Impulse Origin and Propagation in a Bipolar Sensory Neuron

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ABSTRACT Intracellular recording techniques were used to study electrical activity in bipolar sensory cells associated with crayfish tactile receptors. Several lines of evidence indicate that spikes evoked by natural stimulation of the receptor originate at a dendritic locus. Although overshooting spikes are recorded in the soma in response to both natural and antidromic stimulation receptor potentials are observed only rarely, and, when present, their amplitude is less than 5 mv. Impulses propagating centrifugally into the soma following antidromic stimulation always exhibit an inflection in the rising phase of the spike; however, orthodromic spikes are usually uninflected. Occasionally, orthodromic responses (in the soma) exhibit rather unusual wave forms. Such spikes evoked by natural stimuli are indistinguishable from those elicited electrically in the dendrite, but they do not resemble antidromic impulses. Because the axonal and dendritic boundaries of the soma have a low safety factor for spike transmission, at high frequencies invasion of the soma by dendritic spikes is impeded and often blocked. The soma region can thus act as a low-pass filter. The significance of this self-limiting mechanism for the behavior of the animal is not known; it is suggested, however, that this impediment is a potentially critical one, and may, in other situations, have encouraged the evolution of alternative arrangements.

INTRODUCTION

In investigations of complex neurons and systems of neurons, identification of the specific cellular loci concerned with the initiation of impulses is of considerable importance. For this reason, a number of such studies have been undertaken in recent years, and many of them have demonstrated an axonal locus for spike initiation in various types of neurons: spinal motoneurons (Araki and Otani, 1955; Araki and Terzuolo, 1962); crustacean stretch receptors (Edwards and Ottoson, 1958); teleost supramedullary neurons (Bennett, Crain, and Grundfest, 1959); *Aplysia* central interneurons (Tauc, 1962); eccentric cells in the *Limulus* photoreceptor (Tomita, 1957); and Mauthner cells of the goldfish (Furshpan and Furukawa, 1962). Thus, this pattern of impulse initiation has attained the status of a generality.

One major morphological class of nerve cells, the bipolar cells, has never yielded satisfactorily to this type of analysis. Bipolar cells are not only widely distributed in various vertebrate sensory systems, but also are found as primary receptor neurons associated with the cuticular sense organs of arthropods. In practice, however, these cells do not usually lend themselves to analyses of membrane phenomena. Not only are they usually of small size; in vertebrates they are also often located in inaccessible regions of the nervous system. Many of the equivalent cell types in arthropods are closely applied to the inner surface of a tough and often calcified exoskeleton.

Bipolar sensory neurons have recently been described from the freshwater crayfish which occur in pairs and which innervate a single exoskeletal mechanosensory hair of a type which is found in pits on the surface of the thoracic carapace (Mellon, 1963 *a*). Each member of the pair shows opposite directional sensitivity. The somata are up to 100 microns in length. The neurons are easily exposed, and permit recordings with micropipette electrodes from axons and cell bodies. Special attention has been given to an attempt to identify the region of impulse initiation, and the results stand in interesting contrast to previous findings on the crayfish stretch receptor cells (Edwards and Ottoson, 1958). In addition, the electrical characteristics of several areas of the nerve membrane have been examined; it is suggested that the cell body acts as a "valve" to limit the frequency response of the neuron. A preliminary account of this work has appeared previously (Mellon, 1963 *b*).

