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Commentary

Recruitment of Neurons and Loudness

Commentary on "Encoding intensity in ventral cochlear nucleus following acoustic trauma: implications for loudness recruitment" by Cai et al. *J. Assoc. Res. Otolaryngol.* DOI: 10.1007/s10162-008-0142-y

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Loudness recruitment is one of the hallmarks of cochlear impairment. Recruitment refers to the perceptual phenomenon of sounds becoming rapidly louder with increasing sound level, leading to the somewhat paradoxical but common request of people with cochlear disorders "to speak louder" followed by the complaint to "stop shouting" (Moore 2003; Bacon and Oxenham 2004). It is commonly thought that the loudness of a sound reflects some aspect of the overall activation pattern of peripheral structures such as the auditory nerve, e.g., the total number of action potentials (Relkin and Doucet 1997). Therefore, an obvious physiological correlate of recruitment would be an abnormal growth of response with sound level. Many studies, both in patients and in various animal models of cochlear hearing loss, have documented abnormal response growth. However, these studies, which mostly examined gross evoked potentials, are contradictory regarding the lowest level of the auditory pathway where such changes are found. In this issue of *JARO,* Cai and colleagues (Cai et al. 2009) report a breakthrough observation based on careful comparison of how responses in the ventral cochlear nucleus (VCN) differ between normal cats and cats with acoustic trauma. The authors find that different physiological types of VCN neuron are differently affected by acoustic trauma; in particular, neurons with a rhythmic "chopper" response to short tone bursts show abnormally rapid response growth with increasing sound level.

Figure 1 schematically illustrates three different hypothetical changes in peripheral physiology that have been proposed to underlie recruitment. The most straightforward mechanism (Fig. 1A) is a steeper relationship between response and sound level. Such a change would be expected based on the loss of the cochlear compressive nonlinearity consequent to damage to outer hair cells (Robles and Ruggero 2001). A variety of psychophysical results are consistent with an explanation of recruitment in terms of a loss of compression of the amplitude of basilar membrane vibration (reviewed by Bacon and Oxenham 2004). However, studies of neural responses do not support this explanation. Abnormally rapid growth in response amplitude is consistently found in evoked potentials at anatomically higher levels, such as the auditory cortex, but not always at the level of the brainstem and midbrain (e.g., Popelar et al. 1987; Szczepaniak and Møller 1996; Qiu et al. 2000). Perhaps most striking is that rapid response growth, in terms of number of discharges per fiber or across a population of fibers, has not been a consistent finding in studies of the auditory nerve (Phillips 1987). A recent extensive study of auditory-nerve fibers in cats with acoustic trauma failed to find a simple increase in the slope of rate-level functions (plots of discharge rate as a function of sound pressure level) of auditory-nerve fibers (Heinz

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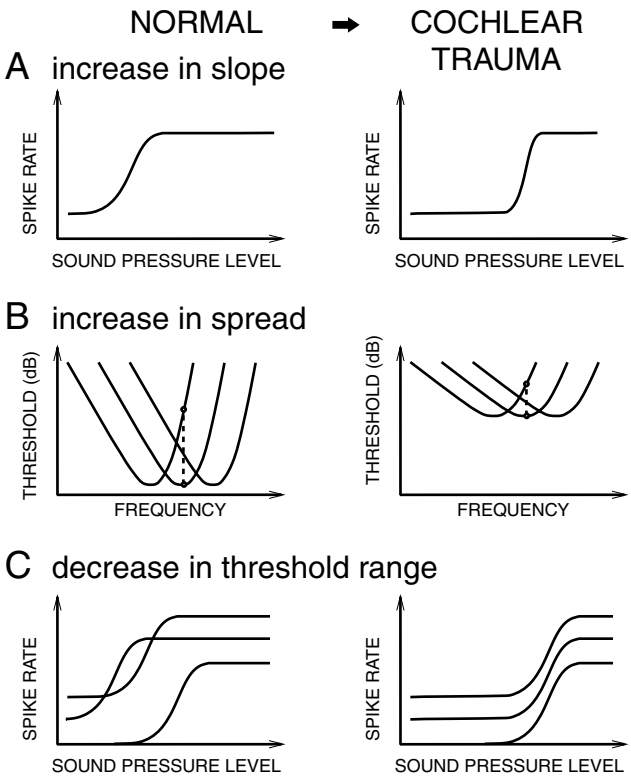

FIG. 1. Three hypotheses for loudness recruitment after cochlear trauma based on changes in auditory-nerve responses. *Left column* normal situation, *right column* after cochlear trauma. **A** Increase in slope of the rate-level function. **B** Spread of excitation. Note that a smaller increase in sound level is required to recruit the three fibers after trauma compared to the normal situation (*vertical dashed lines*). **C** Decrease in the range of thresholds.

and Young 2004). In fact, the nerve fibers showed a decrease rather than an increase in responsiveness at the intensities of relevance for recruitment.

