Modes of Initiation and Propagation of Spikes in the Branching Axons of Molluscan Central Neurons

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ABSTRACT A study has been made of *Aplysia* nerve cells, mainly in the pleural ganglia, in which the main axon divides into at least two branches in the neighbourhood of the soma. Conduction between these branches was investigated by intracellular recordings from the soma following antidromic stimulation *via* the nerves containing the axonal branches. It has been shown that transmission between separate branches need not involve discharge of the soma but only of the axonal region between the soma and the origin of the branches. In some cells, the spike may fail to invade the other axonal branch, whereas transmission in the opposite direction is readily achieved. Often spikes in none of the branches are transmitted to the others, unless facilitated. Indications about the geometry of the neuron in the vicinity of the soma may be obtained from the study of the relative size of the A spikes originated in different branches. These observations, together with the presence of different sizes of A spikes, produced by orthodromic stimulation, provide evidence that spikes initiated at separate axonal "trigger zones" of *Aplysia* neurons may be conducted selectively to the effectors or other neurons innervated by the particular branch.

INTRODUCTION

It is well known that the axons of central nerve cells often divide and in this way a single cell may be in contact with several widely distributed neurons or effectors. In molluscan central neurons, such axonal ramifications frequently occur in the proximity of the cell body, so that the main axonal process is already divided into several branches before it crosses the synaptic field of the neuropile. As there is good reason to believe that no synaptic contacts are present on the cell body (11, 23, 24) it may be assumed that integration of the synaptic input depends not only upon local excitabilities of the neuron membrane but also upon the geometry of the cell in the proximity of the cell body and its relation to the distribution of active synaptic areas along different

axonal branches. However, the molluscan cell in which spike initiation has been most studied (22–25) has no proximal axonal ramifications. This is the giant cell of the abdominal ganglion in *Aplysia*, where the spike is initiated in the axon in the region of the axonal synaptic field. In cells other than this giant neuron evidence has been obtained that spikes may originate at more than one site (17, 18). The present investigations have disclosed that it is very likely that the latter cells have ramifying axons and they have also provided evidence for the importance of the geometrical relationship of the axons. In a single neuron it now appears that spikes may be initiated in several proximal axonal branches by a preferential action of their synapses and that these spikes do not necessarily invade the other axonal branches or the soma (26). Yet the spike presumably always propagates centrifugally in the axon where it was initiated, so that an *Aplysia* neuron is able to send impulses selectively to only a part of the total regions supplied by the axons of a given neuron.

METHODS

The central nervous system of the gastropod mollusc *Aplysia depilans* comprising the cerebral, pleural, pedal, and abdominal ganglia, together with their connectives and most of the nerves were dissected (13), pinned in a suitable wax chamber, and immersed in oxygenated sea water. The protective sheaths of the pleural and abdominal ganglia were removed and the naked cell bodies penetrated with capillary microelectrodes of less than 1 μ tip diameter. The potentials were recorded in the usual way by means of a cathode follower and DC amplifier. A bridge circuit was used (1) to permit modifications of the membrane polarisation by current applied through the recording electrode. The nerves and connectives were placed on chlorided silver wire electrodes, which were used either for stimulation or for recording of activity in the nerves.

All experiments were performed at room temperatures varying from 20 to 22°C.

RESULTS

1. *Identification of the Antidromic Response*

Each of the nerves and connectives emerging from the pleural and abdominal ganglia is composed of both afferent and efferent fibres. When a nerve or connective is electrically stimulated, the nerve cells in these ganglia may be affected not only orthodromically *via* synaptic endings on the cell but also antidromically, if the axon of the cell is present in the stimulated nerve. When the response of the cell is recorded with an intracellular electrode located in the soma, it is not always easy to decide from the simple form of the potential whether an antidromic component is present or not. A definite distinction may be made, however, if the dynamic properties of the synaptic and antidromic events are considered.

It has been shown in the giant cell of the *Aplysia* abdominal ganglion (22–25)

that conduction of an antidromically evoked spike is partially blocked at some distance from the soma, which only becomes invaded after some delay or not at all. These events give characteristic potential changes, the antidromically evoked spike recorded in the soma intracellularly showing an inflection on its rising phase. Under fatigue or when polarisation of the cell is artificially increased, the inflection becomes more prominent and allows the dissociation of an initial small spike representing the axonal discharge (or A spike), followed after some delay by a larger component (or S spike) indicating activation of the somatic membrane itself (Fig. 2, *2*, *3*). Greater hyperpolarisation or fatigue prevents the somatic discharge and the A spike appears alone (*e.g.* Fig. 2, *4*, *5*). In this latter condition, the falling phase of the A spike represents the recharging of the somatic membrane after it had been depolarised but not excited by the axonal spike. Consequently the duration of the A spike depends on the time constant of the somatic membrane. On the other hand this membrane is also depolarised electrotonically following activation of the excitatory synapses located on the axon and therefore the duration of the excitatory postsynaptic potentials (EPSPs) will likewise depend on the electric constants of the membrane. In small cells (under 70 μ in diameter) having a short time constant (7), however, the action of the transmitter surpasses the duration of the electrotonic decay, so that the EPSPs have a longer duration than the A spikes and are easily recognised. With larger sizes of cell body the time constant increases progressively and the forms of the EPSPs and A spikes become more and more similar. However, these two potentials are differently affected by the change of membrane polarisation, and this property is very helpful in their identification.

