



Review

Stability of central binaural sound localization mechanisms in mammals, and the Heffner hypothesis

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ABSTRACT

Heffner (2004) provided an overview of data on the evolutionary pressures on sound localization acuity in mammals. Her most important finding was that sound localization acuity was most strongly correlated with width of field of best vision. This correlation leaves unexplained the mechanism through which evolutionary pressures affect localization acuity in different mammals. A review of the neurophysiology of binaural sound localization cue coding, and the behavioural performance it supports, led us to two hypotheses. First, there is little or no evidence that the neural mechanisms for coding binaural sound location cues, or the dynamic range of the code, vary across mammals. Rather, the neural coding mechanism is remarkably constant both across species, and within species across frequency. Second, there is no need to postulate that evolutionary pressures are exerted on the cue coding mechanism itself. We hypothesize instead that the evolutionary pressure may be on the organism's ability to exploit a 'lower envelope principle' (after Barlow, 1972).

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1. Introduction

Mammals vary widely in the acuity of their behavioural sound localization performance, as measured by the smallest detectable separation of sound sources symmetrically disposed around the midline. Heffner's (2004) thoughtful review on this topic provided a collation of data on the midline acuity of mammals varying widely in head size, hearing range, and ecological status. Her review offered a developing hypothesis of the evolutionary pressures that shape sound localization acuity across mammals. The key factors

examined were head size, limit of high-frequency hearing, and width of the field of best vision as inferred from retinal ganglion cell density measurements (Heffner, 2004).

Historically, head size has been thought to matter for sound localization acuity for two reasons. First, it sets an upper limit on the range of interaural time disparities (ITDs) available to the animal. Because sound travels at the same velocity to the two ears, the interaural distance is important in determining the size of differences in sound travel time to the two ears. ITDs come in two forms: an arrival time disparity or onset time disparity, and for periodic sounds, an interaural phase difference resulting from the imposition of the interaural arrival time delay on all subsequent periods of the stimulus waveform. In what follows, we use the term ITD to mean interaural phase difference, unless otherwise specified. ITDs are arguably most pertinent at low stimulus frequencies, for which the period of the stimulus is long relative to the sound travel time

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between the ears; interaural phase difference becomes ambiguous when it exceeds a half-period of the stimulus (Hartmann, 1999). Second, head size determines the depth of the acoustic shadow cast by the head (and pinnae), and therefore the magnitude of the interaural level difference (ILD) available; in practice, shadows are cast for sounds whose wavelengths are shorter than the head diameter (or pinna size). Animals with large heads thus potentially benefit from the availability of a larger range of both ITDs and ILDs to use in determining source azimuth. The limit of high-frequency hearing matters because animals with small heads are potentially able to capitalize on pinna directionality and head shadow effects if their hearing range extends to frequencies high enough to engage those acoustical factors.

Width of field of best vision, quantified as the horizontal width of the retinal region with the greatest ganglion cell density, also matters. Heffner and Heffner (1992; see also Heffner, 2004) hypothesized that one purpose of sound localization is to provide information for visually orienting to the sound source; it follows that the acuity of sound localization required to bring the source into the region of most acute vision is inversely proportional to the width of the field of best vision (Heffner, 2004).

Perhaps the most surprising feature of Heffner's work was an analysis of the partial correlations between these three factors and sound localization acuity (Heffner and Heffner, 1992). For what follows, most important was the fact that when the correlation between head size and acuity was partialled out, the correlation between width of field of best vision and sound localization acuity remained strong, while the reverse was not true. Indeed, with the visual factor partialled out, the correlation between head size and sound localization acuity was rendered statistically insignificant (Heffner and Heffner, 1992). The finding that sound localization acuity was most strongly correlated with the width of field of best vision stands even after doubling the number of species studied (Heffner et al., 2007).

Heffner was careful to emphasize that her account was of the evolutionary pressures that determined sound localization acuity, and not the mechanisms through which those pressures were exerted. The purpose of this article is to address this lacuna. The most obvious hypothesis linking the two ends of the correlation (width of field of best vision, and sound localization acuity) is that possession of a visual fovea results in, or is associated with, a refinement in the precision of localization cue coding, i.e., a tailoring of the accuracy of ILD and ITD coding to that required by the width of field of best vision. Surprisingly, our review of the neurophysiology of binaural sound location cue coding finds no evidence supporting that hypothesis. Indeed, we find a remarkable stability of binaural cue coding both across mammalian species, and across frequency within species. This evidence is expressed in both neurophysiological studies in animals, and psychophysical studies in man; it leads to a conclusion that mammals as a biological class may have inherited the same mechanisms for ITD coding and the same mechanisms for ILD coding, and they show similar ranges of encoded cue sizes. As such, we propose that evolutionary pressure is exerted not on the cue coding itself, but on the ability of a species to exploit the precision of coding by the 'best' neurons — an extension of Barlow's (1972) 'lower envelope principle.' An ability to base perceptual judgements selectively on the activity of neurons whose responses are the steepest functions of near-zero ITDs and ILDs may be the link between Heffner and Heffner's (1992) data on visual acuity and the stability of ITD and ILD coding mechanisms across mammals seen by others.

The review that follows is necessarily selective. Our focus is on the possible neurophysiological intermediaries between the Heffners' findings on the width of field of best vision on the one hand, and behavioural midline sound localization acuity on the other. Some recent reviews on sound localization mechanisms in general and

their evolution in mammals and other classes can be found elsewhere (Grothe, 2000; McAlpine and Grothe, 2003; Grothe et al., 2010; King and Middlebrooks, 2011).

2. Different mammals have highly similar mechanisms for the coding of binaural sound localization cues

Under normal (i.e., binaural hearing) circumstances, the two main cues used to localize sounds are the ILD and ITD generated by the spatial relationship between the sound source and the two ears. ITDs increase from zero for sources at the midline, almost linearly to azimuths near 90° in the lateral hemifields (Middlebrooks and Green, 1990). Maximal ITDs depend on azimuth, head size, and to some extent frequency, and can reach 750 μ s in man (Middlebrooks and Green, 1990) and 350 μ s in cats (Roth et al., 1980). ILDs also depend on source azimuth, head size, but more heavily than ITDs on frequency. In cats, there is a significant tendency for ILD magnitude to saturate for sources deep in the lateral hemifields (Irvine, 1987). In man, the patterns of ILD magnitude across azimuth and elevation can show some marked individual differences (Middlebrooks et al., 1989), but the general pattern is one of greater ILDs for sources located more laterally, with some tendency for ILDs to be near maxima over relatively broad ranges of lateral azimuths. The tendency of ILDs to saturate in the lateral hemifields renders ILD amplitude somewhat imprecise in specifying source azimuth when the source is in the lateral hemifields, and, indeed, free-field sound localization is less accurate for lateral sources (Makous and Middlebrooks, 1990). Localization of sources in the lateral hemifields is likely based on the dichotic cue information being supplemented by other data, namely the distribution of energy across frequencies (the 'head-related transfer function'), which is potentially available both monaurally and binaurally. In natural settings, this cue information may be supplemented further by patterns of sound reflection off environmental obstacles (Hartmann, 1999). The use of monaural spectral cues is probably mediated by comparison of the received source spectrum with some kind of internal template or representation of the source spectrum itself or of the listener's own head-related transfer function. The temporal dynamics of this processing are quite different to those of the 'online' analysis of ITDs and ILDs (Hofman and Van Opstal, 1998; Ishigami and Phillips, 2008).

In man, behavioural minimal audible angles around the midline supported by these mechanisms can be as small as 1° of auditory azimuth (Mills, 1958). Human free-field acuity is comparable to that in elephants (about 1° of azimuth: Heffner and Heffner, 1982), but most other species studied to date have midline acuities of 5° or worse, and many species have acuities in the range from 10° to 40° (see Heffner et al., 2007). A subtle, but perhaps important, feature of the Heffner et al. (2007) data is that the task required animals to discriminate which of two sound sources symmetrically disposed across the midline was activated. Technically, this is a lateralization (as opposed to a localization) task because the animal is required simply to differentiate left from right. As such, and on the assumption that the stimulus information used in the discrimination is the binaural ITD and/or ILD cue, the task reduces to the detection of a change in sign of the ITD/ILD from favouring one ear to favouring the other.

