Music Perception and Cognition Following Bilateral Lesions of Auditory Cortex

Mark Jude Tramo
Program in Cognitive Neuroscience
Dartmouth Medical School

Jamshed J. Bharucha
Department of Psychology
Dartmouth College

Frank E. Musiek
Division of Audiology, Section of Otolaryngology, Department of Surgery
Dartmouth Medical School

Abstract

■ We present experimental and anatomical data from a case study of impaired auditory perception following bilateral hemispheric strokes. To consider the cortical representation of sensory, perceptual, and cognitive functions mediating tonal information processing in music, pure tone sensation thresholds, spectral intonation judgments, and the associative priming of spectral intonation judgments by harmonic context were examined, and lesion localization was analyzed quantitatively using straight-line two-dimensional maps of the cortical surface reconstructed from magnetic resonance images.

Despite normal pure tone sensation thresholds at 250–8000 Hz, the perception of tonal spectra was severely impaired, such that harmonic structures (major triads) were almost uniformly judged to sound dissonant; yet, the associative priming of spectral intonation judgments by harmonic context was preserved, indicating that cognitive representations of tonal hierarchies in music remained intact and accessible. Brainprints demonstrated complete bilateral lesions of the transverse gyri of Heschl and partial lesions of the right and left superior temporal gyri involving 98 and 20% of their surface areas, respectively. In the right hemisphere, there was partial sparing of the planum temporale, temporoparietal junction, and inferior parietal cortex. In the left hemisphere, all of the superior temporal region anterior to the transverse gyrus and parts of the planum temporale, temporoparietal junction, inferior parietal cortex, and insula were spared.

These observations suggest that (1) sensory, perceptual, and cognitive functions mediating tonal information processing in music are neurologically dissociable; (2) complete bilateral lesions of primary auditory cortex combined with partial bilateral lesions of auditory association cortex chronically impair tonal consonance perception; (3) cognitive functions that hierarchically structure pitch information and generate harmonic expectancies during music perception do not rely on the integrity of primary auditory cortex; and (4) musical priming may be mediated by broadly tuned subcomponents of the thalamocortical auditory system. ■

INTRODUCTION

The orderly arrangement of anatomical zones within the auditory cortex has inspired a number of authors to assign fine structure a role in functional specialization. The belief that structural complexity and connectivity patterns reflect functional complexity and hierachical processing was expressed long ago by Campbell (1905), who designated the granular cortex of the supratemporal plane "audiosensory" and the surrounding belt "audiopsychic." However, empirical data about the organization of cognitive functions mediating auditory information processing outside the verbal domain remain scarce. Although the cortical representation of sensory information (e.g., frequency receptive fields) has been extensively documented in the cat and monkey using neuronal-evoked potential techniques (for reviews see Aitkin et al. 1984; Brugge and Reale 1985), we know little about how physical features of acoustic signals are processed to form psychological representations of sound, and how knowledge about sound contributes to sensory integration and percept formation. The effects of bilateral ablations in animals indicate that these higher levels of processing rely on the integrity of auditory cortex (for reviews see Neff et al. 1975; Whitfield 1985); the relative roles of the primary and secondary areas have not yet been distinguished. In man, physiological studies employing depth electrodes (Celesia 1976), magnetoencephalography (Hari et al. 1980; Romani et al. 1982), and radioisotope imaging (Lauter et al. 1985) have primarily focused on the problems of boundary definition and tonotopicity, and have therefore used elementary acoustic stimuli such as clicks and pure tones to evoke cortical responses. Anatomical data derived from the study of neurological populations are likewise wanting given the rarity of auditory-nonverbal deficits gross enough to cause overt symptoms following focal unilateral lesions, the rarity of focal bilateral and symmetrical lesions, and the proximity and common vascular supply of the primary and secondary auditory areas. Furthermore, brain scans obtained for clinical indications provide a limited view of the supratemporal plane because of their orientation (horizontal) and slice thickness (8–10 mm), and therefore frequently fail to capture the transverse gyri of Heschl, which typically house the primary auditory areas, and the full extent of the superior temporal gyrus, temporoparietal junction, parietal operculum, and temporal pole, which typically house the auditory association areas (Galaburda and Sanides 1980).

In the present study, we examined a well-documented case of cortical hearing loss (Mendez and Geehan 1988) under experimental conditions designed to tap selectively into sensory, perceptual, and cognitive functions mediating tonal information processing. The choice of a musical priming paradigm (Fig. 1; Bharucha and Stoeckig 1986, 1987) reflected the desire to engage cognitive processes that hierarchically structure sensory information and thereby facilitate spectral pattern perception. The cortical mapping procedure of Jouandet et al. (1989) permitted the localization and quantification of damage to the presumed gross anatomical landmarks of the mary auditory cortex and auditory association areas imaged by thin-section coronal magnetic resonance scanning (MR).

RESULTS

Pure Tone Sensation Thresholds

Intensity thresholds for detecting a 500 msec pure tone at 250, 500, 1000, 2000, 4,000, and 8000 Hz were within normal limits for each ear (Fig. 2).

Spectral Intonation Judgments

Intonation judgments about harmonic spectra (major triads) and quasiharmonic spectra (mistuned major triads) were performed better than chance [56%; $t(47) = 2.00$, $p < .05$], but overall accuracy was more than 2 SD below the mean of normals' performance on the more difficult priming task (83 ± 11%; Bharucha and Stoeckig 1987).

Associative Priming of Spectral Intonation Judgments

M.S.'s performance on the musical priming task is illustrated in Figure 3 alongside data previously obtai from 13 normal subjects using the same stimuli and procedure.

Overall accuracy was at chance [48%; $t(95) < 1$] and fell greater than three standard deviations below the mean of the normal population (.07th percentile). There

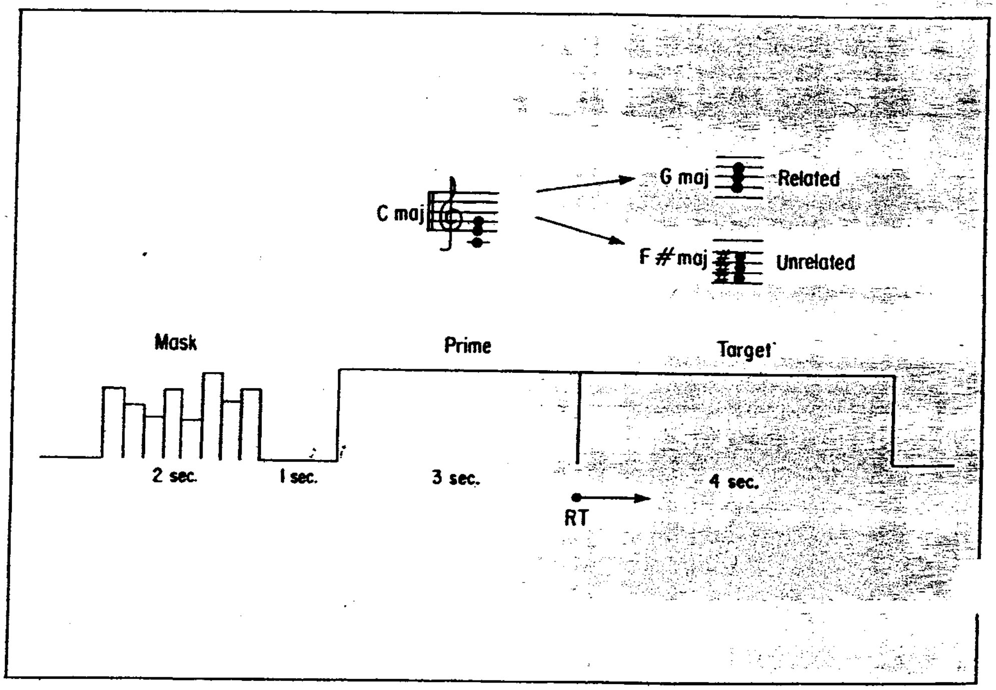
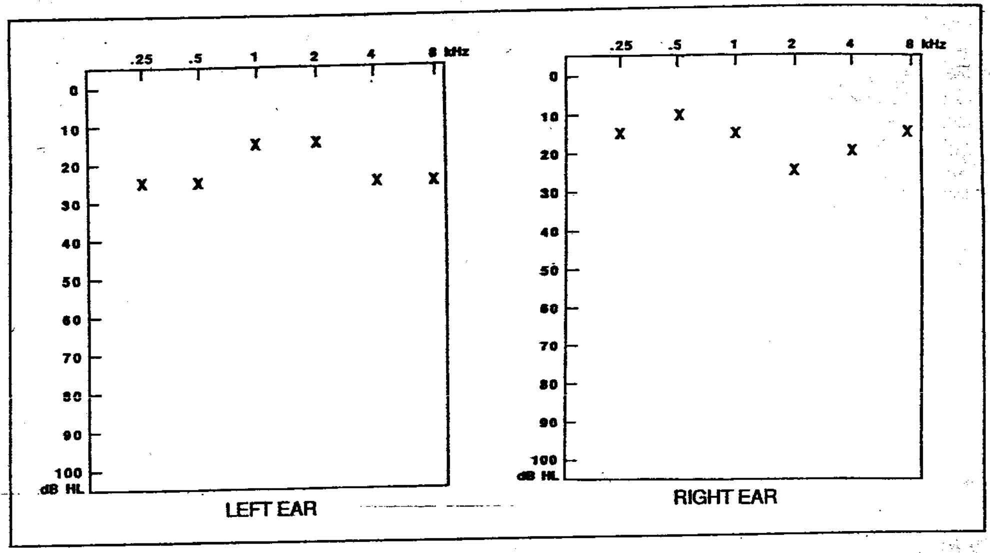


Figure 1. Trial design in the musical priming task. Each trial began with a rapid sequence of randomly selected frequencies to mask the influence of the previous trial. Following a 1 sec pause, a prime chord (major triad) was presented for 3 sec, followed by a target chord, the onset of which marked the start of response recordings. The target was either harmonically related or harmonically unrelated to the prime (defined by high and low transition probabilities in Western tonal music and previous relatedness judgments in normals). The components of each target triad were either in-tune or mistuned by flattening the fifth a fraction of a semitone. The subject's task was to judge whether the target was in-tune or out-of-tune (see Methods for details).

