Identification of Active Membrane Areas in the Giant Neuron of *Aplysia*

L. TAUC

From the Department of Zoology, University of California, Los Angeles, and the Centre d'Etudes de Physiologie nerveuse du Centre National de la Recherche Scientifique, Paris, France

ABSTRACT Intracellular and extracellular potentials were simultaneously recorded from the soma and different parts of the axon of the giant cell of *Aplysia*. Evidence was obtained that for all modes of stimulation the spike originates in the axon at some distance from the cell body. The conduction of the spike is blocked at a distance of 200 to 300 μ from the soma for the antidromic spike, closer to the soma for an orthodromic spike. This event is recorded in the soma as a small or A spike. After some delay, a spike is initiated in the resting part of the axon and in the axon hillock; the soma is invaded only afterwards. The response of these three parts of the neuron is recorded in the soma as the big or S spike.

It was shown in the previous paper (17) that in the giant neuron of *Aplysia* (GN) a two stage invasion of the spike must be taken into consideration: the conduction of the impulse, first initiated in the axon at a relatively great distance from the cell body (about 1.5 mm), is blocked in a transitional zone in the vicinity of the soma; the latter, having the highest threshold of the whole neuron, discharges after a variable delay. Thus the membrane excitability appears to be different in different regions of the cell. This non-uniformity has recently been described in a number of preparations (1–8).

However, the method of intracellular recording employed for our previous study (17) did not permit a precise differentiation of active and inactive zones in the proximity of the cell body. Indeed the impedance of the neuronal membrane is extremely high compared with the resistance of the cytoplasm, so that practically identical potential variations are recorded in an active zone and in a non-active contiguous zone, coupled by a small resistance, as is the case of the soma and axon hillock (7, 12). To avoid the averaging process of electrotonic spread, external electrodes closely applied to the membrane are useful as their polarity in relation to a distant electrode is significant. It is

generally accepted that electrical activity is associated with an inward current flow which produces, under ohmic conditions, a negativity of the proximal extracellular electrode. On the contrary, external positivity denotes that the underlying membrane is either not active or active, but generating in the latter case a smaller EMF than some other proximal active membrane. In the present investigation, simultaneous extracellular and intracellular potentials recorded from the *Aplysia* giant neuron have been analyzed on the basis indicated above, and it has been shown that the somatic membrane and that of the intermediary axonal region are invaded by the spikes; it was also confirmed that the proximal axonal and axon hillock regions have a lower threshold than the soma and that nerve impulses originate in the axon at some distance from the cell body.

METHODS

The research was performed on the molluscs *Aplysia depilans* and *Aplysia californica*. The abdominal ganglion with two pleurovisceral connectives was dissected free from the animal and the collagenous protective membrane covering the giant cell removed. The preparation was immersed in sea water and the intracellular potentials recorded with capillary microelectrodes, simple or double, following the techniques previously described (13). Extracellular recordings were made with capillary microelectrodes having a tip diameter of 2 to 5 μ and filled with sea water to avoid any action on the membrane by the fluid diffusing from the electrode. The microelectrodes were brought into close contact with the soma under visual control; but by reason of the presence of a protective membranous capsule covering the soma a distance of a few microns was always present between the tip of the electrode and the excitable membrane. The presence of this gap explains the fact that the value of the recorded extracellular potentials never has an amplitude greater than a few hundred microvolts. The good contact of the extracellular electrode on the axon, which is covered by other neuronal structures, was indicated by the amplitude of the response in relation to the potentials recorded simultaneously in the soma. The localization of the axon process in the neuropile was known from the previous work (17).

For geometrical reasons, the extrasomatic recordings were free of any interference from the activity of other neurons. To avoid this interference in the extraaxonal recordings, the excitable structures in the proximity of the axon were dissected and destroyed; however, the experiments have shown that this precaution is not necessary if the extraaxonal electrode is placed at a distance not greater than 200 or 300 μ from the soma.

In all figures, for both intracellular and extracellular recordings, positivity is indicated by an upward deflection.