When the membrane potential of the abdominal giant nerve cell is increased, the amplitude of the antidromically evoked A spike recorded in the soma decreases, because the spike is blocked at a greater distance from the soma which is less affected electrotonically by more remote discharges (24, 25). In contrast the same change of polarisation produces an increase in the amplitude of the EPSP (20). This increase has been observed in other nerve cells (5) and has been explained as due to a tendency of the EPSP to reach the equilibrium potential, which is closer to zero potential than to the resting potential. These opposite changes in amplitude of the A spike and EPSPs are ideal for their identification.

Another important property of the antidromic A spike is its non-fatigability and relatively constant amplitude, even if the fibre is stimulated at a fairly high frequency. In contrast, repetitive stimulation usually has a marked effect on the amplitude of the EPSPs which may either increase in size by reason of the potentiation (8) or diminish, if interneurons are present along other pathways (21). It has also been shown (24, 25) in *Aplysia* neurons stimulated either orthodromically, directly, or during spontaneous activity that the spike origi-

nates in the axon at some distance from the cell body, so that conduction of the spike in the proximity of the soma occurs in the same direction as that following antidromic stimulation. The result is that an A spike may be recorded in the soma which has been initiated by afferent input to the cell. However, a synaptically evoked A spike may be easily distinguished from one antidromically evoked because of its fatigability, variation in latency, and a greater sensitivity to the level of membrane polarisation.

2. *Stimulation of Two Axonal Branches*

Nerve cells were impaled in the abdominal and pleural ganglia and the converging nerves stimulated. Using the above criteria for discriminating orthodromic and antidromic activity, it was found that many of the pleural ganglion cells, fewer in the abdominal ganglia, showed an antidromic response to stimulation of more than one nerve (13). This indicates that axonal branches belonging to a single cell are distributed in different nerves. Stimulation of these different axons here referred to as *a* and *b* will produce in the intracellular recording either AS spikes or A spikes alone. It was observed, however, that whereas in some cells stimulation from different directions produced identical antidromic potential changes, in other cells the effects were different under the same conditions. Thus stimulation of *a* might produce an AS spike, while stimulation of *b* produced an A spike of a different size. Frequently none of the antidromic stimuli alone was able to produce an AS spike, so that only A spikes were recorded, often of different amplitudes. The possible combinations of the responses recorded in different somata following separate antidromic stimulation of *a* and *b* axons are defined in the following table:

Nerve or axon stimulated	*a*		*b*
Responses recorded in the cell body	AS		AS
	AS	$\neq$	A
	A		A
	A	$\neq$	A

The sign $\neq$ indicates that the amplitude of the A spikes was different. It is clear, therefore, that in a number of cells the spike propagating antidromically in a single branch is not able to invade the soma. Moreover, the difference in amplitude of the A spikes coming from different directions points to the invasion of different areas of the membrane by the spikes. This means that a spike coming from one axon does not necessarily invade the other axonal branches. One obvious method to investigate this possibility was to stimulate branch *a* and record the propagation of the spike simultaneously in the soma and branch *b* (Fig. 1). The records in this figure show that when the nerve containing branch *a* was stimulated a corresponding A spike appeared

in the intrasomatic recording, but no activity was found in branch *b*. Obviously the spike was not transmitted from *a* to *b*. When the stimulation is repeated, the amplitude of the A spike slightly increases (Fig. 1, *2*). This in-

