The ventral cochlear nucleus (VCN) contains a heterogeneous collection of cell types reflecting the multiple processing tasks undertaken by this nucleus. This in vivo study used intracellular recordings and dye-filling to examine membrane potential changes and firing characteristics of onset neurones to acoustic stimulation (50 ms pure tones, 5 ms rise time, 0.2 Hz repetition). Using rats anaesthetised with urethane (1.3g/kg i.p.), microelectrodes containing 1M KAc and 4% neurobiotin, were inserted into VCN. Stable impalements were made from 11 onset neurones, seven identified as multipolar cells. Neurones responded to characteristic frequency (CF) tones with sustained depolarisation at below spike threshold. Increasing stimulus intensity increased and sharpened the depolarisation occurring in the initial 5 ms of the response from which an onset spike was generated. With the exception of tones presented at the high frequency edge of the cells response area, which resulted in depolarisation and spike at both the onset and offset of the stimulus, off CF tones resulted in a broadening of the initial depolarisation with high stimulus intensities required to initiate an onset spike. The onset spike latency in response to a given frequency decreased with increasing intensity. Presentation of tones off CF resulted in longer latencies. The spike onset latency depended on the rise time of the depolarisation with its latency remaining constant across tones which is consistent with monosynaptic excitation from auditory nerve. Depolarisation rise times decreased with increasing stimulus intensity and increased off CF. These results suggest that multipolar cells receive convergent input from auditory nerve enabling intensity and frequency to be coded through changes in membrane responsiveness.

CENTRAL EFFECTS OF A PROFOUND SENSORINEURAL HEARING LOSS

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The central auditory pathways undergo significant transneuronal change in response to auditory deprivation, particularly during development. An understanding of the functional consequences of auditory deprivation is important for the clinical application of cochlear prostheses. In the present study we have investigated the physiological status of the central auditory system of profoundly deafened cats. Seven adult cats were used: two had normal hearing; four were deafened neonatally, prior to the onset of hearing; two bilaterally and two unilaterally. The remaining animal had a profound bilateral hearing loss of >8 years duration. Under sodium pentobarbital anaesthesia (40 mg/kg i.p.; supplements administered as required), both cochleae of each animal were implanted with bipolar stimulating electrodes, and one inferior colliculus (IC) was surgically exposed. Single unit activity within the IC was recorded with tungsten microelectrodes (1-2MΩ) oriented 45° to the sagittal plane, in response to biphasic current pulses presented at 5 pulses/s. Results were based on responses recorded from 421 neurones. There was no significant difference in response latencies at saturation current intensities between normal and neonatally deafened animals, although latencies were significantly prolonged in the long-term deafened animal (p<0.05; t-test). In the normally hearing and bilaterally deafened animals, response latencies for stimulation of the ipsilateral cochlea were significantly shorter than for contralateral stimulation (p<0.01); however, this trend was reversed when recording from the IC ipsilateral to the deafened cochlea of unilaterally deaf animals (p<0.05). Finally, a rudimentary tonotopic organisation was demonstrated within the IC of animals with no prior auditory experience. These results have important implications for the application of cochlear prostheses in congenitally deaf children.
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Title:
Intracellular responses of onset neurones in the ventral cochlear nucleus to acoustic stimulation [Abstract]

Date:
1998

Citation:

Persistent Link:
http://hdl.handle.net/11343/27029

File Description:
Intracellular responses of onset neurones in the ventral cochlear nucleus to acoustic stimulation [Abstract]

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