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Role of Cat Primary Auditory Cortex for Sound-Localization Behavior

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SUMMARY AND CONCLUSIONS

1. Small lesions designed to completely destroy the cortical zone of representation of a restricted band of frequency were introduced within the primary auditory cortex (AI) in adult cats. Physiological mapping was used to guide placement of lesions. Sound-localization performance was evaluated prior to and after induction of these lesions in a seven-choice free-sound-field apparatus. All tested cats had profound contralateral hemifield deficits for the localization of brief tones at frequencies roughly corresponding to those whose representations were destroyed by the lesion. Sound-localization performance was normal at all other test frequencies.

2. In a single adult cat, a massive lesion destroyed nearly all auditory cortex unilaterally, with only the representation of a narrow band of frequency within AI spared by the lesion. This cat had normal abilities for azimuthal sound localization across that frequency band but a profound contralateral deficit for the azimuthal localization of brief sounds at all other frequencies.

3. Recorded sound-localization deficits were permanent.

4. Localization of long-duration tones was not affected by a unilateral AI lesion.

5. These studies indicate that, at least in cats, *1*) AI is necessary for normal binaural sound-localization behavior; *2*) among auditory cortical fields, AI is sufficient for normal binaural sound-localization behavior; *3*) sound-location representation is organized by frequency channel in the auditory forebrain; and *4*) AI in each hemisphere contributes to only contralateral free-sound-field location representation.

INTRODUCTION

The earliest reports of the consequences of lesions of the auditory cortex described deficits in hearing in the ear opposite the lesion (23, 53). The descriptions of the brain-damaged monkeys and dogs in these historic studies leave little doubt that the lesions resulted in contralateral free-sound-field localization deficits and not in contralateral hearing deficits per se (as originally suggested). Similar apparently straightforward localization deficits have been reported in some studies of humans with unilateral superior temporal (auditory cortex) lesions (49, 72, 92). Surprisingly, less significant or no deficits were recorded in other studies in patients with similar lesions (77, 86). In parallel, cortical ablation-behavioral studies were conducted in cats, using standard behavioral conditioning techniques and using the identification of auditory cortical zones provided by the studies of Rose and Woolsey and colleagues (68–70, 90) as a guide for defining ablation boundaries. Unilateral destruction of virtually all of auditory responsive cortex produced no profound permanent localization deficits in cats tested in a two-choice sound-localization task (78). Thus over the last 100 yr early investigators apparently described deficits in sound localization resulting from unilateral cortical lesions in dogs and monkeys, whereas later investigators found little if any apparent loss in sound-localization ability resulting from such lesions. In humans, significant deficits were observed by some investigators; whereas other subjects with presumably similar large brain lesions were described as having virtually normal sound-localization capabilities.

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Recent studies have emphasized experimental differences that led to these strikingly different conclusions about the possible role of auditory cortex in sound-localization behavior. They have shown that in cats, ferrets, dogs, and monkeys large unilateral lesions of auditory cortex produce profound and apparently permanent deficits in the ability to localize brief sounds in the hemifield contralateral to the brain lesion. No deficit is recorded in the ipsilateral hemifield nor in making a left-right discrimination (a "lateralization") (31, 39, 43, 47, 82). In fact, earlier investigators, who had found a sound-localization deficit, had evaluated the consequences of the cortical lesion for true binaural sound-localization behavior, whereas those recording no deficit had studied the cortical contributions to so-called "lateralization" behavior (43). Following a unilateral lesion, the latter behavior need be no more complicated than the discrimination of a sound field in which the sound is sharply localizable from the sound field in which it is only poorly localizable. Jenkins and Masterton's (43) studies also indicated that true binaural sound localization is processed within the tonotopic "mainline" auditory system, as damage to nuclei all along this axis disrupted the behavior. Consistent with that view, psychophysical studies have long indicated that binaural sound-localization information is processed by frequency channel (6, 12, 22, 32, 64, 74), and it would therefore appear to be necessarily a product of the tonotopic system axis (57).

In the Jenkins and Masterton study (43), a profound contralateral hemifield sound-localization deficit was seen after a massive unilateral lesion of the auditory cortex in the cat. In this present study, we have attempted to extend and refine these results by testing the hypotheses that *1*) the primary cortical field (AI) is necessary for normal azimuthal sound-localization behavior; *2*) an intact AI (but no other auditory cortical field) is sufficient for normal binaural sound-localization behavior; and *3*) sound-localization representation is by frequency channel.

To address these questions *1*) normal sound-localization behavior was studied in individual cats using a seven-speaker behavioral apparatus to assess azimuthal sound-localization ability; *2*) lesions restricted to only a part of AI representing a restricted band of frequencies were then introduced after the relevant internal organization and boundaries of AI were defined in a microelectrode mapping study; and *3*) azimuthal sound localization was then again evaluated, to determine the behavioral consequences of these very small lesions. These studies confirmed and extended the basic cortical ablation-behavioral results of Jenkins and Masterton (43). Somewhat to our surprise, results were consistent with all three hypotheses under test. Preliminary reports of these results have been presented earlier (40, 44).

METHODS

Adult cats with no signs of ear pathology were used as subjects in these experiments. Six cats provided normal and experimental data, whereas two cats provided only normal data. All animals were initially trained to localize the sources of brief noise bursts. All of these normal cats underwent additional sound-localization testing with brief pure-tone pips of selected frequencies. Subsequently, *1*) microelectrode best-frequency maps of auditory cortex were derived; *2*) cortical lesions whose boundaries were guided by these maps were induced; and *3*) after recovery from surgery, postlesion sound-localization testing was conducted. After completion of a postlesion sound-localization test series, an attempt was made to again derive a physiological map of auditory cortex in the region of the lesion. At the conclusion of each experiment, cats were deeply anesthetized with pentobarbital sodium and perfused intracardially. Their cortical lesions were subsequently reconstructed histologically.

Behavioral testing

APPARATUS. The essential features of the multiple-speaker sound-localization test apparatus used in this series have been described in detail elsewhere (43, 80). It consisted of a semicircular enclosure mounted in an Industrial Acoustics acoustical chamber (Fig. 1). Its outer wall and floor were made of wire mesh in order to reduce sound-reflective surfaces. The apparatus was raised off the carpeted floor to further minimize echoes. The inner walls and ceiling of the acoustical chamber were hung with folded burlap.

Seven speakers were mounted 30° apart along 180° of the azimuthal plane. Small basins attached to electrically actuated water valves were mounted below each speaker to deliver the rewards for correct responses.

To effect standard alignment of the cat's head at the beginning of each trial, the start chute was positioned so that when the cat walked through it

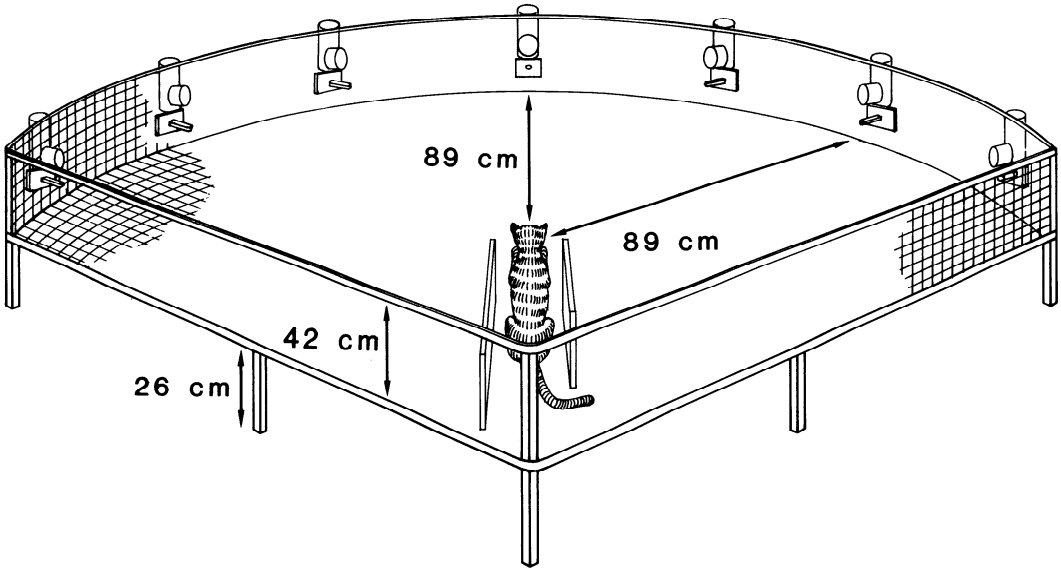


FIG. 1. Seven-speaker behavioral apparatus used for testing azimuthal sound-localization ability in cats. Cat is shown in standard start position.

its head passed through the geometric center of the semicircle of speakers off the end of the chute. An overhead TV camera allowed observation and recording of the head's alignment with the center speaker. When the cat's head was in a static position and a straight-ahead alignment was established, the experimenter initiated the trial. Control of the sound duration and location as well as data collection and reward delivery were accomplished with an Apple II microcomputer and associated interfaces (42).

STIMULI. On each trial, a single-tone pip or noise burst (40-ms duration, 5-ms rise-fall) at ~70-dB sensation level was presented from one of the speakers (61). A duration of 40 ms was selected since reflexive reactions of a cat's head to a sound have a latency of ~40 ms (81), and therefore scanning of the sound field for an intensity gradient that might be correlated with the azimuth of the source is unlikely (see results on the effect of duration, Fig. 13, below). The signal from either an oscillator (H-P 204C) or wide-band noise generator (41) was passed through a rise-fall gate (Coulburn), a wide-range attenuator (H-P 350D), an audio amplifier (Crown D75), an impedance-matching transformer, a switching network, and finally to one of the speakers (Realistic 35 in. 40-1333A). Each speaker was equipped with its own matching attenuator. To provide sufficient output at low frequencies, speakers were mounted in sealed tubular enclosures.

Speakers were oriented toward the center of the semicircle of the array, i.e., toward the cat's standard head position, at which all trials were initiated. A microphone (0.5 in. B & K) suspended at this center point was used to calibrate (match) the intensities of all speakers for each sound stimulus, using a sound level meter (B & K). Measurements of stimulus intensity within 6 cm of the center point did not vary by more than 5 dB (re 20 μPa).

BEHAVIORAL TASK. Repeated sound-localization tests were administered in each case. The apparatus and testing procedures were identical for all tests. Test variables were the frequency, intensity, and duration of the sound.

Each cat was deprived of water for ~23 h, and correct responses were rewarded by the automatic delivery of a measured amount of water to a small basin below each speaker. To begin a trial, the thirsty cat was required to walk through the start chute and align its head toward the 0° speaker.

On any one trial, a noise burst or tone pip too brief to allow either homing or scanning movements of head or pinna was presented at one of the seven speakers (38, 81). If the cat approached and made contact with the water basin mounted beneath the speaker that had sounded, it received 1 ml of water as a reward, and the trial was scored as correct. The cat could then begin another trial by again walking through the start chute. If, instead, the cat first made contact with the water basin beneath a speaker that had not sounded, no reward was given, the trial was scored as incorrect, the response position was recorded, and a brief (usually 5 s) "time-out" occurred. After expiration of the time-out, the cat could begin another trial

by again walking through the start chute. After 42 rewards, the test session was terminated and the cat was allowed 15 min of free access to water in its home cage. This procedure ensured that the cats were not satiated during the test procedure and that they obtained sufficient water to maintain their health.

Both to minimize position biases and to provide intensive training for the sound directions that were the most difficult for the cat to discriminate, the sound locus was not changed after an error. Instead, the trial was repeated until a correct response was made. If the cat had not made a correct response after three attempts, the stimulus duration was set to 50 ms and the rise-fall time to 0 ms. This stimulus (i.e., generating onset and offset clicks) nearly always resulted in a correct response. It ensured that the cat was rewarded an equal number of times at each position and that extra training trials would be provided at any position proving to be a difficult one. However, only trials immediately following a randomization of the sound position were used to assess sound-localization ability. It is the percentage correct on these trials that is the chief behavioral measure in this report.

Pre- and postsurgical behavioral testing always started with use of broad-band noise stimuli. On completion of 10 consecutive daily sessions (42 trials per session) of testing with noise, pure-tone testing was initiated. Selection of stimulus frequency was pseudorandom but remained constant for 10 consecutive daily sessions when another frequency was selected.

To further investigate the nature of the observed behavioral deficits, ancillary tests were administered following completion of basic noise and pure-tone testing. In these studies, either tone intensity or tone duration was varied pseudorandomly from one daily test session to the next until data from five sessions at each intensity or duration had been obtained. For the tone-intensity series, as well as the standard frequency and intensity combinations, measurements of harmonic distortion were obtained for each frequency and intensity combination with a spectrum analyzer (Nicolet, U-500). Harmonic distortion for the standard test series was at least 50 dB below the test frequency.

Surgical and electrophysiological procedures

For surgery and limited prelesion cortical mapping studies, cats were anesthetized with intravenous injections of pentobarbital sodium (40 mg/kg), with supplemental doses given as required to maintain an areflexive state. Body temperature was maintained at 38°C. Sterile techniques were employed throughout this 4 to 6-h procedure.

