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Effect of Restricted Cortical Lesions on Absolute Thresholds and Aphasia-Like Deficits in Japanese Macaques

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The effect of small bilateral cortical lesions on pure-tone audiograms and on the ability to discriminate between two types of Japanese macaque coo vocalizations was determined in four Japanese macaques (*Macaca fuscata*). A lesion that included the middle portion of the superior temporal gyrus of both hemispheres, that is, the primary and secondary auditory areas, resulted in a partial hearing loss as well as an inability to discriminate the vocalizations. Lesions that included the ventral portions of the superior temporal gyrus of both hemispheres but spared auditory cortex on one side also resulted in a partial hearing loss but had either a small effect or no effect on the ability to discriminate the vocalizations. Bilateral ablation of the dorsal superior temporal gyrus and adjacent parietal and occipital areas did not appear to result in a hearing loss and had no effect on the ability to discriminate the vocalizations. These results suggest that a hearing loss may be produced by lesions that involve small portions of the ventral two-thirds of the superior temporal gyrus bilaterally although the resulting loss is not as great as that observed with larger lesions. However, the aphasia-like deficit appears to result from a lesion of primary and/or secondary auditory cortex.

Recent studies have revealed two effects of auditory cortex lesions on hearing in Japanese macaques (*Macaca fuscata*). The first is a substantial hearing loss that results from bilateral lesions of the superior temporal gyrus (H. Heffner & Heffner, 1986b). Typically, this deficit begins as an initial total deafness followed by partial recovery, particularly at lower frequencies, with a permanent hearing loss persisting in the midfrequency range. The second effect is a permanent inability to discriminate species-specific vocalizations (H. Heffner & Heffner, 1984, 1986a). This deficit is demonstrated by the inability of Japanese macaques to discriminate two types of their "coo" call, an inability that suggests that the lesions may have resulted in a deficit analogous to sensory aphasia.

In analyzing these effects, it soon becomes apparent that all the animals involved in these studies received large lesions that included most of the superior temporal gyrus. This observation leads to the question of whether these deficits might be produced with smaller lesions, particularly lesions restricted to areas around or within primary auditory cortex. Further, all of the animals with lesions examined so far have shown both of the effects. Although it does not appear that the inability to discriminate the coos is due simply to the hearing loss (H. Heffner & Heffner, 1986a, in press), the question remains as to whether the two deficits can be dissociated. Thus the purpose of the present report is to describe the results of an initial study on the effect of restricted cortical lesions on the hearing abilities of Japanese macaques.

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Method

Subjects

Five male Japanese macaques (*Macaca fuscata*) ranging in age from 5 to 6 years old (adolescent) were used in this study. Four of the animals (M-136, M-235, M-243, and M-319) received bilateral cortical lesions, whereas the 5th animal (M-337) was used as a normal control. The animals had been born and reared in a 58-acre outdoor primate colony (Arashiyama West Institute). The animals were individually housed in primate cages with free access to food and were trained using water as a reward. The ears of each animal were examined to ensure that they were free of damage or disease.

Four of the animals had been used in previous auditory research, the results of which have been reported elsewhere (M-235, M-243, and M-319 in H. Heffner & Heffner, 1986a; M-337 in H. Heffner & Heffner, 1986b).

Surgical and Histological Procedures

Surgery

Following preoperative training and testing involving the discrimination of primate vocalizations, 4 of the monkeys received two-stage lesions 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 atropine sulfate and 100 mg 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 with the aid of a surgical microscpe. 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 to the pia. The muscle was then sewn together, and a topical antibiotic powder (Neo-Predef, Upjohn) was sprinkled on top of the muscle. The animal was returned to its home cage and was given acepromazine as needed to minimize discomfort.

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. Each 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 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) that a monkey could comfortably place its mouth on both spouts. The two spouts were electrically isolated from each other so that they could be attached to an electronic "contact" switch, which detected when an animal placed its mouth on them and completed the circuit. 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 (Grason-Stadler Model 700) connected to the two spouts. A 60-W light was mounted above the chair, and the entire apparatus was located in a sound chamber (2.7 × 2.5 × 2.0 m). A microcomputer was used for behavioral programming.

Discrimination of Species-Specific Sounds

Acoustic stimuli. The monkey vocalizations used in this discrimination were the same 15 coos previously used to demonstrate the effect of unilateral and bilateral auditory cortex lesions in Japanese macaques (H. Heffner & Heffner, 1984, 1986a, 1986b, in press). These vocalizations were originally recorded by Green (1975) and had been used by Petersen and his colleagues to demonstrate a right-ear advantage in Japanese macaques for the perception of vocalizations (e.g., Petersen et al., 1984; Petersen, Beecher, Zoloth, Moody, & Stebbins, 1978). They consist of seven "smooth early high" (SE) and eight "smooth late high" (SL) coos. The coos are distinguished by the temporal position (i.e., either early or late) of the peak fundamental frequency of the frequency-modulation portion of the call. The SE coo is emitted by all ages and sexes and appears to be a "contact-seeking" call, whereas the SL coo is produced primarily by estrous females seeking male consorts. The two types of coos are thus acoustically similar sounds with different meanings.

The physical characteristics of the coos have been described in detail elsewhere (e.g., Petersen, 1981). They have an average fundamental onset frequency of 625 Hz (ranging from 500 Hz to 750 Hz), with an average peak frequency of 865 Hz (ranging from 600 Hz to 1350 Hz), and their harmonic structure is such that all the significant energy is below 4 kHz.

The tape-recorded vocalizations were filtered (100 Hz to 5 kHz, Krohn-Hite 3202 filter) to prevent harmonic distortion owing to digitization (i.e., aliasing) and digitized with an eight-bit microcomputer (Apple II microcomputer and Mountain Hardware analog-to-digital/digital-to-analog converter card) at a 20-kHz sample rate, edited to remove the background noise that preceded and followed the coos on the tape recording, and stored on computer disk for digital-to-analog playback. The sounds were played back at a 20-kHz sample rate through a band-pass filter (100 Hz to 5 kHz), an amplifier, and a high-fidelity loudspeaker (Acoustic Research 3a) located 1.5 m in front of the primate chair. The quality of the digital reproduction was such that human observers did not notice any significant differences between the taped and digitized reproductions. More significant, monkeys that had been originally trained to discriminate tape-recorded coos transferred without difficulty to the digitized reproductions.

