JOURNAL OF NEUROPHYSIOLOGY
Vol. 64, No. 1, July 1990. *Printed in U.S.A.*

Effect of Bilateral Auditory Cortex Lesions on Absolute Thresholds in Japanese Macaques

HENRY E. HEFFNER AND RICKYE S. HEFFNER
Laboratory of Comparative Hearing, Department of Psychology, University of Toledo, Toledo, Ohio 43606

SUMMARY AND CONCLUSIONS

1. The behavioral audiograms of four Japanese macaques (*Macaca fuscata*) were assessed before and after receiving two-stage bilateral lesions of auditory cortex. Thresholds were assessed for each ear with the use of insertion earphones.

2. The bilateral lesions resulted in a large initial hearing loss followed by partial recovery that left the animals with a permanent hearing loss in both ears.

3. The initial hearing loss consisted of a total insensitivity to sound in the ear contralateral to the second lesion with limited hearing in the other ear. However, the animal with the most complete lesion was initially unable to hear sound in either ear. Broadband noise was often more effective in eliciting a behavioral response than tones.

4. Partial recovery occurred in all animals and was observed as early as the first week after surgery. Most of this recovery occurred during the first 3–7 wk after surgery. This rapid phase of recovery was sometimes followed by a more gradual phase although thresholds were still elevated after 94 wk.

5. The permanent hearing loss, which averaged from 30 to 44 dB, was not constant across frequency. Threshold shifts were smallest at 63 Hz and progressively increased with frequency to a maximum loss from 8 to 25 kHz with slightly less loss at 32 kHz.

6. Analysis of the psychophysical functions and threshold stability gave no indication of any nonsensory deficits in attention or vigilance.

7. These results, taken with those of previous experiments, indicate that each hemisphere is primarily involved in the detection of sound in the contralateral ear and secondarily involved in detection in the ipsilateral ear. This arrangement differs from that seen in sound localization where each hemisphere is involved with the contralateral hemifield as opposed to the contralateral ear. Thus it appears that the functional organization of auditory cortex for sound localization is different from that for the detection and identification of sound itself.

INTRODUCTION

The idea that cortical lesions result in a hearing loss has gone through many changes. Although nineteenth-century investigators believed that ablation of auditory cortex would result in complete and permanent deafness (e.g., James 1890), the failure to find total deafness in animals called this belief into question. Indeed, the observation that cortical lesions in cats and dogs have little permanent effect on absolute thresholds led most experimentalists to reject the notion that the cortex plays a role in the detection of sound (Neff et al. 1975). However, continued reports of hearing loss in humans after bilateral temporal lobe lesions kept the idea of cortical deafness alive, although even among clinicians there were many who were skeptical (for a review, see Heffner and Heffner 1986).

After several decades of belief to the contrary, it has now been established that auditory cortex lesions can result in large and permanent hearing losses in primates, particularly in macaques and humans. That such hearing losses were not reported by previous studies is due to the fact that these studies examined cats and dogs, and it is now clear that there are substantial species differences in the role of the cortex in hearing: unlike primates, carnivores show only a small cortical hearing loss (for reviews, see Heffner and Heffner 1986; Kavanagh and Kelly 1988).

In macaques, however, both unilateral and bilateral lesions of auditory cortex result in a decrease in the ability to detect sound. In the case of unilateral lesions, a hearing loss can be found in the ear contralateral to the lesion, whereas sensitivity in the ipsilateral ear is unaffected (Heffner and Heffner 1989c). Bilateral lesions, on the other hand, result in a hearing loss in both ears (Heffner and Heffner 1986, 1989b).

Both unilateral and bilateral lesions result in a large initial hearing loss followed by substantial recovery that becomes apparent within a few days or weeks after surgery. However, the recovery is incomplete, and the animals are left with a permanent hearing loss.

The effects of unilateral lesions, which have been studied in detail, indicate that the average initial hearing loss ranges from ~20 to 40 dB during the first week after surgery with a period of rapid recovery occurring during the first 3–5 wk after surgery. Subsequent improvements in sensitivity are modest, and the animals are left with a permanent hearing loss that averages from 4 to 10 dB depending on the completeness of the lesion (Heffner and Heffner 1989c).

Bilateral lesions result in an initial hearing loss that can consist of a complete inability to respond to sound (Heffner and Heffner 1986). This is followed by a recovery period, the exact time course of which is unknown. The animals are then left with a permanent hearing loss that averages from ~20 to 40 dB. The fact that this hearing loss is much greater than that after unilateral ablation indicates that a bilateral hearing loss is not simply the sum of two unilateral hearing losses.

The previous studies of the effect of bilateral lesions on absolute sensitivity were conducted with loudspeakers that, although sufficient to describe the sensitivity of an animal, could not specify the effect of the lesions on each ear separately. In addition, the audiograms were not taken frequently enough to enable the recovery process to be exam-

ined in detail. Thus the purpose of the present study was to determine the effect of bilateral auditory cortex ablation on the absolute sensitivity of each ear over a period of time sufficient to obtain a comprehensive picture of the recovery process. This was done by examining the effect of ablation of auditory cortex in the intact hemisphere of animals previously used in the study of unilateral lesions (Heffner and Heffner 1989c).

METHODS

The general design of the study was to first determine the preoperative audiograms of four monkeys for each ear separately with the use of earphones. These audiograms served as the base line against which all subsequent audiograms were compared. The animals received two-stage lesions of the superior temporal gyrus with testing between stages, the results of which have been previously reported (Heffner and Heffner 1989c). After bilateral ablation, the animals were tested periodically for up to 94 wk.

Subjects

Six 7-yr-old (adolescent) male Japanese macaques (*Macaca fuscata*) that had been born and reared in a free-ranging colony (Arashiyama West Institute) were used in this study. Four of the animals, referred to as *M-207, M-214, M-267*, and *M-337* were also used in studies of the effect of cortical ablation on the perception of primate vocalizations and on the effect of unilateral cortical ablation on hearing (Heffner and Heffner 1989a,c). Two additional monkeys were used as normal controls. All animals were individually housed in primate cages with free access to food and were trained with water reward. Each monkey was weighed daily to monitor its health and deprivational level. The ears of each animal were examined during and after testing to ensure that they were free of damage or disease. Auditory-evoked potentials were recorded from each operated animal at the end of the experiment to provide further evaluation of their peripheral hearing (Hood and Heffner 1989).

Surgical and histological procedures

SURGERY. After preoperative training and testing, the four monkeys received two-stage lesions of the temporal lobe. For surgery, a monkey was initially sedated with ketamine (5 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 superior temporal gyrus removed by subpial aspiration. Aseptic procedures were followed throughout surgery.

After removal of cortical tissue, the lesion was packed with Gelfoam (Upjohn) 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 temporal muscle was then apposed and sutured, a topical antibiotic powder (Neo-Predef, Upjohn) was applied, and the scalp incision was closed with silk suture. The animal was placed in a cage located in a dark, quiet room and given acepromazine as needed to minimize discomfort.

HISTOLOGY. After 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 lesions and resulting thalamic degeneration.

Procedure

BEHAVIORAL APPARATUS. A standard primate chair was modified to accommodate a "double" water spout. This spout consisted of two standard sipper tubes mounted parallel and close enough (1 cm apart) so that a monkey could comfortably place its mouth on both spouts. The two spouts, which were electrically isolated from each other, were connected to a contact switch that detected when an animal placed its mouth on them. One of the spouts was attached via plastic tubing to an electrically operated water valve and constant-pressure water reservoir. Use of the double water spout eliminated the need to tie an animal's foot to complete the circuit for the contact switch, thus providing an animal with greater freedom of movement. Mild electric shock was provided by a shock generator connected to the two spouts.

Although an animal was able to turn about in the monkey chair, perfunctory head movements were restricted to prevent accidental dislodging of the insertion earphones through which the stimuli were presented. This was accomplished by mounting two vertical rods on the primate chair so that an animal placed its muzzle between them while drinking from the spout. A horizontal rod mounted on the two vertical rods above the animal's muzzle and a back plate mounted behind the animal's head completed the arrangement. This configuration enabled the animal to easily break contact with the water spout while reducing the possibility of accidentally dislodging the earphones. However, the animal was still able to turn around in the chair and face the door of the test chamber, an act that dislodged the earphones. As this usually occurred late in a session, it was taken as an indication that an animal wished to terminate the session, and it was returned to its cage. Supplemental water was given as needed to maintain good health.

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 was used for behavioral programming.

ACOUSTICAL APPARATUS. Sine waves were produced by a generator (Hewlett-Packard 209A), and noise stimuli were produced by a white noise generator (Grason-Stadler 901A). The signal was switched by a rise-fall gate (Coulbourn S84-04) and pulsed 2 times per second (250 ms on, 250 ms off; 50-ms rise-fall for 63–250 Hz and 20-ms rise-fall for higher frequencies). The signal was then led to an attenuator (Hewlett-Packard 350B), a band-pass filter that was set at ⅓-octave points above and below the test frequency (Krohn-Hite 3202), an amplifier (Crown D75), and finally to headphones (Koss Pro 4X).

The headphones, which were suspended over the animal's head, were fitted with a plexiglas adapter and connected to molded ear pieces via a 25-cm length of plastic tubing (2 mm ID). The use of insertion earphones maximizes the interaural attenuation (cf. Killion et al. 1985) thus allowing each ear to be stimulated independently while allowing the animal to move its head to break contact with the water spout. The use of these particular earphones in a study that found large differences in thresholds between the two ears (Heffner and Heffner 1989c) and unpublished observations involving the discrimination of monkey vocalizations indicated that the attenuation between the two ears was at least 60 dB. Thresholds were taken at octave steps from 63 Hz to 32 kHz with an additional threshold taken at 25 kHz.