Under dichotic conditions, human subjects can detect ITDs as small as 10–15 μ s (Klump and Eady, 1956; Mills, 1958), and they can detect ILDs as small as 1 dB (Mills, 1958, 1960; Grantham, 1984). Unfortunately, there are few comparative studies of the sensitivity of animals to ITDs and ILDs. The data available suggest that despite differences in head size, there are similarities in threshold-level ITDs and ILDs detectable across species. Dichotic studies show that cats can discriminate ITDs as small as 15 μ s and ILDs as small as 1 dB

(Cranford, 1979). Pig-tailed monkeys have slightly higher thresholds (45 μ s and 3.5 dB; Houben and Gourevitch, 1979). Japanese macaque monkeys have ITD and ILD thresholds of about 26 μ s and 1 dB, respectively (Boester, 1994). Cats and macaque monkeys have similar widths of field of best vision (about 5°) while the human width of field of best vision is much smaller (less than 1°; see Heffner et al., 2007). Although preliminary, these data suggest that there is little correlation between width of field of best vision and ITD/ILD acuity. Because these species (e.g., humans and cats) differ in their free-field midline sound localization acuity but not in their ITD/ILD acuity, the data also suggest that the correlation between ITD/ILD acuity and free-field localization acuity may be weak. The only hint that any mammal has greater acuity than those seen in cats and humans comes from a free-field localization study of Jamaican fruit bats (Heffner et al., 2000) in which it was shown that the bat's ability to use ITD cues to distinguish left from right speakers at 30° eccentricity extended to 6.3 kHz. This is remarkable only in that 6.3 kHz is the highest frequency at which mammals have ever been shown to use ITDs. A tone of 6.3 kHz has a period of 160 μ s and the Jamaican fruit bat's interaural distance supports ITDs of over 75 μ s, so while the upper limit of ITD used in this species may be high, it does not constitute evidence of behavioural ITD acuity better than 15 μ s. Interestingly, these species (humans, cats, Jamaican fruit bats) also differ widely in their widths of field of best vision (about one, seven and 35°, respectively; Heffner et al., 2007). Thus, the available behavioural data suggest that variations in head size and widths of field of best vision are not accompanied by variations in sensitivity to the binaural sound localization cues.

2.1. Interaural time differences

In the case of both ITDs and ILDs, neural sensitivity to the stimulus cue information ultimately depends on a tracking of the amplitude of cochlear output on the two sides. The cochlea's transducers are the inner hair cells (IHCs), and the cochlea's output is constituted by spiral ganglion cells, each of which contacts a single IHC (Spoendlin, 1967). IHC depolarization is driven only by upward motions of the basilar membrane. The probability of an action potential being generated in a cochlear ganglion cell depends on the magnitude of the IHC depolarization response. For low tone frequencies, the stimulus period is so long that IHC depolarization can track the amplitude of basilar membrane motion driven by individual cycles of the stimulus; period histograms of auditory nerve responses to low-frequency tones or tone combinations show that the temporal distribution of spike discharges within the stimulus period follows the amplitude of the positive-going portions of the stimulus waveform quite precisely ('phase-locking': Rose et al., 1967; Brugge et al., 1969a). That is, the temporal distribution of spikes has an envelope that is akin to a half-wave rectified version of the stimulus waveform (see below). For high tone frequencies, the IHC depolarization also depends on the amplitude of basilar membrane motion, but the IHC membrane response is dominated by a direct current ('pedestal') depolarization component (Russell and Sellick, 1978). This has the consequence that IHC neurotransmitter release is more nearly continuous, the timing of cochlear nerve cell action potentials is random with respect to phase of basilar membrane motion (see Ruggero, 1992), and auditory nerve responses, i.e., average firing rate, follow the envelope of the stimulus.

Neural comparisons of the cochlear outputs occur first in the superior olivary complex (SOC; Irvine, 1992), although it is embellished at higher levels (Spitzer and Semple, 1998; Phillips, 2000; Park et al., 2004). Indeed, *de novo* binaural interactions occur as high as the auditory cortex (Kitzes et al., 1980). The SOC contains a number of nuclei, the most important of which for the present discussion are the medial (MSO) and lateral (LSO) superior olivary

nuclei. The inputs to both the MSO and LSO preserve the amplitude-coding properties seen at cochlear output. The two nuclei, however, have distinctly different tonotopic organizations in that the frequency representation of the former is highly biased towards low frequencies, while that of the latter is biased towards high frequencies (Guinan et al., 1972). This offers MSO cells a specialized opportunity to compare the instantaneous amplitudes of the two cochlear outputs on a cycle-by-cycle basis for low-frequency stimulus elements, and it affords LSO cells the opportunity to compare the envelope amplitudes of high-frequency components of binaural stimuli.

The MSO and LSO have unique cytoarchitectures and patterns of afferent and efferent connectivity (Schwartz, 1992; Smith et al., 1993). Irvine (1992) raises the question of whether the MSO and LSO circuitries constitute separate, parallel pathways for the processing of ITDs and ILDs respectively. He noted that species with well-developed MSOs and poorly developed or absent LSOs may nevertheless display good ITD discrimination at low frequencies and ILD discrimination at high frequencies (see also below), suggesting that ITD and ILD processing can be executed by different neural populations within a single nucleus. It follows from this that the anatomical separation of MSO/LSO circuitries seen in some animals (e.g., cats) may not be paralleled in other species. An interesting corollary of this point is that the functional distinction between the MSO and LSO, which is usually cast in terms of ITD and ILD coding, may in fact functionally be a distinction in frequency representation, with which the coding of behaviourally-relevant ITDs and ILDs of simple tones is correlated. On the other hand, as we shall see below, in some species the poor development of one or other nucleus does indeed appear to have behavioural correlates for the processing of ITDs or ILDs.

The responses of MSO cells, and the neurons in higher centers deriving their input from the MSO, are a sensitive function of the relative phases of the signals at the two ears (Yin and Kuwada, 1983; McAlpine et al., 2001; Brugge et al., 1970; Brugge and Merzenich, 1973; Spitzer and Semple, 1995, 1998; Hancock and Delgutte, 2004). The firing rate of the binaural cells reflects the extent to which phase-locked inputs from the two ears arrive in temporal coincidence. For simple tones, this sensitivity manifests as a cyclical relation of spike count to interaural delay, with the period of the response cycle being equal to that of the tonal signal. This is what one would expect from a coincidence-detection mechanism operating on phase-locked inputs from the two sides.

At the level of the MSO, this coincidence detection is mediated by interlaced, phase-locked excitatory/inhibitory inputs from the two sides (see Grothe et al., 2010 for review; but see also Spitzer and Semple, 1995). At higher levels, it can be mediated by a host of different binaural interactions. For example, one can find cells with a phase-locked excitatory input from one ear, and an inhibitory one from the other ear (Orman and Phillips, 1984); one can find cells receiving almost sub-(spike)-threshold excitatory inputs from the two sides which sum to generate spike responses in the binaural comparator only when a certain phase relation occurs in the stimuli at the ears, and thus in the inputs to the binaural comparator (Brugge and Merzenich, 1973); one can find cells that receive interlaced excitatory and inhibitory half-periods from one or both sides (Brugge et al., 1970; Orman and Phillips, 1984). These all have in common that stimulus amplitude-dependent responses from the two sides are phase-locked because the long stimulus period permits it, and this enables a moment-by-moment comparison of the stimuli at the two ears within each period of the stimulus, executed as a temporal coincidence detection. The diversity of binaural interactions mediating the interaural correlation likely reflects the convergence of inputs onto higher neurons, including those that support *de novo* binaural interactions (e.g., van Adel et al., 1999; Kitzes et al., 1980).

