

Effect of Unilateral and Bilateral Auditory Cortex Lesions on the Discrimination of Vocalizations by Japanese Macaques

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SUMMARY AND CONCLUSION

1. Ten Japanese macaques were trained to discriminate between two types of Japanese macaque coo vocalizations before and after auditory cortex ablation. Five of the animals were tested following left unilateral ablation, whereas the other five were tested following right unilateral ablation. After postoperative testing, symmetrical lesions were made in the remaining hemisphere in two animals from each group and the effect of bilateral lesions was assessed. The animals were tested using a shock avoidance procedure.

2. Unilateral ablation of left auditory cortex consistently resulted in an initial impairment in the ability to discriminate between the vocalizations with the animals regaining normal performance in 5–15 sessions. In contrast, right unilateral ablation had no detectable effect on the discrimination.

3. Bilateral auditory cortex ablation rendered the animals permanently unable to discriminate between the coos. Although the monkeys could learn to discriminate the coos from noise and from 2- and 4-kHz tones, they had great difficulty in discriminating between the coos and tones in the same frequency range as the coos (i.e., 500 Hz and 1 kHz).

4. The initial impairment following left unilateral lesions indicates that the ability to perceive species-specific vocalizations is lateralized to the left hemisphere. The observation that bilateral lesions abolish the discrimination indicates that the recovery in the left lesion cases was the result of the right hemisphere mediating the discrimination.

INTRODUCTION

The study of cerebral lesions in human patients has shown that the neural mechanisms involved in speech are located predominantly in the left hemisphere (e.g., 11). Specifically, lesions of the left temporal lobe often result in an inability to understand speech. This deficit, part of a syndrome referred to as Wernicke's aphasia, has become associated with a cortical region called Wernicke's area in the left temporal lobe. Although the exact locus of Wernicke's area has been the subject of some debate, it is generally accepted that it is contained within the secondary auditory areas located in the posterior portion of the superior temporal gyrus (3).

The existence of a lateralized cortical speech reception mechanism in humans has led to a search for a similar mechanism in other species, particularly nonhuman primates (4, 13). Early efforts examined the effect of auditory cortex lesions on the perception of speech sounds and found that monkeys were unable to discriminate vowel sounds following bilateral lesions (the effect of unilateral lesions was not assessed) (8). Similarly, squirrel monkeys were unable to discriminate squirrel-monkey calls from other complex sounds following bilateral auditory cortex lesions, but unilateral ablation did not appear to affect the discrimination (21). However, more recent studies by Dewson (5, 6, 7), using an auditory association task, have suggested that left auditory cortex in monkeys might be dominant for some auditory tasks. Thus, while these studies established that auditory cortex is necessary for the

discrimination of complex sounds in primates, the question of laterality was unresolved.

Recently, there has appeared evidence that Japanese macaques (*Macaca fuscata*) may have undergone specialization of the left hemisphere for the perception of their vocal communications. Specifically, Japanese macaques show a consistent right ear advantage for the ability to discriminate two types of their "coo" vocalizations (1, 28–31, 34); types that although are acoustically similar, have different meanings (12). This right ear advantage for the perception of species-specific vocalizations resembles that shown by humans for the perception of speech sounds (22, 23). In humans, this right ear advantage is explained by the fact that input from each ear is dominant in the contralateral hemisphere. As a result, input from the right ear will predominate in Wernicke's area, which is located in the left hemisphere. Thus the presence of a right ear advantage in Japanese macaques suggests that they might also have a left hemisphere specialization for the perception of species-specific vocalizations.

The purpose of the present study was to determine the neuroanatomical basis of the right ear advantage of Japanese macaques by use of the ablation-behavior technique. This report presents a detailed description of the effect of unilateral and bilateral ablation of auditory cortex on the ability of Japanese macaques to discriminate the same coo sounds that were previously used to establish the existence of their right ear advantage (15). It is the second in a series of detailed reports describing the effect of auditory cortex lesions on hearing, the first of which described the hearing loss that accompanies bilateral auditory cortex ablation in monkeys (16).

METHODS

Ten Japanese macaques were tested on their ability to discriminate two types of coos before and after unilateral and bilateral cortical ablation. The animals were tested with a conditioned avoidance procedure in which they demonstrated their ability to discriminate the coos by breaking contact with a water spout when one of the types was presented and maintaining contact when the other was presented.

Subjects

Ten male Japanese macaques (*Macaca fuscata*) ranging in age from 5 to 7 yr (adolescent) were used

in this study. The animals had been born and reared in a 58-acre outdoor primate colony (Arashiyama West Institute). The preoperative audiogram of monkey M-36 and the postoperative audiograms of M-37, M-57, M-150, M-246, and M-252 were determined during the course of this study and have been reported elsewhere (16).

The animals were individually housed in primate cages with free access to food and were trained using water as a reward. The auditory canals of each animal were examined during surgery and the middle ears following death, to ensure that they were free of damage or disease.

Surgical and histological procedures

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

Following removal of cortical tissue, the area 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) sprinkled on the surface of the muscle. The scalp incision was closed with silk suture. The animal was returned to its home cage and was administered acepromazine (Ayerst) as needed to minimize discomfort. Approximately 4–16 wk elapsed before a similar lesion was made in the other hemisphere.

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

The relative sizes of the cortical lesions and the amount of thalamic degeneration were quantified with the aid of a digitizing tablet in order to determine whether there were any systematic differences between the left and right lesions. The cortical lesions were quantified by taking drawings of normal coronal sections 3.6-mm apart onto which the lesions had been mapped and measuring the extent of the lesion in five areas: superior temporal gyrus, middle temporal gyrus, frontal and parietal areas, insula, and subcortical areas. These measurements were then converted to an estimate of the volume of tissue removed in each area (in cubic millimeters). A similar analysis was made of degeneration in the auditory thalamus by taking drawings of a normal thalamus 600- μ m apart onto which the degeneration had been mapped and quantifying the areas of degeneration for the principal division of the medial geniculate, the magnocellular division, and the supragenulate nucleus. The measurements were weighted according to the estimated percentage of cell loss and a volume measure (in cubic millimeters) was derived for each area.

Behavioral procedure

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

ACOUSTIC STIMULI. The vocalizations used in the discrimination were the same 15 coos used by Petersen and his colleagues (1, 30, 31, 34) to demonstrate the right ear advantage. The vocalizations had originally been recorded by Green (12) and are described in detail elsewhere (e.g., Refs. 1, 12). They consisted of seven so-called "smooth early (SE) high" and eight "smooth late (SL) high" coos. The coos are distinguished by the temporal position of the peak fundamental frequency in the frequency-modulation portion of the call. Furthermore, 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 vocalizations were digitized at a 20-kHz sample rate and edited to remove the background noise that preceded and followed the coos on the tape recording. Initially, the sounds were rerecorded onto a four-channel tape for playback during discrimination training. This system, however, was soon replaced by direct digital-to-analog playback, which allowed complete random access to the vocalizations. The sounds were played back through a band-pass filter (100 Hz–5 kHz) to reduce low-frequency noise and prevent aliasing, an amplifier, and a high-fidelity loudspeaker (Acoustic Research 3a), which was placed 1.5 m in front of the primate chair. The coos were presented at an average level of at least 55 dB above threshold as determined by attenuating the sounds until the animals were no longer able to detect them.