Behavioral procedure. An avoidance procedure was used in which a thirsty monkey was seated in the primate chair and trained to place its mouth on the waterspout, an action that 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. The SE coos were arbitrarily designated as the "warning" stimuli, and the monkeys were taught to break contact with the spout whenever an SE coo was presented.

Initial auditory training consisted of presenting one of the SE coos at random intervals from 7 to 49 s apart. Each presentation consisted of playing the coo three times, following which a mild electric shock was delivered through the waterspout, and the overhead light was momentarily turned on. After several stimulus presentations, the animals learned to avoid the shock by breaking contact with the spout whenever the coo was presented and to return to the spout when the light signaled the end of the shock.

The animals were then trained to distinguish one SE coo from one SL coo by presenting one of the two coos every 7 s, with the SE coo presented randomly 25% of the time. Only the SE coo was followed by shock. Once an animal had learned to break contact only when the SE coo was presented, additional examples of both types of coos were presented until the animals had learned to discriminate the entire set of 15 coos. Thus the animals demonstrated the ability to distinguish the two types of coos by breaking contact with the spout following presentation of any SE coo (i.e., a "warning" signal) and by maintaining contact after presentation of any SL coo (a "safe" signal).

The final test procedure consisted of presenting 3.5-s trials once every 7 s (i.e., with a 3.5-s intertrial interval). Each trial was either an SL (safe) trial or an SE (warning) trial. SE trials occurred randomly from one to seven trials after the previous warning trial with occasional "catch" trials inserted in which a safe stimulus was presented on the seventh trial. The number of warning trials given in each of the trial periods was adjusted so that each trial period had the same probability (0.25) of being a warning trial. To ensure optimal performance, the level of shock was varied to determine the setting for each animal that yielded the highest score. A typical session consisted of the presentation of 35 to 56 warning trials and 105 to 168 safe trials.

The response of an animal on each trial, that is, 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 trial. Basing the response criterion on the last 200 ms of the trial allowed the animal sufficient time to break contact with the spout following presentation of a warning stimulus. 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. 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.

The scores for a session were averaged separately for each of the 15 coos and rank ordered to allow a detailed comparison of the relative performances. An overall measure of performance was calculated by taking the average of the SE and SL scores and calculating a performance ratio with the formula $(S - W)/S$; where S is the average of the SL (safe) scores and W is the average of the SE (warning) scores. In a trained animal, this measure ranged from 1.0 (indicating good discrimination of the SE and SL coos) to about 0 (indicating failure to discriminate).

The statistical reliability of the scores was evaluated two ways. First, chance probability for a session was calculated using the binomial distribution by comparing the occurrence of responses during the warning trials with the occurrence of responses during the safe trials (i.e., the false positive rate; Hays, 1963). Second, the scores for the individual coos were rank ordered, and the Mann-Whitney U test (Siegel, 1956) was used to determine the probability that the two groups of coos were being responded to differently.

Absolute Thresholds for Tones

Acoustic stimuli. 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 (250 ms on, 250 ms off, rise–decay of 50 ms for 63 Hz to 500 Hz and 20 ms for higher frequencies). 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 Hz to 500 Hz), a 4-in. (10.2-cm) midrange loudspeaker (for frequencies from 1 Hz to 4 kHz), or a ribbon tweeter (for frequencies from 8 kHz 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 level (re 20 $\mu N/m^2$) for frequencies from 63 Hz to 16 kHz was measured with a 1-in. (2.54-cm) microphone (Brüel & Kjaer 4131), sound level meter (Brüel & Kjaer 2203), and octave filter (Brüel & Kjaer 1613), whereas 32 kHz was measured with a 0.25-in. (0.64-cm) microphone (Brüel & Kjaer 4135), preamplifier (Brüel & Kjaer 2618), microphone amplifier (Brüel & Kjaer 2608), and filter (Brüel & Kjaer 1613 or Krohn-Hite 3202). Sound pressure measurements were taken with the primate chair and waterspouts in their normal position by placing the microphone in the region occupied by an animal's ears during testing.

Psychophysical procedure. The general training and testing procedures were identical to those described earlier for the discrimination of the coos. In this case the warning signal was a 3.5-s train of tone pulses, whereas the safe signal was silence.

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 ensure optimal performance. Threshold was defined as the lowest intensity that could be detected above the 0.01 level of chance, a definition that was usually associated with a performance ratio of 0.20. Testing was considered complete when thresholds obtained in two different sessions were within 4 dB of each other.

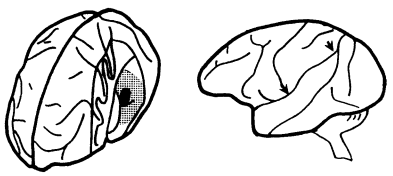

Figure 1. Location of auditory cortex in the macaque. (Left: drawing of a Japanese macaque brain with the frontoparietal operculum removed to show the location of primary [blackened] and the surrounding belt of secondary [stippled] auditory cortex. More secondary auditory cortex is buried in the circular sulcus bounding the insula. Right: drawing shows the lateral surface of a Japanese macaque brain. Arrows mark the approximate limits of the primary and secondary auditory fields of the superior temporal plane [drawings based on the electrophysiological study of Merzenich & Brugge, 1973, and taken from H. Heffner & Heffner, 1986b].)

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 (H. Heffner & Heffner, 1986b).

Results

Anatomical Results

Location of Auditory Cortex

The locus and extent of auditory cortex have been studied in the rhesus macaque by evoked response (Woolsey, 1972; Woolsey & Walzl, 1982), microelectrode recordings (Merzenich & Brugge, 1973), cytoarchitectural analysis (Galaburda & Pandya, 1983; Pandya & Sanides, 1973), and by tracing thalamocortical connections (Mesulam & 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 third of the superior temporal plane and is surrounded by four secondary auditory fields (Figure 1; for a review, see H. Heffner & Heffner, 1986b).

Other parts of the superior temporal gyrus may also be auditory. 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, which indicates that there are at least two other auditory fields (Merzenich & Brugge, 1973). In addition, data on the cytoarchitectonics and cortical connections of the auditory fields suggests that all of the superior temporal gyrus with the exception of the caudal tip, which receives no significant projections from the medial geniculate, may have auditory functions (Galaburda & Pandya, 1983; Mesulam & Pandya, 1973).

