Auditory Cortical Neurons are Sensitive to Static and Continuously Changing Interaural Phase Cues

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SUMMARY AND CONCLUSIONS

1. The interaural-phase-difference (IPD) sensitivity of single neurons in the primary auditory (AI) cortex of the anesthetized cat was studied at stimulus frequencies ranging from 120 to 2,500 Hz. Best frequencies of the 43 AI cells sensitive to IPD ranged from 190 to 2,400 Hz.

2. A static IPD was produced when a pair of low-frequency tone bursts, differing from one another only in starting phase, were presented dichotically. The resulting IPD-sensitivity curves, which plot the number of discharges evoked by the binaural signal as a function of IPD, were deeply modulated circular functions. IPD functions were analyzed for their mean vector length (r) and mean interaural phase ($\bar{\phi}$). Phase sensitivity was relatively independent of best frequency (BF) but highly dependent on stimulus frequency. Regardless of BF or stimulus frequency within the excitatory response area the majority of cells fired maximally when the ipsilateral tone lagged the contralateral signal and fired least when this interaural-phase relationship was reversed.

3. Sensitivity to continuously changing IPD was studied by delivering to the two ears 3-s tones that differed slightly in frequency, resulting in a binaural beat. Approximately 26% of the cells that showed a sensitivity to static changes in IPD also showed a sensitivity to dynamically changing IPD created by this binaural tonal combination. The discharges were highly periodic and tightly synchronized to a particular phase of the binaural beat cycle. High synchrony can be attributed to the fact that cortical neurons typically respond to an excitatory stimulus with but a single spike that is often precisely timed to stimulus onset. A period histogram, binned on the binaural beat frequency (f_b), produced an equivalent IPD-sensitivity function for dynamically changing interaural phase. For neurons sensitive to both static and continuously changing interaural phase there was good correspondence between their static ($\bar{\phi}_s$) and dynamic ($\bar{\phi}_d$) mean interaural phases.

4. All cells responding to a dynamically changing stimulus exhibited a linear relationship between mean interaural phase and beat frequency. Most cells responded equally well to binaural beats regardless of the initial direction of phase change. For a fixed duration stimulus, and at relatively low f_b , the number of spikes evoked increased with increasing f_b , reflecting the increasing number of effective stimulus cycles. At higher f_b , AI neurons were unable to follow the rate at which the most effective phase repeated itself during the 3 s of stimulation.

5. Changing stimulus intensity equally at both ears at BF resulted in relatively little systematic change in the interaural-phase sensitivity when data obtained near threshold and at nonmonotonic levels were excluded from the analysis. In a few cells where the effects of intensity change were studied at a number of excitatory frequencies the results were the same.

6. The IPD functions were converted to interaural-time-difference (ITD) functions. Approximately 85% of the curves had their peaks or troughs within the physiological range of $\pm 400 \mu\text{s}$,

and most (91%) of the peaks occurred when the ipsilateral tone lagged the contralateral (average: $+142 \mu\text{s}$), whereas most (79%) of the troughs occurred at an ipsilateral lead (average: $-144 \mu\text{s}$). The same trends were seen when a neuron's ITD sensitivity was represented by a composite function. For the majority of cells analyzed in this way both the composite peak and trough were within the physiological range of ITD.

7. The slope of the linear regression line fitted to the plot of mean interaural phase-versus-frequency yielded the characteristic delay (CD) for that neuron, and the y -intercept its characteristic phase. When ITD functions from a single cell obtained at different frequencies were plotted on a common time axis, they rarely showed an exact registration near their peaks or troughs; the CDs could be located anywhere along the spike count-versus-ITD function. Of the 14 cells for which such analysis was possible, 12 of them had CDs that fell within the $\pm 300 \mu\text{s}$ range with values almost equally distributed about zero ITD (average: $+49 \mu\text{s}$).

INTRODUCTION

Many central auditory neurons are known to be highly sensitive to interaural disparities in the time (ITD) or intensity (IID) of pure-tone stimuli presented dichotically. These two acoustical cues, among others, are intimately related to the ability of a listener to localize sound sources in space (see e.g., Blauert 1983). For tonal stimuli of low frequency and in the steady state or near steady state, a neuron's ITD sensitivity is generally believed to derive mainly from interactions between time-locked afferent input arising from the two cochleae (Johnson 1980; Rose et al. 1967) and converging on neurons in the medial superior olivary nucleus (Goldberg and Brown 1969; Yin and Chan 1990). Once established, ITD sensitivity is then presumably preserved by neuronal circuits at successively higher levels of the lemniscal pathway including the dorsal nucleus of the lateral lemniscus (Brugge et al. 1970), central nucleus of the inferior colliculus (Kuwada et al. 1979; Rose et al. 1966), ventral division of the medial geniculate body (Aitkin and Webster 1972), and as many as three fields of the auditory cortex (Brugge et al. 1969; Orman and Phillips 1984). It is also apparently preserved by neurons in the cerebellar vermis (Aitkin and Boyd 1975), which receive their input via ITD-sensitive cells in the dorsolateral pontine nuclei (Aitkin and Boyd 1978).

In 1969 Brugge and coworkers reported that neurons of auditory cortical area AI and of cortex on the anterior ectosylvian gyrus [now known as the anterior auditory field (Knight 1977)] exhibited an ITD sensitivity similar to that

described a few years earlier by Rose and his colleagues (1966) for neurons in the inferior colliculus. The discharges of certain cortical cells responding to low-stimulus frequencies were shown to be a periodic function of ITD, and when this property was studied at several different frequencies and intensities within the cell's response area, the peaks or troughs of the response functions tended to remain in alignment; the latter observation gave support to the hypothesis of Rose et al. (1966) that there exists for some ITD-sensitive neurons a "characteristic delay." Later studies established with certainty that ITD-sensitive neurons are also active in the posterior field adjacent to area AI in the anesthetized cat (Orman and Phillips 1984), in the primary auditory field of the anesthetized chinchilla (Benson and Teas 1976), and in field AI of the unanesthetized monkey (Brugge and Merzenich 1973).