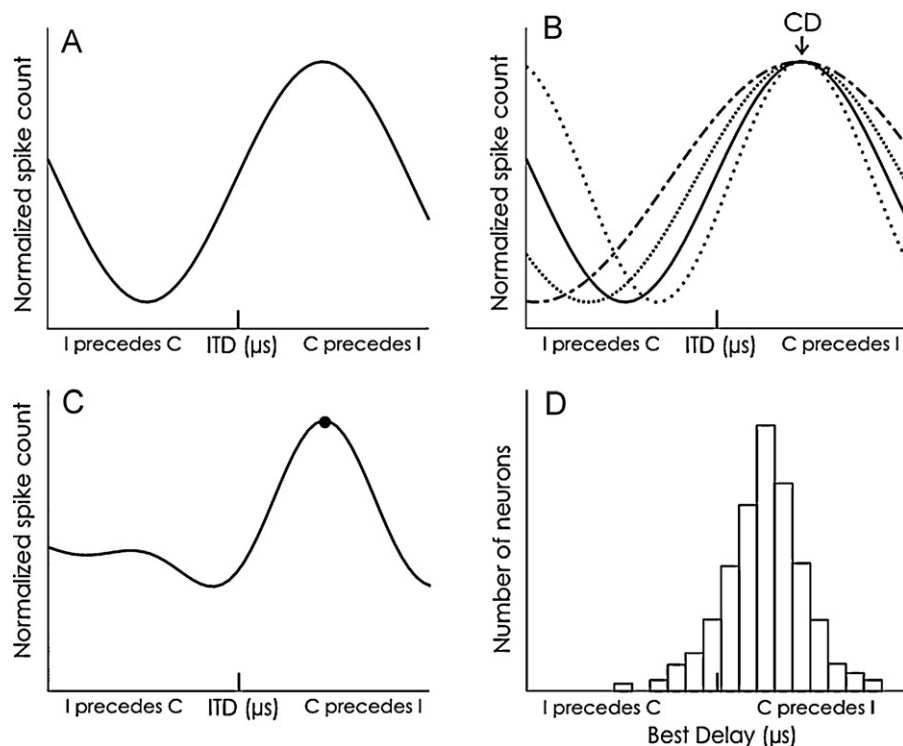


Fig. 1. Schematic diagram illustrating some general features of ITD coding. A shows an idealized spike count versus ITD function for one neuron tested at a single frequency. B shows idealized normalized ITD functions for a single neuron tested separately at four frequencies. In this instance, the response functions coincide at a single ITD, which is termed the 'characteristic delay' (CD) for that neuron. C shows an idealized average of the functions obtained when many frequencies are tested, each weighted by the absolute firing rates characterizing the function; it represents the composite delay function for that neuron. The filled circle depicts the CD for this neuron. D shows a schematic representation of the distribution of best delays typically encountered across a population of neurons. The distribution of characteristic 'peak' delays (of the kind schematically illustrated in B) would look very similar. More detailed description in text.

Central auditory neurons, of course, do not respond only to a single frequency, but to a range of frequencies described by the tuning curve (the envelope of best sensitivities across the neuron's limited effective frequency range). For each frequency within the tuning curve, low-frequency cells can be tested for their sensitivity to ITDs. An important concept to emerge from such studies is that of the 'composite delay function' which is the average of the ITD functions seen at all the frequencies tested, with the function at each frequency weighted by the firing rate characterizing it (Yin and Kuwada, 1983). Fig. 1 schematically illustrates how composite delay functions are obtained. Fig. 1A shows an idealized ITD function for a single neuron tested at its preferred frequency. Fig. 1B shows idealized and normalized ITD functions for the same neuron, this time tested at four frequencies. Note that the functions coincide (in this instance) at the peak of the functions, although in practice, that coincidence can occur at any point on the functions. The ITD associated with that coincidence is termed the 'characteristic delay' (CD) for that neuron. A composite delay function is computed by taking the average of the ITD functions, with each function being weighted by the absolute firing rates that characterize it. Seven frequencies were used to generate the idealized composite delay function shown in Fig. 1C. Note that it preserves the CD. If ITD functions are obtained for a sufficient range of frequencies, then the composite delay function is exceedingly close in form to that obtained with a noise stimulus whose bandwidth spans the effective frequency range of the neuron (Yin et al., 1986). Sometimes, the peak of the composite function is termed the 'best delay' (McAlpine et al., 2001; Hancock and Delgutte, 2004) to distinguish it from the characteristic delay (ITD at which delay functions for tones of different frequencies coincide). Composite delay functions typically, though not always, have peaks favouring the contralateral ear (i.e., contralateral stimulus phase precedes the ipsilateral phase). Fig. 1D

shows an idealized distribution of best delays; it is based on the data of Hancock and Delgutte (2004) and McAlpine et al. (2001). The bias in best delays towards ones favouring the contralateral ear has the result the steeply declining portion of the functions is often centered over zero ITD (compare: McAlpine et al., 2001; Brand et al., 2002; Hancock and Delgutte, 2004). The peaks and troughs of such functions are often outside the behaviourally relevant range of ITDs, suggesting that it is the steep portion of the function straddling zero ITD, rather than the peak (or trough), which is the informative part of the function (see Phillips and Brugge, 1985; Spitzer and Semple, 1995; McAlpine et al., 2001; Brand et al., 2002; Hancock and Delgutte, 2004).

Evidence has recently been presented in guinea pigs (McAlpine et al., 2001), gerbils (Brand et al., 2002) and cats (Hancock and Delgutte, 2004) that best delays are inversely correlated with cell's preferred tone frequencies. Cells with low preferred frequencies have large best ITDs, and cells with higher preferred frequencies have small best ITDs. The relationship is such that the best interaural phase (as opposed to best ITD in units of time) is nearly constant (at about 45°) across cells with different preferred frequencies. The finding has the corollary that the steep portion of the composite ITD function is centered close to zero ITD. The details of the neurophysiological mechanisms supporting the relationship between best frequency and best ITD are still being worked out, but it clearly involves precisely-timed inhibition at the level of the inputs to the MSO (Brand et al., 2002; Pecka et al., 2008; see also Grothe et al., 2010). These findings may require further confirmation because the relationships between best frequency, best ITD and best interaural phase were not reported in earlier detailed studies of ITD coding (e.g., Yin and Kuwada, 1983; Spitzer and Semple, 1995). These new observations are, however, interesting and important for three reasons. One is that the three

species in which the relationships have been reported vary widely in head size and width of field of best vision. The second is that because the preferred interaural phase difference is constant across the (low-frequency) tonotopic array, the mammalian ITD coding mechanisms may have evolved to encode interaural phase rather than interaural time. The third is that because best ITDs are dependent on neural best frequency, any model of sound localization mechanisms cannot be based on an 'identity code,' i.e., the lateral position of a sound source cannot be encoded by which particular neurons are discharging maximally.

These and other data led to the hypothesis that the lateral locus of a sound is encoded by the relative activation of two 'channels' of neurons: those activated maximally by sources with ITDs favouring the contralateral and ipsilateral sides respectively, with steep borders straddling the midline, i.e., zero ITD (see: Boehnke and Phillips, 1999; McAlpine et al., 2001; Phillips and Hall, 2005; Phillips, 2008; Dingle et al., 2010). Thus, as the stimulus ITD shifts from favouring one side to favouring the other, there is a maximal differentiation of response strengths in the two populations; for large ITDs favouring one side, there are less differentiated responses with comparable changes in ITD. This account parallels human behavioural sensitivity, in which azimuthal allocations of a source are steep functions of ITD for near-zero values of ITD, with a 'saturation' of azimuthal assignments for large ITDs (Yost, 1981). The two-channel model has received recent support from human evoked response studies (Magezi and Krumbholz, 2010) and from human magnetoencephalography studies (Salminen et al., 2009, 2010).