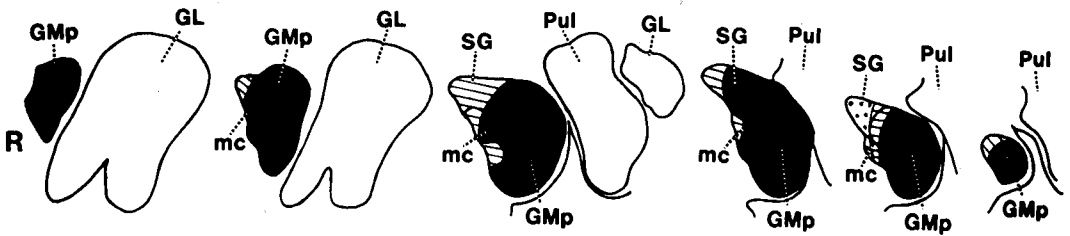
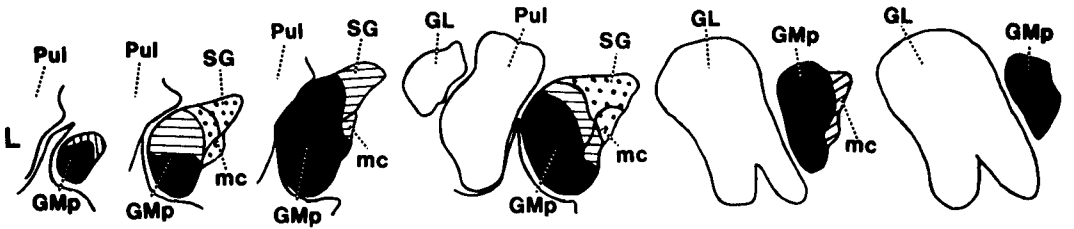
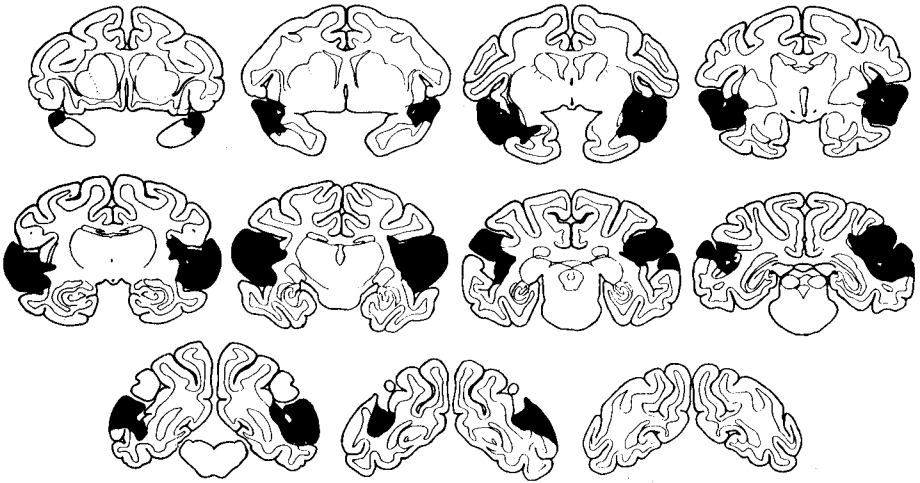
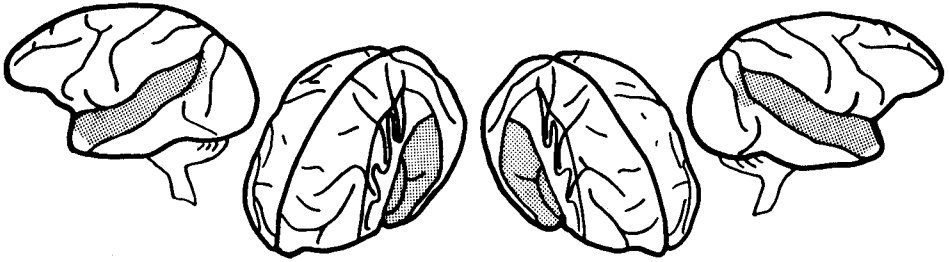
Additional stimuli consisted of tone pips and noise bursts. Tones of 500 Hz, 1, 2, and 4 kHz were produced by a tone generator (Hewlett-Packard 209A), gated with a 10-ms rise fall (Coulbourn S82-04 rise-fall gate), and then digitized for playback. A noise burst was produced by a white noise generator (Grason Stadler 1285), gated with a 10-ms rise fall, and digitized. The duration of the tone pips and noise burst was set at 400 ms, which is within the range of the durations of the coos. All sounds were presented at a level at least 40 dB above threshold.

PSYCHOPHYSICAL PROCEDURE. In the avoidance procedure, a thirsty monkey was seated in the primate chair and trained to place its mouth on the water spout, an action that fixed its head in front of the loudspeaker. This was accomplished by providing a steady trickle of water (3–4 ml/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 water spout and the room lights were momentarily turned off. 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 room lights 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,

M-37



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 with a 3.5-s intertrial interval (i.e., 1 trial every 7 s). 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. The number of SE trials given in each of the seven trial periods was adjusted so that each trial period had the same probability of containing an SE trial (which was 0.25). A typical session consisted of the presentation of 21–35 SE trials and 63–105 SL trials.

The response of an animal on each trial, i.e., whether or not it had made an avoidance response, was determined by whether or not the animal was in contact with the spout during the last 200 ms of the trial. 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 an SE coo. 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.

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 ranges from 1.0 (indicating good discrimination of the safe and warning sounds) to 0 (indicating failure to discriminate the two sets of coos).

It should be noted that this shock-avoidance procedure was chosen because it requires very little

learning and very little motor prowess on the part of the animal. To perform the task, an animal need only drink water from the spout and then momentarily withdraw when a warning signal indicates impending shock. Because of the simplicity of the task, it is easy to shift from one test to another test without significant disruption of the animal's performance. Indeed, this task has been used successfully many times to assess hearing in severely brain-damaged animals and in otherwise intractable animals (e.g., Refs. 18, 32).

RESULTS

Anatomical results

The locus and extent of auditory cortex have been studied in the rhesus macaque with microelectrode recordings (25), cytoarchitectural analysis (10, 27), and by tracing thalamocortical connections (26). 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 (cf. Ref. 16).

Thalamic degeneration and cortical reconstructions are illustrated for each of the 10 cases (Figs. 1–10). All of the lesions removed the entire primary core and secondary surrounding auditory fields. Correspondingly, all of the lesions resulted in severe to total degeneration of the principal division of the medial geniculate. Some variation was present in the extent of the lesions into the temporal pole and its underlying fibers and the degeneration in the caudal tip of the medial geniculate varies accordingly. M-252 and M-235 had the least degeneration in this area among the left cases and M-150 and M-319 had similarly mild degeneration in this area among the right cases. The caudal tips of the other cases were severely to totally degenerated. The magnocellular division of the medial geniculate and the supragenicular nuclei were moderately to totally degenerated in all cases, with M-235 having

FIG. 1. Cortical reconstruction and medial geniculate degeneration for M-37 that received a left unilateral lesion followed by a right lesion. *Top*: reconstruction of the 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- μ m apart. GL, dorsal lateral geniculate; GMp, principal division of the medial geniculate; mc, magnocellular division of the medial geniculate; Pul, pulvinar; SG, supragenicular nucleus. *Blackened area* indicates total degeneration, 95%–100% cell loss; *hatched area* indicates severe degeneration, 70%–95% cell loss; *stippled area* indicates moderate degeneration, 30%–70% cell loss.

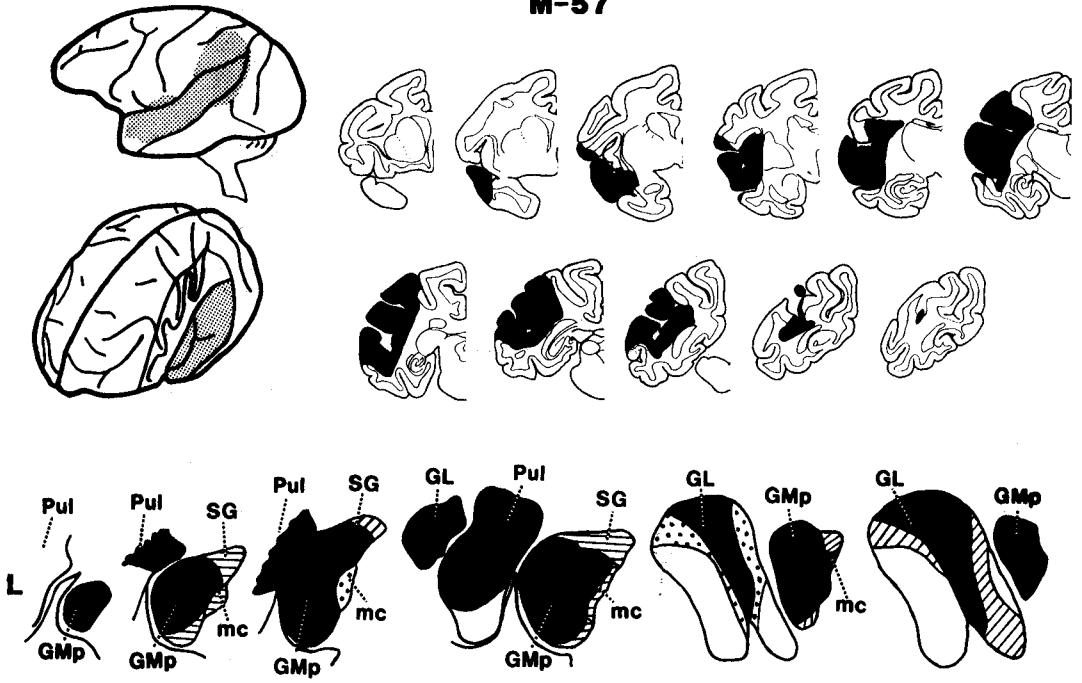
M-57

FIG. 2. Cortical reconstruction and medial geniculate degeneration for M-57 that received a left unilateral lesion. See Fig. 1 for key.