RESULTS

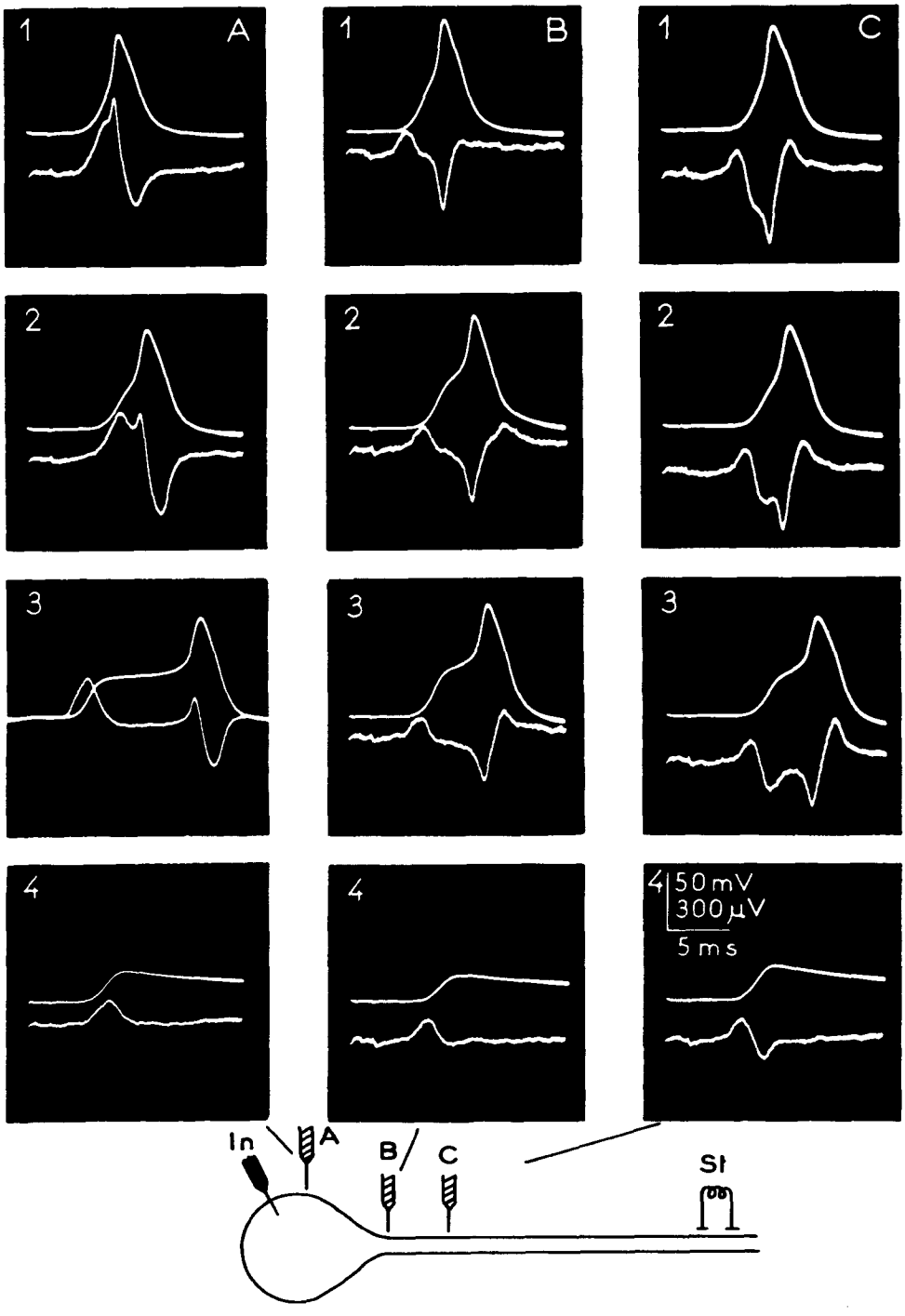
1. *Antidromic Stimulation*

An antidromically evoked spike recorded in the soma shows an inflection on the rising phase (Fig. 1 A, *1*); under fatigue or imposed hyperpolarization, the inflection becomes more prominent or the spike fails to develop, thus leaving a small potential of 20 to 30 mv in amplitude (Fig. 1 A, *4*). It is now known (16, 17) that the small potential or A spike alone indicates the blockage of the impulse in the axon at some distance from the soma, whereas the big spike or S spike represents the response of the somatic region, occurring with some delay. During the A–S interval, local responses develop under the electrotonic influence of the A spike.

The extracellular potential recorded on the soma (Figs. 1 A and 2 A) shows several phases. First appears a positive deflection preceding slightly the intracellular A spike. When the A–S interval is increased the positive inflection is isolated; postivity indicates the discharge of a distant membrane, undoubtedly that of the axon as demonstrated in Fig. 2 B. Here intraaxonal and extrasomatic potentials are compared and show the simultaneity of the A spike with the first positive extracellular deflection (see reference 17).

A second extracellular positive deflection and the following negative phase are connected with the S spike, as is seen from Fig. 1 A, *3* where the A–S interval is long, and from Fig. 1 A, *4* where the positive-negative complex is absent. It seems therefore that during the intracellular S spike, the somatic membrane, before being itself activated, serves as a source for another active membrane. It may be demonstrated that the active membrane whose activity precedes the somatic discharge, is that of the axon hillock and of the proximal axonal region not activated during the A spike. When the extracellular potential is recorded on the axon hillock (Fig. 1 B) or on the proximal axon at 200 μ from the cell body (Fig. 1 C), the potential during the S spike is first negative in both positions (best seen in Fig. 1 B, *3*, 1 C, *3*). The negativity suggests a simultaneous response of these regions at the time corresponding in the intrasomatic record to the beginning of the spike. The subsequent extracellular positivity signals the somatic discharge.

It may be seen in Fig. 1 B that the extracellular potential on the axon hillock corresponding to the A spike is positive, the same as on the extrasomatic records (Fig. 1 A). However, this positive potential is followed at 200 μ from the soma by a negative phase, indicating the proximity of the membrane activated during the A spike. At 300 μ from the soma (Figs. 3 A and 5 C, *3*, *4*) this negative phase is large and shows clearly that in this position the membrane is active at the time when the A spike is recorded in the soma. If the A–S interval is brief, the S spike gives at this place only a very small extracellular potential (Fig. 3 A, *1*), increasing with the A–S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interval. It may be found surprising that the polarity of the deflection is negative, meaning that the membrane is activated both during A and S spikes. But it was shown previously (17, 16) that when the A–S interval exceeds the absolute refractory period of the axonal region discharging during the antidromic A spike, the latter is reactivated by the somatic discharge and an efferent spike originates in the axon. It is then natural that the extra-axonal record should be negative, as apparently the activation of the axon