MATERIALS AND METHODS

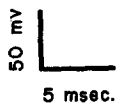
Freshwater crayfish, *Procambarus clarkii*, were collected locally and kept in the laboratory until used. The carapace was removed from one side of the thorax of an animal and isolated in physiological saline (van Harreveld, 1936). The thin transparent cuticle which is closely applied to the hypodermis of the branchiostegites was then carefully stripped off to expose the nervous elements. The receptor hairs could be stimulated mechanically with a fine brush, or often simply by disturbing the interface of the bathing solution. As previously described (Mellon, 1963 *a*), the tracts of sensory fibers from the tactile hairs were easily visualized, and no difficulty was encountered in penetrating axons with micropipettes. However, only under the most advantageous circumstances was it possible to visualize the paired cell bodies in unstained preparations. For the greater part of their length, the sensory axons run in bundles near to the medial surface of the hypodermis; close to their respective somata, however, they branch away from other fiber pairs, decrease in diameter, and course more deeply into the hypodermal tissue where they are usually masked by chromatophores. Lightly pigmented animals in the middle of the moulting cycle were found to offer the best chances of visualizing the somata, and the occasional crayfish meeting these specifications generally contained one or more pairs of cells which could be seen and penetrated. Successful penetration was often confirmed following an experiment in which the recording electrode was left *in situ* and the preparation stained with methylene

blue. Micropipettes filled with 3 M KCl and with resistances of 10 to 20 megohms were used. Signals were led into a high impedance, neutralized capacitance preamplifier (Bioelectric Instruments, Inc.) and then to conventional oscillographic recording equipment.

Stimulating electrodes were pairs of fine silver or platinum wires mounted on micromanipulators. All experiments were performed at temperatures between 20 and 25°C.

FIGURE 1. A pit receptor neuron which had been stained supravitally with methylene blue. Note the abrupt enlargement of the axon proximal to the cell body. The scale marker represents 100 microns.

FIGURE 2. Intracellular records from the axon of a pit receptor cell. Left, a single spike evoked by natural stimulation of the receptor; right, an antidromic impulse.



RESULTS

1. *Axon Responses*

The characteristic morphology of the receptor neurons has been described previously (Mellon, 1963 *a*), and is shown in Fig. 1. The majority of axon penetrations were in the thick segment, several millimeters proximal to the soma. Resting potentials were generally about 70 to 75 mv although values above and below this range were also seen. Axons with resting membrane potentials of 70 mv and higher always gave spikes which overshot the zero potential level in response to natural and antidromic activation; spikes produced in either fashion were similar in appearance (Fig. 2). On occasion, an electrode was inserted into an axon as close as 500 microns to the region of marked diameter change; spikes recorded there were similar in all respects to those observed at more proximal loci, and no evidence for the spread of receptor potentials to this region from the dendrite was ever obtained.

When paired antidromic stimuli were delivered to the axon, the second spike showed some loss in amplitude, and eventually failed, as the stimulus was delivered progressively earlier after the initial response (Fig. 3). This behavior is essentially identical with results obtained in other axons (*e.g.*, that of the crayfish stretch receptor neuron (Eyzaguirre and Kuffler, 1955 *b*)), and it is shown here for later comparison with soma responses evoked under similar conditions.

2. *Soma Responses*

NATURAL STIMULATION Successful intrasomatic recordings were obtained from more than twenty cells. Resting membrane potentials did not differ noticeably from those found in axons, although direct comparisons from both regions of the same neuron were never made. Usually, orthodromic impulses recorded within somata also resembled those seen in axons; except in deteriorating preparations, the spikes overshot the zero level and recovered

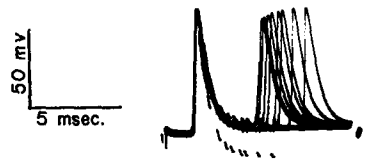


FIGURE 3. Response of an axon to pairs of antidromic stimuli. The shock pairs occurred five times per second, and the film was exposed continuously as the stimulus interval was decreased. The second impulse failed at intervals of less than 3 msec. Intracellular records.

without any postspike hyperpolarization. Only in two instances were slow depolarizations resembling receptor potentials seen to underlie the conducted impulses; in both cases the amplitude of depolarization was less than 5 mv, even with intense natural stimuli. Since the length of the receptor cell dendrite is usually 100 microns or more, it is assumed that considerable attenuation of a receptor potential would occur within this region. In any event, such potentials were below the recording level in the majority of units studied, and the possibility that they evoked spikes in regions of the neuron proximal to the recording site was therefore extremely remote.