The two-channel model has not been without challenge. Harper and McAlpine (2004) modeled optimal coding strategies for ITDs. They concluded that for small-headed animals and/or low-frequency sounds, the two-channel model was optimal. For large-headed animals and/or higher frequencies, a distributed coding system based on a population of neurons with a homogeneous distribution of ITD tunings was optimal. For human beings, the two-channel model was deemed optimal for stimulus frequencies below about 400 Hz, and a distributed population was deemed optimal for higher frequencies. Nevertheless, empirical psychophysical evidence in man supports the two-channel model at frequencies below and above 400 Hz (Phillips and Hall, 2005; Vigneault-MacLean et al., 2007). Magezi and Krumbholz's (2010) human electrophysiological study of responses to 'inward' and 'outward' changes in ITD employed noise stimuli, and also supported the two-channel model. The use of noise stimuli makes it difficult to infer exactly which frequency-specific channels of processing contributed to the responses, but it is perhaps reasonable to assume that it was all of those that supported coding of the ITD stimuli. Salminen et al.'s (2009, 2010) magnetoencephalographic studies in man also produced data favouring a two-channel mechanism. Once again, recall that human beings and the smaller mammals (guinea pigs, gerbils, cats) whose ITD coding mechanisms were used as a neurophysiological basis for the two-channel model have vastly different widths of field of best vision.

A second challenge to the two-channel model is the assertion that it requires integration of information across the two cerebral hemispheres (Joris and Yin, 2007). The challenge is based on three facts. One is that each cerebral hemisphere is dominated by cells encoding spatial information for the contralateral auditory hemifield (e.g., Phillips and Brugge, 1985). The second is that animals with unilateral lesions rostral to the LSO/MSO display sound localization deficits only for sources in the auditory hemifield contralateral to the ablation (Jenkins and Masterton, 1982; Jenkins and Merzenich, 1984; Heffner, 1997). The third is that experimental lesion of the forebrain commissural pathways in animals (Moore et al., 1974) or congenital agenesis of the corpus callosum (or early callosotomy) in man (Lessard et al., 2002) has only minor consequences for sound localization acuity. What this assertion misses,

however, is that each side of the auditory forebrain contains populations of neurons that encode ipsilateral auditory space as well as the more numerous cells that encode contralateral auditory space (e.g., Stecker et al., 2005). The relative activity comparison required by the two-channel model can thus be mediated within a single cerebral hemisphere contralateral to the sound source (Dingle et al., 2010).

A commonality in the neurophysiological literature is the mismatch between the size of ITDs generated by the animal's head size, and the range of ITDs encoded by their central auditory systems. The latter is typically much wider in range than is the former, especially for small-headed animals (see Yin and Kuwada, 1983 [cats, with a relatively small fovea]; Phillips and Brugge, 1985; Kelly and Phillips, 1991 [rats, with a relatively broad width of best vision, and studied with click stimuli]; McAlpine et al., 2001 [guinea pigs]; Spitzer and Semple, 1995; Brand et al., 2002 [gerbils, with an exceptionally broad width of field of best vision]). Indeed, although somewhat anecdotal, the data suggest that the range of ITDs encoded by central mechanisms is relatively constant across mammals. Thus, the distribution of preferred (and characteristic) delays in cats (Yin and Kuwada, 1983; Hancock and Delgutte, 2004), guinea pigs (McAlpine et al., 2001) and gerbils (Spitzer and Semple, 1995) are extraordinarily similar, despite those species having very different head sizes and widths of field of best vision. In all three species, the distribution of best delays is highly peaked, centered near 250–300 μ s favouring the contralateral ear; most cells have best delays between –100 and +400 μ s, although the skirts of the distributions extend from about –500 μ s to +1000 μ s (contralateral stimulus time re: ipsilateral).

The mismatch between the available range of ITDs and those encoded by central neurons in small-headed animals has been apparent for many years, and led to the question of whether maximal responses to ITDs outside the behaviourally relevant range served a purpose other than direct coding of the cue for source azimuth (McFadden, 1973). It could be argued, indeed, that once it has been illustrated that the range of encoded ITDs across mammals is relatively constant, the mismatch is to be expected: in a two-channel model, the existence of neurons with best ITDs outside the behaviourally relevant range is a simple consequence of animal head size and audible frequency range. The alternative possibility, apparently refuted by the empirical evidence, is that the range of encoded ITDs could have covaried with head size. It is the relatively constant range of encoded ITDs across species with greatly different head sizes that first prompted a hypothesis that mammals as a class have a common set of mechanisms for ITD coding, and one which serves larger headed animals better than small headed ones (Phillips and Brugge, 1985; Kelly and Phillips, 1991). That is, large-headed animals have the advantage of being able to exploit a greater portion of the response-vs-ITD dynamic range simply because they have a larger range of ITDs available to them, and this supports greater sound localization acuity. Indeed one sees a modest correlation between head size and sound localization acuity in earlier comparative studies (Heffner and Heffner, 1982). It was only later that midline sound localization acuity was found to be independent of head size when width of field of best vision is partialled out (Heffner and Heffner, 1992).

The further question concerns the stability or generality of ITD coding mechanisms across the frequency domain within species. Historically, ITD coding at behavioural and neurophysiological levels has been studied for low-frequency sounds, presumably because low-frequency sounds are the most obvious instances in which ITDs are behaviourally relevant. In practice, however, even sounds with purely high-frequency spectra can contain low-frequency periodicities, and so it is an open question as to whether such sounds are subject to ITD coding using the same mechanisms as low-frequency tones.

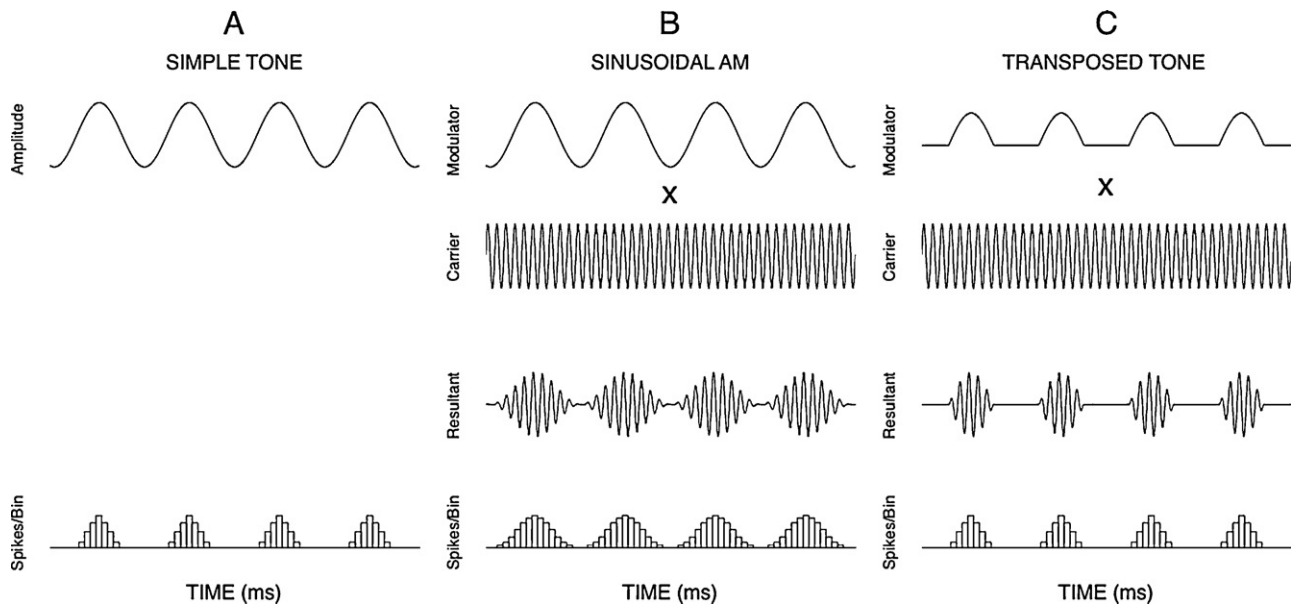


Fig. 2. Schematic diagram illustrating stimuli used in studies of ITD coding, and the average temporal distribution of spikes evoked by them in the cochlear nerve. A shows the case for a simple low-frequency tone. The upper panel shows the time waveform of the stimulus, and the lower panel shows the distribution of spikes, whose form follows from the coupling of spike discharge selectively to upward motions of the basilar membrane. B shows the case for sinusoidally amplitude-modulated tones. The uppermost panel shows the time waveform of the modulator, the next panel shows the waveform of the carrier tone, the third panel shows the waveform of the result of modulating the latter by the former, and the bottom panel shows the expected distribution of spikes driven by the stimulus. Note that the neural response contains no 'half periods' of inactivity of the kind seen in response to simple tones. C shows the case for transposed tones. In this instance, the low-frequency modulator (uppermost panel) is half-wave rectified. Multiplication of the modulator by the carrier tone (next panel) creates a stimulus (next lower panel) with periods of silence between the tone bursts. In turn, the temporal distribution of evoked spikes also contains 'half periods' of inactivity, and thus resembles that seen with a simple low-frequency tone, even though there is no low-frequency energy in the transposed tone stimulus.

