Comments

Comment by Moore

As you note, the hearing loss produced by acoustic trauma (relatively short exposures to high-intensity sounds) can result in both inner hair cell damage and outer hair cell damage. It has been proposed that loudness recruitment is mainly associated with the latter and that inner hair cell damage can lead to threshold elevation without marked loudness recruitment (Moore and Glasberg 1997). Also, steeper basilar-membrane input-output functions appear to be mainly associated with loss of outer hair cell function (Robles and Ruggero 2001). These considerations lead to two questions: (1) Is there any evidence that your noise-exposed cats had loudness recruitment for frequencies close to 2 kHz? (2) To what extent were basilar-membrane input-output functions steeper than normal at places tuned around 2 kHz? Perhaps you could answer the second question using the method proposed by Yates (1990).

Moore, B. C. J. and Glasberg, B. R. (1997) A model of loudness perception applied to cochlear hearing loss. Auditory Neurosci. 3, 289-311.

Robles, L. and Ruggero, M. A. (2001) Mechanics of the mammalian cochlea. Physiol. Rev. 81, 1305-1352.

Yates, G. K. (1990) Basilar membrane nonlinearity and its influence on auditory nerve rate-intensity functions. Hear. Res. 50, 145-162.

Reply:

In previous studies in our lab, normal-hearing cats were trained to perform vowel discrimination (differences in F2) and later exposed to the same acoustic-trauma paradigm used in our current study. In contrast to the ease in which they performed this task prior to noise exposure, these cats were much less willing to perform the F2-discrimination task following noise exposure. This observation was interpreted to be a result of loudness recruitment (Brad May, personal communication), although the evidence is only anecdotal at this point.

As suggested, the growth of basilar-membrane (BM) response was estimated from our data using the method proposed by Yates (1990). Estimates of BM I/O slope in the region of broadened tuning are shown (Fig. A1). BM growth was steeper than normal for BF tones (left panel) following impairment, with a greater loss of compression as the degree of hearing loss increased. Impaired BM response growth was also steeper for broadband noise (right panel). These analyses support the idea that steeper BM responses following sensorineural hearing loss do not produce consistently steeper AN RLFs.

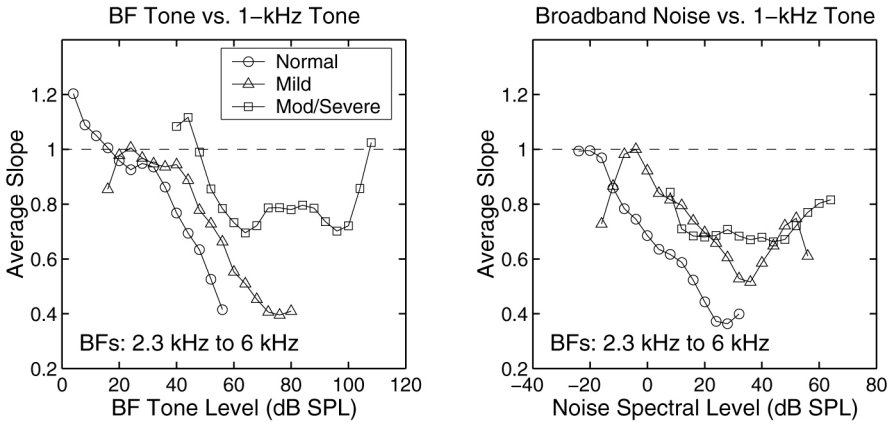

Fig. A1. Estimates of the BM I/O function were made for each AN fiber by comparing RLFs for "on-BF" stimuli to the RLF for a reference tone below BF that is assumed to have linear BM growth (Yates, 1990). Average slope of the BM I/O function (slope of 1 represents linear growth) was computed as a function of level based on all BFs between 2.3 – 6 kHz. Comparisons between BF tones and a 1-kHz tone (left panel) estimate compression at BF, while comparisons between broadband noise and a 1-kHz tone (right panel) include the effects of both compression and suppression.

Comment by Viemeister:

The reduction of the level dependence of phase in hearing damage is interesting partly because it may provide a test of the Carney (1994) model of low-frequency intensity coding. More specifically, because the Carney model relies on the level dependent phase shift at low frequencies, it can be expected, based on the data shown in Fig. 5, that intensity coding would be adversely affected by a hearing loss at low frequencies. One way to examine this psychophysically would be to measure intensity JNDs at low frequencies (less than 1 kHz) in persons with a low-frequency loss. If factors related to spread of excitation can be excluded then the prediction would be that the JNDs should be markedly elevated. However, the data from Schroder *et al*. (1994) for their two listeners with a "flat" hearing loss indicate no degradation in intensity JNDs at 300 Hz, 500 Hz, and 1 kHz both in quiet and, more importantly, in the presence of a highpass noise. Thus, these data, together with the results presented in Fig. 5, appear to contradict the basic assumption of the Carney (1994) model.

Carney, L.H. (1994) Spatiotemporal encoding of sound level: Models for normal encoding and recruitment of loudness. Hear. Res. 76, 31-44.

Schroder, A.C., Viemeister, N.F, and Nelson, D.A. (1994) Intensity discrimination in normal-hearing and hearing-impaired listeners. J. Acoust. Soc. Am. 96, 2683-2693.

Reply:

Yes, it is true that for conditions in which nonlinear phase is the dominant cue for level coding, performance should degrade following impairment. Theoretically, the nonlinear phase cues are dominant when spread of excitation is unavailable and the discharge rates of AN fibers with BFs near the tone frequency are saturated; however, when spread of excitation is available, predicted level discrimination of low-frequency tones is only slightly degraded by the removal of nonlinear phase cues (Heinz *et al*. 2001). Thus, to provide a true test of the Carney (1994) model it must be demonstrated that no spread of excitation was possible in the presence of the high-pass noise used in the Schroder *et al*. (1994) study. In particular, at low frequencies where physiological tuning is more symmetric, spread of excitation may have been possible below the tone frequency.