RESPONSES OF SINGLE AUDITORY UNITS TO RANDOM STIMULATION AND LATERAL INHIBITION

N.G. BIBIKOV

N.N. Andreev Acoustical Institute, Moscow, USSR

Cross-covariance functions (CCFs) between the spike sequence and the low-frequency noise used for the modulation of characteristic frequency (CF) and off-CF tones were obtained for cochlear nucleus neurons of unanesthetized lake frogs. The analysis of CCFs demonstrates the existing of the inhibitory inputs which seem to enlarge the dynamic range of amplitude modulation coding in secondary auditory units comparing with primary ones.

The single secondary-order neurons of the auditory system process the inputs they receive from the auditory nerve fibers. One of the main differences between the spiral ganglion cells and the cochlear nucleus units is the fidelity with which changes in sound amplitude are encoded. The auditory nerve fibers reproduce the stimulus amplitude variations without any considerable amplification. Moreover, this reproduction takes place only in the restricted dynamic operation range: 30-40 dB above the threshold /1/. In contrast the majority of the cochlear nucleus units demonstrate the amplification of the stimulus modulation depth and this amplification is observed over a great dynamic range /1,2/.

It is not difficult to explain the widening of the operation range by the convergency of many excitatory inputs on one cochlear nucleus cell. The fibers which provided these inputs can have different thresholds and different characteristic frequencies. The summed input excitation would be increased by sound amplitude increment regardless of how intense an initial amplitude was.

This mechanism can not, however, explain many important features of the amplitude modulation encoding in cochlear nucleus cells. First of all it doesn't explain the amplification of the stimulus amplitude changes in cochlear nucleus units. On the contrary the relative weight of the input changes evoked by a fixed sound increment has to be decreased as a result of the convergency of many fibers with various thresholds and various dynamic ranges. Also, the rate-intensity function for stationary tone would have the same operation range as that for encoding of small amplitude changes. It is not the case for the majority of the cochlear nucleus. The operation range for modulation encoding as a rule exceeds that of the average rate-intensity function considerably /2/. Finally, the convergency of the excitatory inputs with different characteristic frequencies must lead to the considerable widening of the tuning curve and the worsening of the frequency selectivity. Indeed, in cochlear nucleus neurons some widening of the tip of the tuning curve is observed, but their

edges are usually as sharp as those in auditory nerve fibers. Moreover in many cochlear nucleus cells a low-frequency tail of the tuning curve is absent /3/.

Most of these difficulties, however, could be overridden if the hypothesis of how cochlear nucleus neurons enhance the amplitude modulation information will include the delayed lateral inhibition influences. The amplification of the modulation can be explained by relative decreasing of the average impulsion rate and by the enhancement of the reaction to amplitude changes. The enlargement of the operation range for amplitude-modulation encoding may be treated as a direct consequence of the time difference between the excitatory and inhibitory inputs. For intense stimuli the steady-state rate-intensity function is flat. The stimulus increment, however, causes the initial excitatory input that evokes the spike generation. The succeeded inhibition can not prevent the reaction to the increment. Finally, the absence of the considerable widening of the tuning curves in secondary-order units is easily explained if we assume that the inhibitory inputs come from the larger region of the cochlea than the excitatory ones. It is well known that many cochlear nucleus neurons exhibit strong inhibitory components in their responses to off-characteristic frequency (CF) tone bursts /4/.

In our study we have investigated the dynamic properties of the responses of cochlear nucleus neurons in unanesthetized curarized lake frogs (*Rana ridibunda*). The stimuli were long-duration (100 sec) tones of different intensities and frequencies. They were amplitude-modulated by random low-frequency (0-200 Hz) noise with the modulation depth (root mean square) - 16%. The dynamic properties of the cells were described by cross-covariance functions (CCF) between the recorded discharge sequence and the modulation waveform.

The details of the methodology are described in our previous paper /5/. The sound delivering system was closed and stimuli were delivered on the ipsilateral ear. The registration was extracellular and glass micropipettes filled with 3 M NaCl were used. We used the standard equipment for stimulus generation, control and calibration. The spike activity was amplified, triggered and fed to a ATAG 501-20 (Nihon Kohden) analyzer. The CCFs were obtained by synchronized summation of the stimulus envelope waveforms that preceded the spikes generation. In contrast to Møller's experiments /1,6,7/ we used for modulation not pseudorandom noise but continuous random noise with gaussian amplitude distribution.

