Neurobiology Relevant to Some Central Auditory Processing Disorders

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Abstract

This paper explores how some recent advances in auditory neuroscience might help us understand some of the perceptual sequelae seen in listeners with acquired pathologies of the central auditory nervous system. We achieve this by trying to understand the links between the spectral/temporal structure of a sound, the neural mechanisms that shape that sound's representation in the nervous system, and the perceptual consequences of having that neural representation disrupted by pathological processes. The neural code or representation of a sound may depend on which cells are active, on how many cells are active, and on the timing of the spike activity in those cells. We see examples of this linking between the structure of the stimulus, the nature of the neural code, and the relevant auditory perceptual dimension, in studies of spatial hearing, signal/noise extraction, and pure word deafness.

Introduction

The purpose of this paper is to survey some recent advances in central auditory neurophysiology that may be relevant to understanding the sensory deficits shown by human listeners with some acquired pathologies of the central auditory nervous system. The account that follows is necessarily both selective and simplified, since an exhaustive treatment might legitimately fill a volume (eg., Keith, 1977; Pinheiro & Musiek, 1985). It is for this reason that we have refrained from discussing recent advances in the use of brainstem auditory evoked potentials, and in studies of auditory brainstem neurochemistry and olivocochlear function. Our more limited goal then, is to explore some of the acoustic sensory dimensions in which the auditory neuroscience and central auditory dysfunction literatures are congruent in pointing toward some recognizable principles of auditory function. An understanding of these principles might provide impetus for refinement of central auditory testing procedures in the audiology or neuro-otology clinic, and for the design of rehabilitative or compensatory therapies.

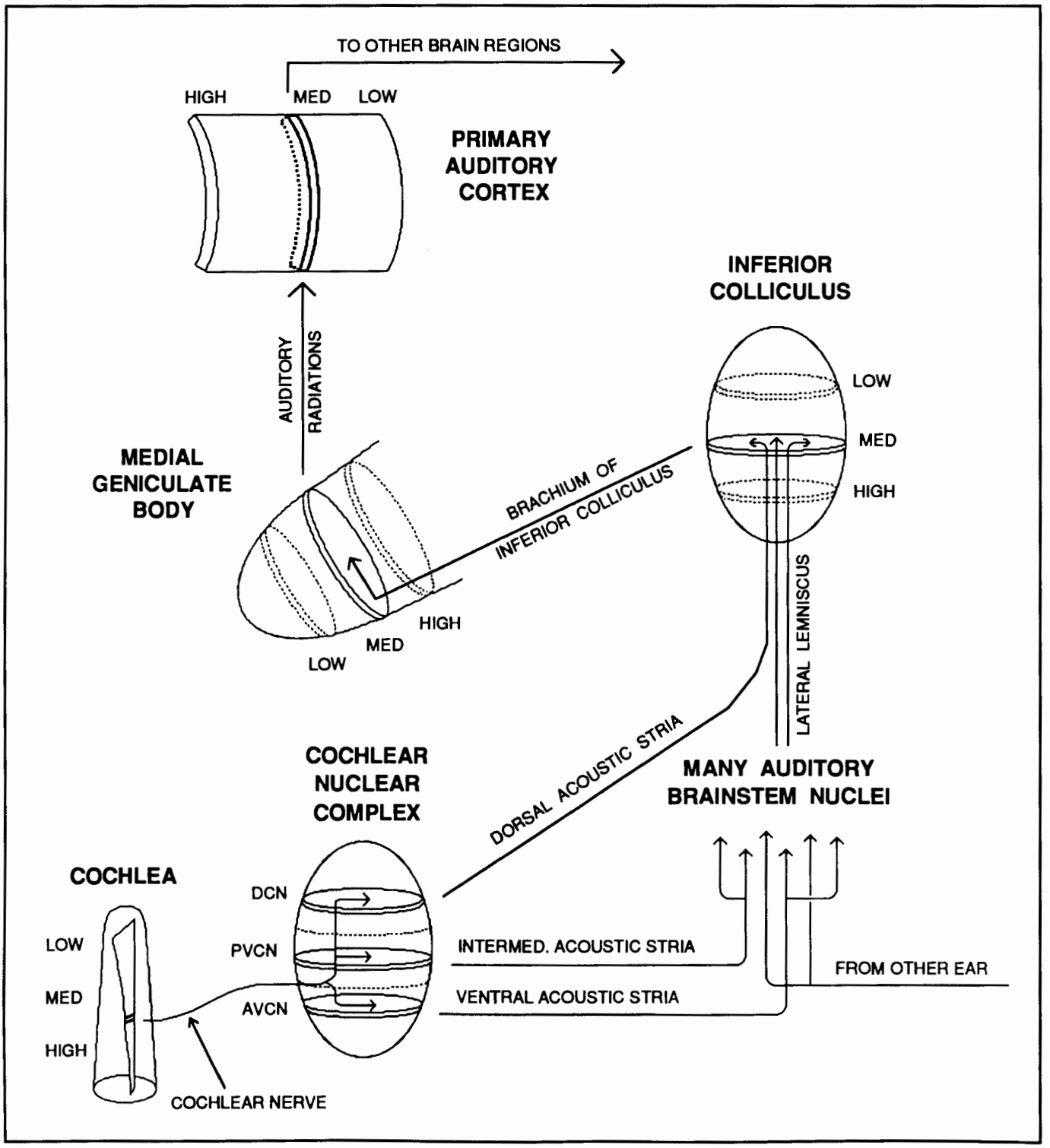
We begin by briefly sketching the organization of the central auditory pathway, since this will provide a framework for what follows. This sketch is a strictly *functional* one, and more detailed accounts of the neuroanatomy and neurophysiology can be found elsewhere (Aitkin et al., 1984; Moller, 1983; Phillips, 1988b). We then turn to examine the consequences of convergence of input from the two ears in the auditory brainstem, to the implications of that convergence for the processing of acoustic spatial information, and to the behavioral instantiations of that processing witnessed in studies of pathological listeners. Next, we look at the neural mechanisms that shape the representation of spectrally and temporally complex sounds in the auditory system. This will lead us to speculate on the contribution of the afferent auditory system to the processing of speech sounds, and in turn, to the correspondence between the conclusions on this issue drawn from the animal neurophysiological studies and those derived from studies of normal and impaired human listeners.

Functional Organization of the Auditory Nervous System

Information about the composition of a sound enters the central auditory nervous system by way of the auditory nerve, which consists in some tens of thousands of fibers (axons of spiral ganglion cells). Each of these fibers links a single inner cochlear hair cell with neurons of the cochlear nuclear complex of the auditory brainstem (Figure 1, bottom left). The driving force on the spike activity of an auditory nerve fiber lies in the motion of the basilar membrane at the site of the hair cell innervating the fiber. The basilar membrane motion at that locus is shaped by the spectral content of the sound, the passive mechanics of the membrane, and the activity of outer cochlear hair cells at that locus (e.g., Pickles, 1985). The response of the hair cell at the stimulated site consists in an a.c. receptor potential that mimics the stimulus waveform at low frequencies, and an increasingly rectified depolarization (d.c.) potential at higher frequencies (Russell & Sellick, 1978). The amplitude of the depolarization response is a saturating function of tone level.

Unidirectional elevations of the basilar membrane evoke the depolarization response, which in turn leads to neurotransmitter release and thus spike activity in the auditory nerve fibers contacting the hair cell. For low frequency signals, to which the hair cell membrane potential responds with a stimulus-mimicking oscillation, this brings about a synchrony of

Figure 1. Highly stylized and simplified representation of the tonotopic organization of the central auditory pathway. Each point along the cochlear partition is linked by cochlear nerve fibers to a sheet of neurons in each of three divisions of the cochlear nuclear complex. Neurons in these nuclei direct their axonal outputs onto numerous nuclei on both sides of the auditory brain stem. The ascending outputs of these nuclei in turn converge, but in a tonotopically constrained fashion, on the single frequency representation of the inferior colliculus. The inferior colliculus (on each side of the brain) directs its ascending axons primarily upon the ipsilateral medial geniculate body, which in turn projects upon the ipsilateral primary auditory cortex. Note that what begins as a "point" representation of tone frequency in the cochlea is transformed into a parallel series of "sheet" representations in the auditory brainstem, and finally a "strip" representation in the primary auditory cortex.

