resulting average audiogram (in SPL) was 40 dB at 63 Hz, 27 dB at 125 Hz, 17 dB at 250 Hz, 8 dB at 500 Hz, 2 dB at 1 kHz, 2 dB at 2 kHz, 3 dB at 4 kHz, −3 dB at 8 kHz, 3 dB at 16 kHz, and 25 dB at 32 kHz.

The conditioned suppression procedure, used for additional comparison in two of the animals, was conducted in the same manner as the avoidance procedure, except that the animals' feet were tied to two separate foot plates, and a warning trial was always followed by shock delivered to the feet.

RESULTS

Anatomical results

Details of the locus and subdivisions of auditory cortex have been studied in the rhesus macaque by microelectrode recordings (39), cytoarchitectural analysis (15, 46), and tracing thalamocortical connections (40). Because Japanese and rhesus macaques are closely related and their brains are similar in appearance, information from these studies is useful in locating auditory cortex in the Japanese macaque.

Though the exact boundaries of auditory cortex can be difficult to define, both cytoarchitectural and electrophysiological methods indicate that there is a central core area (koniocortex or primary auditory cortex) and a surrounding belt region of secondary auditory fields. Primary auditory cortex lies in the depths of the sylvian fissure on the superior temporal plane (Fig. 1). The locus of primary auditory cortex as determined by microelectrode recordings is coextensive with koniocortex (15, 39). This area has been shown to receive essential projections from the anterior two thirds of the parvocellular division of the medial geniculate (GMp) (40). Accordingly, ablation of primary auditory cortex results in severe degeneration of the anterior two thirds of GMp with no noticeable degeneration in the magnocellular division (GMmc) or in the suprageniculate nucleus (SG).

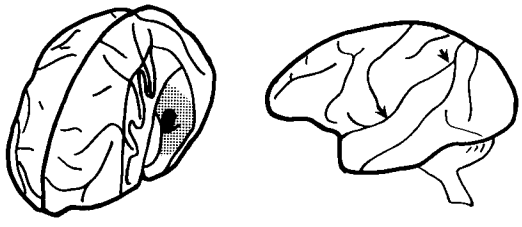

FIG. 1. Drawing on the *left* is of a Japanese macaque brain with the frontoparietal operculum removed to show the location of primary (*blackened*) and the surrounding belt of secondary (*stippled*) auditory cortex. More secondary auditory cortex is buried in the circular sulcus bounding the insula. Drawing on the *right* shows the lateral surface of a Japanese macaque brain. *Arrows* mark the approximate limits of the primary and secondary auditory fields on the superior temporal plane [based on the electrophysiological study of Merzenich and Brugge (39)].

The number and exact location of the secondary auditory fields as determined electrophysiologically is less well known. There appear to be four secondary fields surrounding primary auditory cortex that can be differentiated from it on both anatomical and physiological grounds (39). These fields correspond approximately to the secondary auditory fields of Pandya and Sanides (46). The thalamic projections to these surrounding fields are diffuse and overlapping (40) with the result that ablation of these areas alone results in only mild degeneration. A large lesion encompassing primary as well as these secondary surrounding fields results in severe degeneration throughout GMp, with the exception of the caudal tip, and partial degeneration of GMmc and SG.

Auditory responses have also been recorded on the superior temporal plane rostral to the secondary fields and on the lateral surface of the superior temporal gyrus suggesting that there are at least two other auditory fields (39). In addition, the cytoarchitectonics and cortical connections of the auditory fields suggest that the entire superior temporal gyrus can be considered auditory (15). Ablation of the superior temporal gyrus results in total degeneration of all of GMp including the caudal tip. Severe degeneration occurs in GMmc and SG, although those nuclei have sustaining projections to insula and the parietal operculum and do not completely degenerate (40, 43, 54).

EXTENT OF THE LESIONS. The thalamic degeneration and cortical reconstructions are illustrated for the largest (M-57, Fig. 2) and smallest (M-150, Fig. 3) lesions, while the cortical reconstructions are illustrated for the re-