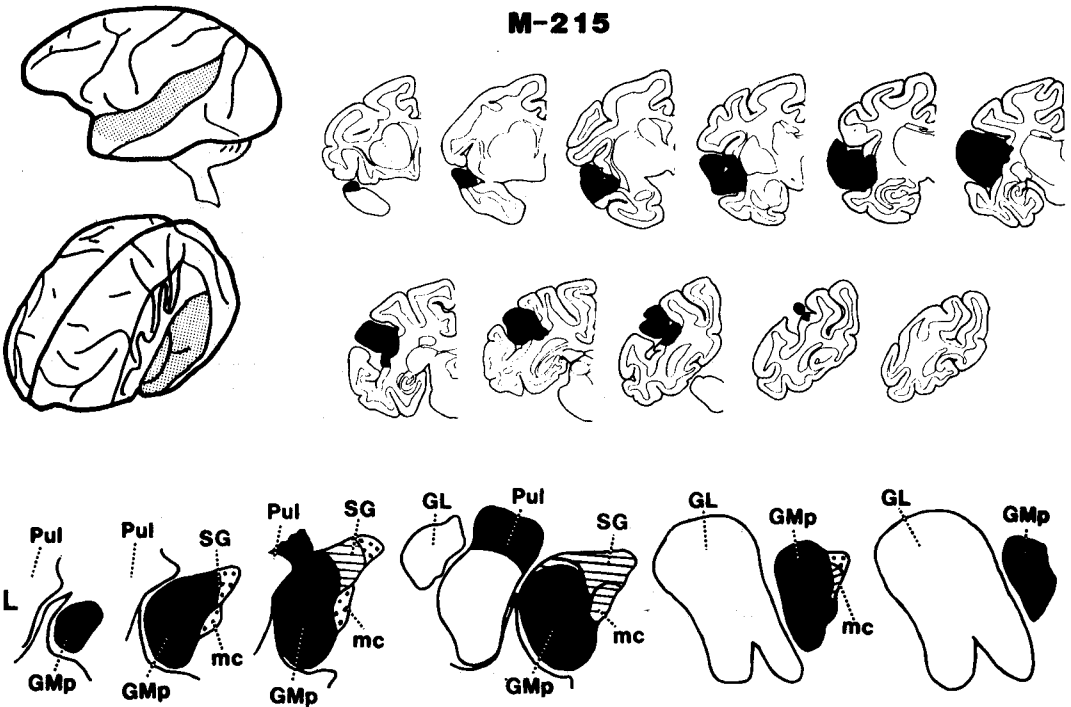
M-215

FIG. 3. Cortical reconstruction and medial geniculate degeneration for M-215 that received a left unilateral lesion. See Fig. 1 for key.

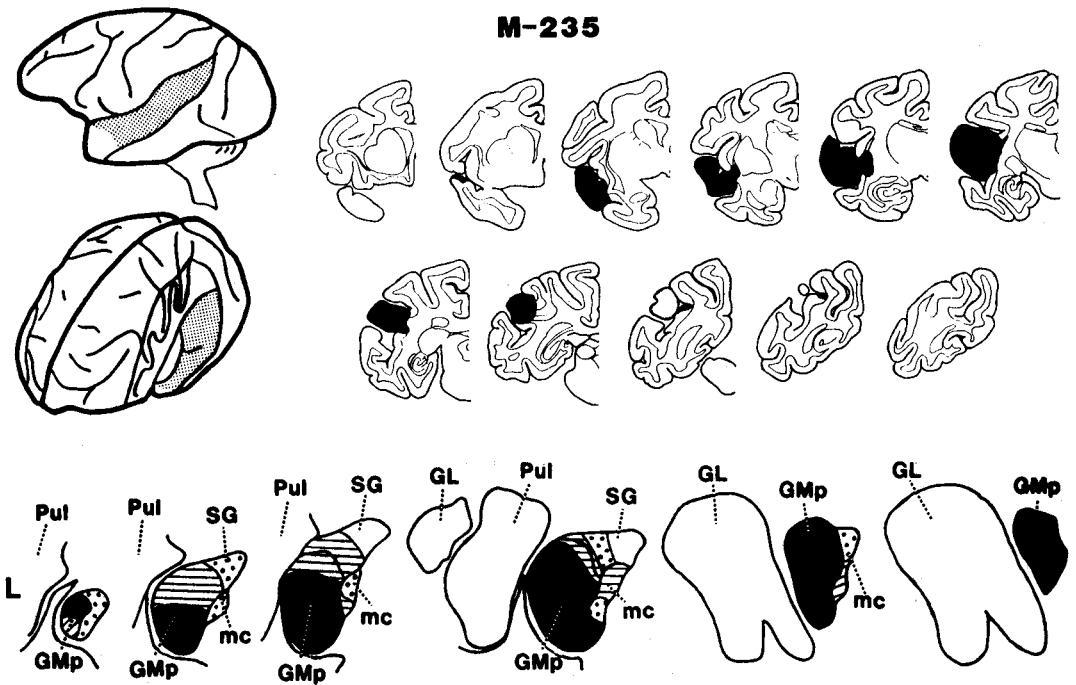


FIG. 4. Cortical reconstruction and medial geniculate degeneration for M-235 that received a left unilateral lesion. See Fig. 1 for key.

the least degeneration among the left cases and M-246 having the least degeneration among the right cases. The lesions usually did not encroach on the visual radiations; M-57 on the left and M-36 on the right being the only exceptions as revealed by partial degeneration of the lateral geniculate.

A detailed analysis was made of the left and right lesions to determine if there were any systematic differences that might be expected to lead to behavioral differences between the left and right unilateral cases. Table 1 shows the sizes of the lesions for five areas: the superior temporal gyrus, middle temporal gyrus, frontal and parietal areas, insula, and subcortical areas, as well as the amount of degeneration in the principle division of the medial geniculate, the magnocellular division, and the supragenicular nucleus. As can be seen in Table 1, there was considerable overlap in the sizes of the lesions in the two groups. Statistical analysis of the differences between the two groups shown in Table 1 indicated that there were no reliable differences in either the size of the lesions or in the degree of degeneration in the medial geniculate nucleus ($P > 0.05$,

Mann-Whitney U). Overall, the auditory lesions for both the left and right cases were complete and the bilateral lesions were symmetrical.

Normal performance

The discrimination of the coos proved to be a relatively easy task for the monkeys to acquire as indicated by their learning times. The 10 monkeys acquired the basic avoidance response after 5–12 training sessions (average 7.5 sessions). Learning to discriminate one pair of coos required an additional 3–10 sessions (average 6.8). The animals were then trained to discriminate all seven SE coos from all eight SL coos to a performance level of at least 0.75 with no overlap in the individual average scores for the two sets of coos. This criterion was reached in an additional 10–42 sessions (average 25.9). Thus the animals learned the entire discrimination in an average of 40 sessions. Further practice sessions were given to ensure that the animals could maintain a high level of performance.

To determine the degree to which the animals might forget the task over time, five of

TABLE 1. Comparison of lesion size and extent of thalamic degeneration in the left and right unilateral cases (in cubic millimeters)

Monkey	Cortical Lesion					Thalamus		
	Superior Temporal Gyrus	Middle Temporal Gyrus	Frontal and Parietal	Insula	Subcortical	GMP	mc	SG
<i>Left</i>								
37	2,560	496	0	156	143	17.54	1.40	2.80
57	2,794	1,078	1,366	458	252	17.67	1.96	3.65
215	2,616	70	193	224	11	18.18	1.67	3.32
235	2,290	0	20	114	39	17.86	1.50	1.67
252	2,749	138	1,130	315	199	17.78	3.88	2.05
<i>Right</i>								
36	2,568	145	112	399	295	18.24	2.06	3.34
150	1,973	330	1,260	386	27	16.97	1.94	2.78
243	2,469	194	27	479	334	17.97	2.08	3.31
246	2,438	0	0	201	121	18.15	2.30	1.77
319	2,450	108	13	303	57	18.09	1.98	3.53

The left and right lesion cases overlapped in both lesion size and degree of thalamic degeneration with the exception that the right lesion cases tended to have more insula removed ($P = 0.028$) and greater degeneration in the magnocellular division of the medial geniculate (mc) ($P = 0.008$). GMP, principle division of the medial geniculate; SG, supragenulate nucleus.

the monkeys were taken off deprivation and given no auditory training for 31–139 days. Three of the animals, M-243, M-36, and M-246, were retested after 31, 40, and 73 days at which times they scored 1.0, 0.96, and 0.91, respectively. M-215, which was tested 91 days later, scored 0.55, 0.67, and 0.86 on the first three retest sessions, whereas M-150, which was tested after 139 days, scored 0.78 and 1.0 on the first two retest sessions. These results indicate that the discrimination was robust and that a decrement in performance would probably not be observed unless training was discontinued for 3 mo or longer.

