gests that it may occur in man under certain undefined conditions. However, at this time there is no evidence that adenovirus infections have been associated with tumors in man.

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Anteroventral Cochlear Nucleus: Wave Forms of Extracellularly Recorded Spike Potentials

Abstract. *Analysis of wave forms of spike potentials from neurons in the anteroventral cochlear nucleus indicates that the spikes are composed of three components. Two appear to be postsynaptic events, and one appears to be presynaptic and to be related to the calyces of Held found in that part of the nucleus.*

Spike potentials recorded extracellularly from neurons in the most rostral region, or "oral pole," of the anteroventral cochlear nucleus (AVCN) have a wave form that is not found in recordings from other regions of the cochlear nucleus. The occurrence of these wave forms in this region appears to be related to the anatomical finding that the AVCN contains large nerve terminals, called calyces (or bulbs) of Held, that are not found in other regions of the nucleus (*1*).

Adult cats were anesthetized (Dial in urethane, 0.75 mg/kg), and the cochlear nucleus was exposed by partial aspiration of the cerebellum. Spike potentials were recorded with indium-filled, platinum-black-tipped microelectrodes with tip diameters from 4 to 12 μ (*2*); the reference electrode was connected to the head-holder. This report is based on observations and recordings of spike potentials of both spontaneous and acoustically stimulated activity from several hundred neurons in the AVCN of more than 25 cats (*3*).

Superposed traces of spike potentials for six different neurons are shown in Fig. 1. The samples shown are representative of the variations in wave forms encountered. The prominent property that distinguishes these spike potentials from those encountered in other regions of the cochlear nucleus is the presence of a positive (P) component early in their wave form. The P components are all-or-nothing in character (that is, for a given neuron, their amplitude is independent of stimulus level); they are usually either monophasic or diphasic (+, −); they each are consistently followed by a prominent negative potential (*4*). The negative potentials, alone, are characteristic of those generally obtained with extracellular recordings of spike discharges of neurons (*5*).

Let us first consider the negative potentials. The inflection on the rising phase of the negative potentials of several of the samples shown suggests that they consist of two components (A and B) that are similar to the composition of spike potentials recorded from spinal motor neurons and elsewhere (*5–7*). For some neurons (about 20 to 25 percent of our sample) the two-component structure is explicit because the second, B, component occasionally fails to develop (Fig. 2). This two-component structure has been distinctly observed, thus far, only for neurons in the "oral pole" of the AVCN, although inflections on leading edges of spike potentials have been observed elsewhere in the cochlear nucleus.

Factors affecting the development of the A and B components of spike potentials have been studied extensively by intracellular techniques in spinal motor neurons (*6*) and cortical neurons (*7*). The A component has been identified as the discharge of the initial segment (IS) or axon hillock of the neuron, and the B component has been identified as the discharge of the soma-dendritic process (SD) (*5*).

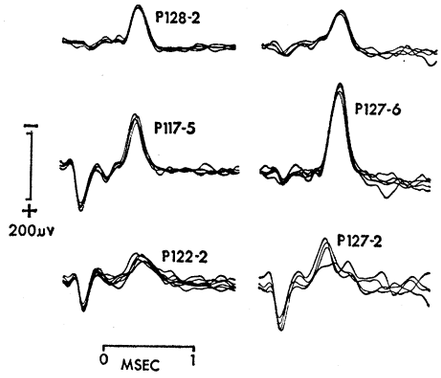

Fig. 1. Superposed traces of spike potentials of six different neurons in the "oral pole" of the anteroventral cochlear nucleus. These illustrate the variety of wave shapes obtained and the consistent appearance of a positive (P) component. These traces were not synchronized to any stimulus but were synchronized to an arbitrary point on the wave form after they had been recorded from the neuron.

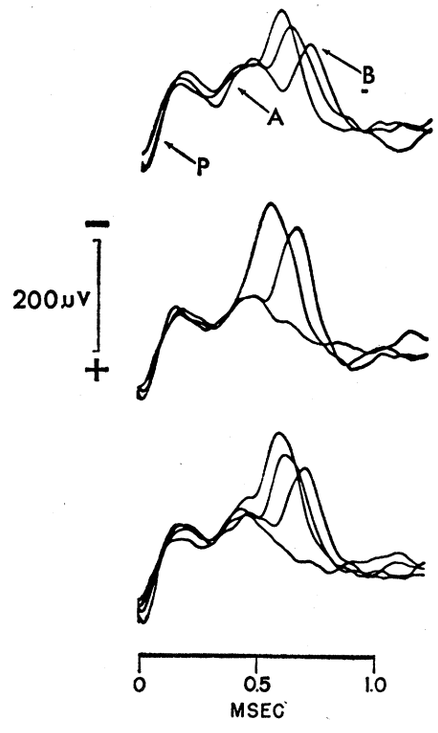

Fig. 2. Superposed traces of spike discharges from a single neuron which illustrate the two-component structure of the negative potential. These spike potentials were recorded in response to a continuous sinusoidal acoustic signal delivered to the ear. In general, the inflection on the rising phase increases in size as the time from previous spike discharge decreases (not shown). The fact that explicit two-component structure is seen for only 20 to 25 percent of the neurons may be related to electrode location relative to the neuron. These traces were synchronized to a point on the leading edge of the P component.