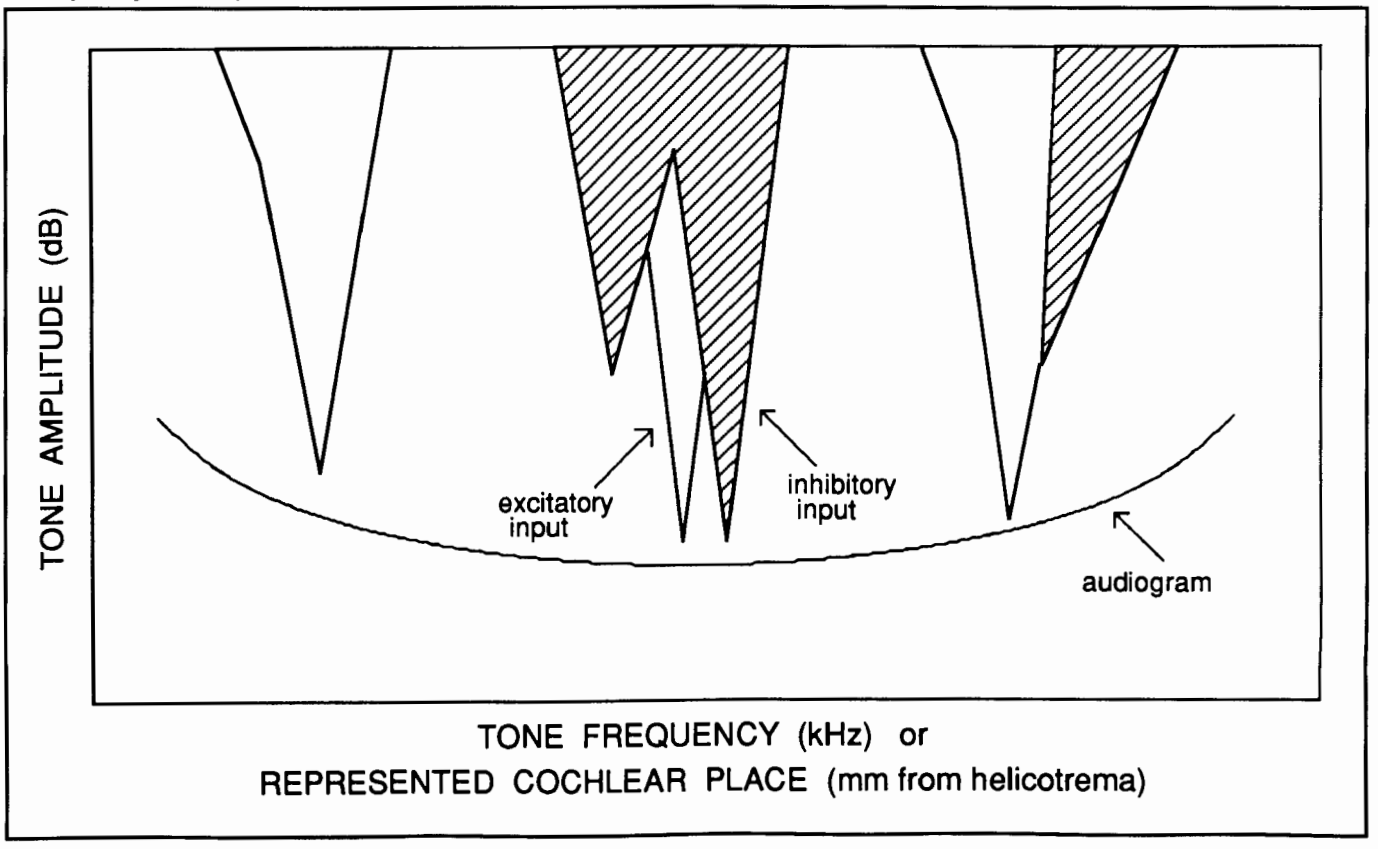


transmitter release with basilar membrane elevation. It follows from this that the instantaneous probability of nerve fiber discharge has a time structure that resembles a partially rectified time waveform of the stimulus (e.g., Rose, 1973). This spike-time code for stimulus frequency content fails when the signal periodicity is too high in frequency to evoke a significant a.c. response from the hair cell (Palmer & Russell, 1986). Above these frequencies (about 3-4 kHz), where the relevant hair cell response is a d.c. level of depolarization, neurotransmitter release is more nearly continuous and auditory nerve fibers respond with spike rates proportional to hair cell response magnitude, and without stimulus-bound temporal periodicities.

The fidelity of this phase-locking neural code in any single cochlear nerve fiber is limited by a second means in which cochlear output represents signal frequency: in the "tuning curve" of each fiber. Each afferent cochlear fiber is narrowly tuned to a characteristic frequency (CF) to which it is most sensitive (Figure 2, left-most neuron). Departures of tone frequency from a neuron's CF evoke excitatory responses with significantly higher threshold sound pressures. A plot of threshold sound pressure as a function of tone frequency thus has a narrow V-shape, at least for sound pressure levels less than about 80 dB. The response *rate* of a neuron at any given tone frequency is usually a monotonic, saturating function of tone level (Kiang et al., 1965). This shape likely reflects both the input-output properties of the hair cell and the organization of synaptic contacts between the hair cell and the nerve fiber. In most instances, the dynamic portions of these functions are less than about 40 dB wide (Liberman, 1978; Palmer & Evans, 1980).

Cochlear output thus consists in an array of fibers each of which, either by means of the periodicities in its spike dis-

Figure 2. Schematic depiction of the excitatory/inhibitory response areas of some central auditory neurons. Neurons with these properties are seen as far caudally as the cochlear nuclear complex, and as far rostrally as the auditory cortex. Each neuron possesses a central excitatory response area which may be flanked on one or both sides by inhibitory response areas. Studied with broadband stimuli, these neurons would show quite different responses; the differences would reflect the overlap between the stimulus spectra and the disposition and sensitivity of the excitatory and inhibitory inputs. The neuron on the left would respond to any broadband signal that contained sufficent energy within the cell's tuning curve. The neuron on the right would show low-pass sensitivity to bandwidth for moderately intense sounds. The middle neuron would probably be unresponsive to broadband signals, and would respond to tonal signals only within a narrow window of frequency and amplitude.



charges, or in its firing rate, indicates the presence of stimulus energy close to its CF.

The pattern of activity across the cochlear nerve fiber population is capable of indicating the spectral content of the stimulus at the tympanum, and in the case of temporally varying sounds, one can imagine shifting patterns of activity across the fiber population following the time course of the stimulus (see Carney & Geisler, 1986; Delgutte, 1980; Delgutte & Kiang, 1984; Kiang et al., 1979; Sachs, 1984). This stimulus representation is obviously limited by the transmission properties of the middle ear, by the non-independence of basilar membrane motion at adjacent sites, and by the tendency of cochlear nerve fibers to firing rate saturation.

Each auditory nerve fiber penetrates the cochlear nucleus, bifurcates, and sends axon branches to each of three divisions of the nuclear complex (anteroventral [AVCN], posteroventral [PVCN], and dorsal [DCN] compartments: see Figure 1). The terminal axon fields deriving from any single cochlear site are disposed in sheet-like fashion, and the "sheets" of fiber input are topographically arrayed according to the cochlear place from which they originate. The result is that each division of the cochlear nucleus contains a complete and orderly representation of cochlear place, and therefore of the audible frequency range. This divergent projection of the auditory nerve upon the cochlear nuclear complex initiates a system of *parallel processing*. By this we mean that each division of the cochlear nucleus may be independently capable of carrying out its own transformations of the cochlear nerve input, and may direct its axonal outputs to separate target nuclei.

Neurons of the medial superior olivary nucleus (MSO) receive axonal connections from neurons in both the left and right AVCNs. The cochlear nerve input to these AVCN cells, and their own biophysical properties, are specialized in fashions that preserve the fine temporal pattern of spikes in the auditory nerve (Brugge & Geisler, 1978; Wu & Oertel, 1984). In particular, these AVCN neurons receive input from very few auditory nerve fibers, but do so by way of synaptic connections termed "end-bulbs of Held." These are very large synaptic endings whose thick, finger-like extensions partially encapsulate the recipient cell soma. In addition, these cochlear nucleus cells have membranes with a low input-resistance. This means that a particularly large synaptic current is required to evoke a post-synaptic spike response, and such a current is likely to be generated only by the synchronous activation of the multiple contacts provided by the end-bulb. It is the conjunction of these properties that makes the auditory nerve fiber input to these AVCN cells the dominant one, and which makes possible a precisely timed "spike-in/spike-out" transmission of information to more central brain regions. This has the consequence that MSO neurons are able to compare the phase relationships of excitatory responses in their two monaural inputs. By this means, the binaural MSO neurons are able to compare the phase relationships of low frequency signals at the two tympanic membranes.