For the cortical exposure, a midline incision was made, the skin and underlying muscle were retracted, and the bone overlying auditory cortex was removed and stored in refrigerated sterile Ringer solution. The dura overlying the auditory cortex was cut and retracted. Auditory cortex was protected under a pool of silicone oil in a well walled by cotton soaked in Ringer solution. The surface of the cortex was photographed and a 20–30× print was produced. The activity of small clusters of units and single units was recorded in multiple intracortical penetrations of parylene-coated tungsten microelectrodes (1 to 2-MΩ impedances). In any given experiment, all penetrations were parallel to each other and were approximately normal to the cortical surface. Electrode penetration locations were sited by cross reference of the brain photograph and the cortical vascular pattern as viewed in a Zeiss operating microscope. "Best frequencies" (and in some experiments binaural response characteristics) were defined in the middle cortical layers, in which reliable responses can be evoked under barbiturate anesthesia.

The sound stimuli were shaped with 5-ms rise-fall time and were 150 ms in duration repeated at 1 per sec. Tones were delivered using calibrated headphones (Beyer) coupled to hollow ear bars that were sealed into the ear canals. The hollow ear bars were specially designed to accept a 0.5 in. B & K microphone for calibration. Additionally, the size and taper of the ear bars did not allow the bars to contact the tympanic membrane. Great care was taken to ensure that the placement of the ear bars did not injure the ear canal wall. At the conclusion of the electrophysiological experiments, the ears were examined. No evidence of significant trauma was observed in any case (also see Histological methods, below). Search stimuli consisted of moderately intense pure tones delivered monoaurally to the contralateral ear. The frequencies and intensities of tone pips were under manual control. The best frequencies of unit clusters and single units were identified by finding the frequency of the lowest intensity tone pip that would consistently evoke a neural response. In our hands, this resulted in the definition of best frequencies at ~5–10 dB above threshold at their characteristic frequencies. Best frequencies were defined at from 25 to 76 cortical sites in these prelesion studies.

A cortical lesion was induced after collection of best-frequency data sufficient to determine the approximate dorsal and ventral boundaries of the primary auditory cortical field (AI), its isofrequency axes, and the frequency-band representation to be included in the planned cortical lesion. To induce the ischemic cortical lesion, the cortex was first rinsed with sterile Ringer solution to remove the

silicone oil. Using microforceps connected to a bipolar electrocoagulator (Codman) and with the aid of the operating microscope, all blood vessels were occluded as they entered the cortical surface in the zone of the intended lesion. Care was taken to preserve major crossing arteries while occluding all of their local branches. The effects of these ischemically induced lesions were immediately apparent by the dramatic change in cortex to a blanched white-gray color. Cortical shrinkage was also dramatic in the lesioned zone.

After lesion induction, the dura was resutured and the bone plug was replaced with use of stainless steel sutures. The retracted muscle, galea, and skin were sutured in place. Again, sterile conditions were maintained throughout these surgical and mapping procedures.

After surgery, animals were maintained on antibiotics, and their temperatures were maintained throughout recovery. Recovery was rapid; each cat appeared to be generally behaviorally normal within a few days. However, in order to ensure that postoperative discomfort played no part in deficits that might be later uncovered, behavioral training and testing was not begun in any case until at least 20 days after surgery.

After the postlesion behavioral test series was completed, an attempt was made to again derive a best-frequency map in the zone of the cortical lesion. Because of dural adhesions, it was usually impossible to complete such maps without risking further cortical damage. For two of seven hemispheres studied in six cats, relatively complete maps were obtained. In others, only partial postlesion maps or no postlesion maps were obtained.

Histological verification of lesions

At the conclusion of the final mapping experiment, each cat was deeply anesthetized with barbiturate and then perfused with normal saline followed by formal-saline. The cranium was removed and the brain was immersed in refrigerated formal-saline for at least 24 h. The outer and middle ears were dissected and examined. In every case both ears appeared normal. Brains were then removed, photographed, and allowed to soak in sugar-formalin until they sank. They were then embedded in gelatin and cut in 33-μm thick sections. Alternate sets of sections were stained for Nissl substance (cresyl violet) or myelin (Weil). The locus and extent of the damaged and degenerated tissue in each case was then reconstructed by microscopic analysis of these sections.

These lesions commonly resulted in the formation of a false sulcus within AI (see Fig. 16). Histological boundaries of lesions were relatively sharp and were usually marked by a transition from normal-appearing cortex to a zone with few or no neurons or with a complete collapse to white matter over a distance not greater than a few tens of microns. In several cases, there were significant areas in the lesioned zone in which there was complete cell loss in superficial cell layers (layer 4 and above) but substantial cell survival in deep layers (especially in layer 6). Further details are provided in RESULTS.

RESULTS

The major result of this report is that restricted unilateral lesions of frequency-band representational sectors within primary auditory cortex (AI) produce corresponding frequency-dependent sound-localization deficits in the contralateral sound field. To validate the interpretation of the results of these experiments, it is first necessary to document the sound-localization abilities of normal cats, studied under conditions identical to those used to evaluate experimental cases. Representative cases with unilateral AI lesions of limited frequency-band sectors will then be described. These cats exhibited frequency-dependent contralateral sound-localization deficits; the nature of those deficits and their relation to specific cortical lesions will be described in detail. Additional results from other special preparations that bear on the nature and on the true site(s) of origin of the observed behavioral deficits will be presented. Finally, some relevant histological features of the small intrafield lesions made in these experimental cases will be described.

Sound-localization ability in normal cats

These experimental studies were undertaken to further test the hypotheses that auditory space is represented *1*) contralaterally, *2*) by frequency channel, and *3*) in the primary auditory cortical field. To evaluate possible deficits, it was necessary to first determine the sound-localization capacity of normal cats for brief pure tones. The sound-localization ability of one normal cat for noise and for eight pure tones is shown over 10 consecutive daily sessions in Fig. 2. There, each data point represents the average performance for the three speakers located either in the cat's left or right hemifield (18 trials per point). The chance line in Fig. 2 is drawn at the 33.3% level to allow for direct comparison with the results for cats with only hemifield deficits. The rational for selecting

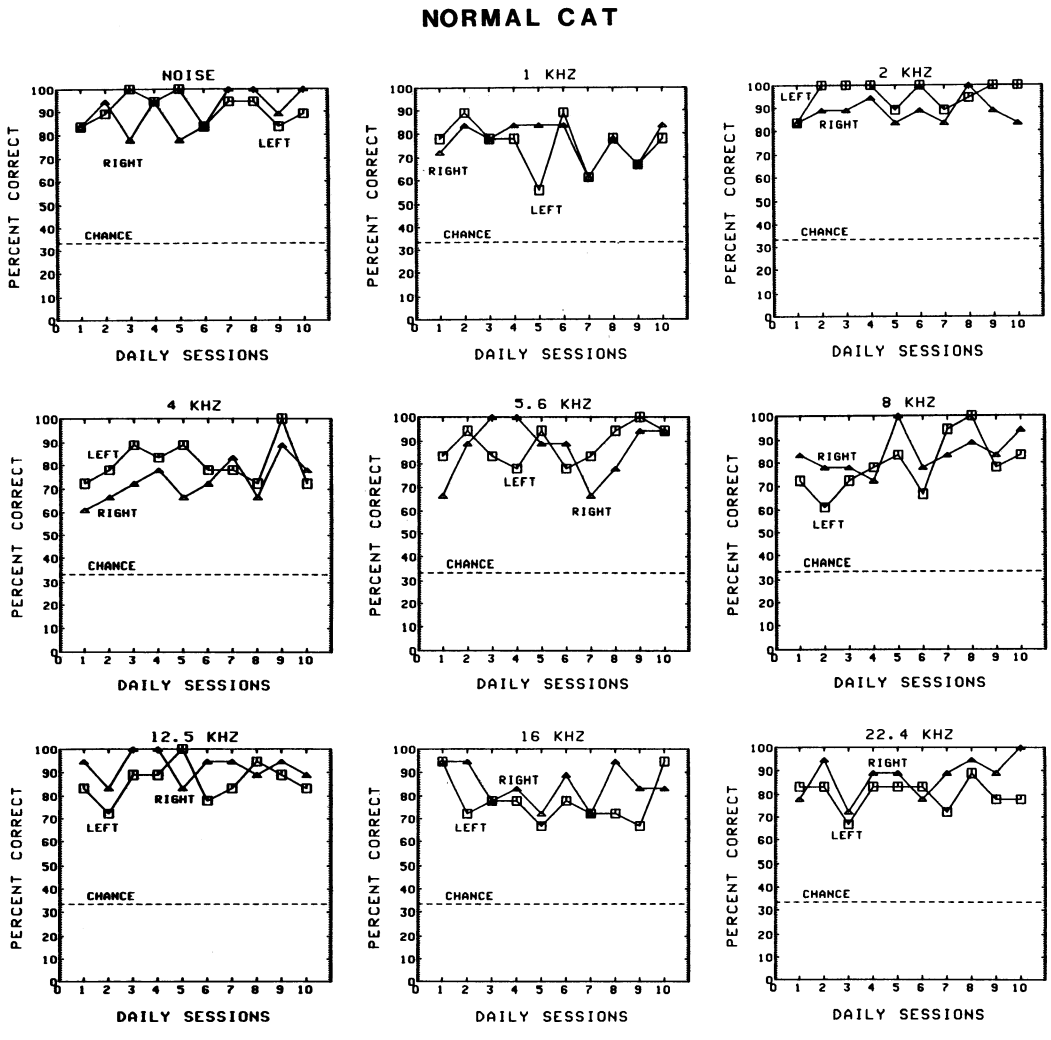


FIG. 2. Sound-localization performance of a normal cat for 40-ms-duration noise bursts and tone pips over 10 consecutive daily sessions. Stimulus frequencies are indicated above each graph. Each data point represents average performance for 3 speakers on either left or right side (18 trials per point). Note that there is little difference in performance between speakers located to left or right of animal at any frequency tested. Chance-level performance for either hemifield is indicated with each plot.

the 33.3% chance level for statistical purposes in cases that exhibit only contralateral hemifield deficits is described in detail elsewhere (43).

Two sources of variability in sound-localization performance for this normal cat are apparent (Fig. 2). First, average performance for speakers located to the cat's left or right hemifield varied from day to day. When both left and right performance levels changed from the previous day, the direction of change was usually the same in both fields. For example, in the case illustrated in Fig. 2, 41 changes in performance level were in the same direction for both left and right fields, whereas 18 changes were in the opposite direction (binomial test, $P = 0.5$, $N = 59$, $p = 0.0009$). This suggests that the day-to-day variation in performance was a result of factors common to both left and right fields.

Performance results also varied as a function of stimulus frequency. For example, compare the results obtained with stimulation at 1 and 2 kHz (Fig. 2). At both of these

frequencies, performance in the left and right fields were similar. Differences in performance as a function of frequency presumably principally reflect variations in binaural cues and their central neural processing that are common to both the left and right fields.

Since there appeared to be some progressive improvement in performance over the ten days of testing at some stimulus frequencies (for example, compare *day 1* vs. *day 10* at 8 kHz in Fig. 2) and since this was sometimes more apparent in experimental cases, only behavioral results from the last 5 days of testing with a particular stimulus condition were used for further analysis. This simplification had no impact on conclusions drawn from the study of normal cats but, as will be shown later, could in a few instances significantly affect the interpretation of data from experimental cases.

Another source of performance variability is illustrated in Fig. 3, where average performance (last 5 test days; 30 trials per point) of the normal cat shown in Fig. 2 is plotted as a function of speaker location. It can be seen that the average performance as a function of speaker position differed under every test condition. Performance as a function of speaker position was never perfectly symmetrical about the midline for any cat tested under any stimulus condition. Furthermore, the asymmetries as a function of speaker position were not consistent between normal cats. This last fact indicates that there were no differences of any consequence between the speakers (e.g., echoes or spectral distortion). For example, the poor performance exhibited by the normal cat at 1 kHz at the 60° position on the animal's right side was not seen in other normal cats. Furthermore, as will be shown below, average performance in the left and right quadrants was similar.

A data matrix indicating both the number and location of correct and incorrect responses is shown adjacent to each plot (Fig. 3). An important point evident in these data matrices is that errors in localization made by a normal cat were usually to adjacent speakers. For example, out of the 1,890 trials depicted in Fig. 2, there were 237 errors (87.5% correct overall); of these, 226 (95.4% of total errors) were to locations adjacent to the correct speaker, whereas only 11 (4.6% of total errors) were to more distant locations. As will be shown later, this clearly differs from the results obtained in experimental cases.

Again, Figs. 2 and 3 illustrate three sources of sound-localization performance variability for a normal cat: *1*) variability across frequency, *2*) variability across daily test sessions, and *3*) variability across individual speaker locations. To minimize these latter two sources of variability in our data analysis, all of the data for left field and right field speaker locations collected over the last 5 days of a particular test condition were separately averaged. Since the paradigm that was employed involved a comparison of the performance for the two auditory hemifields, the average performance in the left field was subtracted from that in the right field. Figure 4 shows the results of this procedure for the same normal cat described earlier. For this cat, the difference between performance in the left and right field did not exceed 10% for any of the eight tested pure tones or noise.

The question arises as to whether or not the small differences between performance in the left and right hemifield for the normal cat illustrated in Fig. 4 are typical. Obviously, if large differences in performance between the left and right field are common for normal cats, then performance changes in only one field that result from a unilateral lesion would be difficult if not impossible to assess. That this is not the case is shown in Fig. 5, where data derived at selected frequencies in eight different cats is summarized. The differences in sound-localization performance between the left and right fields for these normal cats never exceeded 10%.

The performance of all normal cats tested was characterized by an accurate short-latency-orienting response toward the sound source (e.g., see Refs. 43, 73, 81, 83). Following this response, the source was quickly approached and contacted, completing the discrimination response. When an error was made, it was usually to a location adjacent to the correct speaker, and given a second chance on the trial following the occasional error, the cat almost always made the correct response.