Perhaps the simplest high-frequency sounds with low-frequency periodicities are sinusoidally amplitude-modulated tones. Henning (1974) was early to note that in man, such sounds could be lateralized on the basis of an ITD of the modulating waveform quite well. It has since become clear that central auditory neurons encode the ITDs of the low-frequency envelopes of sinusoidally modulated tones using binaural interaction mechanisms very similar to those used for simple low-frequency tones (Yin et al., 1984; Joris and Yin, 1995).

Low-frequency tones evoke auditory nerve responses whose temporal distribution resembles a half-wave rectified version of the stimulus waveform (see Fig. 2A). Sinusoidally amplitude-modulated tones do not, because multiplication of a high-frequency carrier by a low-frequency modulator results in a stimulus more likely to afford a full-wave rectified distribution of auditory nerve action potentials (at half the modulator frequency). This point is illustrated schematically in Fig. 2B. When a high-frequency carrier is multiplied by a low-frequency modulator, the resultant waveform consists of sinusoidally-modulated bursts of the carrier, with a zero-duration silent period between them. The expected average temporal distribution of cochlear nerve spikes driven by such a stimulus also has effectively a zero-duration period of silence between responses.

Bernstein and Trahiotis (2002) used what are termed 'transposed stimuli' in which a low-frequency filtered, half-wave rectified modulator tone was multiplied by a high-frequency carrier. A transposed stimulus affords the auditory nerve an opportunity to phase-lock to the modulated waveform in a fashion much more akin to that seen in response to low-frequency tones. We illustrate this point in Fig. 2C. When a half-wave rectified, low-frequency modulator is multiplied by a high-frequency carrier, the resultant waveform is a series of high-frequency tone bursts with equal-duration silent periods between them. The temporal distribution of phase-locked spikes driven by this stimulus would be expected to resemble much more closely that seen with the simple

low-frequency tone. Behavioural sensitivity to ITDs of the transposed stimuli was typically better than that seen in response to sinusoidally amplitude-modulated tones, and at least equal to that seen in response to low-frequency tones of the modulator frequency (Bernstein and Trahiotis, 2002). Still more recently, it has been revealed that neural sensitivity to ITDs of transposed stimuli follows the same pattern, and led to the conclusion that 'the neural mechanisms that mediate sensitivity to ITDs at high and low frequencies are functionally equivalent' (Griffin et al., 2005). These data suggest that the neural mechanisms of ITD coding are not only stable across mammalian species, but also across frequency within mammalian species.

2.2. Interaural level differences

The neural coding of ILDs is based on a comparison of level-dependent responses from the two ears. It has been studied almost exclusively in central neurons tuned to high tone frequencies, presumably because it is high tone frequencies for which the head and pinnae cast significant acoustic shadows and thus provide significant (i.e., usable, behaviourally-relevant) ILDs. As mentioned above, the lateral superior olive (LSO) has a tonotopic organization biased towards high frequencies and is the first major neural site of convergence of stimulus level information from the two ears (Boudreau and Tsuchitani, 1968). The most common pattern of binaural input mediating this comparison is excitation from one ear and inhibition from the other. Because both of those inputs are level-dependent (and probably saturating), the output of the binaural comparator is most commonly a sigmoidal function of ILD, with the steep portion of the function centered near zero dB ILD. Rostral to the crossed outputs of the LSOs, it is usually, though not always, the case that response rates are at ceiling for ILDs significantly favouring the contralateral ear, and at minima for ILDs favouring the ipsilateral ear (Phillips and Brugge, 1985; Park et al., 2004). Neurons vary in the slopes of their spike rate-vs-ILD functions, but their ILD dynamic

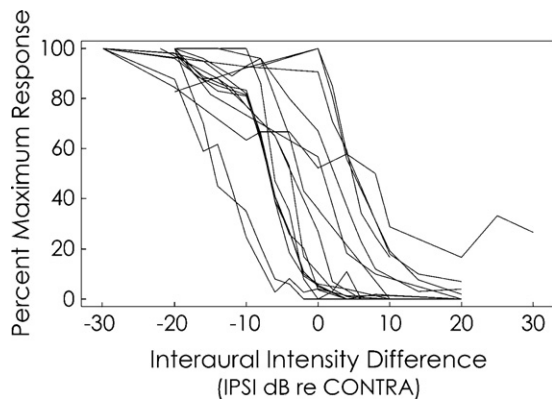


Fig. 3. Normalized spike count vs ILD functions for primary auditory cortical neurons in the cat. Each curve is a sigmoidal function of ILD, although they have somewhat variable slopes and dispositions along the ILD axis. Nevertheless, all are centered over small ILDs which themselves are associated with spatial locations close to the midline. Data are all from the study by Phillips (1980).

ranges are usually less than about 25–30 dB wide. This sigmoidal pattern of responsivity is seen throughout the auditory pathway (Brugge et al., 1969b, 1970; Brugge and Merzenich, 1973; Phillips and Irvine, 1981; Orman and Phillips, 1984).

The more rostral central auditory centers contain cells that encode ILDs using a range of other binaural interactions. These include nearly subthreshold excitatory inputs from the two sides which sum to evoke spikes only for ILDs near zero dB (Kitzes et al., 1980; Phillips and Irvine, 1981; Orman and Phillips, 1984), and cells with a short-latency excitatory and longer-latency inhibitory input from one side and an inhibitory input from the other (Phillips and Irvine, 1981). Note that these are the same patterns of binaural input mediating sensitivity to ITDs (above). Interestingly, at the most rostral levels of the auditory system, neural responses are quite often dominated by an onset transient (Phillips et al., 2002), and one might construe response strength to binaural stimuli as dependent on the degree of temporal synchrony with which inputs from the two sides arrive, i.e., coincidence detection (e.g., Kitzes et al., 1980).

Fig. 3 shows normalized ILD functions for a population of neurons studied in the primary auditory cortex of cats (based on data from Phillips, 1980). The functions are at ceiling for ILDs significantly favouring the contralateral ear, and at minima for ILDs favouring the ipsilateral ear. The functions have somewhat variable slopes, and are disposed across somewhat variable ILD ranges. Individually, neurons usually have ILD dynamic ranges of 10–20 dB (though sometimes much wider). Collectively, the population code spans about 10–15 dB on each side of zero ILD (i.e., a total width of 25–30 dB). Like ITD functions, the steep parts of the ILD functions are most often concentrated around relatively small ILDs, i.e., those that would be generated by sources located near the midline (see Park et al., 2004). Recall that ILDs are a most precise indicator of source azimuth for sources close to the midline. Comparable data have been presented for the rat (Kelly et al., 1998) which has a much wider field of best vision (Heffner et al., 2007). It was the conjunction of findings on the azimuth-ITD/ILD relationship and the ITD/ILD-spike rate relationship that prompted Phillips and Brugge (1985) to suggest that auditory forebrain neurons are most sensitive to cues for source azimuth over cue ranges that themselves most precisely specify source azimuth.

Once again, these data, and data from free-field studies of sound azimuth coding (see especially, Stecker et al., 2005) have led to the hypothesis of a two (Boehnke and Phillips, 1999; Phillips and Hall, 2005; Phillips, 2008) or three (Dingle et al., 2010) channel model of sound azimuth coding. The argument is that perceived sound

azimuth is specified by the relative outputs of two (left, right) or three (left, right, midline) neural channels, much as in the case of ITD coding. The outputs of the neural channels are most differentiated for ILDs near zero (and therefore for source azimuths near the midline), and this contributes to behavioural acuity being greatest for near-midline source locations and poorer for sources in the lateral hemifields.