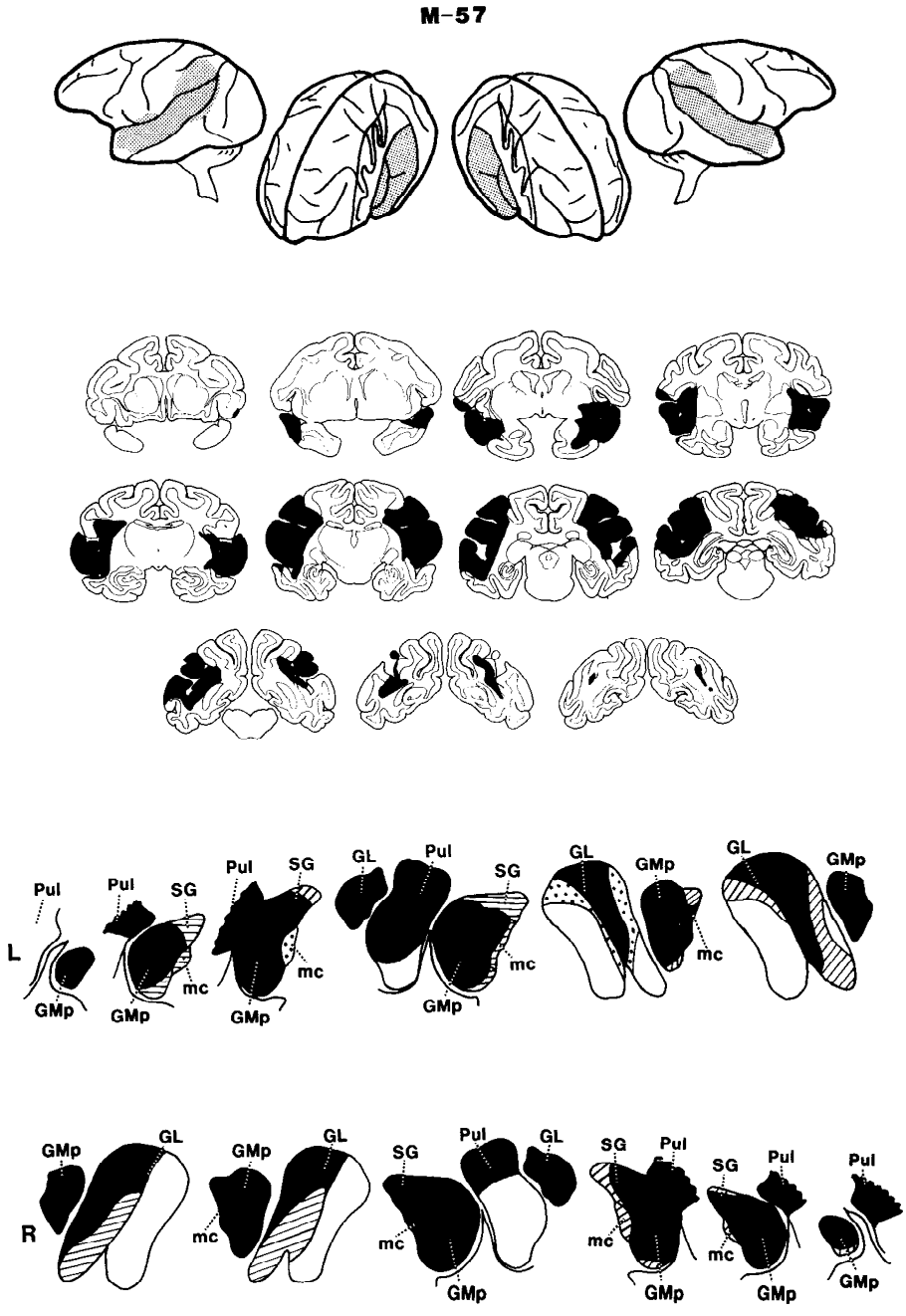

FIG. 2. Cortical reconstruction and medial geniculate degeneration of the monkey (M-57) with the largest lesion. The entire superior temporal gyrus was removed on both sides with the exception of the tip of the temporal pole, which was undercut. The lesions also included the posterior part of the middle temporal gyrus, insula, and part of the inferior parietal gyrus including the parietal operculum. The entire GMp was totally degenerated, while severe degeneration was found in GMmc and SG. In addition, mild to severe degeneration was observed bilaterally in the lateral, medial, and oralis nuclei of the pulvinar, and in the ventroposterior lateral nucleus and lateral geniculate. *Top*: reconstruction of cortical lesion showing surface views and coronal sections. Coronal sections are 3.6 mm apart. *Bottom*: retrograde degeneration in the vicinity of the medial geniculate. The left thalamic sections (*top*) are shown posterior to anterior, while the right sections (*bottom*) are anterior to posterior. Thalamic sections are 600 μm apart. GL, dorsal lateral geniculate; GMp, parvocellular division of the medial geniculate; mc, magnocellular division of the medial geniculate; Pul, pulvinar; SG, supragenicular nucleus. *Blackened area* indicates total degeneration, 95–100% cell loss; *hatched area* indicates severe degeneration, 70–95% cell loss; *stippled area* indicates moderate degeneration, 30–70% cell loss.