Effect of unilateral lesions

Following ablation of left auditory cortex, each of the five animals experienced an initial impairment in their ability to discriminate the coos (Fig. 11). Analysis of the animal's performances indicated that they erred by failing to make an avoidance response when a warning stimulus was presented as well as by making avoidance responses to safe stimuli. However, the impairment was not serious, and with the exception of M-37 and M-57, all of the animals were able to perform the discrimination above chance on their first postopera-

tive session and were consistently performing at preoperative levels within 5–15 sessions.

The transient impairment shown by the left unilateral cases was fairly mild and it would be tempting to ascribe them to general postoperative malaise. However, comparison with the performances of the right unilateral cases suggests otherwise. In contrast to the left unilateral cases, ablation of right auditory cortex had no discernible effect at all on the discrimination even in the first postoperative session (Fig. 12).

The difference between the two groups does not appear to be due to such factors as amount of recovery time before testing, overall time between pre- and postoperative testing, or to differences in the size of the lesions. First, the right lesion cases were generally tested sooner after surgery (3–8 days) than were the left lesion cases (4–11 days). As a result, any transient impairment would have been more likely to have been observed in the right lesion cases than in the left. Second, both groups had approximately the same amount of time between pre- and postoperative testing with the left lesion cases having 6–22 days, whereas the right lesion cases had 4–23 days. Indeed, these times are much less than the 3 mo needed to produce

M-252

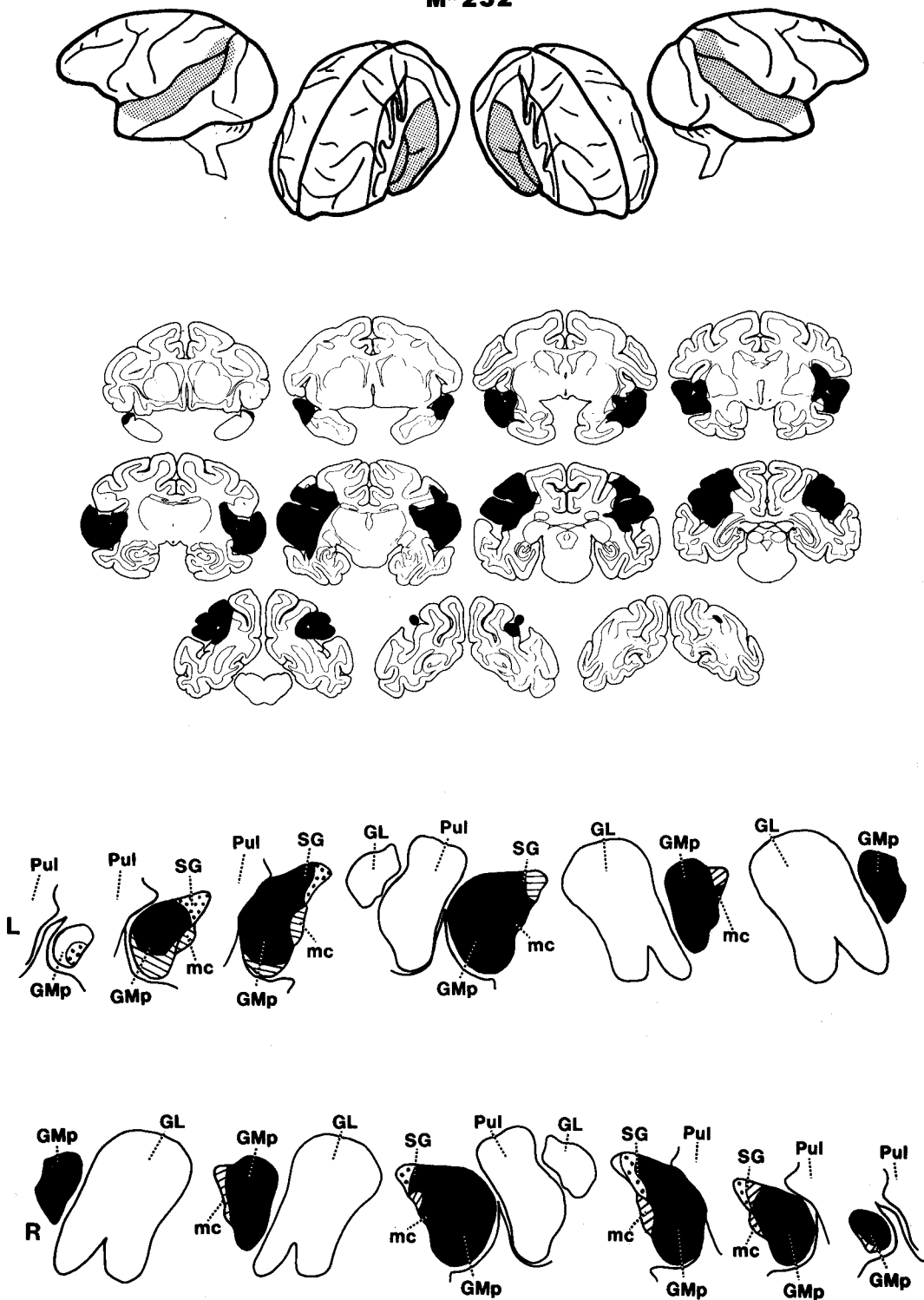


FIG. 5. Cortical reconstruction and medial geniculate degeneration for M-252 that received a left unilateral lesion followed by a right lesion. See Fig. 1 for key.

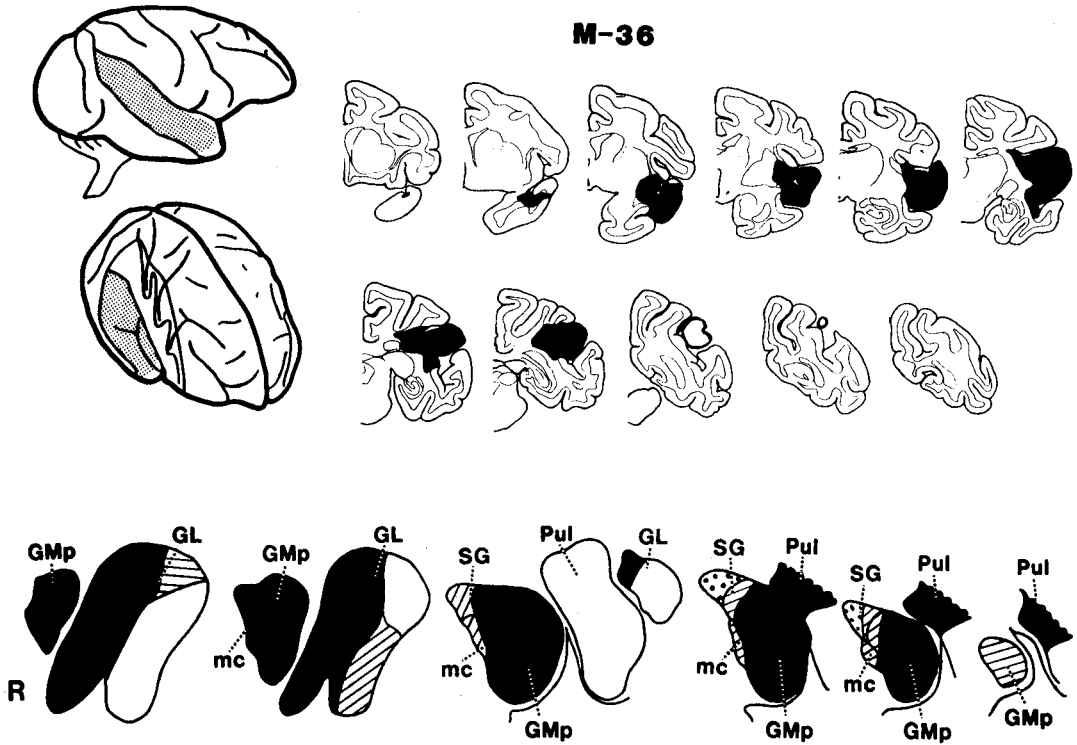


FIG. 6. Cortical reconstruction and medial geniculate degeneration for M-36 that received a right unilateral lesion. See Fig. 1 for key.

a decrement in performance in normal animals. Finally, the lesions were similar with primary and the surrounding auditory fields removed in all cases. Although there was some variation in the degree of damage to the ventral tip of the temporal lobe and in the resulting degeneration of the caudal tip of the medial geniculate, there is sufficient overlap between the two groups to rule out the possibility that the difference between the two groups was the result of any systematic difference in the size and extent of the lesions.