Effects of unilateral strip lesions in AI

Results from a cat with a narrow strip lesion paralleling the isofrequency axis of AI

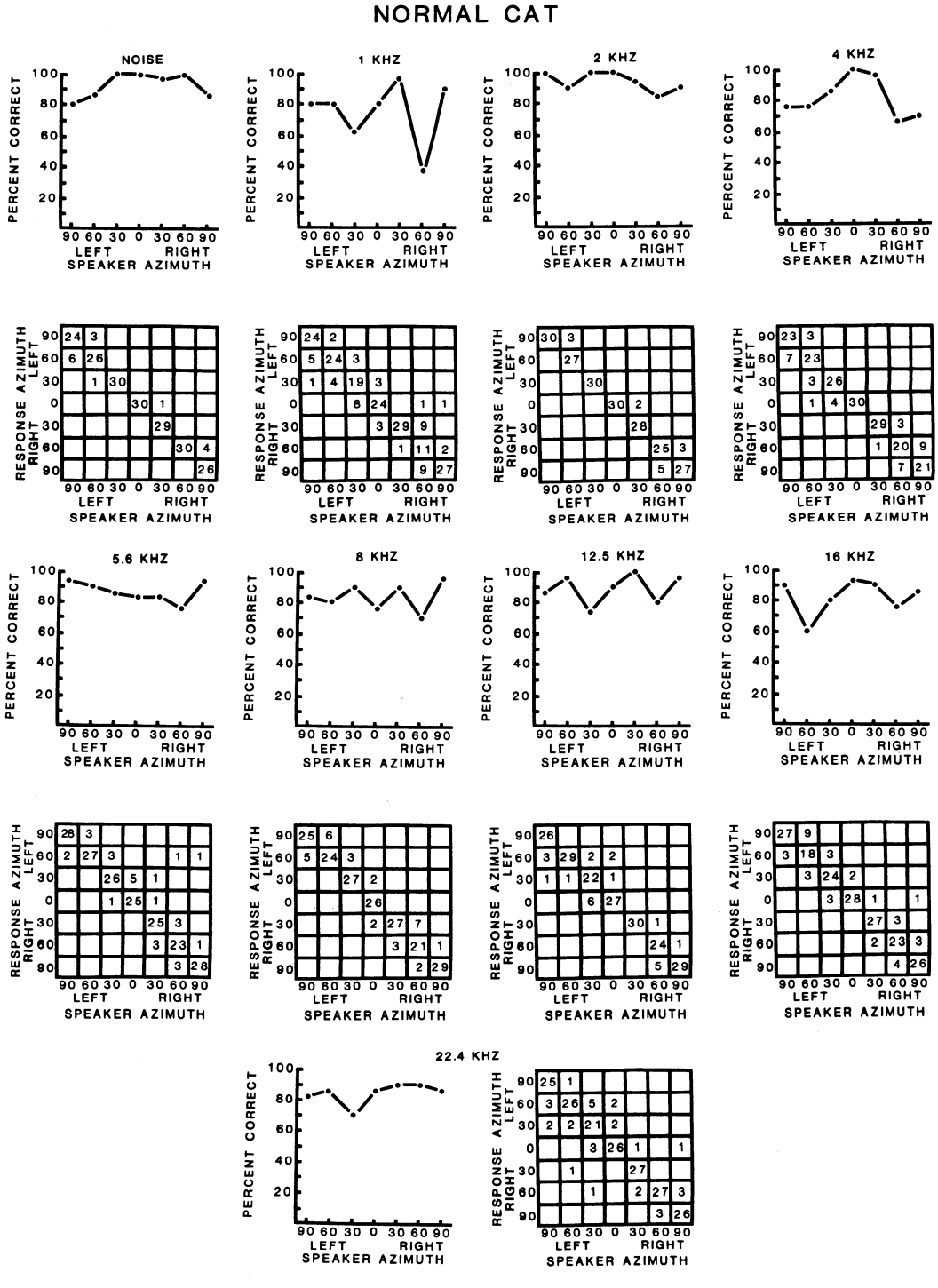


FIG. 3. Average performance (last 5 test days, 30 trials per point) of 1 normal cat for each speaker location using noise and 8 pure tones (as indicated above each plot). Data matrix adjacent to each plot indicates both number and location of correct and incorrect responses. Note differences in performance at different speaker locations for a particular test sound. In data matrices, note that most errors are at locations adjacent to correct response location.

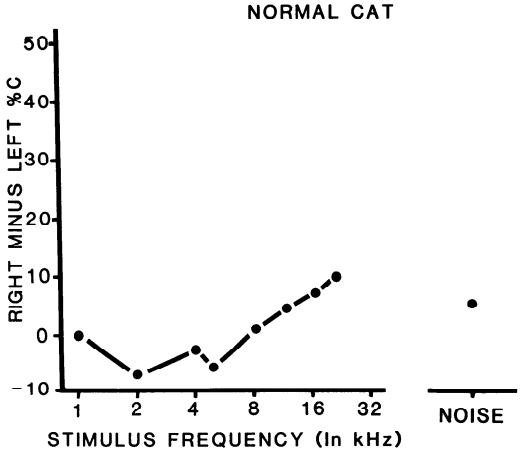


FIG. 4. Differences in sound-localization performance between left and right sound fields of a normal cat for noise and pure tones. Plot was obtained by subtracting average performance over last 5 test days for left-field locations from right-field locations (180 trials per point). Note that differences in performance between left and right field never exceeded 10%. This analysis will be used in subsequent cases to summarize consequences of restricted cortical lesions.

(e.g., see Ref. 56) will be described in detail, to allow a direct comparison between the sound-localization ability of a typical normal cat (illustrated in Figs. 2–4) with that of a typical cat with an AI strip lesion.

Figure 6 shows the results from *cat C8062,* in which a lesion made parallel to the isofrequency axis in AI destroyed the cortical representation of about an octave band of frequency (see Fig. 8 for a reconstruction of lesion). Behavioral results are shown for localization of 40-ms noise and tone pips. There, performance over the 10 daily sessions are shown separately for the field ipsilateral and contralateral to the cortical lesion (18 trials per point). The most striking difference in the behavior of this cat compared to normal cats was the consistently poor performance in the hemifield contralateral to the cortical lesion at 5.6, 8, and 11.2 kHz. Performance in the hemifield ipsilateral to the lesion at these same frequencies was high and similar to that of normal cats. For noise

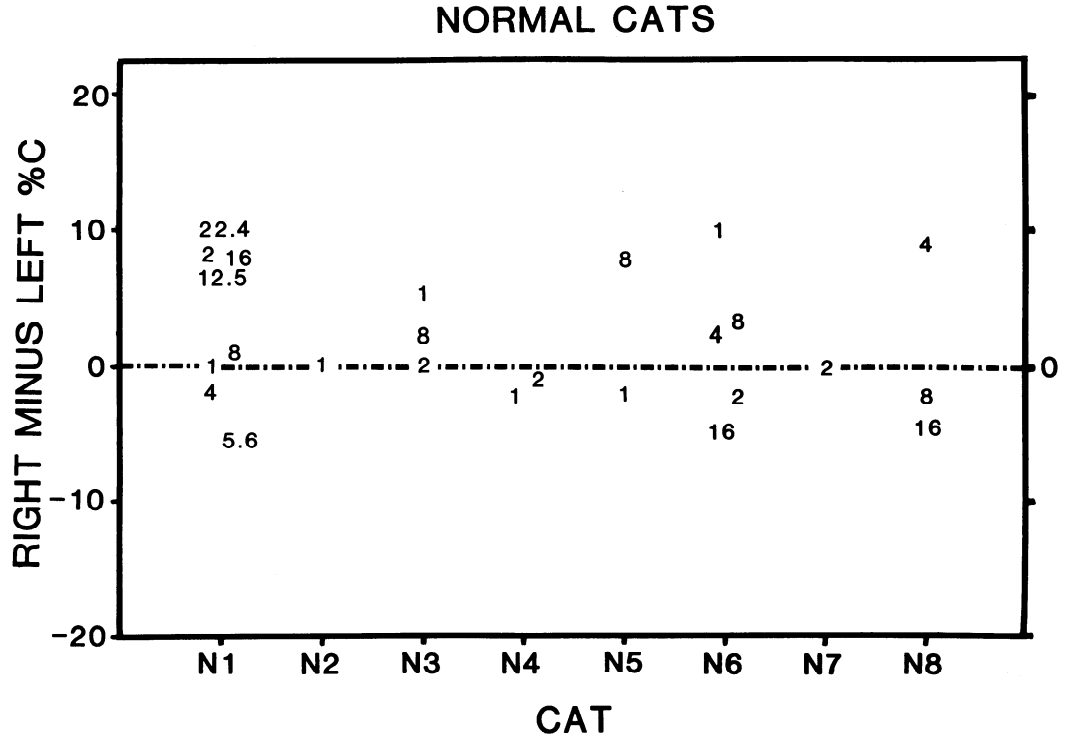


FIG. 5. Difference in sound-localization performance between left and right fields of 8 normal cats. This plot was obtained by subtracting average performance over last 5 test days for left-field locations from that for right-field locations (180 trials per point). Numbers are test frequencies in kHz. N1 is same case illustrated in Figs. 2–4. Note that differences in average left-right performance never exceeded about 10%.

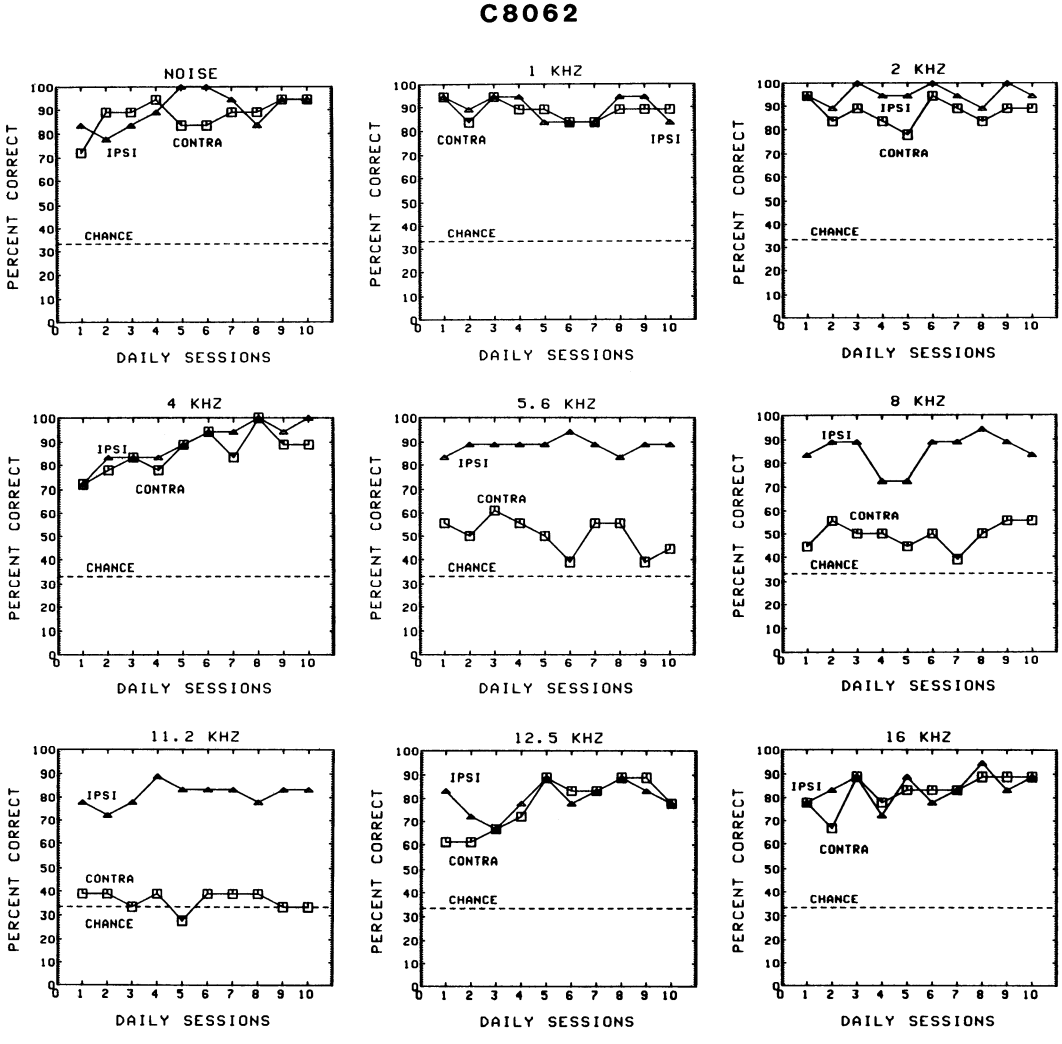


FIG. 6. Behavioral performance of a cat with a narrow strip lesion paralleling isofrequency axis in AI. Performance in localization of brief noise and tonal stimuli are shown for 10 consecutive daily sessions. Frequency of tonal stimuli is indicated at top of each plot.

bursts and for tone pips at 1, 2, 4, and 16 kHz, performance in both the ipsilateral and contralateral field was high and indistinguishable from that seen in studies in normal cats. As in normal cats, this experimental case also shows slight performance variations over the 10 daily test sessions, as well as a progressive and simultaneous improvement in performance for both hemifields over the 10 test days (e.g., 4 kHz). The behavioral results at 12.5 kHz are interesting, as on the first test session performance in the contralateral hemifield was 22.2% below performance in the ipsilateral hemifield. However, by the 3rd test day performance in the two hemifields was similar and subsequently improved. On the one hand, the initial poor contralateral hemifield performance at 12.5 kHz could represent a transient contralateral hemifield deficit. On the other hand, testing at 12.5 kHz was initiated immediately following the completion of testing at 5.6 kHz. Since at 5.6 kHz this cat exhibited a consistent contralateral deficit, it is possible that the initial poor performance at 12.5 kHz merely reflects position habits and strategies adopted by this cat in response to the deficit at 5.6 kHz. Given this example of a progressive improvement in contralateral hemifield performance (additional instances were recorded in other

cases), only behavioral results from the last 5 days of testing with a particular stimulus condition were used for further analysis.