Figure 2. Audiograms documenting the intensity levels (in decibels) at which a 500 msec pure tone was detected at 250–8000 Hz.



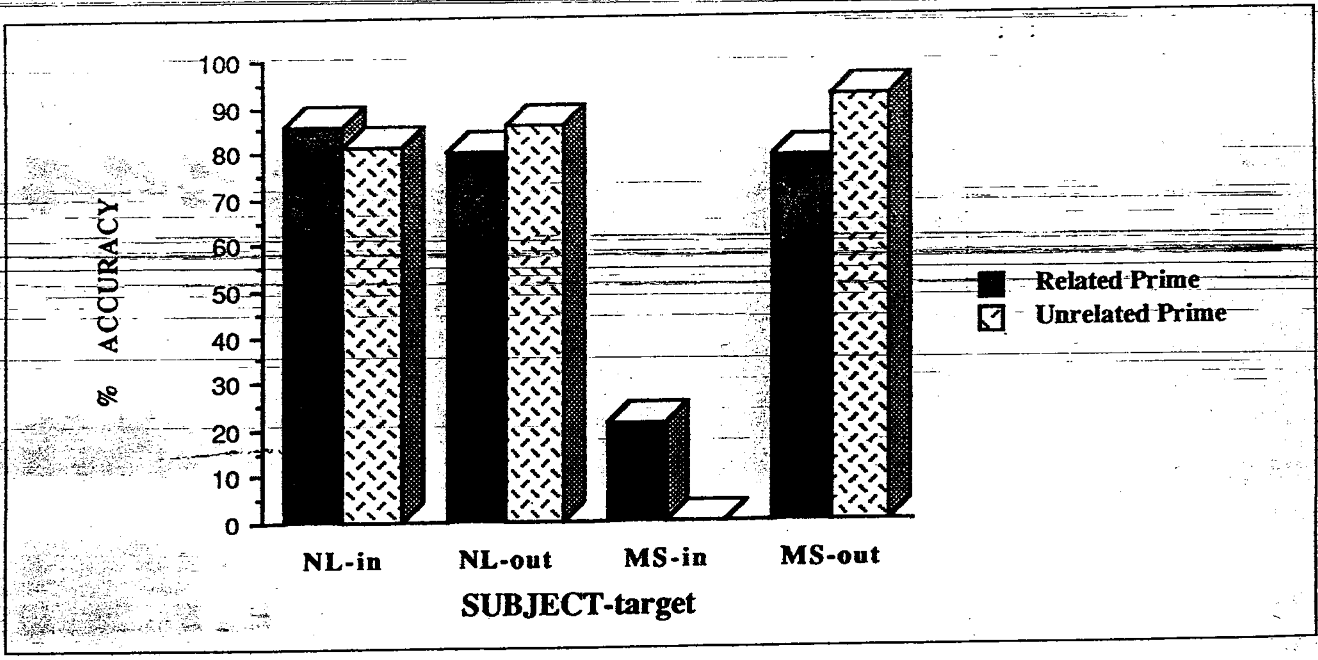


Figure 3. Accuracy of intonation judgments in each of the four prime-target conditions. In patient M.S., there is a marked out-of-tune response bias, yet the interaction between intonation and relatedness is significant in the same direction as the normal interaction previously reported by Bharucha and Stoeckig (1987).

was a marked bias to judge target chords "out-of-tune" [$F(1,23) = 112.66, p < .0001$]. However, the interaction between target intonation and the harmonic relatedness of the prime to the target was significant in the predicted direction [$F(1,23) = 5.41, p < .05$]. As in normals, M.S. was more accurate when (1) in-tune target chords were preceded by a prime chord that was harmonically related to the target; and (2) mistuned target chords were preceded by a prime chord that was not harmonically related to the target. In none of the 24 in-tune/unrelated trials was the target correctly perceived to be in-tune. Statistical analysis of the intonation/relatedness interaction using reaction time data was precluded by the absence of any correct responses in the in-tune/unrelated condition.

Brainprints and Quantitative Lesion Localization

Straight-line two-dimensional surface maps of the unfolded right and left cerebral hemispheres are illustrated in Figures 4 and 5, respectively. Coronal MR sections through the superior temporal region representative of those used to map the auditory cortex are shown in Figure 6. The surface areas (SA) of individual regions of

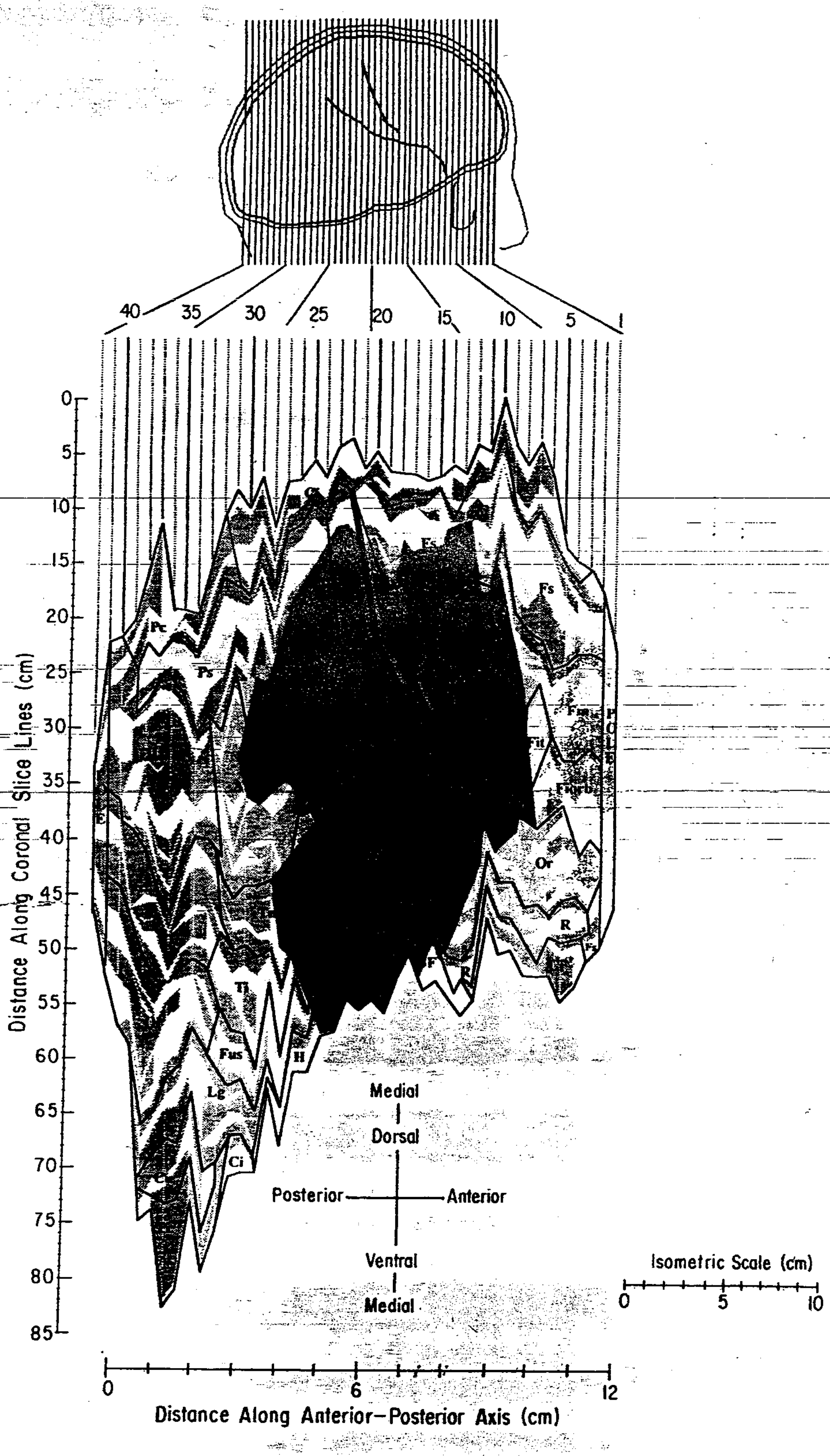


Figure 4. Brainprint of M.S.'s right cerebral cortex. Coronal MR sections are numbered sequentially from anterior to posterior (right to left on the page). Gray areas indicate intrasulcal cortex; yellow area indicates cortex lying within and/or overlying abnormal MR signal. Regional surface area measurements are given in Table 1 and regional lesion measurements in Table 2. For glossary of abbreviations, see Appendix.