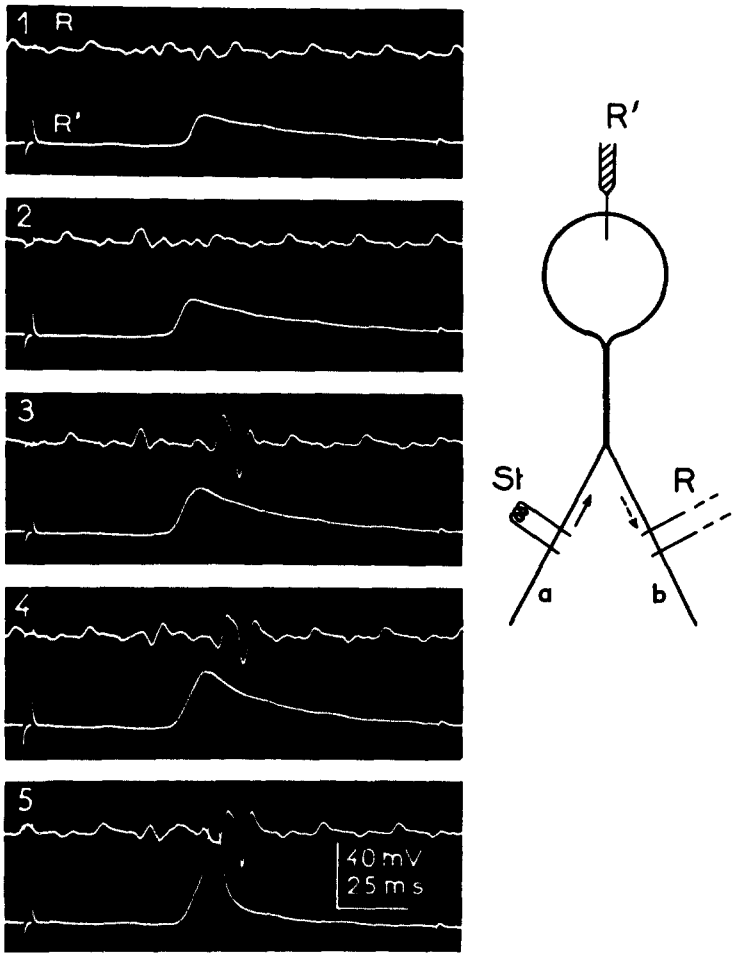

FIGURE 1. Recording from an *Aplysia* neuron (see diagram), whose axonal branch *a* was stimulated (*St*) and the resulting activity simultaneously recorded from the axon *b* (*R*) and in the soma by an intracellular microelectrode (*R'*). From *1* to *5* the cell response was facilitated by repetitive stimulation, showing from *1–4* an increase of the A potential, in *3* the beginning of the spike conduction from *a* to *b*, and in *5* the somatic discharge. Further details in text.

crease has been interpreted (24, 25) as being due to blocking of the conduction of the A spike closer to the soma. The increase in amplitude is progressive only within narrow limits. In Fig. 1, *3*, the spike reaches a greater amplitude with no intermediate size between (*2*) and (*3*), and a corresponding spike is re-

corded in axon *b*. The spike is therefore transmitted from *a* to *b*, but the soma is not invaded. When facilitated by repetition, the A spike may still increase in size within some limits (Fig. 1, *4*) and the soma is invaded (Fig. 1, *5*). Yet the somatic discharge does not alter anything in the conduction of the spike in *b*, which was transmitted from *a* to *b* before the invasion of the cell body, as is indicated by the identical latencies between the A spike and spikes recorded in *b* in Fig. 1, *4* and *5*.

The above results suggest that an intermediary axonal region is present between the bifurcation and the soma (diagram in Fig. 1). In *1*, the spike in *a* stops at some distance from the bifurcation, closer to this latter in *2*. The intermediary axonal region is invaded in *3*, *4*, and *5*, making possible conduction of the spike into axon *b*. Thus these experiments give not only important indications about the propagation of the spike, but also about the morphological structure of the nerve cell in the vicinity of the cell body.

However, only exceptional neurons like the giant nerve cell in the left pleural ganglion (12, 14) have axons of sufficiently large size to make it possible to record selectively their spikes in the whole nerve, which contains several hundreds of other fibres. Because of this difficulty, a different method was more generally used, and this gave satisfactory results. The axonal branch *a* was stimulated and transmission of the spike into branch *b* was tested by stimulation of this branch delayed in such a manner that the antidromic spike evoked in *b* would collide in this branch with the spike coming from *a*, if conduction from *a* to *b* was effective. This event is indicated in the intrasomatic recordings by the absence of the A spike coming from *b*. The test may be applied to both branches and conduction of the spike controlled from *a* to *b* and from *b* to *a*.

In Fig. 2, *1*, stimulation of the nerve containing either *a* or *b* produced AS spikes. When the delay between the two stimuli was decreased, there was an increase in the interval between the A and S spikes produced by the second shock (Fig. 2, *2*, *3*) and finally only the A spike remains (Fig. 2, *4*, *5*). The absence of the S spike is almost certainly due to a postexcitatory refractoriness of the somatic membrane (*cf.* reference 10, 4, for the spinal motoneuron). When the delay is still shorter (Fig. 2, *6*), any response to stimulation of *b* is absent in the somatic record. As the delay in record *6* is longer than the absolute refractory period of the A spike generator (19), it is obvious that the spikes from *a* and *b* collided in the axon *b*. As identical results were obtained when stimulation of *b* preceded that of *a*, it follows that spikes may be transmitted both from *a* to *b* and *vice versa*. The amplitude of the A spike coming from *a* or *b* is identical in this cell and the results point to a similar distribution of axons as in Fig. 1. The antidromic spikes coming from *a* and *b* are both able to excite an intermediary axonal zone, through which the spike invades the other axon and the soma.

A similar morphological arrangement of the axons may be proposed for the cell examined in Fig. 3; here the separate antidromic spikes (*1*) do not invade the soma, but only the intermediary axonal zone. Conduction is effective in both directions at the bifurcation. Indeed the amplitudes of the separate A spikes coming from the axons *a* and *b* are identical (Fig. 3, *1*) and the second