The output of the headphones was periodically checked with a Brüel and Kjaer (B & K) sound level meter (model 2203), 1-in. (2.54-cm) microphone (B & K 4144), octave filter (B & K 1613), and artificial ear (B & K 4152 with 2-cc coupler). Frequencies above 16 kHz were measured with a 0.5-in. (1.27-cm) microphone (ACO 7012), microphone amplifier (B & K 2608), external filter (B & K 1613 or Krohn-Hite 3550), and artificial ear (B & K 4152 with 2-cc coupler). In addition, the output of the microphone amplifier was led to an oscilloscope to verify the presence of a signal. All microphones were calibrated with a pistonphone (B & K 4220).

PSYCHOPHYSICAL PROCEDURE. A thirsty monkey was rewarded for climbing into the primate chair and placing its mouth on the water spout by a steady trickle of water (3–4 ml/min) that was provided as long as the animal maintained contact with the spout. Tones were then presented through the earphones at random intervals and followed at their offset by mild electric shock delivered across 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 turned on and off with the shock to indicate that shock had been delivered and that the animal could return to the spout.

The level of the shock was individually adjusted for each animal to the lowest level that would reliably produce an avoidance response. The mildness of the shock was empirically verified by observing that the animals never developed any fear of the water spout and returned to it without hesitation after receiving a shock.

The test procedure was identical to that used previously (Heffner and Heffner 1989c). This procedure consisted of trials in which a train of tone pulses (250 ms on, 250 ms off) was presented for 3.5 s at random intervals 5–47 s after the previous trial. The occurrence of the tone was restricted to time periods at 7-s intervals (e.g., 5 s, 12 s, 19 s, . . . 47 s) so that there was a maximum of seven time periods during which a tone might occur. The number of times in which a tone occurred in a particular time period was adjusted so that each of the seven periods had the same probability (0.25) of containing a tone. This resulted in some sequences in which no tone occurred so that the probability of the seventh time period containing a tone was also 0.25. Thus a trial would begin 5 s after the previous trial and would end either after presentation of a tone or 50.5 s later (if no tone was presented).

The response of an animal on each trial, i.e., whether it had made an avoidance response, was determined by noting whether the animal was in contact with the spout during the last 200 ms of the time period that contained the tone. Basing the response criterion on the last 200 ms allowed the animal sufficient time to break contact with the spout after presentation of the tone. Responses to a particular intensity level were averaged to arrive at a "hit" rate. A measure of an animal's "false alarm" rate was obtained by recording the animal's response during the last 200 ms of the time periods that did not contain a tone. To reduce the effects of occasional pauses in drinking, a score for a particular time period was automatically discarded if the animal was not in contact with the spout at any time during the 1 s immediately preceding that period.

The hit and false alarm rates for a session were determined separately for each frequency at each intensity. A measure of discrimination was then expressed in the form of a performance ratio, (Hit Rate − False Alarm Rate)/(1 − False Alarm Rate). This formula is a classic method for correcting hit rate for false alarms (Green and Swets 1966) and is identical to the formula (S − W)/S, which we have previously used (Heffner and Heffner 1986). In trained animals, this measure varies from ~0 (failure to detect the tone) to 1 (perfect detection).

Two procedures were used to determine auditory thresholds. Preoperative thresholds were first 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 with the use of the method of constant stimuli 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 score of 0.2. Chance level was calculated by comparing the occurrence of responses during the safe and warning trials with the use of the binomial distribution (Hays 1963).

Once initial preoperative thresholds had been determined, they were rechecked with the use of the descending method of limits. This procedure consisted of decreasing intensity first in 10-dB and then 5-dB steps, with three to five warning trials given at each intensity, until the animal no longer responded to the stimulus. The thresholds obtained with this procedure were in close agreement with those obtained in the initial audiogram. This method was then used for rapid assessment of postoperative thresholds. However, every stimulus for which a threshold shift was noted was tested at least once with the method of constant stimuli. No significant differences were noted between the results of the two procedures.

RESULTS

Anatomic results

The locus and extent of auditory cortex have been studied in the rhesus macaque by evoked response (Woolsey 1972; Woolsey and Walzl 1982), microelectrode recordings (Merzenich and Brugge 1973), cytoarchitectural analysis (Galaburda and Pandya 1983; Pandya and Sanides 1973), and by tracing thalamocortical connections (Mesulam and Pandya 1973). 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.

Although the exact boundaries of auditory cortex can be difficult to define, both cytoarchitectural and electrophysiological studies have indicated that there is a central core area (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 middle one-third of the superior temporal plane and is surrounded by

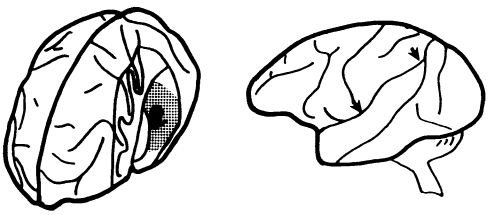

FIG. 1. Location of auditory cortex in the macaque. *Left*: Japanese macaque brain with the frontoparietal operculum removed to show the location of the core (blackened) and the surrounding belt (stippled). More auditory belt is buried in the circular sulcus bounding the insula. *Right*: lateral surface of a Japanese macaque brain. Arrows mark approximate limits of the core and surrounding belt on the superior temporal plane [drawings based on the electrophysiological study of Merzenich and Brugge (1973)].

four secondary auditory fields (Fig. 1) (Merzenich and Brugge 1973).

Other parts of the superior temporal gyrus also appear to be auditory. Auditory responses have been recorded on the superior temporal plane rostral to the secondary fields as well as on the lateral surface of the superior temporal gyrus indicating that there are at least two other auditory fields (Merzenich and Brugge 1973). In addition, the cytoarchitectonics and cortical connections of the auditory fields suggest that all of the superior temporal gyrus including the rostral portion may have auditory functions (Mesulam and Pandya 1973; Pandya and Sanides 1973). This conclusion is supported by behavioral evidence demonstrating that lesions restricted to the portion of the superior temporal gyrus rostral to the primary and surrounding auditory fields produce a hearing loss (Heffner and Heffner 1989b).

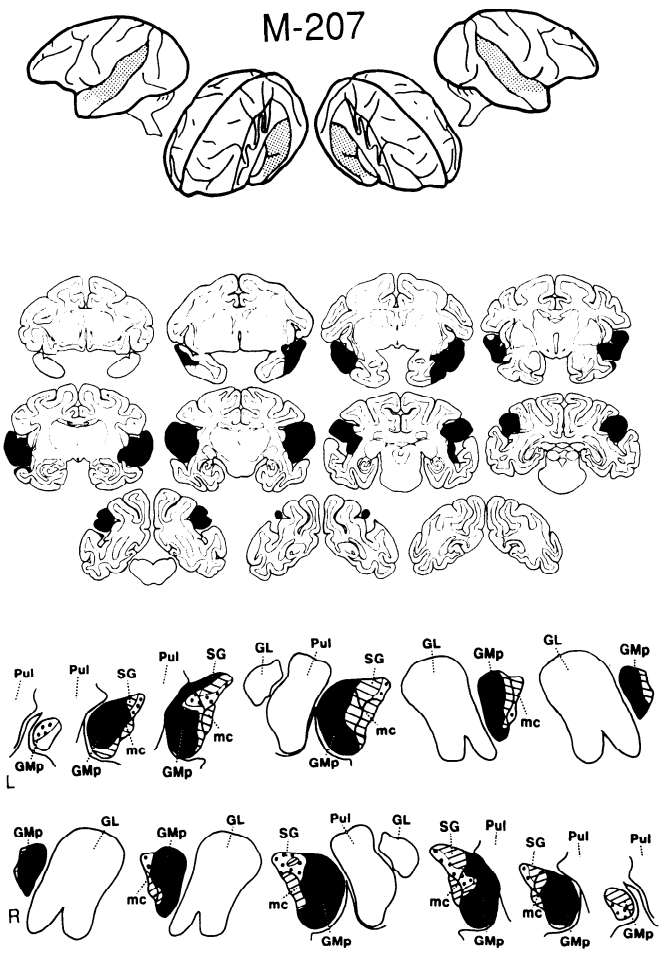

FIG. 2. Cortical reconstruction and medial geniculate degeneration for monkey *M-207*. *Top*: reconstruction of cortical lesion (stippling) showing surface views and views of the superior temporal plane with the parietal operculum removed. *Middle*: coronal sections 3.6 mm apart with ablated areas shown in black. *Bottom*: retrograde degeneration in the vicinity of the medial geniculate. Left thalamic sections (*top*) are shown posterior to anterior whereas right sections (*bottom*) are anterior to posterior. Thalamic sections are 600 μm apart. GL, dorsal lateral geniculate; GMp, principal division of the medial geniculate; mc, magnocellular division of the medial geniculate; Pul, pulvinar; SG, suprageniculate 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.

