COMPARISON OF MONAURAL ACOUSTIC AND ELECTRIC STIMULATION: UNIT TYPES IN THE CAT INFERIOR COLLICULUS

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This study has shown that 1) the ratio of excitatory and inhibitory inputs to units in the central nucleus of the inferior colliculus is altered when one cochlea is neomycin-deafened and/or electrically stimulated; 2) prestimulus threshold shift and lateral inhibition mechanisms can be used to explain these results; and 3) at high current levels a leakage current into the modiolus can evoke unit population responses.

INTRODUCTION

Preliminary investigations\(^1\)\(^2\) indicate there are some qualitative changes in the response of inferior colliculus units for electrical, compared to acoustic, stimulation. These qualitative changes (eg, an increase in the number of units with an onset response or suppression of spontaneous rate) may be the result of a shift in the balance of excitatory and/or inhibitory inputs to inferior colliculus units. This may affect the proportions of observed unit types (contralateral and ipsilateral excitatory [EE], contralateral excitatory only [EO], and ipsilateral excitatory only [OE]). There have been no quantitative investigations of unit types for electrical stimulation. The

REFERENCES

Comparison of monaural acoustic and electric stimulation. A) Spectral distribution, based on unit CF, of EE and OE units for C(acoust)I(acoust), C(elect)I(acoust), and C(acoust)I(elect) stimulation. C(acoust)I(acoust) [EE, n = 57]; C(elect)I(acoust) [EE, n = 42; OE, n = 41]; and C(acoust)I(elect) [EE, n = 34]). B) Spectral distribution based on unit CF. of OE units for C(acoust)I(acoust), C(elect)I(acoust), and C(acoust)I(elect) stimulation. For comparison, C(acoust)I(acoust) OE unit data from Semple and Aitkina are also shown. [C(acoust)I(acoust)][n = 81]; C(elect)I(acoust)[n = 81]; C(acoust)I(elect) OE (including EI units) [n = 81], compiled from their Figs 1-4.

Comparison of unit type proportions for C(acoust)I(acoust), C(elect)I(acoust), and C(acoust)I(elect) stimulation.

<table>
<thead>
<tr>
<th>Type</th>
<th>C(acoust)I(acoust)</th>
<th>C(elect)I(acoust)</th>
<th>C(acoust)I(elect)</th>
<th>C(acoust)I(acoust)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(%)</td>
<td>(%)</td>
<td>(%)</td>
<td>(%)</td>
</tr>
<tr>
<td>EE</td>
<td>35</td>
<td>35</td>
<td>35</td>
<td>40</td>
</tr>
<tr>
<td>OE</td>
<td>2</td>
<td>34</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>EO (EE)</td>
<td>63 (31)</td>
<td>31†</td>
<td>65 (48)</td>
<td>58 (34)</td>
</tr>
<tr>
<td>Total</td>
<td>100 (n = 46§)</td>
<td>100 (n = 121)</td>
<td>100 (n = 98)</td>
<td>100 (n = 582)</td>
</tr>
</tbody>
</table>

*From Semple and Aitkin, after recollection according to monaural response properties.
†There was no experiment to determine EI units for C(elect)(acoust) stimulation.
§91 C(acoust)(acoust) units were recorded from in this study (57 EE, 34 EO). Some tracks searched specifically for EE units. Units on these tracks have not been included in above statistics.

Results

A total of 310 units were recorded from the central nucleus of the inferior colliculus. We recorded 121 under C(elect), 98 under C(acoust), and 91 under C(acoust) stimulus conditions. The unit type proportions are shown in the Table, wherein the C(acoust)I(acoust) unit type proportions are shown to be consistent with those of Semple and Aitkin. In summary, the relative proportions of EE units remained constant across the three stimulus combinations. The C(acoust)I(elect) and C(acoust)I(acoust) population data.
showed similar populations of EE, EO, and OE units, but C(elect)I(acoust) stimulation produced a large increase (32.0% ± 9.4%; 95% confidence interval using normal statistics) in OE and a decrease (−31.6% ± 16.2%) in EO unit proportions. These data suggest an enhanced ipsilateral and reduced contralateral excitatory input for C(elect)I(acoust) stimulation relative to C(acoust)I(elect) or C(acoust)I(acoust) stimulation.