A second hypothesis for recruitment involves widening of tuning, which is a well-known effect of cochlear trauma, documented both in mechanical and in neural responses (Liberman and Kiang 1978; Robles and Ruggero 2001). In response to narrowband stimuli, such widening should lead to a faster recruitment of activated auditory nerve fibers with increasing sound level (Fig. 1B) and, therefore, an abnormal growth in overall activity in the auditory nerve (Kiang et al. 1970; Evans 1975). Although a spread of excitation has indeed been observed in the auditory nerve (Heinz et al. 2005), it does not occur over the range of intensities for which patients show recruitment. In addition, psychophysical masking experiments in patients do not provide strong support for the spread-of-excitation mechanism (Moore et al. 1985).

The reason that steeper slopes (Fig. 1A) and spread of excitation (Fig. 1B) are not prominent after acoustic trauma seems to be a decrease in excitability of auditory nerve fibers, secondary to combined inner and outer hair cell damage (Liberman and Dodds 1984; Liberman and Kiang 1984). Thus, even though the cochlear mechanical response in these damaged ears may indeed show a steeper growth in response amplitude and a faster spread of activation with sound level, transduction of the mechanical response is hampered, so that the overall discharge rate of the auditory nerve, and its growth with sound level, is reduced relative to the normal situation.

A third hypothetical basis for recruitment is a decrease in the spread of thresholds (Moore et al. 1985; Zeng and Turner 1991). In healthy ears, there is some spread in the thresholds and dynamic ranges of auditory-nerve fibers tuned to similar frequencies (Liberman 1978; Schalk and Sachs 1980; Winter et al. 1990), which could be important for the coding of sound level (Delgutte 1996). Similar to the effect of widened frequency tuning, a decrease in the spread of thresholds would cause a steeper growth in response amplitude across the auditory nerve array, but again, empirical support for this mechanism is lacking (Heinz et al. 2005).

In summary, even though auditory-nerve responses of damaged ears are clearly abnormal, the relationship between these pathological changes and loudness recruitment is not as straightforward as suggested by the above hypotheses. It has been proposed that changes in temporal aspects of the responses across the population of nerve fibers are important for the encoding of sound level and that the loss of the compressive nonlinearity and the loss in frequency tuning after cochlear damage change these temporal patterns, leading to loudness recruitment (Carney 1994; Heinz et al. 2005). Such models suggest that changes in discharge rate may be more pronounced at the next synaptic level, where neurons receive convergent input from a population of nerve fibers. Combined with the results from central nervous system recordings after cochlear damage (Phillips 1987), these models point to the cochlear nucleus as a critical site to study. There have been two studies in which animals were exposed to high-level tones, and temporary changes in the activity of single VCN neurons were reported (Lonsbury-Martin and Martin 1981; Boettcher and Salvi 1993). In a minority of neurons, the rate-level functions were steeper, but it is not known whether such changes were confined to a specific class of neurons and whether they would be present after permanent acoustic trauma.

In the study reported in this issue, Cai et al. (2009) induced acoustic trauma in cats by overexposure to an intense narrowband stimulus. They then recorded from the VCN after a recovery period of one month or more. Consistent with previous work (Boettcher and Salvi 1993), they found a preservation of peri-

stimulus time histogram categories to best frequency (BF) tones, allowing statements regarding the effects of acoustic trauma on different cell types. In neurons with responses of the primary-like and primary-like-with-notch categories (which are associated with bushy cells in normal animals), the rate-level functions to tones at BF became shallower after trauma. This is not surprising since shallower slopes were also found in auditory-nerve fibers (Heinz and Young 2004; Heinz et al. 2005), which have powerful axosomatic terminals on bushy neurons. However, the rate-level functions were steeper in non-primary-like neurons: in neurons with chopper responses, likely stellate cells, as well as in some neurons that were difficult to classify ("unusual" response types in Blackburn and Sachs 1989). The rate-level functions of these non-primary-like neurons to broadband noise were also steeper. Thus, even though the mechanism of Fig. 1A is not found in the auditory nerve, it is present in at least one class of neuron in the VCN.

Cai et al. also studied spread of excitation (Fig. 1B) in VCN neurons, which was larger after acoustic trauma than in normal controls and larger in VCN than in the auditory nerve of traumatized ears. "Rate-balance" functions were constructed by comparing the sound levels required to obtain equal discharge rates in normal and traumatized ears; for non-primary-like neurons, such functions resemble loudness–balance functions in humans with cochlear hearing impairment. Another finding of interest was an increase in the variability of rate in VCN neurons in the animals with cochlear hearing loss (Cai 2007), which may explain why recruitment is not accompanied by improved behavioral detection of changes in stimulus level (Florentine et al. 1993). There was no indication for a reduction in spread of thresholds (Fig. 1C) of VCN neurons after acoustic trauma.

In conclusion, some hypotheses for recruitment that have been postulated, but not validated, at the level of the auditory nerve seem to hold for some classes of neurons in the VCN. At a general level, these results fit with the notion that parallel functional pathways are set up at the level of the cochlear nucleus (Young and Oertel 2004) and that stellate cells have a special role in encoding sound level over a wide range (Blackburn and Sachs 1990). The results of Cai et al. do not give insight into the mechanisms that underlie the changes in slope. As they discuss, acoustic trauma may induce functional changes in synaptic properties of auditory-nerve fibers or in electrical properties of stellate cells, or perhaps anatomical changes, such as axonal sprouting and synaptogenesis. Additional work is needed to identify the factors responsible and to understand which abnormalities in the inputs to these neurons trigger functional or morphological changes subsequent to cochlear trauma.

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