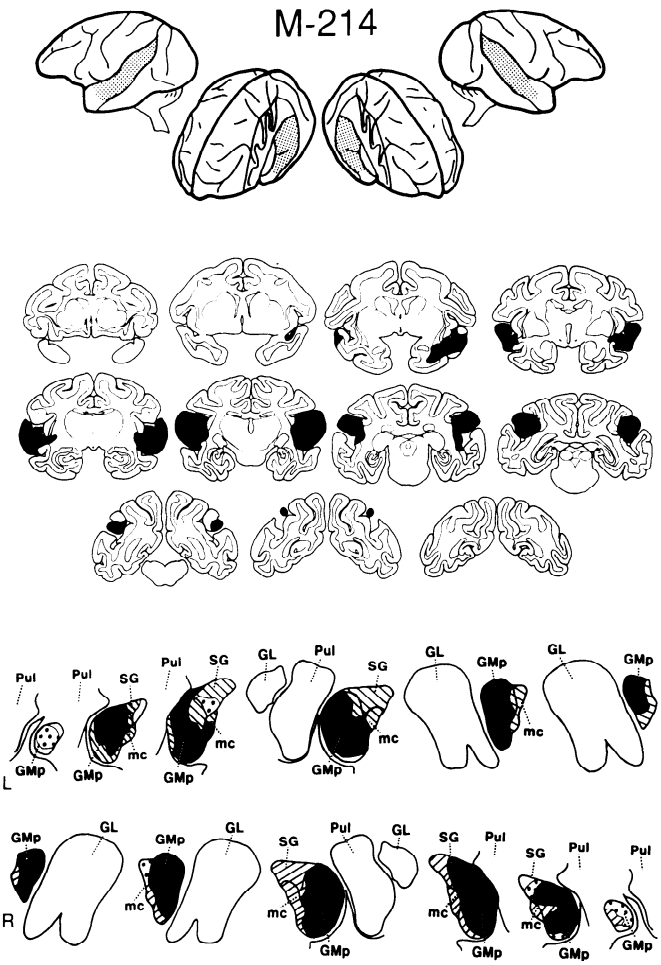

FIG. 3. Cortical reconstruction and medial geniculate degeneration for *M-214*. (See Fig. 2 for key.)

Although precise placement of auditory lesions requires prior electrophysiological mapping, the pattern of thalamic degeneration that follows cortical ablation gives a useful picture of the extent of a lesion (for details, see Heffner and Heffner 1986). Ablation of primary auditory cortex results in severe degeneration of the anterior two-thirds of the principal division of the medial geniculate (GMp) with no noticeable degeneration in the magnocellular division (GMmc) or in the suprageniculate nucleus (SG). Ablation of the primary and surrounding secondary auditory fields results in severe degeneration throughout GMp, with the exception of the caudal tip, and partial degeneration of GMmc and SG. Finally, ablation of the entire superior temporal gyrus results in total degeneration of all of GMp, including the caudal tip, with severe degeneration in GMmc and SG.

EXTENT OF THE LESIONS. Cortical reconstructions and thalamic degeneration are illustrated for each of the four cases (Figs. 2–5). The lesions were essentially confined to the superior temporal gyrus and differed primarily in the involvement of the rostral tip of the gyrus. All of the animals appeared to have complete lesions of the primary and surrounding auditory fields with the possible exception of *M-267* in which part of the rostral auditory field on the left side may have been spared (Fig. 4).

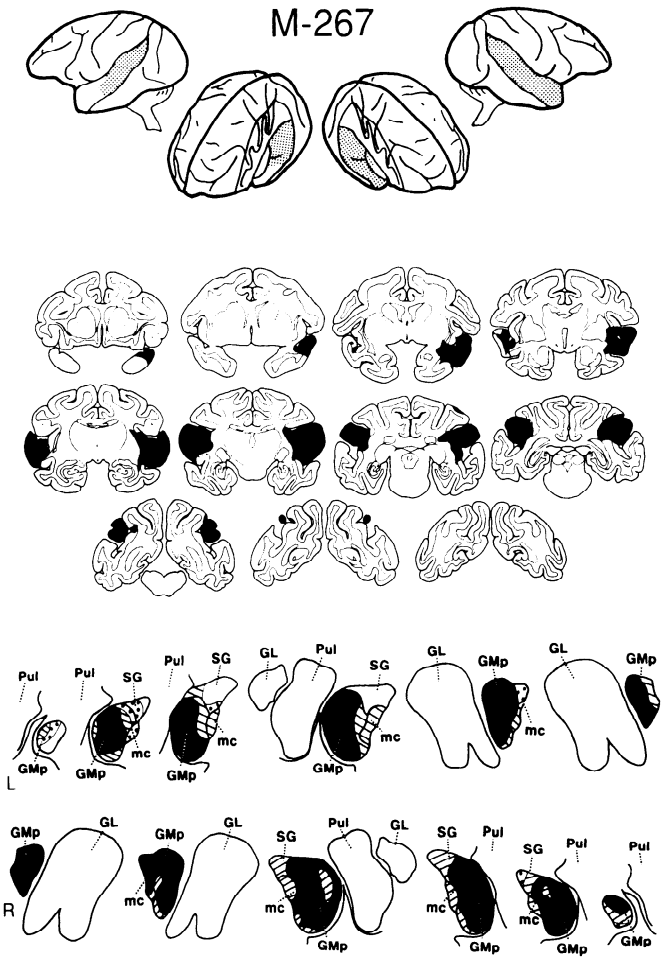

FIG. 4. Cortical reconstruction and medial geniculate degeneration for *M-267*. (See Fig. 2 for key.)

Before turning to the behavioral results, it is necessary to consider two factors: the order in which the two-stage lesions were done and the size of the lesions. First, the order in which the lesions were made was left-right for *M-214* and *M-267*, and right-left for *M-207* and *M-337*. As will be seen, the bilateral hearing loss was consistently larger in the ear contralateral to the second lesion.

Second, although the lesions were confined primarily to the superior temporal gyrus and did not differ significantly in their caudal extent, they did differ in the involvement of the rostral portion of the gyrus. Examination of the serial sections (Figs. 2–5) reveals that *M-267* received the smallest lesion because of sparing of the rostral tip in the left hemisphere. *M-337*, on the other hand, received the largest bilateral lesion of the four animals. As will be seen, *M-337* had the largest initial and permanent hearing loss, whereas *M-267* had the smallest.

Behavioral results

Bilateral ablation of auditory cortex consistently resulted in a large initial hearing loss in both ears (Figs. 6–9). The animals showed rapid but partial recovery of their hearing during the first 3–7 wk after surgery. Further improvement in hearing was minor, and the animals were left with permanent hearing losses averaging 30–44 dB.

INITIAL HEARING LOSS. Previous work with the use of tonal stimuli has shown that bilateral ablation of auditory cortex results in a large initial hearing loss that in some cases renders an animal totally unresponsive to sound for days or even weeks (Heffner and Heffner 1986). To determine whether the current animals showed any early response to either loud broadband noise or tones, testing was begun 3 days after surgery.

The results of these tests show that each animal was initially completely unresponsive to sounds presented to the ear contralateral to the second lesion (Table 1). However, only the monkey with the largest bilateral lesion (*M-337*) was completely unresponsive to sounds presented to either ear.

Another finding was that broadband noise was occasionally more effective in eliciting the first signs of hearing than were tones. This was particularly true in the case of *M-337*, which was unable to respond to tones for 5–7 days after it had begun to respond to the noise.

These results provide further demonstration that bilateral auditory cortex lesions can result in a transient total deafness in both ears (cf. Heffner and Heffner 1986). However, its occurrence may depend on the completeness of the lesion, and its duration may depend on the type of stimuli used to elicit a response. Furthermore, it would appear that

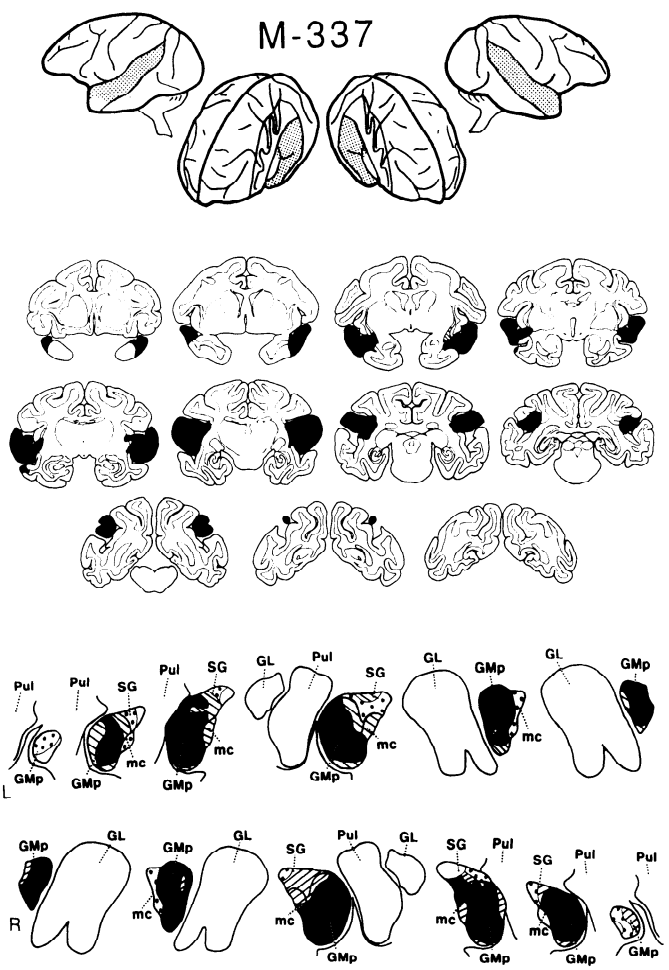

FIG. 5. Cortical reconstruction and medial geniculate degeneration for *M-337*. (See Fig. 2 for key.)