Although precise localization of auditory lesions requires electrophysiological mapping before making the lesion, the resulting pattern of thalamic degeneration gives a general

picture of the extent of a lesion (for details, see H. Heffner & Heffner, 1986b). 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

In order to avoid the problem of trying to make small bilaterally symmetrical lesions, 3 of the monkeys (M-243, M-235, and M-319) received a large lesion of the superior temporal gyrus in one hemisphere followed by a restricted superior temporal gyrus lesion in the other hemisphere. Because performance on the tasks used here is not permanently impaired by unilateral lesions, it is assumed that any deficit was due to the bilaterally ablated areas.

M-136. This monkey received a left hemisphere lesion that included the dorsal half of the superior temporal gyrus and adjacent parietal, preoccipital, and middle temporal areas (Figure 2). Cortical reconstruction and thalamic degeneration indicated that the primary auditory and dorsal secondary auditory fields were removed. The animal received a similar though smaller lesion in the right hemisphere 10 weeks later. This second lesion included only a small part of the primary and dorsal secondary auditory fields. The common feature of these lesions is the removal of the superior temporal gyrus dorsal to primary and secondary auditory cortex and the immediately adjacent parietal, occipital, and temporal areas.

M-243. This monkey received a right hemisphere lesion of the superior temporal gyrus that spared the temporal tip (Figure 3). Analysis of the lesion indicated that the primary and secondary auditory fields were completely removed. The animal received a much smaller lesion of the left superior temporal gyrus 31 weeks later that removed primary auditory cortex and most of the surrounding secondary auditory fields. The common feature of these lesions is the removal of primary and secondary auditory cortex.

M-235. This monkey received a left lesion of the superior temporal gyrus, sparing the temporal tip, that removed all of primary and secondary auditory cortex (Figure 4). The animal received a much smaller lesion of the right superior temporal gyrus 30 weeks later. This second lesion was restricted to the ventral portion of the superior temporal gyrus outside the primary and secondary auditory fields. The serial reconstruction (Figure 4) indicates that it extended somewhat more ventrally than the left lesion with the result that the common feature of these lesions is a ventral superior temporal gyrus lesion of slightly smaller extent than indicated by the size of the right lesion itself.

M-319. This monkey received a right lesion of the superior temporal gyrus, sparing the temporal tip, that removed all of primary and secondary auditory cortex (Figure 5). This animal received a smaller left lesion 6 weeks later that was restricted to the portion of the superior temporal gyrus ventral to the primary and surrounding secondary auditory fields. The common feature of these lesions is the removal of a portion of the superior temporal gyrus ventral to the primary and secondary auditory fields. Although this lesion is similar to that of M-235, the serial reconstruction indicates a greater amount of overlap in the locus of the left and right lesions, resulting in a larger bilateral lesion.

Behavioral Results

Absolute Thresholds

Previous work had indicated that large bilateral lesions of the superior temporal gyrus initially result in a total inability to respond to sound (H. Heffner & Heffner, 1986b). In the present experiment, none of the animals ever appeared to be totally deaf and each was able to hear the coos when first tested either 5 days (M-235 and M-243) or 7 days (M-136 and M-319) after surgery. However, 3 of the animals did have hearing losses, which were revealed by their pure tone thresholds (Figure 6).

The animal with bilateral removal of primary and secondary auditory cortex (M-243) had a hearing loss as did the 2 monkeys with bilateral lesions of the ventral superior temporal gyrus (M-235 and M-319). Of the two latter cases, the animal with the larger bilateral lesion (M-319) had the greater hearing loss. The fourth animal, M-136, which received a dorsal superior gyrus lesion was unimpaired in its ability to hear 4 kHz 3 weeks after surgery as its threshold at this frequency had not changed significantly from a previous audiogram taken following the unilateral ablation. Although it is possible that a hearing loss may have occurred at other frequencies, 4 kHz has been consistently affected in all previous cases of cortical hearing loss (H. Heffner & Heffner, 1986b, and in this study). It is therefore likely that this animal's hearing was not impaired by the bilateral lesion.

Discrimination of Species-Specific Sounds

Previous work has demonstrated that large bilateral lesions of the superior temporal gyrus result in a permanent inability to discriminate between the coos (H. Heffner & Heffner, 1986a). In the present study only the monkey in which primary and secondary auditory cortex were removed bilaterally (M-243) had this deficit. As shown in Figure 7, M-243 was unable to discriminate the coos when tested during the first week after surgery. Following determination of the animal's audiogram, the monkey was given training on single pairs of coo sounds, and the intensity of the coos was increased by 15 dB to compensate for its hearing loss. At no time was this animal able to discriminate one SE coo from one SL coo despite weeks of training. The animal was then tested on the full set of coos for seven more sessions, during which it again failed to discriminate them.

The 2 animals with bilateral ventral lesions that spared auditory cortex on one side were successfully able to discriminate the coos when first tested 5 days (M-235) and 10