ANTIDROMIC INVASION Impulses recorded within the soma due to antidromic stimulation invariably showed an inflection on the rising phase of the spike. Occasionally, orthodromic spikes were also inflected, although direct comparison of the two usually showed the inflection on the antidromic impulse to be more pronounced (Fig. 4). The magnitude of the sudden change in slope of the rising potential was somewhat variable, depending on the particular preparation being studied and its physiological condition. Inflected

spikes have been recorded from a variety of other neuron somata, and their properties have been thoroughly described (Brock *et al.*, 1953; Araki and Otani, 1955; Bennett, Crain, and Grundfest, 1959; Tauc, 1962). Adjacent regions of neural membrane may show differences in electrical excitability; moreover, any morphological discontinuity may produce delays by loading the adjacent excited region with an expanded membrane area. This is, of course, exactly what happens to antidromic spikes at the axon-soma boundary,

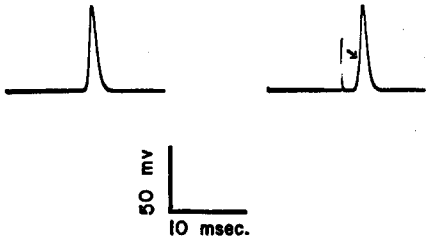


FIGURE 4. Intrasomatic records from a pit receptor cell. The left-hand record is an orthodromic spike evoked by natural stimulation. Right, an antidromic response. Arrow marks approximate point of inflection.

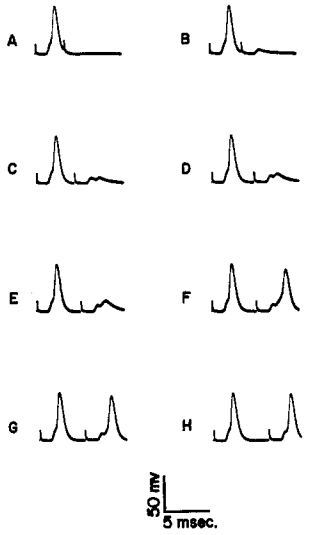


FIGURE 5. Responses recorded within the soma of a receptor cell to pairs of antidromic stimuli delivered to the axon. In A, the stimulus interval was small and the axon refractory to the second stimulus. The interval was gradually increased (B–H), evoking a second axon impulse in B, a local soma response in C, and a second soma spike in F. Further description in text.

and it is often possible to accentuate the invasion delay (and hence, the inflection) by adding relative refractoriness to the impediment due to morphology (Fuortes, Frank, and Becker, 1957). This was accomplished in the pit receptor neurons with paired antidromic stimuli (Fig. 5). At intervals of less than 3 msec., a single inflected spike was recorded in the cell body. It must be assumed that a second spike was prevented by the refractory period of the axon (*cf.* Fig. 3). At an increased stimulus interval (Fig. 5B) a small potential suddenly appeared following the second shock. This response resembled the A′ spike recorded by Tauc (1962) in the giant neuron of *Aplysia* and also the A spike of spinal motoneurons (Fuortes, Frank, and Becker, 1957); it appar-

ently was the electrotonic potential from an impulse occurring in an active region of the axon proximal to the soma. The impulse has been prevented from actively invading the soma by the reduced safety factor at the boundary region. To be consistent with previous terminology we will refer to this small potential as the A spike. As the stimulus interval was increased still further, a small graded component rose out of the A spike. The peak latency of this response was quite variable and decreased with an increasing stimulus interval. This potential may have been a somatic local response, for it subsequently triggered the soma impulse (Fig. 5F) and was afterwards lost in the rising phase of the spike. The intermediate response was not apparent in every preparation studied; a particularly interesting case of this kind is shown in the records of Fig. 6. The A spike following the second stimulus was characteristic,