Other neurons in the ventral cochlear nucleus direct their axons upon the ipsilateral lateral superior olivary nucleus (LSO) and contralaterally upon the medial nucleus of the trapezoid body (MNTB). The MNTB cells in turn project to the LSO of the same side. One principal function of the MNTB is to receive the excitatory input from the cochlear nucleus and to express that input as an inhibitory influence on the target LSO cells. As a result, each LSO neuron is characterized by contralateral-inhibitory/ ipsilateral-excitatory pattern of binaural input. As in the case of the MSO, these inputs are constrained tonotopically; moreover, the MNTB relay is made up of a calyx-like synapse that ensures rapid transmission (Morest, 1973). The strengths of the excitatory and inhibitory inputs to the LSO cells are each a sensitive function of tympanic sound pressure level. The net result for the LSO is that it contains cells receiving intensity-sensitive excitatory and inhibitory inputs of similar CF from the ears, and by these means, those cells are able to encode differences in the amplitudes of signals at the ears.

Within the dorsal cochlear nucleus (DCN), inhibitory circuitry developed by local interneurons confers complex excitatory/inhibitory "response areas" on the output cells (Shofner & Young, 1985; Young & Brownell, 1976: see Figure 2). By "response area", we mean the total frequency-intensity domain within which a tonal stimulus is able to influence the spike discharge rate of a neuron. The domain of tone frequency-intensity conjunctions that excites cochlear nerve fibers — all of those inside the limits of the frequency tuning curve — is, in the DCN, dramatically shaped by inhibitory inputs from neurons of adjacent CF. In some cases, the excitatory tone response area of a DCN neuron is a narrow sliver or "island" in a much broader stimulus domain of those providing inhibitory inputs (e.g., Figure 2, middle). This imposes not only narrow frequency tuning, but in many cases, an apparent tuning to tone pulse amplitude. The same imposition of flanking inhibitory stimulus domains around the excitatory one centered at CF provides a mechanism conferring sensitivity to the spectral bandwidth of acoustic signals. This follows from the fact that broadband signals may simultaneously activate both excitatory and inhibitory inputs to a DCN neuron: its spike output, in the simplest cases, will reflect the net balance of the excitatory and inhibitory influences. Again, this processing takes place within a tonotopic framework: while the range of tone frequencies (cochlear places) effective in evoking excitatory or inhibitory responses may be significantly greater than the width of a cochlear fiber tuning curve, those ranges are still narrow by comparison with the whole audiogram (Figure 2).

The axonal outputs of the DCN, MSO, LSO, and a number of other brainstem nuclei to which cochlear output is indirectly disseminated, ultimately converge on the inferior colliculus (Figure 1), principally through the dorsal acoustic stria (DCN) or lateral lemniscus (MSO, LSO). The MSO projections are almost exclusively ipsilateral; the DCN efferents are exclusively crossed, and those of the LSO predominantly contralateral. Most of these inputs terminate on neurons of the central nucleus of the inferior colliculus (ICC), which has a three-dimensional structure in which cells of similar CF are disposed in sheets orthogonal to a single dorso- ventral tonotopic axis (Figure 1, upper right). Within this single tonotopic framework, the convergence of ascending input is incomplete, so that within any single iso-CF neuronal sheet, local sectors are apparently dominated by inputs from subsets of the brainstem nuclei that send axons to that sheet (Semple & Aitkin, 1981). Nevertheless, the convergence of input that does occur at this locus exemplifies a second feature of the ascending auditory system: the serial, *heirarchical processing* principle. By this means, some neurons receiving monosynaptic input from the DCN, and which show DCN-like properties in their responses to contralateral stimuli, also receive input from the LSO, and are therefore binaurally influenced.

Neurons of the ICC direct their ascending axons to the ventral division of the medial geniculate body (MGv). This thalamic nucleus, like the ICC, is a laminated structure with a single tonotopic representation; the ICC-MGv projection might usefully be thought of as a sheet-to-sheet one (Figure 1). The MGv is surrounded by a number of related nuclei, and it is thought that these might be the targets of ascending axons from cell groups surrounding the ICC (Calford & Aitkin, 1983). The ICC also provides input to surrounding nuclei, notably the external nucleus of the inferior colliculus. This nucleus is one of a number that provides acoustic input to structures mediating eye and head movements to auditory targets.

The MGv projects directly upon the ipsilateral primary auditory cortex (AI), and less densely upon a number of adjacent cortical fields which are interconnected with AI on a tonotopic basis (Brugge & Reale, 1985; Imig & Reale, 1980;). The various cortical fields are distinguished by their cytoarchitecture, their afferent and efferent connectivities, and by their spatial distributions of neural CFs (e.g., Brugge & Reale, 1985; Seldon, 1981a,b). In the primary auditory cortex, and in some adjacent fields, tonotopic organization is expressed in the form of iso-CF strips, and its thalamic afferent projection is thus a convergent, sheet-to-strip one (Figure 1, top left). The convergence of input onto these cortical strips is far from undifferentiated: within an isofrequency strip, there are local territories showing physiologies that reflect the activity of only small subsets of the potentially available inputs (Phillips, 1988c). The primary auditory cortex might be conceptualized as having a continuous strip-like representation of cochlear place, overlaid on which is a mosaic of discontinuous, partially overlapping territories specialized for one or another form of stimulus processing.

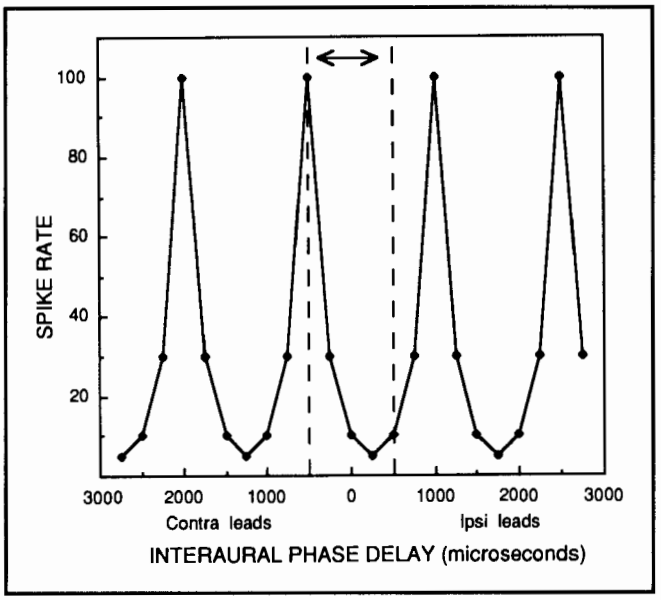
The tonotopic organization of AI (and some other cortical fields) is the cortical manifestation of the place code developed in the auditory periphery, and it likely extends to humans (Romani et al., 1982). The tonotopic constraints that characterize the divergent and convergent connectivities of the central auditory system seem necessarily to imply that, at least to the level of AI, information processing occurs along frequency-specific channels that are preservations and extensions of those formed at the periphery. The fact that there are multiple cortical fields, probably with relatively independent thalamic connectivities, is a prime example of parallel processing in the auditory system. It is likely that these various auditory cortical fields will make different contributions to the hearing process. We know this to be true in the bat (Suga, 1982). In the cat, we know that neurons "tuned" jointly to tone frequency and tone amplitude are in the minority in AI, but are in the majority in an adjacent tonotopic field (Phillips & Orman, 1984). Likewise, in contrast to AI in which most neurons are narrowly tuned to tone frequency, neurons in one secondary auditory field appear to be broadly tuned to frequency (Schreiner & Cynader, 1984). Serial connectivity, described above for the brainstem, is equally evident in the cortex in the connections between the cortical fields (Brugge & Reale, 1985), and perhaps in their outputs to more remote non-auditory structures (Irvine & Phillips, 1982).

Binaural Processing

Binaural stimulus processing is of special interest because it is binaural stimulus information that provides to the listener some important cues to the spatial location of a sound source. This cue information is broadly divisible into interaural time disparities, which derive largely from differences in the travel distance of the sound to the two ears, and interaural intensity differences, which are brought about by the sound- shadowing effect of the head and the directional properties of the pinnae (Phillips & Brugge, 1985). The temporal cue is itself separable into an arrival time difference, present for all signal frequencies, and the on- going phase difference, which can be encoded only for signals with frequencies lower than the upper limit of neural phase-locking. These two time cues are likely coded by different neural populations.