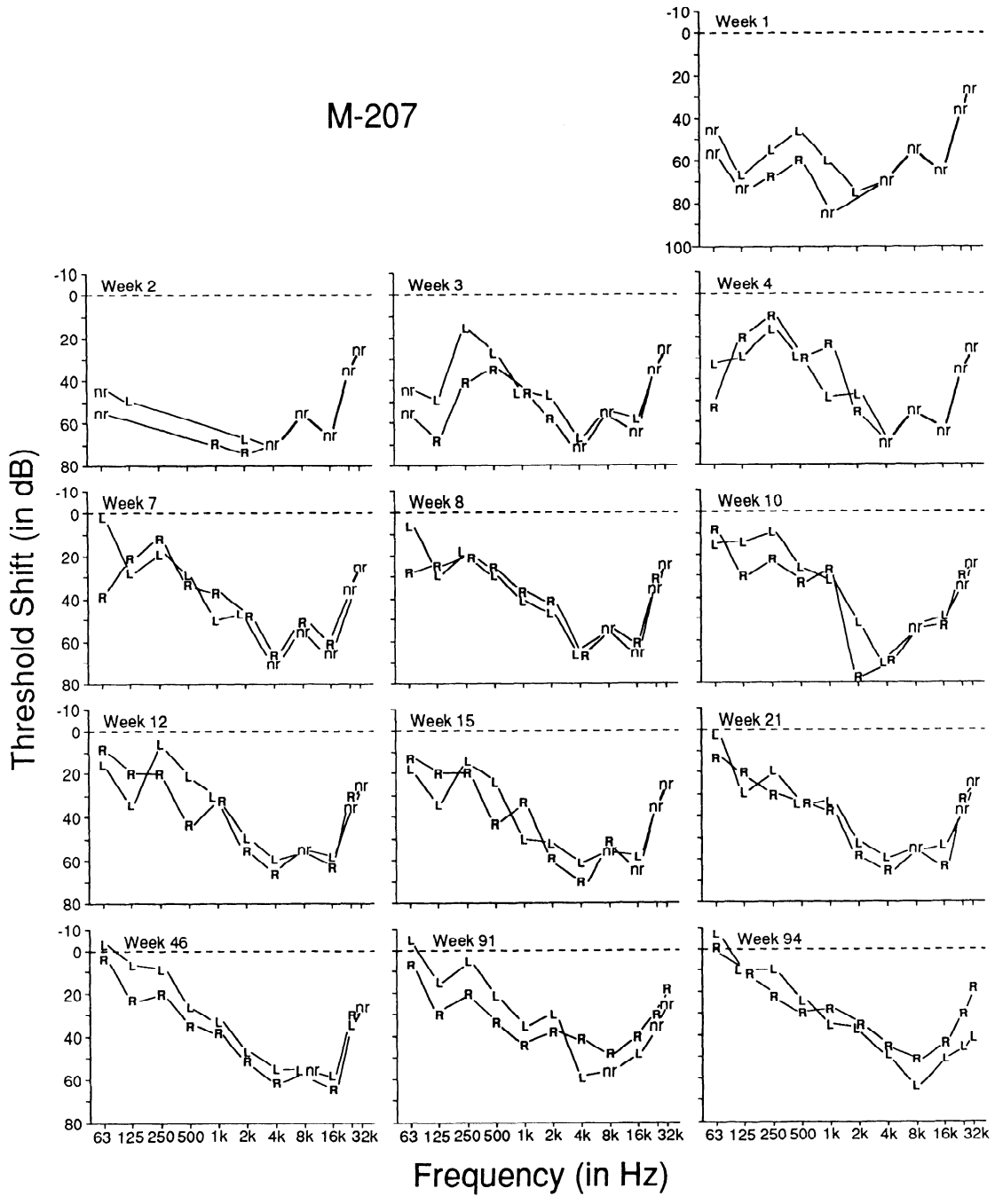

FIG. 6. Audiograms for the left (L) and right (R) ears of *M-207* taken at various times after bilateral auditory cortex lesions. Surgery was conducted in 2 stages with the right lesion made 16 wk before the left. Zero level (dashed lines) indicates the animal's preoperative hearing level; nr indicates that the animal was unable to respond to a tone at maximum intensity.

a two-stage lesion does not affect both ears equally. In particular, the ear contralateral to the first lesion was slightly less affected, suggesting that some recovery took place between surgeries that benefited only this ear.

RECOVERY. Examination of the individual audiograms indicates that the animals showed substantial recovery over time (Figs. 6–9). This recovery is shown more clearly in Fig. 10 in which the average threshold shift is plotted as a function of time (with thresholds at frequencies for which no response could be obtained estimated as 10 dB above the maximum intensity of the test tone). As shown in this figure, there was an initial period of rapid recovery followed by an abrupt decrease in recovery rate. The end of this initial phase, defined as the first week in which average thresholds failed to improve by at least 2 dB, occurred at 3 wk for *M-214* and *M-267*, 6 wk for *M-337*, and 7 wk for *M-207*.

Although the audiogram of *M-267* showed no subsequent improvement, the average audiograms of the other three animals showed gradual improvement of as much as 18 dB (*M-337*) by the end of testing. Because the animals

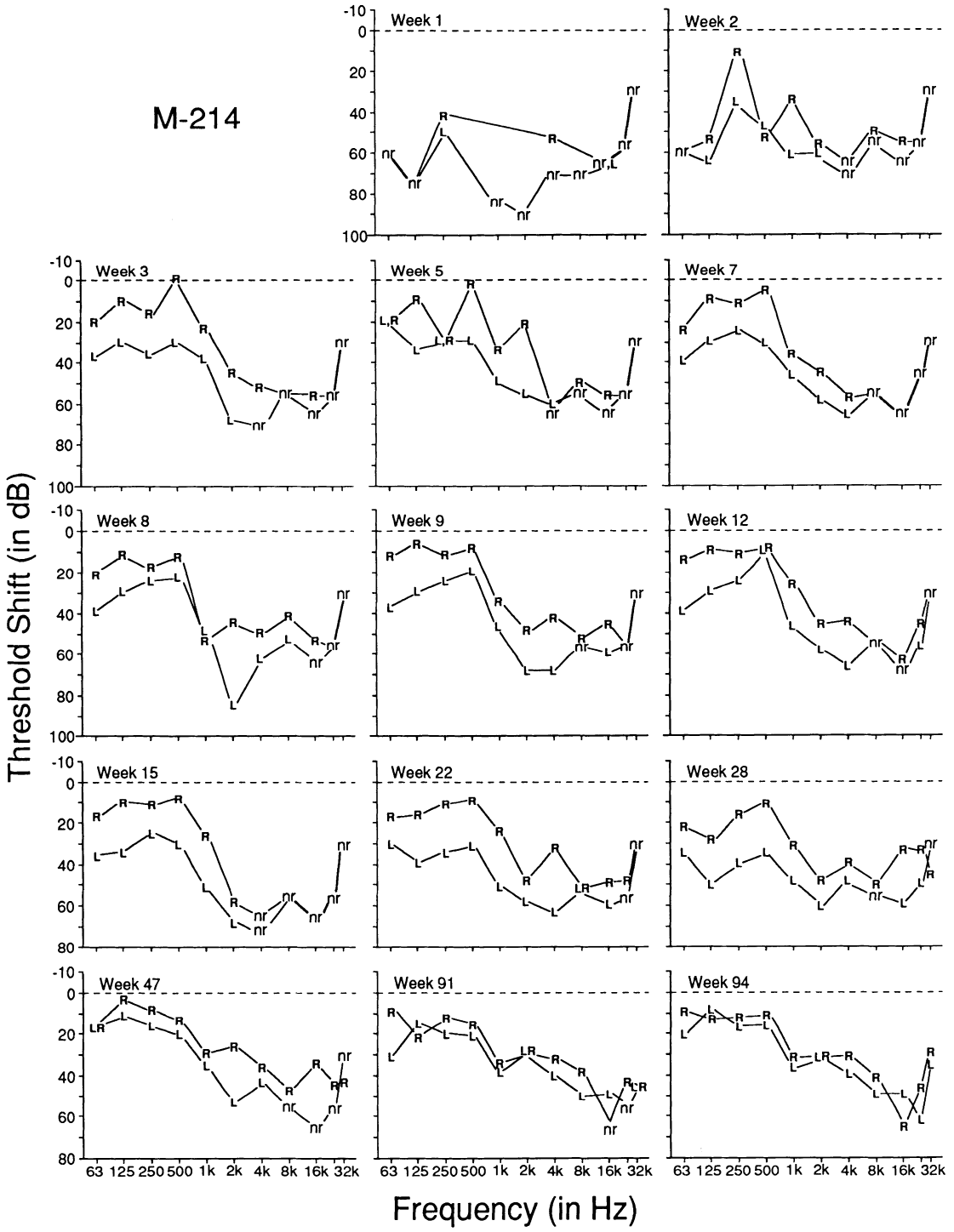

FIG. 7. Audiograms for the left (L) and right (R) ears of *M-214* taken at various times after bilateral auditory cortex lesions. Surgery was conducted in 2 stages with the left lesion made 16 wk before the right. Zero level (dashed lines) indicates the animal's preoperative hearing level; nr indicates that the animal was unable to respond to a tone at maximum intensity.

were unable to respond to a number of frequencies at maximum intensity early in testing, it is not possible to quantify precisely the improvement in hearing that occurred during the initial recovery phase. However, it is apparent from Fig. 10 that a large and rapid improvement in sensitivity occurred during the first few weeks after surgery.