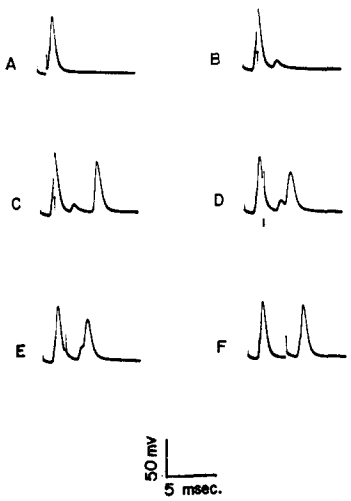


FIGURE 6. Soma responses to paired antidromic stimuli. As in Fig. 5, the stimulus interval was progressively increased. In C, a second soma impulse occurs following the small A spike. See text.

but the second soma impulse initially occurred *after* the A spike had decayed to the baseline; it was uninflected and thus resembled an orthodromic impulse. It is tentatively suggested that, in such cases, the second spike originated in the dendritic portion of the cell and subsequently propagated orthodromically into the cell body. Results to be presented below (Fig. 9) suggest that the dendritic membrane recovers more rapidly than the soma after the passage of a conducted impulse, and the possibility thus exists that current spread from the A spike directly excited some low threshold region of the dendrite and initiated a spike. With an increase in stimulus interval, the site of origin of this spike shifted toward the cell body as this region recovered from the first response (Fig. 6D). As the conduction delay became small the spike occurred earlier in the recovery cycle of the soma membrane and decreased in amplitude; full recovery was attained only at stimulus intervals greater than 5 msec. However, confirmation of this proposal would depend upon

knowledge, which we now lack, of variations in threshold and of the space constant for electrotonic spread in different regions of dendrite and soma.

Whatever the details of events which underlie the complex wave form of antidromic spikes, it is clear that they differ characteristically from normal orthodromic spikes. Since receptor potentials do not spread to the axonal regions, there can be no safety factor increase due to such depolarization (Coombs *et al.*, 1957; Fatt, 1957; Fuortes *et al.*, 1957; Tauc, 1962); the results therefore show that orthodromic activity must be initiated either at the recording site (*i.e.*, the soma) or distal to it on the dendrite.

ORTHODROMIC ELECTRICAL STIMULATION If orthodromic impulses normally originate at a dendritic locus, spikes evoked by electrical stimulation of the dendrite should have a wave form identical to that of spikes due to mechanical stimuli. To test this, a pair of stimulating electrodes was placed

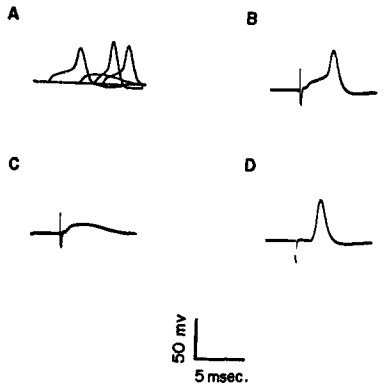


FIGURE 7. Responses from a deteriorating soma. A, a multiple exposure showing three inflected impulses and a dendritic A spike evoked by natural stimulation. B, an inflected spike, and C, a dendritic A spike elicited by electrical stimulation of the dendrite. D is an antidromic impulse. The responses in A were retouched.

on the preparation with the cathode near to the dendritic process and the anode distal to it. Short (0.1 msec.) electrical stimuli were effective in evoking responses whose wave form, regardless of the physiological condition of the preparation, was identical to that of the mechanically evoked spikes. In deteriorating preparations which had been injured by electrode penetration, the safety factor for impulse invasion of the soma was lowered considerably. Eventually, spikes were blocked altogether at the soma boundaries, but sometimes rather bizarre changes in wave form occurred first. Records from such cells were especially suitable for differentiating the antidromic and orthodromic spikes, and, consequently, for demonstrating the identical wave form characteristics of the mechanically and electrically evoked orthodromic impulses. In the examples shown, electrically evoked spikes (Fig. 7B, C) were identical in wave form to those arising from mechanical stimulation of the receptor (Fig. 7A), but different from those elicited by antidromic stimulation (Fig. 7D). Occasionally, invasion of the cell body by an orthodromic impulse

was blocked, and only a small potential was recorded by the electrode (Fig. 7A, C); this small potential seems entirely analogous to the antidromic A spike, and it thus will be referred to as the dendritic, or orthodromic, A spike. Its occurrence is undoubtedly due to a region of low safety factor at the dendrite-soma boundary; the unmasking of this impediment to impulse invasion in deteriorating cells is further evidence that a dendritic conduction pathway is involved in the propagation of orthodromic activity.