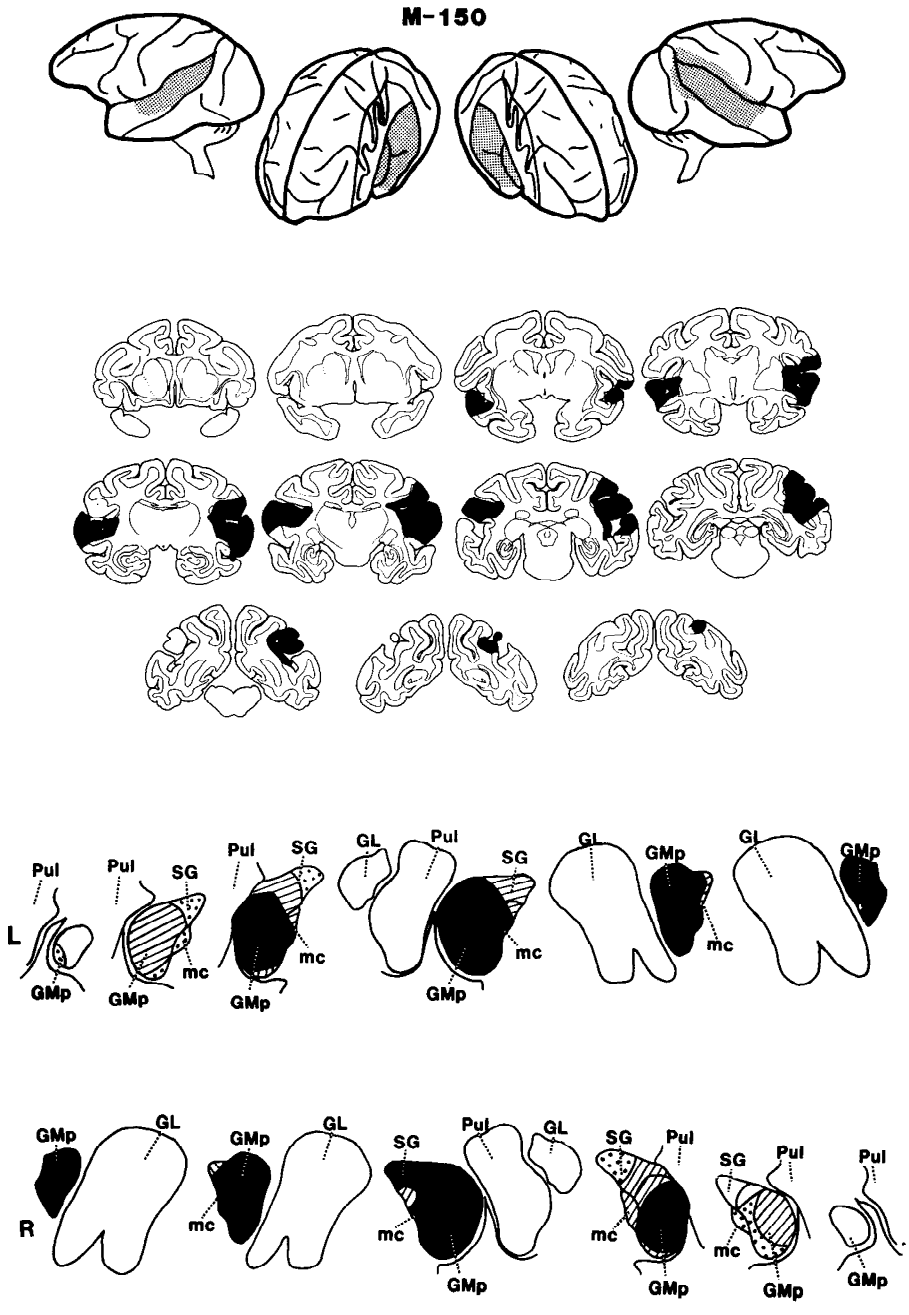

FIG. 3. Cortical reconstruction and medial geniculate degeneration of the monkey (M-150) with the smallest lesion. The left lesion was confined to the superior temporal gyrus and insula while the right included part of the parietal operculum, inferior parietal gyrus and posterior portion of the middle temporal gyrus. Both lesions were smaller than the other cases in that a significant portion of the temporal pole of the superior temporal gyrus was spared. As a result, the caudal third of GMp showed moderate to no degeneration. The anterior two thirds of GMp, however, underwent severe to total degeneration. Some degeneration was also observed in GMmc and SG, but all other thalamic nuclei appeared intact. See Fig. 2 for abbreviations.

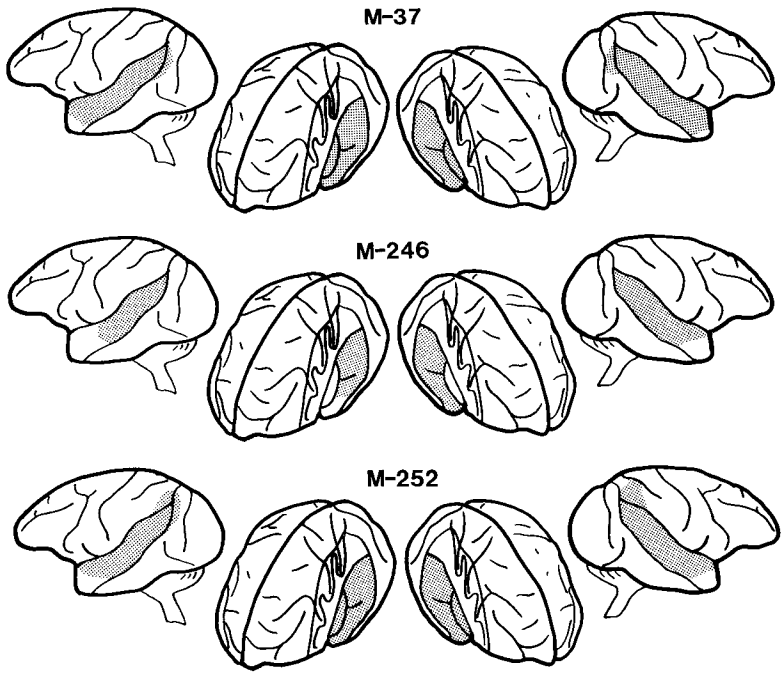

FIG. 4. Cortical reconstruction of the lesions of 3 monkeys. Area of a lesion is indicated by *stippling*. M37: the lesions included the superior temporal gyrus sparing only a small portion of the temporal pole ventrally. In the medial geniculate, the entire GMp was severely to totally degenerated on both sides including the caudal tip with mild to moderate degeneration in GMmc and SG. M-246: the lesions were confined to the superior temporal gyrus with the right lesion extending further anteriorly than the left. In the medial geniculate, GMp was completely degenerated on both sides with the exception of the caudal tip, which underwent moderate to total degeneration. Mild to moderate degeneration was present in GMmc and SG. M-252: the lesions included the superior temporal gyrus sparing only the temporal tip. In the medial geniculate, GMp was completely degenerated bilaterally except for the extreme caudal tip on the left side, which had some mild degeneration. SG and GMmc were severely degenerated.

maining three cases (M-37, M-246, M-252, Fig. 4). All of the lesions involved the entire primary core and secondary surrounding auditory fields. In terms of degeneration of the medial geniculate, M-57 had the largest lesion followed by M-37 and M-246, whose lesions were similar, followed by M-252 and M-150. As will be seen, all of the animals suffered substantial hearing losses.

Behavioral results

The *unilateral* lesions had no noticeable affect on the monkeys' free-field hearing, though no audiograms were taken at the time. A threshold at 4 kHz was obtained for one animal (M-150) and found to be normal. Though the left unilateral lesions caused an initial impairment in the discrimination of primate vocalizations (22), the animals had no difficulty detecting those sounds in a free-field test. However, preliminary evidence from more recent cases suggests that monkeys suffer a mild hearing loss in the ear contralateral to an auditory cortex lesion, which would not be detectable in a free-field test (24).