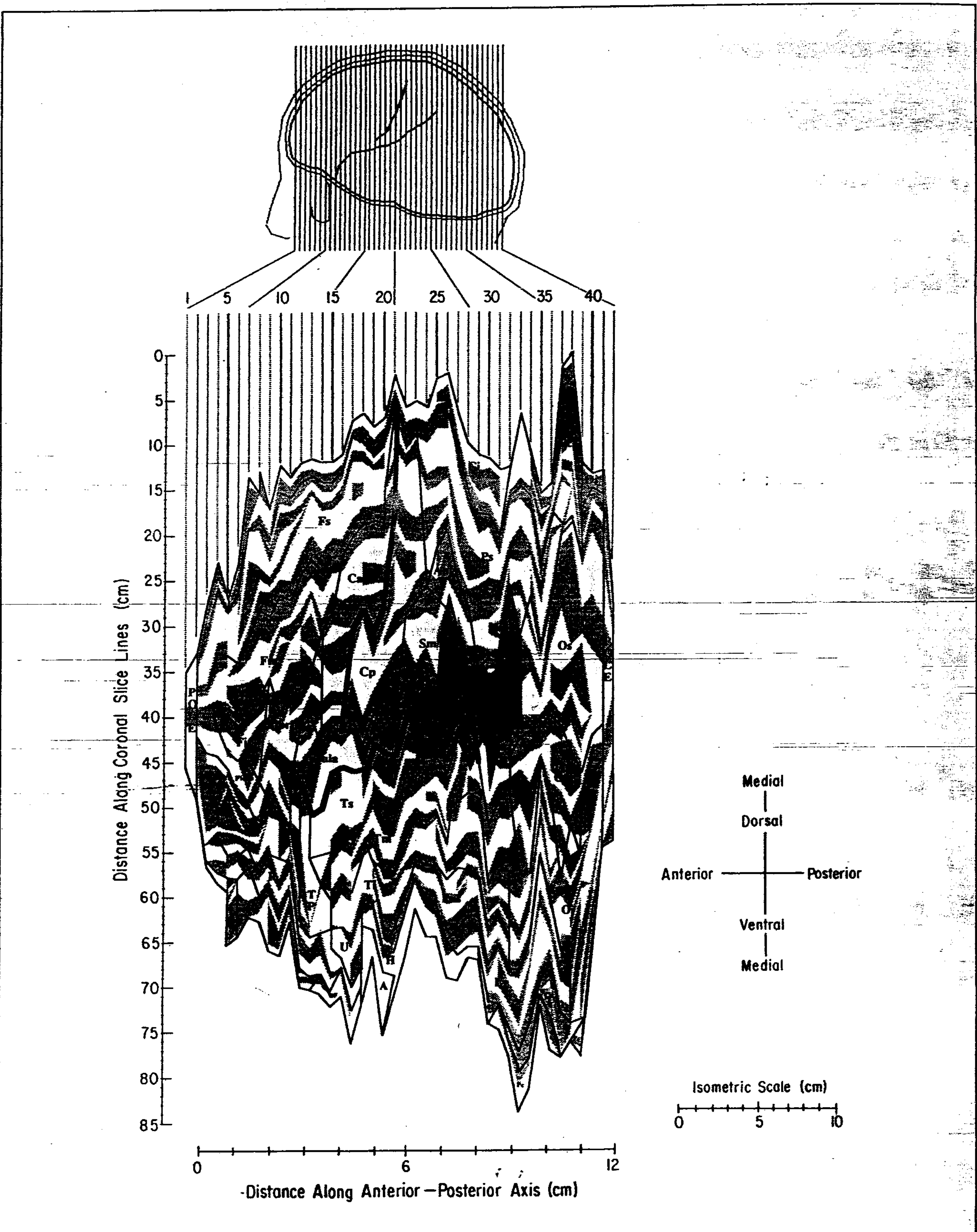


Figure 5. Brainprint of M.S.'s left hemisphere. Coronal MR sections are numbered sequentially from anterior to posterior (left to right on the page). See Figure 4 for details about graphics, Tables 1 and 2 for quantitative measures, and the Appendix for abbreviations.

Table 1. Regional Cortical Surface Area Measurements for the Right and Left Hemispheres

	Right Hemisphere		*Left Hemisphere*	
	cm²	*% hemisphere*	*cm²*	*% hemisphere*
Temporal Lobe	159	20	176	20
Transverse gyrus	3		3	
Superior temporal gyrus	27		33	
Temporal pole	9		8	
Middle temporal gyrus	32		43	
Inferior temporal gyrus	40		41	
Hippocampal region	15		41	
Uncus	5		5	
Amygdala	2		7	
Fusiform	26		21	
Parietal Lobe	183	23	208	23
Postcentral gyrus	32		44	
Supramarginal gyrus	12		21	
Angular gyrus	45		35	
Superior parietal lobule	63		72	
Precuneus	31		36	
Frontal Lobe	236	30	247	27
Superior frontal gyrus	74		72	
Middle frontal gyrus	46		43	
Inferior frontal gyrus				
Pars orbitalis	16		10	
Pars triangularis	6		9	
Pars opercularis	22		22	
Precentral gyrus	22		37	
Orbitofrontal gyri	33		38	
Rectal gyrus	17		16	
Occipital Lobe	151	19	190	21
Lateral occipital gyri	113		149	
Cuneus	12		11	
Lingual	26		30	
Other/Paralimbic				
Insula	17		28	
Parainsular area	0.7		0.9	
Cingulate gyrus	63		58	
Basal forebrain	7		5	
Total Hemisphere	800		899	

Table 2. Percent Regional Cortical Surface Area Contained within or Undercut by the Lesion

	Right Hemisphere			*Left Hemisphere*		
	% Lesion	*% within*	*% undercut*	*% Lesion*	*% within*	*% undercut*
Temporal Lobe	64	27	37	7	7	—
Tranverse gyrus(i)	100	100	—	100	100	—
Superior temporal gyrus	98	76	22	20	20	—
Temporal pole	100	100	—	—	—	—
Middle temporal gyrus	70	43	27	4	4	—
Inferior temporal gyrus	53	12	41	—	—	—
Hippocampal region	68		68	—	—	—
Uncus	100	33	67	—	—	—
Amygdala	100	—	100	—	—	—
Fusiform	35	—	35	—	—	—
Parietal Lobe	45	14	31	15	9	6
Postcentral gyrus	91	48	43	—	—	—
Supramarginal gyrus	100	53	47	32	32	—
Angular gyrus	46	2	44	56	33	23
Superior parietal lobule	29	3	26	5	—	5
Precuneus	—	—	—	—	—	—
Frontal Lobe	40	19	21	—	—	—
Superior frontal gyrus	15	—	15			
Middle frontal gyrus	48	23	25			
Inferior frontal gyrus						
Pars orbitalis	9	—	9			
Pars triangularis	59	—	59			
Pars opercularis	100	88	12			
Precentral gyrus	94	49	45			
Orbitofrontal gyri	35	11	24			
Rectal gyrus	5	—	5			
Occipital Lobe	—	—	—	2	1	1
Lateral occipital gyri				4	2	2
Precuneus				—	—	—
Cuneus				—	—	—
Lingual				—	—	—
Fusiform				—	—	—
Other/Paralimbic						
Insula	100	100	—	9	9	—
Parainsular area	100	100	—	100	100	—
Cingulate gyrus	—	—	—	—	—	—
Basal forebrain	4	4	—	—	—	—
Total Hemisphere	36	17	19	5	4	1

interest are listed in Table 1, and the proportion of each region that was affected by the lesion is listed in Table 2.

There were complete, bilateral lesions of the transverse gyri of Heschl. Their SA measured 3 cm^2 on each side, almost 2 SD below the mean of previous normal measurements obtained by the same method (7.6 ± 2.5 cm^2, $N = 12$; Jouandet et al. 1989, and unpublished data).

In the right hemisphere (Figure 4), almost all superior temporal gyrus SA (98%) and all temporal pole SA were contained within and/or overlay areas of abnormal MR signal. Most of the planum temporale was undercut by the lesion; a small portion of its posterior extension into and including the temporoparietal junction was spared. Elsewhere in the temporal lobe, 70% of middle temporal gyrus SA and 53% of inferior temporal gyrus SA were affected by the lesion; there was extensive involvement of medial temporal structures, including 68% of the hippocampal region, all of the amygdala, and all of the uncus. In the parietal lobe, all of the operculum was involved; 100% of supramarginal gyrus SA, 91% of postcentral gyrus SA, and 46% of angular gyrus SA were within or undercut by the lesion. All of the insula was involved. In the frontal lobe, the pars opercularis was completely infarcted; in addition, the posterior portions of the pars orbitalis (38% SA), pars triangularis (59%), middle frontal gyrus (48% SA), superior frontal gyrus (15% SA), and orbitofrontal gyri (35% SA) and almost all of the precentral gyrus (94% SA) were involved. Occipital lobe structures were completely spared.

In the left hemisphere (Figure 5), the posterior 20% of superior temporal gyrus SA, including all but small posterolateral segments of the planum temporale and temporoparietal junction, was contained within areas of abnormal MR signal; the superior temporal gyrus anterior to the transverse gyrus and all of the temporal pole were spared. Four percent of adjacent middle temporal gyrus SA was affected by the lesion, mostly within the superior temporal sulcus. In the parietal lobe, much of the operculum was lesioned posteriorly; 32% of supramarginal gyrus SA and 56% of angular gyrus SA were involved. The anterior 91% of insula SA was spared. Small portions of the superior parietal lobule (5% SA) and anterior lateral occipital gyri (<4% SA) were involved.

In the right hemisphere, the lesion extended deep into the subcortical white matter, putamen, caudate nucleus, globus pallidus, claustrum, internal capsule, external capsule, and extreme capsule (Figure 6); it comprised 27% of total right hemisphere volume. The left hemisphere lesion was largely superficial and comprised 4% of total left hemisphere volume.

DISCUSSION

To summarize the results: (1) pure tone sensation thresholds were normal; (2) intonation judgments about harmonic spectra and mistuned harmonic spectra were grossly inaccurate, and there was a response bias indicating that tonal consonance perception was sever impaired; and (3) the associative priming of spectral intonation judgments by harmonic context was preserved. Brainprints demonstrated (1) complete bilateral lesions of the transverse gyri of Heschl, and (2) partial bilateral lesions of the superior temporal gyri involving 98% of the cortical surface area on the right and 20% on the left. The right hemisphere infarct spared portions of the planum temporale, temporoparietal junction, and inferior parietal cortex; the left hemisphere infarct spared all of the anterior superior temporal gyrus and temporal pole and parts of the planum temporale, temporoparietal junction, inferior parietal cortex, and insula.

Functional Dissociations

The perception of tonal consonance is determined by the frequency relationships among simultaneous elements within tonal spectra (Helmholtz 1863/1954; Levelt et al. 1966; Terhardt 1974, 1984) and is influenced by the harmonic context established by prior spectra (Bharucha and Stoeckig 1986, 1987; Marin and Barnes 1985). Normal subjects exhibit a dissociation between spectral content and associative priming as they influence intonation judgments: degrading the spectrum by removing some harmonics renders the intonation judgment more difficult but does not diminish the priming effect (Bharucha Stoeckig 1987). In M.S., frequency information could detected at normal thresholds, but fine-grained analysis of the frequency relationships among major triad components was severely impaired. Still, "top-down" influences on tonal consonance perception remained operative. Sensory, perceptual, and cognitive functions mediating tonal information processing thus appear to be neurologically dissociable.

A number of authors have proposed that auditory percepts are reconstructed from fragments of sound at higher, associative levels of processing (for reviews see Hartmann 1988; Whitfield 1985). That associative processes which generate harmonic expectancies in music can be activated even when elementary components of musical stimuli are degraded or the capacity to make discriminations at this level is lost suggests that synthetic processes (e.g., the integration of frequency relationships into a harmonic whole) do not depend entirely upon fine-grained analytic processes (e.g., the ability to resolve frequency differences between individual spectral components) in music perception.