Interaural phase differences are encoded by MSO neurons which, we recall, receive bilateral input from AVCN neurons specialized for faithful transmission of spike cadences from the auditory nerve. In practice, it has been difficult to record from MSO cells directly, and our best evidence on the coding of interaural phase disparities has come from studies of nuclei

Figure 3. Idealized impression of the responses of a single neuron to variations in the interaural delay of continuous sinusoids of the same frequency and amplitude at the two ears. The curve plots the spike rate of a neuron as a function of interaural delay, expressed in microseconds. Note that the response rate is a periodic function of interaural delay, and that the period of the function is 1.5 ms. (This means that the frequency of the sinusoids presented to the ears was 666 Hz.) Dashed vertical lines linked by the arrows roughly indicate the behaviorally relevant range of delays, with the qualification that the width of this range depends on head size. Within this range, response rate is greatest when the contralateral phase leads the ipsilateral.



to which the MSO directly or indirectly projects (Brugge et al., 1970; Yin & Kuwada, 1983). Those studies revealed that, when studied with low-frequency tonal stimuli, each monaural input to the binaural cells consists in interlaced, alternating excitatory half-periods and inhibitory half-periods. The excitatory half-periods may be thought of as preservations of the phase-locking of spike discharges seen in the auditory nerve. The inhibitory half-periods are inserted by central auditory synaptic connections. The spike output of the binaural neuron reflects a coincidence detection of the times of arrival of the excitatory/inhibitory afferent volleys in the monaural inputs. If continuous sinusoids are presented to the ears, and only the interaural phase delay is varied, then the response rate of the binaural neuron is a periodic, cyclical function of interaural delay, with the period of the response cycle being equal to that of the carrier tone: it reflects a cycle-by-cycle comparison of the phases of the monaural inputs, and therefore of the stimuli at the two ears (Figure 3). Note that once the convergence of input has taken place, there is no need for more rostral auditory nuclei to preserve the spike timing seen in the monaural inputs to the binaural comparator. The comparison itself is one of the timing of excitatory and inhibitory neural events, but the resulting neural representation of interaural disparity (and therefore sound source azimuth) need only be in the firing rates of the relevant neurons. In this respect, forebrain neurons whose spike rates are sensitive to interaural phase delays often do not demonstrate phase-locking of their spike discharges (Brugge & Merzenich, 1973).

Because the interaural phase comparison is performed on a cycle-by- cycle basis for as long as the stimuli are present at the ears, the interaural delay function can be extended for many periods of the delay favoring either ear (Figure 3). The *behaviorally relevant* range of delays is probably only a few hundred microseconds wide (the range of delays indicated by the arrows and dashed lines in Figure 3). Within this range of delays, neural firing rates are usually maximal when the delay favors the contralateral ear, and minimal when the delay favors the ipsilateral ear. These observations carry a special significance because they suggest that the interaural delays, and therefore the sound source azimuths, evoking the greatest response rates are associated with *contralateral auditory* space. This is because a sound generating disparities favoring the contralateral ear can only be located in contralateral space. Moreover, the neuron's response rate is often most sensitive to changes in interaural delay over ranges near zero microseconds, i.e., close to those associated with midline azimuths. Interestingly, the interaural time cue is itself most precise in specifying sound source azimuth for sources within about 45 degrees of the midline (see Phillips & Brugge, 1985).

The interaural intensity difference is a cue for sound source azimuth for signals with wavelengths shorter than the interaural distance. Recall that LSO neurons receive an excitatory input from the ipsilateral ear, and an inhibitory input of similar CF from the contralateral ear. Each of these inputs has a rate response which is a sensitive function of tympanic sound pressure. The LSO cells are thus able to encode interaural intensity differences, since their firing rates will reflect the net balance of the excitatory and inhibitory inputs, which in turn reflects the relative levels of the stimuli at the two ears.

Rostral to the crossed output of the LSO, the pattern of binaural input obviously reverses to a contralateral-excitatory/ipsilateral- inhibitory one. The spike rates of these cells are sigmoidal functions of interaural disparity, with maximal responses occurring when the intensity difference favors the contralateral ear (Figure 4). The dynamic portion of the sigmoidal function typically occurs over ranges near zero dB or slightly favoring the contralateral ear (Phillips & Irvine, 1981). As in the case of interaural time differences, the interaural intensity disparity is most precise in specifying sound source azimuth for sources near the midline; we again see that neural sensitivity to small differences in cue magnitude is greatest where small changes in cue magnitude themselves provide the most accurate spatial information. Because of the well-known

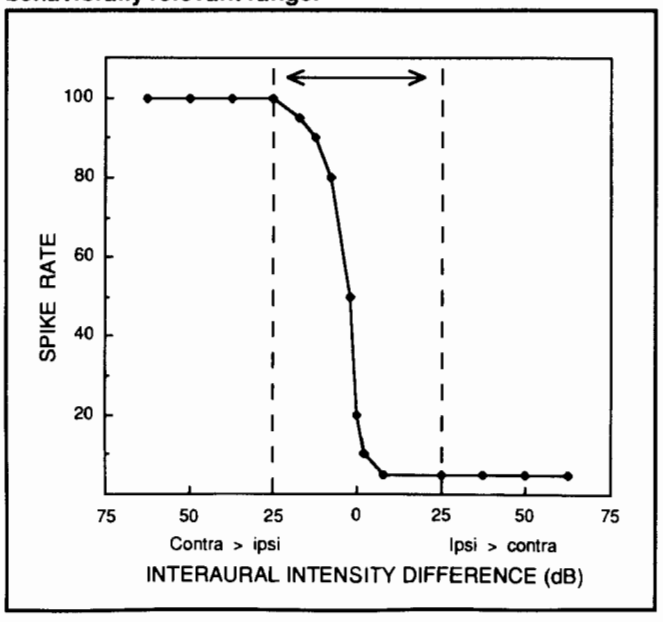
latency-intensity relation, it is likely that the neural code for interaural intensity differences in brief-duration sounds relies as much on the timing of the excitatory/inhibitory inputs as on their relative amplitudes. Indeed, for neural and psychophysical responses to transient stimulus elements, it has been possible to show that the effects of an amplitude disparity favoring one ear may be offset by an arrival time disparity favoring the other (time-intensity trading). In nature, these two cues work in concert, and it is likely that these excitatory/inhibitory neurons encode both cues, although they are by no means the only neurons that do so (Phillips & Brugge, 1985).

A number of important principles emerge from these analyses. First, at levels rostral to the superior olivary complex, there is a striking contralaterality in the neural representation of the cues for sound source azimuth. It is manifested in the cue ranges (microseconds or dB) associated with maximal firing rates and those associated with the dynamic portions of the response rate-disparity functions. It is common to the coding of interaural phase and interaural intensity disparities. Note that this contralaterality does not derive from any "dominance" of the contralateral "ear" *per se*: it is an emergent property of the binaural comparison, and it refers to contralateral *space*.

It follows from this that each side of the auditory brain rostral to the superior olivary complex may be independently capable of localizing sounds in the contralateral auditory hemifield (Jenkins & Masterton, 1982; Phillips & Brugge, 1981; Phillips & Irvine, 1981). This is in contrast to an earlier view (van Bergeijk, 1962) which proposed that it was a comparison of the outputs of the left and right olivary nuclei that provided the basis for the discrimination of sound source laterality. It now seems more likely that the relevant comparison is of the stimuli at the two ears; once that comparison has been made, the emergent contralaterality conferred upon higher neurons renders any further comparison (between the sides of the brain) unnecessary. In the following section, we will see a powerful instantiation of this hypothesis.

Finally, recall that all of these interaural comparisons are performed along frequency-specific channels (within the tonotopic framework). This has the advantage that for spectrally complex sounds, the interaural disparities associated with any given spectral element may be analyzed independently. The advantage derives from the fact that the magnitude of the interaural time or amplitude difference generated by any given source locus varies with signal frequency: the separate analysis of different spectral elements may thus be the basis of superior localization performance for complex sounds.

Figure 4. Idealized impression of the responses of a single neuron to variations in the interaural intensity disparity of a tonal signal presented to the two ears. Details as for Figure 3. Again note that the spike output of the neuron is least ambiguous in specifying the intensity difference when the size of the disparity is small, and within the behaviorally relevant range.