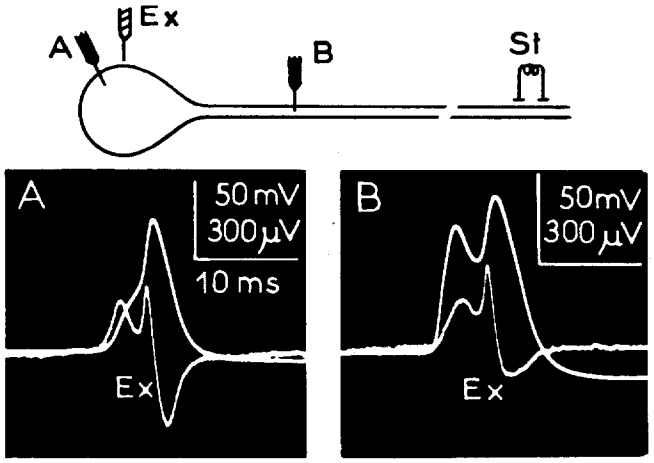

FIGURE 2. Spike potentials evoked in the giant cell by antidromic stimulation. Upper records give the intracellular potentials recorded in A in the soma, in B in the axon at 400 μ from the soma. Lower records in both A and B represent the extracellular potentials recorded on the somatic membrane (electrode *Ex*). The double intracellular positive deflection in B represents the sequence of axonal and somatic discharges (see previous paper). Note in B the simultaneity of the A spike with the first positive extracellular deflection.

hillock and of the proximal axonal membrane during the rising phase of the intrasomatic S spike gives no positive potential (Fig. 3 A, *1*).

It is interesting to analyze the influence of the somatic discharge on the extraaxonal potential as a function of the A–S interval. This distant activation of the somatic membrane is evidenced by a positive extracellular potential only when the A–S interval is very brief (Fig. 5 C, *3*) or when it is long

FIGURE 1. Simultaneous intracellular and extracellular recordings of the antidromically evoked spike of the same giant cell. All upper records represent the activity recorded in the soma by the electrode *In*, whereas the lower records give the extracellular potentials recorded in A on the soma, in B on the axon hillock, and in C on the axon at 200 μ from the soma (see schema). From *1* to *3* A–S interval increasing because of fatigue. In *4* the somatic discharge fails to develop. Further description in text.

In this and the follcwing figures positivity is indicated by an upward deflection for all records.

(Fig. 3 A, *3–5*). For a brief A–S interval no active changes occur in the axon during the somatic discharge and a positive deflection is recorded (Fig. 5 C, *3*). When the somatic spike appears with a delay of 7 msec. as in Fig. 3 A, *1*, active membrane changes develop in the axon during the S spike. The beginning of the negative extraaxonal deflection follows only slightly the beginning of the somatic discharge (which as was demonstrated above follows the begin-

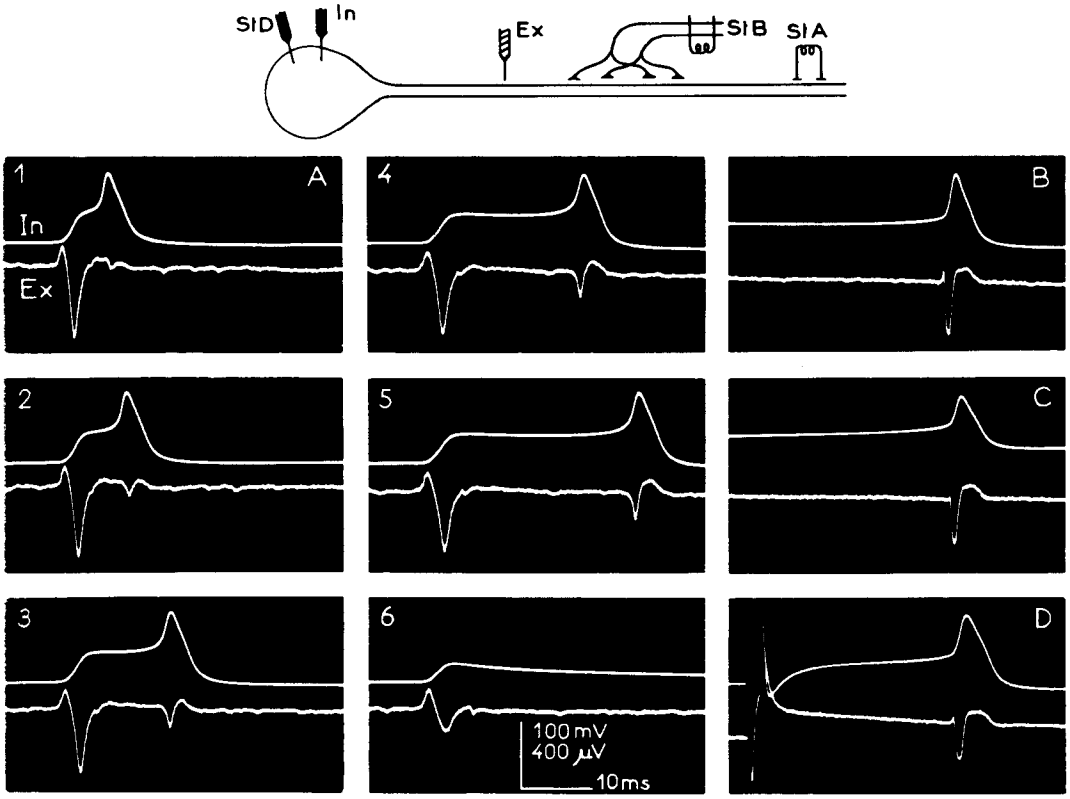

FIGURE 3. Simultaneous intrasomatic (*In*) and extraaxonal (*Ex*, at 300 μ from the soma) recordings of the same giant cell stimulated antidromically in A, synaptically in B, directly in D, and during spontaneous activity in C. In A, from *1* to *5*, A–S interval increasing because of fatigue; in *6* the soma is not invaded. Further description in text.

ning of the S spike recorded in the soma representing a compound response of the axon hillock and the soma). For an A–S interval of 9 msec. (Fig. 3A, *2*), the negative extraaxonal wave is simultaneous with the somatic discharge; thus the axon is reactivated by the discharge of the proximal axon and axon hillock and not by that of the soma. Since both phenomena occur at almost the same time, the positive extracellular sign of the distant somatic activity is depressed by the activity of the underlying membrane.