Effect of bilateral lesions

Following testing, symmetrical lesions were placed in the intact hemisphere of five of the monkeys (M-37, M-57, M-150, M-246, and M-252). The results of the bilateral auditory cortex lesions were twofold. First, they resulted in a hearing loss, the details of which have been reported elsewhere (16). Briefly, the animals were initially unable to respond to sound with the first responses to sound appearing as

late as 13 wk after surgery (M-57). Hearing levels improved gradually over time with maximal recovery by 24–35 wk. Recovery was most pronounced for low frequencies (63–250 Hz) and very high frequencies (32 kHz), which generally returned to normal or near-normal levels. However, the monkeys suffered a permanent hearing loss throughout most of their hearing range that was most severe in the mid-frequencies. In the frequency range of the coos (i.e., 125 Hz to 4 kHz), the average hearing losses 24 wk or more after surgery ranged from 21 to 26 dB.

Once the monkeys had recovered hearing sufficient for testing, it became apparent that a second effect of the lesions was to render them unable to discriminate between the two types of coos (Figs. 11 and 12). The animals were observed for a period of months during which they were given various tests designed to assess their ability to discriminate sounds (with occasional time off while their audiograms were assessed) and also given various

M-150

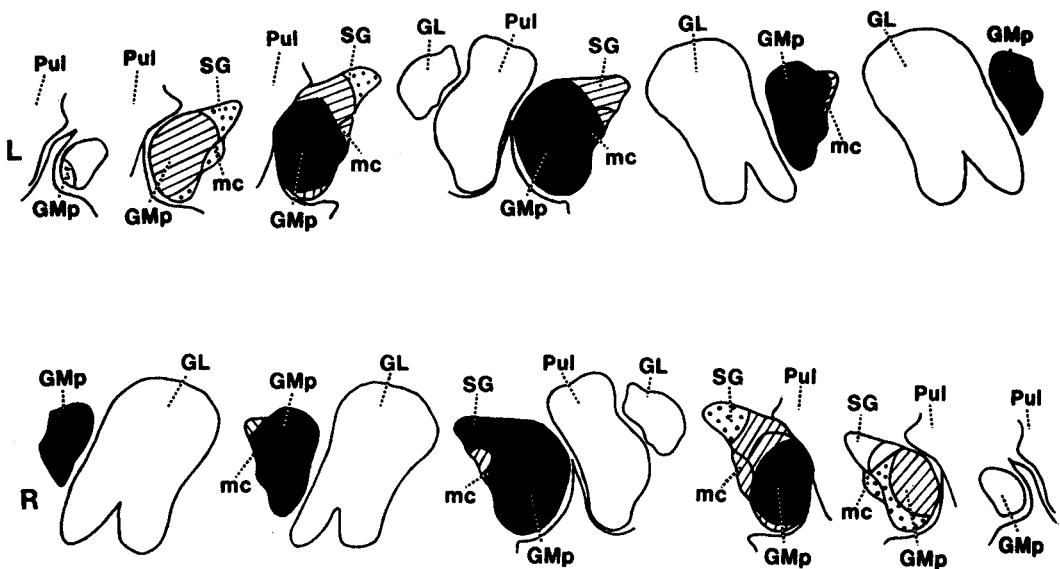
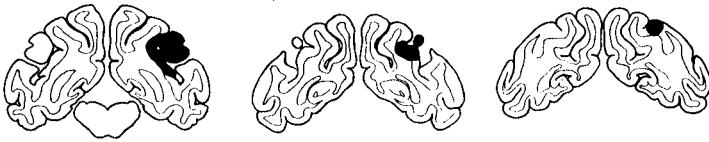
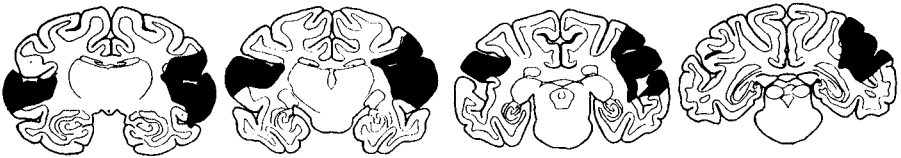
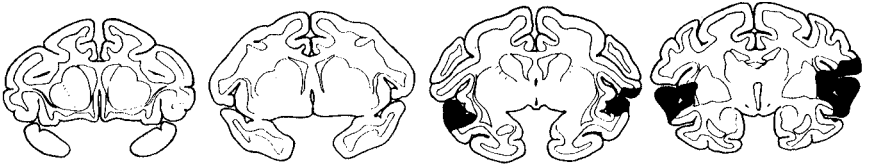
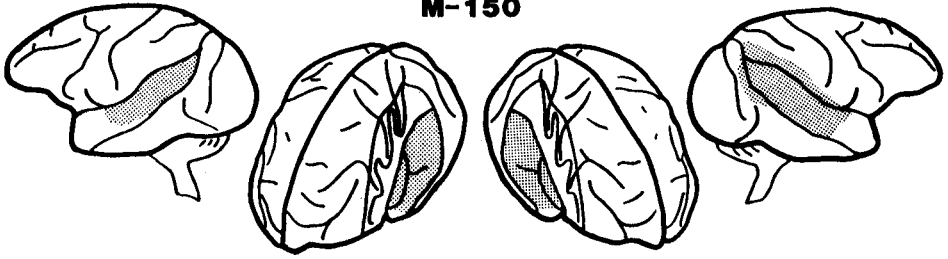


FIG. 7. Cortical reconstruction and medial geniculate degeneration for M-150 that received a right unilateral lesion followed by a left lesion. See Fig. 1 for key.

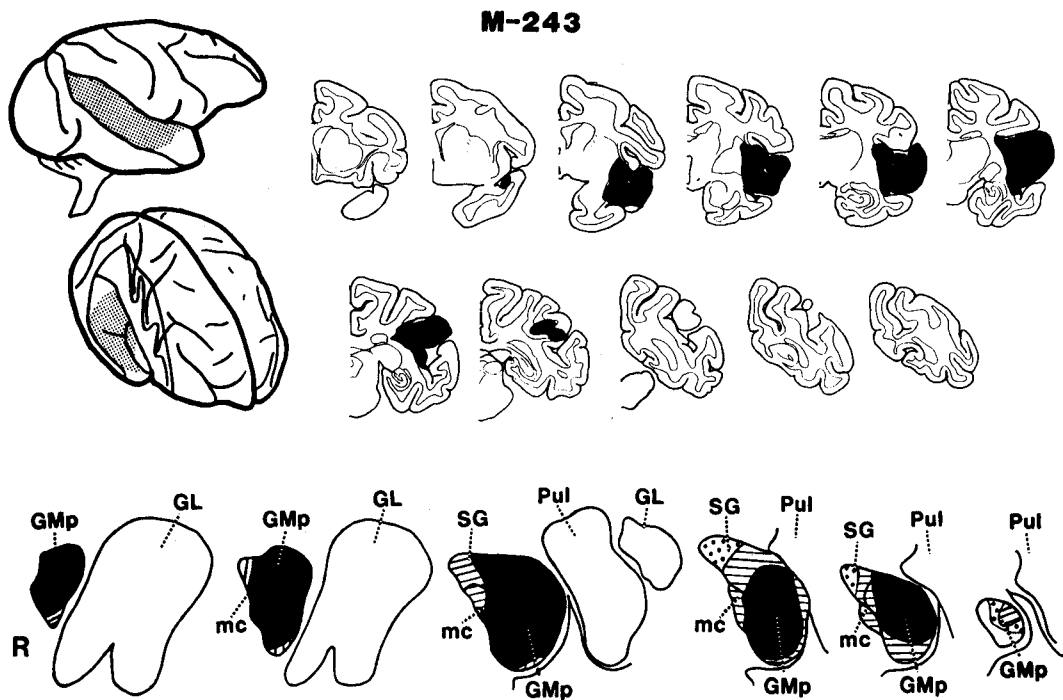


FIG. 8. Cortical reconstruction and medial geniculate degeneration for M-243 that received a right unilateral lesion. See Fig. 1 for key.

training procedures in an attempt to retrain them on the original coo discrimination. The following section presents the results of these tests on four of the monkeys. The fifth monkey, M-57, sustained such a severe and long-lasting hearing loss that its inability to discriminate the coos could not be confidently separated from the loss of sensitivity.