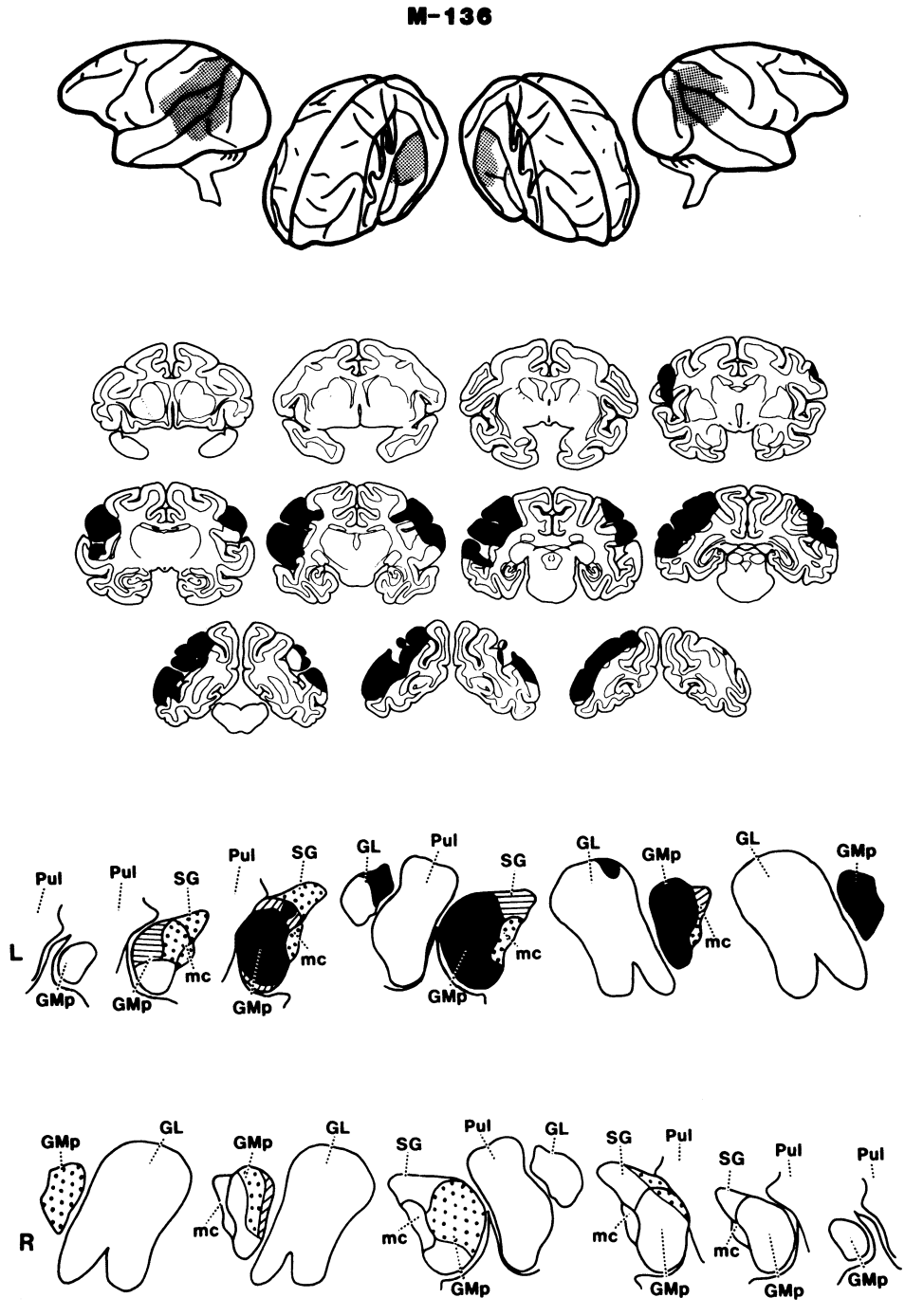

Figure 2. Cortical reconstruction and medial geniculate degeneration for M-136. (Top: reconstruction of cortical lesion [stippled areas] showing surface views and views of the superior temporal plane with the parietal operculum removed. Middle: coronal sections 3.6 mm apart with ablated areas shown in black. Bottom: retrograde degeneration in the vicinity of the medial geniculate. The left thalamic sections [top] are shown posterior to anterior and the 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, supragenículate 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.)

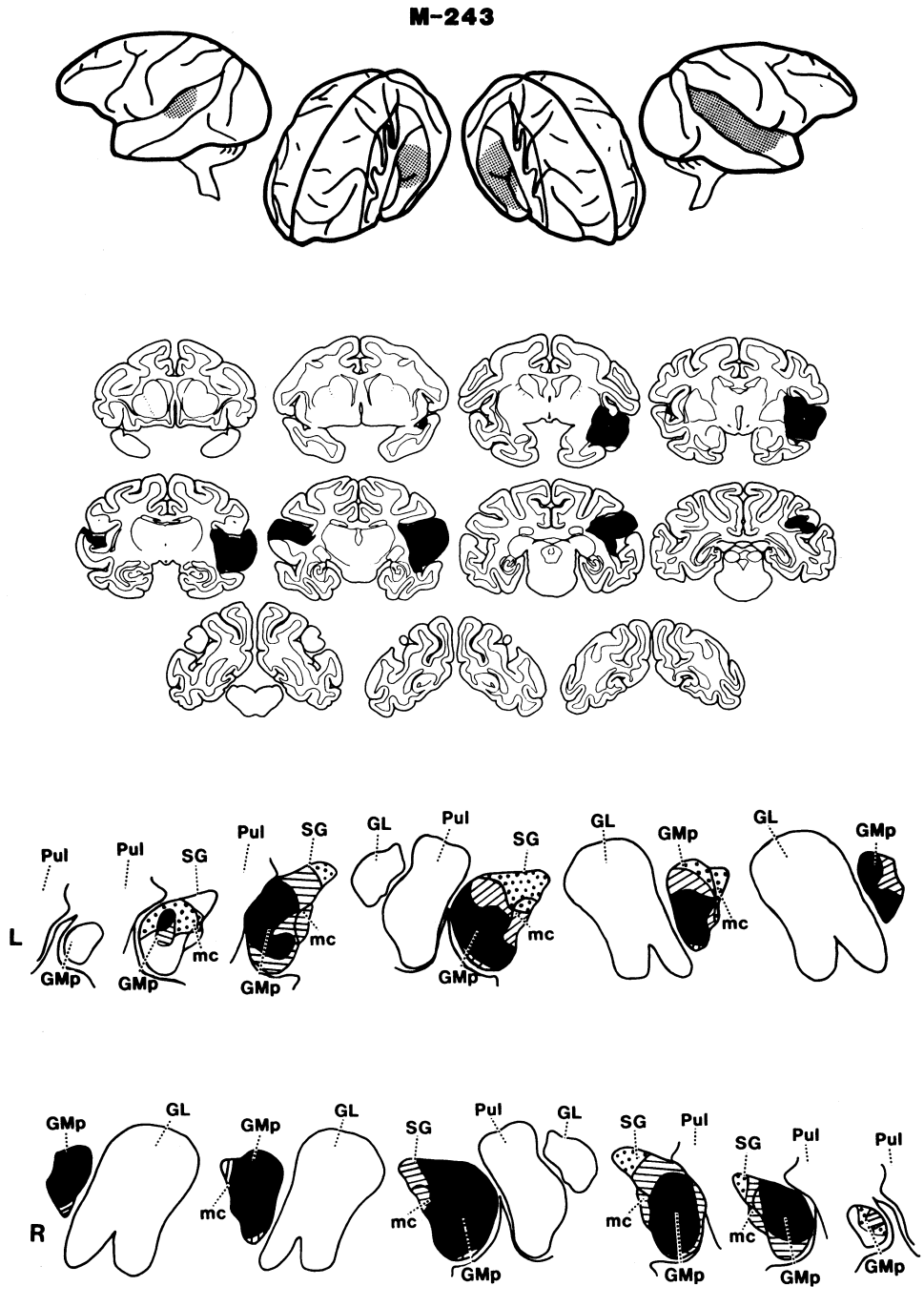

Figure 3. Cortical reconstruction and medial geniculate degeneration for M-243. (See Figure 2 for key.)

days (M-319) after surgery. However, M-235 quickly reached normal levels of performance, but M-319, with a larger lesion, was initially unable to perform at normal levels. Although that animal's performance gradually improved, it was never able to perform consistently at preoperative levels and appears to have suffered a minor but permanent impairment.