The axonal discharge is even simultaneous with that of the proximal axon and axon hillock when the A–S interval is greater than 20 msec.: in Fig. 3 A, *5*, the extraaxonal negative deflection starts at the same time as the intrasomatic S spike, the beginning of which, as we have seen above, is precisely simultaneous with the activation of the proximal axon and axon hillock.

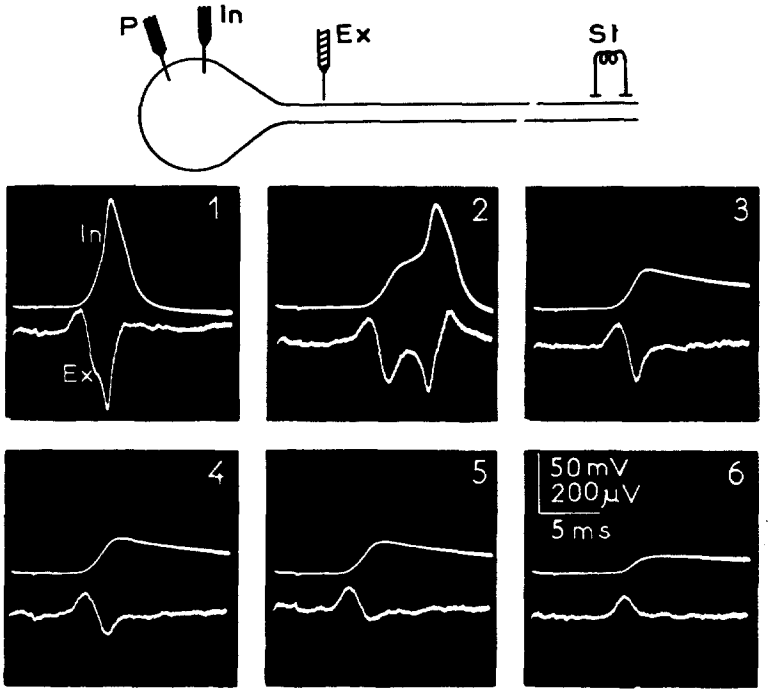

FIGURE 4. Antidromically evoked activity recorded simultaneously with an intracellular electrode (*In*) in the soma and extracellularly (electrode *Ex*) on the axon at 200 μ from the soma. From *1* to *6* the membrane polarization was gradually increased by hyperpolarizing current through P. Note on the extracellular records the progressive diminution of the negative phase corresponding to the A spike.

This second axonal response originates under the electrotonic influence of the depolarization level maintained in the non-activated S membrane by subliminal local responses induced by the A spike. It must therefore be assumed that the axonal A spike membrane has a higher excitability than the S spike membrane. This confirms previous conclusions from intracellular recordings (17). It may also be seen in Fig. 3 A, *5* that with this change in time sequence a clear positive deflection appears on the extraaxonal record, indicating the activation of the somatic membrane and perhaps also the reactivation of the distant axonal process.