Following *bilateral* ablation, the monkeys were first tested for their ability to discriminate primate vocalizations (for a preliminary report, see Ref. 22). When it appeared that the animals were initially unable to hear the vocalizations, absolute thresholds were assessed throughout the postoperative period and compared with those of normal monkeys.

NORMAL MONKEYS. The audiograms of two normal monkeys, M-36 and M-337, are shown in Fig. 5. M-36 was tested at seven frequencies ranging from 250 Hz to 16 kHz. Its thresholds showed good agreement with the macaque average, deviating by no more than 3 dB and being on the average 1 dB more sensitive. M-337 was tested at all 10 frequencies. This animal averaged 3 dB less sensitive than the

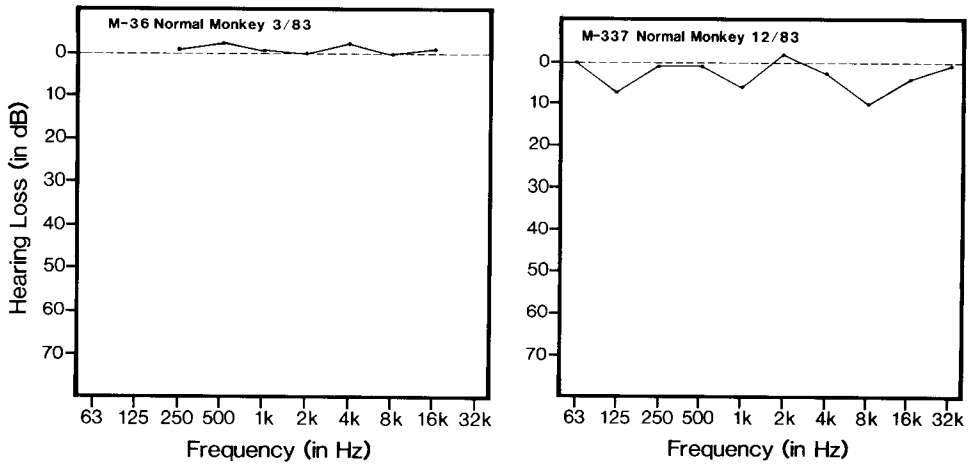

FIG. 5. Audiograms of 2 normal monkeys, M-36 and M-337.

normal average with a maximum difference of 10 dB at 8 kHz. Thus, normal monkeys tested in our apparatus showed close agreement with the macaque average.

MONKEY-37. First tested 6 days after bilateral ablation, M-37 was at this time completely unable to respond to the primate vocalizations. At 11 days, the animal showed signs of being able to detect the vocalizations when they were presented 70 dB above normal detection threshold. Two weeks after surgery the monkey was unable to detect a 1-kHz tone presented at 66 dB (Fig. 6). At 12 wk pure-tone thresholds obtained from 500 Hz to 4 kHz showed an average hearing loss of 41 dB ranging from 29 dB at 500 Hz to 47 dB at 4 kHz.

Thresholds from 500 Hz to 16 kHz were assessed 24 wk after surgery using conditioned suppression, and thresholds at 2 kHz, 4 kHz, and 8 kHz were replicated using conditioned avoidance. No consistent differences between the results of the two methods were found, and the data were combined into a single audiogram (Fig. 6). The average hearing loss at 24 wk was 30 dB and ranged from 19 dB at 500 Hz to 38 dB at 8 and 16 kHz.

The final audiogram assessed 52 wk after surgery showed normal or near-normal hearing at low (63- to 250-Hz) and high (32-kHz) frequencies. A comparison of the threshold from 500 Hz to 16 kHz with those taken at 24 wk, however, showed only a 1-dB improvement. The average hearing loss was 20 dB and ranged from 0 dB at 63 Hz to 44 dB at 8 kHz. Recovery appears to have reached asymptote at 24 wk, and the lower average threshold of the 52-wk audiogram is due to the fact that the loss was smallest at the very low and high frequencies which were tested for the first time.

MONKEY-57. When first tested 12 days after surgery, M-57 was unable to respond to tones. An audiogram attempted 4 wk postoperatively revealed that the monkey was unable to respond to frequencies from 500 Hz to 8 kHz (Fig. 6). Indeed, it was unable to respond to any of the tones or to the primate vocalizations until 11 wk after surgery. This inability was not due to a general difficulty in responding, as was demonstrated by its successful avoidance responses to a light cue. At 13 wk, the animal was able to detect 4 kHz reliably at 60 dB SPL. No further tests were carried out on this animal.

MONKEY-150. The absolute threshold for 4 kHz was assessed 3 wk after the right unilateral lesion (1 wk before the bilateral lesion). The animal was found to have a normal threshold at this frequency (−1 dB hearing level), and no further testing was conducted.