The hypothesis that common sets of neural coding mechanisms characterizes sound localization behaviour across the mammalian frequency range arguably requires that ILD sensitivity be manifested for low-frequency stimuli, even in the event that low-frequency stimuli do not generate significant ILDs. This is indeed the case. First, the human scalp-recorded frequency following response (an index of the phase-locked responses to low frequency stimuli) is sensitive to ILD over a range of ILDs that matches the dynamic ranges of central neurons (Krishnan and McDaniel, 1998). Second, human scalp-recorded steady state responses to periodic reversals of the interaural phase of a noise stimulus are sensitive to ILDs over roughly the same 25 dB range (Massoud et al., 2011). This particular response to ILDs can be mediated only by low-frequency neurons because only low-frequency neurons will be sensitive to phase reversals of a noise carrier. Third, not only do humans have ILD detection thresholds of less than 1 dB at frequencies well under 1 kHz (Mills, 1960), but they show a mapping of ILD to intracranial auditory azimuth at low frequencies as they do for high frequencies (Yost, 1981). Fourth, neurophysiological studies in cats show that low-frequency central neurons are directly sensitive to ILD, and the ITD sensitivity of low-frequency neurons is dramatically affected by the imposition of an ILD, in the form of ILD-driven modulations of the depth of the ITD function, or a time (latency)-intensity trading (Yin and Chan, 1990; Tollin and Yin, 2005).

The foregoing data render it unequivocal that the neural ILD sensitivity seen in response to high-frequency stimuli extends to low-frequency sounds. We had previously seen that the neural sensitivity to ITDs, typically studied for low-frequency sounds, extends to the amplitude envelopes of high frequency carriers, i.e., high-frequency cells can possess the same ITD coding mechanisms seen in responses to single tones by low-frequency cells. Moreover, at their root, both ITD and ILD coding depend on a comparison of basilar membrane motion amplitude on the two sides; for low frequency stimulus elements, the periods are sufficiently long that cochlear output can track the phase of basilar membrane motion, and this in turn permits central neurons to compare the phase of the stimulus at the two ears in a process that can be described as a temporal coincidence detection. Next, the ITD and ILD coding mechanisms are phenomenologically similar across species of different head sizes (and widths of field of best vision), and the data available suggest that there are not major differences in the dynamic ranges of the ITD or ILD cues the species encode. Finally, behavioural data in man (Klump and Eady, 1956; Grantham, 1984) and in cats (Cranford, 1979) which have a much smaller head size, indicate that the smallest detectable ITDs and ILDs are close to 10–15 μ s and 1 dB, respectively. Taken together, these data suggest that the mammalian biological class has common sets of mechanisms for the encoding of ITDs and ILDs.

The foregoing might be interpreted as a challenge to the duplex theory of sound localization. The duplex theory is based on free-field studies of human minimal audible angles revealed in studies using pure tones (see Moore, 2003 for review). It argues that we use ITDs at low frequencies and ILDs at high frequencies. Free field studies (e.g., Mills, 1958) show that humans have small errors in midline localization at low frequencies and at high ones, but not at intermediate ones (around 2 kHz). The reasons for this are (1), the intermediate frequencies are above those for which neural coding of ITDs of simple tones is possible, and (2), the intermediate

frequencies are ones for which the head does not cast a significant acoustic shadow, so no ILDs are available to use to perform the task. Nothing in our argument disputes this. The fact that we can contrive high-frequency stimuli (e.g., transposed tones) to reveal high-frequency sensitivity to ITDs does not dispute that the nervous system is insensitive to ITDs of simple high-frequency tones. The fact that the neural machinery for processing of low-frequency stimuli is sensitive to ILDs does not dispute that ILDs do not exist for low frequency sounds. Our point is only that the neural machinery serving the processing of all frequencies is capable of encoding ITDs and ILDs if the stimulus design offers it opportunity to do so.

2.3. Departures from the general model

There is behavioural evidence, however, of exceptions to this general rule. In particular, hedgehogs (Masterton et al., 1975) and some bats (e.g., Koay et al., 1998) have extraordinarily poor abilities to use interaural phase difference cues to execute free-field sound localization. In addition, horses (Heffner and Heffner, 1986) and pigs (Heffner and Heffner, 1989) are very poor in their ability to use ILDs to localize free-field sources. In each of these cases, animals were tested for their ability to identify which of two sound sources, located symmetrically about the frontal midline, was activated. Tonal stimuli were used, and the frequencies were selected to explore the ability of the animals to use ITD or ILD cues, by covering the frequency range over which ITD and ILD cues were predicted to be useful (or not) based on the animal's head size.

For all of these species, sound localization performance could be trained to excellence over the part of the frequency range expected to support one or other of ITD or ILD coding, but was extremely poor (performance at chance level) over the other. This suggests a wholesale loss of the use of one or other cue. Because of the within-subject design, it is difficult to make case that the animals were simply more motivated to respond to one cue than to the other. In the case of at least some species (hedgehogs, horses), the poor behavioural performance over the affected frequency range was paralleled by a poorly developed (or absent) MSO or LSO. This was not so clear in the case of others (e.g., pigs), although it is possible that anatomical defects, if present, were simply too subtle to be detected by the methods used.

For the purposes of the present article, the important point is that these species' deviations from the general pattern are not so much quantitative adjustments of a single mechanism or model, but, apparently, the wholesale loss of one part of it. This is important because it prompts the view that binaural interaction mechanisms for ITD and ILD coding are already as optimal as the mammalian nervous system can provide, so that the only deviations from that optimum are impoverishments; these appear to take the form of losses of MSO or LSO (or frequency-specific) function. At the other end of the spectrum, extraordinary advances in sound localization behaviour appear to have been achieved by the addition of a wholly new mechanism, e.g., echolocation (Suga, 1982). To date, there is no compelling evidence that the central coding of passive sound localization cues in mammals in some way adjusts to species head size or width of field of best vision.

3. A 'lower envelope principle' can explain the relation between Heffner's evolutionary account and the central neurophysiological data

The obvious question that emerges from the foregoing concerns the nature of any variations in central neural coding mechanisms that might explain the relationship between sound localization acuity on the one hand, and width of field of best vision on the other. The foregoing review offered no evidence of specializations

of sound localization circuitry or range of cue sizes encoded in animals with vastly different widths of field of best vision and/or significantly different head sizes. Appeals to the possibility that Heffner (2004) failed to identify all of the relevant evolutionary pressures on midline sound localization acuity do not help because such appeals still leave unanswered the identity of the mediating auditory neural factor(s) that link an evolutionary pressure with behavioural acuity. Our working hypothesis is that one does not need to postulate covariation of behavioural sound localization acuity and ITD/ILD coding mechanisms. Rather, there is a continuous, if limited, distribution of the range of ITDs and ILDs encoded, and in the precision with which central neural responses specify the stimulus disparity size, and thus source azimuth. Evolutionary differences in acuity may reflect adjustments not of that encoded range or coding precision, but in the extent to which the animal is selectively able to base behavioural performance on the subset of neurons with the most informative code of small cue values.