DETECTION OF THE COOS. The animals were tested for their ability to detect the presentation of the SE coos against a background of silence. All four of the monkeys were eventually able to perform this task perfectly (i.e., with a performance ratio of 1.0). M-37 demonstrated this ability 15 wk after the second surgery, M-150 at 2.5 wk, M-246 at 38 wk, and M-252 at 9 wk. It should be noted that these times probably represent an underestimate of the speed of recovery as the animals could not be tested on this task daily because their audiograms were also being determined during this time.

DISCRIMINATION OF THE COOS. The monkeys were tested for their ability to discriminate the seven SE and eight SL coos at various times

throughout their postoperative recovery period. This discrimination was last tested in M-37 at 29 wk after surgery, in M-150 at 35 wk, in M-246 at 40 wk, and in M-252 at 47 wk. At no time did any of the four animals show any indication of being able to perform the discrimination above chance levels even when the intensity of the coos was increased to compensate for their hearing loss (Figs. 11 and 12). Thus the animals appeared to have permanently lost the ability to discriminate the two types of coos.

DISCRIMINATION OF A SINGLE PAIR OF COOS. Once it became clear that the monkeys could not discriminate the entire set of coos, we attempted to train them to discriminate a single SE coo from a single SL coo. However, in spite of over 1,000 training trials, none of the four animals could reliably discriminate a single pair of coos. Although occasionally an animal's performance on a particular pair might rise above chance, varying the intensity of the coos over a 6-dB range reduced performance to chance indicating that the animal was using

M-246

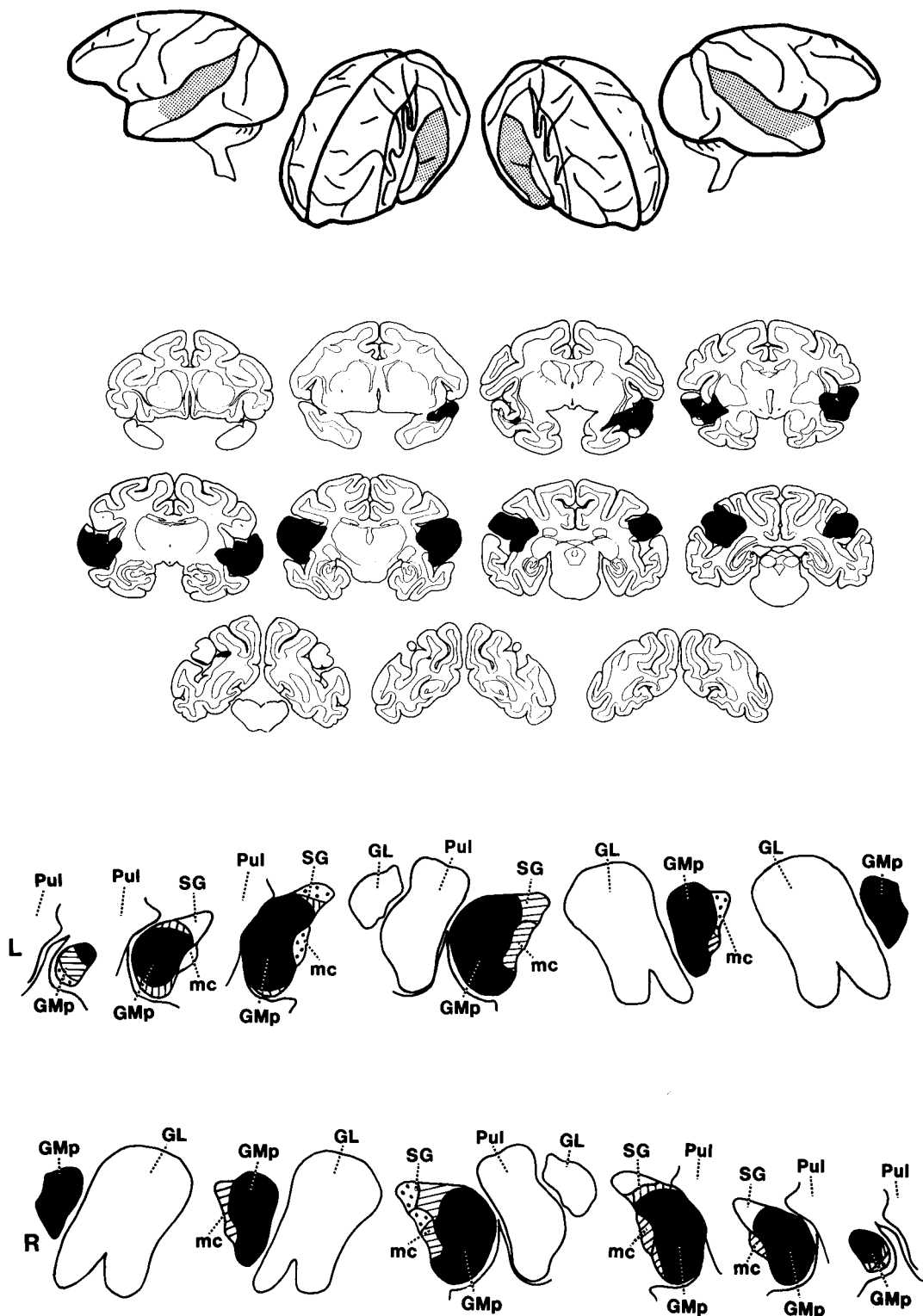


FIG. 9. Cortical reconstruction and medial geniculate degeneration of M-246 that received a right unilateral lesion followed by a left lesion. See Fig. 1 for key.

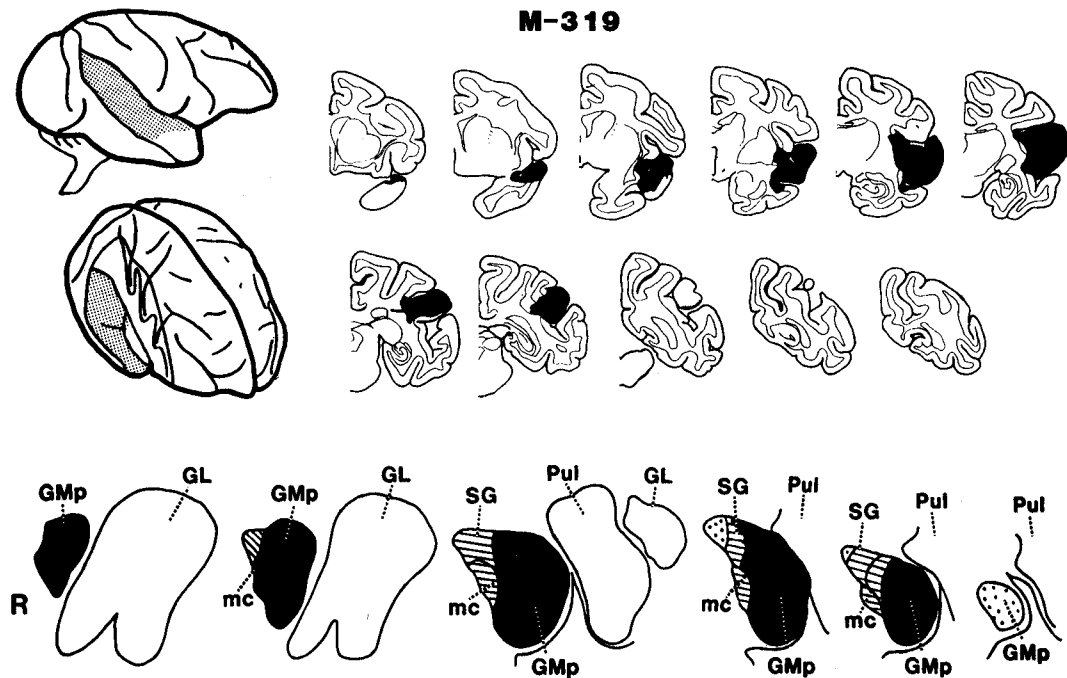


FIG. 10. Cortical reconstruction and medial geniculate degeneration for M-319 that received a right unilateral lesion. See Fig. 1 for key.

an intensity difference between the two particular coos in order to discriminate between them. This inability to discriminate a single pair of coos was last demonstrated in M-37 at 30 wk after surgery, in M-150 at 29 wk, in M-246 at 42 wk, and in M-252 at 44 wk.