The ITD established between the two ears by the arrival of a sound originating from a distant source not centered on the midline is viewed as composed of two delays. The onset-time disparity is the difference in arrival time between the first wavefronts of the sound at the near and far ears. For a pure tone this onset disparity also produces a simultaneous interaural-phase difference (IPD) that is maintained for the duration of the stimulus. It is now known that IPD, and not onset-time disparity, is the critical parameter in most neurons' ITD sensitivity at the level of the medial superior olive (Yin and Chan 1990) and central nucleus of the inferior colliculus (Kuwada and Yin 1983). Neurons of auditory cortex are also able to detect the IPD of low-frequency tones, although very little else is known of the properties of these cells (Benson and Teas 1976; Brugge et al. 1969; Brugge and Merzenich 1973; Orman and Phillips 1984).

Numerous other studies over the past decade have added greatly to our knowledge of the binaural interactions exhibited by single auditory neurons, of the brain stem mechanisms that underlie these interactions, and of the possible functions these interactions may serve in sound-localization behavior (for recent reviews see Phillips and Brugge 1985; Yin and Chan 1988; Yin and Kuwada 1984). In the present study, we found that the IPD sensitivity of some AI neurons is remarkably similar to that of IPD-sensitive neurons in the medial superior olive and central nucleus of the inferior colliculus. These results lead us to conclude that sensitivity to IPD is transmitted from the lower brain stem to the primary auditory cortex where it is preserved essentially undistorted.

METHODS

Experiments were performed on 24 healthy, clean-eared adult cats anesthetized with an intraperitoneal injection of pentobarbital sodium (40 mg/kg). The animals were maintained at a surgical level of anesthesia by supplemental injections (10 mg/kg) of the drug at regular intervals throughout the experiment. For this paper, data were chosen from eight of these animals whose cortical single-cell thresholds to stimulation of each ear were within ~ 10 dB.

The methods of animal preparation, stimulus presentation, acoustic calibration, and microelectrode recording were essentially the same as those employed in earlier studies of auditory cortex in this laboratory (Brugge et al. 1969; Imig and Brugge

1978; Reale and Imig 1980; Reale and Kettner 1986). Briefly, the pinnae were removed, and a hollow ear piece was sealed into each truncated ear canal close to the tympanic membrane. The ear pieces conducted sounds transduced by Yamaha OM-2 earphones. For each experiment the sound delivery system was calibrated in situ. The amplitude and phase of the tone, at frequencies between 50 and 30,000 Hz, were stored by computer (PDP 11/23 or MicroVax GPX). These calibration data were used together with a digital stimulus system (Olson et al. 1985; Rhode 1976b) to produce sinusoidal signals of any desired frequency, intensity, and initial-starting phase. Stimuli used were pure tones delivered to one or both ears. Stimulus configurations are described in detail at appropriate places in RESULTS. All intensities are expressed in dB SPL re: 20 μ Pa. Recordings were made in a sound-shielded room with the animal positioned on a vibration isolation table.

The ectosylvian cortical region was exposed, and the dura was removed. A plastic chamber was cemented to the skull over the exposed area and filled with warm silicone fluid (Dow Corning 200 Fluid). Glass-insulated tungsten microelectrodes were advanced into the cortex under microscopic control by a Davies microdrive mounted on the chamber.

Area AI in the cat generally extends across the region between, and on the banks of, the anterior and posterior ectosylvian sulci (Merzenich et al. 1975; Reale and Imig 1980). The best frequencies (BF) (i.e., that stimulus frequency to which a neuron is most sensitive at a sound pressure level (SPL) near the lowest-threshold intensity) of AI neurons form a decreasing progression along the anterior-to-posterior axis of the field. To locate and determine the boundaries of the low-frequency representation of AI, we first mapped in each experiment the relevant portion of the tonotopic representation with a systematic series of electrode penetrations in which the BFs of single neurons or neuron clusters were determined. The location of the area occupied by neurons with BFs below ~ 3 kHz varied in different animals. Most often this portion of the frequency representation was largely buried on the banks of the posterior ectosylvian sulcus, although in a few animals the adjacent gyral surfaces also contained the representation. Although we are confident from our mapping studies that most of the data reported here were from single neurons located in AI, for some cells with very low BFs one is less certain. This is because the low-frequency representation of AI abuts the low-frequency representation of the adjacent posterior auditory field (Reale and Imig 1980).

RESULTS

Our principal aim was to study in a systematic way within the excitatory response area a cortical cell's sensitivity to static and continuously changing IPD. Each of the 43 cells represented in this study was tested with the use of stimulating tones set equal to that cell's BF, and for 92% of these neurons the responses at two stimulating frequencies or intensities, at least, were studied. Sensitivity functions representing five or more stimulating frequencies were obtained from 29% of our sample. BFs of these cells ranged from 190 to 2,400 Hz. Data collection from a single cell required satisfactory contact for 1–5 h.

Most of the neurons studied were excited by stimulation of either ear. When studied monaurally, spike count was either a monotonic or nonmonotonic function of stimulus level (see also Phillips and Irvine 1981). For neurons exhibiting nonmonotonic spike count-versus-level functions the dynamic range was rather narrow, ~ 10 –20 dB. These are neurons that would typically exhibit circumscribed response areas (i.e., the loci of all frequency-intensity pairings

that elicit a neuron's discharge are bounded by a closed contour). For monotonic neurons the response areas are usually V-shaped; the dynamic range of these cells rarely exceeded 40 dB (see also Phillips et al. 1985). The dynamic range need not be extended under conditions of binaural stimulation; and, indeed, this range may even be foreshortened. Thus for many AI cells in our sample there existed, even at BF, only a relatively small intensity domain within which the effects of IPD could be studied.

The curve relating spike count to IPD is, by definition, a function of a circular variable and may, therefore, be analyzed by the use of circular statistical metrics (Goldberg and Brown 1969; Rayleigh 1929; Rhode 1976a) for its mean vector length (r) and mean interaural phase ($\bar{\phi}$). With some restrictions, ($\bar{\phi}$) expresses the average phase relation between the cell's response and the interaural phase of the stimulus, whereas (r), the mean vector length (or vector strength), provides a measure of the strength of that relationship.

Binaural interactions studied with static IPD

A static IPD was produced by presenting low-frequency tone bursts to each ear simultaneously. A burst was usually 100 ms in duration and repeated once every 700 ms. The initial phase angle of one member of the pair was shifted systematically, but otherwise the tones to the two ears were

identical in frequency and constant in intensity as the static IPD was incremented in steps of 30° to cover the range from 0 to 360° . Data were collected during 20- or 30-stimulus trials at each 30° -increment.