The reactivation of the A spike membrane is also certainly responsible for

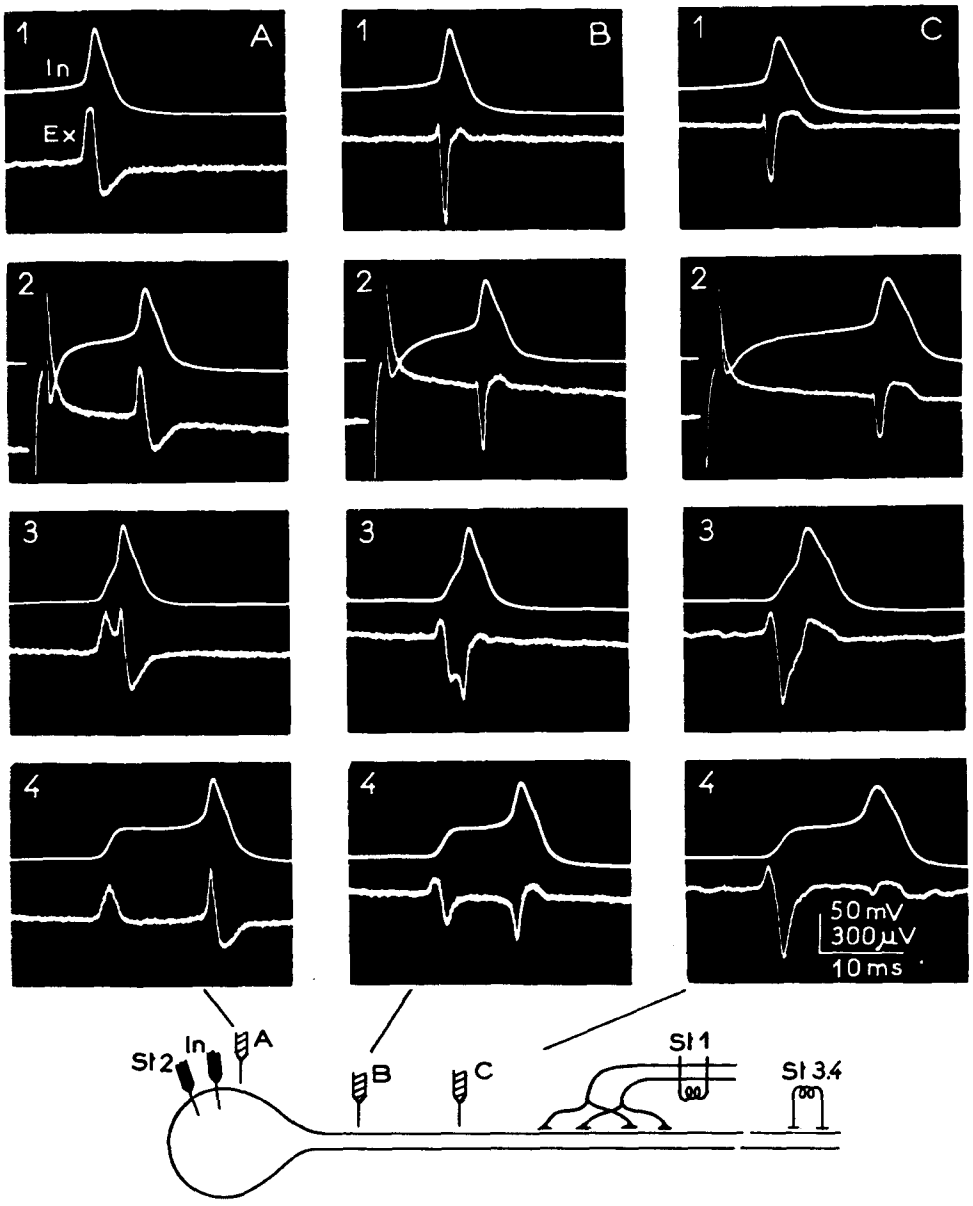

FIGURE 5. Simultaneous intracellular (*In*) and extracellular (*Ex*) recordings of spikes evoked in the same giant cell by different modes of stimulation: synaptically in *1*, directly in *2*, and antidromically in *3* and *4*. In all records, the intracellular electrode is located in the soma, but the extracellular potentials are recorded in A on the soma, in B on the axon at about 200 μ from the soma, and in C on the axon at about 300 μ from the soma (see schema). Further description in text.

the increase in the amplitude of the positive deflection recorded on the soma during the S spike when the A–S interval is long (compare Fig. 5 A, *3* and 5 A, *4*). The currents coming to the axon hillock and proximal axon membranes will be strengthened by the currents to the simultaneously reactivated A spike membrane, thus increasing the positivity recorded on the not yet active somatic membrane.

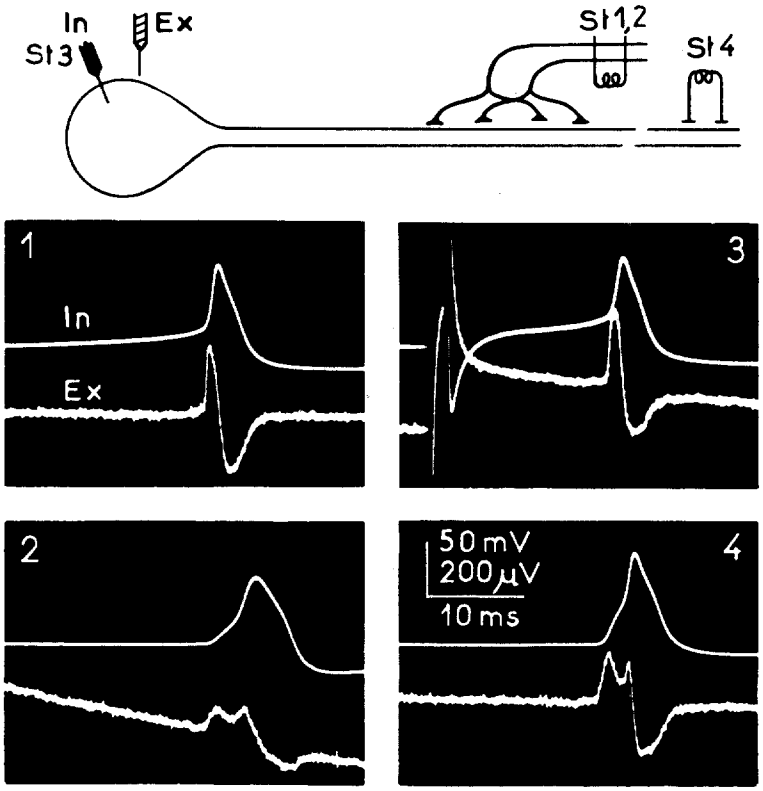

FIGURE 6. Simultaneous intrasomatic (*In*) and extrasomatic (*Ex*) recordings of spikes evoked in the same giant cell synaptically in *1* and *2*, directly in *3*, and antidromically in *4*. The synaptic spike in *2* was obtained under fatigue; note the dissociation of the extracellular positive wave into two components and compare with antidromic spike in *4*.

It may be shown that there is a relation between the duration of the A–S interval and the distance from the soma at which the conducted spike is blocked. The evidence derives not only from differences in the amplitude of the A spike recorded in the soma (Fig. 4, upper records, also reference 17), but also from a progressive diminution or disappearance of the negative extracellular A spike component, recorded on the axon at 200 μ from the soma, when the excitability has been decreased by artificially applied hyper-

polarizing current (Fig. 4, lower records). Consequently the blocking of the conducted spike in the axon does not occur in a fixed region, but is functionally determined by momentary conditions of membrane excitability (see reference 17).