A comparison of C(acoust)I(acoust), C(elect)I(acoust), and C(acoust)I(elect) EE, EO, and OE unit CF distributions (see Figure) was undertaken to help explain the above results. The results showed that compared to C(acoust)I(acoust) stimulation, electrical stimulation and/or neomycin deafening of either the ipsilateral or contralateral cochlea resulted in a shifted unit CF distribution. In particular, the results showed 1) there was an increase ($\chi^2 = 31.4, df(u) = 4, \alpha = 0.05$) in low CF C(elect)I(acoust) EO unit density; 2) there was an increase ($\chi^2 = 8.9, v = 3, \alpha = 0.05$) in high CF C(acoust)I(acoust) EE, OE, and EO unit CF distribution, resulting in a bimodal distribution, and 3) the C(elect)I(acoust) OE unit CF distribution was almost identical ($\chi^2 = 0.9, v = 4, \alpha = 0.05$) to that of the C(acoust)I(acoust) EE units. This study's C(acoust)I(acoust) EE unit CF distribution is not dissimilar to that of Sempel and Atkin$^4$ ($\chi^2 = 1.8, v = 4, \alpha = 0.05$; see Figure, B) or that of Roth et al$^5$ ($\chi^2 = 1.0, v = 3, \alpha = 0.05$).

DISCUSSION

Unlike the monomodal C(acoust)I(acoust) EO CF distribution, the C(elect)I(acoust) EO CF distribution is bimodal, with peaks in the 0.5- to 1-kHz and 5- to 10-kHz frequency bands. The sharp, large, well-demarcated peak in the 0.5- to 1-kHz frequency band could be explained by a spread of current, particularly at higher current levels, through the modiolus to the spiral ganglion cells of a more apical region. The modiolar fibers affected correspond to the fibers originating from the region of the cochlear turn immediately above the stimulating electrodes. The C(acoust)I(elect) EO CF distribution is similar ($\chi^2 = 4.7, v = 4, \alpha = 0.05$) to that obtained with C(acoust)I(acoust) stimulation, except for a slight dip in CF distribution in the 0.5- to 1-kHz frequency band. This could be due to the modiolar spread of current providing inhibitory rather than excitatory input.

Unlike the unimodal C(acoust)I(acoust) EE unit CF distribution, the C(acoust)I(elect) EE CF distribution is bimodal, with peaks in the 0.5- to 1-kHz and 5- to 10-kHz frequency bands. The C(acoust)I(elect) distribution low-frequency peak occurs in a lower band. This could be a consequence of modiolar stimulation. The C(acoust)I(elect) EE unit distribution supplemental high-CF peak is located in the same CF region as that of the EO distribution. This suggests this high-CF peak population may be derived from units that would respond as EO under C(acoust)I(acoust) stimulus conditions. Many OE units are not just influenced by ipsilateral inhibitory and contralateral excitatory inputs; the overall response of these units is a balance of contralateral and ipsilateral inhibitory and excitatory inputs.$^6$ The application of an electric instead of an acoustic stimulus may inherently alter this balance. Alternatively, it may be argued that as a consequence of neomycin deafening of the ipsilateral side, an EI unit receives more spontaneous input from the contralateral side, resulting in the unit's becoming more depolarized prior to stimulation (ie, a prestimulus threshold shift). This may mean that a "weak" ipsilateral excitatory stimulus normally not evoking an excitatory response might be capable of doing so, particularly if its latency was shorter than that of the inhibitory input. The C(elect)I(acoust) EE units have a predominantly low-frequency monomodal CF distribution. This is not markedly different ($\chi^2 = 3.5, v = 4, \alpha = 0.05$) from the C(acoust)I(acoust) EE CF distribution, except that the peak is larger and its location slightly lower than that of the C(acoust)I(acoust) EE CF distribution. Modiolar stimulation can be used to help explain these differences in peak location and size.

An analysis of the excitatory drive to C(elect)I(acoust) units suggests that as a result of neomycin deafening and/or electrical stimulation, the efficacy of the electrically stimulated side in evoking an excitatory response is reduced (−31.5% ± 8.5%), while that of the acoustically stimulated side is enhanced (+26.0% ± 9.2%). The enhanced ipsilateral (relative to contralateral) stimulus efficacy seen with C(elect)I(acoust) stimulation means collicular units likely receive more low-CF excitatory and high-CF inhibitory drive from the ipsilateral side. This could lead to an increase in the excitatory drive to low-CF, ipsilaterally driven EE units, while some higher CF EI units may appear more inhibited or unresponsive to contralateral stimuli. A reduced contralateral (relative to ipsilateral) stimulus efficacy may result in some EE and EO units' requiring a more intense contralateral stimulus to evoke an excitatory response (or may appear OE responding or unresponsive, respectively).