Figure 1 shows the relationship between spike count and IPD for six AI neurons having BFs ranging from 550 to 2,400 Hz. For each neuron illustrated, IPD sensitivity was studied at a number of different frequencies across the cell's excitatory response area. For four of these neurons (Fig. 1, A–D) sound pressure level was held constant some 20–30 dB above BF threshold at all frequencies studied; for two cells (Fig. 1, E and F) intensity was adjusted equally at the two ears to maintain a nearly constant level above threshold at each stimulus frequency. There are several features of note. First, regardless of the BF the spike count exhibits a regular modulation when plotted as a function of IPD for stimulus frequencies as high as 2,500 Hz. For the curves illustrated in Fig. 1, vector strength ranged from 0.17 to 0.74, and all were significantly different from zero ($P < 0.001$ for all frequencies except 2,500 Hz where $P < 0.01$). For any given cell the degree of phase synchrony depended on the stimulus frequency. Neurons with BFs below $\sim 1,000$ Hz (Fig. 1, A and B) generally exhibited deeply modulated IPD functions even near the extreme limits of their response areas. For cells with higher BFs, the sensitivity to IPD is reduced for frequencies at and above

FIG. 1. Spike count plotted as a function of static interaural phase difference for 6 neurons having best frequencies between 550 and 2,400 Hz. Each curve in families of curves represents a single frequency within the neuron's response area. Dashed curves represent data obtained at best frequency. Best frequency and the SPL at the contralateral (C) and ipsilateral (I) ears shown above each panel. Individual frequencies or range of frequencies covered shown on each panel.

BF. Thus, for example, the curve shown in Fig. 1*F* that was obtained at 2,500 Hz exhibits a relatively weak phase sensitivity as compared with curves obtained at lower frequencies within the response area. We have observed significant IPD sensitivity at stimulus frequencies as low as 120 Hz and as high as 2,500 Hz as evidenced by a regular modulation of spike count with IPD and a statistically significant ($P < 0.005$) vector strength measured by the Rayleigh test (Rayleigh 1929; Rhode 1976a).

Second, for any given neuron the positions of the peaks of the curves may remain relatively constant, as shown in Fig. 1, or they may shift more abruptly as stimulus frequency changes. Regardless of whether these shifts are small or large, neurons whose phase sensitivity is linearly related to stimulus frequency are said to exhibit a "characteristic delay" (Rose et al. 1966; Yin and Kuwada 1983b), a property of certain IPD-sensitive AI cells, which we describe in greater detail later in this paper.

Third, there was a tendency for the average location of the peaks in a family of IPD functions to shift toward larger IPDs with increasing BF. In the present convention, this would imply that cells with the lowest BFs generally have their highest probability of firing when the contralateral tone leads the ipsilateral tone by less than $\sim 90^\circ$, whereas this favorable phase lead tends to be $>90^\circ$ for cells with the highest BFs.

Fourth, although spike count was a deeply modulated function of the IPD of a periodic stimulus, the curves were rarely perfectly sinusoidal in shape; often the peaks or troughs of the curves were not well defined, with the functions reaching broad plateaus at their maximal and minimal values, especially at the lowest stimulus frequencies.

Although not illustrated for reasons of clarity, there was usually a marked facilitation at the most favorable IPDs as evidenced by a spike count that was typically greater than the summed count produced by stimulation of either ear alone. At the least favorable IPDs this facilitation was not evident, and the binaural spike count was often equal to or even less than that of the monaural output, suggesting an out-of-phase inhibition.

Binaural interactions studied with dynamic IPD

Twenty-six percent of the neurons sensitive to static IPD were also highly sensitive to continuously changing interaural phase, which was produced by presenting 3-s suprathreshold tones of slightly different frequency to the two ears. Under these stimulus conditions the IPD changed smoothly over each complete cycle of the difference frequency. This also created a "binaural beat," the frequency (f_b) of which was equal to the frequency separation of the binaural tones (Kuwada et al. 1979; Yin and Kuwada 1983a). The spike discharges of this population of IPD-sensitive AI cells were time-locked around a particular phase of each binaural-beat cycle.

Figure 2 illustrates this discharge synchronization for one such neuron studied with a 600-Hz tone delivered to one ear and 607-Hz tone delivered to the other, thereby creating a binaural beat of 7 Hz. The BF for this neuron was 600 Hz, and its threshold was 35 dB. This cell, like many other AI neurons studied under general anesthesia,

FIG. 2. *A*: dot raster illustrating the temporal locking of the discharge to individual cycles of a binaural-beat signal created by presenting 10 identical 3-s tones at 5-s intervals to 2 ears separated in frequency by 7 Hz. *B*: peristimulus time histogram constructed from the same data as in *A* showing 21 major peaks corresponding to 21 binaural-beat cycles that occur during 3-s tones. *C*: period histogram plotted over 1 cycle of the binaural beat superimposed on the spike count-vs.-IPD function obtained with the use of static tones at the same SPLs. θ_s , static mean interaural phase; θ_d , dynamic mean interaural phase; r_s , static mean vector length; r_d , dynamic mean vector length.

discharged only one or two spikes shortly after the onset of a BF tone burst.

For the binaural-beat frequency of 7 Hz and an observation time of 3 s, there are 21 complete 360° cycles of IPD. The dot raster in Fig. 2*A* shows that under these conditions the neuron discharged but a single spike locked to the phase of each effective beat cycle. Not all cycles were effec-

FIG. 3. Dot rasters and period histograms derived from binaural beat paradigm. Binaural beat frequency (f_b) shown on each graph was created by delivering a 600-Hz tone to one ear and a slightly higher frequency ($600 + f_b$) to the other. $\bar{\phi}_d$, mean interaural phase; r_d , mean vector length; N , number of spikes evoked during 10 presentations of 3-s tone.

tive in evoking a discharge; indeed, perfect one-to-one entrainment was rarely observed over a full 3-s period of stimulation. Figure 2B is a peristimulus time histogram constructed from these same data, which more clearly illustrates the 21 major groupings corresponding to the 21 binaural-beat cycles into which the discharges were organized.

The period histogram in Fig. 2C, also derived from the same discharge trains, shows the distribution of spikes collapsed over one period of the binaural-beat cycle. The static IPD function obtained from this cell is plotted on the same axis, having adjusted the position of the period histogram to account for the phase shift that typically occurs with changing f_b (see Figs. 3 and 4 below). As with the spike count-versus-IPD function obtained under static conditions, it is possible under this dynamic condition to derive from the period histogram a mean interaural phase ($\bar{\phi}_d$) and a vector strength (r_d), which are shown on this graph. The peaks of the static and dynamic functions are in close registration, with $\bar{\phi}_d$ differing from the static mean interaural phase ($\bar{\phi}_s$) by no more than 6°.