Figure 7 shows the average performance (last 5 test days; 30 trials per point) for *cat C8062* with a right AI strip lesion paralleling

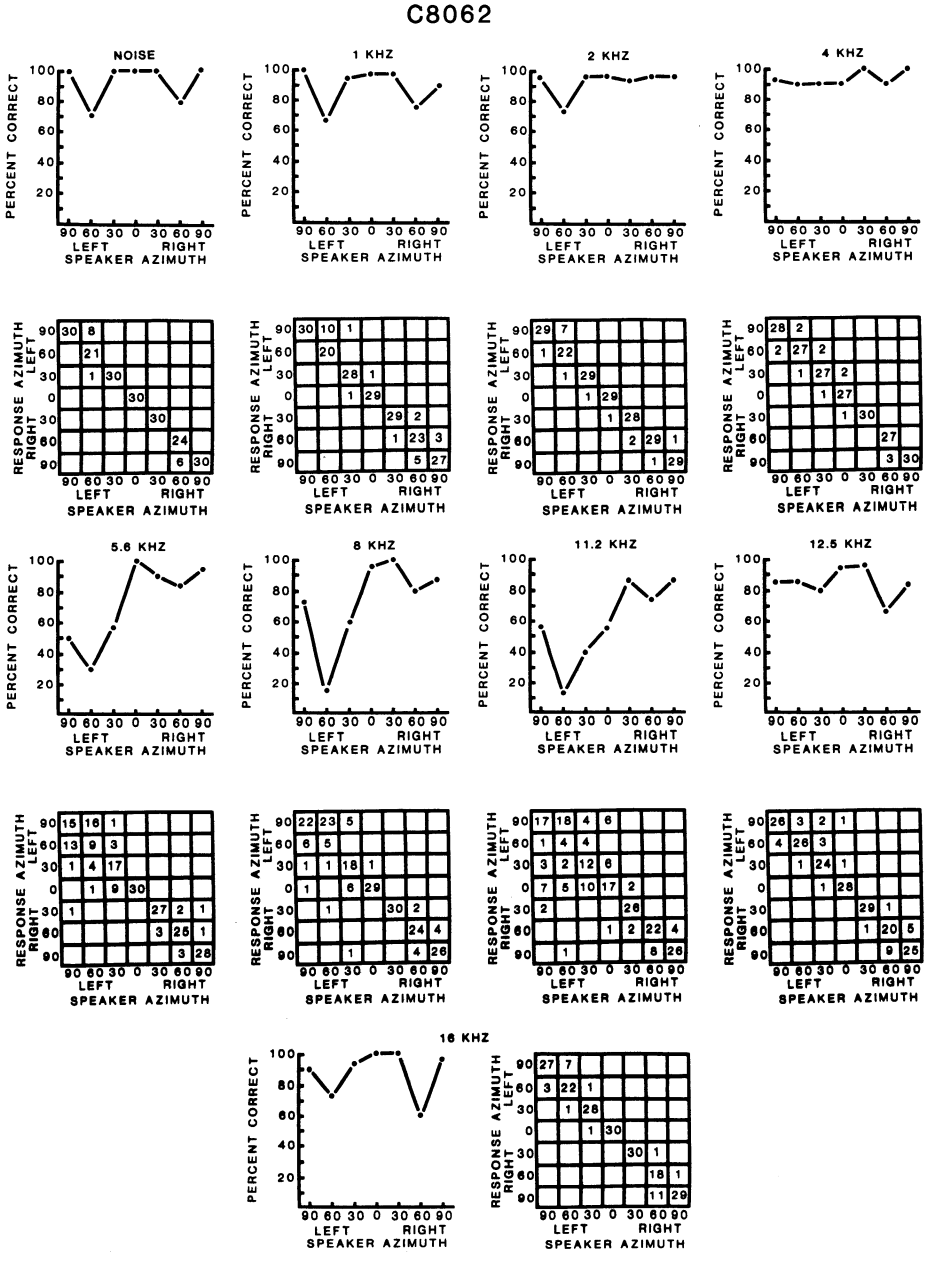


FIG. 7. Average performance (last 5 test days, 30 trials per point) for *cat 8062* with a right AI frequency-band lesion, for each speaker location. A data matrix indicating both number and location of correct and incorrect responses is shown for each stimulus condition. Note that for noise and for all frequencies except 5.6, 8, and 11.2 kHz, the variability in performance across speaker locations for a particular test sound was similar to that seen in normal cats (see Fig. 3). For 5.6, 8, and 11.2 kHz, the performance at speaker locations to the cat's left side (contralateral to AI cortical lesion) was poor. For these 3 frequencies, there was a consistent dip in performance at 60° contralateral position. Performance differences at 3 contralateral speaker locations are probably consequent from a behavioral strategy rather than a location-specific deficit. However, this behavioral test cannot distinguish between these 2 possibilities.

the isofrequency axis, for each speaker position for noise bursts and for tone pips at each of eight frequencies. A data matrix indicating both the number and location of correct and incorrect responses is shown adjacent to each plot. The behavioral performance of this cat was normal for noise stimuli and for tonal stimuli at 1, 2, 4, 12.5, and 16 kHz. At these frequencies, performance in both the ipsilateral and contralateral hemifield was high. When an error was made (as in the normal case) it was always to an adjacent speaker location, and when given a second chance on the subsequent trial the cat almost always made the correct response. Average performance as a function of speaker location varied slightly.

However, unlike normal cats, this cat exhibited a profound contralateral sound-localization deficit at 5.6, 8, and 11.2 kHz. For these three frequencies, there was a consistent dip in performance at the 60° contralateral position. Performance differences at the three contralateral speaker locations are probably consequent from a behavioral strategy rather than a location-specific deficit. However, this behavioral test cannot distinguish between these two possibilities. Performance at the center speaker was normal at 5.6 and 8 kHz, but not at 11.2 kHz. At these three frequencies, many more errors were made when the sound source was located in the hemifield contralateral to the cortical lesion than when the sound source was located in the ipsilateral hemifield. Another difference was a significantly (χ-square = 6.853; df = 1; $p < 0.0089$) higher number of errors made to locations not adjacent to the correct location. For example, in 270 trials per hemifield at 5.6, 8, and 11.2 kHz, 23.8% of errors in the contralateral hemifield were to nonadjacent positions; in the ipsilateral hemifield, only 2.8% of errors were to nonadjacent positions. In all experimental cases, there was a significantly higher than normal percentage of errors to nonadjacent locations in the hemifield contralateral to the cortical lesion, for all frequencies at which a contralateral localization deficit was apparent. It is also important to note that unlike normal cats (or at frequencies represented in AI cortical zones outside the lesion, or at all frequencies in the hemifield ipsilateral to the cortical lesion) repetitive errors were common for frequencies at which a contralateral hemifield deficit was apparent.

A summary of the results from *cat C8062* is shown in Fig. 8. A lateral view of the right cortex in this adult cat shows the approximate location of AI (stippled area) and the induced cortical lesion (black area). The approximate locations of other auditory cortical fields are indicated. A reconstruction of an auditory cortical mapping experiment preceding the ablation is shown in *B*. There, dots represent electrode-penetration sites. Adjacent numbers are best frequencies in kHz defined for neurons recorded at those sites. The dashed line defines the border of the subsequent cortical lesion. Observed cortical landmarks are also indicated. The best frequency of neurons in penetrations in AI as a function of distance across the cortical surface in the frequency-representational dimension (i.e., orthogonal to the isofrequency axis), defined 16 mo after the induction of the lesion, is illustrated in Fig. 8*C*. The approximate frequency band over which a profound sound-localization deficit was observed is indicated by the heavy line segment of the curve fitting these post-behavioral data. The cortex was silent in the zone of the lesion in this concluding mapping experiment, except for two penetrations along the low-frequency edge of the cortical lesion. No neurons were encountered with best frequencies at around 3 kHz. This appears as a gap in data points for frequencies outside the cortical lesion. Due to dural adhesion, surgical exposure of cortex was difficult. Local cortical damage occurred as a result of this postlesion postbehavior mapping experiment in a small region caudal to the cortical lesion. This region was intentionally avoided in this mapping experiment and thus accounts for this gap.

The differences in sound-localization performance for sound sources in the field ipsilateral compared with those contralateral to the AI lesion are shown in Fig. 8*D*. This plot was obtained by subtracting the average performance over the last 5 test days for contralateral field locations from ipsilateral field locations (180 trials per point) for each test sound. This plot indicates that this cat had a profound contralateral hemifield sound-localization deficit for 5.6, 8, and 11.2 kHz. From the results of the mapping experiments, there was a close correspondence between

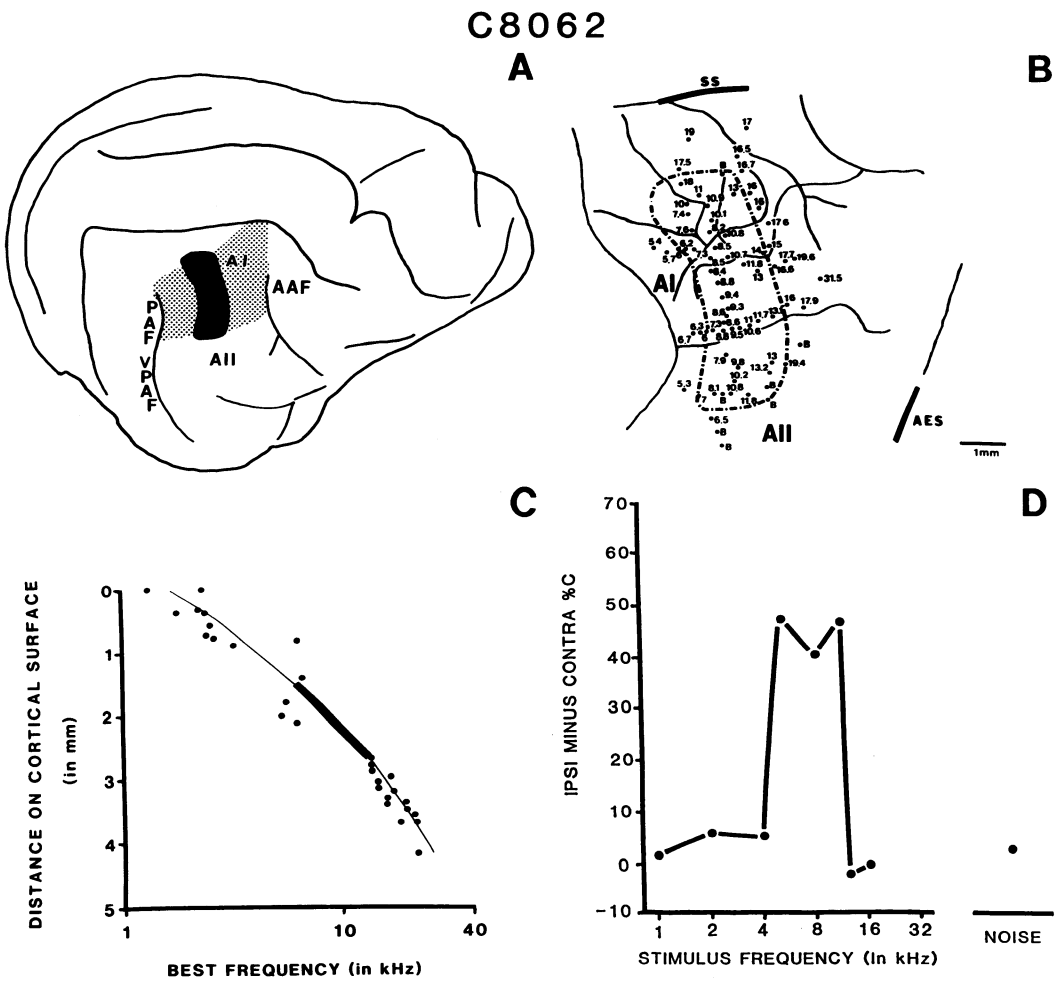


FIG. 8. A profound frequency-specific contralateral hemifield sound-localization deficit results from a lesion destroying the representation of those frequencies in AI. *A*: lateral view of hemisphere in an adult cat, illustrating approximate location of AI (stippled area) and a cortical lesion extending across mediolateral (isofrequency) axis of AI (black area). Approximate locations of other auditory cortical fields are indicated. AAF, anterior auditory field; PAF, posterior auditory field; VPAF, ventral posterior auditory field; AII. *B*: reconstruction of an auditory cortical mapping experiment preceding ablation. Dots represent electrode penetration sites. Adjacent numbers are best frequencies in kHz defined for neurons recorded at those sites. "B" adjacent to dots refers to units for which best frequencies were difficult to define since units appeared to be broadly tuned. Dashed line defines border of subsequent cortical lesion. Cortical landmarks are also indicated: SS, supersylvian sulcus; PES, posterior ectosylvian sulcus; AES, anterior ectosylvian sulcus. *C*: best frequency of neurons in penetrations into AI as a function of distance across cortical surface in frequency representational dimension, defined 16 mo after induction of lesion shown in *A*. Frequencies for which a profound sound-localization deficit were observed are indicated by heavy line segment of curve fitting the data. In zone of lesion spanning these frequencies in this concluding mapping experiment, cortex was silent, except for 2 penetrations near low-frequency edge of cortical lesion. *D*: differences in sound-localization performance for sound sources in field ipsilateral compared with those contralateral to AI lesion. Plot was obtained by subtracting average performance over last 5 test days for contralateral field locations from ipsilateral field locations (180 trials per point) for each test sound. Overall contralateral field performance levels at deficit frequencies were at or near chance.