The C(elect)I(acoust) OE and C(elect)I(acoust) EE units have remarkably similar CF distribution plots (see Figure, A). This suggests that OE units may be derived from units that would respond as EE under C(acoust)I(acoust) stimulus conditions. A reduced contralateral excitatory drive, possibly the result of many units' having a prestimulus threshold shift induced by deafening the contralateral side, would help explain this notion. The Table shows the C(elect)I(acoust) EO population to be significantly smaller than that derived with C(acoust)I(acoust) stimulation. This, too, can be explained by EO units becoming nonresponders as a consequence of a reduced contralateral excitatory drive, prestimulus threshold shifts, and monaural deafening.

Lateral inhibition is postulated as an additional explanation for our C(elect)I(acoust) OE data in particular. The current density apical to the most apical stimulating electrode pair (electrodes 3 and 4, about 6 mm into the cat cochlea) will be such that higher-frequency regions (nearer the electrode pair) are likely to receive more intense stimuli than lower-frequency regions (distal to the electrode pair). Nearly all contralateral tuning curves for collicular units have higher-threshold inhibitory sidebands.$^7$ For many units, for all excitatory suprathreshold electrical stimuli, both the dominant higher-frequency, high-threshold inhibitory sideband and the lower-threshold, lower-frequency excitatory region of the tuning curve will be simultaneously stimulated. Thus, a large proportion of units more apical to the stimulating electrode pair may show no excitatory drive. For contralateral electrical stimulation, this implies a reduced EE (some appearing OE-responding or unresponsive, respectively).
This study shows that a large number of units, when electrically driven, show no excitatory response. Finally, this study has important consequences for masking of electrical stimuli, particularly for the sequential progression of electrodes in the basal to apical direction.

REFERENCES


MODEL OF DISCHARGE RATE FROM AUDITORY NERVE FIBERS RESPONDING TO ELECTRICAL STIMULATION OF THE COCHLEA: IDENTIFICATION OF CUES FOR CURRENT AND TIME-INTERVAL CODING

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A model of the response of auditory nerve fibers to electrical stimulation of the cochlea is presented. Auditory nerve fiber responses are described in terms of cochlear regions activated by the stimulus: region A, in which the discharge rate equals a value of the pulse rate plus spontaneous activity, and region B, in which the discharge rate is less than pulse rate plus spontaneous activity but greater than spontaneous activity. The cues for intensity and time-interval coding provided by regions A and B are discussed.

INTRODUCTION

This is a descriptive model of responses of the auditory nerve to pulsatile electrical stimulation of the cochlea using stimulus parameters set within the operative range of a cochlear implant (biphasic charge-balanced current pulses, 100 to 200 microseconds per phase, amplitude 0.2 to 2 mA, pulse rates 50 to 400 pulses per second). The model looks at responses to electrical stimulation as populations of auditory nerve fibers (ANFs) in two cochlear regions, A and B. Region A encompasses the cochlear region around the stimulating electrodes, in which discharge rate is "saturated" at a value equal to the pulse rate plus the spontaneous activity. Region B is a population of ANFs in which the discharge rate is less than the pulse rate but greater than the spontaneous discharge rate. Region B surrounds region A and is therefore more distant from the stimulating electrode. The model is described mathematically, and cues for the coding of intensity (current) and time intervals (pulse rate) are identified.

MATHEMATICAL DESCRIPTION OF AUDITORY NERVE MODEL

Figure 1 illustrates the input-output function of an ANF to electrical stimulation of the cochlea at pulse rates of 100, 200, 300, and 400 pulses per second. Javel et al. noted from these data that the curves lie virtually on top of each other, which implies 1) that there are no differences in growth rate for different pulse rates, 2) eliciting a discharge rate is dependent on stimulus current and not pulse rate, and 3) the growth of discharge rate is logarithmic against current. Thus,

\[ R = k(I - IT) + S \]

where

\[ R = \text{discharge rate (spikes per second)} \]
\[ k = \text{the growth rate of discharge (50 to 127 spikes per second per decibel, mean } = 70, \sigma = 20, \text{ reanalysis of auditory nerve data from this laboratory} \]
\[ I = \log \text{stimulus current (dB re 1 \mu A)} \]
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