Binaural Processing Disorders

It is abundantly clear that binaural convergence provides a major basis for the identification of sound source azimuth. This in no sense implies that the sole end to which these computations are put is to localize sound *per se*. Once the sound source locus is established, presumably in the firing rates of the relevant neurons, then that spatial information is available for other purposes, notably as a basis for selective attention (e.g., cocktail party effect).

In the case of neural, and therefore perceptual, sensitivity to both forms of interaural time differences, it is obvious that accurate coding relies not only on convergence of input from the two ears, but also on the precision of neural spike timing in the monaural inputs to the site of convergence. In cats (Masterton et al., 1967), section of the trapezoid body, which contains the fibers mediating interaural time comparisons, results in elevations in smallest discriminable time disparities from the normal range (less than about 50 microseconds) to values an order of magnitude greater (more than 500 microseconds). This is likely beyond the interaural fusion threshold, and suggests that the lesioned animals may be detecting the order of stimulus presentation to the two ears, rather than the apparent (spatial) azimuth of a single source.

In humans, presumed or demonstrable pontine foci of dysmyelination may result in a disturbance of the brainstem auditory evoked response, suggesting that the timing of brainstem auditory neuron discharges has been compromised by the

disease process, presumably by variably interfering with axonal conduction velocities (Waxman, 1982). One might reasonably expect that objective evidence of pathology in neural conduction times should be linked to detriments in interaural time discriminations, and there is preliminary evidence that such is in fact the case (Hausler & Levine, 1980; Zerlin & Mowry, 1980). Interestingly, while pontine dysmyelination may impair discrimination of interaural time differences for transients, the same patients may retain roughly normal discrimination of interaural intensity differences of noise pulses (Hausler & Levine, 1980). There are two reasons why we might expect this separability. One is that interaural time and amplitude disparities are encoded in spatially separated pathways which are independently susceptible to dysmyelinating (or other) disease processes; the other is that the neural coding of interaural intensity differences in signals with steady-state components may rely less on spike timing than on spike rate.

The binaural interaction data reviewed above may also help understand the phenomenon of binaural masking level differences and their vulnerability to brainstem pathology (see Lynn et al., 1981). The test comes in a variety of forms, but in principle, it is based on the fact that the masking effect of a binaural noise background on the detectability of a simultaneously-presented, binaural tonal (or other) signal is lessened by introducing an interaural phase reversal of either the signal or the noise. It is not unreasonable to speculate that the interaural phase reversal of one of the stimulus components will alter the population of neurons excited by that component, while the neurons driven by the other element will remain unchanged. This separation in the identities of the discharging neurons might underlie the perceptual segregation of the two signals. By the same line of argument, any process that disrupts the neural coding of interaural phase disparity must also impair the segregation of signal and noise neural representations. Lynn et al.'s (1981) data suggest that this may indeed have been the case. They showed that pontine foci of dysmyelination, which revealed themselves in late waves of the brainstem auditory evoked response, severely reduced the perceptual benefit obtained by the interaural phase reversal. Pathology of higher auditory centers, rostral to the sites of binaural convergence, was without effect on the consequences of phase reversal. At these more rostral levels, the neural representation of this binaural information may, as mentioned, reside in the discharge rates of the relevant neurons, rather than in the temporal spacing of those cells' spike responses. This would be compatible with the relative immunity of the code from the effects of disease processes that disrupt neural timing. On the other hand, few of Lynn et al.'s patients with rostral lesions had diagnoses of specifically dysmyelinating disease, so the two groups of patients differed not only in lesion locus, but also in the pathophysiology of the lesions.

There is agreement that, in animals, focal unilateral lesion of auditory structures rostral to the olivary nuclei profoundly disturbs sound localization performance (Jenkins & Masterton, 1982; Kavanagh & Kelly, 1987; Phillips & Brugge, 1985). In a two-choice task, the animals typically retain the ability to discriminate sound source laterality (i.e., to discriminate between left and right), but are unable to localize sounds within the acoustic hemifield contralateral to the lesion. This finding suggests that the animal's perception of auditory space contralateral to the ablation has become undifferentiated. The deficit in performance extends to both acoustic hemifields in animals with bilateral lesions (Kavanagh & Kelly, 1987).

An important, but less often cited, finding from studies of animals with bilateral (cortical) lesions is that the deficit in sound localization performance might be task-dependent (Heffner, 1978; Heffner & Masterton, 1975). Primates and dogs that have received bilateral cortical ablations are unable to indicate even sound source laterality by approaching a remote sound source; they are, however, able to indicate source laterality using other motor responses. This indicates that the lesioned animal has available the sensory spatial information, but cannot express that information in the performance of some localization tasks. To our knowledge, this type of investigation has been limited to the two- speaker (laterality) task. Whether the phenomenon extends to the more complex localization (within a hemifield) task is not known. Moreover, at present, we are unable to predict precisely which motor responses will be affected by the lesions.

Nevertheless, some significant conclusions might be drawn from these studies. The first is that the neural processes that mediate discrimination of sound source laterality are different from those that mediate localization within an acoustic hemifield. This follows from the fact that the former ability survives unilateral (and probably bilateral) lesions while the latter ability probably does not. Second, the animal data provide strong support for the notion of contralaterality of *spatial* representation in the rostral auditory nervous system, which we derived (above) from studies of central auditory neurophysiology. Finally, Heffner's studies point to the need for great attention to procedural variables when attempting to assess the contribution of a given neural structure to the performance of a task requiring sensory and motor (and intervening) processes.

Jenkins and Merzenich (1984) provided a further refinement in our knowledge of sound localization mechanisms. In cats, they physiologically mapped the tonotopic organization of the auditory cortex in one cerebral hemisphere. They then ablated only a single, narrow sector of the frequency representation. Following this surgery, they tested the cats in a free-field sound localization task using pure tone stimuli. The cats showed deficits in sound localization performance only for

sources contralateral to the lesion, and only for signal frequencies deprived of cortical representation. These data not only confirm the contralaterality of spatial representation in the auditory system, but provide an elegant demonstration of the processing of acoustic information along frequency- specific channels.

The extant evidence on the acoustic spatial abilities of human beings with lesions of rostral auditory centers is less concordant. Early studies of patients with unilateral temporal lobe insults pointed towards a contralateral, specifically spatial, deficit that was not understandable in terms of asymmetric peripheral sensitivities (Sanchez-Longo & Forster, 1958; Wortis & Pfeffer, 1948). Jerger et al. (1969) presented a detailed case study of a patient, with bilateral lesions of the temporal lobe, who showed poor localization ability in both hemifields. Patients with unilateral cerebral injuries further removed from the classical auditory pathway (inferior parietal and frontal lobes) sometimes display a "neglect" for stimuli in auditory space contralateral to the damage, particularly when relevant stimuli occur in both hemifields simultaneously (De Renzi et al., 1984; Heilman & Valenstein, 1972). In some other cases, poor localization ability in the contralateral auditory hemifield following these more remote lesions is less obviously due to a hemineglect (Klingon & Bontecou, 1966). These data are certainly compatible with the general notion of contralaterality of spatial representation in the auditory forebrain. It must be emphasized, however, that these observations do not indicate that the normal functional role of these structures is the localization of sounds per se. It is equally likely that auditory processing in general might be performed within a contralateral spatial framework. This would appear to be the most parsimonious account of auditory hemi-inattention: perhaps the neural processes mediating directed attention rely on the spatial information obtained from the sensory cortex of the same hemisphere, and in the case of the auditory system, this spatial information is contralateral in reference.

There have, however, been a number of reports describing patients with unilateral cortical lesions whose auditory spatial deficits do not conform to the contralaterality hypothesis advanced here (Altman et al., 1979; Bisiach et al., 1984; Ruff et al., 1981). A failure to detect any deficits in spatial hearing after forebrain lesions may not be damaging to the hypothesis, given the host of factors that may influence the detectability or severity of the deficit (lesion locus, transient nature of the deficit, test procedures, etc.). The difficulty raised by the reports cited above is two-fold. First, these authors report that spatial deficits tend to occur more often after right hemisphere damage than left. Second, in such cases, the deficits may extend to both auditory hemifields. It is not at all clear that the contralaterality hypothesis can easily deal with these findings.

Complex Sound Processing

Sounds can be complex in the spectral, temporal, or in both domains. In this section we examine only one of a number of possible conceptual frameworks in which we might think about the central processing of complex sounds, including speech, and their constituent acoustic elements. The coverage that follows is based largely on studies of single cortical neurons, and mostly in anesthetized animals; it is presented with the qualification that the use of general anesthesia in these studies might conceal some of the response diversity that one can encounter in alert animals, either by silencing some neurons, or by modulating the responses of others.