Rate and direction of change in IPD

In the binaural-beat paradigm the rate of interaural-phase change is independent of the carrier frequency but

directly proportional to the magnitude of the binaural-beat frequency, because IPD goes through one complete cycle for each period of the f_b . Moreover, because the phase of the tone with the slightly higher frequency will change faster than that of the tone in the opposite ear, the interaural phase will increase at a constant rate until the two mismatched frequencies are 180° out of phase and then begin to decrease again at the same rate to 0°. During this

FIG. 4. Mean interaural phase ($\bar{\phi}_d$) plotted as a function of binaural beat frequency for 3 cortical cells. Data are fitted by a simple linear regression equation. Inset: compares the mean interaural phase obtained from each unit under static ($\bar{\phi}_s$) and dynamic ($\bar{\phi}_d$) conditions. Negative $\bar{\phi}_d$ can be converted to a positive value by adding 360°.

complete cycle of IPD change, the sound is perceived as moving toward the side with the slightly higher frequency, and this direction may be reversed simply by switching the frequency of the tones presented to each ear (see Kuwada et al. 1979). For most cells in this study, there was found a selectivity to the rate of interaural-phase change as measured by the number of spikes evoked over the 3-s stimulus period. On the other hand, we encountered no marked selectivity for the direction of phase shift.

The effect of varying the rate of change of interaural phase, by increasing f_b from 1 to 35 Hz, is illustrated in Fig. 3 with dot-rasters and companion IPD functions (period histograms) derived from the same neuron illustrated in Fig. 2. Strong locking to the binaural-beat cycle was observed over the full range of f_b shown, although the total number of spikes evoked (N) at each f_b varied greatly. At any f_b only 1 or 2 spikes were evoked by an effective stimulus cycle. Considering the transient nature of the discharge and the rather uniform distribution of spikes over the 3-s stimulus period at f_b below ~ 13 Hz, it follows that in this range the spike number was limited mainly by the number of stimulus cycles available during 3 s of stimulation. At higher f_b , however, this seems not to be the mechanism. Rather, the gradual fall in spike number with increasing f_b was associated with a reduction in the number of cycles that were effective in evoking spikes during the latter portions of the 3-s tones. Regardless of the total spike count, one can see in the period histograms of Fig. 3 that the spikes remained solidly locked to the IPD at f_b as high as 35 Hz. The highest f_b tested in our experiments was 45 Hz, and at that f_b the relatively few spikes evoked by that stimulus were locked to the beat cycle.

There is also shown in Fig. 3 a progressive and systematic shift in the peak of the period histograms as a function of f_b . The function that relates ϕ_d to f_b is shown in Fig. 4 for three cortical neurons studied with both dynamic and static changes in IPD. The data points in Fig. 4 are derived from that part of the experiment employing the dynamic binaural-beat stimuli. A simple linear regression line appears to fit the data points; correlation coefficients exceed 0.95. For a comparable carrier frequency and intensity level, the ϕ_d at the y intercept of the regression lines obtained by the use of this dynamic stimulus is expected to match reasonably well ϕ_s , the mean interaural phase derived from the IPD functions obtained under static conditions. The *inset* shows this comparison for the cells depicted on this figure.

To produce the binaural-beat frequencies shown in Fig. 4, the tonal frequency at the ipsilateral ear was held constant while the tone presented to the contralateral ear was incremented in frequency. By convention, this was defined as a positive f_b , whereas a negative f_b represented the reversed presentation of the same tones. Neurons responsive to dynamically changing IPD were usually tested with both directions of interaural-phase change. Figure 5 presents phase-versus-frequency functions obtained from four cells that were tested in this way as well as under static conditions. There were no systematic changes in the number of spikes evoked with changing the sign of f_b that would suggest a sensitivity to direction of interaural-phase change. Moreover, as illustrated in Fig. 5, a single regression line was found to provide a satisfactory fit for points derived

from both negative and positive f_b , and a close correspondence between ϕ_d (located at the vertical $--$) and the same cell's ϕ_s (see *insets*) derived from data taken under dynamic and static conditions, respectively. This is exactly what is expected if the cell's response is solely dependent on the IPD.

FIG. 5. Mean interaural phase (ϕ_d) plotted as a function of binaural beat frequency for 4 cortical cells. Points to the *right* of 0 (vertical $--$) correspond to a direction of sound movement toward the contralateral ear, points to the *left* to a direction toward the ipsilateral ear. Data are fitted by a single simple linear regression equation. *Inset*: compares the mean interaural phase obtained from each unit under static (ϕ_s) and dynamic (ϕ_d) conditions.

Binaural interactions studied with different stimulus levels

The effects of changing stimulus intensity equally at both ears on the interaural-phase sensitivity of a single AI neuron were comprehensively studied in a smaller number of cells. As discussed previously, the dynamic range of many of our neurons was severely limited. In general, we found that over this dynamic range neurons exhibited relatively little systematic change in their interaural-phase sensitivity.

The IPD functions in Fig. 6A show this in one of the few neurons for which we have extensive data taken under these stimulus conditions. This neuron was highly phase sensitive over a range of some 45 dB at its BF of 550 Hz. Except for the 40-dB curve obtained near threshold and the 85-dB curve obtained in the nonmonotonic region of the response area, the functions tend to overlay one another. From 40 to 75 dB the peaks of the periodic functions differ from one another in no systematic way as indicated by the mean interaural phases shown in the figure *inset*. One notes, however, that the 85-dB function in Fig. 6A, although deeply modulated, departs noticeably in its position from those cyclic functions obtained at lower levels within the cell's dynamic range. Whereas the ϕ_s obtained from 40 to 75 dB varied by no more than 15°, the ϕ_s derived from the 85-dB curve differed from the others by as much as 86°.

Changes in the mean interaural phase with intensity will result in concomitant changes in ITDs. Figure 6B illus-

FIG. 6. *A*: effects of changing SPL on the static IPD-sensitivity curve. Data presented at 6 SPLs at the best frequency of 550 Hz. *B*: mean interaural phase plotted as a function of stimulus frequency at 2 SPLs within the cell's response area. Data fitted by a linear regression equation.