Lesion Localization

If correlations between gross surface structure landmarks and microscopically distinct zones of auditory cortex be cautiously entertained on the basis of normative data from six hemispheres (Galaburda and Sanides 1980), the topography of M.S.'s lesions (Figures 4 and 5) indicates

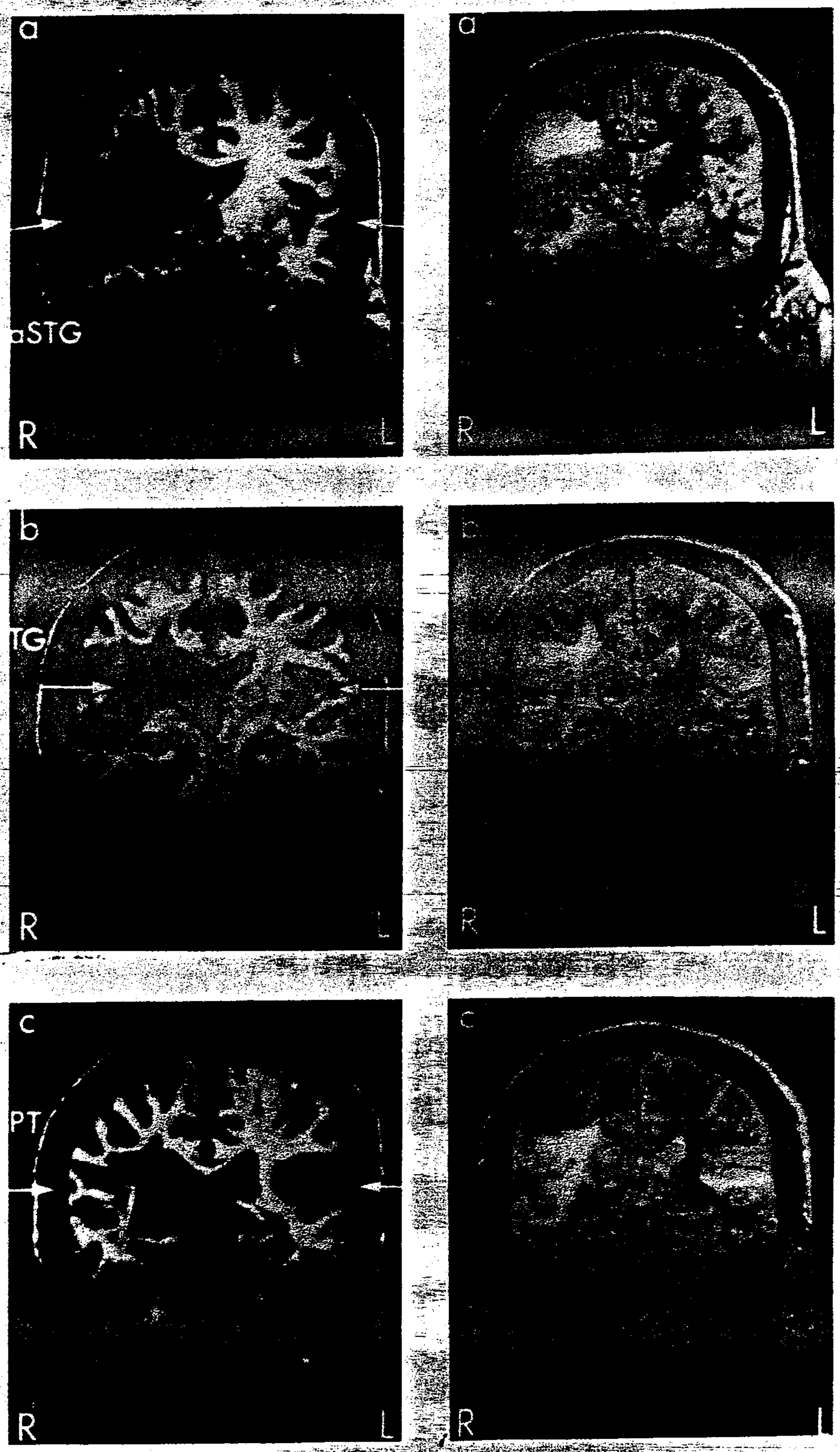


Figure 6. Coronal MR images of the perisylvian region representative of the 20 sections through the area depicted in the brainprints. MRs a and a′ are T1- and T2-weighted images, respectively, of the anterior superior temporal gyrus (aSTG). MRs b and b′ are sections through the transverse gyri of Heschl (TG); the T2-weighted image clearly demonstrates left- as well as right-sided hyperintense signal abnormalities. Sections c and c′ show the planum temporale (PT), that portion of the superior temporal gyrus lying posterior to the transverse gyrus of Heschl. Note the extensive destruction of the deep white matter and basal ganglia in the right hemisphere.

complete, bilateral lesions of the koniocortical fields. In the right hemisphere, some of the temporoparietal isocortex and perhaps a small posterior portion of the external parakoniocortex may have been spared. Much of the remaining posterior association belt on the right, including most of the external and all of the internal and caudodorsal parakoniocortex, appears to be undercut by the lesion. Right perisylvian structures anterior and medial to the transverse gyrus, including the rostral parakoniocortex, prokoniocortex, rostral isocortex, parainsular zone, and temporopolar proisocortex, appear to be infarcted. In the left hemisphere, small posterolateral portions of temporoparietal isocortex and perhaps external parakoniocortex may have been spared; much or all of the rostral parakoniocortex, isocortex, and proisocortex along the anterior superior temporal gyrus, temporal pole, and parainsular zone appear to be spared. All of the internal, external, and caudodorsal parakoniocortex as well as the prokoniocortex on the left may have been infarcted.

The physiological properties of neurons within the perisylvian areas that are spared in M.S. can only tentatively be surmised on the basis of their response characteristics in presumably homologous regions of the monkey, which themselves have only partially been worked out (Imig et al. 1977; Katsuki et al. 1962; Merzenich and Brugge 1973; Pfingst et al. 1977; Vaadia et al. 1982). If all or part of the left rostral parakoniocortex was spared in M.S., and if the tonotopically organized rostral field (R of Imig et al. 1977) of the monkey is functionally as well as anatomically (Galaburda and Sanides 1980) homologous to the rostral parakoniocortex in man, sharply tuned, tonotopically organized frequency information may have been available for processing; the flow of information would be limited posteriorly by the lesion but could proceed rostrally to higher order modality-specific and heteromodal association areas. However, even if portions of sharply tuned, tonotopically organized regions of association cortex were spared in M.S., the principal source of such information, the primary area, was lesioned bilaterally. The afferent input to the spared auditory association areas might therefore be limited to projections from the thalamus.

The differential distribution of thalamic afferents to the various auditory fields is uncertain in man. The connectivity patterns of presumably homologous regions in the monkey (Burton and Jones 1976; Jones and Burton 1976; Mesulam and Pandya 1973; Trojanowski and Jacobson 1975) show that the auditory association areas receive direct input from the thalamus. In M.S., sensory information may have reached the cortex almost exclusively via projections from dorsal and medial regions of the medial geniculate body (MGB) and from the pulvinar-posterior nuclear complex. In the right hemisphere, the deep capsular extension of the infarct may have interrupted the thalamocortical projections altogether. Given that many of the neurons in these thalamic regions show inconsistent frequency responses and irregular tonotopicity in both the monkey (Gross et al. 1974) a cat (Aitkin 1973; Calford 1983; Phillips and Irvine 1979), no sharply tuned, tonotopically organized frequency information may have been available to the spared cortical association areas; at the very least, it was likely incomplete, perhaps owing to projections from sharply tuned cells in some regions of magnocellular MGB (Aitkin 1973) and lateral pulvinar (Phillips and Irvine 1979).

The corticocortical connectivity patterns of the auditory areas that appeared to be spared in M.S. may likewise be considered with regard to presumably homologous areas in the monkey (for review see Pandya and Yeterian 1985). In the right hemisphere, the intrinsic rostral connections of the spared posterior association areas with the primary area and anterior superior temporal region appear to be completely interrupted; long projections to and from prefrontal cortex may have likewise been destroyed, but reciprocal connections of the posterior association areas with the posterior cingulate gyrus and heteromodal temporoparietal regions may have been at least partially spared. In the left hemisphere, caudal connections of the spared anterior association areas were likely interrupted, but the intrinsic connections of the rostral parakoniocortex, isocortex, and proisocortex along the anterior superior temporal region and the long connections of these areas to prefrontal cortex and medial temporal structures may have been spared in th entirety. The island of preserved posterior association cortex on the left appears to be completely disconnected from the rostral primary and association areas, but its reciprocal connections with posterior cingulate gyrus and heteromodal temporoparietal cortex may have been at least partially spared.

Structure–Function Considerations

There has been some controversy in the animal literature as to whether or not bilateral lesions of auditory cortex permanently impair pure tone sensation (for review see Neff et al. 1975), but recent psychoacoustic measurements in monkeys have documented increased detection thresholds, most prominently in the mid-frequency range, as long as 1 year after extensive bilateral ablations (Heffner and Heffner 1986). In the neurological literature, long-term follow-up measurements of pure tone sensation thresholds in patients with cortical hearing loss are few, especially in young individuals in whom peripheral hearing loss secondary to aging can be ruled out. Earnest et al. (1977) reported the case of a 27-year-old man who manifested increased pure tone thresholds more than 3 years after successive, contralateral, middle cerebral artery strokes; judging from the published computerized transaxial tomograms and Matsui and Hiran (1978) atlas, there were bilateral infarcts of the transverse gyri, superior temporal gyri, frontoparietal opercula, inferior parietal areas, and insulae. Jerger et al. (1969)

reported persistent but improving hearing loss at 3 months follow-up in a 20-year-old man following successive, contralateral middle cerebral artery strokes; no anatomical data were available beyond angiography and technetium brain scan. The present case was previously reported to show complete deafness 3 weeks following his second stroke and moderately increased (and variable) pure tone thresholds 18 months out (Mendez and Geehan 1988). Apparently, recovery of sensory functions can continue and attain normal levels more than a year and a half after onset. In view of the lesion localization, the normal sensory thresholds may be attributed to sparing of the thalamus (beyond any possible retrograde degeneration not imaged by MR), partial sparing of association cortex, or both.