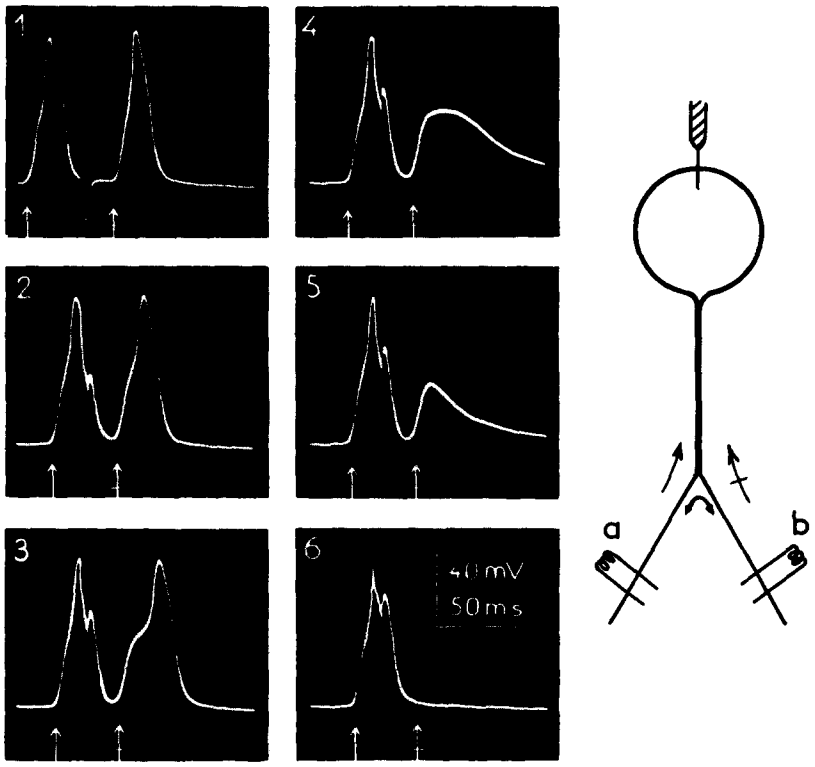

FIGURE 2. Intracellular recording from an *Aplysia* neuron, whose axonal branches *a* and *b* were stimulated at different intervals and the responses recorded in the soma. Note from *1* to *5* the progressive dissociation of the second spike into A and S components and in *6* the absence of the second response, due to the collision of impulses in branch *b*.

response is absent when the delay is brief, whichever axon was first stimulated (Fig. 3, *4*, *5*). Consequently, in both cases, the A spike represents the discharge of the same intermediary axonal region. The difference in conduction times of the spike coming from *a* and *b* is due to the fact that the stimulating electrodes were closer to the ganglion in *b* than in *a*. When the delay is brief but insufficient to induce collision of the spikes (Fig. 3, *2*), the second A spike increases in size. This is apparently due to the persistence of the depolarisation produced by the first A spike on the now discharged membrane which results

in facilitation of the conduction of the second A spike which is able to approach closer to the soma before it is blocked. When this facilitation is sufficient, the soma itself becomes invaded (Fig. 3, *3*).

A different functional distribution is observed in the nerve cell of Fig. 4, where the A spikes produced by antidromic stimulation of the two branches are dissimilar in size (Fig. 4, *1* and *2*). Analysis of the records shows that

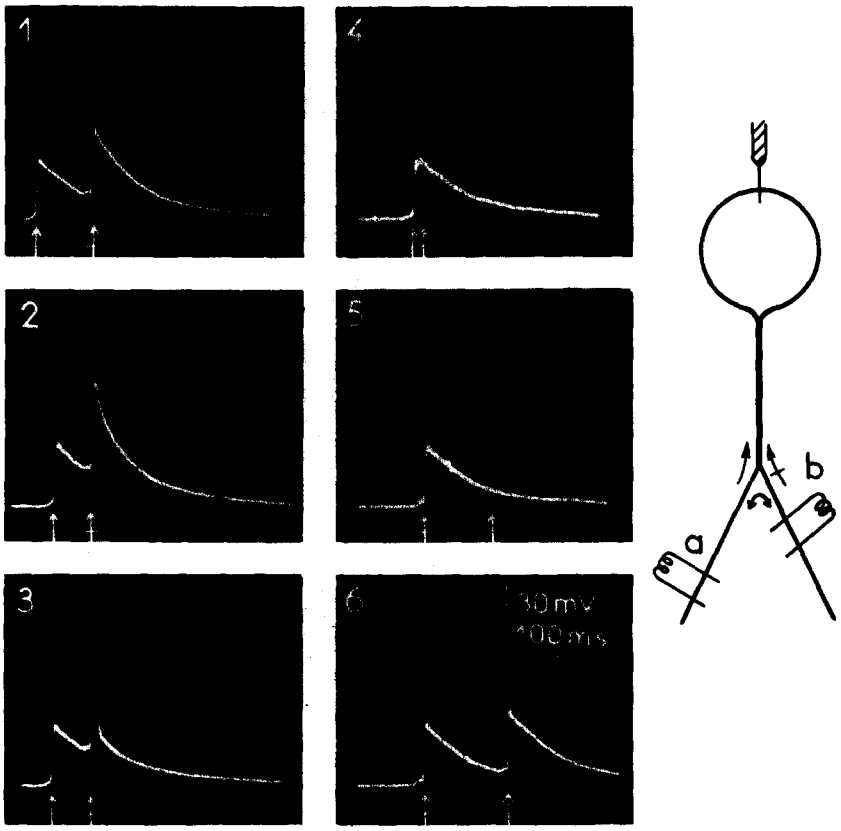

FIGURE 3. A spikes recorded in a slightly hyperpolarised *Aplysia* cell whose axonal branches *a* and *b* were stimulated antidromically. The absence of a second response in *4* and *5* indicates the conduction of an impulse from *a* to *b* and *vice versa* and the collision of spikes in these branches. Further details in text.

whereas the spike from branch *a* is transmitted into *b* (see the absence of response coming from *b* in *7* and *8*), the spike coming from *b* is blocked before the bifurcation. The A spike from *a*, larger in size, is always present if preceded by the A spike from *b*, even if the delay is very brief (Fig. 4, *3–5*). On the other hand its amplitude is increased by facilitation (*3* and *4*) and at short delays the soma is invaded (*5*). It may be concluded that in this cell the spike

coming from branch *a* is able to excite an intermediary axonal zone and propagate along *b* without exciting the soma, whereas the spike from branch *b* is unable to excite this axonal zone, perhaps by reason of its smaller diameter (see Discussion).