(The A and B components of spike potentials of neurons in the "oral pole" could also be IS and SD spikes respectively, but an alternative is that the A component is an excitatory postsynaptic potential rather than an IS discharge.) The absence or failure of the SD component has been found to be due to a threshold level of depolarization that is higher than that required for the IS component (*6*). Furthermore, failures of the SD component occur more frequently as time from last spike discharge decreases (*6*, *7*).

The present observations are consistent with those made on motor neurons in that the B component can be made to fail more frequently, for a given neuron, by increasing the sound pressure level of acoustic stimuli. The application of such a stimulus causes the discharge rate to increase, which leads to a decrease in interspike intervals. Properties of the A and B components are strikingly similar to those of the IS and SD components, and extracellularly recorded spike potentials of their polarity and wave form have been unequivocally identified as records of postsynaptic events (*5*); therefore, I am led to conclude that the A and B components are postsynaptic.

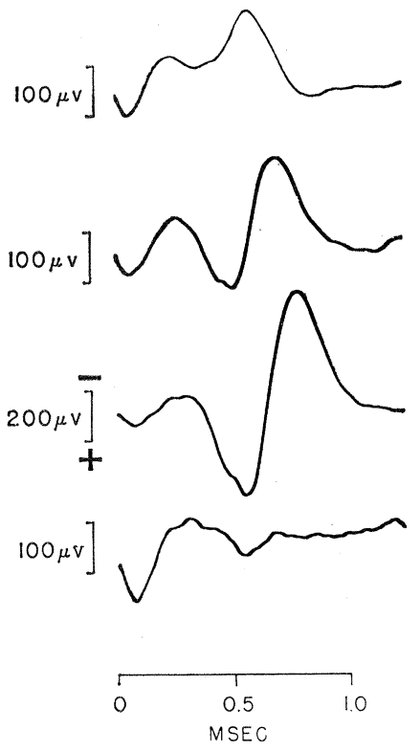

Fig. 3. Four traces from a single neuron during the "injury" process. The top trace shows the wave form of the spike potential before the "injury" began. As the position of the electrode is advanced, the wave form changes shape. The negative potential changes to a positive-negative sequence and increases in amplitude (note scale change). Finally, in the bottom trace the large injury potential disappears. However, the P component is relatively unaffected throughout the seqeunce.

The time interval between the initiation of the P component and the initiation of the A-B sequence (approximately 0.4 to 0.6 msec) is consistent with times between the arrival of presynaptic impulses at presynaptic terminals and the onset of postsynaptic depolarization for a wide variety of synapses (*8*). Hence, if the A and B components are assumed to be postsynaptic, one is led to conclude that the P component is *presynaptic*.

This interpretation, as well as the interpretation of the A and B components, is further supported by the changes in the wave form of spike potentials caused by "injury." In the present experiments, "injury" discharges have been detected for more than 25 neurons, presumably owing to the physical advancement of the electrode. I have consistently observed that the P components remain unaffected by such "injuries." On the other hand, the A and B components undergo a characteristic change of amplitude, polarity, and shape—then disappear (Fig. 3) (*9*). Thus, the P component appears to be associated with a structure different from that with which the A and B components are associated and one that is not affected by "injury."

The detection of individual presynaptic events with extracellular electrodes is not unusual (*10*). For example, spike potentials from large presynaptic terminals have been recorded extracellularly from giant synapses of squid stellate ganglia (*11*), nerve terminals in rat diaphragm (*12*), and motor nerve terminals of crayfish (*13*). To the best of my knowledge, however, the oral pole of the AVCN is the first place where individual presynaptic events have been recorded extracellularly in the mammalian central nervous system.

The fact that wave forms with P components have not been detected elsewhere in the cochlear nucleus is consistent with the absence of calyces of Held in the other regions. Calyces of Held, however, are found in the nucleus of the trapezoid body (*14*). (Although the endings are not identical in structure with those in the AVCN, their appearance is similar enough that they too are called calyces of Held.) In a single experiment I have recorded from two neurons in the nucleus of the trapezoid body (*15*); in the wave form of their spike potentials, a P component was also present, a finding that supports an apparent relationship between wave form of spike potential and neuronal morphology.

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