Cortical auditory neurons are sensitive to both the spectral and the temporal structure of sounds. The afferent pathways to the cortex preserve many of the spectral/amplitude tuning properties developed in the auditory brainstem, so that in the primary field, most cells have narrow excitatory response areas tuned to a CF, and these can be variously flanked by inhibitory response areas (Figure 2; Phillips, 1988c). Thus, whether or not a stimulus excites a cortical neuron depends in part on the overlap between the stimulus spectrum, and the disposition and sensitivity of the neuron's excitatory and inhibitory inputs. The temporal structure of a sound is represented by the timing of a cortical neuron's spike discharges, since these may recall the timing of acoustic events in the stimulus. This code probably is competent only for stimulus elements less than about 100 Hz, simply because of the imprecision in spike timing that derives from the number of serial synapses in the afferent pathway to the cortex (Phillips, 1989b).

Primary auditory cortex neurons in mammals (including primates) respond briskly to brief stimulus events, and show rather poorer (or no) spike response rates to maintained, invariant acoustic signals. Phillips and his colleagues have explored the neural mechanisms that shape the responses of cat cortical neurons to brief acoustic events, and for conditions where those events occur in isolation, against a noise background, or as modulations of tonal carrier signals (Phillips, 1987; 1988a,c; Phillips & Hall, 1987). One strictly qualitative interpretation of these studies is as follows. The responses of cortical neurons, at least in anesthetized animals, appear to reflect stimulus energy integrated over a relatively narrow time window, probably only a few tens of milliseconds wide at most. In the presence of an invariant signal, these neurons appear to adapt, i.e., adjust their threshold sensitivity to the effective level of the maintained signal. In order for a stimulus event to evoke spike discharges from a cortical neuron in the presence (adapted state) or absence (unadapted state) of a maintained signal, the stimulus event must provide an excitatory afferent volley, which either by spatial or temporal summation, exceeds the threshold of the neuron. The effectiveness of a stimulus event (be it the onset of a tone or noise pulse, an incidental amplitude or frequency modulation, etc.) de-

pends on the net balance of excitatory and inhibitory afferent responses which is evoked by overlap of the short-term spectral/amplitude properties of the stimulus on the one hand, and the adaptive state and response area organization of the neuron, on the other.

A recent experiment on cortical neurons in anesthetized cats illustrates some of these phenomena (Phillips, 1988a). Typically, cat cortical neurons respond with spike discharges transiently at the onset of a CF tone pulse. Some of these neurons, notably those that show an apparent tuning to tone pulse amplitude, are known to possess inhibitory response areas flanking the excitatory one at CF, and they are sensitive to the spectral bandwidth of acoustic signals. Other cortical neurons, notably those whose properties resemble those of cochlear nerve fibers, do not possess such sideband inhibition, and are not suppressed by wideband signals. We might reason that if cortical neurons respond only to the tone pulse onset transient, then they should be sensitive to the rise-time of the tone pulse. (This is because the faster the rise-time, the broader is the short-term stimulus spectrum at tone onset, and the more likely is the effective stimulus to activate sideband inhibitory inputs.) At suprathreshold tone pulse levels, this was indeed the case. Tone pulses with amplitudes that evoked maximal responses for medium or long rise-time signals were suppressive if the rise times were significantly shortened, but only in neurons that were independently suspected to possess sideband inhibitory response areas. The same general argument applies to the coding of transient amplitude modulations in ongoing carrier tones (Phillips & Hall, 1987). These data emphasize the sensitivity of cortical neurons to relatively brief stimulus events, and to the short-term spectra of those signals.

A second consequence of the sensitivity of cortical cells to short- term stimulus events is seen in the responses of those cells to signals presented against continuous noise backgrounds (see Phillips, 1987, 1989a for review). Continuous noise evokes little or no background spike activity in cortical neurons. Rather, the neuron's threshold for any signal presented against the continuous background is elevated to the effective level of the noise. Once the noise level is above threshold for producing this signal sensitivity shift, then any further increment in noise level is matched by equivalent signal sensitivity shifts. These neural data parallel psychophysical sensitivity shifts for signals presented against noise backgrounds. The underlying mechanism is likely a short-term neural adaptation to the effective level of the continuous noise (Phillips, 1985, 1989a). The net result of the mechanism is that even in the presence of moderately intense noise backgrounds, a cortical neuron's full spike-rate dynamic range can be available to encode the occurrence of any acoustic event against that background. Even if the threshold for the detection of the signal is raised, a salient cortical response is evoked by that signal. The data further suggest that the sound pressure level of a signal is not what defines the threshold of a cortical response - it may be signal level *re* background, or the amplitude "contrast". It is tempting to speculate that this property of cortical neurons may be the sensory mechanism compromised by cortical lesions in humans, which result in degraded performance at detection or discrimination of signals against noise backgrounds (Heilman et al., 1973; Olsen et al., 1975; see also Phillips, 1987, 1989a).

A second line of research, in large part prompted by Wollberg and Newman (1972), has explored the coding of species-specific vocalizations by neurons in the auditory cortex of awake primates (see Phillips, 1988c for review). Although these vocalizations are obviously devoid of linguistic content, some of them are nonetheless comparable to human speech sounds in being temporally complex concatenations of spectrally rich, closely spaced transients. In any given cortical neuron, the timing of spike discharges evoked by these signals is markedly nonrandom, and likely follows the time course of brief events in the stimulus. The fact that these responses occur during the stimulus indicates that the neurons are being driven by the acoustic content of the signal, and not its behavioral significance. The fact that spikes are precisely timed within the response again emphasizes the sensitivity of cortical neurons to transients. Both of these conclusions are confirmed by studying the responses of the same neurons to reversed vocalizations, in which the cadence of spike discharges is sometimes the reverse of that seen in response to the normal vocalization (Glass & Wollberg, 1983).

Different cortical neurons are strikingly individual in the temporal pattern of their spike discharges evoked by any given vocalization, and they are equally idiosyncratic in their response strengths to different vocalizations (Newman & Wollberg, 1973). It is perhaps reasonable to suppose that this individuality reflects the composition of those cells' respective excitatory/inhibitory response areas, since this factor will determine, in part, which components of the vocalization will be effective in evoking spike responses. Equally important, however, is the fact that the identity of the vocalization will not be encoded by "vocalization- specific" neurons ("grandmother cells"): rather, the acoustic structure of the vocalization will be encoded in the temporal pattern of spike activity across the mosaic of cortical elements deriving inputs from the cochlear places whose outputs are excited by the stimulus.

This general line of research has been extended to include examination of the extent to which cortical responses to any given component in the vocalization are set, or conditioned, by responses to preceding stimulus elements, i.e., the question of independence of the responses evoked by closely spaced acoustic events. The monkey vocalization data might be regarded as still preliminary on this issue, although evidence from a number of laboratories indicate that there are significant

interactions between responses to temporally adjacent auditory events within a vocalization (Newman & Symmes, 1979; Steinschneider et al., 1981; Wollberg & Newman, 1972). Most commonly, the presence of one stimulus element depresses sensitivity to a subsequent one, although some facilitative interactions have also been described. The mechanisms mediating these temporal interactions are still being worked out, but at least two forms of interaction seem likely (Phillips, 1988c). One is the time course of the excitatory or inhibitory response evoked by a given stimulus element, since this may outlast the duration of the triggering stimulus event. The second is a short-term adaptation to the effective level of the earlier stimulus element. Irrespective of the (synaptic) nature of these mechanisms, however, it is clear that some cortical cells are indeed sensitive to the temporal spacing or ordering of stimulus components, especially in the short-term sense. This sensitivity will shape the fashion in which a vocalization, or other temporally complex sound, is represented in the auditory cortex.

(By way of a digression here, let us ask ourselves the following question. If the responses of neurons in the primary auditory cortex are so dominated by transient stimulus events, then how does the cortex "process" steady-state signals? A tentative, but intriguing, answer to this question might be that it doesn't. As will be seen in the following section, and in the earlier discussion of binaural processing disorders, a remarkable number of "basic" auditory discriminations survive bilateral lesions of the primary auditory cortex. This raises the extraordinary, and perhaps neurophilosophical question, of *where* (in the brain) hearing takes place. Perhaps the safest speculation is that the various dimensions of hearing — e.g., spectral, spatial, temporal — are mediated by different brain structures, so that "hearing" should not be thought of as a unitary sensory-perceptual skill.)