the frequencies formerly represented in the AI cortical lesion and the frequencies for which a sound-localization deficit was observed. Based on the initial mapping experiment and the location of the lesion (Fig. 8*B*), the sound-localization deficit might be expected to extend up to 13–14 kHz. The postbehavior mapping experiment revealed

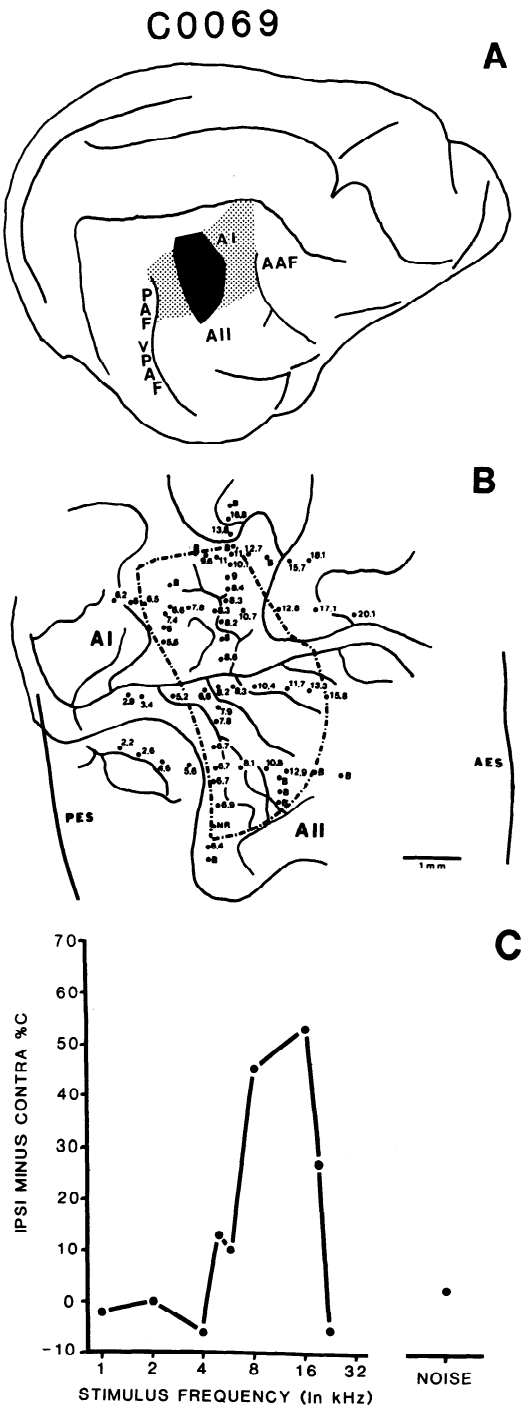
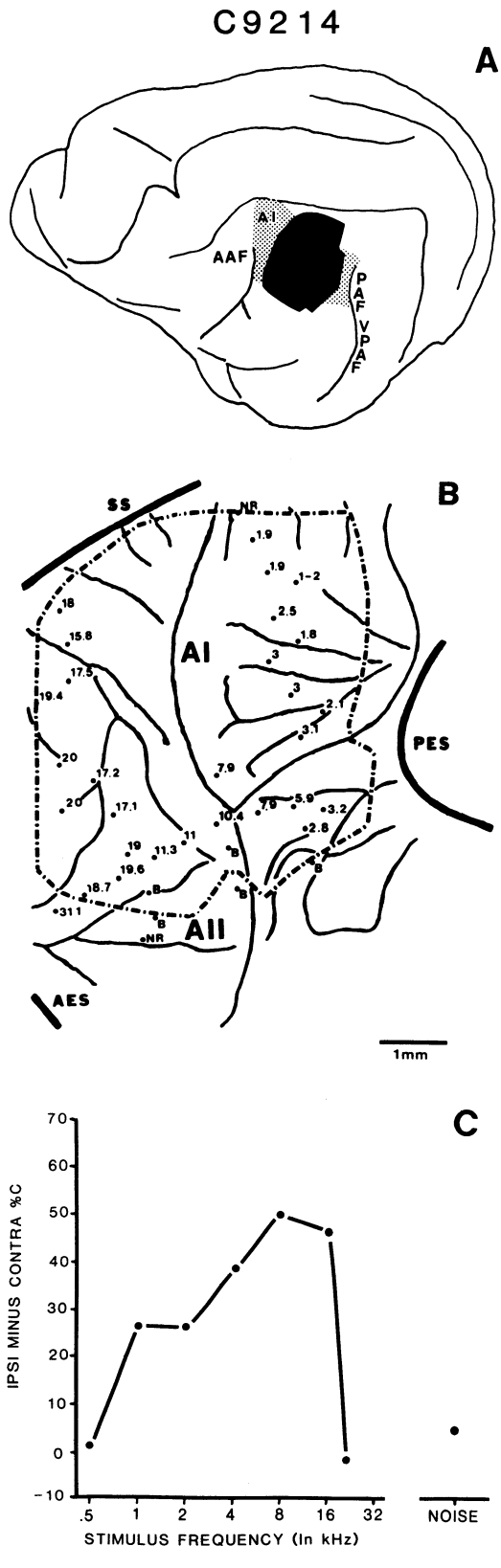


FIG. 9. A second case illustrating a profound frequency-specific contralateral-hemifield sound-localization deficit resulting from a restricted lesion parallel to isofrequency representational axis of AI. *A*: lateral view of right cortex in an adult cat, showing approximate location of AI (stippled area) and a cortical lesion (black area). Approximate locations of other auditory cortical fields are indicated. *B*: reconstruction of an auditory cortical mapping experiment preceding ablation. There, dots represent electrode penetration sites. Adjacent numbers are best frequencies in kHz defined for neurons recorded at those sites. "B" refers to broadly tuned. Dashed line defines border of subsequent cortical lesion. *C*: differences in sound-localization performance for sound sources in field ipsilateral compared with those contralateral to AI lesion. Plot was obtained by subtracting average performance over last 5 test days for contralateral field locations from ipsilateral field locations (180 trials per point) for each test sound. Note rough correspondence between frequency representational region of the AI cortical lesion and frequencies at which large ipsilateral vs. contralateral performance differences were observed.

units with best frequencies as low as 13.2 kHz located along the high-frequency lesion border. Postlesion sound-localization performance at 12.5 kHz was apparently normal. These results indicate that there are small discrepancies in the lesion border based on the initial mapping experiment and the postlesion postbehavior mapping experiment. Performance at 12.5 kHz was normal, in spite of the fact that no units with best frequencies between 12–13 kHz were observed in the postlesion mapping experiment. At least two possibilities could account for this discrepancy. First, it is possible that units with best frequencies between 12–13 kHz did exist but due to a sampling bias were not identified. Second, it is also possible that the normal performance seen at 12.5 kHz depended on units with slightly higher best frequencies (i.e., greater than 13 kHz). It is important to note that for noise and frequencies represented on either side of the cortical lesion, no sound-localization deficit was observed.

A second cat (*C0069*) with a similar lesion paralleling the isofrequency representational axis in AI is shown in Fig. 9. In this study, a postbehavior cortical mapping experiment was attempted but was aborted due to the severity of dural adhesions and a concern for the preservation of cortical architecture for subsequent histological verification of the locus and extent of the cortical lesion. The behavioral results from this case indicate that for noise bursts and for brief tone pips at 1, 2, 4, 22.4, and possibly 5.6 and 6.3 kHz there was no apparent sound-localization deficit. However, at 8, 16, and 18 kHz, there was a contralateral hemifield sound-localization deficit. The deficit was especially pronounced at 8 and 16 kHz. At 18 kHz, the contralateral hemifield deficit was less severe.



Sixteen and 18 kHz were represented near the high-frequency border of the introduced cortical lesion.

This case is interesting on several grounds. First, as in the previously described cat, this case showed a rough correspondence between the frequencies formerly represented in the AI cortical lesion and the extent of the subsequent contralateral hemifield sound-localization deficit. Second, it revealed a limitation of resolution in the ability to predict a sound-localization deficit based on the prebehavior mapping experiment. On the basis of the prelesion map, a greater deficit at 6.3 kHz might have been expected (the posterior edge of the lesion passed down the 5.8–6.0 kHz line), and no deficit at 18 kHz might be expected (the lesion passed across the 13–16 kHz line). Given these discrepancies and the lack of a postbehavior best-frequency map, the reasons for nearly normal performance at 6.3 kHz or clearly abnormal performance at 18 kHz remain moot. A similar ambiguity occurred in a case presented below (*C0057*) in which a postbehavior best-frequency map was obtained; in that case, the postbehavior map was more consistent with the identified behavioral deficit frequencies.

The behavioral results at 18 kHz in *cat C0069* are also interesting because they represent a relatively small partial octave step in test frequency from a profound deficit frequency (16 kHz) to a normal frequency (22.4 kHz). The behavioral results at 18 kHz indicate a contralateral deficit, which was roughly midway between the results at 16 kHz and those at 22.4 kHz.

A third case (*C9214*) with a relatively large lesion in AI is shown in Fig. 10. The cortical lesion in this cat included the frequency representation in AI extending from ~1 kHz

FIG. 10. A profound frequency-specific contralateral-hemifield sound-localization deficit results from a large lesion in AI. *A*: estimated location of AI (stippled area) and cortical lesion (black area). Approximate locations of other auditory cortical fields are indicated. *B*: reconstruction of an auditory cortical mapping experiment preceding the ablation. Best frequencies, penetration sites and border of cortical lesion (dashed line) are indicated. "B" refers to broadly tuned. "NR" adjacent to dot indicates no driven response observed. Note that this lesion includes AI frequency representation for frequencies between roughly 1 and 18–22 kHz. *C*: differences in sound-localization performance between locations ipsilateral and locations contralateral to the cortical lesion (180 trials per point).

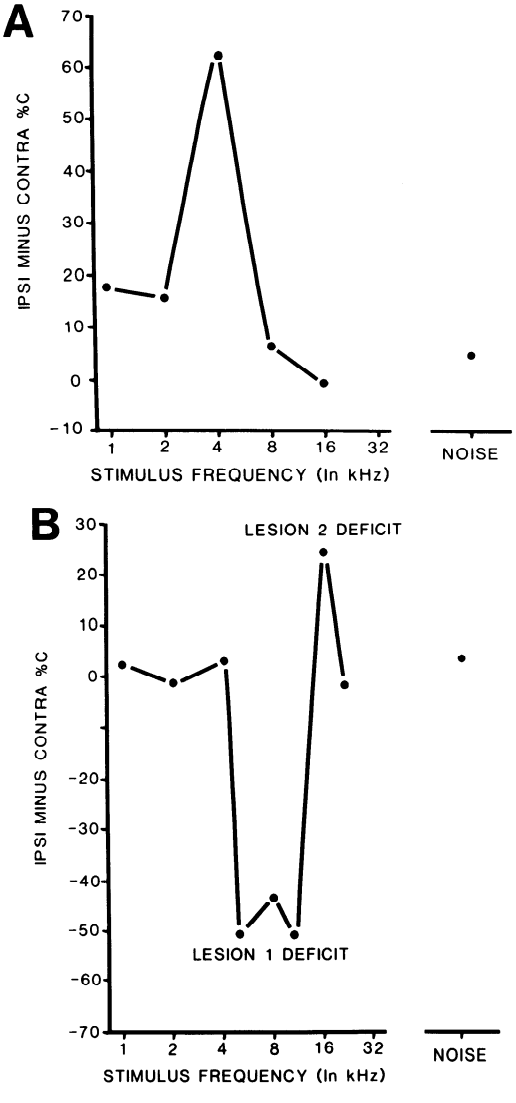


FIG. 11. *A*: Summary of behavioral results from *cat C0057*. Frequency representation in AI was completely destroyed from roughly 3.2–4.7 kHz. A profound sound-localization deficit limited to hemifield contralateral to lesion was observed at 4 kHz. *B*: summary of behavioral results from *cat C8062* after a second AI lesion in cortex contralateral to first lesion. Sound-localization deficit labeled "lesion 1 deficit" replicates behavioral deficit recorded after first lesion (see Fig. 8). Second cortical lesion extended across representation of adjacent octaves in upper and lower halves of AI. That is, in upper half of AI, a lower-frequency band was destroyed, while in lower half of AI, a higher-frequency band was destroyed. A deficit in field contralateral to this second lesion was recorded only at 16 kHz (labeled "lesion 2 deficit"), i.e., in narrow representational band in common for these 2 continuous lesions. Both *A* and *B* show differences in sound-localization performance between location ipsilateral and locations contralateral to cortical lesion (180 trials per point). For *B*, ipsilateral and contralateral refer to second lesion.

to 16 kHz. The postlesion behavioral results reveal a contralateral sound-localization deficit for frequencies all across this range. Contrariwise, normal sound-localization performance was observed for brief 500 Hz and 22.4 kHz tone pips and for noise bursts. Note that even with large subtotal AI lesions (this lesion destroyed ~70% of AI), apparently normal sound-localization ability is recorded under some stimulus conditions (e.g., with clicks, noises, or tones of appropriate frequency). The contralateral hemifield sound-localization deficits recorded in this case again correlated roughly with the AI frequency representational zone destroyed by the cortical lesion.