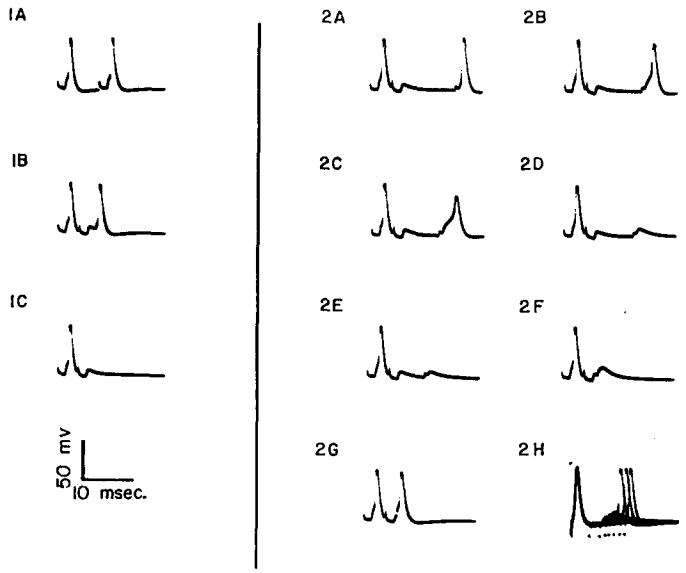


FIGURE 8. 1A–C, soma responses to paired antidromic stimulation. The stimulus interval was decreased progressively. 2A–G, same antidromic stimulation interval as in 1C; an orthodromic spike was evoked electrically in the dendrite at decreasing intervals following the antidromic A spike. In 2D–F the orthodromic spike failed to invade the soma; in 2G, however, the dendritic A spike summated with the antidromic A spike and evoked a soma impulse. 2H, response of the soma to paired electrical stimuli delivered to the dendrite. Intracellular records.

ANTIDROMIC-ORTHODROMIC INTERACTIONS Additional support for this conclusion was obtained in other experiments by interacting orthodromic and antidromic stimuli. Antidromic stimuli were paired to give a small A spike (Fig. 8, 1A–C), and an orthodromic response was then evoked electrically in the dendrite at varying intervals (Fig. 8, 2A–H). As the orthodromic response was initiated progressively earlier after the pair of antidromic responses, it encountered a refractory condition in the dendritic membrane brought about by the previous passage of the antidromic spike. As a result, the safety factor at the soma boundary region, inherently low because of the expanded membrane area, was reduced still further, and the orthodromic

spike became increasingly inflected and finally failed to invade the soma (Fig. 8, 2D). A small dendritic A spike remained, however, and when this occurred simultaneously with the antidromic A spike, the summated depolarization was of sufficient amplitude to fire the soma (Fig. 8, 2G). Since the small potentials both must have arisen from actively responding parts of the cell and were capable of summating, they must have occurred in separate regions of the cell.

The soma appeared to be capable of supporting higher impulse frequencies than could be successfully passed into it across the dendritic-soma boundary. In Fig. 8, 2G, the summated small potentials evoked a second soma spike 5 msec. after the first spike, corresponding to a frequency of 200/sec.; with