This hypothesis is fundamentally similar to Barlow's (1972) 'lower envelope principle.' In its simplest formulation, this states that the detectability of an auditory event follows the envelope of the sensitivities of the neurons with the greatest sensitivities to that event. Consider the behavioural audiogram. Cochlear neurons serving any given position along the cochlea have varying absolute sensitivities (Ruggero, 1992; see his Fig. 2.4). The behavioural audiogram, which depicts the maximum sensitivity of the animal across the frequency domain, has a shape that reflects the thresholds ('follows the envelope') of the most sensitive neurons across the frequency range (e.g., Ruggero, 1992; see his Fig. 2.2). It is a small step to extend this principle from detectability of an event *per se* to discriminability of an event *change*. In this case, the lower envelope principle now makes the general argument that the lowest psychophysical threshold in discrimination reflects the activity of the subset of neurons whose own 'acuity' is also the best (see also Eggermont, 1998). At *suprathreshold* cue values, the population response may well be a vector sum of the outputs of the contributing neurons (Eggermont, 1998; Eggermont and Mossop, 1998). That vector sum population response is the neural instantiation of the two or three neural 'channels' whose outputs are compared in lateralization judgments in the perceptual architecture proposed by others (Phillips and Hall, 2005; Stecker et al., 2005; Vigneault-MacLean et al., 2007; Phillips, 2008; Dingle et al., 2010). Heffner's measurements of sound localization acuity are by design efforts to measure the smallest discriminable (i.e., *threshold*) change in source location around the midline. This might be construed as equivalent to measuring the smallest ITD/ILD for which a change in sign (from left-favouring to right-favouring) is detectable. The neurons in the two channels that are best capable of indicating the change in location are those with the steepest spike rate functions for midline azimuths (or the cues for them). Our hypothesis is that in the absence of evidence that the neural coding of sound localization cues varies across species, evolutionary forces may operate instead on the selectivity with which the animal can access the 'best' neurons. Heffner's argument (Heffner, 2004; Heffner and Heffner, 1992; Heffner et al., 2007) is that the force shaping sound localization acuity is the width of field of best vision. Thus, animals with broad visual streaks require only relatively poor sound localization acuity to bring that region of the retinas in line with the auditory target; such animals are under only weak pressure to selectively access the 'best' auditory neurons. In contrast, animals with small foveas require highly accurate information about sound source location to align the foveas with the target; these species are under greater pressure to access the outputs of the 'best' auditory neurons. In a sound localization threshold study, this means that the behavioural performance must be based on the subset of neurons with the lowest neural thresholds for discriminating source position. Eggermont (1999) has previously appealed to a lower

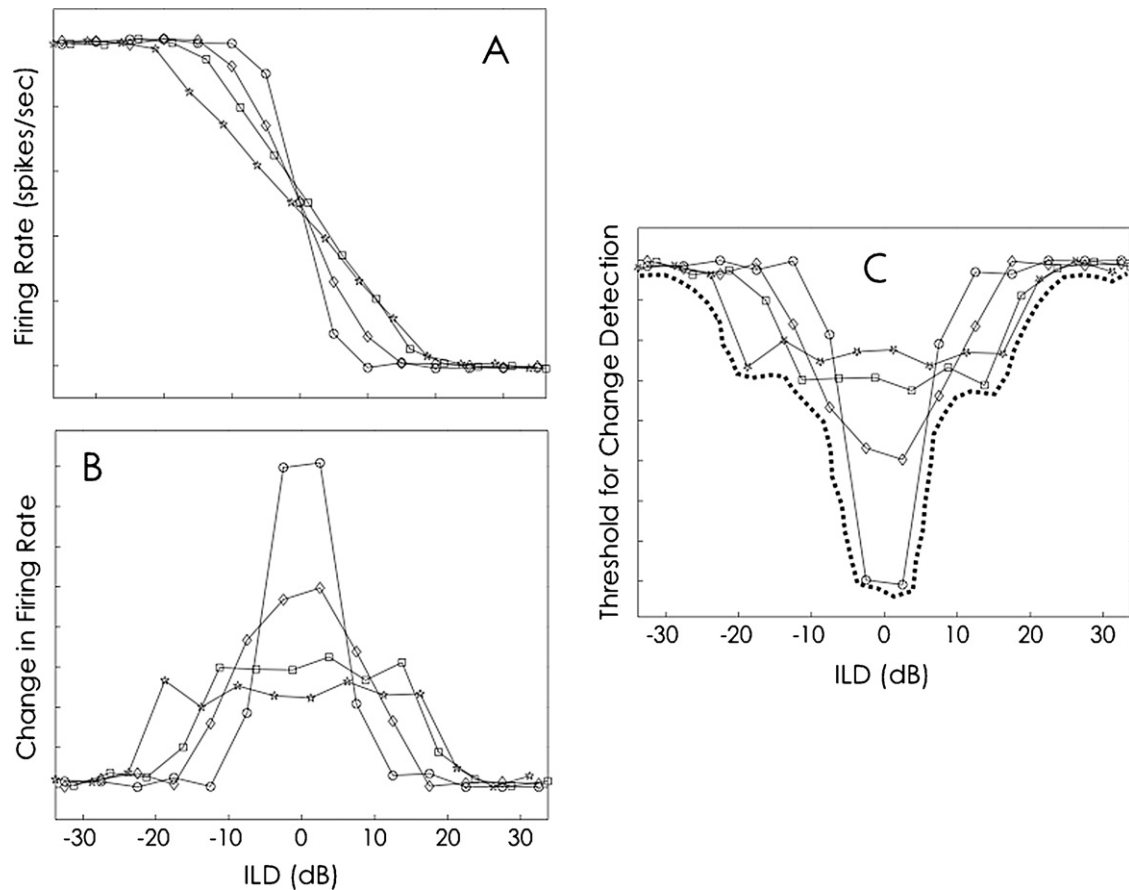


Fig. 4. Schematic illustration showing how Barlow's (1972) lower envelope principle might be expressed in the ILD stimulus domain. A shows idealized spike rate-vs-ILD functions for neurons with different ILD dynamic ranges. B shows the same data, this time expressed as the instantaneous slope of the ILD function, measured over a very small ILD range. Sensitivity to ILD change (or detection of the presence of an ILD at all) reflects the inverse of the functions in B, shown in C as the dashed line. A listener whose perceptual judgement is based selectively on the responses of the most sensitive neurons will have the lowest threshold for ILD detection. Any other weighting of the outputs of the neurons will result in a poorer (higher) threshold for ILD detection.

envelope principle to link human auditory temporal gap detection thresholds with the responses of cortical neurons studied with gap detection stimuli.

We illustrate our argument schematically in Fig. 4. Fig. 4A shows idealized curves intended to represent the spike rate-vs-ILD (or, in principle, ITD) functions of neurons with different dynamic ranges, and for the purpose of simplicity, all are centered on 0 dB ILD. These are the responses of neurons contributing to one of the two channels in the two-channel model. Some neurons have narrow, steep functions, while others have broader encoded ranges and thus shallower slopes in their response functions. The ability of a neuron to respond differentially to slightly different ILDs is expressed as the instantaneous slope of the function for the stimulus range over which the small ILD change occurs. For the functions shown in Fig. 4A, these slopes are shown in Fig. 4B. As one would expect, the neurons with the greatest change in firing rate for a given change in ILD are those whose spike count-vs-ILD functions are the steepest. The best sensitivity of the organism to a small change in ILD is the envelope drawn around the functions of Fig. 4B when they are inverted (dotted line in Fig. 4C). In the two-channel account, this is of benefit because as stimulus azimuth around the midline alters, the rate of change of difference in channel spike output will be greatest for precisely those neurons. The listener whose perceptual judgments are based selectively on the activity of the neurons with the greatest ILD discriminating power will have the lowest threshold for ILD change detection, and by extension, the smallest behavioural minimum audible angle. Any other weighting of the outputs of the ILD-sensitive neurons

results in a poorer ILD change-detection threshold, and by extension, a larger behavioural minimum audible angle. Parenthetically, the danger in this argument lies in the assumption that it is only sensitivity to binaural cues that mediates free-field sound lateralization acuity at the midline. In principle, it is possible that monaural cues also play a role. It is, however, precisely at midline azimuths that the binaural cues themselves are the most sensitive function of source eccentricity, and it is difficult to imagine that monaural cues could be informative enough to support empirically observed sound lateralization thresholds. In this regard, one recent study showed that even the best long-term monaural listeners do not have azimuthal localization judgments for near-midline sources as good as do normal-hearing control listeners (Slattery and Middlebrooks, 1994).