Bilateral lesions of auditory cortex in animals are known to cause permanent deficits affecting the discrimination of various complex spectra, including intermittent vs. steady white noise (Symmes 1966), human vowel sounds (Dewson 1964; Dewson et al. 1969), and species-specific vocalizations (Heffner and Heffner 1988). The clinical literature contains a number of case reports in which the perception of complex tonal spectra, including musical sounds, is impaired out of proportion to hearing loss (at least overtly) following bilateral temporal lobe lesions (for reviews see Benton 1977; Hecaen and Albert 1978; Marin 1982; Vignolo 1969; Wertheim 1969). Two previous experiments are particularly relevant to the present observations. Whitfield (1980) demonstrated in the cat that the capacity to derive the harmonic "best-fit" of a schedoharmonic series was permanently abolished following bilateral ablations of primary auditory cortex and part of the surrounding fields, while relative pitch discriminations based on differences between individual spectral components could be relearned. In man, Zatorre (1988) has recently reported that removal of all or part of the right transverse gyrus(i) along with all of the anterior superior temporal region commonly impairs the perception of the "missing fundamental" (Schouten 1938), which like tonal consonance perception requires the capacity to abstract constancies among the frequency relationships of simultaneous spectral components. These latter cortical regions were infarcted in M.S.

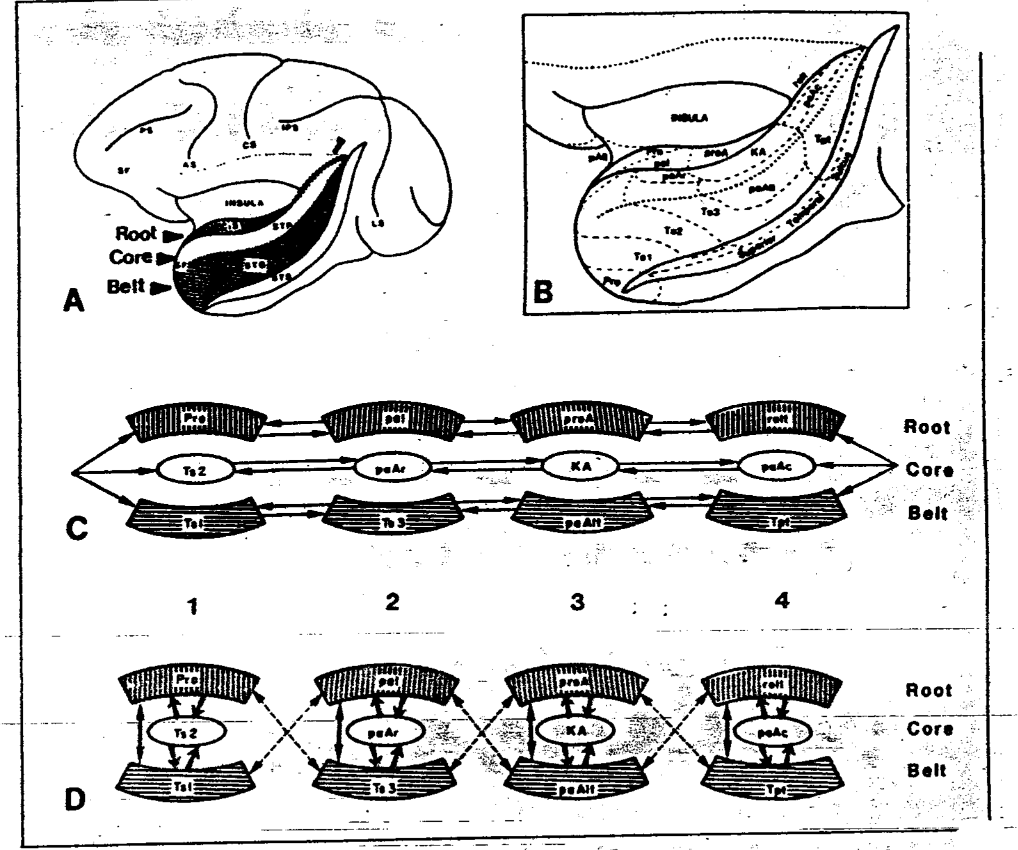
Several authors have commented on the possible correlation between the severity of auditory deficits caused by cortical lesions and lesion size within specialized anatomical zones (Allen 1945; Dewson et al. 1969; Symmes 1966). Colombo et al. (1990) have recently raised the possibility that the size of lesions limited to auditory association cortex may influence the recovery of short-term memory for pitch. In man, there is evidence that the quantity of damage to the left auditory cortex influences the recovery of single word comprehension (Selnes et al. 1984) and sentence comprehension (Naeser et al. 1987) following middle cerebral artery stroke. In M.S., the lesions of the primary areas were complete, suggesting that their role in frequency information processing is essential to tonal consonance perception. The functional significance of the different amounts of sparing within the right and left association areas in the present case cannot be ascertained beyond the assertion that they were sufficient to sustain musical priming.

A number of confounding variables preclude broad-ranging structure–function correlations in the present case. Because M.S. suffered his second stroke 9 years ago, some reorganization of auditory functions within the spared anatomical zones may have taken place. Furthermore, the uncertainties surrounding hemispheric specialization in auditory-nonverbal functions make it difficult to ascertain whether the spared posterior association areas in the right or left hemispheres, spared anterior association areas in the left hemisphere, or all the spared areas combined subserved residual functions. Our recent observation of musical priming by the right hemispheres of two split-brain patients (Tramo and Bharucha 1990) is too limited to permit generalizations concerning laterality effects in the present case. Even if laterality effects in right-handers were known, M.S.'s mixed handedness would preclude a straightforward conclusion. Finally, there are the uncertainties surrounding the interspecies comparisons entertained above, which are especially troublesome in view of the uniquely strong lateralization of auditory-verbal functions in man. The most parsimonious interpretation of the present data is that (1) the perception of tonal consonance is chronically impaired following complete bilateral lesions of primary auditory cortex combined with partial bilateral lesions of auditory association cortex; and (2) musical priming does not rely on the integrity of primary auditory cortex.

Neural Networks in Music

On the basis of the cytoarchitectonic and connectional organization of the superior temporal region in the rhesus monkey, Galaburda and Pandya (1983) have proposed that the auditory cortex comprises three parallel processing streams, the "root," "core," and "belt" lines, which are oriented rostrocaudally along the circular sulcus (Fig. 7). Within each line, cytoarchitectonic differentiation progresses caudally in a stepwise fashion across four stages. Each stage is richly and reciprocally connected to adjacent stages within each line and to its corresponding stages across lines. Pandya and colleagues have hypothesized that the existence of multiple stages of auditory representation signifies hierarchical levels of information processing, such that the rostral flow of connections from cortical layer III to layer IV mediates the successive elaboration of thalamic input and the caudal flow of connections from layers V and VI to layer I mediates the integration of limbic influences on perception (Galaburda and Pandya 1983; Pandya and Yeterian 1985). If a similar organization characterizes human auditory cortex, as Galaburda and Sanides' (1980) work

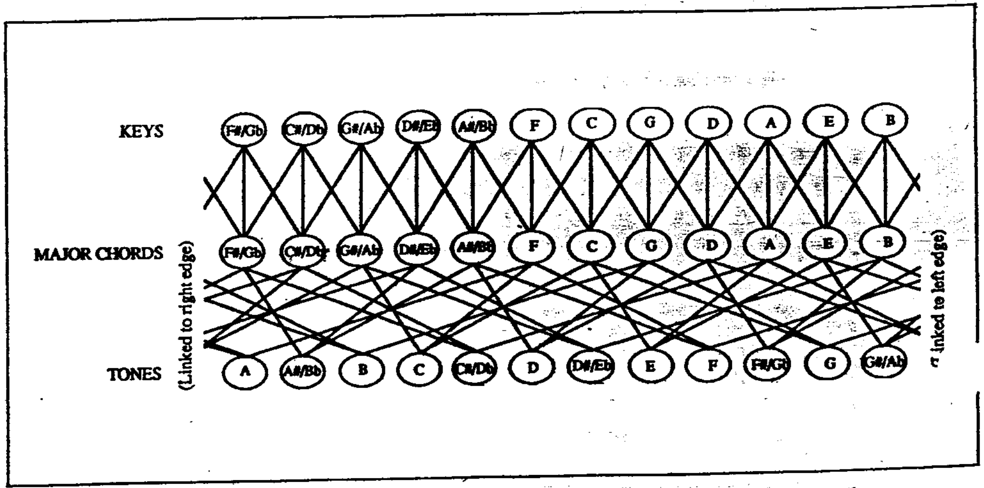
Figure 7. Anatomical model of the auditory cortex proposed by Galaburda and Pandya (1983) on the basis of cytoarchitectonic trends and connectivity patterns. See text for description. Cis, circular sulcus; KA, koniocortex; paAc, caudal parakoniocortex; paAlt, lateral parakoniocortex; paAr, rostral parakoniocortex; paI, parainsular area; pAll, periallocortex; Pro, proisocortex of the temporal pole; proA, prokoniocortex; reit, retroinsular area; STG, superior temporal gyrus; STP, supratemporal plane; STS, superior temporal sulcus; Tpt, temporoparietal isocortex; Ts_1, Ts_2, Ts_3, rostral superior temporal isocortex. (From Pandya and Yeterian 1985, Copyright by Plenum Publishing Corp. Reprinted by permission.)



suggests, all three streams were disrupted bilaterally in M.S. by lesions within the koniocortex and adjacent association areas; stages lying at the rostral and caudal extents of each stream, which have features of isocortex and proisocortex and interact with paralimbic and heteromodal association areas, were spared to varying degrees in each hemisphere.