A lesion that bilaterally removes the area of the superior temporal gyrus dorsal to auditory cortex also has little effect on the discrimination of the coos. As illustrated in Figure 7, M-136 that received a bilateral lesion that included the areas dorsal to auditory cortex was not impaired in its ability to discriminate the coos. An interesting feature of this case was that although the left lesion encompassed the primary and dorsal secondary auditory fields, this animal did not show the transient impairment that has been observed following larger unilateral lesions of the left superior temporal gyrus

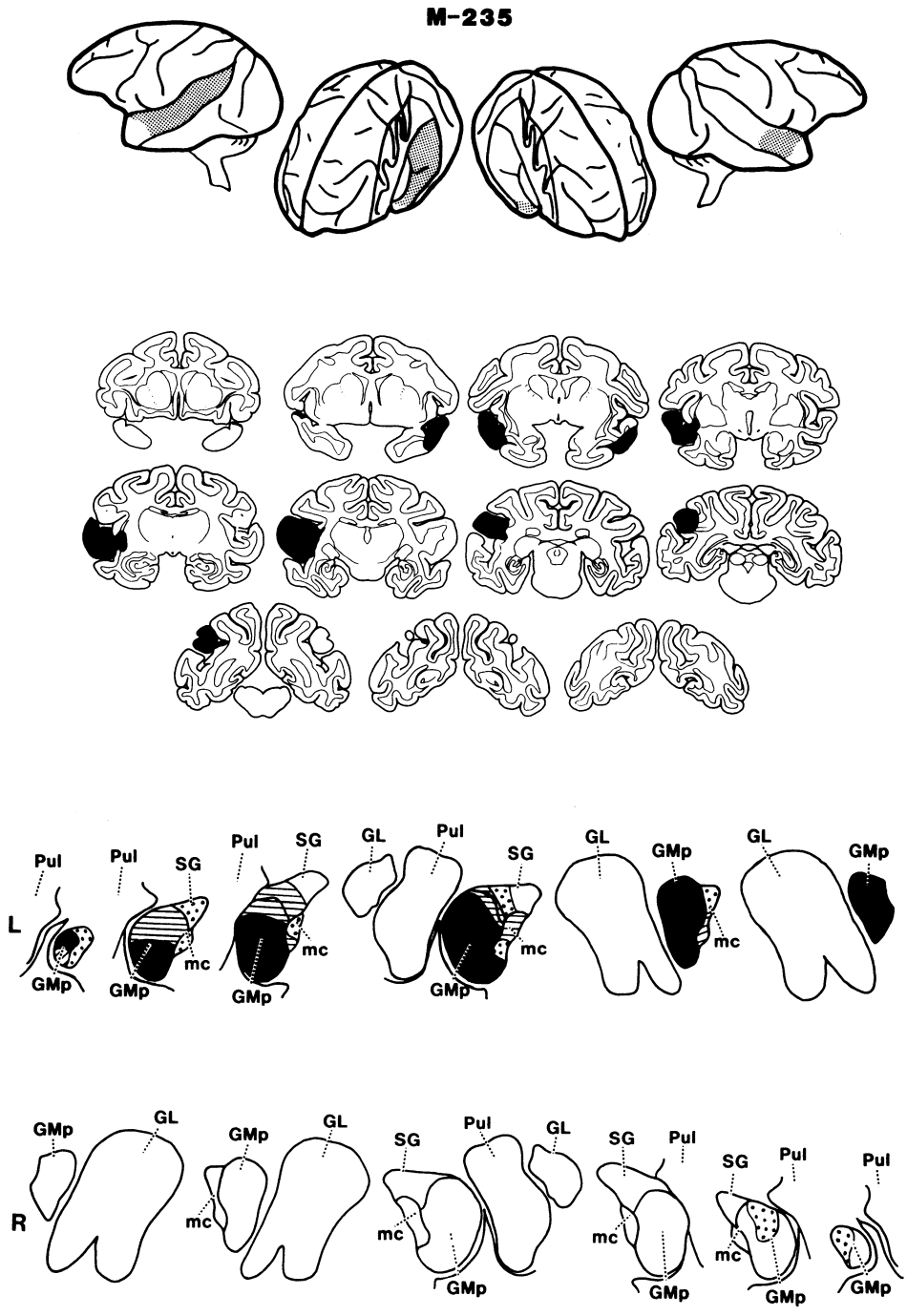

Figure 4. Cortical reconstruction and medial geniculate degeneration for M-235. (See Figure 2 for key.)

(H. Heffner & Heffner, 1986a). Whether this means that an effective left unilateral lesion must include the areas ventral to primary auditory cortex remains to be determined.

Absolute Thresholds for Detecting the Coos

After completion of the previous tests, the 4 operated animals and one normal animal (M-337) were tested to determine their absolute thresholds for detecting the coos. This was done by training the animals to discriminate the SE or warning coos from silence and then reducing the intensity of the coos to determine their threshold. An additional threshold was obtained for the normal monkey for discriminating between the SE and SL coos.

The results of this test, shown in Figure 8, illustrate three points. First, the detection and discrimination thresholds (us-