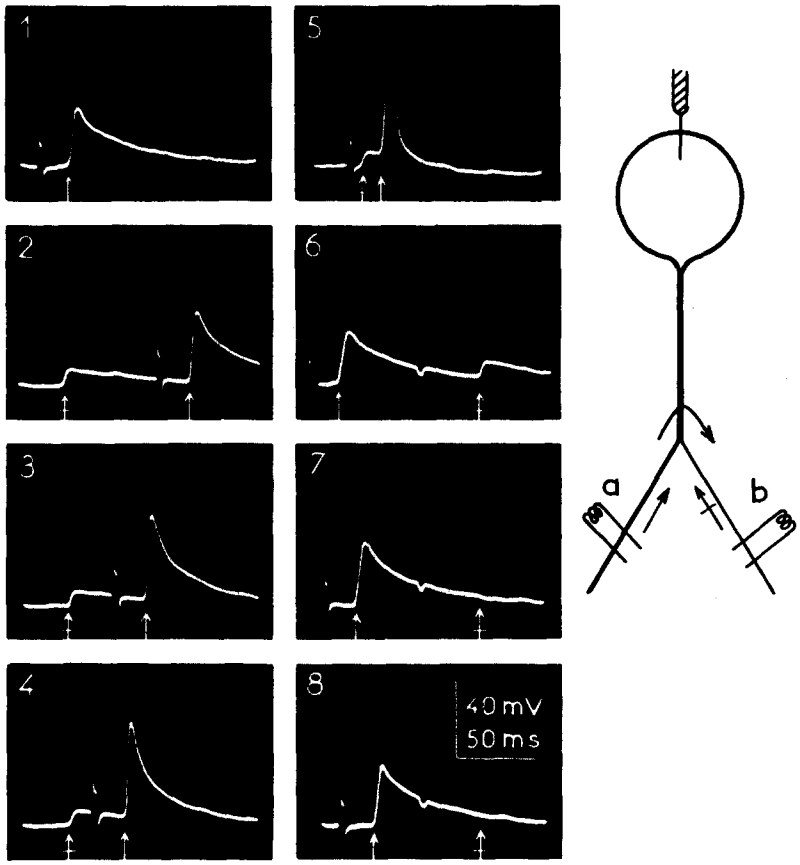

FIGURE 4. A spikes of different size produced by stimulation of two different branches of an *Aplysia* neuron, recorded by an intracellular electrode in the soma. The absence of a response in *7* and *8* shows that the spike from *a* is transmitted to *b*. But the spike from *b* is blocked before the bifurcation and facilitates conduction of the spike in branch *a*, as may be seen from the increase of the second A spike in *2* to *4* and the somatic response in *5*.

Other complementary information may be obtained when polarisation of the membrane is increased by an artificially applied current passed through the recording electrode. Under these conditions, thanks to the electrotonic gradient thus introduced, conduction of the antidromic spike may be stopped further from the soma. The cell studied in Fig. 5 presented the same pattern of response as that shown by the cell of Fig. 2 in the absence of any polarising

current; *i.e.*, mainly AS spikes with identical amplitudes of A spikes following stimulation of both *a* and *b*. When this cell was hyperpolarised, the A spikes observed were of much smaller amplitude without any intermediate stage between them and the previously recorded A spike and they differed in size when coming from *a* or *b*. Moreover, the spike did not conduct from branch *a* to *b* or *vice versa* (Fig. 5, *4* and *5*), yet when the membrane excitability was facilitated at short delays (Fig. 5, *6*) a big A spike appeared, undoubtedly rep-

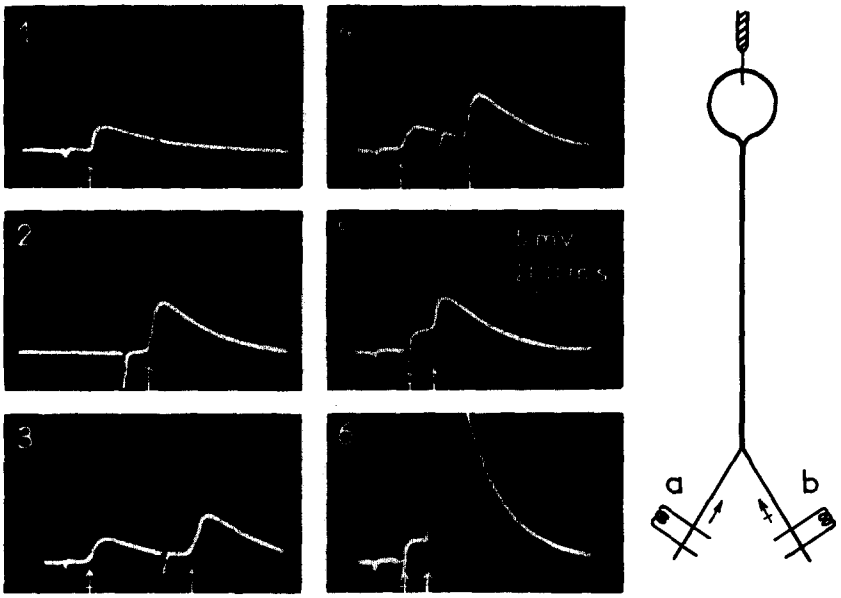

FIGURE 5. Small A spikes dissimilar in size, produced by the stimulation of two different branches of an artificially hyperpolarised neuron. The spikes do not conduct from *a* to *b* or *vice versa*, but when arriving close together (*6*) they may induce discharge of the axonal region located between the branching and the soma. Further description in text.

resenting the discharge of an intermediary axonal region. The diminutive size of the small A spikes suggests a distant localisation of the ramification rather than a small diameter of the branches as the spike of each of these branches was able under normal conditions of polarisation to excite the intermediary axonal zone.