The set of the CCFs obtained in typical cochlear nucleus cells when the carrier was the tone at unit's characteristic frequency is shown in Fig 1. With the low-intensity carriers (lower curves) the CCFs have only a single positive peak. Such shape of the CCF indicates that a brief increment in stimulus intensity gives rise to an elevation in the neuron discharge rate. When the mean stimulus intensity is increased a negative deflection is usually appeared after this positive CCF peak. In some neurons at high intensities this deflection determines the whole shape of the CCF.

We obtained the relative values of the CCF peaks by the dividing of peaks amplitude upon the mean impulsion rate. In fig 2 these relative peak values are shown as a function of carrier intensities for two cochlear nucleus units. When the carrier intensity increases the relative value of the positive CCF peak saturates 10 dB above threshold. The relative value of the negative peak, however, monotonically increases through the whole investigated intensity range. Thus the role of the delayed inhibitory input increases with the intensity of the CF tone.

Fig.1. Cross-covariance functions for two units in response to CF tone modulated by 200 Hz low-pass noise. The carrier intensity is shown near the curves in dB re unit threshold. Neuron 80-10-5 (left row): CF 0.8 kHz; threshold 32 dB SPL. Neuron 81-6-3 (right row): CF 0.65 kHz; threshold 19 dB SPL.

Fig 2. Mean impulsion rates (filled circles), positive CCF peak amplitudes (circles) and negative CCF peak amplitudes (crosses) are plotted for two units as a function of the carrier intensity in dB re units threshold. The values of the CCF peaks are shown in relative values. Neuron 81-9-10 (left row): CF 0.8 kHz; threshold 43 dB SPL. Neuron 81-9-12 (right row): CF 1.2 kHz; threshold 54 dB.

The role of the inhibitory inputs becomes more evident when off-CF tones are used as a carrier. The set of the CCFs obtained in typical cochlear nucleus cell at various carrier frequencies is shown in Fig 3. With the best-frequency carriers (0.5 kHz) the CCF reveals a positive peak that succeeded by a negative one. The shape of the CCF is radically changed when the carrier frequency moves far enough from the CF. For 0.3 kHz tone it is the negative CCF peak that preceded the positive one. At the edges of the frequency response range (0.2 kHz and 0.9 kHz) the negative peak

Fig.3. Cross-covariance functions at two intensity levels (50 dB SPL - left row; 70 dB SPL - right row) obtained for tones modulated by 200 Hz low-pass noise. The carrier frequencies in kHz are shown near the curves. Neuron 80-13-18: CF - 0,5 kHz, threshold-36 dB SPL.

remains the only notable component of the CCF. Such shape of the CCF indicates that for these frequencies this neuron generates the spike discharges in response not to the increment but in fact to the decrements of the sound envelope. It is of interest that the mean impulsation rate for amplitude modulated off-CF tones which evoke such "off-response" can be fairly high.

The analysis of CCFs obtained in cochlear nucleus neurons of the frog allows to suggest that the complex interaction of the excitatory and inhibitory inputs takes place during the action of the amplitude-modulated CF and off-CF tones. For high intensity CF tones delayed negative peak of CCF could demonstrate the delayed inhibitory input. For off-CF tones this inhibitory input becomes the leading one. Now it may be treated as a typical representation of lateral inhibition mechanisms. These findings suggest that the same underlying cellular mechanism may be responsible for the lateral inhibition and the delayed CF-inhibition. The latter effect can be used for the explanation of the enormous operation range for amplitude-modulation encoding in many cochlear nucleus neurons.

Some models of cochlear nucleus neurons assume independent inhibitory system which participate in response-strength limitation. From the other hand the lateral inhibition is usually treated as a response property that improves the frequency selectivity by the limitation of the excitatory response area. Our evidence allow to suggest the important role of the lateral inhibition both in the response-strength limitation and in the enlargement of the dynamic operation range during the action of complex modulated sounds.

1. MOLLER AR, Acta Physiol. Scand. 98 (1975) 157-167.
2. FRISINA, R., SMITH, R., CHAMBERLAIN S, Hear. Res. 44 (1990) 123-142.
3. RHODE, W., SMITH P, J. Neurophysiol. 56 (1985) 287-307.
4. MARTIN, M., DICKSON, J.W., Hear. Res., 9, (1983) 35-42.
5. BIBIKOV NG, Sensory Systems, 1 (1987) 353-361.
6. MOLLER AR, J. Physiol. 259. (1975) 63-82.
7. MOLLER AR Exp. Brain Res. 25 (1976) 307-321.