FIG. 7. Dot rasters and period histograms obtained with a binaural-beat paradigm at 6 SPLs within the cell's response area. dB SPL refers to level at 2 ears. ϕ_d , mean interaural phase in degrees; r_d , mean vector length; N , number of spikes evoked during 10 presentations of 3-s tones.

trates the linear and nearly overlapping phase-versus-frequency curves obtained at 45 and 65 dB for the cell whose curves are shown above. The slopes of the lines, expressed as ITD, differed by only 23 μ s. This would represent about a 1% variation at a stimulus frequency equal to the BF of this cell. We calculated for all cells studied the change in ITD for a 10-dB change in intensity taking place at different frequencies and intensities, excluding those points at the extremes of a cell's dynamic range. These values ranged from 2 to 80 μ s/10 dB. The mean value was 22 μ s/10 dB, which was close to the mode (26 μ s/10 dB) of the distribution. For three cells it was possible to assess the effects of intensity at stimulus frequencies systematically spanning the entire response area. In these cases the effects of intensity change at multiple frequencies within the cell's response area appeared to mimic the effects observed at a stimulus frequency that produced the most deeply modulated IPD function.

Figure 7 illustrates the same phase stability with intensity level for another neuron obtained under continuously changing phase conditions. The mean interaural phases

(ϕ_d) differed by no more than $\sim 15^\circ$, over the 45 dB range of this cell. Again the largest difference occurs when comparisons involve the lowest or highest intensity levels.

Laterality of the response

Regardless of the stimulus frequencies or intensities we employed within each neuron's excitatory response area, there was a marked tendency for that cell to fire maximally when the phase of the tone to the ipsilateral ear lagged that to the contralateral (positive IPD) ear and for cell firing to reach its minimum when the reverse was true (negative IPD). This finding is illustrated in Fig. 8*A* where the mean interaural phase at the peak (closed squares) and at the trough (open squares) of an IPD-sensitivity function, calculated for all neurons and at all stimulus frequencies in our sample, is plotted as a function of stimulus frequency. Although IPD functions are often irregular in shape, peaks and troughs remain close to 180° out of phase with each other. The modal values for the peak and trough distributions of ϕ are 107° and -73° , respectively. The relationship between the ϕ at the peak and at the trough of individual IPD-sensitivity functions is perhaps more clearly seen in Fig. 8*B* where we show the near-perfect symmetry of the positions of the peaks and troughs of these functions over one IPD cycle.

FIG. 8. *A*: distribution of the mean interaural phase (ϕ) in degrees as a function of stimulus frequency derived from all spike count-vs.-IPD functions for all 43 phase-sensitive neurons in our sample. Filled squares represent peaks in curves; open squares, troughs. *B*: relationship between ϕ at peaks and ϕ at troughs of spike count-vs.-IPD curves. Dashed line is the diagonal.

Binaural interactions expressed as functions of ITD

Rose et al. (1966), and later Yin and his colleagues (1983b), found that for certain cells of the cat central nucleus of the inferior colliculus there existed an ITD that evoked the same relative discharge rate regardless of the stimulus frequency. Rose and his co-workers (1966) referred to this ITD as the characteristic delay (CD) and argued that it may be a property of some binaural neurons that allows them to detect the location of a sound source in space. Similar but less extensive studies on cells of the cat auditory cortex have been consistent with the notion that the CD arising in the brain stem is preserved at the cortical level (Brugge et al. 1969; Brugge and Merzenich 1973; Orman and Phillips 1984), although the data have so far not been extensive enough to prove the point. Benson and Teas (1976) were unable to find in their results from chinchilla auditory cortex a registration of peaks or troughs in spike count-versus-IPD functions taken at three different frequencies. In the present experiments ITD sensitivity of a single neuron was analyzed at a relatively large number of frequencies within the response area, and further consideration was given to the cortical presence of a CD in light of previous work in the inferior colliculus by Yin and Kuwada (1984).

We were able to study 14 IPD-sensitive neurons at a number of frequencies within the excitatory response area and to examine the extent to which they exhibited a CD. To do this we first converted the IPD functions to ITD functions by simply multiplying the IPD values on the abscissa, expressed as a fraction of one period, by the period of the stimulus frequency. We then normalized each of the resulting ITD functions and plotted them on the same time axis, where the family of curves could be examined for a clear point of registration (i.e., a CD). Figure 9 presents data from four neurons in which this was done; a positive ITD represents an interaural-time lead at the contralateral ear. BF and stimulus frequency and intensity are given in each box. As IPD functions were typically deeply modulated at stimulus frequencies across the excitatory portion of the cell's response area, so too are their ITD counterparts. As mentioned earlier, less deeply modulated functions are obtained near the extremes of the response area and for stimulus frequencies above $\sim 2,000$ Hz, even when the latter frequencies are near BF.

For each of the four neurons an average response to all frequencies studied within a neuron's response area has been represented by a "composite" ITD function (Fig. 9, *E-H*). These composite functions were constructed by combining the family of normalized ITD curves and averaging each data point with its four closest neighbors (5 point smoothing). Only that portion of the composite function from -400 to $+400 \mu s$ is displayed because it is within this range of ITDs that the composite peaks and troughs for these four neurons, as well as for the vast majority of recorded cells, are located. Although restricted to a $\pm 400 \mu s$ range, the peak-to-trough dynamic range for each cell is seen to span a different portion of the ITD axis. We return to a consideration of the composite functions later.

For some cells (e.g., Fig. 9, *B* and *C*), the presence of a CD is clearly suggested by an apparent point of registration

FIG. 9. *A-D*: normalized spike count plotted as a function of interaural time difference for 4 cortical neurons, illustrating the presence of a characteristic delay. *E-H*: normalized composite functions of the same 4 cortical neurons. Vertical arrows indicate the position of the CD estimated from the slope of the ϕ -vs.-frequency plot seen in Fig. 10A.

among the normalized ITD functions, especially if this relative point appears at sharp peaks. However, even in these cases the troughs of functions were often fairly broad plateaus having low spike counts in registration within the physiological range of some ± 400 μ s. In other cells (e.g., Fig. 9A), an ITD between the peak and trough seems just as appropriate as the point of registration. In Fig. 9D the wavering among ITD functions seems to make the choice of registration based on inspection of the curves almost arbitrary.

The CD may also be estimated from the slope of the function which relates ϕ to stimulating frequency (Yin and Kuwada 1983b). If a given neuron possess a CD then this function will be linear, and its slope will represent the change in phase with respect to frequency that occurs be-

cause of the invariant time delay (i.e., the CD). The y -intercept of this linear function, referred to as the characteristic phase, will be associated with the CD. The characteristic phase will be at the peaks for a y -intercept of 0° , at the trough for a y -intercept of 180° , or between peak and trough for intermediate values.