The intermediary axonal region is apparently absent in some nerve cells. In the cell illustrated by Fig. 6, stimulation of branch *a* gave a larger spike (*2*) probably somatic in nature because of its large amplitude and brief time course. The small spike coming from *b* (*1*) is undoubtedly an A spike. The records show that spikes can be transmitted from *a* to *b* (*3*) but not in the opposite direction (*4–6*). When this cell was slightly hyperpolarised (Fig. 7), the

amplitude of the spike following stimulation of *a* became considerably reduced (Fig. 7, *2*) which confirms the AS character of the spike obtained from *a* stimulation in Fig. 6. However, the amplitude of the A spike from *b* was unchanged by this increase in polarisation (compare Fig. 6, *1*; and Fig. 7, *1*). The amplitudes of the A spikes from the two branches are now different and the spike from *a* no longer propagates into *b* (Fig. 7, *7* to *8*). Spikes coming from *a* and *b* remained independent of one another and under these conditions

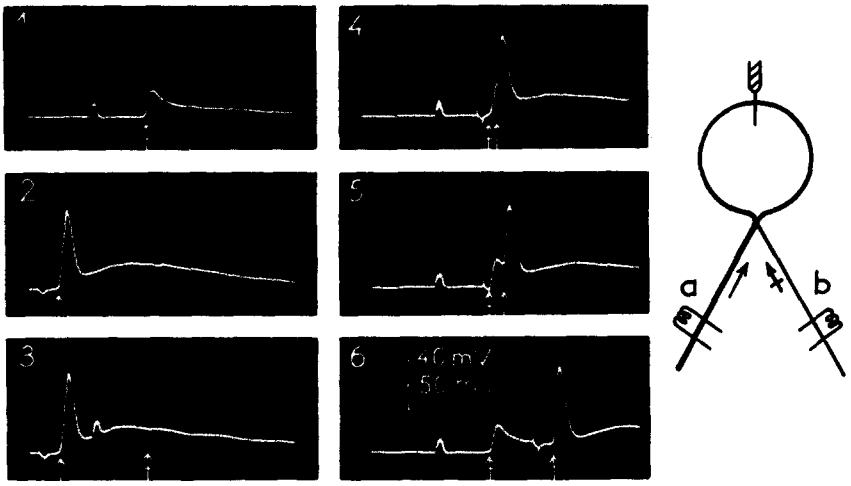

FIGURE 6. Intrasomatic records in an *Aplysia* neuron stimulated antidromically through axonal branches *a* and *b*. The analysis of the records, given in the text, suggests a neuronal structure of the type shown in the diagram.

neither was able to abolish the other, but when arriving in close succession or simultaneously they were able to excite the soma or a part of it (Fig. 7, *4*, *5*). The diagram acccmpanying Fig. 6 gives the probable axonal distribution in this cell. The great amplitude of the *a* potential under hyperpolarisation makes it necessary to suppose that blocking in conduction occurs in the close vicinity of the soma. The difference in amplitude of the two A spikes, and the fact that under normal conditions, stimulation of branch *a* gives a somatic spike, indicate the absence of any intermediary zone. The axon *b* is probably smaller in size than *a*, as the A spike from *b* is unable to invade the soma by itself.

The different situations that have been shown to exist concerning transmission of an antidromic spike between the two axonal branches *a* and *b* may be summarised as follows:—

1. The spike is transmitted from *a* to *b* and in the reverse direction through the excitation of an intermediary axonal zone. The somatic discharge is not necessary for transmission in this case.

2. The spike is transmitted from *a* to *b* through the excitation of an inter-

mediary axonal zone, but the spike from *b* is blocked before it can excite this zone so that transmission can only take place in one direction.

3. The spike is transmitted from *a* to *b* through the excitation of the cell body, but the spike from *b* is not transmitted to *a*. Somatic discharge is necessary for transmission in the one direction in which it is possible, for no intermediary zone is present in this type.