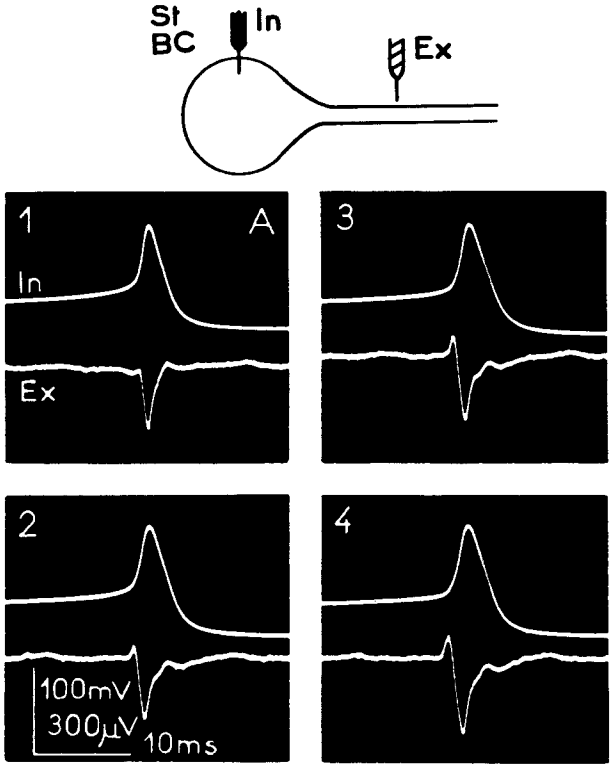

FIGURE 7. Simultaneous intrasomatic (*In*) and extraaxonal (*Ex*, at about 200 μ from the soma) recordings of spikes during rhythmic activity induced by a depolarizing current applied to the soma through the recording electrode *In*. In *1* the first spike, in *2* the 5th, in *3* the 10th, and in *4* the 16th and final spike. Note on the extracellular records the progressive appearance of a positive initial wave indicating a displacement of the trigger zone.

2. *Orthodromic Activation*

When the neuron is activated orthodromically, the membrane excitability all over the ganglionic part of the neuron is increased by the EPSP's, so that the A spike propagates very nearly to the soma and the A–S interval is brief or virtually non-existent, and thus usually no inflection will be observed on the rising phase of the intrasomatic spike (Fig. 5, *1*, and Fig. 6, *1*). The extracellular potential recorded on the soma shows only a simple positive deflection, probably corresponding to the activation of both axon and axon hillock mem-

branes, followed by a normal negative wave indicating the somatic discharge During fatigue, it is possible to obtain the dissociation of the positive deflection into two components (Fig. 6, *2*), so that the extrasomatic record becomes

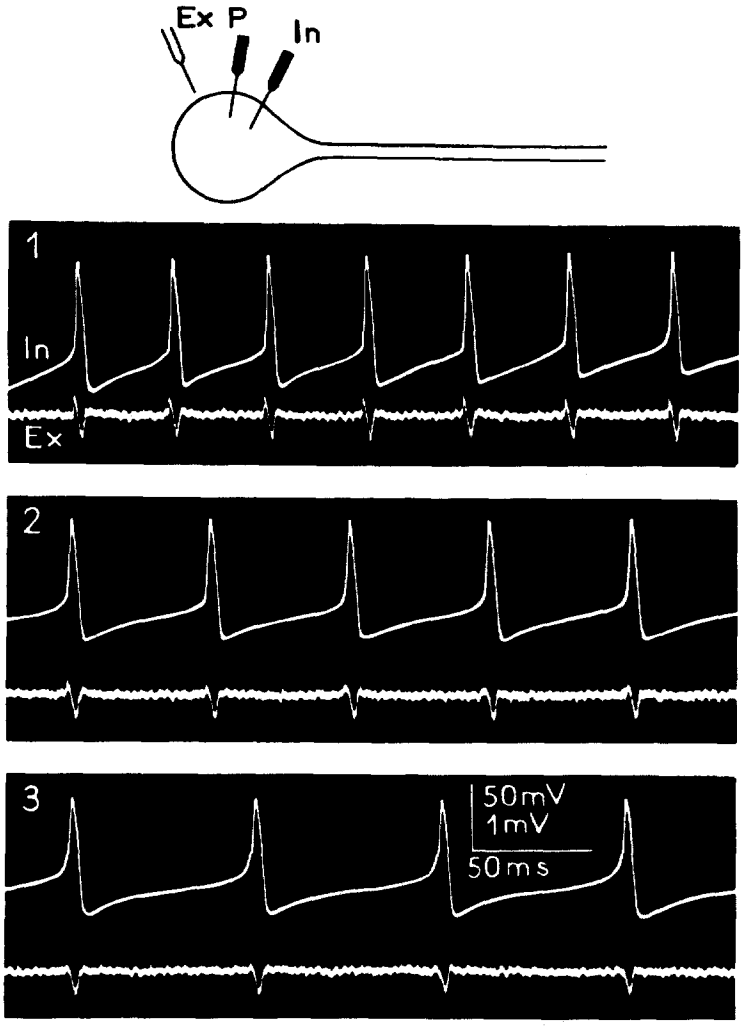

FIGURE 8. Simultaneous intrasomatic (*In*) and extrasomatic (*Ex*) recording of a rhythmic activity induced by a strong depolarizing current applied through electrode P. The record is not continuous and a gap of 20 sec. exists between *1* and *2* and *2* and *3*. Note, from *1* to *3*, the progressive modification of the extracellular potential from a positive-negative wave to a purely negative deflection.

similar to that obtained by antidromic stimulation (Fig. 6, *4*). The amplitude of the positive extracellular deflection accompanying the orthodromic spike is also comparable to that of an antidromically initiated S spike appearing

after a long A–S interval (Fig. 5 A, compare *1* and *4*) when it is accompanied by the A spike membrane reactivation as well as the activation of the proximal axon and axon hillock.