The hierarchical organization of auditory information processing, as it pertains to harmony perception in music, has also been modeled as a parallel distributed system on computational grounds (Bharucha 1987; Fig. 8). Three stages of processing are identified: the tone level, whose units abstract octave-equivalent pitch information; the chord level, whose units abstract more complex harmonic features; and the key level, whose units abstract even more complex harmonic features. The connectivity patterns between different stages of processing can be learned by extensive exposure to music on the basis of a number of available neural net learning algorithms, including competitive learning (Rumelhart and Zipser 1986). Once learned, the network embodies hierarchical feature relationships that can achieve (1) bottom-up ac-

Figure 8. Computational model representing the schematic organization of tonal hierarchies in music. Letters designate scalar nodes within the key, chord, and tone levels of representation. Links between nodes reflect the membership of tones in chords and the membership of chords in keys. Sensory input activates the network from bottom-up, and associative influences on information processing are exerted top-down. (From Bharucha 1987, Copyright by the Cognitive Science Society. Reprinted by permission.)



tivation of representations of complex features such as tone chroma, chords, and keys form the sounded tones; and (2) top-down activation of representations of chords and tone chroma from the activated keys and, consequently, the observed facilitation of chord intonation judgments. In the context of Galaburda and Pandya's anatomical model of auditory cortex, and in view of the present findings, the neural substrate for top-down influences in musical priming may be distributed within the rostral and/or caudal reaches of auditory association cortex. Given this localization, and given that cognitive representations of tonal hierarchies in music are presumably learned, it is noteworthy that frequency-specific response plasticity during associative learning is a common property of neurons in the auditory association cortex of the cat (Diamond and Weinberger 1984; Weinberger and Diamond 1988); cells within both the primary and secondary fields manifest frequency-specific response plasticity, and they comprise a greater proportion of the sampled population in the latter than in the former.

The existence of cortical feature detectors that are selectively responsive to musical sounds (Katsuki et al. 1962) and to the harmonic composition of sounds (Winter and Funkenstein 1973) has been previously hypothesized on the basis of neuronal responses to complex spectra in monkeys. Response selectivity to acoustic transients in human speech sounds, including fundamental frequency, has been reported (Steinschneider et al. 1982). Neurons in the secondary fields appear to be slightly more selective for complex features than those in the primary field, but a considerable degree of variability in both response selectivity and response patterns has been observed at the single cell level (Manley and Mueller-Preuss 1978; Newman and Wollberg 1973). Unit responses to simultaneous pure tones cannot be predicted with certainty even on the basis of that unit's responses to each pure tone element (Katsuki et al. 1962). This variability has led Manley and Mueller-Preuss (1978) to postulate the existence of "detection networks" whose units respond in a probabilistic rather than deterministic fashion and collectively contribute to percept formation based on the conglomerate pattern of unit discharges.

That contextual influences on chord intonation judgments remained operative in M.S. even though the cortical association areas may have been deafferented from finely tuned, tonotopically organized frequency information raises the possibility that broadly tuned subcomponents of the thalamocortical auditory system mediate musical priming. From a cognitive perspective, broad tuning is not so much a lack of fine tuning as an attribute that is well-suited to a system that abstracts constancies among the spectral features of different acoustic signals and ignores small differences among elementary acoustic parameters that are insignificant to categorical processing and stimulus recognition. Given the observed interaction between incoming sensory information and prior acoustic events in musical priming, it is interesting that the responses of broadly tuned cortical auditory neurons to a given frequency are often influenced by the stimulus' relationship to a prior frequency (McKenna et al. 1989; Whitfield and Evans 1965).

Of course, these parallels between anatomical, physiological, and computational models of tonal information processing must remain conjectural in anticipation of further empirical justification.

METHODS

Neurological History and Examination

The details of the neurological history have been previously published by Mendez and Geehan (1988). Briefly, M.S. is a 30-year-old man who in 1980 and 1981 suffered focal infarcts in the distribution of the right and left middle cerebral arteries, respectively, presumably secondary to cardiogenic emboli. His first stroke presented with left hemiparesis and his second with transient deafness and persistent deficits involving both speech and environmental sound perception.

At the time of the present observations, M.S. reported a pervasive sense that "everything doesn't sound clear." He had great difficulty with figure-ground separation; for example, he stated he had trouble understanding speech when more than one person was talking, and when he rode in his car with the window down he had to greatly increase the loudness of his radio in order to apprehend the music being played. His wife, a speech therapist, noted that in the face of competing sounds his speech comprehension was aided by facing him and by talking somewhat slowly.

M.S. almost exclusively listened to music sold before his strokes. He denied that music unfamiliar to him had an unpleasant, dysacusic, or dissonant quality. He had difficulty understanding the lyrics of new songs, but he remembered the lyrics (and music) of familiar old favorites well enough to recognize them when he heard them on the radio. Although he spent fewer hours per day listening to music than before his strokes, he attributed this to a change in life-style rather than a lessened appreciation of music. He denied ongoing difficulty recognizing environmental sounds, and remarked that he was able to differentiate foreign car horns from American ones by their tonal quality.

The neurological examination was remarkable for dysprosodic speech, a spastic left hemiparesis, and left hemisensory loss to all tactile modalities. M.S. was alert, attentive, and highly motivated to perform well on all tasks. His speech was fluent and well-articulated and he followed spoken commands quickly and accurately. He distributed spatial attention normally. He was ambulatory with a cane and independent in most activities of daily life. Medications were limited to coumadin. The Edinburgh Inventory (Oldfield 1971) indicated mixed handedness with right predominance premorbidly (Laterality Quotient = +64, Decile R2).

M.S.'s premorbid level of musicality corresponds approximately to Grison's (1972) third level of musical culture, which lies in the middle of her classification scheme. M.S. took guitar lessons for 1 year at age 15; he was not trained in theory and had not played the guitar for several years before his first stroke. In his teens and early twenties, he was an avid listener of popular music; he accumulated a large record collection and listened attentively to music several hours a day.

Standard Audiometric and Psychometric Tests

Pure Tone Sensation Thresholds

Audiometry was performed with the subject seated in a sound-treated room (IAC). Acoustic stimuli were generated using a Grason-Stadler 16 Diagnostic Audiometer and presented through TDH-49 earphones. Detection thresholds for a 500 msec pure tone at 250–8000 Hz (Fig. 2) were established using the modified Hughson–Westlake procedure (Carhart and Jerger 1959).

Speech Perception

The Northwestern University List Number 6 (Tillman and Carhart 1966) was presented monaurally 35 dB above average pure tone threshold. A response accuracy of 90% and interear differences of 12% lie within the 95% confidence interval in normals (Thornton and Rafflin 1978). M.S. repeated 4/50 words accurately in left ear trials and 23/50 in right ear trials.

Psychometric Tests

The Boston Diagnostic Aphasia Examination (BDAE; Goodglass and Kaplan 1983), Boston Naming Test (Goodglass et al. 1983), Performance Subtest of the Revised Wechsler Adult Intelligence Scale (Wechsler 1981), and the Revised Wechsler Memory Scale (Wechsler 1987) were administered in standard fashion in a quiet room. During the presentation of the auditory-verbal subtests of the BDAE, the examiner took care to face away from the patient and speak at a customary rate and loudness.

Performance on the BDAE showed normal single word recognition (36/36 correct). However, M.S. made three paraphasic errors on the sentence repetition subtest, the nature of which are pertinent to the present experimental observations. He said "The bat leaps" for "The vat leaks," "The Chinese man had a rare emerald" for "The Chinese fan had a rare emerald," and "They heard him speak from the radio" for "They heard him speak on the radio." The subject substitution errors in the first two instances are far more frequent in the lexicon (Francis and Kučera 1978), suggesting that M.S. was activating cognitive representations of speech sounds via a "degraded" signal in an attempt to enhance his impoverished consonant–vowel phoneme discrimination. Consist with this interpretation is the observation that he substituted "leaps" for "leaks" to follow his error "bat," presumably to resolve the impending semantic incongruity, yet failed to correct the open-class substitution error "from," which did not alter the meaning of the sentence.

Performance on the Boston Naming Test was within normal limits (59/60 correct), the Performance IQ was 95, and the Memory Quotient was 91. On the W.M.S. he scored below the 50th percentile on the logical memory subtests and below the 25th percentile on the visual reproduction subtests; of course, performance on the former could have been affected by impaired speech discrimination.

Musical Priming Task

Stimuli and Apparatus

The stimuli used in the present experiment have been previously described by Bharucha and Stoeckig (1987). To summarize, chord spectra were synthesized using an Apple Macintosh microcomputer. Each prime chord and "in-tune" target chord is a major triad composed of the tonic (f_0), mediant ($2^{4/12} \times f_0$), and dominant ($2^{7/12} \times f_0$) across the equal-tempered scale. "Out-of-tune" targets are major triads that are mistuned by flattening the fifth degree by a fraction of a semitone (a frequency facto $2^{n/96}$). Chord components were sampled from a five tave range (65.41–4186.2 Hz; $A_4 = 440$ Hz) with an amplitude envelope in the frequency domain, such that the lowest and highest frequency components tapered off to the threshold of hearing as determined by the Fletcher and Munsen (1933) isoloudness curves. This procedure minimizes the salience of the lowest and highest spectral components (Shepard 1964; Krumhansl et al. 1982b). The waveform of each of the 15 component tones contained the first four harmonics with equal amplitudes.

The degree of association (harmonic relatedness) between the prime chord and target chord was varied in accordance with previous experimental analyses of harmonic relatedness judgments in normal subjects (Bharucha and Krumhansl 1983; Krumhansl et al. 1982a), which corroborate theoretical accounts of harmonic structure in Western music (for review see Piston 1978). Related pairs (e.g. A^{maj} and G^{maj}) shared parent keys, but they did not share component tones. Primes were 3 seconds in duration and targets were 4 seconds in duration.

M.S. performed one block of 96 trials. Trial presentations were internally randomized by the computer. Brief pauses lasting up to 1 min were interspersed approximately every 12 trials. Each of the 12 major chords occurred four times as a prime. The prime was follo equally by each of the following target conditions: in-tune/related, (2) in-tune/unrelated, (3) out-of-tune related, and (4) out-of-tune unrelated.

Stimuli were presented in free field in a quiet room using a Macintosh II and a Sansui A-707 amplifier and speaker system. Stimulus frequencies lay within the high end of the pure tone frequencies used to measure sensation levels, and stimulus intensity was adjusted to 60–65 dB using a Quest sound level meter. Response accuracy and reaction time measurements were internally synchronized with chord target onsets.

Procedure

A training session was conducted during which feedback was given after each practice trial. The amount of mistuning was increased by a frequency factor of $2^{1/96}$ until M.S. was to get five consecutive trials correct, but it became apparent that he had difficulty with "in-tune" targets, even at a factor of $2^{1/24}$, the maximal level of deviation from the equally tempered scale. We then tested him with single chords, that is, targets without primes, but he still did not reach criterion. In collecting data for statistical analysis, we decided to mistune the out-of-tune targets by a factor of $2^{1/24}$, the maximal level of mistuning.