DISCRIMINATION OF COOS VS. NOISE. To determine whether the monkeys were capable of performing any auditory discrimination, three of the animals were tested for their ability to discriminate the seven SE coos (warning signal) from a noise burst (safe signal) that was of the same general duration as the coos. All three of the monkeys easily made this discrimination. M-37 had an asymptotic performance of 0.88 at 17 wk after surgery, M-150 performed at 0.97 at 12 wk, and M-252 performed at 0.96 at 26 wk. Thus the animals were able to demonstrate some auditory discriminatory ability.

DISCRIMINATION OF COOS VS. TONES. In a final test, three of the monkeys (M-37, M-150, M-252) were tested for their ability to discrim-

inate the SE (warning signal) coos from tone pips (safe signal) of the same general duration as the coos (400 ms). The animals were required to discriminate the seven SE calls from four tones (500 Hz, 1, 2, and 4 kHz), which were presented in the same session. The results of this test indicated that discriminability was related to frequency with the monkeys being able to discriminate the higher frequencies from the coos, but not the lower frequencies (Table 2). Specifically, all three animals confused 500 Hz and 1 kHz with the coos and only 2 and 4 kHz could be reliably (though not perfectly) discriminated from the coos. Although performance could be improved by presenting only one of the tones during a session, two of the monkeys (M-37 and M-252) were unable to distinguish 500 Hz from the coos even in this simplified situation.

The difficulty experienced by the monkeys in attempting to discriminate 500 Hz and 1 kHz from the coos is probably due to the fact that these two tones are close to the fundamental frequencies of the coos whose starting frequencies ranged from ~500 to 750 Hz (1).

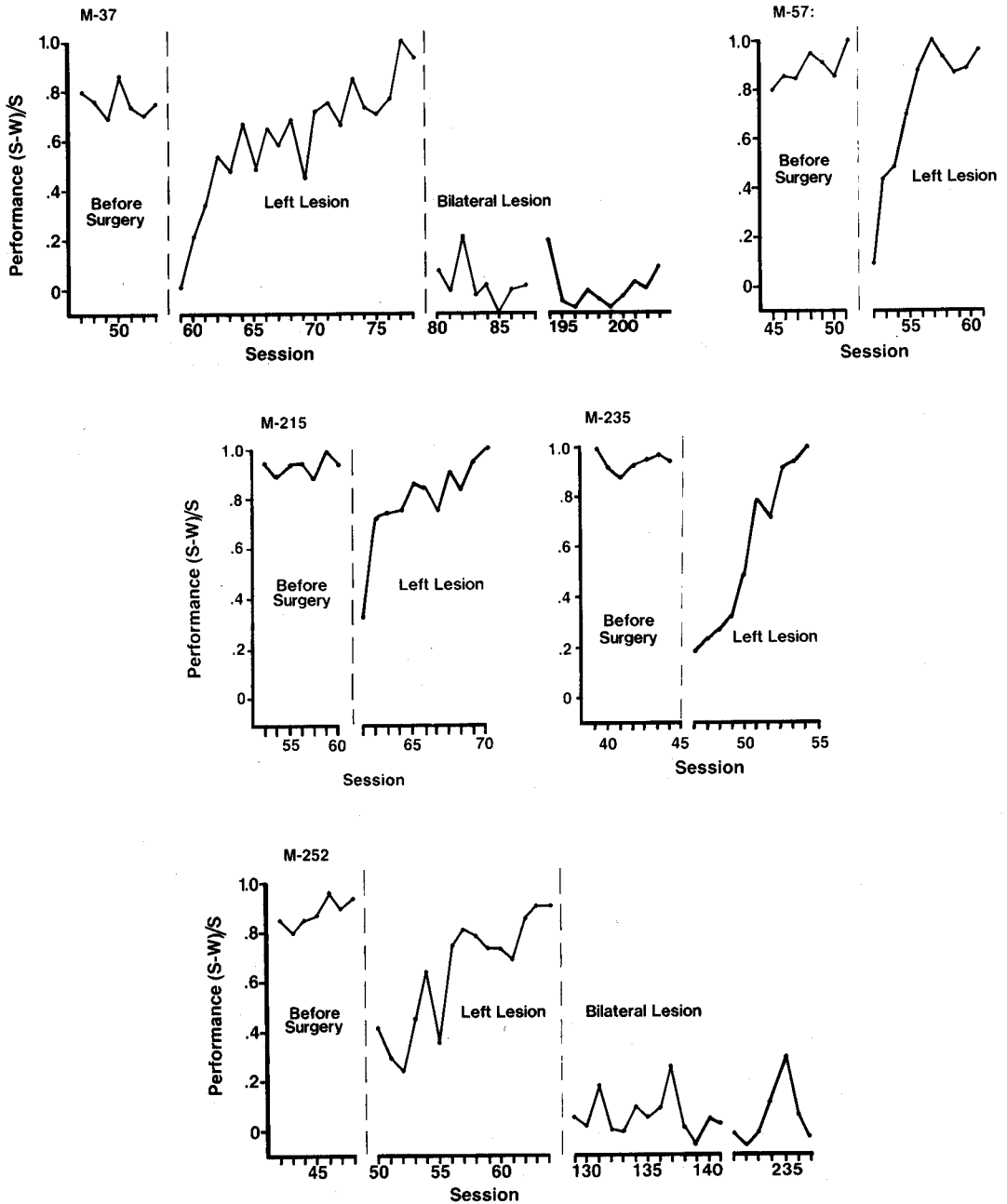


FIG. 11. Effect of *left* unilateral and bilateral auditory cortex lesions on the ability to discriminate the coos. Each of the 5 monkeys showed an initial deficit following unilateral ablation. Bilateral ablation rendered the animals permanently unable to discriminate the coos (M-37 and M-252).

While the fact that the animals had little difficulty distinguishing 2 and 4 kHz from the coos indicates that they could perform a gross

frequency discrimination, their difficulty with the lower frequencies suggests a general impairment in the ability to distinguish sounds.