After bilateral ablation the monkey was able to detect primate vocalizations just above chance level on the first postoperative session 11 days after surgery. A complete audiogram assessed 33 wk after surgery (Fig. 6) indicated a hearing loss at middle and high frequencies with normal hearing at the low frequencies. The average hearing loss was 25 dB with thresholds ranging from −1 dB at 63 Hz to 52

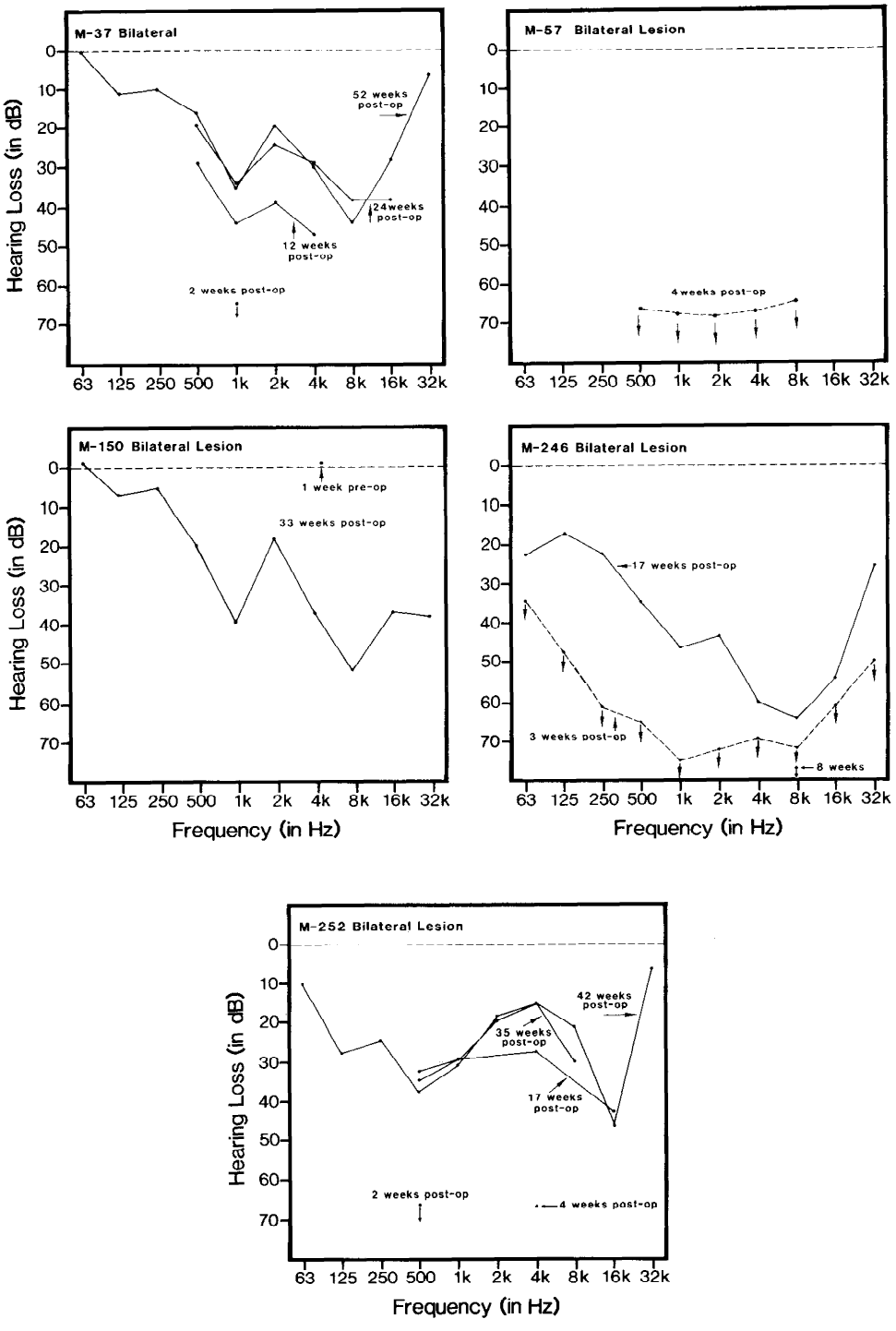

FIG. 6. Audiograms of 5 monkeys with bilateral auditory cortex lesions. *Dashed lines* and *downward arrows* indicate that an animal was unable to respond to the tones at maximum intensity. Hearing improved over time but did not reach normal levels even a year after surgery (M-37). Maximal recovery occurred at low frequencies and, in 3 cases, at 32 kHz. Note 4-kHz preoperative threshold for M-150.

dB at 8 kHz. Because it was able to detect primate vocalizations soon after surgery, this animal is believed to have had the smallest initial hearing loss.

MONKEY-246. This animal showed no response to primate vocalizations when testing began 6 days after surgery. An audiogram attempted 3 wk postoperatively failed to elicit any response to frequencies from 63 Hz to 32 kHz (Fig. 6). The animal gave its first auditory response to 75-dB white noise 8 wk after surgery, though it was still unable to respond to an 8-kHz tone presented at 82 dB SPL. An audiogram conducted at 17 wk revealed a hearing loss that was most pronounced in the midfrequency range from 1 kHz to 16 kHz. The animal's average hearing loss was 39 dB and ranged from 17 dB at 125 Hz to 64 dB at 8 kHz.

MONKEY-252. This animal was unable to respond to primate vocalizations or a 500-Hz tone (Fig. 6) when tested 13–16 days after surgery. When tested again at 4 wk, the monkey responded to a 4-kHz tone presented at 72 dB SPL.

Both conditioned suppression and conditioned avoidance were used to obtain absolute thresholds at 17 wk for 500 Hz, 1 kHz, 4 kHz, and 16 kHz. As in the case of M-37 no consistent difference between the results of the two methods was found, and the data were combined. The resulting thresholds demonstrated considerable recovery with the hearing loss averaging 33 dB and ranging from 27 dB at 4 kHz to 43 dB at 16 kHz. Further recovery was noted at 35 wk, at which time the monkey's average loss was 25 dB and ranged from 19 dB at 2 kHz to 34 dB at 500 Hz. A comparison of the 17- and 35-wk threshold for the three frequencies tested on both occasions showed an average improvement of 3 dB. A final and complete audiogram determined at 42 wk showed near-normal hearing at 63 Hz and 32 kHz. The animal's average hearing loss was 24 dB and ranged from 6 dB at 32 kHz to 46 dB at 16 kHz. Comparing the 42- and 35-wk audiogram shows an average of only 1 dB improvement in hearing for frequencies from 500 Hz to 8 kHz.