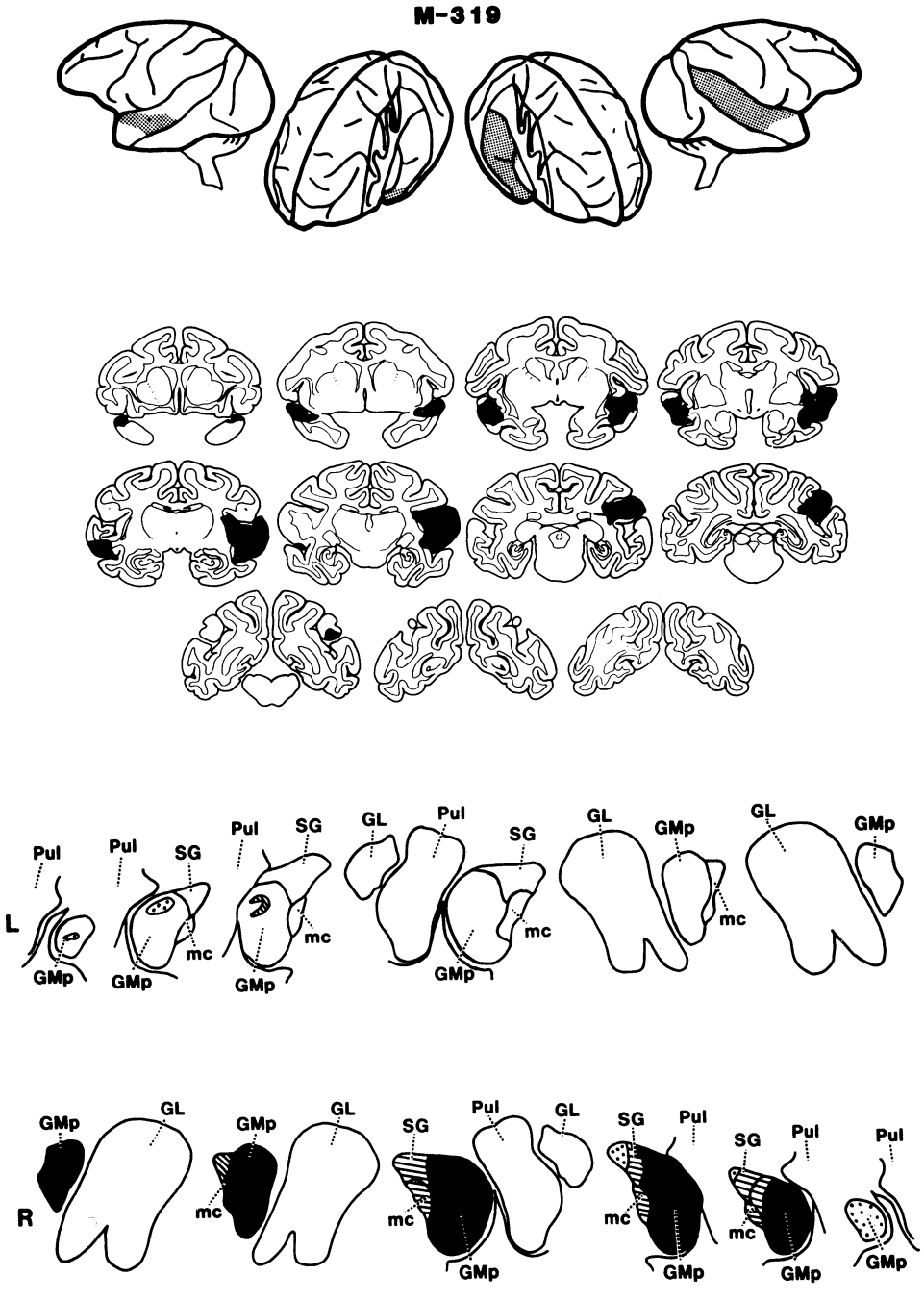

Figure 5. Cortical reconstruction and medial geniculate degeneration for M-319. (See Figure 2 for key.)

ing the 0.50 performance level) for the normal monkey differ by only 2 dB. This result suggests that an animal should be able to discriminate any sound that it can easily detect. Second, all of the monkeys could easily detect the coos when they were attenuated by 30 dB, which demonstrates that the coos were clearly audible to them at the standard (0 dB attenuation) presentation level. Third, although M-243 was 10 dB less sensitive than the normal monkey, it had shown that it could not discriminate the coos even when the intensity was increased 15 dB over the standard level. These results provide further evidence that the inability to discriminate the coos following bilateral cortical ablation is not due to any difficulty in hearing them (H. Heffner & Heffner, in press).

Discussion

Cortical Hearing Loss

We have previously demonstrated that large bilateral lesions of the superior temporal gyrus in Japanese macaques