Returning to our theme then, the neurophysiological studies suggest that the contribution of the auditory cortex to the processing of complex sounds is an acoustic one. Cortical neurons are sensitive to the spectral composition of acoustic transients, and to their spacing. The behavioral state of the animal (e.g., Brugge & Merzenich, 1973) or the behavioral relevance of the stimulus (e.g., Miller et al., 1972) may alter the strength (and sometimes the patterning) of the responses to a given signal, or perhaps the salience of the evoked response against the background of spontaneous spike activity. The meaning of the stimulus, however, appears not to be a factor in the representation of that stimulus in the auditory system.

It is worthwhile at this point to insert a caveat to this argument. At least some of the nonprimary cortical auditory fields (Reale & Imig, 1980) and cortical regions far removed from the primary sensory areas (Irvine & Phillips, 1982) contain neurons that receive extraordinarily convergent input, as revealed in their very broad frequency tuning. Many of these neurons are labile and variable in their responsiveness to a given stimulus over time (e.g., Manley & Muller-Preuss, 1978). It is possible that this non-stationarity is a medium through which the behavioral significance of a stimulus might modify the stimulus selectivity of a neuron. Weinberger and Diamond (1987) have provided some preliminary evidence on this issue. They showed in cats that some cortical neurons which were equivalently responsive to a broad range of tone frequencies exhibited a highly selective enhancement of responsivity to a particular frequency if that frequency was made behaviorally relevant to the animal. Although not, apparently, a property of AI neurons, this plasticity may be a potentially important mechanism in auditory learning.

The question of whether these neurophysiological conclusions are germane to the human auditory system is obviously an important one. We have reason to believe that the cytoarchitecture of the human auditory cortex shares features common to that of nonhuman species (compare: Brugge, 1975; Galaburda et al., 1978; Seldon, 1981a,b). It is likely that the human cortex contains multiple auditory fields (Celesia, 1976), and evidence from neuromagnetic response studies suggests that there is at least one tonotopically-organized field in the region of the superior temporal plane on which the primary auditory cortex is located (Romani et al., 1982). There is also recent evidence that the cortical coding of consonant-vowel syllables in the human cortex (Kaukoranta et al., 1987) might follow rules common to those seen in primate cortex (Phillips, 1988c; Steinschneider et al., 1981). By this we mean that the responses to the components of the speech sounds seem to be dictated by strictly acoustic and neural response parameters, also seen in studies with simpler sounds, and not by the phonetic identity of the components.

All of these data might properly be regarded as suggestive rather than definitive. If we choose to conclude that the general principles of auditory cortex function seen in the higher mammals may be common to humans, then we are prompted to two speculations. First, the process of speech (complex sound) perception might have an acoustic phase which is separable from higher-level (phonetic) phases. Second, the contribution of the auditory system, including the primary cortex, to this process is a specifically acoustic, sensory-analytic one.

Certainly, the former speculation is compatible with recent accounts of human speech perception (Blumstein & Stevens, 1979, 1980; Kurowski & Blumstein, 1987; McClelland & Elman, 1986; Stevens, 1980; Stevens & Blumstein, 1978). In contrast to some earlier models which proposed that the acoustic and phonetic analyses of speech sounds were not readily separable (e.g., Liberman et al., 1967), these more recent studies provide evidence that many phonetic categories

have specific short-term acoustic spectra. The model suggests that the process of speech perception involves a sliding temporal window that samples short-term spectra, and matches the samples against internal templates for subsequent phonetic and other analyses. This is not to imply that specifically linguistic functions do not penetrate the lowest levels of speech recognition processes, since there are obvious contraindications, particularly where the acoustic signal is ambiguous (McClelland & Elman, 1986; Warren, 1970). It does, however, point to separable acoustic and phonetic analyses in the speech recognition process, and this separability allows the possibility of pathology selective to either one of them. This general line of argument, is, of course, familiar to students of acquired language disorders (see Caplan, 1987; Caramazza, 1988).

Complex Sound Processing Disorders: Cortical Lesions and Word Deafness

Of the instances in which it has been possible to link the development of a central hearing disorder to pathology of a single auditory brain region, that of word deafness and the auditory sensory cortex is among the strongest (see Goldstein, 1974; Mendez & Geehan, 1988 for review). Rather than engage in discussion over the nosology of cortical hearing disorders (see Buchman et al., 1986; Leicester, 1980; Michel et al., 1980; Rapin, 1985), we indicate that by "word deafness" we mean poor discrimination of speech in the absence of a peripheral auditory pathology of a kind that could account for the impairment. Note that this deficit may occur in relative isolation ("pure word deafness"), that in some patients the discrimination deficit clearly extends to nonverbal sounds ("auditory agnosia"), and that in other patients, it is part of a more generalized unresponsivity to sound ("cortical deafness").

We preface what follows with some cautionary remarks. First, pathologies of the cerebral cortex do not respect the boundaries of functionally-defined neural territories. Moreover, the locus of the primary (or any other) field with respect to the fissural pattern is probably not static, so that two patients with spatially coextensive foci of damage need not have the same functional cortical regions compromised. Second, significant destruction of a cortical region often has the further consequence of retrograde degeneration of some of its afferent inputs. This is such a profound effect that it was used to advantage for studying cerebral connectivity in the 1950s and 1960s; the phenomenon raises the question of whether a functional impairment consequent to cerebral injury should be attributed to the death of the cortical region concerned, or to the retrogradely-degenerated sources of afferent supply.

There is some agreement that word deafness is probably pathognomic of bilateral lesion of the primary auditory cortex (Adams et al., 1977; Auerbach et al., 1982; Buchman et al., 1986; Coslett et al., 1984; Earnest et al., 1977; Jerger et al., 1969; Kanshepolsky et al., 1973; Miceli, 1982; Rosati et al., 1982; Yaqub et al., 1988; see, however: Metz-Lutz & Dahl, 1984; Saffran et al., 1976). Particularly as it pertains to the "pure" cases, this conclusion is of great importance because it imputes a specifically linguistic function to a classically sensory brain region. This view seems quite at variance with the animal neurophysiology because we concluded from those studies that the cortex has a strictly sensory analytic function. On the other hand, animals are probably linguistically incompetent (Fodor et al., 1974; Terrace et al., 1979), so the physiology of animals' primary auditory cortex might be viewed as irrelevant to this issue. However, the speech perception literature suggests the existence of separable acoustic and linguistic levels of analysis, and if the sensory role is to be ascribed to any brain system, then surely it must be to the classical auditory sensory one. It is for these reasons that the notion of word deafness being attributable to primary auditory cortex lesions is so provocative.

Commonly, word deafness is a stage in the resolution from a more complete unresponsivity to sound, during which there may be remarkable recovery of audiometric sensitivity (Earnest et al., 1977; Mendez & Geehan, 1988; Miceli, 1982; Tabira et al., 1981). The recovery of tone thresholds in the presence of maintained deficits in other acoustic sensory-perceptual performances is seen in the animal cortical lesion literature. Kavanagh and Kelly (1987, 1988) reported that ferrets with bilateral cortical lesions showed transient deficits in behavioral tone audiograms but permanent and severe losses in sound localization ability. Equally interesting in the present context are the studies by Heffner and Heffner (1986a,b; 1989b) who report that Japanese macaque monkeys surgically deprived of the auditory cortex bilaterally, lose the ability to discriminate conspecific vocalizations, while showing considerable recovery in tone audiograms. These authors recently presented two independent lines of evidence that any residual (audiometric) hearing loss in these animals could not account for the vocalization discrimination deficit (Heffner & Heffner, 1989a). Compensation for the hearing loss by selective amplification of the relevant spectral elements failed to improve discrimination performance in the lesioned animals, and filtering of the vocalizations to simulate a cortical hearing loss did not significantly impair performance in normal monkeys. This suggests that the compromise of some auditory sensory-perceptual dimension other than absolute sensitivity underlies the vocalization discrimination deficit in the lesioned animals.

Interestingly, primates with large bilateral cortical lesions also show deficits in the discrimination of behaviorally-irrelevant complex sounds (Symmes, 1966), human vowel sounds (Dewson et al., 1969), and in the discrimination of conspecific vocalizations from other animal calls (Hupfer et al., 1977). Sensitivity to long-term tonal patterns may survive bilateral AI lesions in animals; it is lost when the lesions extend to include

more remote insular and temporal regions (Colavita et al., 1974; Kelly, 1973). In man, the speech perception deficit also often occurs in the presence of auditory processing difficulties for non-linguistic material (Adams et al., 1977; Buchman et al., 1986; Jerger et al., 1969; Mendez and Geehan, 1988; Miceli, 1982; Rosati et al., 1982;). It is not completely clear whether the generality of the deficit is correlated with the size of the lesions.