It was previously noted that a transient total deafness consistently appeared only in the ear contralateral to the second of the two-stage lesions (Table 1). This difference between the two ears was also reflected in the average thresholds during the initial recovery period (Fig. 10). The fact that the ear contralateral to the first lesion, which already had a small hearing loss before the second lesion, showed a slight but consistent superiority to the other ear

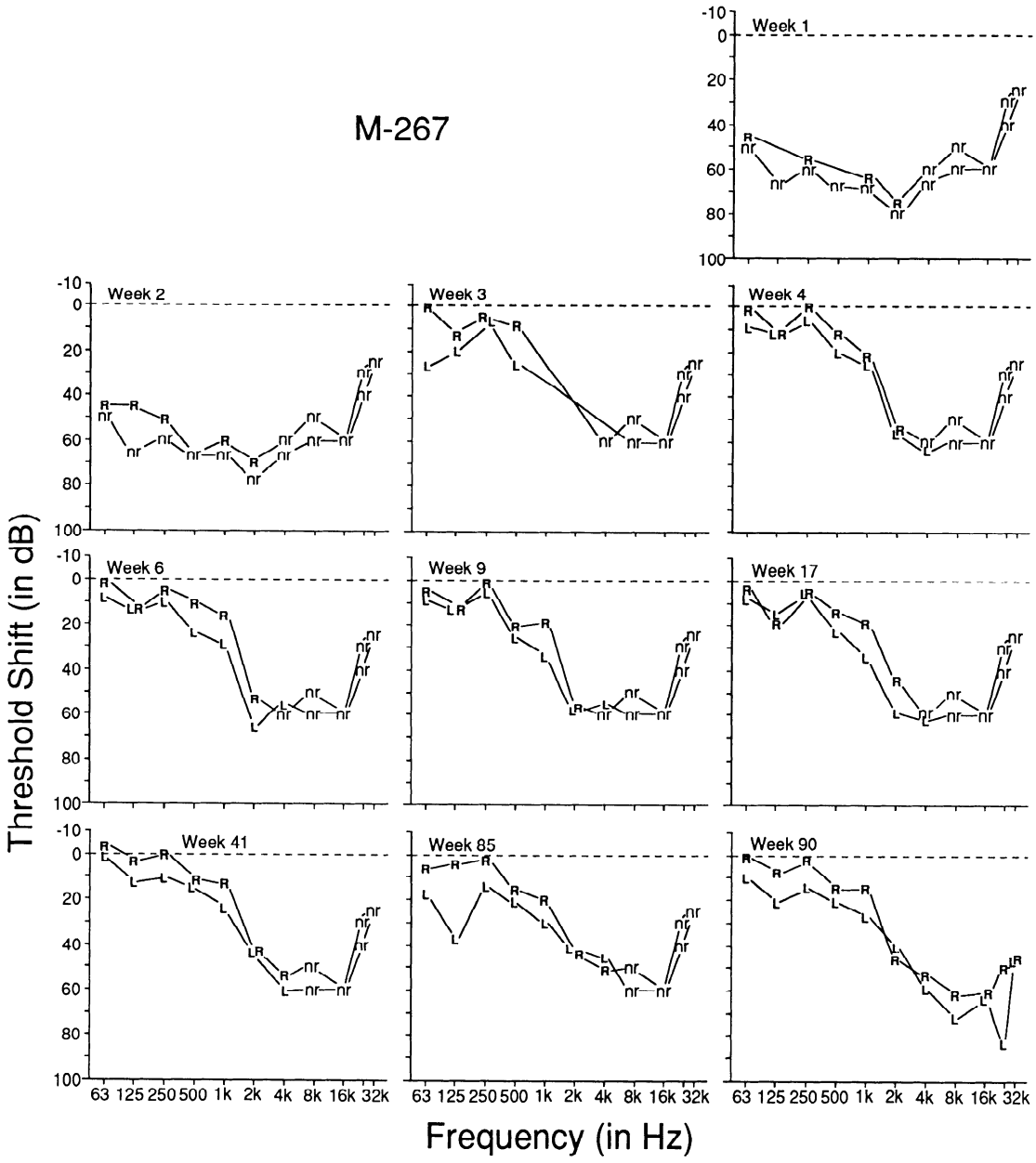

FIG. 8. Audiograms for the left (L) and right (R) ears of *M-267* taken at various times after bilateral auditory cortex lesions. Surgery was conducted in 2 stages with the left lesion made 6 wk before the right. Zero level (dashed lines) indicates the animal's preoperative hearing level; nr indicates that the animal was unable to respond to a tone at maximum intensity.

suggests that some unilateral recovery occurred between surgeries that reduced the impact of the second lesion on that ear.

PERMANENT HEARING LOSS. The effect of the lesions was to leave the monkeys with a permanent hearing loss. Interestingly, the magnitude of the loss was not the same for all frequencies nor was it random. Specifically, the loss was smallest at low frequencies, increased with frequency to a maximum in the midrange, and then decreased at higher frequencies (cf., Figs. 6–9).

The pattern of the permanent hearing loss can best be seen in the final audiograms. At this time the intensity of the test tones was increased where necessary so that thresholds could be obtained for all of the test frequencies. Averaging these audiograms reveals that the hearing loss began as a small loss at 63 Hz and progressively increased with frequency to a maximum loss in the range from 8 to 25 kHz, followed by an improvement in sensitivity at 32 kHz (Fig. 11).

The resulting pattern of hearing loss appears to be a consistent effect of bilateral auditory cortex lesions on hearing. Indeed, the same pattern was found in an earlier study in which the audiograms of monkeys with bilateral auditory cortex lesions were obtained with the use of loudspeakers instead of earphones (Heffner and Heffner 1986). The main difference between present and previous results is that the present audiograms show less variation between animals than the earlier ones, a result that may be due to the similarity of the lesions and the availability of preopera-

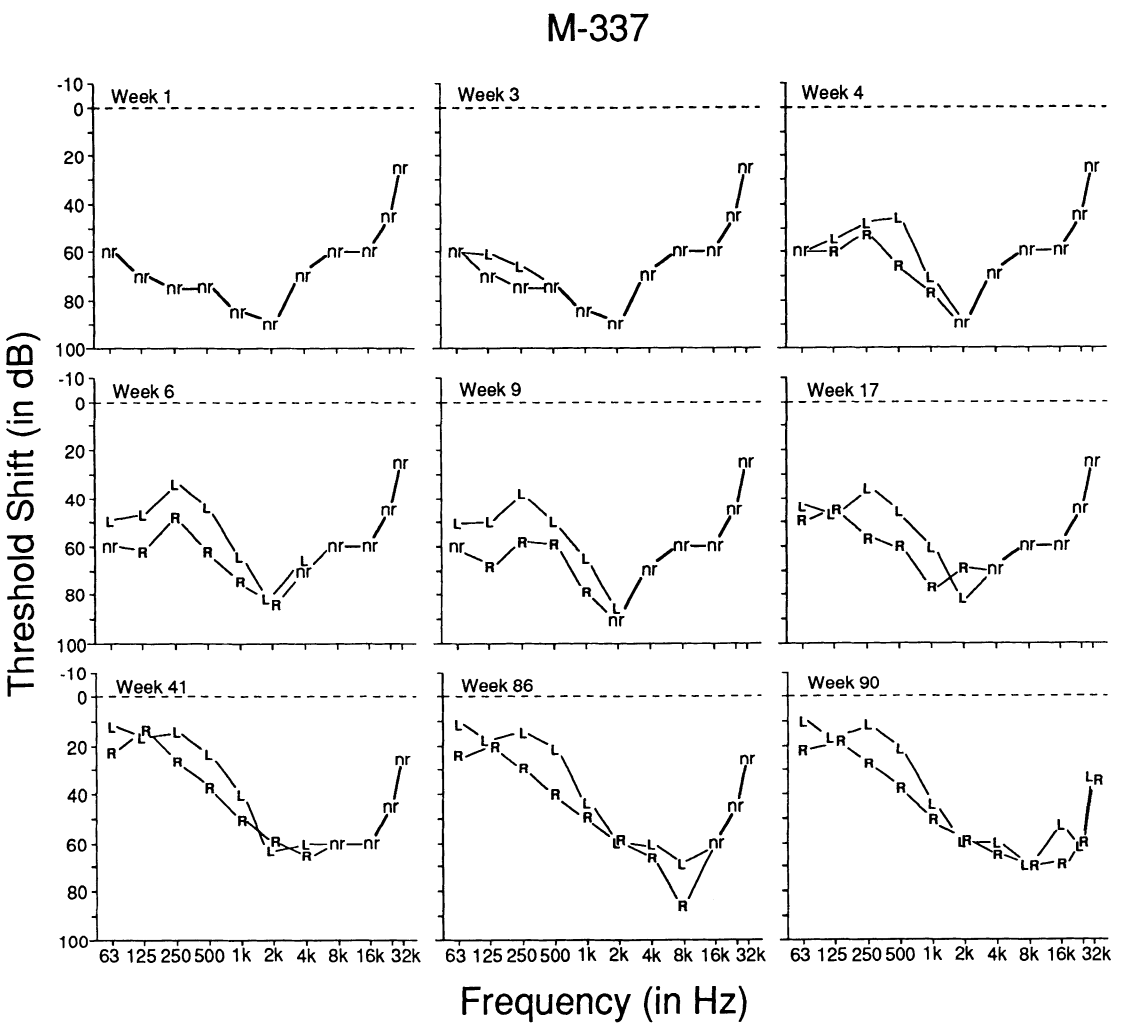

FIG. 9. Audiograms for the left (L) and right (R) ears of *M-337* taken at various times after bilateral auditory cortex lesions. Surgery was conducted in 2 stages with the right lesion made 6 wk before the left. Zero level (dashed lines) indicates the animal's preoperative hearing level; nr indicates that the animal was unable to respond to a tone at maximum intensity.

tive audiograms. Thus this pattern is not peculiar to any particular experiment but is a replicable phenomenon.

Another way of looking at the hearing loss is to graph the audiogram in terms of absolute sound pressure level (SPL) instead of threshold shift. This analysis demonstrates that the maximum hearing loss occurs in the monkey's best range of hearing (Fig. 12). Why cortical ablation should result in this pattern of hearing loss, as opposed to a constant decrease in sensitivity at all frequencies, is not known.