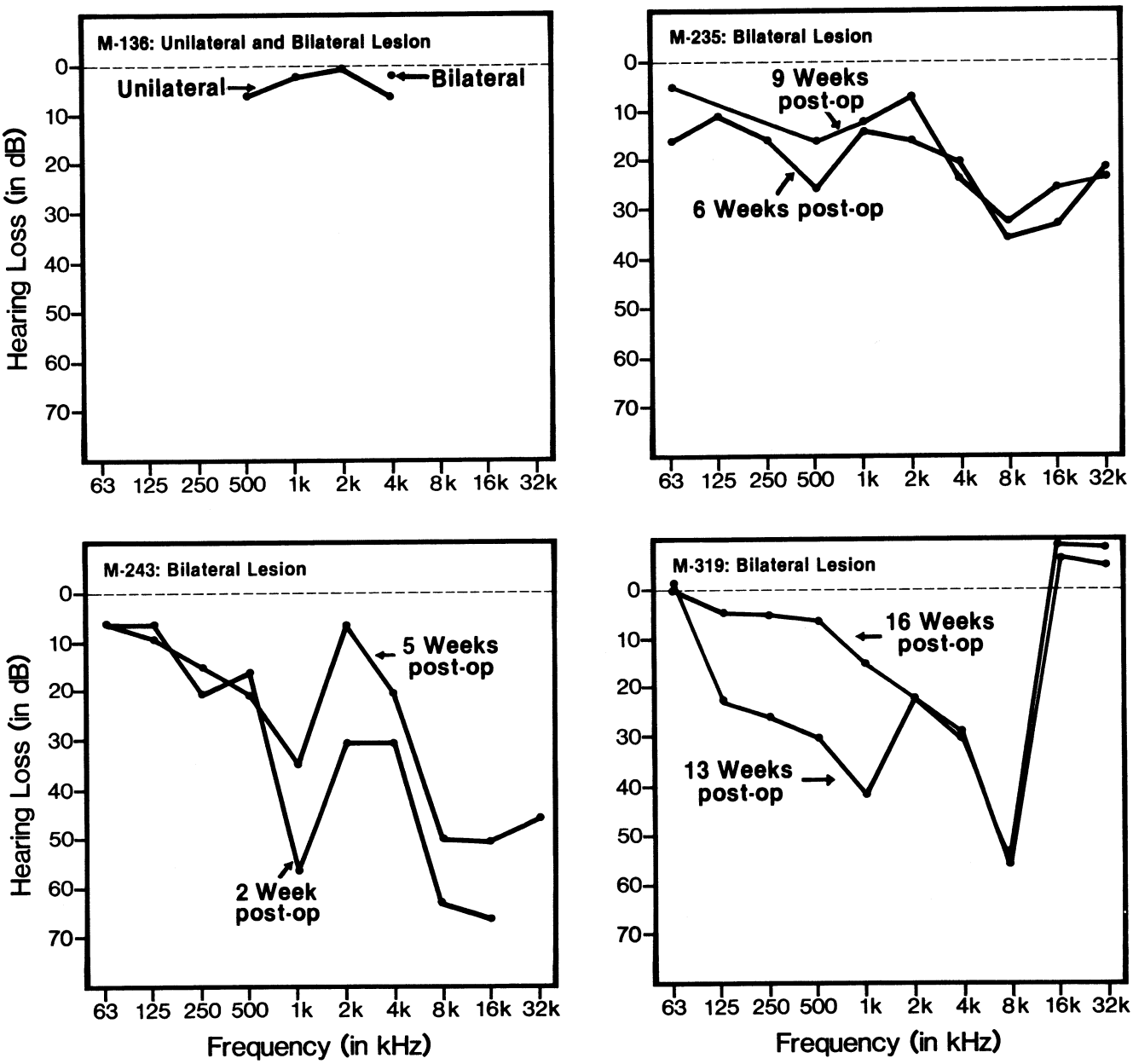

Figure 6. Audiograms of the four monkeys. (Note that all but M-136 showed a hearing loss. Post-op, postoperative.)

result in a dramatic hearing loss (H. Heffner & Heffner, 1986b). Such lesions, which include the posterior two-thirds of the superior temporal gyrus, result in an initial complete deafness followed by partial recovery of hearing. The present results extend these findings by demonstrating that a cortical hearing loss can be produced by much smaller lesions. However, the hearing losses produced by the restricted lesions differ quantitatively from the previous ones. Unlike the larger lesions, the restricted lesions did not result in a transient total deafness, and the animals showed greater and more rapid recovery of their hearing.

In analyzing the locus of the lesions, there appears to be two general areas that, when ablated, result in a hearing loss. First, lesions that bilaterally remove the primary and secondary auditory fields (as defined by thalamic degeneration) result in an obvious hearing loss as demonstrated by M-243. Whether such a loss would result from lesions of only primary or secondary auditory cortex remains to be determined. Second, bilateral lesions of the superior temporal gyrus rostral to primary and secondary auditory cortex also result in a hearing loss (M-235 and M-319). Although the exact anatomical areas of these lesions is difficult to determine, they probably include area Ts2 of Pandya and Sanides (1973).

The areas rostral to primary and secondary auditory cortex have previously been linked with the auditory system. Unit responses to tonal stimuli have been found in these areas,

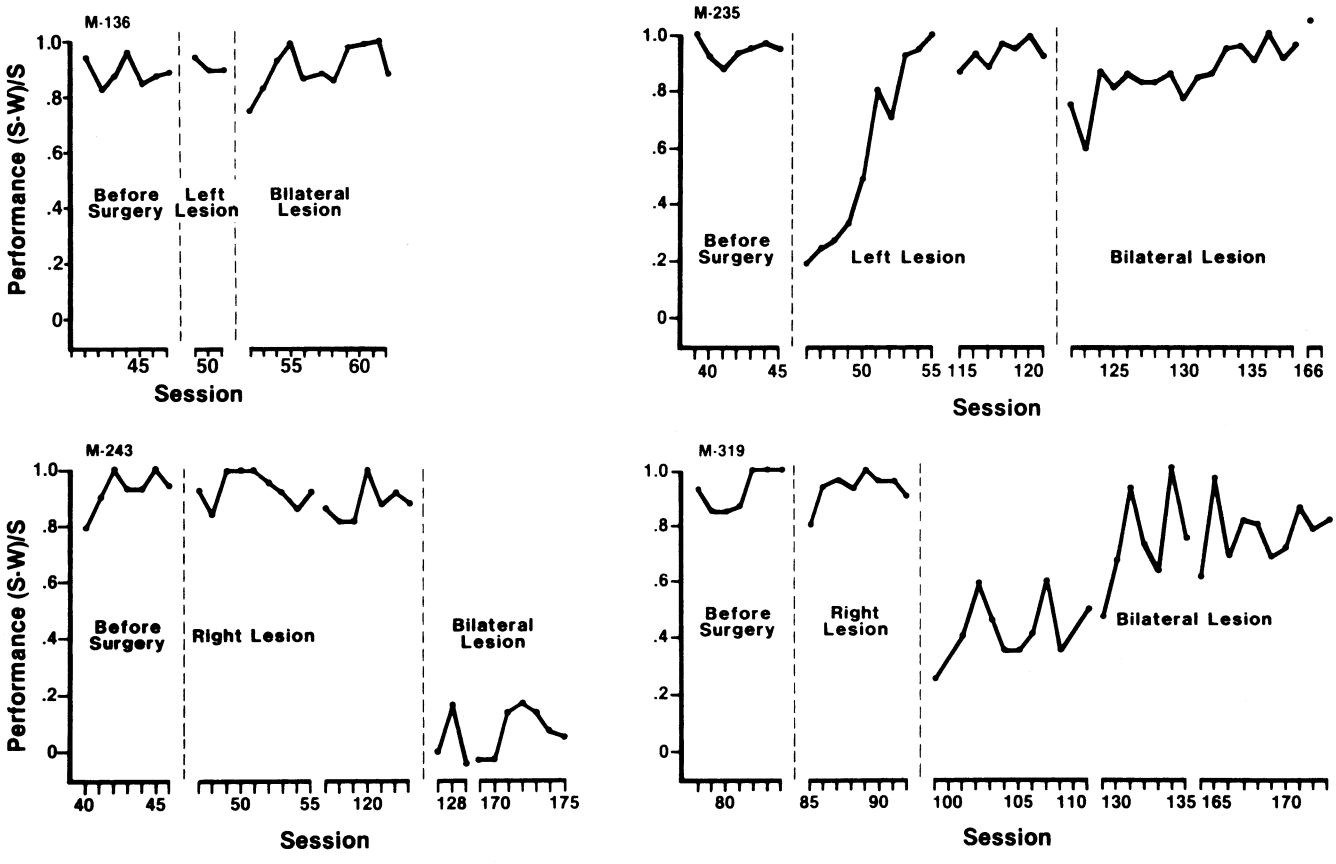

Figure 7. Effect of cortical ablation on the ability to discriminate the coo vocalizations. (Note the permanent inability of M-243 to perform the discrimination successfully.)

and it is possible that an orderly tonotopic representation may exist there (Merzenich & Brugge, 1973). That these areas have connections with primary and secondary auditory cortex has been demonstrated by neuroanatomical studies (Galaburda & Pandya, 1983; Mesulam & Pandya, 1973).