4. The spike is not transmitted between branches in either direction.

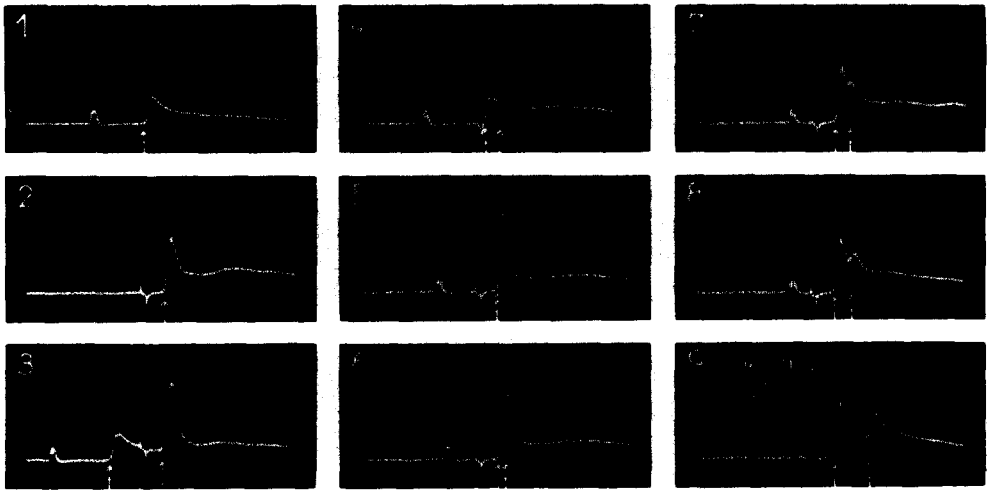

FIGURE 7. Same neuron as in Fig. 6, but slightly hyperpolarised. Further description in text.

DISCUSSION

The principal observation of the present study is that an antidromically evoked spike in addition to not necessarily invading the soma, may also fail to invade other axonal branches of the same neuron in the vicinity of the soma. Consequently the safety factor for continuous antidromic spike conduction in an axonal branch is low in the vicinity of such ramifications. This may be explained at least in part by the local increase in the surface to be invaded by the spike. The loading effect of the not excited membrane on the A spike membrane diminishes the amplitude of the spike and thus the safety factor. Certainly the relative sizes of the constituent branches before and after a bifurcation are very important, as the loading currents depend upon the proportion between the area of the surface already excited and that of the resting membrane, which are coupled by a highly conductive cytoplasm. In addition it is also possible that the excitability of the axon decreases as it approaches the soma. In these aspects conduction of the spike in the region of these ramifications may be compared to that between the axon and soma (9, 4, 24, 25).

The possibility that an antidromic spike may fail to be transmitted between axonal branches is important in view of results previously obtained on the giant cell of the abdominal ganglion of *Aplysia* (24, 25). Here it was disclosed that under normal conditions of excitability the spike originates at some distance from the cell body in an axonal region whose membrane has the lowest threshold of the whole neuron and that this occurs during spontaneous firing or following orthodromic stimulation. The spike then propagates to one side of this trigger region in an efferent direction but also on the other side towards the soma exactly like an antidromically evoked spike. Thus in the vicinity of the soma the direction of spike conduction is similar whatever its origin. Like

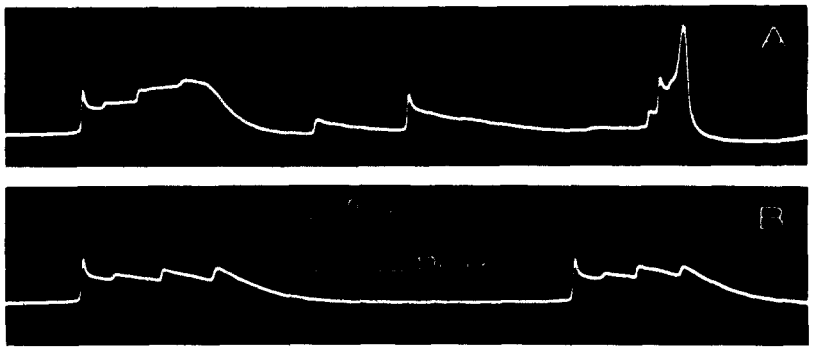

FIGURE 8. A spikes induced in an *Aplysia* neuron by orthodromic stimulation (A) and appearing spontaneously (B). Their different amplitudes indicate the presence of several trigger zones in different axonal branches. Intrasomatic recordings.

an antidromically evoked spike, the orthodromic spike also shows on its rising phase an inflection characteristic of the two-stage invasion of the neuron. When polarisation of the somatic membrane is artificially increased, it is possible to prevent the somatic discharge and record an orthodromically initiated A potential giving a normal efferent response easily recorded on the distant axon just as has been shown in the present study for the giant cell of the left pleural ganglion (Fig. 1, *3–4*). The somatic discharge is not necessary to originate an efferent response, however, for this may be initiated even when the soma is removed because all synaptic contacts are located on the axon in the region where the spike takes its origin (23–25). Thus under normal conditions of excitation an A spike recorded in the soma of the giant cells indicates that an efferent response has been initiated in the axon. This is of fundamental importance when we remember (17, 18, 20) that in many other *Aplysia* or *Helix* central neurons, A spikes alone appear in the somatic recordings in the absence of any polarisation either during spontaneous activity or following orthodromic stimulation. These A spikes were called "pseudospikes" and often several of these having different amplitudes may be observed in a single

neuron (Fig. 8). In the light of the present work it is clear that such pseudospikes represent responses initiated in the axonal branches, but unable to invade the soma.

Furthermore, a failure of transmission between different axonal branches should always be considered when the pseudospikes appear to have different amplitudes, because the conditions for their propagation towards the soma are identical to those observed during antidromic stimulation (*e.g.*, Fig. 4). Yet they will always propagate in an orthodromic direction in the branch in which they were initiated (see reference 24). Moreover, the presence of several pseudospikes of different amplitudes recorded in a single neuron, resulting from orthodromic stimulation (Fig. 8) indicates a plurality of loci for spike initiation, located in different axonal branches. Such a plurality has been postulated not only for *Aplysia* cells (17, 18) but also for the crustacean heart ganglion cells (3) and the cells of the sixth abdominal ganglion of the crayfish (16). Spike initiation in spatially separate synaptic regions located in individual ganglia of the abdominal chain has also been demonstrated for crayfish interneurons (15).