TABLE 1. *Duration of total inability to respond to sound after bilateral auditory cortex ablation*

Monkey	Ear	Duration of Total Deafness, days
M-207	Left	—
	Right	3
M-214	Left	4
	Right	—
M-267	Left	3
	Right	—
M-337	Left	13
	Right	18

Dashes indicate that the animal responded to sound when first tested 3 days after surgery. Note that the lesions were made in 2 stages, with the largest hearing loss occurring in the ear contralateral to the 2nd lesion.

EFFECT OF LESION SIZE. The lesions of the four animals were similar in that primary auditory cortex and the surrounding auditory fields were removed in all cases with the exception of *M-267* in which a small portion of the rostral auditory field of the left hemisphere was probably spared. Their hearing losses were also similar, and there does not appear to be any simple relation between the size of the lesions, as determined either by cortical reconstruction or thalamic degeneration, and the degree of the final hearing loss. However, it may be noted that the animal that had the longest lasting total deafness, *M-337*, also had the most complete lesions in that they included the rostral portion of the superior temporal gyrus.

SIMILARITY BETWEEN THE LEFT AND RIGHT AUDIOGRAMS OF AN ANIMAL. In each of the four monkeys it was noted that their final left and right audiograms were remarkably similar. Because the severity of a hearing loss is determined by the completeness of the lesion (Heffner and Heffner 1989b), this correspondence is probably due to the fact that

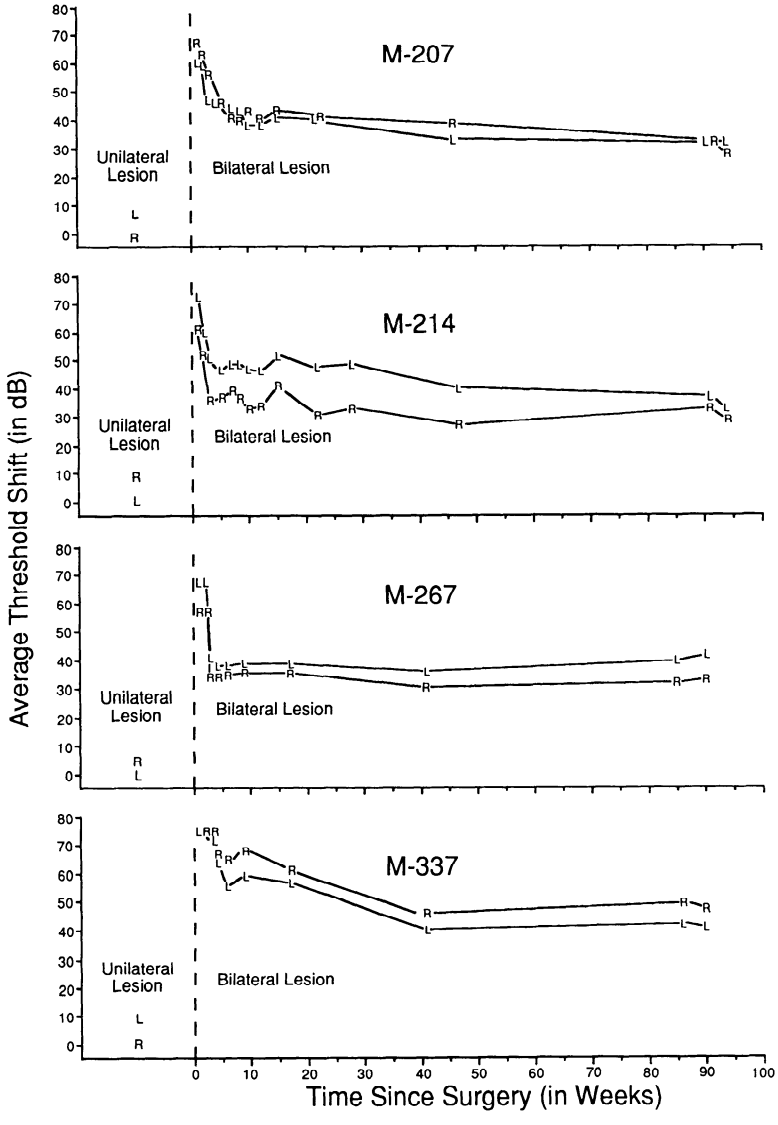

FIG. 10. Average threshold shifts of the 4 monkeys (*M-207, M-214, M-267,* and *M-337*) plotted over time. Maximal recovery occurred within 3–7 wk. L and R indicate average left and right ear thresholds, respectively. Also shown are the average threshold shifts after unilateral ablation taken during the week preceding the second lesion.

the lesions themselves were similar. However, an additional factor appears to be operating that reduces the difference between the two ears. Specifically, although sparing part of auditory cortex in one hemisphere reduces the hearing loss in the contralateral ear, to a lesser extent it also reduces the hearing loss in the ipsilateral ear. Consider for a moment the extreme case of a lesion that removes all of auditory cortex in one hemisphere while sparing all of audi-

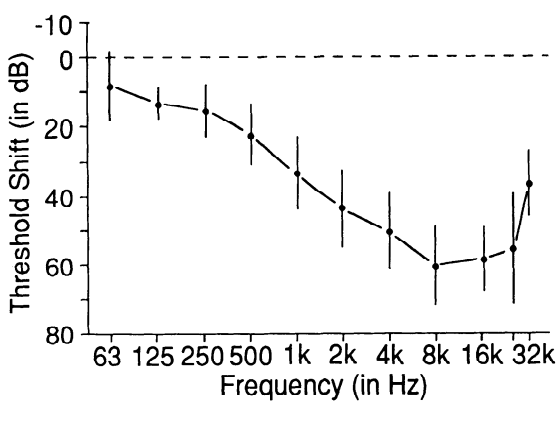

FIG. 11. Average and standard deviation of hearing losses of all 4 monkeys.

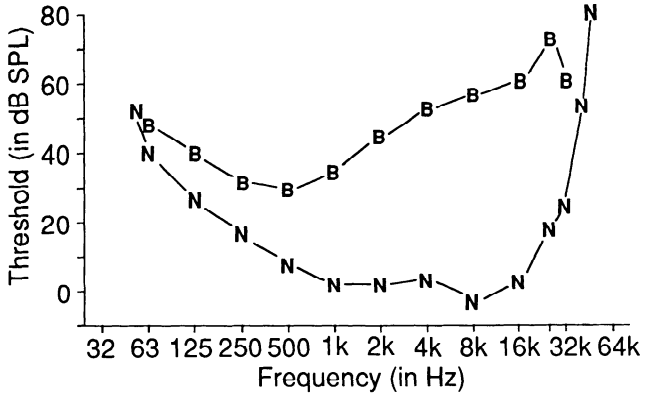

FIG. 12. Normal (N) macaque and average bilateral (B) hearing loss audiograms of 4 monkeys plotted in terms of absolute sound pressure level.

tory cortex in the other hemisphere, i.e., a unilateral lesion. Such a lesion results in a contralateral hearing loss that is much smaller than that resulting from a bilateral lesion (Heffner and Heffner 1989c). Thus the principle of primary processing of contralateral input and secondary processing of ipsilateral input by auditory cortex works to reduce the difference in hearing loss between the two ears.

The hypothesis, then, was that the final audiograms of an animal's two ears would match each other more closely than they would match the audiograms of the other animals. This was tested by determining the similarity between two audiograms by taking the sum of the absolute differences between the hearing losses at each frequency. That is, if the hearing loss at 1 kHz was 45 dB for one audiogram and 52 dB for another, the absolute difference would be 7 dB. An index of similarity could then be obtained by summing the absolute differences for all 11 frequencies. The similarity between the left and right audiograms of each animal was then compared with their similarity to those of the other animals using the Mann-Whitney *U* test.

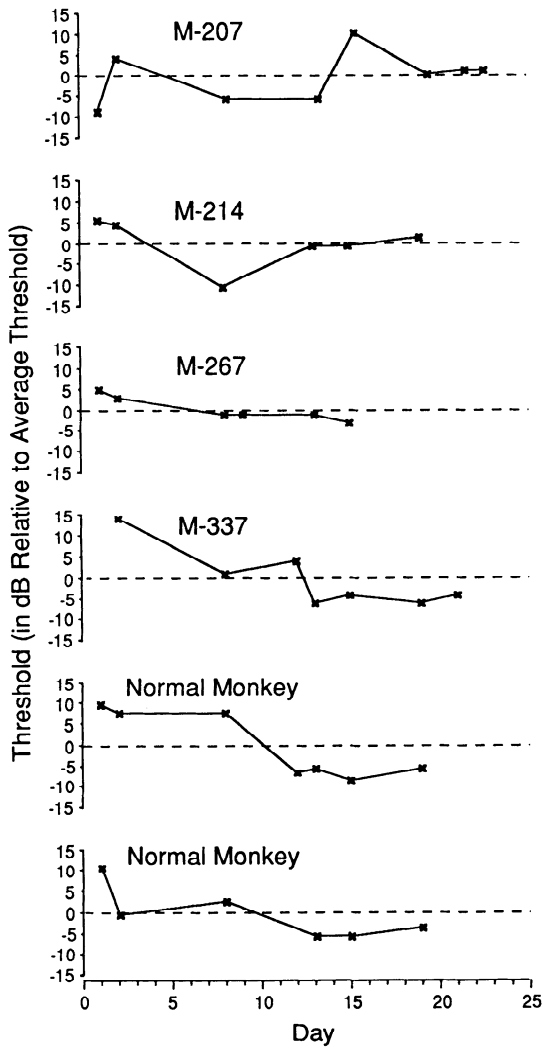

FIG. 13. Thresholds for 2 kHz taken in the left ears of 4 operated and 2 normal monkeys. Note that operated monkeys' thresholds did not show any consistent increase in variability.