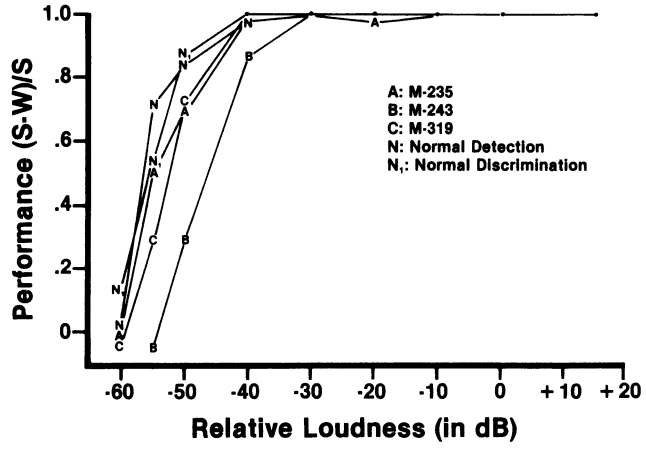

Figure 8. Effect of loudness on the ability to detect and discriminate the coo vocalizations. (A, B, C, and N indicate the scores of four monkeys on the detection of the SE coos. N_1 indicates the scores of the normal monkey on the discrimination of SE and SL coos.)

The results of one animal, M-136, suggest that lesions caudal to primary and secondary auditory cortex do not result in a hearing loss. This conclusion is based on the animal's 4 kHz threshold, which was determined 3 weeks after surgery. Although we cannot rule out the possibility that the monkey may have had a hearing loss at other frequencies, it should be noted that a hearing loss at 4 kHz of at least 20 dB has been observed in all of the monkeys with cortical hearing losses in this and a previous study (H. Heffner & Heffner, 1986b). As a result, there is reason to believe that the normal threshold at 4 kHz is representative of the animal's sensitivity in general. This point is of some importance because the failure to find a hearing loss gives functional significance to the observation that the caudal tip of the superior temporal plane (Tpt) receives no significant projection from the medial geniculate (Mesulam & Pandya, 1973) and therefore may have no auditory function.

Finally, the observed variation in hearing loss as a function of the location and size of the lesion is relevant to the understanding of hearing loss following bilateral temporal lobe lesions in humans. For a number of years, there has been some uncertainty as to whether and to what extent such lesions affect hearing. This uncertainty was due in part to the fact that bilateral temporal lobe lesions were reported to

result in effects ranging from total deafness to no hearing loss at all (for a review, see H. Heffner & Heffner, 1986b).

As the results of the present study show, such variation in hearing loss can be accounted for by variation in the size and location of the lesions. That is, complete bilateral superior temporal gyrus lesions result in an initial complete deafness followed by slow and partial recovery. Bilateral lesions of the rostral and middle thirds of the superior temporal gyrus result in a partial hearing loss, and the animals show more rapid and greater recovery of their hearing. On the other hand, bilateral lesions of the caudal third of the superior temporal gyrus may result in little or no loss of hearing. Given that different lesions have different effects and that bilateral lesions must affect the same area in both hemispheres in order to produce a hearing loss, it is not surprising that the diversity of naturally occurring bilateral temporal lobe lesions in humans results in widely varying effects on hearing.

Discrimination of Species-Specific Sounds

Previous work has indicated that the cortical mechanism involved in the discrimination of species-specific sounds is located in the posterior two-thirds of the superior temporal gyrus (H. Heffner & Heffner, 1986a). Furthermore, this discrimination is mediated primarily by the left hemisphere with the right hemisphere able to perform the discrimination within a few days following removal of the left superior temporal gyrus. Although the present study does not pertain to the lateralization of this ability, it does have a bearing on the locus of the cortical mechanism.

The results of this study indicate that the discrimination of species-specific sounds may be mediated by primary and/or secondary auditory cortex. Specifically, a lesion that removed primary and secondary auditory cortex in both hemispheres abolished the ability to discriminate the coos (M-243). In contrast, lesions of the superior temporal gyrus that included bilateral removal of areas rostral or caudal to auditory cortex, but spared auditory cortex on one side had little or no effect. Because the lesion that included auditory cortex bilaterally also included the posterior two-thirds of the superior temporal gyrus in one hemisphere, we cannot rule out the possibility that the deficit would not have occurred if the lesion had been restricted to auditory cortex on both sides. However, we believe that the critical factor in producing the deficit in this case was the removal of primary and secondary auditory cortex bilaterally. Thus a deficit that is similar to sensory aphasia in humans may be produced by small bilateral lesions restricted to the middle third of the superior temporal plane.

Another study that examined the effect of temporal lobe lesions on hearing is one by Dewson and his colleagues (Dewson, Pribram, & Lynch, 1969). They found that bilateral lesions of primary and secondary auditory cortex abolished the ability of rhesus monkeys to discriminate among human speech sounds, whereas lesions of the superior temporal sulcus or of inferotemporal cortex did not abolish the discrimination. Because human speech sounds are not "species-specific" for monkeys, their results suggest that auditory cortex is necessary for the discrimination of sounds that cannot be discriminated along simple frequency or intensity dimensions, that is, "complex" sounds. Thus it appears likely that auditory cortex plays a role in the discrimination of complex sounds in general and species-specific vocalizations in particular.

Finally, because bilateral auditory cortex lesions result in a hearing loss as well as an inability to discriminate coos, the question arises as to whether the deficit in the coo discrimination is a result of the cortical hearing loss. In a previous study, we demonstrated that amplifying and equalizing the coos to compensate for their hearing losses did not improve the ability of monkeys with bilateral superior temporal lesions to discriminate the coos (H. Heffner & Heffner, in press). Furthermore, the ability of normal monkeys was not disrupted by attenuating and filtering the coos to simulate a cortical hearing loss. With the present results, we now have evidence that bilateral superior temporal lesions can result in a hearing loss with little or no effect on the ability to discriminate the coos. This not only provides further evidence that a cortical hearing loss does not in itself result in sensory aphasia, but it also demonstrates that the two deficits can be partly dissociated.

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