Figure 10A presents plots of ϕ -versus-stimulus frequency for the four neurons illustrated in Fig. 9. The correspondence between them is indicated by the letters associated with each fitted curve. Each function is fitted with a linear regression line. The slopes are the CDs and are indicated by the vertical arrows on both the corresponding individual and composite ITD functions in Fig. 9. The cell represented in Fig. 9, *B* and *F*, and the neuron in Fig. 9, *C* and *G*, both yield a CD near where the functions reach

their peaks. The results illustrated in Fig. 9, *A* and *E*, and Fig. 9, *D* and *H*, show a CD located between the peak and trough.

Results obtained by the use of this method of analysis for six other neurons studied under static or dynamic conditions are illustrated in Fig. 10, *B* and *C*, respectively. We used the method of Yin and Kuwada (1983b) to assess whether the data points were significantly ($P < 0.005$) linear. All but two of the neurons (not shown here) subjected to this analysis met or exceeded this criterion of linearity. Although the scatter of values of ϕ about the regression line was assumed to be random, the degree to which data points were distributed about a regression line showed considerable variation. Often noticeably regular oscillations were present (e.g., ■ in Fig. 10), and these could be large enough to suggest that the function was not linear (see Kuwada et al. 1987). In a few cells, only one or two of the sampled stimulus frequencies yielded mean phase angles that appeared markedly distant from the fitted line. Finally, even though the fitted function was judged to be

FIG. 10. *A–C*: mean interaural phase (ϕ) plotted as a function of stimulus frequency for 10 cortical neurons. Data fit by a linear regression equation. Functions labeled “A,” “B,” “C,” and “D” in *A* correspond to 4 neurons illustrated in Fig. 9.

FIG. 11. *A*: scatter plot showing the distribution of mean ITD as a function of stimulus frequency for all spike count-vs.-ITD curves from all neurons in the sample. Filled squares represent positions of peaks of spike count-vs.-ITD curves; open squares, troughs. Horizontal dashed lines are placed at a delay of $\pm 400 \mu s$, which is the approximate physiological limit for the adult cat. *B*: distribution of the ITD at peaks and troughs of composite ITD functions.

linear, the estimated CD could be well outside the expected physiological range (e.g., Fig. 10C, □).

The CD values (in microseconds) derived from 13 neurons with linear ϕ -versus-frequency functions were as follows: $-1,225$, -210 , -139 , -138 , -52 , -38 , $+33$, $+56$, $+102$, $+147$, $+174$, $+260$, and $+303$. These data reflect the observation that the majority (87%) of neurons tested possessed a CD and that most (92%) estimates of CD were within the range of $\pm 300 \mu s$ and rather equally distributed about zero ITD (average = $+49 \mu s$). Furthermore, the distribution of characteristic phases (-160 , -78 , -62 , -19 , $+29$, $+30$, $+33$, $+47$, $+58$, $+92$, $+104$, $+130$, and $+150^\circ$) was also rather uniform with no prominent modes near 0° (peak) or 180° (trough).

Figure 11A illustrates in the form of a scatter plot the ITD corresponding to the peaks and troughs of ITD functions derived from responses to all of the individual stimulus frequencies employed in the study. Approximately 85% of all curves had their peak or trough located within $\pm 400 \mu s$ and above ~ 800 Hz the value is closer to 99%. Most (91%) of those peaks occurred at a positive ITD (average, $+142 \mu s$), whereas most (79%) of the troughs had a negative ITD (average, $-144 \mu s$).

Composite functions obtained from either the simple (Yin and Kuwada 1983b) or weighted (Kuwada et al. 1987) averaging of ITD curves obtained at nearly equal stimulus

levels may be a more useful metric of a neuron's ITD sensitivity. The distribution of ITDs at the peaks and troughs of composite ITD functions obtained from the 21 cells studied with three or more frequencies in this experiment is shown in Fig. 11*B*. Because positive ITD corresponds to a contralateral stimulus lead, most of these neurons would be expected to respond maximally to a sound source located in the contralateral sound field. Further analyses of the distributions shown in Fig. 11 indicated that for 81% of the cells tested both the composite peak and trough were within the $\pm 400 \mu\text{s}$ range of ITD and, therefore the dynamic range of these cells is wholly positioned within the normal physiological range (Roth et al. 1980).

DISCUSSION

In the course of these studies we discovered that sensitivity of a subset of cat AI neurons to the IPD of low-frequency tones is very similar to that recorded in the medial superior olive (Yin and Chan 1990) and central nucleus of the inferior colliculus (Kuwada et al. 1979; Yin and Kuwada 1983*a,b*). In all these areas IPD-sensitive cells display a number of common attributes including a strong dependence on the effective stimulus frequency but relative independence of the neuron's BF; an insensitivity to the onset-time-disparity cue that normally accompanies the interaural-phase-disparity cue of time-delayed tones; the demonstration of a CD for some cells, and the ability of certain neurons to respond to continuously changing as well as to static IPDs.

No cortical neuron in our sample, and none so far reported in the literature, have been shown to exhibit a phase-locked response to monaural-tonal stimulation, which would be expected if a major site of binaural interaction underlying IPD sensitivity were located at the cortical level. Very few cells in the medial geniculate body (Rouiller et al. 1979) are found to phase lock to monaural tones, and only a small proportion of inferior colliculus neurons are known to behave this way (Rose et al. 1966; Kuwada and Yin 1983). In the medial superior olive, on the other hand, monaural responses of IPD-sensitive neurons evoked by low-frequency stimulation of either ear are strongly phase locked to the stimulus cycle (Goldberg and Brown 1969; Yin and Chan 1990), which is what would be predicted for cells that cross correlate the phase-locked binaural input they receive from the large spherical cells of the anteroventral cochlear nucleus.

Sensitivity to IPD

IPD sensitivity is observed in neurons situated across the entire low-frequency cochleotopic array in AI; IPD-sensitive neurons had BFs that ranged from 190 to 2,400 Hz. In the inferior colliculus, Kuwada and Yin (1983) studied IPD sensitivity in neurons with BFs as low as 60 Hz and as high as $\sim 3,000$ Hz. Benson and Teas (1976) described such neurons in chinchilla auditory cortex with BFs between 250 and 1,800 Hz.