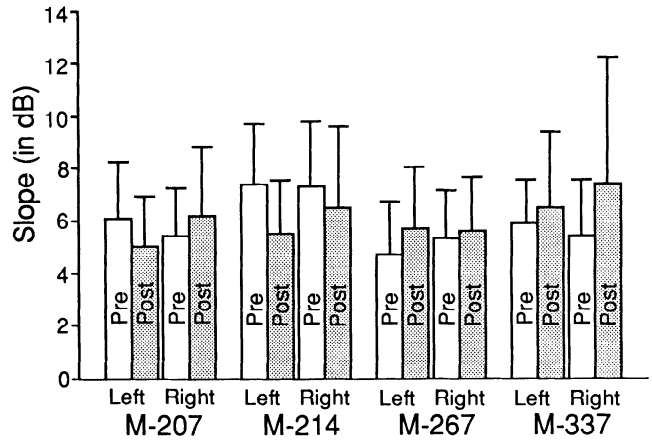

FIG. 14. Average and standard deviation of pre- and postoperative slopes of the psychophysical functions for 4 monkeys (*M-207, M-214, M-267,* and *M-337*). There was no consistent change in slopes of psychophysical functions after surgery.

The results indicated that the highest degree of similarity (i.e., the smallest differences) were between the left and right audiograms of the same animal ($P < 0.004$). Only in the case of *M-267*, which had the most asymmetrical lesion, was there a closer match with another animal. This animal's left ear more closely matched that of *M-337's* left ear, and its right ear more closely matched that of *M-207's* left ear. However, the second closest match for each of *M-267's* ears was its own opposite ear. Thus, although the severity of a bilateral hearing loss is primarily determined by the size of the contralateral lesion, the fact that each hemisphere can process input from both ears serves to reduce the difference between the two ears.

STABILITY OF THRESHOLDS. Although the overall sensitivity of the animals generally improved with time, there were occasional reversals in which sensitivity decreased. This variability led us to consider the possibility that the cortical lesions might have resulted in an increase in threshold variability.

An estimate of the short-term variability of the animals' thresholds was determined by taking repeated measures at 250 Hz and 2 kHz for both ears. These thresholds were then compared with those taken on two normal monkeys. The operated monkeys' thresholds were taken during a 3-wk period that began ~35 wk (*M-267* and *M-337*) and 40 wk (*M-207* and *M-214*) after surgery. Thus the thresholds were taken well after the initial recovery period and constituted a measure of variability of the permanent hearing loss.

The results of this analysis failed to reveal any difference in threshold variability between the normal and operated animals ($P > 0.20$, Mann-Whitney *U*). In each case the variability of the operated animals was no greater than that of the normal animals (Fig. 13). Thus it appears that the permanent hearing loss is not accompanied by any large increase in variability.

IS THERE A NONSENSORY COMPONENT TO THE HEARING LOSS? It has previously been noted that lesions of the auditory system may result in nonsensory deficits, such as deficits in vigilance or attention (Heffner and Heffner

1989c; Masterton and Granger 1988). Such a deficit could cause performance to decrease at higher stimulus intensities while leaving the intensity at which detection falls to chance unchanged thus resulting in a shallower slope to the psychophysical function. Although such a change would not have affected the thresholds in this study, which were defined as the intensity at which performance fell to chance, the slopes of psychophysical functions were analyzed to determine if the presence of a nonsensory deficit could be detected.

The monkey's pre- and postoperative psychophysical functions were analyzed by determining the range of intensity over which performance declined from 1.0 (perfect performance) to 0.2 (chance performance). Separate samples of post-operative performance were taken from both the first few weeks of testing and from the final week. These samples were compared with preoperative samples with the use of the Mann-Whitney *U* test. The results, however, did not show any major change in the slopes after bilateral ablation (Fig. 14). In only one instance was there a significant change (*M-214*, left ear), and this was a slight sharpening of the postoperative slope ($P < .01$). Thus we could find no indication that nonsensory factors played a role in the cortical deficit.

DISCUSSION

The hearing loss resulting from cortical ablation, taken together with previous studies of the role of the cortex in the detection, localization, and identification of sound, can be used to construct a picture of the function of auditory cortex in primates. The following discussion consists of a description of the hearing loss and the partial recovery of hearing, a review of cortical deafness in other mammals, and the significance of these results to our views on the role that each hemisphere plays in processing information from the two ears.

Cortical hearing loss

The present results show that bilateral ablation of the superior temporal gyrus results in a hearing loss in both ears. This hearing loss can be divided into initial and permanent stages. Initially, the animals show a large hearing loss that, in the case of a complete lesion, may be a total inability to respond to sound. In this stage, the animals undergo a period of rapid recovery during which their hearing shows considerable improvement. This stage ends within 3–7 wk after surgery after which some animals show further gradual improvement. However, the animals are left with a large permanent hearing loss that is still apparent 21 months after surgery.

The magnitude of the permanent hearing loss is not the same at all frequencies nor is it random. The average threshold shift, illustrated in Fig. 11, is smallest at 63 Hz, the lowest frequency tested, increases progressively with frequency to a maximum in the range from 8 to 25 kHz and is followed by some improvement in sensitivity at 32 kHz, the highest frequency tested. Overall, the size of the average hearing loss ranges from 8 dB at 63 Hz to 61 dB at 16 kHz. Just why there should be such variation is not apparent.

Because pure tones generally do not occur in nature, the question arises as to whether the audiograms shown here are representative of the animals' abilities to respond to natural sounds. That is, the animals may have lost the ability to respond to or to remember nonnatural or abstract sounds. That this does not appear to be the case, however, is indicated by two observations. First, the monkeys in the present study were impaired in their ability to hear broadband noise indicating that their hearing loss was not limited to tones. Second, the monkeys in a previous study were impaired in their ability to detect the vocalizations of other monkeys demonstrating the hearing loss also affects the detection of natural sounds (Heffner and Heffner 1986).

Finally, there is no indication that the deficit is the result of nonsensory factors such as attention, vigilance, or sensory neglect. As has previously been noted, nonsensory factors can affect the slope of the psychophysical function such that performance begins to decline at higher stimulus intensities, whereas threshold itself may not change (Heffner and Heffner 1989c; Masterton and Granger 1988). However, the slopes of the psychophysical functions in the present study showed no such change. Furthermore, examination of the stability of the thresholds after the initial recovery stage yielded no consistent evidence of unusual variation as might be expected in disorders of attention and vigilance. Thus the cortical hearing loss does not appear to differ from a sensory deficit such as that resulting from direct damage to the ear.

Although the cortical hearing loss resembles a peripheral hearing loss, it should be noted that the animals had no sign of peripheral impairment. First, careful postmortem inspection of their ears failed to reveal any sign of peripheral damage. Second, auditory-evoked potentials, recorded after completion of behavioral testing, gave frequency-specific auditory brain stem response latency-intensity functions that were consistent with normal peripheral auditory sensitivity (Hood and Heffner 1989).

Recovery

RECOVERY RATE. One of the main features of the cortical hearing loss is the occurrence of an initial period of rapid recovery followed by an abrupt decrease in recovery rate (see Fig. 10). In the present study this initial period lasted from 3 to 7 wk. It should be noted that the initial recovery period for the contralateral ear after unilateral ablation was 3–5 wk (Heffner and Heffner 1989c). The fact that both unilateral and bilateral lesions produce an initial recovery period of approximately the same duration suggests that similar processes of recovery are involved in both cases.

BASIS OF RECOVERY. There are a number of explanations that have been advanced to account for recovery of function (e.g., Finger et al. 1988), and their potential role in the study of auditory cortex lesions has been previously addressed (Heffner and Heffner 1989c). Two questions that any explanation of recovery must address is why cortical lesions do not permanently abolish hearing and what accounts for the initial rapid recovery.

There are two principal explanations for the partial recovery of hearing, and they are based on differing views of the role of auditory cortex in the detection of sound. The

first is that although the detection of sound is primarily mediated by subcortical neurons, these neurons require input from auditory cortex to function normally. The large initial hearing loss could be due to the disruption of these neurons brought on by the sudden removal of cortical input, i.e., diaschisis. Accordingly, the initial period of recovery could be due to the rapid but incomplete adjustment of these neurons to the loss of cortical input thus leaving the animal with elevated thresholds.

The second explanation is that auditory cortex is necessary for the detection of sound and that the persistence of hearing after bilateral ablation is the sign of an incomplete lesion. According to this view, the initial large hearing loss is due to the loss of functioning of the remaining auditory cortical neurons as the result of surgical shock. The subsequent partial recovery occurs as the cortex recovers from surgery and, perhaps, as the remaining neurons take over some of the functions of the ablated neurons.

Although the lesions in this study are considered complete by current standards, it must be noted that auditory cortex in the macaque has not been completely explored electrophysiologically (Brugge 1982). This is partly due to the fact that it lies buried in the sylvian fissure making access difficult. As a result some areas of auditory cortex have never been described in detail. An example of this is the rostral portion of the superior temporal gyrus, which is known to be auditory by virtue of its cytoarchitecture and cortical connections as well as by the fact that ablation of this area results in a hearing loss (Heffner and Heffner 1989b; Mesulam and Pandya 1973; Pandya and Sanides 1973). Thus it can be argued that the extent of auditory cortex in the macaque is not precisely known.