An objection to our use of a lower envelope principle in the present context might be derived from the work of Hancock and Delgutte (2004). They studied ITD coding in the cat's auditory mid-brain, and then used modeling techniques to try to explain human ITD discrimination based on the slopes of neurophysiological ITD functions. In particular, they were concerned with the fact that ITD acuity is greater for small ITDs than for large ones, especially for noise stimuli. Their modeling data, far from supporting a lower envelope principle, in fact suggested that pooling of neural outputs across frequency provided a better account of human ITD discrimination, and for both tonal and noise stimuli. This was due, at least in part, to the alignment of ITD functions over zero ITD (see Hancock and Delgutte, 2004, for details). In practice, however, their finding does not contradict the present hypothesis. Variance in ITD

discrimination at different base ITDs within a species is simply not the same thing as differences in lateralization acuity (i.e., detection of a change in sign of ITD/ILD from favouring one ear to favouring the other, which is what the Heffner and Heffner task likely measures) across species that are equipped with the same ITD/ILD coding mechanisms. In a sense, these are almost orthogonal dimensions. It is quite possible that a pooling principle explains acuity differences for ITD acuity across different base ITDs on one or the other side, while differences in the application of a lower envelope principle explain the acuity differences for left-right discriminations. In this regard, acuity of left-right discriminations tend to survive cortical lesions in cats (Cranford, 1979) and ferrets (Kavanagh and Kelly, 1987), while localization discriminations within the affected auditory hemifield(s) do not, in cats (Jenkins and Masterton, 1982; Jenkins and Merzenich, 1984), primates (Heffner, 1997) or ferrets (Kavanagh and Kelly, 1987). These data suggest that left-right laterality discriminations are mediated by fundamentally different neural mechanisms than those mediating localization discriminations, and thus support the contention that our hypothesis is not at all incompatible with that of Hancock and Delgutte (2004).

A second objection to our argument might be that a lower envelope principle in fact contradicts the two- (or three-) channel localization model. This is because the model is based on the premise that it is in some way the aggregate or vector response of neuron response rates in the channels that contributes to relative firing rate comparison, while our application of a lower envelope principle counters this by suggesting that the discrimination is based selectively on the responses of the 'best' neurons. In practice, this is precisely our point: that 'expert' *threshold* performance develops from the ability to differentially weight the activity of the 'best' neurons in the channels contributing to the perceptual decision-making process. That is, the neurons within a channel that are most critical in signaling a change in location will be those with the steepest functions and it is the ability of a particular species to differentially weight the responses of this group of neurons in the decision-making process that determines their localization/lateralization acuity.

It is not clear what mechanisms the brain implements to exploit a lower envelope principle. It is, however, an axiom of perception and psychophysics that subjects improve their thresholds for detection or discrimination with practice. At least in part, that improvement likely reflects that the superior ('trained') perceptual performance is being based on better selective access to, i.e., more heavy weighting of, the responses of the neurons with the best thresholds rather than on any vector weighting of the responses of a larger population of neurons. This is not to dispute that perceptual development and perceptual learning cannot exploit neural strategies that do indeed 'fine tune' neural circuits serving discriminations, especially complex ones (e.g., face recognition, object recognition, speech discrimination). Indeed, the construction of perceptual prototypes or templates is perhaps fundamental to complex perceptual discriminations, and that construction must have neural correlates. However, practice effects in acuity along basic psychophysical dimensions (that is, dimensions that are neurophysiologically close to parameters mapped by the sensory transduction process), may not require appeal to this kind of perceptual learning process; they may require only improved efficiency at basing the perceptual judgement on the responses of the 'best' neurons.

There are examples in the literature of apparently superior sound localization performance among exceptionally practiced listeners, including orchestra conductors (Münste et al., 2001) and persons with early onset blindness (Lessard et al., 1998; Ohuchi et al., 2006). It is important briefly to consider these, to question whether the apparently superior performance reflects a genuine shift in the sensitivity of ITD/ILD coding mechanisms (some kind of

auditory 'hyperacuity') or some other practice effect. In this regard, superior accuracy in sound localization by blind listeners is not without dispute (e.g., Zwiers et al., 2001), although their processing efficiency might be enhanced (e.g., shorter reaction times: Liotti et al., 1998). The bulk of evidence to date suggests that any performance superiority in blind listeners may be restricted to monaural or other listening conditions that rely less on the coding of ITD/ILD cues than on spectral or other information; this is because sound localization under binaural conditions permitting use of dichotic cues (e.g., at the frontal midline) is already at ceiling in both sighted and blind listeners (Lessard et al., 1998; Zwiers et al., 2001). The performance advantage in the blind listener is restricted to sources in the lateral hemifields. That is, the blind listener becomes expert in using information residing in details of the head-related transfer function and/or in sound reflections from the local environment that are used for localizing sources away from the midline.

The same conclusion is drawn from studies of minimum audible angles in visually-deprived ferrets (King and Parsons, 1999). These animals show no difference from control animals for minimum audible angles around the midline, but better than control performance (i.e., smaller minimum audible angles) for sources centered on 45° in the lateral hemifields (King and Parsons, 1999). Compatible data have been described for cats deprived of vision from birth (Rauschecker and Knipert, 1994). In this experiment, control and visually-deprived cats had to walk towards whichever of eight hidden free-field speakers, disposed at 45° eccentricity spacings, was activated in each trial. Performance was more accurate for the visually deprived animals for all source locations, but mostly so for sources at lateral azimuths or behind the animal.

Arguably the simplest interpretation of the evidence from visually-impaired listeners is that extreme practice has little or no effect on the use of ITD or ILD cues, but increases the listener's perceptual weighting of the more subtle cues of monaural spectrum or sound reflections. Parenthetically, even if we acknowledge that blind listeners who exercise extreme practice are more often at the superior end of the performance spectrum, it is far from clear that the same performance levels would not be reached by regular listeners given equivalent practice. For the present hypothesis, it is significant that studies of blind individuals have not yet offered evidence of a modulation in the neural coding of basic ITD and ILD cues.

4. Summary

Across mammalian species, there is an inverse relation between width of field of best vision and sound localization acuity around the midline, and it is independent of the animal's head size (Heffner and Heffner, 1992; Heffner, 2004; Heffner et al., 2007). This raises the question of what it is about the central processing of sound localization cue coding that varies among these species. Under optimal stimulus conditions, human beings have minimal audible angles as low as about 1° of azimuth (Mills, 1958), can detect ITDs as small as 10–15 μ s (Klump and Eady, 1956; Mills, 1958), and can detect ILDs as small as 1 dB (Mills, 1958; Grantham, 1984). Cats can discriminate ITDs as small as 15 μ s and ILDs as small as 1 dB (Cranford, 1979), despite the fact that cats have a much smaller head size than human beings. Likewise, Japanese macaque monkeys have comparable thresholds (26 μ s and 1 dB; Boester, 1994). These data suggest that the fidelity of binaural spatial cue coding in the mammalian central auditory system is as fine as it can get, and no advantage is accrued by increases in interaural distance beyond that required to be optimally exploited by the coding mechanism.

The neurophysiological data reviewed above suggest a stability of ITD/ILD coding mechanisms across mammalian species, and across frequency within species. The general form of the mechanisms is a sensitivity to the amplitude of basilar membrane motion

on the two sides, with the extension that for low-frequency sound components, signal period is so long that cochlear output can track basilar membrane motion within the cycle, enabling a central comparison of those tracking responses in a form described as an ongoing temporal coincidence detection. This set of mechanisms is general across the frequency range, with the exception of some species that appear unable to use one or other of the cues. The behavioural data also prompt the view that the neurophysiological performance supported by this machinery is the best that mammalian mechanisms can provide.

These findings collectively are the basis of our view that mammals as a biological class have acquired common sets of mechanisms for the coding of the dichotic sound localization cues (ITD and ILD). This stability raises a question about the central neural source of the species differences in free-field sound localization acuity that is coupled so strongly to width of field of best vision (Heffner and Heffner, 1992). Our hypothesis is that there is no direct adjustment or variation in strictly central auditory processes correlated with the visual factor. All that is required to relate the two findings (width of field of best vision, sound localization acuity) is evolutionary pressure on the need to exploit Barlow's (1972) lower envelope principle, namely pressure on the need to base perceptual judgments selectively on the outputs of the central neurons with the best discriminative performance. There is little such pressure in the case of species with broad visual streaks, but a great deal of it in the case of species with small foveas.

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