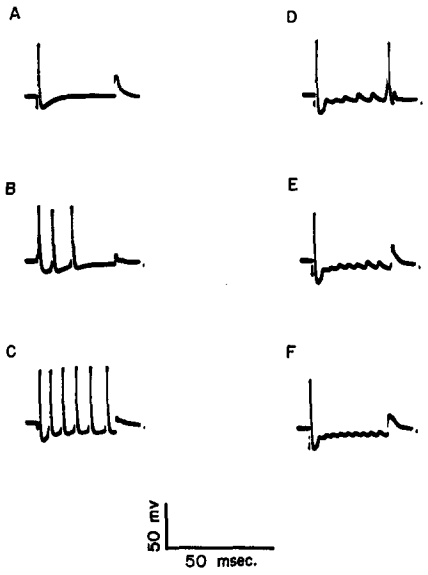


FIGURE 9. Repetitive activity in the soma following stimulation of the dendrite with 35 msec. current pulses. A–F, increasing stimulus intensity. Spikes do not invade the soma at frequencies above 200/sec. Same cell as in Fig. 8.

paired stimuli dendritic spikes were blocked at an interval corresponding to 125/sec. (Fig. 8, 2H). The precise values for the blocking interval often differed from one preparation to another, and even within a single neuron at different times.

DC STIMULATION OF THE DENDRITE REGION When long duration electrical stimuli were delivered to the dendrites phasic trains of impulses were produced (Fig. 9). As the current strength was increased, the number and frequency of impulses appearing in the soma increased to a value represented by the response of Fig. 9C, where the frequency of the first pair of spikes is 200/sec. In the presence of stronger currents there was an initial soma spike followed by trains of small potentials whose amplitude showed an inverse relationship with their frequency of occurrence. It is proposed that these

potentials represent all-or-none responses evoked by the stimulating current in the distal regions of the dendrite but unable to invade the soma. With a decline in frequency, however, the membrane intervening between the electrode and the distal active portion would recover to increasing extents before the arrival of a subsequent spike, and it is likely that the impulse would then approach the soma more closely. At high frequencies it seemed clear that an active response train occurring in the dendrite could be effectively blocked from invading the soma.

By placing stimulating electrodes on different regions of the neuron it was shown that the dendritic portion was specifically involved in the production of trains of impulses in response to long current pulses (Fig. 10). The frequency

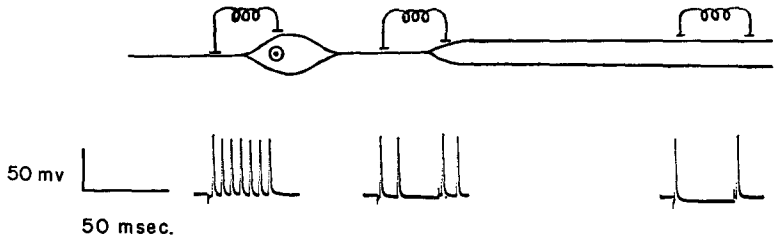


FIGURE 10. Comparison showing the response of three different regions of a pit receptor neuron to equal intensity, 35 msec. current pulses. The recording electrode was within the axon about 1 cm proximal to the soma. Spike trains were evoked when the cathode was in the vicinity of the cell body; at more proximal locations the stimulus efficacy was considerably reduced. Note latency shift of the first spike as the stimulating electrodes were moved centrally.

and number of impulses generated by 35 msec. current pulse depended critically upon the proximity of the stimulating electrodes to the dendritic region.

DISCUSSION

The ability of the dendrites of these bipolar cells to support regenerative electrical activity is not without precedent. Evidence that retrograde propagation of spikes occurs in the dendrites of bipolar chemoreceptor cells of insects has been reported (Morita, 1959; Hanson and Wolbarsht, 1962). Dendritic spikes are also reported to occur in hippocampal neurons (Spencer and Kandel, 1961). Quite recently, Mendelson (1963) has presented evidence which suggests that conducted impulses are initiated in the dendrites of bipolar cells innervating movement receptors in the crab leg. The technical difficulties encountered with that preparation are apparently severe, however, and extensive intracellular recordings were not made.

Since the receptor potential in the thoracic sensory cells is so attenuated at

the soma as to be absent, no relationship between its magnitude and the frequency of conducted impulses could be determined. As would be expected, D.C. stimulation of the dendritic region does produce spike trains whose frequency increases with current strength, suggesting that the usual relationship between generator currents and impulse discharge exists in the dendrite.