SUMMARY OF BEHAVIORAL RESULTS. Following unilateral temporal lobe lesion, none of the five monkeys showed any sign of a bilateral hearing loss, as indicated by their ability to hear primate vocalizations presented via a loudspeaker. One monkey, which was tested at 4 kHz 3 wk after a right unilateral lesion (M-150), had a normal free-field threshold. Recent evidence, however, suggests that the animals probably suffered a mild hearing loss in the ear contralateral to the lesion (24).

After the lesions had been made bilateral, however, four of the five monkeys initially were unable to respond to the sounds, and the fifth animal (M-150) could do so only with difficulty. All of the monkeys eventually regained the ability to respond to sound with the first signs of recovery occurring 2–11 wk after surgery. It should be noted, however, that during this time, the animals were tested with a variety of stimuli among which white noise proved to be the easiest to detect. Furthermore, the results indicate that recovery proceeds unevenly with hearing improving more rapidly at some frequencies than at others. It is possible, then, that had a loud white-noise stimulus been consistently used during this time, the first signs of hearing would have been demonstrated sooner than they were.

Maximum recovery of hearing appeared within 24 to 35 wk with little or no improvement thereafter. Recovery was most pronounced for the high and low frequencies, which returned to normal or near-normal levels in three of the cases. It is of interest to note that the recovery at high frequencies was apparent only at 32 kHz. Had we not tested this frequency, the resulting hearing loss would have resembled the high-frequency hearing loss that often accompanies aging in humans (i.e., presbycusis).

The final audiograms of four of the monkeys graphed in terms of sound pressure level are shown in Fig. 7. Displayed in this way, it appears that the effect of the lesions was to "flatten" the audiograms. That is, the animals showed a frequency-dependent loss with the largest losses occurring in the midrange of the audiogram where monkeys are normally most sensitive. Of the four audiograms, the one for M-246 shows the largest loss probably because it was obtained only 17 wk after surgery before the animal had reached maximal recovery. It appears from the other cases that the greatest sensitivity at any frequency was ~20 dB SPL. This flattening effect of central auditory system lesions has previously been noted following bilateral lesions of the brachium of the inferior colliculus and medial geniculate in dogs (27). During postoperative testing, we noticed that

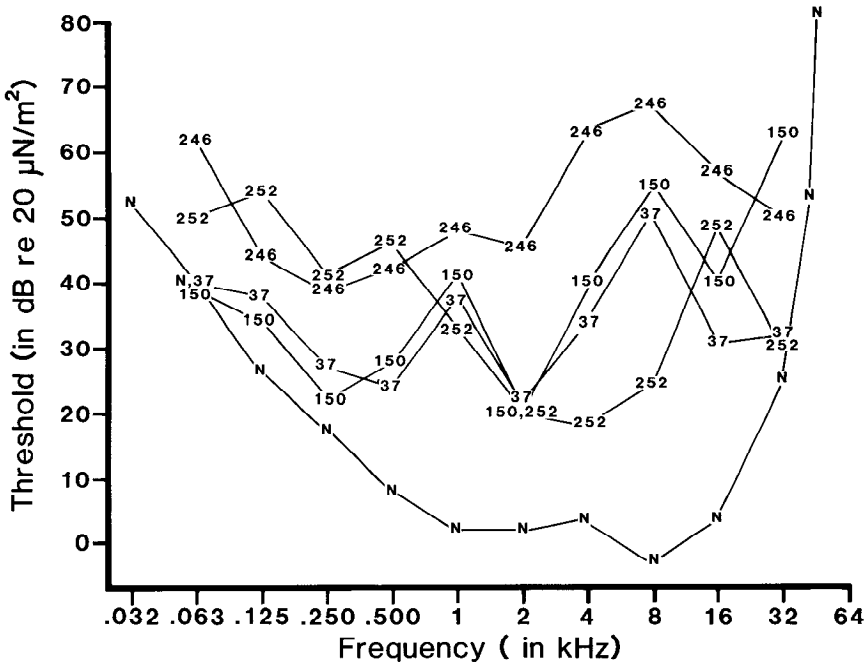

FIG. 7. Final audiograms of 4 of the monkeys (M-37, M-150, M-246, M-252) graphed in terms of absolute sound pressure level. Note that the lesions had a flattening effect on the audiograms.

the monkeys' thresholds were unusually variable, especially in the early tests. In particular, we found that it took more sessions than usual to obtain two thresholds per frequency that were within 4 dB of each other. Though we did not specifically study this effect, we gained the impression that there were short-term as well as long-term fluctuations in hearing, occurring during the early stages of recovery.

Finally, with regard to the relation between lesion size and hearing loss it can be noted that the animal with the largest lesion (M-57) took the longest to show initial recovery, whereas the monkey with the smallest lesion (M-150) appears to have had the earliest recovery of hearing. Other than this observation, the present data do not show any other relation between the locus and extent of the lesions and the degree of the hearing loss, though this may be due to the fact that the lesions were quite similar.

DISCUSSION

These results show that bilateral ablation of primary and secondary auditory cortex in macaques produces a consistent impairment in the ability to detect sound. Initially there is a total inability to respond to sound. With time the animals show marked improvement, although their hearing does not return to normal. While the monkeys eventually regain normal or near-normal hearing at low and very high frequencies, they appear to have a permanent midfrequency hearing loss.