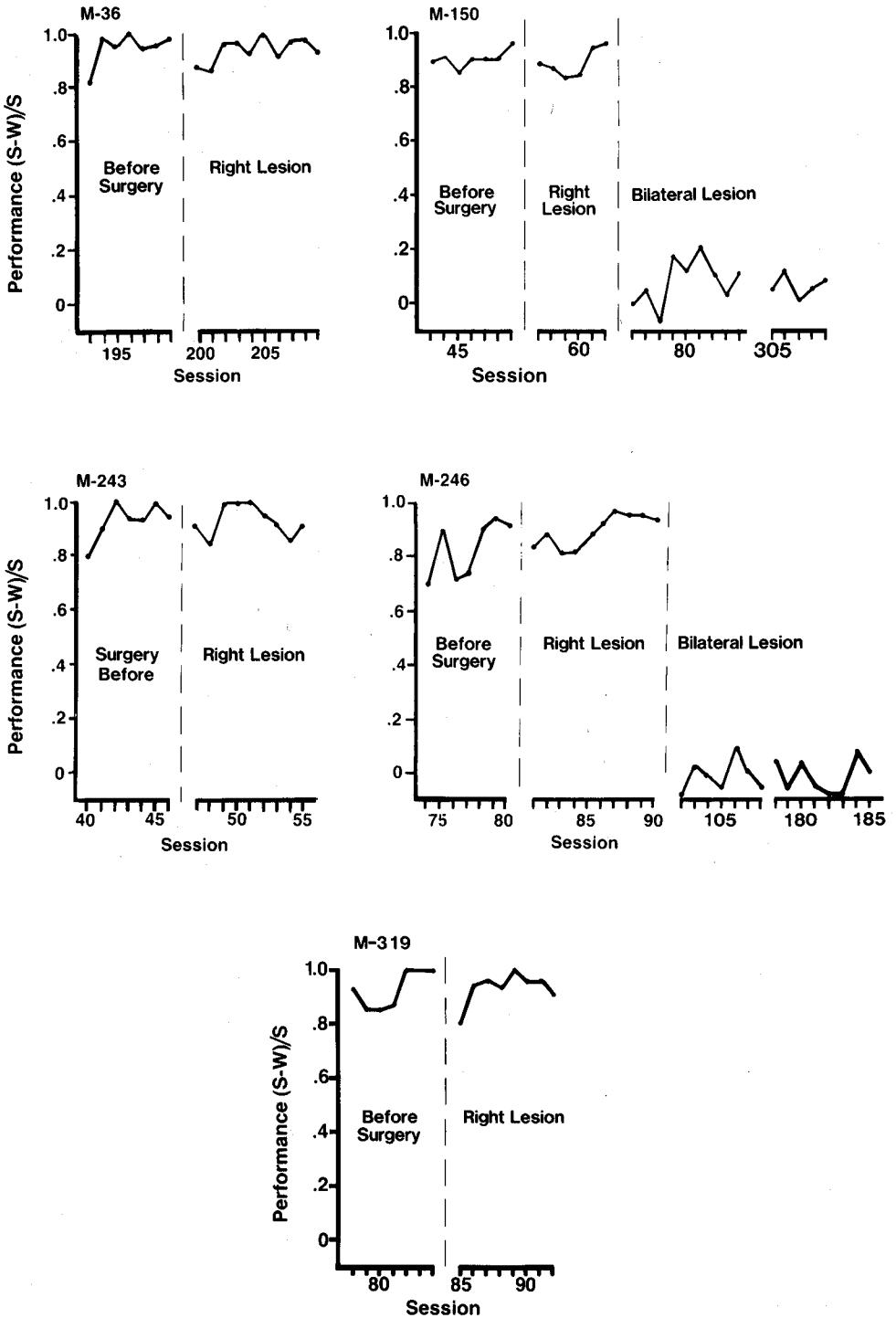


FIG. 12. Effect of *right* unilateral and bilateral auditory cortex lesions on the ability to discriminate the coos. Right unilateral ablation had no noticeable effect on the discrimination. Subsequent bilateral ablation rendered the animals permanently unable to discriminate the coos (M-150 and M-246).

TABLE 2. *Percent correct responses to tones during tone vs. coo discrimination for three monkeys*

Stimulus	M-37	M-150	M-252
500 Hz	0	5	3
1 kHz	13	22	28
2 kHz	53*	45*	66*
4 kHz	48*	50*	62*

The four tones were presented as the "safe" signals during a single session for each monkey. * Tones that could be reliably discriminated from the coos ($P < 0.01$).

DISCUSSION

Unilateral deficit

The transient impairment in the discrimination of the coos following ablation of left auditory cortex suggests that the perception of vocalizations is mediated primarily by the left hemisphere. However, the rapid recovery of the animals indicates that another area can assume this function. The fact that subsequent ablation of right auditory cortex resulted in a complete and permanent deficit is evidence that right auditory cortex can take over this function following ablation of left auditory cortex.

Recently, we have noted that unilateral ablation of auditory cortex results in a hearing loss in the ear contralateral to the lesion (17, 19). While the possibility that such a hearing loss may have contributed to the deficit in discriminating the coos, there are two reasons that suggest that this is not a likely possibility. First, the largest unilateral hearing losses observed so far have been relatively mild, averaging from 21 to 36 dB. The coos, on the other hand, were presented at least 55 dB above the 50% detection level and could be attenuated 40 dB without degrading the normal animals' performances. Second, thresholds in the ipsilateral (left) ear are unaffected, and listening with this ear alone should be sufficient to perform the discrimination easily as the right ear advantage for this task is quite subtle. Nor, for that matter, should a unilateral hearing loss in itself disrupt the discrimination, since the right lesion cases had no such difficulty. Thus the observed deficit in the perception of coos following left auditory cortex lesions appears to be due to the fact that this function is later-

alized to the left hemisphere with the right hemisphere compensating for left hemisphere damage.

The discovery of a left hemisphere deficit is consistent with the observation of a small right ear advantage for the perception of the coos (1, 30, 31, 34). Furthermore, these results suggest that the neural mechanisms involved in the perception of vocalizations in monkeys are similar to those of humans. In both there is a right ear advantage for the perception of communication sounds, and left auditory cortex lesions result in an impairment in the ability to perceive these sounds. One difference between the two species, however, is that while such lesions in humans result in an incapacitating disability in the perception of speech (e.g., Refs. 2, 14), the monkeys in this study showed only a minor and transient disability. Yet the discrimination of two types of coos in an avoidance situation is a fairly simple task and coos constitute a small part of the Japanese macaque's vocal repertoire (cf. Ref. 12). Therefore, it is perhaps premature to conclude that the lateralization in monkeys is much less strong than that which occurs in humans.

Two other studies have been conducted into the lateralization of auditory function in primates. In a series of papers, Dewson (5, 6, 7) described a deficit in auditory association that results from left, but not right, auditory cortex ablation. Briefly, left hemisphere lesions in macaques resulted in a decrement in performance on a two-choice task when a delay is inserted between the presentation of the stimuli and the animal's response. This result was interpreted as indicating a left hemisphere specialization for auditory processing.

A second study investigated the effect of auditory cortex lesions on the ability of squirrel monkeys to discriminate squirrel-monkey vocalizations from other sounds (21). The results of this study did not show any effect of unilateral ablation, whereas large bilateral lesions permanently abolished the discrimination. The fact that this study failed to find any effect resulting from ablation of left auditory cortex may be due to a lack of lateralization of auditory function in squirrel monkeys. However, the deficit observed in Japanese macaques is not large and could be easily overlooked. In addition, only two of the squirrel monkeys received lesions large enough to result in a deficit

when they were made bilateral. Therefore, it is not impossible that further study may reveal lateralization in squirrel monkeys.

There are at least three aspects of the unilateral deficit in Japanese macaques that remain to be explored. First, the question arises as to the exact location of the area involved in the perception of vocalizations. In humans, this area (Wernicke's area) is generally considered to lie in the secondary auditory fields just dorsal to primary auditory cortex (3). It might be expected, then, that smaller lesions in macaques might also be sufficient to produce the deficit seen here. Second, the nature of the impairment needs to be explored to determine whether it is peculiar to the perception of vocalizations or whether other aspects of auditory perception are also lateralized. Finally, it is possible that the right hemisphere is not completely able to compensate for damage to the left hemisphere. The question thus arises as to whether there is some permanent deficit following left auditory cortex ablation.

Bilateral deficit

Bilateral auditory cortex lesions rendered the monkeys completely unable to discriminate the coos. Not only could they no longer discriminate the two sets of coos, but they were unable to learn to discriminate between a single pair of coos. Yet the animals had clearly not lost the ability to perform all auditory discriminations as each of the three monkeys were able to discriminate the SE coos from a noise burst and from 2 and 4 kHz tones. However, the animals had great difficulty in discriminating the coos from 500 Hz and 1 kHz.

The difficulty experienced by the animals in discriminating the coos from low-frequency tones is probably not due to a simple deficit in frequency discrimination. Numerous studies have demonstrated that monkeys with auditory cortex lesions retain the ability to perform at least a basic frequency discrimination

(e.g., Refs. 9, 20, 24). However, the coo discrimination differs from a simple frequency discrimination in that it consists of the discrimination of sounds that are acoustically complex, but biologically simple. That is, although the spectra of the coos are physically complex, the coos are easily discriminated by the monkeys according to the biological relevance of the sounds. As a result, it is tempting to conclude that the effect of the cortical lesions is to abolish the ability of the monkeys to discriminate the coos on the basis of their biological relevance and that they lack the ability to discriminate the sounds solely on their acoustic differences.

Along this line, it is important to note that auditory cortex lesions have been shown to disrupt the ability of monkeys to discriminate acoustically complex sounds that lack biological relevance. Macaques with bilateral auditory cortex lesions have been shown to be unable to discriminate both intermittent noise (33) and human speech sounds (8). These results suggest that one effect of cortical ablation is to disrupt the discrimination of acoustically complex sounds per se. Thus the question of whether the deficit in the discrimination of the coos is the result of an inability to perceive the biological relevance of sounds, an inability to discriminate the acoustic properties of complex sounds, or a combination of the two remains to be determined.

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