The idea that the recovery of hearing may be mediated by cortical sparing is supported by the observation that incomplete cortical lesions result in a smaller hearing loss. This fact was demonstrated in a previous study that showed that smaller hearing losses could be produced by bilateral lesions that spared the rostral portion of the superior temporal gyrus on one side (Heffner and Heffner 1989b). Thus the sparing of cortex that lies outside the central auditory core and those belt regions that have been explored contributes to the preservation of hearing.

At the present time it is not possible to decide conclusively between the two explanations. Although we favor the idea that the detection of sound is primarily a subcortical function, we cannot rule out the possibility that larger cortical lesions might result in a complete hearing loss. Aside from exploring the effect of larger lesions, one line of investigation that would shed light on this problem would be to determine whether cortical lesions affect the response properties of subcortical auditory neurons.

Cortical deafness in mammals

It should be noted that there are large species differences in the effect of cortical lesions on absolute thresholds. Indeed, these differences are so large that it is no longer possible to refer to the function of auditory cortex without specifying a particular species. Bilateral ablation of neocortex in the Virginia opossum has no effect on absolute thresholds, whereas bilateral auditory cortex lesions in cats, dogs, and ferrets result in small hearing losses (e.g., Heffner and Heffner 1984, 1986; Kavanagh and Kelly 1988; Ravizza and Masterton 1972). The one other species in which bilateral lesions result in hearing losses of the magnitude seen in macaques is man.

In spite of the rarity of bilateral temporal lobe lesions in humans, a number of such cases have been reported (for a review, see Heffner and Heffner 1986). Although the exact symptoms depend on the locus and extent of the lesions, it is not difficult to find cases that show the same features as those seen in monkeys. First, a sudden loss of hearing, described as a total inability to hear sounds, is often reported coincident with the cerebrovascular accident. Indeed, patients have reported that the world became "strangely silent" and that they were unable to hear the sound of their automobile engine or of water from a faucet (e.g., Albert et al. 1972; Jerger et al. 1969; Leicester 1980). Second, the hearing ability of the patients eventually improves although the time course of this recovery has rarely been followed. Finally, the recovery is incomplete, and the patients show a hearing loss that, like the monkeys', becomes progressively greater at higher frequencies (e.g., Albert et al. 1972; Leicester 1980; Michel et al. 1980).

The increasing effect of cortical lesions on absolute thresholds as one moves from opossum to carnivores to macaques and humans suggests that there has been a progressive encephalization of the detection of sound in man's lineage. This does not necessarily mean that the lower auditory centers have relinquished their role in detection but only that the cortex has come to play a more essential role in that task. Indeed, the principle of encephalization has long been evoked to explain the observation that cortical lesions result in greater auditory impairments in monkeys and humans than in other animals (e.g., Davis 1951).

Although it is generally agreed that there has been a proportional increase in the size of neocortex in man's lineage, i.e., that structural encephalization has occurred, the concept of functional encephalization and the corticalization of function has been severely criticized (e.g., Jerison 1976). One criticism is that many animals, such as birds, are able to detect sounds without the benefit of neocortex (e.g., Jenkins and Masterton 1979), and it would seem unlikely that a newly evolved structure would take over a function that phylogenetically older structures were capable of handling. Nonetheless, the fact remains that the increase in the relative size of neocortex does seem to be accompanied by an increase in its role in the detection of sound. A more serious criticism, however, is that too few species have been examined to test this concept properly. Indeed, the sequence of "opossum, cat/dog/ferret, monkey, human" is only a slight improvement over the "rat, cat, monkey, human" sequence that has been used in past discussions of encephalization. However, functional encephalization is currently believed to apply to the somatosensory and motor systems and species differences in the effect of lesions in visual cortex suggest that it could apply to vision as well (e.g., Masterton and Berkley 1974; Masterton and Glendenning 1978). In any case, it is clear that the role of auditory cortex varies significantly between species and that the belief that macaques are an appropriate species to use if one wishes to extrapolate to humans remains valid (cf. Ferrier 1878).

Role of each hemisphere in the processing of information from the two ears

Although each hemisphere receives information from both ears, the auditory system, and auditory cortex in particular, has been described as being functionally unilateral (Glendenning and Masterton 1983). This view is based on the observation that the majority of cells in auditory cortex respond best to sounds in the contralateral sound field and, more compellingly, on the fact that unilateral damage to auditory cortex results in sound-localization deficits confined to the hemifield contralateral to the lesion (Jenkins and Masterton 1982; Jenkins and Merzenich 1984; Kavanaugh and Kelly 1987; Phillips and Irvine 1983; Thompson and Masterton 1978). The present results taken with those of previous studies suggest that, although this view may be valid for sound localization, it does not necessarily apply to other auditory abilities such as sound detection and recognition. For these abilities auditory cortex may be more appropriately characterized as being asymmetrically bilateral—that is, although each hemisphere is involved with both ears, the degree of involvement is greater with the contralateral ear.

The concept that auditory cortex is functionally bilateral with each hemisphere more strongly involved with the contralateral ear is based on several lines of research. First, electrophysiological studies have shown that, although unit activity is driven only or driven best by contralateral sounds, a large number of units are excited by ipsilateral stimuli. For example, in a study of unit activity in cat auditory cortex (AI), Phillips and Irvine (1983) found that 43% of the units were only excited by stimulation of the contralateral ear whereas the number that could be excited only by ipsilateral stimulation was 6%, a finding that demonstrates the predominately contralateral nature of the auditory system. However, 39% of the units could be excited by stimulation of either ear (with contralateral stimulation usually producing the strongest response), whereas the remaining 12% were excited by binaural stimulation. Thus, although the cortex is strongly contralateral, the fact that almost one-half of the units in AI can be excited by ipsilateral stimulation alone indicates that there is a large and significant ipsilateral excitatory component.

Second, the effects of auditory cortex lesions on the detection of sound clearly indicate the presence of bilateral representation with a contralateral ear advantage. Unilateral ablation of auditory cortex results in a hearing loss for sounds presented to the ear contralateral to the lesion, whereas thresholds in the ipsilateral ear are unaffected (Heffner and Heffner 1989c). This result is a demonstration of the strong involvement of the cortex in the detection of sounds presented to the contralateral ear.

Bilateral involvement of the cortex in sound detection is demonstrated by the fact that the hearing loss after a unilateral lesion is much smaller than that after a bilateral lesion. That is, the size of a cortical hearing loss is greatly reduced by the presence of an intact ipsilateral hemisphere. Just how much it is reduced is demonstrated by noting the increase in the hearing loss when the ipsilateral cortex is subsequently removed. This can be seen in Fig. 10 in which ablation of auditory cortex in the previously intact hemisphere resulted in a large increase in the average hearing loss that, on average, rose from 8 to 34 dB. Thus each hemisphere plays a significant role in the detection of sounds presented to the ipsilateral ear that serves to limit the effect of a unilateral lesion.

These results suggest that auditory cortex may be organized differently for different auditory functions. For the localization of sound, the evidence indicates that each hemisphere is involved in locating sound sources in the contralateral hemifield. The detection of sound, on the other hand, is based not on hemifields, but on ears—each hemisphere is primarily involved in the detection of sound by the contralateral ear and secondarily involved with detection by the ipsilateral ear. In addition, the notion that different auditory abilities are processed differently is also suggested by a third line of research regarding the perception of communicative vocalizations by Japanese macaques. We have shown elsewhere that the perception of vocalizations by Japanese macaques is a cortically dependent task that is weakly lateralized to the left hemisphere. Specifically, lesions of left auditory cortex disrupt the ability of monkeys to discriminate vocalizations, but the right hemisphere soon takes over and the animals recover their ability to make the discrimination. That this discrimination is dependent on the cortex is demonstrated by the fact that bilateral ablation totally and permanently abolishes the monkeys' ability to discriminate the vocalizations.

Recently, we have been investigating the effect of unilateral ablation on the ability of monkeys to discriminate vocalizations that are presented independently to each ear. The results of this study show that the animals are impaired only when the vocalizations are presented to the ear contralateral to the lesion (Heffner and Mooney 1989a). However, when the vocalizations are presented dichotically with a sufficient time and/or intensity difference to produce a lateralized effect, there is no deficit in performance regardless of the hemifield to which the vocalizations are lateralized. That is, although the vocalizations are lateralized to the hemifield contralateral to the lesion and are therefore not localizeable, the animals retain the ability to perceive them. In other words, they can recognize sounds that they cannot localize (Heffner and Mooney 1989b).

What this result suggests is that different types of information are processed differently in the auditory system. Specifically, locus information is analyzed at the level of the superior olivary complex and sent to the hemisphere contralateral to the hemifield in which the sound occurred. Information necessary for detection and recognition is sent to the hemisphere contralateral to the ear receiving the sound—if it is audible in both ears then it is sent to both hemispheres. Thus there appears to exist separate and parallel processing of information within the auditory system such that locus information is processed by one hemisphere, whereas nonspatial perceptual information may be processed by either or both hemispheres.

This view of the auditory system gives rise to the following picture: A macaque with a unilateral auditory cortex lesion would show only slight impairment in its ability to detect a communication call emanating from the hemifield contralateral to its lesion and would be able to recognize the call as long as it was audible in the ear ipsilateral to the

lesion but would be unable to locate its source. Calls emanating from the hemifield ipsilateral to the lesion would be detected, recognized, and localized normally. An animal with a bilateral lesion, however, would be seriously impaired in its ability to detect a call and would be unable to recognize it or locate its source regardless of location.

We thank B. Porter for help in this study.

This research was supported by National Institutes of Health Grants NS-12992 and HD-02528 to the Bureau of Child Research, University of Kansas.

Address for reprint requests: H. E. Heffner, Dept. of Psychology, University of Toledo, Toledo, OH 43606.

Received 28 August 1989; accepted in final form 14 February 1990.

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