M.S. was instructed to press one of two horizontally adjacent keys on the computer keyboard that were labeled "IN" and "OUT." He used his right hand, which was his preferred hand for fine motor control premorbidly. Power, rapid alternating movements, and proprioception at the fingers were normal, and there was no evidence of response disinhibition on informal "go/no-go" testing.

To begin each trial, the examiner pressed the space bar on the computer keyboard. The trial began with a 2 sec mask consisting of 16 tones of random pitch, followed by a 1 sec pause, then the prime chord, then the target (Fig. 1).

Error rates were analyzed using an analysis of variance with replications as the random factor.

Spectral Intonation Judgments in the Absence of a Prime

The stimuli, apparatus, and procedure were the same as described in the priming task, except that the mask was followed by a single chord, that is, an in-tune or out-of-tune major triad target without a prime. M.S. heard each target twice and performed one block of 48 trials.

Brainprints

MR scans were obtained using a Siemens 1.0 Tesla Magnetom MR System. Forty-two contiguous T1-weighted coronal sections were obtained in the coronal plane (TR = 40 msec; TE = 20 msec; slice thickness = 3.8 mm; gap = 0; acquisitions = 4; in-plane resolution 1.2 × 1.2 mm). In addition, two sets of 20 T2-weighted images were obtained (TR = 2500 msec; TE = 45, 90 msec; slice thickness = 6.0 mm; gap = 1.5 mm; acquisitions = 1; in-plane resolution 1.2 × 1.2 mm). T2 weightings are routinely more sensitive than T1 for infarct imaging, and the TR/TE 2500/45 weighting was particularly helpful for discriminating infarcted tissue from cerebrospinal fluid within ex vacuo change. Mid-sagittal scouts were obtained at the outset to align and mark the planes of section on an internalized grid and ruler and to measure the anterior–posterior distance between the frontal and occipital poles.

The computer reconstruction method used to flat map each hemisphere was originally detailed by Jouandet et al. (1989) and will be only briefly described here, along with minor modifications adopted for pathoanatomical analysis. Copies of each T1-weighted image were placed in a Beseler 23IIC photographic enlarger and projected onto paper; the pial surface of each hemisphere and the borders of the lesion (if it appeared on that slice) were traced. T2-weighted images were projected onto the closest corresponding T1-weighted tracings, from which they were 1.7–2.0 mm away, to further estimate the pial margins of necrotic tissue and the borders of abnormal signal not apparent on the T1-weighted images. Reference points demarcating individual gyri, intrasulcal vs. superficial cortex, surface structures lying within areas of abnormal MR signal, and surface structures overlying areas of abnormal signal in subjacent white matter were numbered sequentially from dorsomedial to ventromedial. The cortical convolutions on each section were labeled in accordance with the coronal atlases of Matsui and Hirano (1978) and Krieg (1963). The tracing was then entered into the computer via a Kurta IS/One digital graphics tablet for straightening, alignment, connection, scaling, and supplemental graphics. The surface areas of individual regions of interest were measured using a digitalized planimeter. Hemispheric volumes were measured by tracing the surface of each hemisphere on each slice using the planimeter, then tracing the ventricles (when necessary), subtracting the ventricular surface area from total hemisphere surface area, and multiplying by slice thickness.

Acknowledgments

This work was supported by N.I.H. 5-PO1-NS17778-08 (M.J.T.), O.N.R. N00014-89-J-3035 (M.J.T.), and N.S.F. BNS-8910778 (J.J.B.). The authors gratefully acknowledge Dr. Michael Gazzaniga, Dr. George Geehan, Dr. Marc Jouandet, Catherine Thomas, Nancy Epifano, William Loftus, Patrick Brown, Dr. John Weaver, Dr. Laurence Cromwell, Barbara Baker, Cari Iven, Robert Ferranti, Joan Thomson, and, especially, patient M.S. and his family.

REFERENCES

Aitkin, L. M. (1973). Medial geniculate body of the cat: Responses to tonal stimuli of neurons in medial division. *Journal of Neurophysiology, 36,* 275–283.

Aitkin, L. M., Irvine, D. R. F., & Webster, W. R. (1984). Central neural mechanisms of hearing. In I. Darian-Smith (Ed.), *Handbook of Physiology* (Section 1: The Nervous System, Volume III. Sensory Processes, Part 2). Bethesda: American Physiological Society Press, 675–737.

Allen, W. F. (1945). Effect of destroying three localized cerebral cortical areas for sound on correct conditioned differential responses of the dog's foreleg. *American Journal of Physiology, 144*, 415–428.

Benton, A. L. (1977). The amusias. In M. Critchley & R. A. Henson (Eds.), *Music and the Brain*. London: Heinemann, 378–397.

Bharucha, J. J. (1987). Music cognition and perceptual facilitation: A connectionist framework. *Music Perception, 5*, 1–30.

Bharucha, J. J., & Krumhansl, C. L. (1983). The representation of harmonic structure in music: Hierarchies of stability as a function of context. *Cognition, 13*, 63–102.

Bharucha, J. J., & Stoeckig, K. (1986). Reaction time and musical expectancy: Priming of chords. *Journal of Experimental Psychology: Human Perception and Performance, 12*, 403–410.

Bharucha, J. J., & Stoeckig, K. (1987). Priming of chords: Spreading activation or overlapping frequency spectra? *Perception and Psychophysics, 41*, 519–524.

Brugge, J. F., & Reale, R. A. (1985). Auditory cortex. In A. Peters & E. G. Jones (Eds.), *Cerebral Cortex* (Volume 4, Association and Auditory Cortices). NY: Plenum, 229–271.

Burton, H., & Jones, E. G. (1976). The posterior thalamic region and its cortical projection in New World and Old Word monkeys. *Journal of Comparative Neurology, 168*, 249–302.

Calford, M. B. (1983). The parcellation of the medial geniculate body of the cat defined by the auditory response properties of single units. *Journal of Neuroscience, 3*, 2350–2364.

Campbell, A. W. (1905). *Histological Studies on the Localisation of Cerebral Function*. Cambridge: Cambridge Univ. Press. Cited by Aitkin, Irvine, & Webster 1984.

Carhart, R., & Jerger, J. F. (1959). Preferred method for clinical determination of pure-tone thresholds. *Journal of Speech and Hearing Disorders, 24*, 330–345.

Celesia, G. G. (1976). Organization of auditory cortical areas in man. *Brain, 99*, 403–414.

Colombo, M., D'Amato, M. R., Rodman, H. R. & Gross, C. G. (1990). Auditory association cortex lesions impair auditory short-term memory in monkeys. *Science, 247*, 336–338.

Dewson, J. H. (1964). Speech sound discrimination by cats. *Science, 144*, 555–556.

Dewson, J. H., Pribram, K. H., & Lynch, J. C. (1969). Effects of ablations of temporal cortex upon speech sound discrimination in the monkey. *Experimental Neurology, 24*, 579–591.

Diamond, D. M., & Weinberger, N. M. (1984). Physiological plasticity of single neurons in auditory cortex of the cat during acquisition of the pupillary conditioned response. II. Secondary field (AII). *Behavioral Neuroscience, 98*, 189–210.

Earnest, M. P., Monroe, P. A., & Yarnell, P. R. (1977). Cortical deafness: Demonstration of the pathologic anatomy by CT scan. *Neurology, 27*, 1172–1175.

Fletcher, H., & Munsen, W. A. (1933). Loudness, its definition, measurement and calculation. *Journal of the Acoustical Society of America, 5*, 82–108.

Francis, W. N., & Kučera, H. (1982). *Frequency Analysis of English Usage: Lexicon and Grammar*. Boston: Houghton Mifflin.

Galaburda, A. M., & Pandya, D. N. (1983). The intrinsic architectonic and connectional organization of the superior temporal region of the rhesus monkey. *Journal of Comparative Neurology, 221*, 169–184.

Galaburda, A., & Sanides, F. (1980). Cytoarchitectonic organization of the human auditory cortex. *Journal of Comparative Neurology, 190*, 597–610.

Goodglass, H., & Kaplan, E. (1983). *The Assessment of Aphas and Related Disorders*, 2nd ed. Philadelphia: Lea & Feiberger.

Goodglass, H., Kaplan, E., & Weintraub, S. (1983). *Boston Naming Test*, 2nd ed. Philadelphia: Lea & Feiberger.

Grison, B. (1972). Une etude sur les alterations musicales au cours des lesions hemispheriques. These, Paris. Cited by Benton 1977.

Gross,, N. B., Lifschitz, W. S., & Anderson, D. J. (1974). The tonotopic organization of the auditory thalamus of the squirrel monkey (Saimiri sciureus). *Brain Research, 65*, 323–332.

Hari, R., Aittoniemi, K., Jarvinen, M. L., Katila, T., & Varpula, T. (1980). Auditory evoked transient and sustained magnetic fields of the human brain. Localization of neural generators. *Experimental Brain Research, 40*, 237–240.

Hartmann, W. M. (1988). Pitch perception and the segregation and integration of auditory entities. In G. M. Edelman, W. E. Gall, & W. M. Cowan (Eds.), *Auditory Function. Neurobiological Basis of Hearing*. New York: Wiley, 623–645.

Hecaen, H., & Albert, M. L. (1978). Disorders of auditory perception. In *Human Neuropsychology*. New York: Wiley, 248–276.

Heffner, H. E., & Heffner, R. S. (1986). Hearing loss in Japanese macaques following bilateral auditory cortex lesions. *Journal of Neurophysiology, 55*, 256–271.

Heffner, H. E., & Heffner, R. S. (1988). Effect of unilateral and bilateral auditory cortex lesions on the discrimination of vocalizations from Japanese macaques. *Journal of Neurophysiology, 56*, 683–701.

Helmholtz, H. (1863/1954). *On the Sensations of Tone* (A. J Ellis, translator). New York: Dover, 1954. Original public ton 1863.

Imig, T. J., Ruggero, M. A., Kitzes, L. M., Javel, E., & Brugge, J. F. (1977). Organization of auditory cortex in the owl monkey (Aotus trivirgatus). *Journal of Comparative Neurology, 171*, 111–128.

Jerger, J., Wekers, N. J., Sharbrough, F. W., & Jerger, S. (1969). Bilateral lesions of the temporal lobe. *Acta Otolaryngolica Supplement, 258*, 1–51.

Jones, E. G., & Burton, H. (1976). Areal differences in the laminar distribution of thalamic afferents in the cortical fields of the insular, parietal, and temporal regions of primates. *Journal of Comparative Neurology, 168*, 197–248.