Significant IPD sensitivity was recorded from AI neurons at stimulus frequencies from 120 to 2,500 Hz. At low BF, below $\sim 1,500$ Hz, AI neurons exhibit strong IPD-sensitivity across the excitatory-response area, whereas at

higher BFs the IPD-sensitivity curves, which exhibit deep modulation, are flattened out at BF and frequencies beyond. These results indicate that the sensitivity to IPD depends more on the position of the stimulus in the excitatory response area than on the neuron's BF per se. Although we did not extend our study to neurons with BFs $> 2,500$ Hz, we think that this BF need not be the upper limit for cortical IPD sensitivity. Regardless of BF, at effective stimulus frequencies above $\sim 2,000$ Hz the sensitivity to IPD drops off demonstrably, which is expected if the mechanism that underlies IPD sensitivity at the cortex is limited primarily by the interactions in the lower brain stem of converging phase-locked afferent input from the two ears. Auditory nerve phase locking, which underlies IPD sensitivity, is exhibited at low-frequencies by high-BF fibers (Johnson 1980; Rose et al. 1967). In the inferior colliculus, IPD sensitivity is observed at stimulus frequencies as high as 3,100 Hz, but above 2,500 Hz the number of such cells is reported to decrease markedly (Kuwada and Yin 1983). In the dorsal nucleus of the lateral lemniscus (Brugge et al. 1970), IPD sensitivity has been recorded at the low-frequency edge of the response area of a neuron with a BF of 11.5 kHz.

Although IPD-sensitive cortical cells have been isolated in cat (Brugge et al. 1969; Orman and Phillips 1984), monkey (Brugge and Merzenich 1973) and chinchilla (Benson and Teas 1976), few detailed systematic studies of these neurons' properties were made. Because we were searching specifically for such neurons our sample is undoubtedly biased in that direction. Nonetheless, we found these cells in relatively high proportions (84%) in the middle cortical layers of AI. For these cells, spike count was a deeply modulated function of IPD with evidence of facilitation occurring at the most favorable IPD and, for some cells, out-of-phase inhibition occurring at the least favorable IPD. By comparison, $\sim 80\%$ of neurons in both medial superior olive (Goldberg and Brown 1969; Yin and Chan 1990) and inferior colliculus (Kuwada and Yin 1983) exhibit IPD sensitivity. In the inferior colliculus only 7% were reported to be affected by the onset-time delay of the tonal signal, another possible property of AI cells that we did not explore in this study.

It is of interest to consider changes that might occur between the phase-locked inputs to a binaural comparator and its phase-locked output. Yin and Chan (1990) showed that in the response to monaural low-frequency tones the degree of phase locking exhibited by medial superior olive cells was the same or exceeded that recorded in auditory-nerve fibers of the same CF for CFs below ~ 1 kHz. Cortical cells, unlike medial superior olive neurons, do not phase lock to monaural low-frequency sinusoidal stimuli. However, we hypothesize that the circular spike count-versus-IPD function exhibited by an AI cell is primarily an expression of the phase locking in the auditory-nerve fibers from which it is ultimately derived. In this treatment, we find that vector strength measured on the IPD functions from AI neurons is similar to that derived from period histograms generated by auditory-nerve fibers responding to the same stimulus frequency. Vector strengths equal to 0.7 or higher were measured on the IPD functions of AI cells for frequencies $< 1,700$ Hz, which is comparable to

that exhibited by a population of auditory-nerve fibers (Johnson 1980).

Sensitivity to dynamically changing IPD

We discovered that about one-fourth of the IPD-sensitive neurons isolated in AI responded to continuously changing IPD. In the inferior colliculus, on the other hand, >90% of IPD-sensitive neurons respond both to static and dynamic phase shifts (Yin and Kuwada 1983a). If one considers only those cells in the inferior colliculus showing an onset pattern of discharge, which is the typical response of a cortical neuron to tones, this difference in proportions between AI and inferior colliculus is reduced or disappears, for all inferior colliculus cells that were exclusively sensitive to static IPD also possessed an onset rather than a sustained-discharge pattern (Yin and Kuwada 1983a).

Cortical-IPD sensitivity to the dynamic binaural-beat stimulus appears to be related in a rather straightforward way to that displayed when static IPD are used. Estimates of the most favorable and least favorable IPD using dynamic stimuli were comparable with those obtained with static IPDs. In the inferior colliculus the same situation pertains (Kuwada et al. 1979; Yin and Kuwada 1983a,b).

Most AI (91%) and inferior colliculus (70%) neurons responded with maximal firing when the tone to the ipsilateral ear lagged that to the contralateral one. When converted to an ITD, the average peak value of inferior colliculus cells occurred at +181 μ s (Kuwada and Yin 1983) and the average trough value at -238 μ s. By comparison the results from this study of auditory cortex yield values of +142 μ s for the peak and -144 μ s for the trough. Some 85% of AI curves had peaks that fell within the physiological range of ± 400 μ s. This compares with 71% for the inferior colliculus (Kuwada and Yin 1983) and 92% for the medial superior olive (Yin and Chan 1990). Considering the differences in sampling methods and sample size among the three studies, the differences in results are small.

Unlike the case for most IPD-sensitive inferior colliculus cells, which respond to tones in a sustained manner, the function derived from responses of AI cells to dynamic phase shifts is markedly more peaked than the one obtained under static conditions. In some cases, all spikes in the period histogram fall into one or a few bins. This feature, which can be attributed to the fact that IPD-sensitive cortical cells respond only transiently to each cycle of the binaural beat, may be a mechanism whereby the cortex sharpens temporal coding by enhancing its synchrony to continuously changing phase cues.

We encountered two neurons that exhibited IPD sensitivity when the binaural-beat stimulus was employed but failed to do so in response to static shifts in IPD. A similarly small proportion of neurons displaying this property have been reported in the inferior colliculus of the cat (Kuwada and Yin 1983) and kitten (Blatchley and Brugge 1990).

Like the situation in the inferior colliculus (Yin and Kuwada 1983a), a relatively small proportion of IPD-sensitive AI cells exhibited a sensitivity to the rate of change of interaural phase caused by a change in the binaural-beat frequency. For relatively low binaural-beat frequencies, AI cortical cells exhibited a high probability of responding

each time the most favorable interaural-phase condition occurred in the dynamic stimulus. Consequently, for a fixed-duration stimulus, the number of spikes evoked during the 3-s tone to successively higher beat frequencies became greater because more potentially effective cycles were available. This situation pertains to moderate beat rates, but at higher rates there appears to be an adaptation to the stimulus. This would be expected of AI cells under anesthesia if we view the periodic excitatory input to an AI neuron created by a binaural-beat stimulus to be equivalent to that created by a series of repeated tone pulses. We found no sensitivity to direction of binaural beats, which are reported in small number in the inferior colliculus (Yin and Kuwada 1983a, 1984). Neither rate- nor direction-sensitivity is reported in the medial superior olive (Yin and Chan 1990).