The extracellular records obtained from the axon at different distances from the soma (Fig. 3 B, and Fig. 5 B, *1*; C *1*) point to a rapid activation of

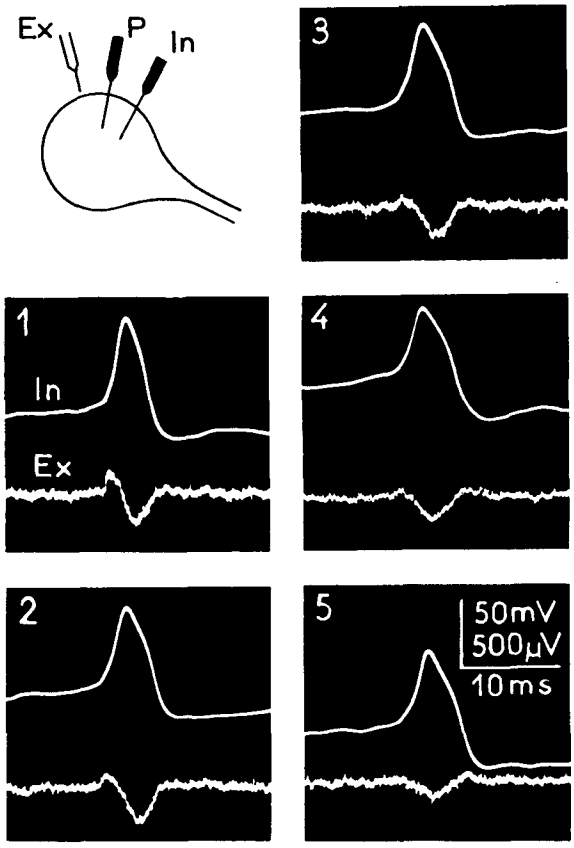

FIGURE 9. Simultaneous intrasomatic (*In*) and extrasomatic (*Ex*) recordings of spikes resulting from a rhythmic activity induced as in Fig. 8 by a strong depolarizing current applied through electrode P. Spikes from *1* to *5* were chosen with an approximate interval of 10 sec. Note, as in Fig. 8, the modifications of the extracellular potential during activity.

these regions; however, a small positive deflection preceding the large negative wave suggests a more distant localization of the membrane originating the spike.

3. *Direct Stimulation and Spontaneous Activity*

The extracellular potentials corresponding to a spike evoked by direct stimulation or appearing spontaneously (Figs. 3 C, D; 5, *2*; 6, *3*; 7) are similar to

those obtained by orthodromic stimulation. Their polarity indicates that the spike is normally initiated in the axon, but the region of origin of spike may be displaced under different conditions of excitability. When rhythmic activity is induced by a depolarizing current applied to the soma, the extent of the trigger zone varies with time. Fig. 7 shows simultaneously recorded intrasomatic and extraaxonal spikes taken in the beginning, during, and at the end of such a train of activity. It may be seen clearly that while the form of the intracellular spike is virtually the same in all records, the extracellular potential changes in a significant manner. A progressive evolution of a positive initial deflection, absent for the first spike, implies a progressive movement of the trigger zone away from the regions where the extracellular potential is recorded, probably by reason of accommodative properties of the membrane. The initial location of the trigger zone in the proximal axon is favored by the electrotonic gradient and is undoubtedly shifted further into the axon, as has already been demonstrated by intracellular recordings (17). A similar transfer or shrinking of the region of spike origin is observed for a directly evoked spike, which is initiated close to the soma, when the stimulus is strong, but is initiated further away by weaker stimuli.

Very different results are obtained when the depolarizing current applied through the intrasomatic electrode is strong, inducing a rhythmic activity of relatively high frequency. Under these conditions, while the form of the intrasomatic spike is only slightly affected, the extracellular potential recorded close to the somatic membrane is greatly modified. The initial positive deflection which is present at the beginning of the activity (Fig. 8, *1*; Fig. 9, *1*) is like that of a normal spike (see for instance Fig. 6, *1*, *3*). This positive phase decreases progressively (Fig. 8, *2*; Fig. 9, *2*, *4*), and disappears, and after some time even becomes negative (Fig. 8, *3*; Fig. 9, *5*). The negative wave appearing simultaneously with the intrasomatic spike (Fig. 9, *5*) indicates either an enlargement of the trigger zone which now includes the soma or a transfer of the trigger zone into the soma. The positive deflection which develops after the initial negative wave (Fig. 9, *5*), never observed before (Fig. 9, *1–4*), may indicate propagation out of the soma into the axon.

DISCUSSION

The interpretation of our results is based on the principle which considers the extracellular negativity to be a sign of the activity of the underlying membrane, while during a positive deflection the membrane would be passive and the recorded potential would indicate activity at a distance. We think the concept applicable to our results as we know the position of the electrode in relation to the membrane. It was observed that the resistance between the tip of the extracellular electrode and the bathing fluid containing the indiffer-

ent electrode remains very small as the electrode is placed in position for recording. It may be presumed that the electrode is held at a short distance from the active membrane by the protective layer surrounding these cells and that this layer has a high conductivity. In addition the ability to see the cell directly permits one to avoid dimpling the membrane by the electrode. It is possible that such dimpling is responsible for the giant extracellular spikes observed on motor horn cells by Freygang and Frank (7). Such spikes were never observed in the present study.