In a fourth experiment (*C0057*), there was good correspondence between the postlesion electrophysiological recordings and the frequencies at which profound sound-localization deficits were recorded (Fig. 11*A*). At the time of induction of the lesion, it was designed to involve a narrower band, but the silent zone was subsequently found to extend several kHz lower than originally estimated. The postlesion map indicated that the cortical lesion completely destroyed the AI frequency representation roughly from 3.2 to 4.7 kHz. In the zone of representation of slightly higher frequencies, there were patches where apparently normal driven responses were observed and patches where no driven response could be seen. That is, the estimated lesion at these higher frequencies did not extend fully across the isofrequency axes of AI. A profound sound-localization deficit limited to the hemifield contralateral to the lesion was observed only at 4 kHz.

In a fifth experiment (*C8062*), an introduced lesion extended across the representation of adjacent octaves in the upper and lower halves of AI (Fig. 11*B*). That is, in the upper half of AI a lower-frequency band was destroyed, whereas in the lower half of AI a higher-frequency band was destroyed. A contralateral deficit was recorded only at 16 kHz, i.e., in the narrow representational band in common for these two continuous lesions. No evidence of a sound-localization deficit was seen for frequencies within each of the adjacent half-frequency band lesions. The cat used for this experiment had already undergone previous extensive behavioral testing after an AI lesion (see Fig. 8). The lesion described above was introduced in the cortex

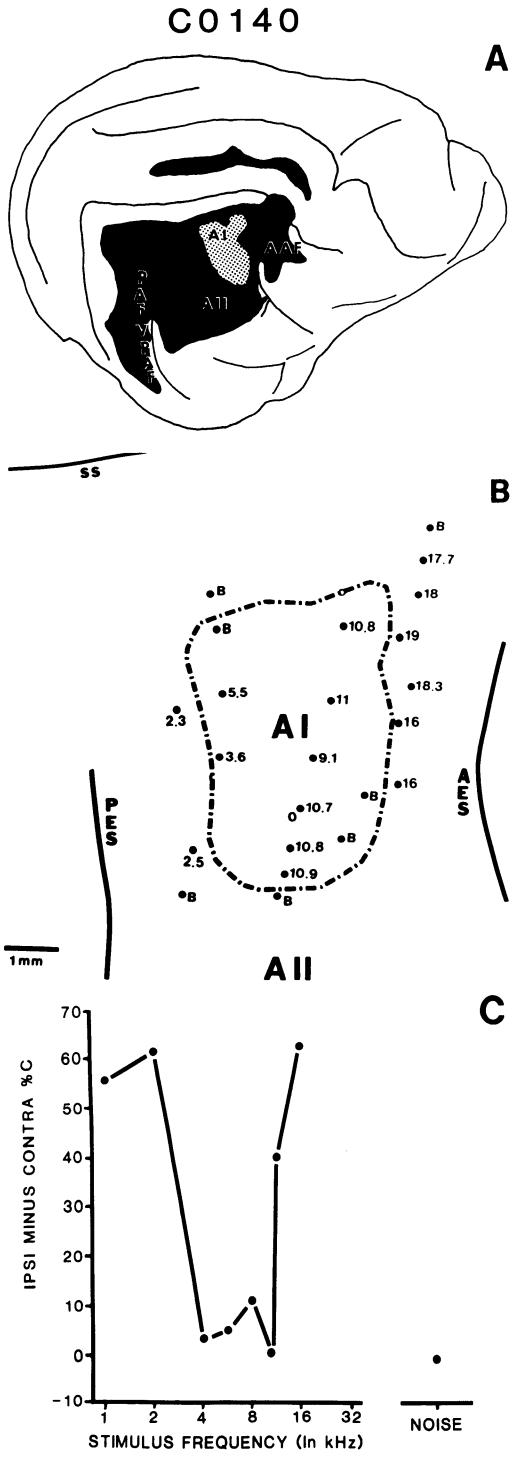


FIG. 12. This case illustrates the effect on sound-localization performance of a lesion of most of auditory cortex, which included both the tonotopic and nontonotopic auditory cortical fields, except for a narrow frequency-band representational sector within AI. *A*: lateral view of right hemisphere of an adult cat, showing cortical lesion (black) and spared region of AI (stippled area). *B*: reconstruction of a cortical mapping experimental preceding ablation. Penetration sites and best frequencies are indicated. "B" refers to broadly tuned. This lesion spared isofrequency representation in AI for frequencies extending from roughly 3.5 to 15–16 kHz. *C*: differences in sound-localization performance for sound sources in field ipsilateral vs. those in field contralateral to cortical lesion (180 trials per point). Note that for noise and frequencies represented in surviving AI cortical zone, no deficit was seen, but for higher and lower frequencies (outside surviving zone) a profound contralateral sound-localization deficit was apparent.

opposite the first lesion. The behavioral results obtained after the second cortical lesion also replicated the results shown in Fig. 8.

As documented below, deficits recorded with lesions extending across the full width of AI are permanent. From these behavioral results, it is apparent that AI is necessary for normal sound-localization performance. The contribution of AI to sound-localization behavior would appear to occur within relatively selective frequency channels. The question arises as to whether AI alone is sufficient for normal sound-localization behavior or is merely a "stepping stone" to other auditory cortical areas where further sound-localization processing occurs.

Consequences of a lesion sparing a narrow strip in AI

To address this issue, another case was prepared in which an attempt was made to unilaterally destroy all auditory cortical fields except a narrow sector of isofrequency representation within AI. The results of this study (*C0140*) are presented in Fig. 12. This case illustrates the affect on sound-localization performance of a lesion of most of auditory cortex, including both the tonotopic and nontonotopic auditory cortical fields but preserving a narrow frequency-band representational zone within AI. It should be noted that portions of the ventral posterior auditory field (VPAF) and the most ventral portion of AII remained intact. Also, cortical damage occurred medial to the suprasylvian sulcus. A reconstruction of a cortical mapping experiment preceding the ablation is shown in Fig. 12*B*. Penetration sites and best frequencies encountered at those sites are indicated. This lesion spared a frequency-band representational strip in AI for frequencies between ~4–11 kHz. The differences in sound-localization performance for sound sources in the

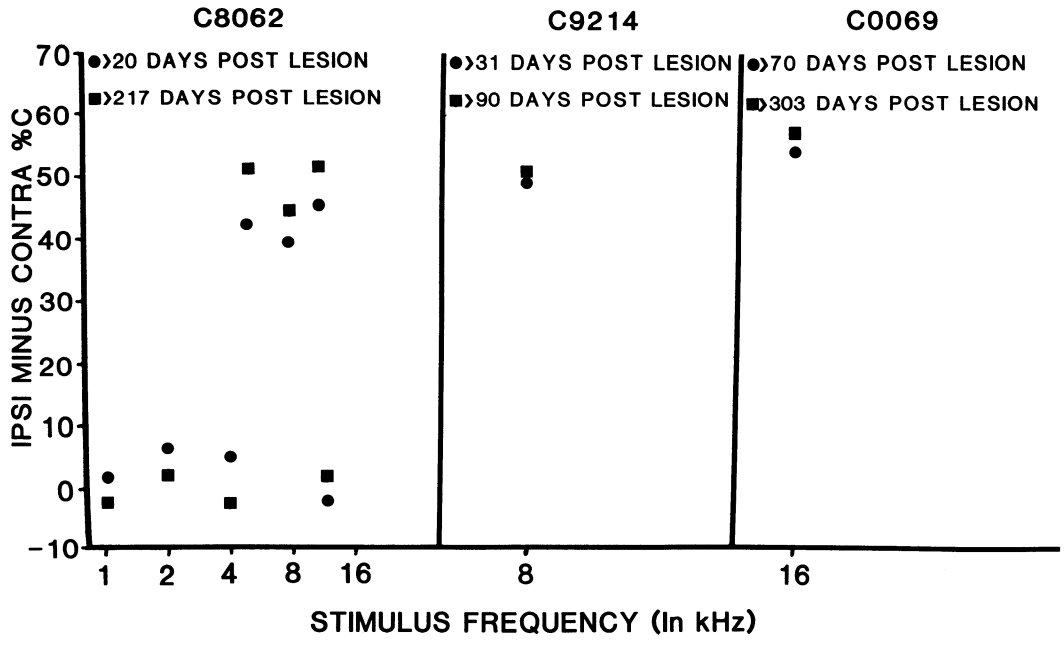


FIG. 13. Behavioral deficit observed after unilateral lesions of restricted isofrequency sectors of AI is permanent. For each of 3 illustrated cases, a complete frequency series of postablation sound-localization performance measures was obtained (dots). Selected frequencies were subsequently tested at later times (squares). There were 180 trials per point in both studies.

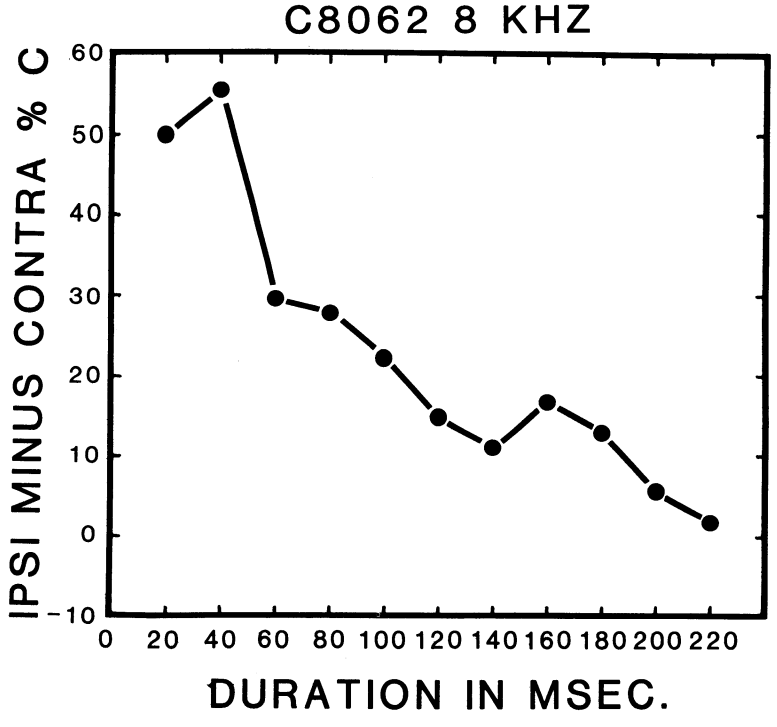


FIG. 14. Effect of tone duration on behavioral deficit for case shown in Fig. 8 at 8 kHz and at same intensity level used for Fig. 8 data. Data for each duration was obtained over 5 daily sessions (180 trials per point) and depicts difference in average sound-localization performance between locations in field ipsilateral vs. those contralateral to cortical lesion.

field ipsilateral vs. those in the field contralateral to this cortical lesion (180 trials per point) are shown in Fig. 12*C*. There, for noise and frequencies represented in the surviving AI cortical zone (i.e., for 4, 6.3, 8, and 11.2 kHz tone pips), no deficit was seen. For frequencies formerly represented outside this surviving zone (i.e., at 1, 2, 12.5, and 16 kHz), a profound contralateral sound-localization deficit was observed. Given the limitations of the cortical lesion (i.e., there was some possible significant sparing of parts of VPAF and AII), the approximate correspondence between the frequency representation in the surviving sector of AI and the normal localization performance at these frequencies strongly suggests that AI and its subcortical connections are sufficient to support normal azimuthal sound-localization behavior.

Ancillary behavioral tests

Several other important questions arise in considering the nature of the behavioral sound-localization deficits that occur as a result of frequency-band representation lesions in primary auditory cortex. First, were the sound-localization deficits recorded after AI strip lesions really permanent? Second, what was the effect of stimulus duration on the sound-localization deficits? Finally, what was the effect of stimulus intensity on the sound-localization deficits? Preliminary answers to these questions are presented below.