Changes in intensity levels

Our experiments investigated variations in the average intensity produced by equal changes to both ears. In three neurons this intensity variation was carried out across the response area of the cell, whereas the remainder of our observations were limited to but one or two stimulus frequencies. Over much of the dynamic range, the peak of the ITD function associated with any neuron was relatively insensitive to changes in binaural intensity level; a change of 26 μ s in peak ITD for a 10-dB change in intensity level was typical. Larger changes in peak ITD were seen to occur near threshold and at intensity levels within the nonmonotonic portion of the spike count-versus-intensity function. In a much larger sample of neurons in the inferior colliculus, Yin and Kuwada (1984) report a high proportion of cells relatively insensitive to change in average or interaural-intensity change, between 0 and ~ 50 μ s/10 dB. In addition, they report a substantial number of cells in which SPL clearly had a strong influence on the most effective IPD. On the other hand, composite ITD functions were reported to be rather insensitive to changes in overall level. Brugge et al. (1969) in cat, Brugge and Merzenich (1973) in monkey, and Benson and Teas (1976) in chinchilla also found that the most effective IPD shifted little with changes in SPL, but again, their sample sizes were quite small. Observations at the level of the inferior colliculus in the unanesthetized rabbit also show an inconsistent change in ITD when average intensity is changed together with small mean changes in ITD (5–35 μ s) for a 10-dB change in binaural level (Kuwada et al. 1987).

Presence of a cortical CD

Finally, we found that IPD-sensitive AI neurons may exhibit a CD similar to that derived from inferior colliculus and medial superior olive cells. Benson and Teas (1976), in their study of auditory cortex of the chinchilla, found that IPD sensitivity examined over three stimulus frequencies failed to reveal a CD positioned at the maxima or minima of the spike count-versus-IPD functions, and thus they dismissed the presence of a cortical CD. The results of Brugge et al. (1969) in cat and Brugge and Merzenich (1973) in monkey were not adequate to resolve the issue. In this study we show that ITD functions derived from as many as

11 stimulus frequencies within the cell's excitatory response area were each deeply modulated but rarely in register at their peaks or troughs. Indeed, it was often difficult to determine by visual inspection whether or where such registration took place. However, for 13 of the 14 cells for which such multiple-frequency data were available, the mean interaural phase of the discharge was a linear function of stimulus frequency. Yin and Kuwada (1983b), who studied this mechanism in a much larger population of IPD-sensitive inferior colliculus neurons, showed convincingly that the slope of the simple regression line fitting this relationship is the CD for that cell, as first postulated by Rose et al. (1966), and that the y -intercept points to the location on the periodic spike-count functions where registration occurs.

Yin and Chan (1990) recently compared the IPD sensitivity of medial superior olive cells with that previously studied in the inferior colliculus under identical experimental conditions (Yin and Kuwada 1984; Yin and Chan 1988). Many similarities were found including a high incidence of IPD sensitive cells, apparent phase-related facilitation and inhibition, binaural-beat sensitivity, and the presence of a CD. Thus, although there may be possible differences between the two data sets in the proportions of cell classes represented and in the distributions of CDs, characteristic phases, and composite peaks, there is little, if any, distortion in IPD sensitivity per se between medial superior olive and inferior colliculus. In the same way, we find that the IPD sensitivity in the cortex differs little from that recorded in the inferior colliculus or medial superior olive, although several features of the response may vary from those typically observed there. With the use of these same criteria for comparison, we find that auditory cortical neurons, like cells in medial superior olive and inferior colliculus, are exquisitely sensitive to static and/or dynamic IPDs, exhibit a CD, and that these CDs may occur at peaks, troughs, or any intermediate interaural phase. Moreover, the distribution of CDs for AI cells is within that of CDs in the medial superior olive (Yin and Chan 1990), which in turn is very similar to the distribution found in the inferior colliculus (Yin and Kuwada 1983b). Our sample size is, however, simply too small to consider differences in the breadths of the distributions. These findings, taken together, are interpreted to mean that IPD sensitivity expressed in the lemniscal pathway is primarily the result of binaural interactions occurring within the medial superior olive, and that this sensitivity is relayed to and mapped on a subset of cortical neurons without demonstrable degradation at any of the intervening synaptic stations in the auditory pathways through which it is transmitted.

Relationship to sound-localization mechanisms

Certain primary auditory cortical neurons exhibit a CD, as originally defined in studies of the inferior colliculus by Rose et al. (1966) and later redefined by Yin and Kuwada (1983b). We also found that some AI neurons are extremely sensitive to continuously changing ITD, which would occur with a stimulus moving across the azimuths (see Kuwada et al. 1979). Furthermore, we emphasized that the peaks and troughs of deeply modulated individual

and composite ITD-sensitivity functions are, on average, contained within the $\pm 400 \mu\text{s}$ physiological range of ITDs seen in a normal adult cat (Roth et al. 1980). The composite function, which is a simple average of individual curves obtained from responses to pure tones, can be considered to represent the ITD-sensitivity function obtained from response to a more natural sound such as noise or multi-tonal complex (Yin et al. 1986). The ITD dynamic range, i.e., the ITD between peak and trough of the composite curve, differs among neurons and spans ITDs on both sides of the midline azimuthal representation of $0 \mu\text{s}$ ITD (see Figs. 9 and 10). Thus along with CD, the ITD dynamic range coupled with a cell's sensitivity to moving sources, as simulated by the binaural-beat stimulus, may provide additional cues to sound-source location.

It is clear that some localization behaviors are dependent on the integrity of AI (Jenkins and Merzenich 1984). To the extent that both static and dynamic ITD sensitivity signal the location of a source of sound in space, we may say that such information is available at the cortical level. We also know from other studies that information with respect to IID is available to AI cells as well (Phillips and Irvine 1981). And because sensitivities to both ITD and IID are expressed in spike output, they are also available to the targets of these neurons. Thus both ITD and IID information, which are extracted at lower brain stem levels, are available essentially undistorted to cortical circuits.

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