The impairment appears to be a true inability to hear sound and not the result of nonsensory or performance variables. As has been argued elsewhere, a frequency-dependent hearing loss of the type observed here cannot easily be explained in terms of an attentional or emotional deficit (27). Such deficits would be expected to cause a general decrease in responding at all frequencies, whereas the hearing loss here was frequency dependent. Furthermore, the complete lack of a response to sound immediately after surgery is corroborated by human patients who have reported complete deafness immediately after suffering bilateral injury to the temporal lobes (e.g., Refs. 9, 17, 18, 41, 45, 56).

Nor does it appear likely that the failure to elicit normal thresholds is a function of the behavioral procedure used here. The conditioned avoidance and conditioned suppression

techniques have both been used successfully to train difficult-to-test and brain-damaged animals. The required response of ceasing to drink in the presence of a warning signal is easy to learn and can be acquired in a few minutes (e.g., Ref. 23). The task is a simple motor response requiring little cognitive ability and can be performed by decorticate mammals (49). These procedures are therefore well suited to determining the optimal ability of brain-damaged animals. In short, the observed deficits are not easily attributable to nonsensory factors but instead appear to be genuine hearing losses.

Previous animal studies

PRIMATES. The question of whether auditory cortex ablation results in deafness had its beginnings in the latter half of the nineteenth century. At that time, the effects of visual cortex lesions had led investigators to believe that the cortex was responsible for all sensation, and they therefore assumed that auditory cortex lesions would result in complete and permanent deafness. With this view, Ferrier sought to determine the location of auditory cortex by making a series of lesions in macaques and assessing their effect on hearing and other senses (11). By observing unconditioned reactions to sound, he found that temporal lobe lesions resulted in deafness, and indeed, one of his cases, which was exhibited at the International Medical Congress of 1881, was reputed to be permanently deaf (5, 13). On the basis of these studies, Ferrier correctly placed auditory cortex in the superior temporal gyrus (12).

However, Ferrier's findings were disputed by Schäfer, who was unable to replicate them (5). Indeed, because he could find no cases of total deafness, Schäfer stated that auditory cortex could not be located in the superior temporal gyrus of the monkey. There followed an acrimonious debate in which Schäfer at first suggested that the monkey which Ferrier exhibited was deaf prior to surgery but then later claimed that Ferrier's own observations demonstrated that the monkey was not deaf after surgery (5, 51, 52). Ferrier, on the other hand, claimed that Schäfer's lesions were incomplete and went on to note the care that must be taken when assessing hearing (12).

In retrospect, it is understandable that such controversy should arise. The total deafness arising from cortical ablation as seen in our cases is both transient and no doubt dependent on the completeness of the lesion. Ferrier probably encountered complete deafness in his early cases which survived for a few hours to a few days (11). His single case of long-term deafness could well have had a large residual hearing loss and, in addition, might have become somewhat habituated to sound by repeated testing for unconditioned reactions. In addition, complete removal of auditory cortex in the macaque is complicated by the fact that it lies within the depths of the sylvian fissure, and Schäfer's lesions could easily have been incomplete. Even so, the test results of at least one of Schäfer's animals suggest the existence of a partial hearing loss (52). Thus, of the two views, it appears that the conclusions drawn by Ferrier concerning the locus of auditory cortex and at least the initial effect of lesions on hearing are surprisingly accurate.

Since then, a number of studies have examined the effect of cortical ablation on hearing in monkeys (for reviews, see Refs. 43, 55). Yet, in no case was a detailed examination of the effect on auditory thresholds ever conducted. While most of the studies looked at other aspects of hearing, such as frequency or intensity discrimination, the few that did determine absolute thresholds often didn't show the lesions or, for that matter, the resulting audiogram. In studies involving auditory discriminations, the animals were often not tested until several weeks after surgery, by which time the animal would be recovering from the transient total deafness. On the other hand, it is not unusual to find reports of "postoperative amnesia" (10, 44) and "loss of learned habit" (32), suggesting that these animals may have experienced transient deafness. But, whereas nineteenth-century investigators assumed that such lesions would result in complete deafness, twentieth-century investigators assumed that there was no effect at all on absolute thresholds and attributed lack of responsiveness to nonsensory factors. Indeed, the possibility that auditory cortex lesions have an effect on absolute sensitivity was not considered in more recent studies (cf., Refs. 25, 26).

CARNIVORES. Both cats and dogs have been widely used in auditory ablation studies, and a number of partial audiograms have been obtained following cortical ablation (for reviews, see Refs. 35, 43). We have reviewed these studies elsewhere and have noted that while

some have failed to find any effect on absolute thresholds, most have found significant hearing losses following bilateral cortical ablation (27). In particular, one study found a hearing loss in cats in which thresholds improved with time, but remained 5–20 dB below normal (38). Another study, in which the auditory input to cortex in dogs was interrupted by lesions of the medial geniculate, found a permanent hearing loss of up to 37 dB at midfrequencies with normal or near normal thresholds at low and very high frequencies (27). These studies, then, suggest that bilateral auditory cortex ablation may produce a hearing loss in carnivores similar to that described here for monkeys.

OTHER SPECIES. The effect of cortical ablation on absolute thresholds has been examined in only two other species, the laboratory rat and the Virginia opossum. In a preliminary investigation of the rat, it was found that bilateral auditory cortex ablation resulted in a hearing loss of ~30 dB at 4 kHz and 16 kHz (33). However, the animals showed little or no loss at 1 kHz and 42 kHz, suggesting that, like the monkeys, they had suffered a midfrequency hearing loss. On the other hand, no significant hearing loss was found in a decorticate opossum tested at 500 Hz, 2 kHz, 8 kHz, and 32 kHz (49). At the present time, the information on the rats is based on only two cases, and that on the opossum is from a single case. Additional cases are needed to determine more fully the effects of cortical ablation on absolute sensitivity. However, since it is already known that there are species differences in the effect of cortical ablation on other auditory abilities (34), it would not be surprising if similar species variation were found with regard to the cortical role in absolute sensitivity.