It may be noted that the evolution of the extracellular potentials in the vicinity of the soma (for example in Fig. 2 A) is similar to that to be expected if the intrasomatic spikes were transformed by an appropriate capacity-resistance network (equivalent circuit of Freygang and Frank). It seems, however, that this is a coincidence and results from the timing of the reactivation of different parts of the neuron. The most important evidence for this assumption is the modification of the extracellular spike when a strong depolarizing current is applied to the soma (Figs. 8 and 9). The extracellular negative wave cannot be reproduced by applying the intracellular spike to the equivalent circuit for the resting membrane and therefore does not result from passive properties of the somatic membrane. Consequently we conclude that the somatic membrane is active; this is not surprising if we remember that the soma deprived of its axon is still directly excitable (17).

The axonal A spike activity gives a relatively large positive deflection on the extrasomatic record, whereas the somatic discharge causes relatively smaller potential changes on the axonal records (Fig. 5). This may be due to the fact that because of the geometrical arrangement of the cell, the axon provides only relatively weak recovery currents to the soma (see Lorente De Nó, 1947).

The following is a synthesis of the present results with those described in the preceding paper concerned with the origins of the spike (17). In Fig. 10, the *Aplysia* neuron is divided into five sections, I to V, each responsible for a part of the extracellular potential recorded on the soma during the spike. An antidromic spike was selected as an example by reason of the clear dissociation of A and S elements; it was shown above (Fig. 6, *2*) and in the preceding paper (17) that in conditions of decreased excitability the orthodromic and antidromic responses have similar form for both intra- and extracellular recordings.

The orthodromically evoked spike is initiated in zone IV which has the lowest threshold of the whole neuron. This zone is placed at 1.5 mm from the soma (17). The spike then propagates normally in the orthodromic direction in zone V, while its conduction in zone III may be stopped because of the excessive drop of the safety factor resulting from an increase in threshold in this region and also from the decrease of the spike amplitude due to heavy

loading by the not yet excited soma. The spike is blocked at a distance from the soma which depends upon the momentary level of excitability of the neuronal membrane. The antidromic A spike normally progresses to a distance of 200 to 300 μ from the cell body, whereas the conduction of the orthodromic spike may be blocked at the limit of the axon hillock. Under the electrotonic influence of the A spike, local response evolves in the S membrane, composed of the non-excited part of zones II and I.

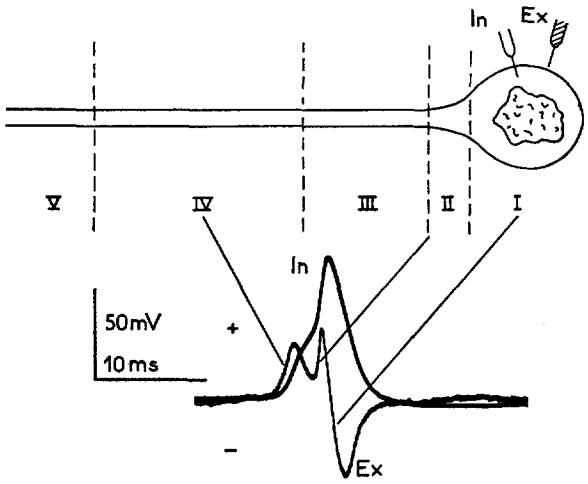

FIGURE 10. The regions of the giant nerve cell corresponding to different components of the antidromic spike recorded simultaneously from the soma with intracellular (*In*) and extracellular (*Ex*) electrodes. I, soma; II, axon hillock; III, transitional zone; IV, normal site of origin of spike; V, distant axon. Further description in text.

After some delay a new spike is first initiated in the resting part of zone III and in II; the soma discharges only afterward, having undoubtedly a very high threshold.

The A–S interval depends also upon the momentary excitability of the neuron; it is brief and sometimes virtually non-existent for an orthodromic spike, whereas it may last several tens of milliseconds for an antidromic spike. The delayed spike may reactivate the A spike membrane and give origin to a second efferent spike.

Zone III represents an intermediary region between the highly excitable membrane initiating the spike and the somatic membrane with high threshold. The variability of the site where the conducted spike is blocked points to an absence of any fixed barrier. Possibly no relation exists between the properties of excitability and the histological structure of the neuron. The differences may be functional; it was shown for different membranes (in Grundfest, 1958, also Gerschenfeld and Tauc, 1961), that electrophysiological

as well as pharmacological differences do not imply observable differences in the structure.

The results of our study indicate that in the *Aplysia* neuron the whole membrane is electrically excitable; however, an "all or nothing" response does not inevitably invade the soma. This may happen in an antidromically stimulated giant neuron (*e.g.* in Fig. 4), but other neurons were found in the same ganglion whose somata, although excitable, were not always invaded by an orthodromic spike initiated in the axonal ramifications. These so initiated A spikes were called by us pseudospikes (14, 15). It was demonstrated by Tauc and Hughes (18) that without facilitation the propagation of a pseudospike is blocked not only towards the soma, but also in the direction of other branches. Yet it propagates normally toward the periphery in the branch where it is initiated. Therefore some of the *Aplysia* neurons are able to initiate a partial response conveyed only to a fraction of the effectors which the neuron innervates. In the light of present results, this behavior may be attributed to the differences in threshold of different parts of the neuron and also to the geometrical conditions which determine the intensity of the depressing recovery currents, both of these factors influencing the safety factor for spike propagation.

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