One insight into the nature of the phenomenology of the deficit in man comes from the patients' own descriptions of the perceptual events evoked by speech (and other complex) sounds. In most instances, the deficit appears not to be in *naming* the source of the sound, as might result from a disconnection syndrome (see, however, Denes and Semenza (1975) for one counter-example). The patients' descriptions suggest that their acoustic experiences may be relatively undifferentiated. Patients with bilateral lesions typically describe speech (and other complex) sounds as "buzzes" or "static" or "noise." This suggests that they detect the presence of the sounds, but that their perceptual differentiation of the sounds is collapsed. This is in contrast to the reports of some patients with unilateral (typically left-sided) lesions near the auditory cortex, who describe speech as not "registering" (Saffran et al., 1976), or who complain that they "hear the sound of your voice but can't understand what you are telling me" (Denes & Semenza, 1975). These latter descriptions are perhaps more consistent with a disconnection of the cerebral auditory processor from an upstream linguistic target.

There have been a number of recent descriptions of patients with purer forms of word deafness in which the ability to recognize or identify non- verbal sounds appears only modestly, if at all, to be compromised (Auerbach et al., 1982; Coslett et al., 1984; Metz-Lutz and Dahl, 1984; Saffran et al., 1976; Yaqub et al., 1988). Typically, the relative preservation of non-verbal sound recognition is tested by a confrontation method in which the patient is presented with a common environmental sound (e.g., a door slamming, hands clapping, a telephone ringing), and is required to indicate the identity of the sound by verbal or other means. Patients with the purer forms of word deafness may perform these identifications quite well while being almost completely unable to perform the same tasks using speech stimuli. Before we accept this contrasting performance as evidence of a verbal/nonverbal dissociation, we must ask if there is any other qualitative or quantitative difference between verbal material and the environmental sounds used. We might reasonably ask if any of the nonverbal sounds employed in these studies has a spectrotemporal microstructure which matches that of speech sounds. In what sense is the sound of a door slamming acoustically comparable to the temporal concatenations of transients in speech?

This is not a trivial enquiry. There is abundant evidence that patients with temporal lobe lesions have significant difficulty with short- term temporal discrimination and temporal resolution (Auerbach et al., 1982; Chedru et al., 1978; Kurdziel et al., 1976; Lackner & Teuber, 1973; Swisher & Hirsh, 1972). If the human auditory cortex is to be implicated in some specifically linguistic processing, then there is a pressure to demonstrate that the deficit in word deafness is truly restricted to verbal material. A comparison of identification performance for speech and environmental sounds obviously succeeds in contrasting performance for verbal and nonverbal material. It might, however, also succeed in contrasting performance for sounds of very different short-term spectrotemporal complexity.

There have been a number of recent studies that have probed more analytically the acoustic processing deficits in word deaf patients with bilateral lesions (Albert & Bear, 1974; Auerbach et al., 1982; Miceli, 1982; von Stockert, 1982; Yaqub et al., 1988). In all of these cases, the patients showed auditory processing deficits for brief stimuli and/or the temporal sequencing of acoustic events (see also: Buchman et al., 1986). Auerbach et al. (1982) and Yaqub et al. (1988) presented evidence on the temporal resolving powers of their patients. Click fusion thresholds (i.e., minimum temporal spacing between two clicks required for their perceptual separation) are normally in the order of two to three milliseconds, but in the two word-deaf patients, fusion thresholds were close to 30, and 16, milliseconds respectively. Similarly, whereas normal listeners are able to count click stimuli presented at rates up to 10/second, the two word-deaf patients were unable to perform this task for click rates in excess of 2/second. Since the patients are able to "hear" clicks presented in isolation, these psychoacoustic data suggest that the cortex has a special role in the perceptual elaboration of short-term temporal event sequences. Note that the time frame of the deficit is close to that of the acoustic events in speech (cf. Stevens, 1980).

Auerbach et al. (1982) took this analysis a step further by obtaining voice onset time (VOT) identification and discrimination functions, for a single consonantal place of articulation, from their patient. They found that the VOT identification function (labelling task) was preserved, with the phonetic boundary at its normal locus. In contrast to normal listeners, however, their patient was unable to discriminate consonant-vowel syllables on the same or opposite side of the phonetic boundary, which differed in VOT by 20 milliseconds. Miceli (1982) reported a similar pattern of speech perception deficits in a patient with a more generalized cortical auditory disorder. These data are again compatible with a deficit in temporal processing in the milliseconds to tens-of-milliseconds range. Unfortunately, no word-deaf patient has been tested more exhaustively with these kinds of procedures, so the limits of

their temporal resolution in speech perception tasks is unknown.

Further circumstantial evidence on the temporal grain of the processing function(s) lost after bilateral lesions comes from the agreement that the discrimination of steady-state vowels survives the cortical pathology (Auerbach et al., 1982; Miceli, 1982; Yaqub et al., 1988). These sounds have a demonstrably periodic fine time structure, although it is in the millisecond or sub-millisecond time range. While it is clear that the timing of spike discharges in the auditory nerve is sufficiently precise to encode much of this structure in spike times (e.g., Carney & Geisler, 1986; Delgutte, 1980; Young & Sachs, 1979), cortical neurons show greater variability in their spike timing and likely cannot support a temporal code for elements much in excess of 100 Hz (Phillips, 1989b). This suggests that the cortical representation of vowels may be a spectral one rather than a temporal one, and that the cortex does not have a special role in the representation of temporal events in the sub-millisecond time frame.

Our investigations into the detailed nature of the auditory processing deficits resulting from bilateral primary cortex lesions are only just beginning. Insofar as word deafness (in its various manifestations) is concerned, we have little reason to believe that it is more than the most obvious and debilitating consequence of a more general spectral/temporal processing disorder. In particular, we are led, by a number of lines of evidence, to suspect that the primary auditory cortex has an important role in the processing of stimuli with temporal variations in the milliseconds to tens-of-milliseconds time frame. Deprived of cortical representation, sounds with a complex short-term temporal structure appear to evoke relatively undifferentiated or unstructured auditory perceptions. Precisely what it is about cortical architecture that makes it important for this perceptual function is unknown. We should, however, note that the deficit resulting from its loss may be quite different from that which characterizes some aphasic patients (Blumstein et al., 1977; Oscar-Berman et al., 1975). In these cases, the strictly acoustic analysis of speech (and other) sounds may be relatively unimpaired, but their linguistic elaboration is not.

Conclusions

In the preceding pages, we have sketched some principles of central auditory function derived from studies in basic auditory neuroscience and human central auditory dysfunction. We have seen that the auditory nervous system has a strikingly parallel organization in which individual nuclei, or local territories within those nuclei, have functional specializations for different aspects of acoustic stimulus processing. The neural representation of a sound may exploit any of a variety of neural coding mechanisms — the identity of the discharging neurons, the rate of spike discharge, the temporal ordering of spike discharges. The nature of a neural representation will shape its susceptibility to various pathophysiological processes. Note that we have not even considered here levels of representation not seen in the behaviour of single neurons: that there may be information expressed in the behaviour of large ensembles of neurons which is simply inaccessible to the microelectrode (just as the frequency tuning of a cortical neuron cannot be gauged by patch-clamp studies of any of that neuron's membrane channels).

Our data on auditory neuroscience as it pertains to central dysfunctions, and our knowledge of the nature of auditory perceptual consequences of central pathology are frustratingly incomplete, even in the restricted domains that we have examined here. An important lesson to be learned from these lacunae is this: that the depth of our understanding of central auditory processing disorders, and therefore our ability to manage these listeners, will be markedly enhanced when we begin more competently to link the structure of our stimuli, the neurobiology of the compromised brain structure, and behavioural performance.

Acknowledgements

Some of the research summarized in this report was funded by NSERC grants U0442, E2745 and URF0035035, and by a Dalhousie University Research Development Award to the author. H.E. Heffner first raised with me the question of "where" hearing takes place. Special thanks are due to S.E. Hall who collaborated in some of the recent experiments described here, to two reviewers for their helpful comments on a previous version of this paper, and to A. Keith for much-needed support during the long recording experiments.

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