By analogy with what is known for the giant cell of the abdominal ganglion of *Aplysia*, it may reasonably be assumed for cells having axons ramifying close to the cell body, that the synaptic contacts are distributed along different branches some of which have a special region of high excitability where the spike may be initiated. Because of the electrotonic gradient this trigger region would be more affected by synapses activated in the same branch than by those of other branches. It is probable that different axonal branches may be affected preferentially by certain afferent pathways (compare reference 6, 2, on frog motoneurons) and that perhaps these correspond to some distinct function. Furthermore, in some neurons, a spike initiated in one branch cannot be transmitted to other branches, unless these are themselves sufficiently synaptically affected; it follows that only this one part of the neuron will give an efferent discharge which will be conducted selectively to the effectors or other neurons innervated by this particular branch. The same may happen during spontaneous activity of an axonal pacemaker. A single neuron is therefore able to give fractional responses sometimes with a very definite directional effect, the axons often being distributed to different nerves emerging from the ganglia and innervating separate effectors.

It is equally clear that in many cases an all-or-none response in a branch would facilitate the response in other branches; indeed a pseudospike amplifies the synaptic action by an additional depolarisation spreading in all the branches in the proximity of the cell body and in the cell body itself. These would mainly be electrotonic effects of pseudospikes which may summate so that usually the response of a few branches would induce general invasion of the whole neuron by the spike (see diagram in Fig. 9). In this way the *Aplysia* nerve cell integrates not only synaptic input, but also the all-or-none activity

locally induced as a result of the synaptic input. The response of each neuron under these conditions will be determined by its structure, by the distribution of the afferent endings on the neuron, and by local conditions of excitability.

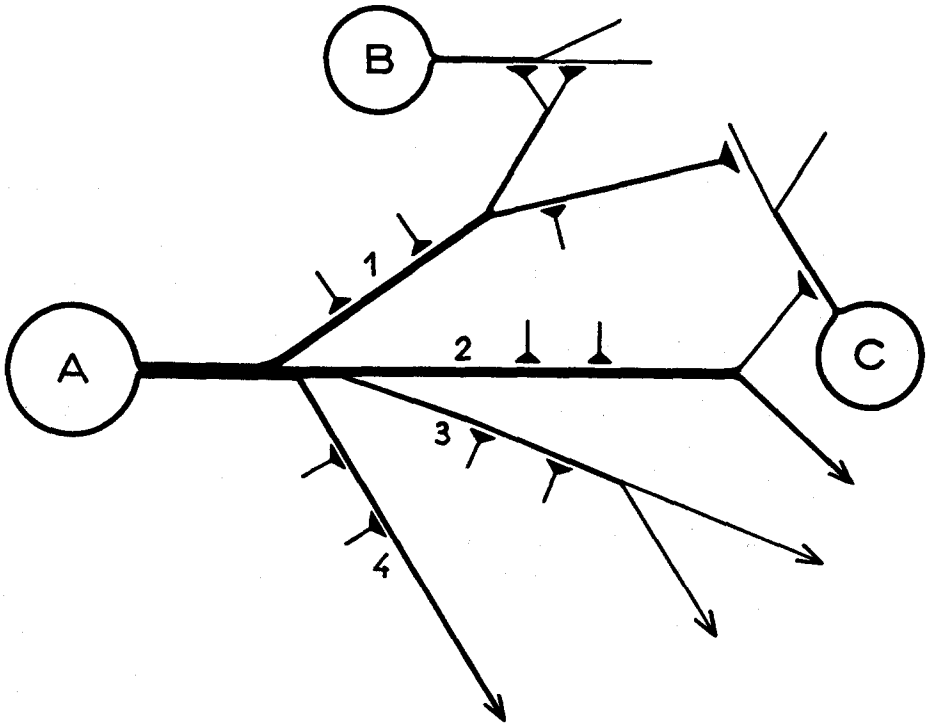

FIGURE 9. Schematic representation of a gastropod central neuron (A) with its ramifying axonal process. The synaptic input on an axonal branch may induce an "all-or-nothing" response in a trigger zone found in each branch which would normally propagate towards the normal effectors which this branch innervates (cell B for instance if the spike originates in branch *1*), but other axonal branches would not necessarily be invaded unless their trigger regions are themselves facilitated through the synaptic endings in the vicinity. Because of the electrotonic effect of the axonal spike which amplifies the synaptic action, the response of a few branches would induce general invasion of the whole neuron. However, if a large branch (*2*) is excited in the first place, it may be able alone to transmit the spike to other axonal branches and to the soma. Further description in text.

It follows from this study that a molluscan nerve cell may assume the functions of several neurons if these are considered in their classical sense as units of nervous activity. Some indication has been given of the degree of independence which is possible between such subdivisions of a neuron's activity, which in turn indicates the different degrees of convergence which a given neuron may allow between the effects of its different afferent inputs. This suggests some saving in the number of neurons needed for the performance of the

functions of the central nervous system, but perhaps it should also be considered as an important mechanism furthering the integrative properties of this system.

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