The permanent nature of the behavioral deficit seen after unilateral lesions of restricted frequency-band sectors of AI is illustrated in Fig. 13. For each of these three illustrated cases, a complete frequency series of postab-

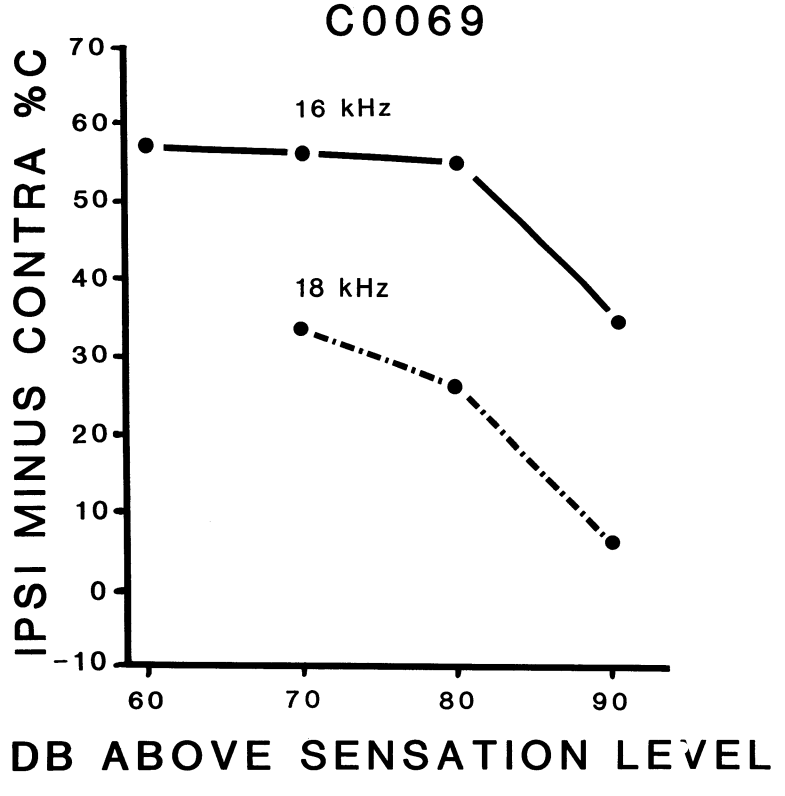


FIG. 15. Effect of intensity level on behavioral performance for same case shown in Fig. 8 at 16 and 18 kHz (tone duration 40 ms). Data for each intensity level was obtained over 5 daily sessions (180 trials per point) and depicts difference in average sound-localization performance between locations in field ipsilateral vs. contralateral to cortical lesion. Note that for 16 kHz, frequency of greater deficit, performance improved markedly only at highest intensity. Note also that for 18 kHz, a frequency that was represented closer to lesion border than 16 kHz, deficit was less marked than that seen at 16 kHz at comparable sensation levels. For 18 kHz at 90 dB SL, there was little difference in performance between 2 hemifields. Decrease in difference between ipsilateral minus contralateral performance was due to improved performance levels for contralateral locations.

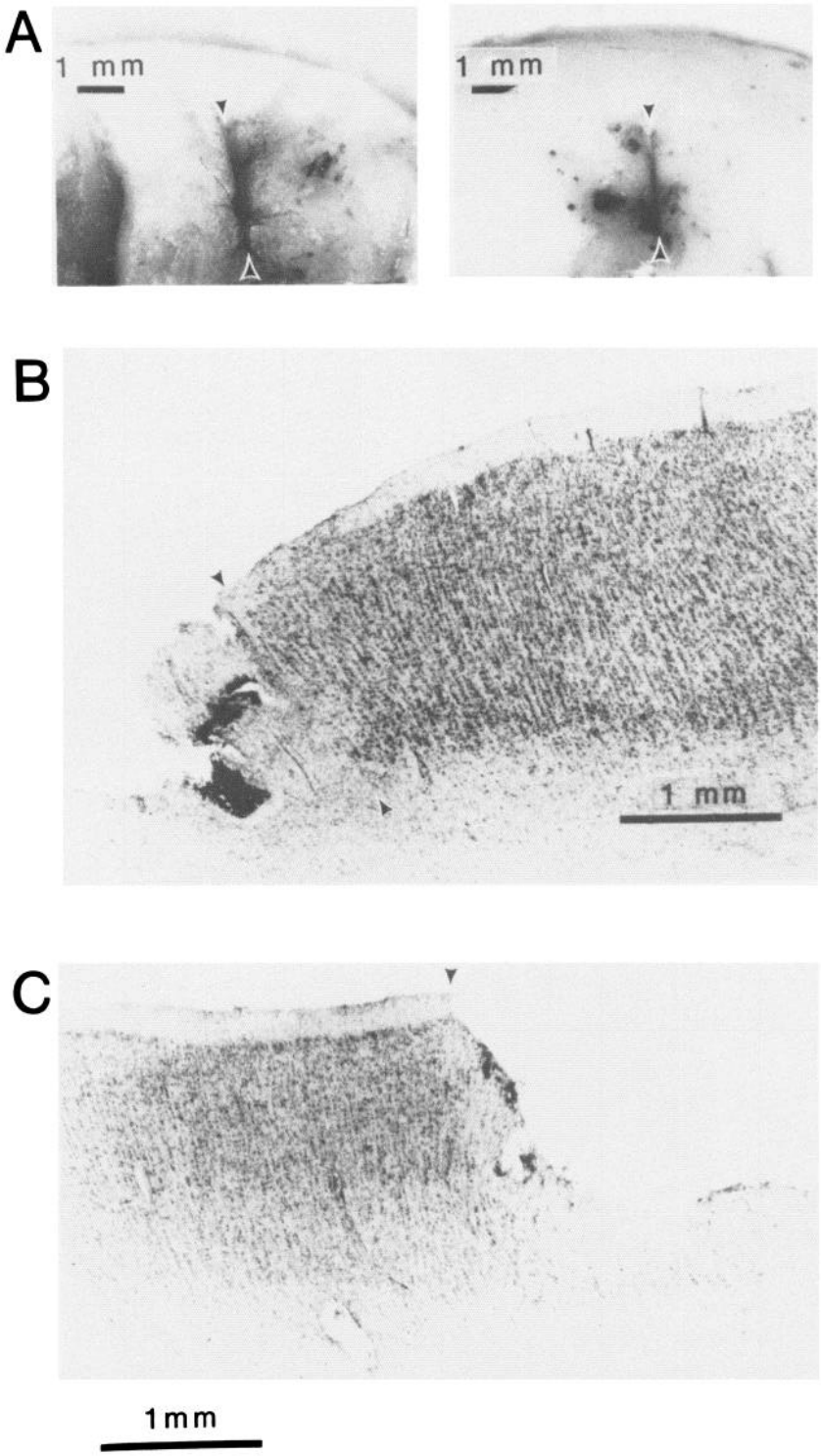


FIG. 16. Features of electrocautery-induced lesions. *A*: typical surface views of auditory cortex of cats with frequency-band strip lesions restricted to AI. Positions of false sulci, recorded in every such case, are marked by arrows. Sites of hemorrhage principally due to removal of adherent dura in postbehavior physiological mapping study can also be seen. Case illustrated at right (*cat C8062*) represents 1 of 2 most extreme cases of dural adhesion.

lation sound-localization performance measures was obtained. Subsequently, some selected frequencies were again tested. Even though the sound-localization tests were repeated in some instances many months after the initial test, the differences between sound-localization performance in the hemifield ipsilateral vs. the hemifield contralateral to the cortical lesion remain virtually unchanged. The only observed change in performance was a slight improvement in localization performance for sound sources in the hemifield ipsilateral to the cortical lesion. This is evident in Fig. 13 by the slight increase on the repeated tests in the ipsilateral minus contralateral percent correct values over the initial test.

The effect of tone duration on the behavioral deficit seen in *cat C8062* (Fig. 8) at 8 kHz derived at the same intensity level used for Fig. 8 data is shown in Fig. 14. The data for each duration was obtained over five daily sessions (180 trials per point) and depicts the differences in average sound-localization performance between locations in the field ipsilateral and locations contralateral to the cortical lesion. There was a progressive decrease in the difference between ipsilateral vs. contralateral performance for durations longer than 40 ms, from a near-chance level to the ipsilateral normal performance level. This occurred as a direct result of progressive improvements in performance for contralateral locations. It is probable that this improvement in localization ability in the hemifield contralateral to the cortical lesion as a function of tone duration was dependent on orienting movements, which have a latency of ~40 ms (81). Observation of this cat's behavior on the video monitor indicated that orientation movements of the head were made with the pinna held in a fixed position. Accurate measurements of the relation of head and pinna movement relative to the speaker location during presentation of long-duration sounds were not possible with the apparatus used in these studies.

The effect of the intensity level on the sound-localization deficit for *cat C0069* (Fig. 9) at 16 and 18 kHz (tone duration 40 ms) is illustrated in Fig. 15. The data for each intensity level was obtained over five daily sessions (180 trials per point) and depicts the difference in average sound-localization performance between locations in the field ipsilateral and contralateral to the cortical lesion. Intensity levels were randomized daily. At 16 kHz, a frequency at which a profound deficit was recorded, performance improved significantly only at the highest intensity. At 18 kHz, a frequency that was represented closer to the lesion border than 16 kHz, the deficit was less than that seen for 16 kHz at comparable sensation levels, and performance at the highest-tested intensity level indicated no contralateral deficit. The spectral characteristics of the speakers were measured at each intensity level in order to determine if a change in spectral content could account for the observed changes in performance. As expected, the greatest harmonic distortion was observed at the highest stimulus level for both 16 and 18 kHz tones. However, at 90 dB sensation level (SL), the relative harmonic distortion at 16 kHz was ~30 dB above that seen at 18 kHz. Furthermore, harmonic distortion increased more rapidly with increasing intensity levels at 16 kHz than it did at 18 kHz. These results suggest that increases in harmonic distortion associated with increased intensity levels were not the basis for the improved performance.

Controls for potential stimulus artifacts

One of the major problems in any psychophysical experiment is the possibility that stimulus cues other than the one intentionally being varied provide a basis for the discriminatory response. In the present series of experiments, this reduces to two categories: *1*) nonacoustic artifacts and *2*) acoustic artifacts.

To control for these potential artifacts, the following types of trials were occasionally

Nonetheless, postbehavior maps were successfully derived in this cat. *B*: typical lesion border, illustrating usual sharp transition (arrows) from normal or nearly normal to aneuronal cortex. In many instances, false sulcus wall paralleled columnar axis of cortex, with normal or nearly normal neurons extending up to wall. In other instances, as illustrated here, a relatively thick aneuronal zone formed false sulcus wall. From *cat C0069*. *C*: in some cases, border of lesion was relatively abrupt for superficial cortical layers, but there was some neuronal survival in deep layers (especially, in layer 6). From *cat C9214*. In both *C* and *B*, sections were transverse, paralleling false sulci. Significant distances along floors of these sulci are shown. In all cases, either all neurons were destroyed in floor of these false sulci; or over some regions in some cases, only neurons within deep layers survived lesion.

presented to both normal and experimental cats: *1*) the wide-range attenuator was set to 110-dB attenuation; *2*) the voltage output of either the oscillator or the noise generator was reduced to 0 volts; *3*) input to the rise-fall gate was disconnected from the oscillator or noise generator and allowed to "float" or was grounded; and *4*) the rise-fall gate was set to 0 or 10 ms. In every configuration except for manipulation of the rise-fall gate, both normal and experimental cats failed to respond on presentation of a "trial." These results indicate that there were no usable nonacoustic or obvious acoustic artifacts. When the rise-fall gate was set to 0 ms (at which a broad-spectrum click was generated) there was an immediate improvement in performance that was most obvious for the experimental cats tested at a frequency at which there was a profound deficit. Performance of either the normal or experimental cats did not change when the rise-fall gate was set to 10 ms. The last potential acoustic artifact is the possibility that the cats were responding to the harmonics of the test frequency rather than the test frequency itself. Although it was not possible to entirely eliminate harmonic distortion, measurements of harmonic distortion for the test tones used in these experiments indicated that the harmonics were at least 50 dB below the test frequency (at ~70 dB SL).

Features of induced cortical lesions

The cortical sites of these very small lesions were marked on the cortical surface by the appearance of a narrow false sulcus centered in the lesion zone. This collapse of the cortical gray matter to form a narrow terminated slit was seen in every case except the massive multiple-field lesion recorded in *C0140*.

Examples are shown in Figure 16*A*. In most instances, there was a very sharp transition from normal-appearing cortex to completely reduced cortex on the margins of these false sulci. In some cases, the cortex retained its full thickness over a region bordering the false sulcus, but in a bordering zone there were few if any surviving neurons and glia had greatly proliferated. Again, the boundary of normal-appearing and aneuronal cortex was sharp (Fig. 16*B*). In several cases, there were zones of a sharply delimited lesion in which cell loss was total or near total through layer 4, whereas neurons in deeper layers (especially 6) partially or largely survived the lesion (Fig. 16*C*). Such zones were recorded, for example, in *cats C8062* and *C0069*.

From alternate Weil series, substantial demyelination and loss of myelinated fibers were seen in the auditory radiations. In demyelinated zones, some normal-appearing fibers appear with demyelinated fibers. The thalamus was examined in every cat. In the two largest lesions cases (*cats C0140* and *C9214*), retrograde cell loss was marked. In all cats with narrow frequency band-strip lesions, it was very difficult to identify a region of cell loss or retrograde cell degeneration, a difficulty likely compounded by the relatively long postlesion survival times in these cases. There were no observable signs of degenerated fibers in the region of primary auditory cortex surrounding the cortical lesions.

DISCUSSION

Cats with small unilateral cortical lesions destroying cortical zones representing limited bands of frequencies within AI could not localize brief tones at those frequencies within the hemifield contralateral to the lesion. Sound localization within the ipsilateral hemifield remained normal, as did localization of tones represented in spared AI sectors. In an additional cat, a unilateral cortical lesion destroyed most of auditory cortex but spared the representation of a narrow band of frequencies in AI. This cat had a profound azimuthal contralateral hemifield deficit for the localization of brief tones at all test frequencies except those whose representations were spared within this narrow strip of AI.

On the basis of these results, the following conclusions are made. *1*) Primary auditory cortex (AI) is essential for the normal localization of brief sounds. *2*) At the level of auditory cortex, AI is probably sufficient for normal binaural azimuthal sound-localization behavior as measured by these experiments. *3*) Only contralateral sound-field deficits result from unilateral cortical lesions (see also 43). *4*) Neuronal mechanisms subserving azimuthal sound localization operate by frequency channel at high levels in the central auditory nervous system.