Human bilateral temporal lobe lesions

Though bilateral temporal lobe lesions are considered to be rare in humans, a number of cases have been reported (for reviews, see Refs. 16, 18, 31). Indeed, since 1969, at least 16 cases have been described in which detailed audiological examinations were given (1, 2, 7, 9, 17, 18, 30, 31, 36, 37, 41, 42, 45, 47, 50, 56). The degree of hearing loss in these cases has varied over a range from complete deafness to a mild hearing loss. Of these 16 cases 13 noted the sudden onset of severe or total deafness coincident with the lesion (1, 7, 9, 17, 18, 30, 31, 36, 37, 41, 42, 45, 56), while the other 3 either didn't mention a hearing loss or else noted that the major complaint was an inability to understand speech (2, 47, 50). In the 13 cases that noted a sudden hearing loss, 9 reported varying degrees of partial recovery (9, 17, 18, 30, 31, 37, 41, 45, 56), while the rest did not specify whether or not such recovery occurred. Finally, 9 of the 16 cases had a noticeably smaller hearing loss at low frequencies than at middle or high frequencies (1, 2, 36, 37, 41, 42, 45, 47, 56), while the remaining cases either showed no frequency-dependent loss or else didn't specify the hearing loss by frequency.

Comparing the loss in these human studies with the hearing loss observed in our monkeys, three major points of agreement emerge. First, the monkeys experienced a sudden hearing loss following bilateral ablation, as did many of the human cases. That the degree of the deafness in humans varies from case to case is not surprising and is most likely due to differences in the size and extent of the lesions. Second, the monkeys showed a partial recovery of hearing over time, as has been noted in the majority of human cases. That such recovery has not always been noted may be due to the failure to look for it or to the possibility that hearing had stabilized by the time the first audiogram was determined. Finally, the monkeys had a frequency-dependent hearing loss in which both low frequencies (63–250 Hz) and very high frequencies (32 kHz) showed better recovery than the middle frequencies. Comparing monkeys with the human cases on this point, however, is complicated by the fact that most human audiograms cover only the portion of human hearing from 250 Hz to 8 kHz. To cover a range equivalent to that for which the monkeys were tested would require testing from 50 Hz to 16 kHz. Nevertheless, most of the studies of humans indicate that low-frequency hearing is less impaired than high-frequency hearing, and one study which determined thresholds down to 63 Hz found that hearing improved noticeably at the lower frequencies (1; see also Ref. 36). Indeed, the tendency towards less impairment at low frequencies may explain why a patient may be able to hear low-intensity environmental sounds even though a conventional audiogram indicates a large hearing loss (cf. Ref. 17). That no one has reported very high frequencies to be less impaired may be due to the fact that humans are virtually never tested above 8 kHz

(cf. Ref. 28). Thus the cortical hearing loss observed in monkeys is remarkably similar to that found in humans.

In spite of such evidence there has been uncertainty as to whether and to what extent bilateral temporal lesions affect hearing (cf. Refs. 2, 7, 16, 17, 31, 50). In general, the arguments against cortical deafness revolve around three issues. First, there is the question of whether the hearing loss is due to a premorbid factor such as age, drugs, noise exposure, or familial hearing loss (e.g., Ref. 31). However, in two of the human cases, premorbid audiograms were available which indicated that the observed hearing losses were of recent origin (30, 31). This evidence plus the fact that in most cases the hearing loss was of sudden onset coincident with the lesion indicate that the hearing loss was a result of the lesion and not a preexisting condition.

Second, the animal literature is often cited as indicating that bilateral auditory cortex lesions do not produce hearing loss (e.g., Refs. 2, 7, 18). However, as our results demonstrate, bilateral lesions in macaques produce a hearing loss similar to that seen in humans.

Finally, humans with bilateral temporal lesions usually have other deficits, such as word deafness, aphasia, and agnosia. Since it was once thought that such deficits were a secondary result of hearing loss (cf. Ref. 16), investigators have been required to demonstrate that their patients had sufficient sensitivity to hear the test sounds with the result that any accompanying hearing loss was deemphasized (e.g., Refs. 1, 7, 50). However, it seems possible for both a hearing loss and a deficit in recognizing sounds to coexist in the same patient. Indeed, the monkeys in this study could not discriminate primate vocalizations even after their hearing had recovered sufficiently to detect them easily (22).

In conclusion, we can find no valid reason for rejecting the idea that bilateral auditory cortex lesions result in a hearing loss in humans. While clinical cases are often difficult to interpret for a variety of reasons, not the least of which is the difficulty in determining the exact locus and extent of the lesions, sufficient cases have been reported that do show such a hearing loss. With the present finding of a similar deficit in macaques, the issue of whether or not cortical deafness occurs has now been resolved.

Role of auditory cortex in hearing

There are at least two possible explanations for the hearing loss following auditory cortex ablation. One is that the deafness is due to the sudden disconnection of cortex from lower auditory centers more directly involved in the detection of sound. This is the phenomenon of diaschisis, which refers to an unresponsiveness of neurons due to a state of shock brought about by damage to areas to which they give or receive projections either directly or indirectly. The recovery of hearing over time is considered to be due to a restabilization as neurons adapt to the loss of auditory cortex.

A second possibility is that auditory cortex is necessary for sound detection. According to this view, the recovery of hearing is a result of lesions that are less than total, and recovery does not occur following very large bilateral lesions (e.g., Ref. 18). A variant of this view is that the recovery of hearing following bilateral auditory cortex lesions is due to adjacent cortical areas taking over auditory function (38). Just what the precise role of auditory cortex is in the detection of sound, however, remains to be determined.

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