An interesting consequence of the morphology of these bipolar neurons is that the regions at the dendritic and axonal boundaries of the soma are incapable of passing spikes at frequencies much greater than 200/sec. Not only does this impose a limit on the firing frequency of the neuron as a whole; it can, when the dendrite is strongly excited, result in an abrupt and complete failure of all transmission to the central nervous system. A similar phenomenon in the crayfish stretch receptor cells was first reported by Eyzaguirre and Kuffler (1955 *a*). Excessive stretch applied to the dendritic terminals of these neurons increased impulse frequency up to a point, and then produced cathodal block at the impulse-generating locus. In the pit receptor cells, spike blockage is apparently due to morphological features which cause loading of the dendrite by the soma, and which are independent of the spread of receptor potentials. Although the possibility exists that penetration of the soma by a microelectrode in some way is responsible for the low safety factor of this region, this argument does not appear valid. In previous experiments (*cf.* Mellon, 1963 *a*) impulses were recorded extracellularly from sensory axons; stimulating currents applied to the dendrites through the exoskeleton failed to evoke spike trains at frequencies greater than 200/sec. Spike blockage was presumably involved, since impulse activity often ceased abruptly during passage of the stimulating current. There is also a slight possibility that anodal current spread actually hyperpolarizes a critical membrane region and impedes impulse invasion. This argument does not seem very likely either, since spikes actually invaded the soma at higher frequencies during DC stimulation (Fig. 9) than when pairs of short duration stimuli were used (Fig. 8, 2H).

The significance of frequency limitation in the physiology of this receptor system is unclear. Natural stimuli rarely evoke impulse trains approaching 200/sec. in the pit receptors, which usually produce arhythmic spike sequences at much lower frequencies (Mellon, 1963 *a*). It is therefore possible that the action of the cell body as a low-pass filter is without any functional significance in the normal behavior of the crayfish. A question does arise in regard to findings such as those of Wolbarsht and Dethier (1958) that spike frequencies as high as 600/sec. occur in some bipolar mechanoreceptor neurons on the blowfly wings. Extracellular recording techniques were employed using micropipettes placed over the tips of cuticular sensilla; it now seems possible that dendritic spike activity alone was monitored, and further experiments are

thus desirable to discover whether the axons themselves are capable of transmitting such high frequency spike trains.

In myelinated vertebrate sensory axons, which are inherently capable of higher impulse frequencies (up to 1000/sec.), the presence of a bulky obstruction such as a cell body in the transmitting pathway might impose severe limitations on the upper working range of the fibers. It is not altogether surprising that many vertebrate sensory cells are monopolar structures with their somata located off the main conduction pathway at the termination of an axonal branch.

The multichanneled input to some central and peripheral nerve cells places an important geometrical restriction on the origin of output signals. In many instances, the cell body itself is an important integrating center where information from several spatially discrete cellular extensions or loci is collected and encoded in the output. The initiation of impulses in these cells must logically occur at a point proximal to the soma, if the integrative capacities are to be realized; otherwise, spikes arising initially in one dendritic branch might subsequently invade neighboring branches and block their activity, thereby effectively limiting the output frequency of the neuron to that of a single input channel. In some central neurons where branching axons and multiple synaptic loci would appear to make such problems especially acute, a single neuron may have several spike-initiating zones (Bennett, Crain, and Grundfest, 1959), and preferential conduction of impulses from these sites insures against the invasion of neighboring synaptic centers by disruptive antidromic activity (Tauc and Hughs, 1963). No such spatial restrictions apply to these bipolar neurons; the presence of a single input channel precludes any possible ambiguity concerning the origin of sensory information, and there are no valid reasons why the spike-generating locus should not be as close as is physically possible to the transducer region.

This work has been supported by grants (to Donald Kennedy) from the United States Public Health Service (B-2944) and the Air Force Office of Scientific Research (AF-AFOSR 62-147).
Dr. Mellon was a post-doctoral fellow of the United States Public Health Service.
Received for publication, June 12, 1963.

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