Relation to previous studies

The role of auditory cortex in discriminations requiring localization of sound in space using modern psychophysical techniques were first described by Neff and his colleagues (63). The conclusion that primary auditory cortex (AI) is necessary for normal sound-localization ability is consistent with the conclusions of earlier studies on the contributions of various subdivisions of auditory cortex to sound-localization behavior. Strominger (78) and Wegener (87, 88) reported that bilateral ablations of AI in cats and monkeys produced permanent impairments in sound-localization abilities. In Strominger's cats with bilateral AI lesions, it was suggested that part of AI on at least one side may have been spared, accounting for the transient nature of the deficit observed in one cat. Cats with large bilateral ablations of any other auditory cortical areas that spared AI were unimpaired. The stimulus parameters and behavioral tests used in these studies were inappropriate for defining true binaural sound-localization abilities (see 43). Long-duration stimuli would allow for monaurally based localization involving scanning (Fig. 14), and the two-choice behavioral test defined "lateralization" or left-right discrimination deficits but not necessarily binaural localization deficits (43). It is nonetheless interesting that AI in a single hemisphere may be necessary and sufficient to support these other behaviors as well.

More recently, experiments using multiple speakers or modifications of the two-speaker test have revealed that large cortical lesions result in contralateral-hemifield, true-binaural sound-localization deficits in a variety of species (31, 39, 43, 47, 82). However, none have defined the cortical field(s) actually essential for this behavior. In this study, that requisite field in the cat is determined to be the primary auditory cortex, AI.

Observations on nature of deficit

If AI is indeed both necessary and sufficient for normal azimuthal sound-localization behavior, then several important questions arise. What specifically might AI contribute to sound-localization behavior? Are the behavioral deficits that ensue from AI lesions the result of the loss of some critical function performed by AI itself, or do these deficits arise from secondary effects occurring at some other cortical or subcortical site that has direct connections with AI?

One potential cause for the disruption of sound-localization behavior after auditory cortex lesions may be an increase in absolute threshold. By this view, an animal with an auditory cortex lesion cannot localize sound because of a consequent monaural hearing deficit (e.g., 48, 76). With a unilateral lesion, the threshold for contralateral ear stimulation may be elevated, resulting in some way in an abnormal binaural cue extraction or representation. Several investigators agree that for durations longer than 100–200 ms, absolute thresholds are normal (4, 16, 17, 19, 25, 45, 46). For stimuli of shorter duration, several investigators have reported small upward threshold shifts (4, 25, 45, 46, 92), whereas others have not recorded significant differences from normal thresholds (16, 17, 19). If such shifts are present, they might be especially important for models of sound-localization processes that involve callosal or crossed comparisons.

All recorded shifts in threshold are only of the order of a few decibels. The behavior of the cats in the present experiments reveal that they have no difficulty hearing a sound originating from a contralateral source at a deficit frequency, as they move rapidly, albeit inaccurately and nearly randomly to one of the speaker locations in the contralateral hemifield. At a severe deficit frequency, performance was little affected by significant changes in stimulus intensity. The deficit was unilateral. Changes in binaural cues introduced at the periphery (e.g., changes in interaural time or intensity difference cues) produce errors in localization throughout the sound field (43). Moreover, such errors introduced peripherally are compensated for, at least in humans (e.g., see Refs. 24, 33). Thus, for example, patients with unilateral sensorineural hearing losses ranging up to 35–60 dB are capable of apparently normal sound localization. Animals with callosal or other midline lesions, except for the trapezoid body, have normal sound-localization capabilities (39, 62). For these and other reasons, it is not reasonable to conclude that the contralateral localization deficits reported here result from any simple elevation in absolute thresholds for stimulation of the contralateral ear.

Other investigators have suggested that

sound-localization deficits consequent from cortical lesions may be due to a defect in auditory attention or memory (29, 66). Following this line of reasoning, the experimental animals cannot remember where the sound originated or are otherwise distracted before they can make a discrimination response, even though they perceive the correct sound location. However, the results of Heffner (30) in dogs with large bilateral auditory cortex lesions and Cowey and Weiskrantz (15) in monkeys with unilateral or bilateral superior temporal cortex lesions indicate that large cortical lesions do not much affect auditory memory or attention. It could be argued that auditory cortex lesions produce a specific sound-location memory deficit. The results of Heffner (30) indicate that this explanation is not tenable, since he was unable to detect any sign that the animals ever solved a localization task in a normal manner. That is, even when the sound remained on until a discrimination response was made, performance did not reach normal levels and the animal's behavior (principally scanning head movements) indicated that they were using monaural and not binaural sound-localization cues.

It has also been suggested that auditory cortex lesions may isolate sound-localization mechanisms from motor mechanisms necessary for some but not all behavioral responses (29, 30, 65, 66). Thus, by this view, animals with cortical lesions are incapable of making the complex motor responses necessary for free-field localization tests of the type employed in these studies but may be capable of indicating the locus of a sound by making simple lever presses (29) or by making reflexive or orienting responses (5, 81, 83). This conclusion is based, in part, on the observation that cats with cortical lesions retain the ability to detect interaural intensity and phase differences, and it thus provides additional evidence that the neocortex has no primary sensory role in sound localization (18). However, these studies by no means provide a proof that these cats have a normal perception of sound space.

In this experimental series, motor responses were not much different from normal at deficit frequencies; these cats simply make large unsystematic errors in identifying the correct speaker at deficit frequencies. It is difficult to absolutely rule out "auditory-motor disconnection." However, it seems more likely that cats, like humans, have lost or have a degraded perception of auditory space following a cortical lesion.

Possible neural sources of behavioral deficit

Given that AI is both necessary and sufficient for binaural sound-localization ability and that lesions along an isofrequency line within AI produce corresponding frequency-dependent sound-localization deficits, the question arises as to the site(s) of action(s) of these lesions that are the basis for the observed behavioral deficits. AI has tonotopic reciprocal connects with the homolateral anterior auditory field, the posterior auditory field, and the ventral posterior auditory field, and with the heterolateral AI, anterior auditory field, and posterior auditory field (8, 34, 36). The corticothalamic and thalamocortical connections of AI are reciprocal and tonotopically organized and include several divisions of the medial geniculate body (i.e., ventral division, deep dorsal division, and medial division) as well as from the posterior group (2, 13, 20, 21, 35, 36, 59, 70, 89). There are also tonotopic projections from AI to the basal ganglia (14, 67) as well as to the pericentral nucleus and dorsomedial aspect of the central nucleus of the inferior colliculus (3). Thus a lesion along an isofrequency line within AI produces a plethora of possible affected sites that might be involved in binaural sound-localization behavior.

The results of *C0140,* with only a surviving narrow sector of isofrequency representation within AI, suggest that the connections of AI to the homolateral cortical fields are probably not necessary for binaural sound-localization ability. Partial unilateral or bilateral lesions of the inferior colliculus that include all of the dorsomedial aspect and most or all of the pericentral nucleus but spare some of the central nucleus of the inferior colliculus do not affect the ability to localize brief broad-spectrum sounds (37, 43). It would appear that the descending connections of AI to the inferior colliculus are probably not essential for binaural sound localization. Transection of the corpus callosum does not disrupt the ability to localize brief sounds under conditions identical to those used in these experiments (39) nor does it cause elevations in the minimum audible angle (unfortunately

measured only with long-duration sounds) (62). These studies indicate that the connections of AI to contralateral auditory cortical fields are not necessary for normal sound-localization ability. Possible crucial roles of two principal AI projection targets cannot be eliminated. AI is the target of an essential projection from the lateral part of the medial geniculate body (MGB), i.e., this nucleus retrogradely degenerates after an AI lesion (70). Both the MGB and basal ganglia receive topographically ordered projections from AI. There is no other basis for evaluating whether ultimate forms of sound-localization representations might be located at either location, within AI, or some combination thereof. Thus, although it is clear that the integrity of AI is requisite for binaural sound-localization ability, the sufficiency of AI for normal binaural sound-localization ability must include the possibility that the interactions of AI with other forebrain neural structures constitutes the true cause of observed binaural sound-localization deficits and accounts for the perceptual representation of free-sound-field location.

Binaural sound-localization is processed within frequency channels

The present results indicate that *1*) AI is organized to represent the contralateral hemifield of auditory space, and *2*) this spatial organization occurs within frequency-representation channels. That is, unilateral lesions along an isofrequency representational zone within AI produce corresponding contralateral frequency-specific sound-localization deficits. Frequency-dependent contralateral deficits in apparent location based on interaural time differences have been previously reported in humans with central lesions at the level of the ventral brain stem up to and including the temporal lobe. More peripheral brain stem lesions produce ipsilateral deficits (54). Some additional support for this contralateral frequency-channel organization can be found in the human binaural psychophysical literature. It has been reported that within the frequency domain there exist critical bands for auditory lateralization (74). For diotically presented tones with given onset time differences, the lateral position of a sound source is constant when components are added within a critical band. With separations beyond the critical band, a monotonic increase in onset time differences was necessary for a lateralization judgement. As two diotically presented tones were progressively separated by more than one critical band, they became harder to lateralize, i.e., the spatial perception of the apparent source became more diffuse. Lateralization of amplitude-modulated tones, whose carrier frequencies differed, indicated that performance remained constant when the carrier frequencies were within a critical band. Beyond a critical band, performance deteriorated (32, 64). The results from numerous human psychophysical studies support the hypothesis that neural resolution of interaural signal differences on which binaural sound-localization ability depends is performed in frequency channels, in that it is the components within critical band distances that are interaurally compared (for reviews see Refs. 6, 12, 22).

Electrophysiological studies of the response properties of neurons in the central auditory nervous system also lend support to the notion of binaural processing by frequency channel. It has been demonstrated that at the first sites in the central auditory nervous system at which binaural interactions occur (i.e., in the superior olivary complex) *1*) the characteristic frequencies of neurons driven from ipsilateral or contralateral stimulation are similar; and *2*) these nuclei are tonotopically organized (27, 28, 84). More recent studies have shown that other features of response, such as fields of excitation, inhibition, inhibitory sidebands, disinhibitory sidebands, etc. are approximately binaurally matched re frequency or are complementary re frequency (7, 10). These binaural nuclei provide principal inputs to midbrain nuclei of the mainline (tonotopically organized) ascending auditory system ultimately feeding AI (1, 2, 9, 11, 26, 35, 59, 71). On this projection pathway, from the first sites of binaural interaction within the hindbrain up to and including AI, neurons with binaural response properties are organized tonotopically, and the inputs from both ears to these neurons are at least roughly matched binaurally re frequency (7, 10, 27, 55).

Where are sound source maps located?

One puzzling aspect of studies of the auditory forebrain has been the difficulty in demonstrating the presence of a topographic representation of sound location in the most

intensively studied species, the cat. Topographic representations of a number of features of sound stimuli have been described in bats (79), and sound-location maps have been recorded in the tecta of owls (50, 51). Several investigators have recently conducted free-sound-field mapping studies in presumably homologous structures in cats. They have found the following. *1*) Unlike the owl, no topographic representation of sound location is evident in any inferior colliculus subdivision in cats (52, 75). *2*) A map of sound location can be demonstrated in the superior colliculus of cats only with a quantitative analysis of relative response rates (60). *3*) Some AI neurons have location-specific response properties, but a strict topographic representation of location has not yet been demonstrated in the cat (58).

Perhaps this is due to the practical limitations of these mapping experiments. Perhaps simple maps are present in a derived parametric form, e.g., as initially suggested by Tunturi (85), and are not in the simple form of continuous topographic representation of sound location. Perhaps the map is manifest in the ensemble response of neurons but less evident in analysis of single units. Perhaps simple continuous maps of sound location are not requisite for normal localization behavior.

Possible basis of compensation for a partial loss of AI representation

It should be noted that some of the results from *cat C0057* and the second lesion in *cat C8062* with adjacent partial frequency-band lesions suggested that only complete lesions of frequency-band representations within AI produced permanent sound-localization deficits. The second lesion in *cat C8062* destroyed the representation of an octave of frequency over the upper half of AI and the representation of the next higher octave of frequency over the lower half. A deficit was recorded only at the frequency (16 kHz) at which the lesions overlapped (i.e., extended all the way across AI). We guessed that temporary deficits at other frequencies might have occurred but were missed because it was many weeks after the lesion before localization performance was evaluated at these other test frequencies whose AI representations had been only partially destroyed. At least two explanations could account for these results. First, a partial frequency representation within AI may be sufficient for apparently normal sound-localization performance on the behavioral test used in these studies. Second, a partial lesion of the frequency representation within AI may produce a transient sound-localization deficit that depends on either compensatory behavioral strategies, or on reorganization within the remaining partial frequency representation.

An additional experiment was conducted to test these hypotheses. In this cat, a lesion destroyed the upper half of the AI representation of a relatively low frequency band, and the cat's behavioral sound-localization performance was immediately tested at a suspected deficit frequency. At this frequency, there was an initial deficit, but it was temporary. Behavioral performance recovered gradually to normal levels over a several week period.

Studies conducted principally in the somatosensory system (57) indicate that central remapping may underly compensation following a peripheral or central lesion or deprivation. If there is a similar capacity for representational plasticity in AI, any substantial part of AI may have the capacity to effect, on reorganization, a relatively complete representation of the sound field. Thus we hypothesize (57) that when a part of the isofrequency representation in AI is destroyed, the remainder may have the capacity of remapping a complete sound-field representation within it. Obviously, confirmation of that interesting possibility requires further experimentation.

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