Jouandet, M. L., Tramo, M. J., Herron, D. M., Hermann, A., Loftus, W. C., Bazell, J., & Gazzaniga, M. S. (1989). Brainprints: Computer-generated two-dimensional maps of the human cerebral cortex in vivo. *Journal of Cognitive Neuroscience, 1*, 88–117.

Katsuki, Y., Suga, N., & Kanno, Y. (1962). Neural mechanisms of the peripheral and central auditory system in monkeys. *Journal of the Acoustical Society of America, 34*, 1396–1410.

Krieg, W. J. S. (1963). *Connections of the Cerebral Cortex*. Evanston, IL: Brain Brooks.

Krumhansl, C. L., Bharucha, J., & Castellano, M. A. (1982a). Key distance effects on perceived harmonic structure in music. *Perception and Psychophysics, 32*, 96–108.

Krumhansl, C. L., Bharucha, J. J., Kessler, E. J. (1982b). Perceived harmonic structure of chords in three related musical keys. *Journal of Experimental Psychology: Human Perception and Performance, 8*, 24–36.

Lauter, J. L., Hersovitch, P., Formby, C., & Raichle, M. E. (1985). Tonotopic organization in the human auditory cortex revealed by positron emission tomography. *Hearing Research, 20*, 199–205.

Levelt, W. J. M., van de Geer, J. P., & Plomp, R. (1966). Triadic comparisons of musical intervals. *British Journal of Mathematical and Statistical Psychology, 19*, 163–179.

Manley, J. A., & Meuller-Preuss, P. (1978). Response variability in the mammalian auditory cortex: An objection to feature detection? *Federation Proceedings, 37*, 2355–2359.

Marin, O. S. M. (1982). Neurological aspects of musical perception and performance. In D. Deutsch (Ed.), *The Psychology of Music*. Orlando: Academic Press, 453–477.

Marin, O. S. M., & Barnes, S. (1985). Evidence for higher cognitive structures in music perception. Paper presented at the Fifth Workshop on Physical and Neuropsychological Foundations of Music, Ossiach, Austria.

Matsui, T., & Hirano, A. (1978). *An Atlas of the Human Brain for Computerized Tomography*. New York: Igaku-Shoin Medical Publishers, Inc.

McKenna, T. M., Weinberger, N. M., & Diamond, D. M. (1989). Responses of single auditory cortical neurons to tone sequences. *Brain Research, 481*, 142–153.

Mendez, M. F., & Geehan, G. R. (1988). Cortical auditory disorders: Clinical and psychoacoustic features. *Journal of Neurology, Neurosurgery, and Psychiatry, 51*, 1–9.

Merzenich, M. M., & Brugge, J. F. (1973). Representation of the cochlear partition on the superior temporal plane of the macaque monkey. *Brain Research, 50*, 275–296.

Mesulam, M.-M., & Pandya, D. N. (1973). The projections of the medial geniculate complex within the Sylvian fissure of the rhesus monkey. *Brain Research, 60*, 315–333.

Naeser, M. A., Helm-Estabrooks, N., Haas, G., Auerbach, S., & Srinivasan, M. (1987). Relationship between lesion extent in "Wernicke's area" on computed tomographic scan and predicting recovery of comprehension in Wernicke's aphasia. *Archives of Neurology, 44*, 73–82.

Neff, W. D., Diamond, I. T., & Casseday, J. H. (1975). Behavioral studies of auditory discrimination: Central nervous system. In W. D. Keidel & W. D. Neff (Eds.), *Handbook of Sensory Physiology. Auditory System, Physiology (CNS)* (Vol. 5, Part 2). Berlin: Springer-Verlag, 307–400.

Newman, J. D., & Wollberg, Z. (1973). Multiple coding of species-specific vocalizations in the auditory cortex of squirrel monkeys. *Brain Research, 54*, 287–304.

Oldfield, R. C. (1971). The assessment and analysis of handedness: The Edinburgh Inventory. *Neuropsychologia, 9*, 97–113.

Pandya, D. N., & Yeterian, E. H. (1985). Architecture and connections of cortical association areas. In A. Peters & E. G. Jones (Eds.), *Cerebral Cortex* (Volume 4, Association and Auditory Cortices). New York: Plenum, 3–61.

Pfingst, B. E., O'Connor, T. A., & Miller, J. M. (1977). Response plasticity of neurons in auditory cortex of the rhesus monkey. *Experimental Brain Research, 29*, 393–404.

Phillips, D. P., & Irvine, D. R. F. (1979). Acoustic input to single neurons in pulvinar-posterior complex of cat thalamus. *Journal of Neurophysiology, 42*, 123–136.

Piston, W. (1978). *Harmony*, 4th ed. Revised and expanded by M. DeVoto. New York: Norton.

Romani, G. L., Williamson, S. J., & Kaufman, L. (1982). Tonotopic organization of the human cerebral cortex. *Science, 216*, 1339–1340.

Rumelhart, D. E., & Zipser, P. (1986). Feature discovery by competitive learning. In D. E. Rumelhart & J. L. McClelland (Eds.), *Parallel Distributed Processing: Explorations in the Microstructure of Cognition*, Vol. 1. Cambridge: MIT Press, 151–193.

Schouten, J. J. (1938). The perception of subjective tones. *Konnkliijke Nederlandse Akademie van Wetenschappen Proceedings, 41*, 1086–1093.

Selnes, O. A., Niccum, N., Knopman, D. S., & Rubens, A. B. (1984). Recovery of single word comprehension: CT-scan correlates. *Brain and Language, 21*, 72–84.

Shepard, R. N. (1964). Circularity in judgments of relative pitch. *Journal of the Acoustical Society of America, 36*, 2346–2353.

Steinschneider, M., Arezzo, J., & Vaughan, H. G. (1982). Speech evoked activity in the auditory radiations and cortex of the awake monkey. *Brain Research, 252*, 353–365.

Symmes, D. (1966). Discrimination of intermittent noise by macaques following lesions of the temporal lobe. *Experimental Neurology, 16*, 201–214.

Terhardt, E. (1974). Pitch, consonance, and harmony. *Journal of the Acoustical Society of America, 55*, 1061–1069.

Terhardt, E. (1984). The concept of musical consonance: A link between music and psychoacoustics. *Music Perception, 1*, 276–295.

Thornton, A., & Rafflin, M. (1978). Speech discrimination scores modeled as a binomial variable. *Journal of Speech and Hearing Research, 21*, 507–518.

Tilman, T., & Carhart, R. (1966). An expanded test for speech discrimination utilizing CNC monosyllabic words: Northwestern University Auditory Test #6. United States Air Force School of Aerospace Medicine Report, 55–66.

Tramo, M. J., & Bharucha, J. J. (1990). Musical priming by the right hemisphere post-callosotomy. Submitted.

Trojanowski, J. Q., & Jacobson, S. (1975). A combined horseradish peroxidase-autoradiographic investigation of reciprocal connections between superior temporal gyrus and pulvinar in squirrel monkey brain. *Brain Research, 85*, 347–353.

Vaadia, E., Gottlieb, Y., & Abeles, M. (1982). Single-unit activity related to sensorimotor association in auditory cortex of a monkey. *Journal of Neurophysiology, 48*, 1201–1213.

Vignolo, L. A. (1969). Auditory agnosia: A review and report of recent evidence. In A. L. Benton (Ed.), *Contributions to Clinical Neuropsychology*. Chicago: Aldine, 172–208.

Wechsler, D. (1981). *WAIS-R Manual: Wechsler Adult Intelligence Scale—Revised*. New York: Psychological Corporation.

Wechsler, D. (1987). *Wechsler Memory Scale—Revised*. San Antonio: Psychological Corporation.

Weinberger, N. M., & Diamond, D. M. (1988). Dynamic modulation of the auditory system by associative learning. In G. M. Edelman, W. E. Gall, & W. M. Cowan (Eds.), *Auditory Function. Neurobiological Basis of Hearing*. New York: Wiley, 485–512.

Wertheim, N. (1969). The amusias. In P. J. Vinken & G. W. Bruyn (Eds.), *Handbook of Clinical Neurology*, Vol. 4. Amsterdam: North Holland, 195–205.

Whitfield, I. C. (1980). Auditory cortex and the pitch of complex tones. *Journal of the Acoustical Society of America, 67*, 644–647.

Whitfield, I. C. (1985). The role of the auditory cortex in behavior. In A. Peters & E. G. Jones (Eds.), *Cerebral Cortex* (Volume 4, Association and Auditory Cortices). New York: Plenum, 329–349.

Whitfield, I. C., & Evans, E. F. (1965). Responses of auditory cortical neurons to stimuli of changing frequency. *Journal of Neurophysiology, 28*, 655–672.

Winter, P., & Funkenstein, H. H. (1973). The effect of species-specific vocalization on the discharge of auditory cortical cells in the awake squirrel monkey (Saimiri sciureus). *Experimental Brain Research, 18*, 489–504.

Zatorre, R. J. (1988). Pitch perception of complex tones and human temporal-lobe function. *Journal of the Acoustical Society of America, 84*, 566–572.

APPENDIX
Glossary of Abbreviations for Brainprint Regions of Interest

A	Amygdala
Ang	Angular gyrus
BF	Basal forebrain
Ca	Precentral gyrus
Ci	Cingulate gyrus
Cp	Postcentral gyrus
Cu	Cuneus
Fiop	Inferior frontal gyrus, pars opercularis
Fiorb	Inferior frontal gyrus, pars orbitalis
Fit	Inferior frontal gyrus, pars triangularis
Fm	Middle frontal gyrus
Fs	Superior frontal gyrus
Fus	Fusiform gyrus
H	Hippocampal region, including the parahippocampal gyrus
Hg	Transverse gyrus(i) of Heschl
Lg	Lingual gyrus
Oi	Inferior lateral occipital gyrus
Om	Middle lateral occipital gyrus
Or	Orbitofrontal gyri
Os	Superior lateral occipital gyrus
Pc	Precuneus
Ps	Superior parietal lobule
R	Rectal gyrus
Sm	Supramarginal gyrus
Ti	Inferior temporal gyrus
Tm	Middle temporal gyrus
TP	Temporal pole
Ts	Superior temporal gyrus
U	Uncus